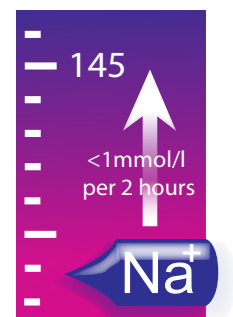
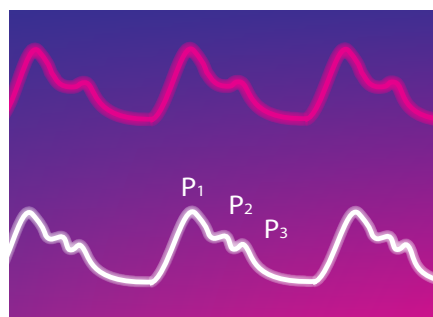
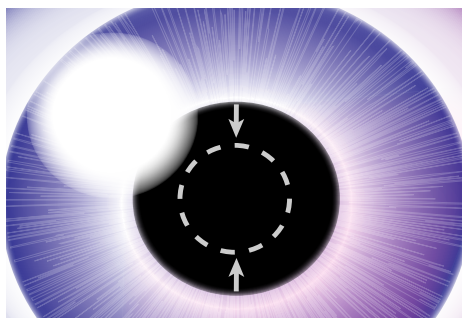


Wessex Neuro ICU

# Guidelines 2014

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# **Wessex Neuro ICU**

# **Guidelines**

# **2014**

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Wessex Neuro ICU

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# Introduction

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These guidelines represent the current policies and practice of The Wessex Neuro ICU. They have been designed to act as a framework and guidance for the management of commonly encountered situations in our daily work. No attempt has been made to reference any of the information provided, as much of it has developed as a consensus of management, heavily influenced by discussion and reflection at our monthly morbidity and mortality meetings.

This is a practical guide which concentrates on the most basic aspects of everyday care of the whole patient, aiming to ensure that management is consistent from day to day. Where possible we have tried to provide brief explanations as to why a particular treatment is recommended, eg explaining why a patient with a high spinal cord injury will ventilate better lying flat rather than sat or tilted head up. In every situation where there remains doubt about the best course of management for an individual patient, specialist advice should be sought.

There will be times when it is entirely appropriate to deviate from these guidelines to individualise management to the patient, their condition, and their circumstances. A guiding principle must always be to seek to return the patient to a quality of life that they would find acceptable. This may not have been achieved by time of discharge from the unit, and will require the support of their local hospital team, rehabilitation and community services. Where this is not deemed possible, we endeavour to ensure that a plan is in place for the timing and institution of palliative care. Fortunately the regular visits by patients who have regained their quality of life following serious brain injuries, act as a constant source of inspiration to the whole team.

We recognise the importance and influence of Professor Menon's work developing protocols for Addenbrooke's Neurocritical Care Unit. These formed the foundations for the protocol book that Dr Eynon developed with colleagues, which helped unify management on Wessex Neuro ICU. We are keen to acknowledge all those who have contributed to these historical documents, and in addition are grateful to all of our colleagues who have assisted in the development of these new guidelines. However, we accept responsibility for any inaccuracies in the text and will happily correct anything that is not accurate or appropriate.

Matthew Cordingly & John Hell

June 2013



# Patient care on Neuro ICU

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## Introduction

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Responsibilities for the Neurosciences Intensive Care Unit (Neuro ICU) team include: the day to day management of all aspects of patient care for the duration of their time on the unit, organising diagnostic and screening tests, and communicating with patients and relatives. A key role of the Neuro ICU team is the co-ordination of input from neuroscience and non-neuroscience specialist teams (e.g. neurosurgery, neurology, microbiology, orthopaedics etc).

The admitting specialty team is responsible for specific management of the underlying condition and, in the case of neurosurgeons, for placement and maintenance of intracranial monitoring devices and external ventricular drains.

To maintain efficient and effective shared care, where possible there should be daily communication between the specialist team members and the Neuro ICU team. Ideally this will take the form of a joint review of the patient by members of the specialty team and the Neuro ICU team. All decisions made **MUST** be clearly documented in the patients notes, including time and date.

It is acceptable for any member of the treating teams to prescribe medication for the patients, however, all changes must be discussed with the Neuro ICU team.

This document is intended to provide a framework for the care of patients on Neuro ICU. Although it is designed to guide management, there will be instances when it may be in the patient's best interests to deviate from these guidelines.

## Neuro ICU daily timetable

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### Weekdays

Time	Activity
<b>07.45 - 08.15</b>	Ward round (Neuro ICU consultant, Neuro ICU trainees, nurse in charge, nurse looking after patient & physios)
<b>08.15 - 11.00</b>	Daily review of all patients by Neuro ICU trainees.
<b>08.30 - 09.30</b>	Joint review by specialty team members where possible
<b>11.00 - 13.00</b>	Neuro ICU teaching ward round (Neuro ICU trainees, Neuro ICU consultant, nurse in charge & nurse looking after patient)
<b>16.00 - 16.30</b>	Hand-over ward round (Neuro ICU consultant, Neuro ICU trainees, duty neurosurgical trainee & senior nurse)
<b>22.00 (20:00 on Friday)</b>	Hand-over ward between the late Neuro ICU trainee and the night Neuro ICU trainee
<b>22.30 - 07.30</b>	All patients should have a night review documented in the notes

### Saturday / Sunday

Time	Activity
<b>08.00 - 08.30</b>	Hand-over ward round between Neuro ICU trainees and senior nurse.
	The duty Neuro ICU Consultant will conduct a ward round during the course of the day
<b>20.00</b>	Hand-over ward round to night time Neuro ICU trainee

Neurosciences patients on the general intensive care unit will be reviewed at the end of the Neuro ICU ward round when appropriate.

# Neuro ICU admission and discharge

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# 3

## Admission guidelines

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The nurse in charge and the Neuro ICU consultant covering the unit must be fully informed of, and agree to, the admission of all patients.

All patients that require Level 2 or Level 3 care with a primary neurosurgical or neurological condition should be considered for admission. All firms within Wessex Neurological Centre have equal right of access. Neuro ICU operates as a part of critical care, allowing nursing staff to be deployed flexibly to manage surges in demand. Any patient that cannot be accommodated on Neuro ICU and has consequently been admitted to GICU, but is under the care of a specialist neurosciences team, should be transferred to Neuro ICU at the earliest possible opportunity. This may require cancelling elective post-operative admissions.

All elective admissions must be booked. Unfortunately, this cannot guarantee the availability of a bed. Prior to starting any neurosurgical procedure that would require admission, the availability of a bed must be confirmed.

## Levels of care

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0	Patients whose needs can be met through normal ward care in an acute hospital
1	Patients at risk of deteriorating, or those recently re-located from higher levels of care, whose needs can be met on an acute ward with additional advice and support from the critical care team
2	Non-invasive ventilation, $\text{FiO}_2 \geq 0.6$ , suctioning 2 hourly or more frequently Invasive haemodynamic monitoring, single vasoactive agent, support for a single failing organ
3	Invasive ventilation More than one vasoactive agent Patients requiring intensive nursing care (eg agitation or unstable spinal injury.)

## Admission criteria for major trauma patients

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Adequate resuscitation of all CNS injured patients is essential to maximise their chance of survival and recovery. The avoidance of secondary brain injury is the most important aspect of management of all major trauma patients with CNS involvement.

All patients with major trauma and CNS injury that could benefit from specialist neurosurgical and neurointensive care are eligible for admission, once they have been resuscitated to:

- Secure airway
- Adequate ventilation and treated pneumothorax (when present) with a chest drain
  - $\text{PaO}_2 \geq 13\text{kPa}$
  - $\text{PaCO}_2$  4.5-5.0kPa
- Cardiovascular stability with all haemorrhage controlled
  - $\text{MAP} \geq 90\text{mmHg}$
  - $\text{Hb} \geq 10\text{g/dl}$
  - Abnormal coagulation corrected

- 30° head up tilt, provided the mechanism of injury is known & unlikely to have caused injury to the thoracolumbar spine. Where this is not the case, tilt whole trolley or bed, rather than sitting patient up, until the thoracolumbar spine has been radiologically confirmed to have no acute injury.
- Seizures controlled
- Core temperature  $\geq 35^{\circ}$

Any deviation from these parameters requires full discussion with the Neuro ICU duty consultant before consideration of transfer for admission.

Any patient >75 years old with any significant co-morbidity and a motor score of 1 or 2, would be unlikely to benefit from specialist neurosurgical or neurointensive care management, so should be thoroughly discussed with the neurosurgeons prior to consideration of transfer for admission.

NB: Limitations to resuscitation in the Wessex Neurological Centre:

- No blood is stored within Wessex Neuro
- No rapid infusers are present
- No anaesthetists are present on Neuro ICU out-of-hours
- No operating theatre or recovery staff are present out-of-hours

## **Admission procedure for all patients**

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Appropriate spinal management of all patients with a history of trauma should be assessed at time of admission to unit. See spinal management chapter.

- All patients must be reviewed by a Neuro ICU fellow on admission to the unit
- Neuro ICU fellow should be available to take verbal handover from the transfer team
- Neuro ICU admission proforma must be fully completed
- Summary of admission history, past medical conditions etc
  - Handedness
  - Occupation
  - Functional status

- Full examination of patient, including secondary survey for all trauma patients. Secondary survey must include clear documentation of all abrasions/contusions etc (May be required by police/coroner)
- Neuro ICU prescription
  - All patients should be started on gut protection and laxatives.
  - Transcribe appropriate pre-existing medication (eg continue beta-blockers, anticonvulsants, antibiotics and steroids)
- Chest X-ray for all patients:
  - Expected to stay longer than 24hrs
  - Ventilated or with abnormal gas exchange
  - Following central line placement
- ECG reviewed and signed



Venous-thromboembolism form must be completed for all patients admitted to Neuro ICU. All patients must have anti-embolic stockings (AES) and intermittent pneumatic compression (IPC) devices unless contra-indicated.

- Ensure full set of bloods including FBC, U&E, coagulation screen and group & save.
- Arterial blood gas if arterial line present or appropriate.
- All patients should be reviewed by the Neuro ICU consultant within 12 hours of admission to the unit. This must be documented clearly to ensure an appropriate management plan is in place.

## Discharge criteria

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All patients with major trauma and CNS injury that no longer need specialist neuro-surgical and neurointensive care are appropriate for discharge, once they are stable.

Transfer to ward care if:

- Maintained airway
- Tracheostomy acceptable once established and adequately secured
- Requiring chest physiotherapy and suctioning > 2 hrly
- $\text{FiO}_2 < 0.6$  with  $\text{SpO}_2 \geq 95\%$  for 24 hrs

- Haemodynamically stable off inotropes / vasopressors with urine output > 0.5ml/kg/hr
- GCS stable > 8 and protecting clear airway
- Agitation controlled with oral medication and manageable with a single appropriate healthcare professional
- Spinally injured patient with (stable or operatively stabilised) injury below C5 and stable neurological deficit or no deficit
- Patient considered inappropriate for further critical care or escalation of current treatment
- Patient only for palliative care
- Pain controlled with oral analgesia or patient controlled analgesia (PCA)

### **Transfer to patient's local critical care facility**

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As soon as patients no longer require specialist neurosciences care (eg presence of external ventricular drain / requirement for plasma exchange), explicit consideration of the repatriation of the patient to their local hospital should occur daily.

Transfer to another Level 2 or Level 3 facility requires the patient to have been accepted under the care of an appropriate named consultant, bed available, and a suitably trained and qualified transfer team available to transfer the patient safely. Both Neuro ICU consultant and specialty consultant should be in agreement that transfer is appropriate at that time. Many neurosurgical patients require a CT Brain scan prior to transfer from definitive care.

### **Discharge summary**

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All patients must have a thorough and accurate discharge summary, which has been checked by the duty Neuro ICU consultant, prior to transfer out of Neuro ICU.

This should include:

- 1** Primary neurosurgical / neurological / stroke consultant
- 2** Primary diagnosis (eg subarachnoid haemorrhage / diffuse axonal injury)
- 3** Co-morbidities (eg diabetes mellitus, hypertension)
- 4** Summary of key events during admission, including any complications

- 5** Relevant investigations (eg CT scans, LP results)
- 6** Insertion dates of any invasive lines (eg arterial lines) / endotracheal tube
- 7** All microbiological results and treatment
- 8** Medication (with appropriate stop dates eg nimodipine at 21 days / aspirin at 6 weeks)
- 9** GCS and neurology on discharge with a plan for investigation if any deterioration occurs
- 10** Plan to include:
  - a** Duration of antimicrobial treatment and whether LP indicated as part of septic screen
  - b** Spinal management plan (eg cervical collar duration)
  - c** VTE mechanical prophylaxis (AES & IPCs), with start date for enoxaparin if not already commenced
  - d** Date for removal of sutures / staples
  - e** Nurse 30° / 45° head up or keep flat for patients with spinal cord injury (reliant on diaphragm excursion)
  - f** Timing of further imaging (eg CT Scan / MRI / cerebral angiogram)
  - g** Targets (see below)
- 11** Contact details for Neuro ICU & primary consultant



### Targets for TBI patients

Examples have been given below, these should be individualised on a patient by patient basis.

	Current	Target
<b>MAP</b>	95 mmHg	>80 mmHg
<b>PaO<sub>2</sub></b>	12.7 kPa	>12 kPa
<b>PaCO<sub>2</sub></b>	5.9 kPa	4.5 - 6.5 kPa
<b>Serum Na</b>	142 mmol/l	140-145 mmol/l
<b>Nutrition (140% normal)</b>	30 kCal/kg/24hrs	30 kCal/kg/24hrs
<b>Blood glucose</b>	6.8 mmol/l	4-8 mmol/l
<b>GCS</b>	E3 VT M5	If motor score drops 2 points CT Scan

### Targets for SAH patients

Examples have been given below, these should be individualised on a patient by patient basis.

	Current	Target
<b>MAP</b>	120 mmHg	100 - 140 mmHg ie do not start antihypertensive treatment within 6 weeks unless evidence of myocardial ischaemia or MAP >140mmHg
<b>PaO<sub>2</sub></b>	12.7 kPa	>12 kPa
<b>PaCO<sub>2</sub></b>	5.9 kPa	4.5 - 6.5 kPa
<b>Serum Na</b>	142 mmol/l	140-145 mmol/l
<b>Nutrition</b>	25 kCal/kg/24hrs	25 kCal/kg/24hrs
<b>Blood glucose</b>	6.8 mmol/l	4-8 mmol/l
<b>GCS</b>	E3 VT M5	If motor score drops 2 points CT Scan

# Patient assessment

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## Patient review

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The patient must be systematically reviewed, including:

- Full completion of admission or daily review proforma
- Patient should have full examination of all systems. We recommend that this examination should occur with the nurse present, both to ensure completeness (eg allowing the nurse to highlight any areas of concern found during turning the patient) and to act as a chaperone. During the examination there should be particular emphasis on:
  - Neurological examination: It is paramount that a baseline neurological level is determined daily, as rapid changes can occur (e.g. development of lateralizing signs with vasospasm, altered GCS with hydrocephalus, neurological deterioration with spinal injury.)
    - GCS including break down of score into Eyes (E), Voice (V), Motor(M)
    - Pupils
    - Examination of scalp (e.g. wound/drain site, sutures/clips, craniectomy site fullness - a patient with a tense craniectomy site requires urgent attention)
    - Cranial nerve examination appropriate to underlying condition
    - Visual fields (e.g. post pituitary surgery, ICH or CVA)
    - Eye movement to all four quadrants (e.g. may be abnormal with PCom aneurysm)
    - Facial symmetry / movement (e.g. eye closure post acoustic surgery - many patients have a 7th nerve palsy following acoustic surgery)
    - Gag / tongue movement (e.g. posterior fossa conditions)
    - Tone / power / sensation / reflexes within limits of patient co-operation. (ASIA score in patients with spinal cord injury.)
  - Cardiovascular examination

- Rate, rhythm, blood pressure & heart sounds
- Respiratory
  - Airway: adequacy and position of adjuncts (e.g. endotracheal tube length at teeth)
  - Chest signs including full auscultation of back and front of chest
  - Ventilatory support parameters
- Gastrointestinal
  - Full abdominal examination (e.g. distended, bowel sounds, wound sites)
  - Adequate nutrition including calorific intake
  - Bowels last open
- Genito-urinary
  - Hourly urine output for last 4 hours
  - Fluid balance
- Venous thromboembolism risk
  - Calf examination
  - Presence of anti-embolic stockings and functioning intermittent pneumatic compression devices.
- On admission, completion of secondary survey for all trauma patients
- Review and document in flow charts the current blood / CSF / microbiology results. Review transcranial doppler (TCD) results in patients post subarachnoid haemorrhage (increased TCD velocities may indicate a patient has vasospasm). Check any ECGs.
- Review of imaging including chest X-ray and CT/MRI scans
- Review of all prescriptions documenting any allergies and ensuring:
  - **All** drugs:
    - are currently appropriate (eg withholding of ACE inhibitor in vasospasm)
    - have been given at appropriate times
    - are at the appropriate dosage (eg weaning of steroids)
  - **No** drugs:
    - have been omitted inappropriately (eg steroids, anticonvulsants or Beta-blockers.)

- are contra-indicated (eg antiplatelet and anticoagulants in traumatic brain injury)
- All patients have appropriate gut protection prescribed
- Any patient that is not prescribed LMWH must have a plan documented in the notes with a timing for review / commencement.

## **Glasgow coma scale (GCS)**

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This is the standard method of assessing level of consciousness in neuroscience patients. Assessment must be performed in a reproducible manner by all staff to ensure consistency. This will allow important changes to be detected rapidly, triggering appropriate interventions in a timely manner.

### **Basic principles of assessment**

- Patient should be woken from sleep prior to assessment
- It is not possible to assess GCS in sedated and paralysed patients, although it is usually possible to follow a trend of responsiveness in patients that are sedated to a minimum level to tolerate an endotracheal tube.
- When assessing motor response to commands, such as asking the patient to squeeze fingers, they must both squeeze and release the examiner's fingers on command. Many patients will just grip objects placed in their hands as a reflex response.
- Response to painful stimulus should be to a standard central painful stimulus. This should be a trapezius pinch. This involves squeezing the trapezius muscle at the base of the neck, between thumb and fingers firmly enough to elicit pain in a normal individual.
- The use of sternal rub, supra-orbital nerve pressure or other potentially painful stimuli is not recommended because of the risk of leaving obvious marks on the patient.
- The use of peripheral painful stimuli (eg nail bed compression) should not be considered a part of GCS estimation, because it may elicit spinal reflexes that may appear to falsely increase the score.
- On occasion, an experienced member of staff may use a peripheral painful stimulus to ensure that a patient is able to move that limb. When this is done it should be by pressure against the side of a fingertip, NOT pressure over the nail bed. Pressure to the nail bed can result in obvious bruising.

- The scale is a range from 3 to 15, where 3 is the deepest level of coma, and 15 is a fully conscious and orientated patient. The total GCS score is less important than the breakdown of Eyes, Verbal and Motor Response.

E.g.

A patient may be fully conscious but unable to speak because of a lesion affecting Broca's area. This would give them a GCS of 11, but the information is more usefully expressed as E4V1M6

## Eyes

- 4** Eyes open spontaneously
- 3** Eyes open to voice
- 2** Eyes open to standard central painful stimulus
- 1** Eyes not open

## Verbal

- 5** Patient is fully orientated to time, place and person
- 4** Patient speaking in sentences, but not fully orientated
- 3** Monosyllabic words
- 2** Incomprehensible sounds
- 1** No sound

## Motor

- 6** Obeying commands (eg squeeze & release examiner's fingers, stick out tongue or wiggle toes)
- 5** Localising to central painful stimulus. The patient's upper limb must flex to bring the hand above the nipple line.
- 4** Normal flexion. The patient's upper limb must flex, without the wrist pronating.
- 3** Abnormal flexion. The patient's upper limb flexes, with the wrist pronating. This may be referred to as decorticate posturing.
- 2** Extension. The patient's upper limbs extend at the elbow, and the lower limbs extend fully. This may be referred to as decerebrate posturing.
- 1** No movement

Normal flexion (M4) is sometimes referred to as withdrawing. However, care must be taken when using this term as it implies a movement away from a peripheral painful stimulus. Peripheral painful stimulation is not part of standard GCS assessment.

Pitfalls in assessment of GCS:

- In locked in syndrome or patients with high spinal cord lesions, it is essential to open the eyelids and look for any eye movement
- Beware assessing GCS in deaf patients who may not respond to verbal command
- Dominant hemisphere lesions that affect eloquent cortex may prevent patients from obeying commands (receptive dysphasia)

## Assessment of pupils

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- The size and response of both pupils to light must be recorded accurately as part of every neurological assessment.
- The presence of a unilateral fixed and dilated pupil is most commonly an indication of significantly raised intracranial pressure from a lesion on that side of the head.



The presence of a single fixed and dilated pupil may occur from uncal herniation (eg temporal lobe swelling/ pressure from an acute subdural haematoma) in the absence of globally raised ICP.

- In the presence of severe trauma to the face or orbit, the pupil may be fixed and dilated as a result of local trauma to the nerve on that side, rather than an indication of raised intracranial pressure.
- Beware the patient that is already blind in one or both eyes, or has undergone previous ocular surgery. The presence of unequal sized pupils, or lack of reaction to light, may be normal for that patient, and not indicative of any new intracranial pathology.
- The presence of bilaterally fixed and dilated pupils is most commonly an indication of severely raised intracranial pressure with brainstem compression. Unless this situation is immediately reversed, haemorrhage or infarction within the brainstem and brainstem death will result. It is associated with greater than 95% mortality.
- Assessment of pupillary reaction may be difficult and painful in patients with severely swollen eyelids (eg following frontal craniotomy), and may be unnecessary if the patient is obeying commands, and vocalising. However, if the patient is otherwise unresponsive and the presence of fixed and dilated pupils is to be used to decide whether to operate or withdraw treatment, then strenuous attempts must be made to assess both pupils.
- Pupil reaction and size is not affected by neuromuscular junction blockers, which may be used to paralyse ventilated patients.

- The presence of a painful III<sup>rd</sup> nerve palsy, causing pupillary dilation, ptosis and loss of eye movement, should always be assumed to have been caused by a PCom aneurysm until formally excluded.

# Ventilation

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## Invasive ventilation

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### Endotracheal tube

Patients requiring ventilation on admission will usually have been intubated and ventilated at their local hospital, in the emergency department or in theatre.

It is essential to check:

- The type of endotracheal tube:
  - Standard COETT
  - Flexometallic (commonly referred to as 'reinforced tube', because of the presence of a metal spiral within the tube wall. The metal spiral reduces the chance of the tube becoming obstructed by 'kinking' of the tube in theatre)



Patients that are not fully anaesthetised may bite down onto flexometallic tubes, completely obstructing the lumen. The lumen will not then reopen when they release the pressure because of deformation of the metal spiral. The patient will not be able to be ventilated, suctioned, and bougie will not usually pass through the obstruction. They will therefore require emergency re-intubation. For this reason patients should **not** be admitted postoperatively without changing these tubes to a standard COETT.

- South facing RAE. These are preformed plastic tubes which may look similar to a standard tube. They are designed to angle downwards after leaving the mouth, rather than continuing the curvature of a standard tube. They are commonly used for anaesthesia during cerebral angiography and intervention.





It may be difficult to suction via RAE tubes and there are no length markings to indicate that the tube is placed to the correct depth. This risks the tube irritating the carina if it is too long for the patient, which will make weaning from ventilation impossible because of uncontrollable coughing. Consequently, patients should not be admitted postoperatively without changing these tubes to a standard COETT.

- COETT with subglottic suction port. These new tubes are increasingly being used by our referring hospitals, as they are reported to be associated with less aspiration on ICU. Unfortunately, currently they only have a single suction port above the cuff which frequently abuts the mucosa preventing aspiration of secretions. Gentle suctioning via the access port with a 10ml syringe may allow some secretions to be removed and reduce the chance of aspiration. NB It is essential not to aggressively suction since this risks damage to delicate mucosa within the larynx or trachea.
- Length of endotracheal tube, measured at teeth. (Tubes will often move during an admission, and therefore it is important to document their position regularly, which is performed on each shift by nursing staff.)
- Position of tip of endotracheal tube on chest X-Ray. This should be assessed as soon after admission as practical. The tip of the tube should be at the lower limit of the clavicular heads, 2cm above the carina. When correctly positioned on chest X-Ray, the length of the tube at the teeth should be noted.
- Cuff pressure must be assessed on admission, and is recorded by the nursing staff on each shift. In patients with poor lung compliance that require high levels of PEEP, it is essential that the cuff pressure exceeds the maximum inspiratory pressure by 5cmH<sub>2</sub>O.
  - Inadequate cuff pressure risks loss of ventilation and aspiration of potentially infected oral secretions.
  - Excessive cuff pressure (>40cmH<sub>2</sub>O) will cause ischaemia of the tracheal mucosa, and may lead to tracheal stenosis or tracheomalacia.
- The endotracheal tube is securely tied into position with a standard tube tie.
  - It is not necessary to tape tubes since the tie passes above any area where the internal jugular vein could be significantly compressed. Tapes are less secure and frequently result in allergic reactions on delicate facial tissue if left for any significant length of time.
- That there is a heat and moisture exchange (HME) filter in the circuit, until the patient is ventilated on a warmed humidified circuit, when it should be removed.

### THE STANDARD KNOT USED ON NEURO ICU

- 1** Fold the tape in half
- 2** Pass the folded end around the tube
- 3** Pass the two loose ends through the loop created and tighten the slip knot against the tube at the patients mouth. The knot should lie against the cranial aspect of the tube (i.e. the side of the tube closest to the patients nose)
- 4** Split the two loose ends of the tape and wrap each back around the tube and tie in a simple knot under the tube securely
- 5** Pass one of the free ends around the back of the patients neck and tie securely with the other end in a simple reef knot on the cheek.

### Initial ventilator settings

Initial ventilator settings for a sedated and paralysed patient should either be:

- The settings that the patient has been transferred on, so long as these are achieving appropriate oxygenation & ventilation parameters (see below).
- Or, we recommend:
  - Mode : PCV-VG: This mode targets the set tidal volume whilst minimising peak airway pressures.
  - $\text{FiO}_2$ : 0.5 (unless patient severely hypoxic)
  - PEEP: 5cmH<sub>2</sub>O
  - Tidal Volume : 6-8ml/kg of ideal body weight (i.e. 300-600ml)
  - Respiratory rate: 12 breaths per minute
  - I:E ratio 1:2
  - Check an arterial blood gas within 15 minutes of starting ventilation on these settings, change these as necessary to achieve target  $\text{PaO}_2$  and  $\text{PaCO}_2$ . Correlate  $\text{PaO}_2$  with  $\text{SpO}_2$  and  $\text{PaCO}_2$  with  $\text{EtCO}_2$ .

### Oxygenation

- Target for oxygenation
  - Acute brain injury:  $\text{PaO}_2 \geq 13\text{kPa}$  ( $\text{SpO}_2 \geq 97\%$ )
  - In acute lung injury, once intracranial pressure is controlled, the  $\text{PaO}_2$  target may be relaxed, but should always be  $\geq 10\text{kPa}$  (except in severe lung injury - see below)

- In severe lung injury requiring high levels of PEEP and inverse ratios (I:E ratio 2:1), it may be appropriate to aim for a  $\text{PaO}_2 \geq 8\text{kPa}$
- Maintaining oxygenation target
  - $\text{PaO}_2$  is related to mean airway pressure and  $\text{FiO}_2$
  - Set PEEP appropriate to lung pathology (eg collapse / consolidation on CXR) and body habitus.
  - In a patient with a normal CXR and normal BMI, start PEEP at 5cm
  - Increase PEEP incrementally ensuring ICP is not affected adversely to a maximum of 15cm  $\text{H}_2\text{O}$ . (Up to 20cm $\text{H}_2\text{O}$  in extreme cases)
  - Set  $\text{FiO}_2$  to achieve  $\text{PaO}_2$  target
  - As oxygenation improves, initially reduce  $\text{FiO}_2$  sequentially down to 0.28, maintaining PEEP. Only reduce PEEP once  $\text{FiO}_2 < 0.3$  with  $\text{PaO}_2 > \text{target}$
  - Consider
    - Treatment of bronchospasm with salbutamol nebs
    - Fiberoptic bronchoscopy if segmental or lobar collapse
    - Drainage of pneumothorax / haemothorax

## Ventilation

- Target  $\text{PaCO}_2$ 
  - Acute brain injury: Target  $\text{PaCO}_2$  4.5-5.0kPa
  - If severe acute rise in intracranial pressure causing abnormal pupillary change (i.e. fixation and dilation of one or both pupils), it may be necessary to acutely hyperventilate to a  $\text{PaCO}_2$  4.0kPa, whilst awaiting response to an osmotically active drug (e.g. Mannitol or hypertonic saline). As soon as ICP is lowered to a safe level, ventilation should be reduced to allow  $\text{PaCO}_2$  to rise gradually to 4.5-5.0kPa
  - In acute lung injury, once intracranial pressure is controlled, the  $\text{PaCO}_2$  may be allowed to rise slowly, keeping within the normal range
  - In severe lung injury, where there is no concern over intracranial pressure,  $\text{PaCO}_2$  may be allowed to rise gradually to 8kPa provided that pH remains  $\geq 7.2$ .
- Maintaining  $\text{PaCO}_2$  target
  - $\text{PaCO}_2$  is inversely related to minute ventilation
    - (minute ventilation = tidal volume x respiratory rate)

- Set tidal volume appropriate to lung pathology (eg collapse / consolidation on CXR) and body habitus.
  - Patients with lung pathology require lower tidal volumes to prevent exacerbating lung injury. Aim for 6ml/kg of ideal body weight.
  - In patients with no evidence of lung injury aim for 7-8ml/kg of ideal body weight.
- Set respiratory rate to control PaCO<sub>2</sub>. Start at 12 breaths/minute (range 10-24 breaths per minute)
- Some patients may synchronise better with the ventilator if the BiLevel mode is used.
- When weaning from ventilation to spontaneous breathing with PCV-VG, it is important to ensure that the inspiratory time ( $T_{\text{insp}}$ ) is not too long (i.e.  $\leq 1.1\text{s}$ ) rather than setting the I:E ratio at 1:2. This may be most easily achieved by using SIMV or BiLevel, setting  $T_{\text{insp}}$  and weaning the respiratory rate until the patient is instigating breaths.
  - Eg During PCV-VG, the ventilator sets the inspiratory time for all mandatory breaths depending on the respiratory rate and I:E ratio. It takes no account of any spontaneous breaths that occur between those mandatory breaths set. Hence when the set respiratory rate is reduced, whilst maintaining an I:E ratio of 1:2, the inspiratory time will increase and the patient may fail to synchronise with the ventilator.
- Spontaneously breathing patients may be supported via the ventilator using the CPAP/PS mode.
- If PaCO<sub>2</sub> is rising whilst patient is breathing spontaneously, ensure that they are not receiving excessive doses of opioids or other sedatives.



The presence of pneumothorax must be suspected in any patient with elevated airway pressures on positive pressure ventilation. Beware that it is much more difficult to diagnose bilateral pneumothoraces.

## Non-invasive ventilation

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- Patients with increasing oxygen requirements on a face mask may benefit from CPAP (Continuous Positive Airway Pressure). The patient typically wears a tight fitting mask with the oxygen/air mix maintained at a pressure of 5-10cmH<sub>2</sub>O. This will usually improve oxygenation and should help reduce areas of atelectasis / collapse in the lungs.
  - CPAP is commonly used at 5, 7.5 or 10cmH<sub>2</sub>O
  - It is best tolerated by using the separate CPAP circuit or NIV ventilator, rather than through non-invasive settings of the ICU ventilator

- Patients achieving insufficient tidal volumes (with increasing  $\text{PaCO}_2$ ), when self ventilating on a face mask, may benefit from BiPAP. This is similar to CPAP, but provides additional support during inspiration.
  - The pressure applied during expiration is EPAP (Expiratory Positive Airway Pressure), and is equivalent to CPAP
  - The pressure applied during inspiration is IPAP (Inspiratory Positive Airway Pressure), and is set to be greater than the EPAP.
  - The difference between the EPAP and the IPAP is the additional pressure the patient receives during inspiration i.e. an EPAP of  $10\text{cmH}_2\text{O}$ , with IPAP of  $15\text{cmH}_2\text{O}$ , produces  $5\text{cmH}_2\text{O}$  of additional pressure to aid inspiration
  - BiPAP is better tolerated using the separate NIV ventilator, rather than through the ICU ventilator
- All high spinal cord injury (SCI) patients, not already on invasive ventilation, must be considered for early nasal/facial BiPAP
  - NIV should be started immediately on any patient with a high SCI, and weakness affecting respiratory muscles
  - Delay in starting NIV can result in the development of lung atelectasis & collapse, increasing likelihood of the patient requiring invasive ventilation
  - Patients with SCI must be kept flat to aid diaphragmatic movement. (See Chapter on Spinal Cord Injury)
- Patients with Guillain-Barré syndrome (GBS) & Myasthenia Gravis (MG) must have their respiratory rate, vital capacity, arterial blood gases (ABG) and chest X-ray (CXR) monitored. NIV should be commenced early, before changes in ABG or CXR occur, to avoid intubation and ventilation whilst awaiting effect of IVIg or plasma exchange.
- Consider inserting NG tube in any patient receiving NIV, to allow suctioning of any gastric air, and prevent the subsequent development of gastric over-distension.
  - NG tubes can be used in patients who are receiving nasal BiPAP
- Patients with lower cranial nerve palsies must have an NG tube inserted and aspirated 1-2 hourly, and may not be suitable for NIV
- Do not attempt to wean IPAP or EPAP until the patient is symptomatically better and less tired
- Plan the weaning of SCI, GBS & MG patients from invasive ventilation onto NIV with the physio team to give best chance of success. Extubate early in the day, and at the beginning of the week.



Patients who are post op transphenoidal pituitary surgery MUST NOT receive non-invasive ventilation. This is likely to force gas intracranially though the surgical approach in the nasopharynx. Any such patient must be considered for early intubation if failing to ventilate adequately on a face mask. (The use of the Bird respirator via a mouth piece, with the physiotherapists, may also be possible.)

## Tracheostomy care

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### Insertion of tracheostomy

Tracheostomies may be inserted either percutaneously on the Neuro ICU, or surgically in theatre. Patients with concerns about neck stability or potentially difficult anatomy will need to be considered for a surgical tracheostomy.

Prior to tracheostomy insertion:

- The neurosurgical / neurological consultant must be in agreement with the Neuro ICU team that a tracheostomy is appropriate for the patient.
- The procedure must have been discussed with (and documented in the notes):
  - the patient, if they are able to comprehend the procedure and consent
  - the patient's family (complete a consent form 4, if patient unable to consent)
- The patient's feed should be stopped 6 hours before the procedure
- A group and save should have been sent, unless blood transfusion already has serum saved
- Sedation should be started prior to the procedure or transfer to theatre

Following the tracheostomy insertion:

- Sedation must be continued until any paralysing agent has worn off
- Consider sedating overnight or longer in agitated patients that may dislodge the tracheostomy tube
- Chest X-Ray must be performed and reviewed immediately following procedure



If the tracheostomy tube becomes dislodged within the first 10 days, the patient should be oxygenated and ventilated by face mask. Attempts to re-insert a tracheostomy tube at this stage may create a false passage and fail to oxygenate the patient. Urgent re-intubation (via oral route) may be necessary, although the priority remains adequate oxygenation and ventilation.

### Change of tracheostomy tube

- Portex tubes should be changed at 7 days, while 'Trachoe twist' tubes should be changed at 1 month
- Any change of tracheostomy tube must be done over an appropriate guide such as a suction catheter, until the tract is fully established.



Beware using a hard disposable bougie via a tracheostomy tube, because of the risk of causing damage to the tracheal / bronchial mucosa

### Management of chest drains

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- Any patient with a pneumothorax, particularly if being ventilated, should be considered for emergent insertion of a chest drain
- Following insertion of a chest drain, a chest X-Ray must be obtained immediately.
- Any patient with a chest drain in-situ must have daily chest x-rays while on Neuro ICU
- Chest drains should not be clamped
- Suction should only be applied after discussion with the cardiothoracic registrar / consultant.



Suction **must not** be standard wall suction. There are dedicated thoracic low pressure suction devices available which can be set up by the medical technicians. Suction must be less than 5kPa at all times.

- Chest drains should not be removed until they have stopped bubbling for more than 24 hours and a chest X-ray demonstrates no residual pneumothorax
- Following removal of the drain the patient must have a chest x-ray within 2 hours if on positive pressure ventilation or at 12 hours if breathing spontaneously

The three circulations that are essential for survival: cerebral, renal, and coronary, are protected from fluctuations in blood pressure by autoregulation. Autoregulation ensures a constant blood flow through these circulations over a range of blood pressures. In a normotensive individual this mechanism ensures an adequate but not excessive flow over a range of mean arterial blood pressure (MAP) of 50-150mmHg.

Untreated hypertensive patients reset this range to a higher level and may need a MAP >70 or 80mmHg to maintain adequate blood flow for perfusion through these circulations.

Adequate cerebral circulatory flow in the awake patient may be demonstrated by normal mentation. Unfortunately this may be impossible to assess in many of the patients admitted with intracranial pathology to Neuro ICU. Adequate renal circulatory flow may be demonstrated by a urine output  $\geq 0.5\text{ml/kg/hr}$  in the absence of any diuretics (eg mannitol) or diabetes insipidus (DI).

Inadequate coronary circulation may be demonstrated by evidence of ischaemia on a 12 lead ECG. However ECG changes are almost universal after subarachnoid haemorrhage, making further interpretation difficult.

## Blood pressure targets

Patients on Neuro ICU may have a blood pressure target set. This generally falls into one of three categories :

- 1** Cerebral Perfusion Pressure (CPP) target:
  - $\text{CPP} = \text{MAP} - \text{ICP}$
  - Traumatic brain injury (TBI) patients that have their ICP monitored must maintain a  $\text{CPP} > 60\text{mmHg}$ , to maximise their chance of good recovery.



- Some patients with TBI appear to respond better to a higher CPP target, and there is currently debate as to whether a dynamic target should be set (eg guided by markers of global cerebral autoregulation such as the pressure reactivity index (PRx))
- CPP targets are not appropriate in spontaneous SAH
- There is no evidence for maintenance of CPP affecting outcome in any intracranial condition except TBI.

## 2 Mean Arterial Pressure (MAP) target:

- **Traumatic brain injury** patients that do not have their ICP monitored should maintain a MAP > 90 mmHg, to maximise their chance of good recovery. (This allows for an ICP ≤ 30 mmHg)
- **Subarachnoid haemorrhage** patients that present with severe hypertension have traditionally had their blood pressure reduced to a systolic of ≤ 160 mmHg. The experience of most senior staff in The Wessex Neurological Centre is that this may not be beneficial in reducing the risk of rebleeding and may result in inadequate perfusion around any areas of haematoma. The ICP may be as high as 50 mmHg in these patients. (See chapter on Subarachnoid haemorrhage). However hypertension with a MAP > 140 mmHg should be controlled.
- **Vasospasm** may be the cause of neurological deterioration occurring > 72 hours after subarachnoid haemorrhage.
  - This will usually present as focal neurology related to inadequate perfusion of an arterial territory.
  - A CT scan must be performed to exclude significant hydrocephalus, rebleed or increased oedema around any intracerebral haematoma.
  - This may require pharmacological elevation of the blood pressure to maintain perfusion through vasoconstricted arteries.
  - The MAP target should be sequentially elevated to reverse any new neurological deficit, to a maximum of 140 mmHg while actively seeking clinical or ECG signs of coronary ischaemia
- **Intracerebral haemorrhage** patients may be severely hypertensive
  - They may need their MAP reduced slowly to decrease the likelihood of further haemorrhage, whilst maintaining adequate perfusion of the penumbra around the haematoma.
  - An ECG should be performed, looking for left ventricular hypertrophy.
  - If the ECG or an echo indicates that hypertension is long standing and has not previously been diagnosed or treated, then full investigation of this is indicated, including: urinary catecholamines, renal artery dopplers, etc.

- If the patient potentially has a phaeochromocytoma, hypertension should be gradually controlled with an infusion of phentolamine, rather than labetalol.
- In other cases, lowering the blood pressure may require labetalol, by intravenous bolus or infusion, prior to the introduction of oral antihypertensive agents
- An initial aim should be to reduce the MAP below 140mmHg
- Gradual control of hypertension may be appropriate over the following days and weeks with oral antihypertensive agents

### 3 No MAP / CPP target

- Awake patients should maintain a blood pressure that is adequate for normal mentation and urine output. This will normally require a MAP $\geq$ 50mmHg in a previously normotensive patient.
- Sedated patients should maintain a blood pressure that is adequate for appropriate urine output in the absence of any diuretic. This will normally be on average  $>0.5\text{ml/kg/hour}$  over a 4 hour period, but in a small elderly patient with normal renal function it may be appropriate to accept less. This will usually require a MAP $\geq$ 50mmHg in a previously normotensive patient.



Brain injured patients may develop significant hypertension. This may be appropriate to maintain perfusion of ischaemic brain in the face of raised intracranial pressure, hydrocephalus, or vasospasm. However there are risks associated with severe hypertension, eg expansion of ICH, cardiac ischaemia. Hypertension with a MAP $>140\text{mmHg}$  is an indication for gradual control of the blood pressure.

## Elevating blood pressure

- 1 Ensure adequate volume expansion using boluses of 0.9% saline. Suspect hypovolaemia in all trauma patients and those with significant aspiration pneumonitis.
- 2 1st line vasopressor : phenylephrine infusion (can be given via peripheral line)
  - phenylephrine 10mg in 500ml 0.9% Saline at rate 0-180ml/hr
- 3 2nd line vasopressor : noradrenaline infusion (via central line only)



The subclavian line is the preferred site for central venous catheters on Neuro ICU, in view of use of hard collars. However if a subclavian line cannot be easily inserted, or there is abnormal clotting or platelets, then internal jugular or femoral access should be used. Ultrasound should always be used to confirm the position of the vein, but need not be used for insertion.



If there has been an unsuccessful attempt to insert a subclavian line into a patient **never** attempt a subclavian line on the opposite side within 24 hours. The patient will require a chest X-Ray to look for pneumothorax. Bilateral subclavian attempts risks bilateral pneumothoraces, with potentially catastrophic consequences. Central venous access may be safely achieved by the internal jugular or femoral route.

- noradrenaline 20mg in 250ml 5% glucose at 0-40ml/hr
- Ensure baseline 12 lead ECG has been performed prior to starting noradrenaline, and repeat 6 hourly until MAP / CPP target has been achieved. All ECGs must be reviewed and signed by a doctor within 1 hour.
- Once noradrenaline infusion >10ml/hr
  - Start hydrocortisone 100mg IV tds
  - Consider fludrocortisone 100mcg PO /NG tds
  - Convert ranitidine to a proton pump inhibitor (PPI), if not already on PPI (Increased risk of GI haemorrhage in patient with vasoconstrictor and steroids)



Choosing the right proton pump inhibitor on Neuro ICU

Oral /NG route: lansoprazole 30mg NG od  
IV route: pantoprazole 40mg IV od

- Set up LiDCO
- Ensure ST segment monitoring is operational on monitor
- Daily 12 lead ECGs, checking for ischaemic changes



Any patient with sudden onset hypotension must have the presence of pneumothorax excluded. NB this is more likely with positive pressure ventilation.

## Patients admitted on anti-hypertensive medication

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- Most patients should have their anti-hypertensive medications withheld initially.
- The exception to this rule, is that all beta-blockers must be continued to avoid precipitating myocardial ischaemia.
- Most patients that are established on a beta-blocker have had it started for ischaemic heart disease rather than control of hypertension.
- Beta-blockade will lead to up-regulation of beta-receptors. Stopping beta-blockers acutely in the presence of raised catecholamines associated with intracranial hypertension, may lead to myocardial ischaemia or infarction.
- Beta-blockers should be continued even when vasopressors are being infused to achieve a high MAP / CPP target.

# Fluid management

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## Enteral feeding

Aim to start enteral feeding as soon as possible to provide appropriate caloric and fluid intake. Enteral feeding also promotes the integrity of the gastrointestinal mucosa. The enteral feeding guidelines are described in the following chapter.

## Intravenous fluid therapy

- Check to ensure that renal function is normal
- If renal function is normal, prescribe 0.9% saline 1000 ml with KCl 3 g (40 mmol) at 100-125ml/hr based on body weight, age and habitus
- In any patient with renal impairment, potassium should be reduced/withheld if serum potassium elevated
- Liaise with nursing staff to slow rate of IV fluids as enteral feeding becomes established

## Electrolyte disorders

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### Hypernatraemia

The commonest causes of hypernatraemia on Neuro ICU are cranial diabetes insipidus, water dehydration from inadequate intake in patients with impaired GCS, and following administration of mannitol or hypertonic saline for the treatment of cerebral oedema / raised ICP.

Acute hypernatraemia (eg occurring over a time period shorter than 6hrs) can be corrected acutely. In chronic hypernatraemia, correction of plasma sodium should proceed more cautiously; brain cells form ideogenic osmoles that act as a strong osmotic

force when exposed to more hypotonic plasma fluid. Rapid correction leads to brain oedema. Use of dextrose- containing IV fluids **must** be avoided for this reason.

Moderate hypernatraemia is acceptable in patients with raised ICP (serum sodium <154mmol/l or serum osmolality <320mosmol/kg)



Rate of change of serum sodium, whether increase or decrease, should NOT exceed 0.5mmol/l per hour, i.e. no more than 12mmol/l in 24hrs.

### CRANIAL DIABETES INSIPIDUS

- Excess loss of free water secondary to inadequate secretion of ADH.
- Common in craniopharyngioma, less common in other pituitary surgery, but may be seen with any intracranial condition, particularly if associated with raised intracranial pressure.
- Diagnosis is suggested by a urine output > 200 ml/hr for ≥ 2 hrs (with urine specific gravity < 1.01) and a rapidly rising serum sodium.
- Confirmation of diagnosis by markedly negative fluid balance over 4 hrs with high serum osmolality (> 300 mosmol), low urine osmolality and low urinary electrolyte concentrations, is not usually necessary
- Treatment should only be considered when serum sodium > 145 mmol/l, but may be withheld in patients with raised ICP until serum sodium >150mmol/l (DDAVP will prevent free water loss, which may worsen cerebral oedema.)
  - Treatment: DDAVP 0.5 µg IV PRN (Max 1hrly)
- Any patient with urine output > 200 ml/hr for ≥ 2 hrs should have blood gases performed immediately to check serum sodium.
- Replace volume with 0.9% saline (± KCl). Recheck sodium regularly to check it is returning to normal (at least 6 hourly).
- If diagnosis is delayed and serum sodium > 150 mmol/l,
  - Addition of water to enteral feed may allow gradual correction of serum sodium
  - IV titration of hypotonic saline (0.45%) should only be used to slowly correct serum sodium (by < 0.5 mmol/hr), if enteral water is not tolerated



Never give DDAVP and hypotonic IV fluids concurrently.

- Occasionally, patients may have partial diabetes insipidus. In these cases, there is an isolated increase in plasma sodium without a high urine output. Treatment should be discussed with the duty consultant but the condition responds to low doses of DDAVP.

## Hyponatraemia

Hyponatraemia in Neuro ICU may be caused by increased retention of water by the kidneys due to SIADH, or excess loss of sodium (cerebral salt wasting, diuretic use or adrenocortical failure). It can also result from long-term anticonvulsant use (e.g carbamazepine). Symptoms are rare until plasma sodium falls below 125 mmol/l, and consist of: headache, nausea, confusion, disorientation, coma and seizures.

Differentiating the underlying cause of hyponatraemia on Neuro ICU is practically impossible in most cases.

## SIADH

- Persistent secretion of ADH without an osmotic trigger.
- May result from CNS disease (trauma, infection, tumours, SAH), other malignancies, pulmonary disease, drugs, hypothyroidism, Addison's disease, porphyria.
- Diagnosis may be suggested by hyponatraemia, oliguria in context of euvolaemia and a normal to low urea and creatinine.
- Diagnosis depends on:
  - urine osmolality > serum osmolality.
  - urinary  $\text{Na}^+$  > 20 mmol/l.
  - serum  $\text{Na}^+$  < 130 mmol/l.
  - normal renal, hepatic, cardiac, pituitary, adrenal and thyroid function.
  - absence of hypovolaemia, hypotension, oedema, drugs affecting ADH secretion.

- Although medical management of SIADH commonly involves fluid restriction, this is not usually indicated for patients on Neuro ICU due to the risk of intravascular depletion causing inadequate cerebral perfusion. This is compounded by the potential for cerebral salt wasting to exist concurrently.

### CEREBRAL SALT WASTING

- Syndrome associated with neurological disease where natriuretic compounds are released by the atria of the heart and possibly periventricular areas of the brain, and induce renal sodium loss.
- Hyponatraemia may be potentiated by appropriate ADH secretion to compensate for hypovolaemia.
- Diagnosis is suggested by hyponatraemia with normal to high urine output, normal to high urea and creatinine, negative fluid balance.
- Confirmation of diagnosis requires demonstrating a natriuresis in the presence of hypovolaemia. This is a situation that can never be tolerated in acutely unwell neurosurgical patients because of the risk of impairing cerebral perfusion. Hence making the diagnosis is practically impossible.
- Diagnosis also requires the absence of aldosterone deficiency, diuretics, renal tubular damage or Bartter's syndrome.

### MANAGEMENT OF HYPONATRAEMIA ( $\text{Na} < 135 \text{mmol/L}$ )

- 1 Ensure adequate sodium and potassium intake by prescribing oral/enteral sodium and potassium supplementation (Slow sodium 4 tablets qds, Slow/Sando K 4 tablets qds)
- 2 Minimize enteral free water, by flushing enteral drugs with either a minimum amount of water, or with 0.9% sodium chloride solution. Discourage patients from drinking water, although tea, coffee and juices are allowed.



Removing the patient's water jug and glass from their bedside will help prevent excess water consumption. To ensure patient comfort they should be permitted to drink tea, coffee and fruit juices. Tea and coffee are both diuretics and will only be supplied on request. They will also usually be made with milk, which contains electrolytes. Fruit juices may also be consumed in moderation since these also contain electrolytes.

- 3 Target a maintenance total fluid intake of 1ml/kg/hr for patients >60 years. Include dietary fluid in balance.
- 4 Chronic low sodium  $< 125 \text{mmol/L}$  should be corrected gradually with 0.9% saline, with 3 grams KCl per litre.



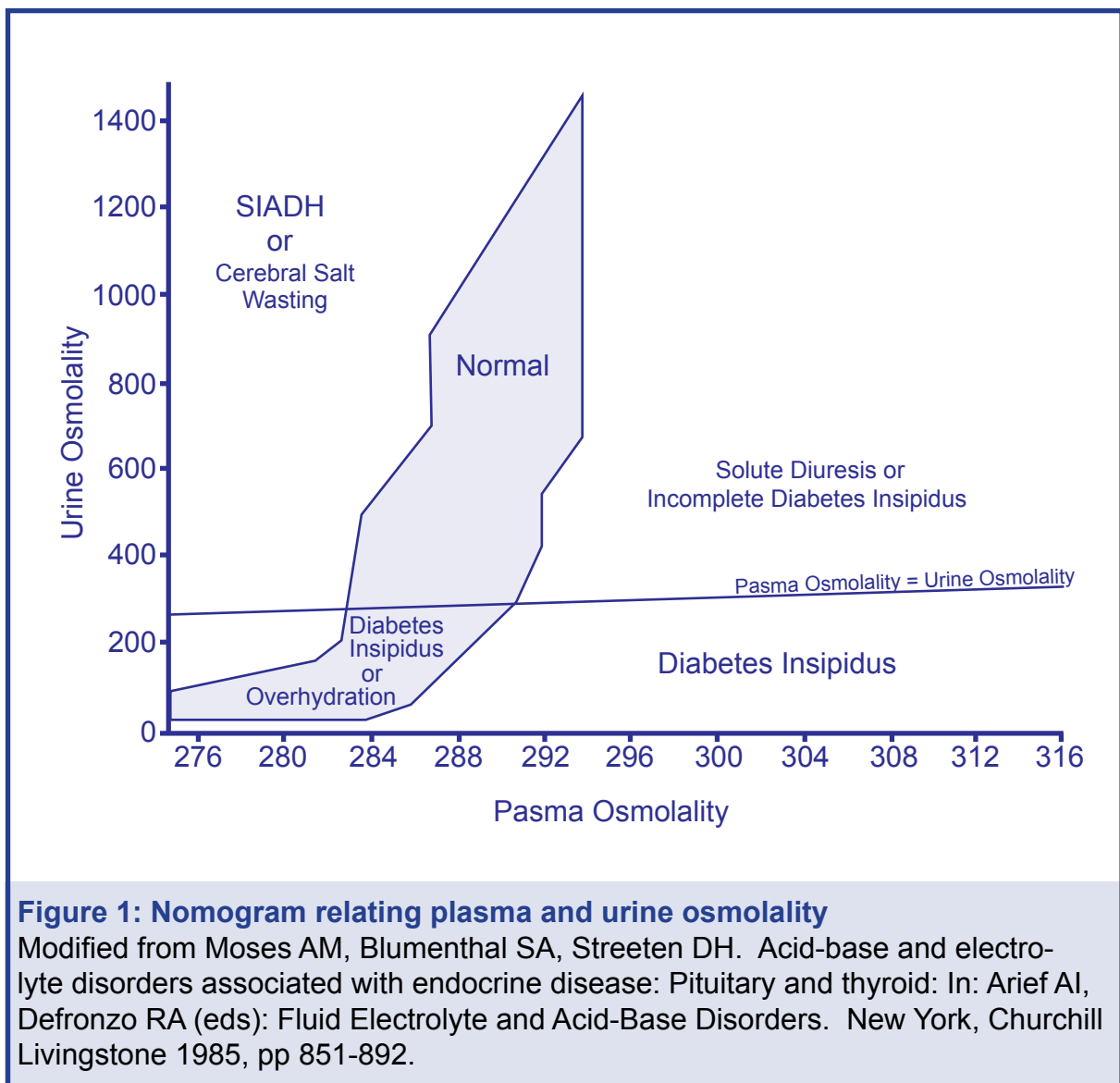
- 5** Moderate hyponatraemia may be corrected with 1.8% saline 50ml/hr, which may be administered by a peripheral IV cannula. If serum sodium does not gradually increase, then consider 5% saline 20-50ml/hr via a central line.



Rate of change of serum sodium, whether increase or decrease, should NOT exceed 0.5mmol/l per hour, i.e. no more than 12mmol/l in 24hrs.



Rapid correction risks central pontine myelinolysis, which may result in permanent neurological impairment; ranging from weakness to locked in syndrome.



- 6** Start **fludrocortisone 100mcg PO/NG tds** in any patient that may have pituitary insufficiency, or is at risk of cerebral salt wasting.

Non-neurosurgical patients that are likely to have SIADH may be considered for fluid restriction (1500ml/day), or in severe cases demeclocycline (impairs action of ADH on the kidney) can be used.



Tolvaptan should NOT be used on Neuro ICU (risk of excessively rapid rise in serum sodium)

## CNS injured patients with diabetes mellitus

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- Patients with Type 1 diabetes mellitus will usually have been commenced on insulin at the time of diagnosis. This is an autoimmune condition with destruction of insulin producing cells. These patients have an absolute requirement for insulin.
- Patients with Type 2 diabetes mellitus will usually produce some insulin. They may require modification of diet, tablets or insulin to control their blood sugar.

Principles of managing the diabetic patient:

- Blood sugar should be maintained 4-8mmol/l. This is most reliably achieved with an insulin infusion whilst on Neuro ICU.
- Type 1 diabetics should receive insulin continuously, even if their blood sugar is within the normal range. This is to prevent the development of diabetic ketoacidosis. These patients may require IV glucose if they are being fasted.
- Intravenous glucose must not be given in the form of hypotonic fluids (eg 5% glucose) in patients with CNS injury. Therefore, if intravenous glucose is required, it should be given as 500ml bags of fluid containing: **5% glucose with 0.9% saline & 0.3% KCl** at an appropriate maintenance rate (eg 100ml/hr).



The use of hypotonic solutions containing glucose (eg 5% glucose, 0.18% saline/4% glucose) as a part of diabetic management with an insulin infusion has been associated with worsening of cerebral oedema.

- Patients should not receive subcutaneous insulin whilst unwell on Neuro ICU. This risks the storage of large amounts of insulin in fat that is inadequately perfused for reliable absorption. Reperfusion of this fat when the patient recovers will cause an excessive delivery of insulin and potentially untreatable hypoglycaemia.
- Patients admitted with diabetic ketoacidosis will be profoundly dehydrated and will require fluid resuscitation with normal saline (+/-KCl) in addition to an insulin infusion and 500ml bags of fluid containing: **5% glucose with 0.9% saline & 0.3% KCl** at an appropriate maintenance rate (eg 100ml/hr).

- Beware rapid reduction in serum glucose. This may cause an excessively fast reduction in serum osmolality, provoking CNS oedema. Serum glucose should be reduced at a controlled rate. Consideration should be given to increasing the serum sodium to offset the reduction in serum osmolality. (Consider allowing sodium to rise by 1mmol/l for every reduction in glucose of 2mmol/l)

# Enteral feeding

- ICU patients should normally receive 25kCal per kilogram per 24hrs.
  - A recent study has shown that best outcomes are observed when patients receive a minimum of 25kcal/kg/24hrs. (10kcal/kg/24hr less was associated with 30-40% increase in mortality)
  - Government recommendations are that females receive 2000kCal per 24hrs, and males 2500kCal per 24hrs.



Nutrison standard and multifibre feeds contain 1kCal/ml

Nutrison energy and energy multifibre feeds contain 1.5kCal/ml

Nutrison protein plus feed contains 1.25 kCal/ml

- All nutrison feeds are gluten free, and contain a full complement of vitamins, iron and trace elements.
- Patients should be fed continuously over 24 hours to help protect gastric mucosa and allow more stable blood glucose concentrations when insulin is required.
- Patients with traumatic brain injury increase their metabolic rate by 40%. Hence use of energy feed rather than standard in this group. Prescribe feed at the same rate, but energy feed rather than standard feed.
- Early establishment of adequate nutrition in head injured patients is associated with reduced intercurrent infections and improved survival.
- Enteral feeding has many advantages over parenteral feeding and should be established as early as possible. Intragastric feeding may fail to be absorbed due to gastric stasis and pyloric closure secondary to high dose opioids.

## Establishing enteral feed

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- 1 A Malnutrition Universal Screening Tool (MUST) must be completed by the nursing staff on admission. This must include an accurate height and weight, either reported by relatives, or measured.
- 2 If not already present, insert a wide bore (14-16G) feeding tube either nasally or, if anterior base of skull fracture present, orally.



### NG tubes and base of skull fractures?

A feeding tube may be placed via the nose provided the base of skull fracture does not involve sphenoid sinus, there is no rhinorrhea, and any facial fractures do not involve nose or nasopharynx. This is not usually performed until shortly before extubation.

In these circumstances the feeding tube should be placed by the Neuro ICU consultant and may require use of a fibrescope to visualise correct passage of the tube through the nasopharynx.



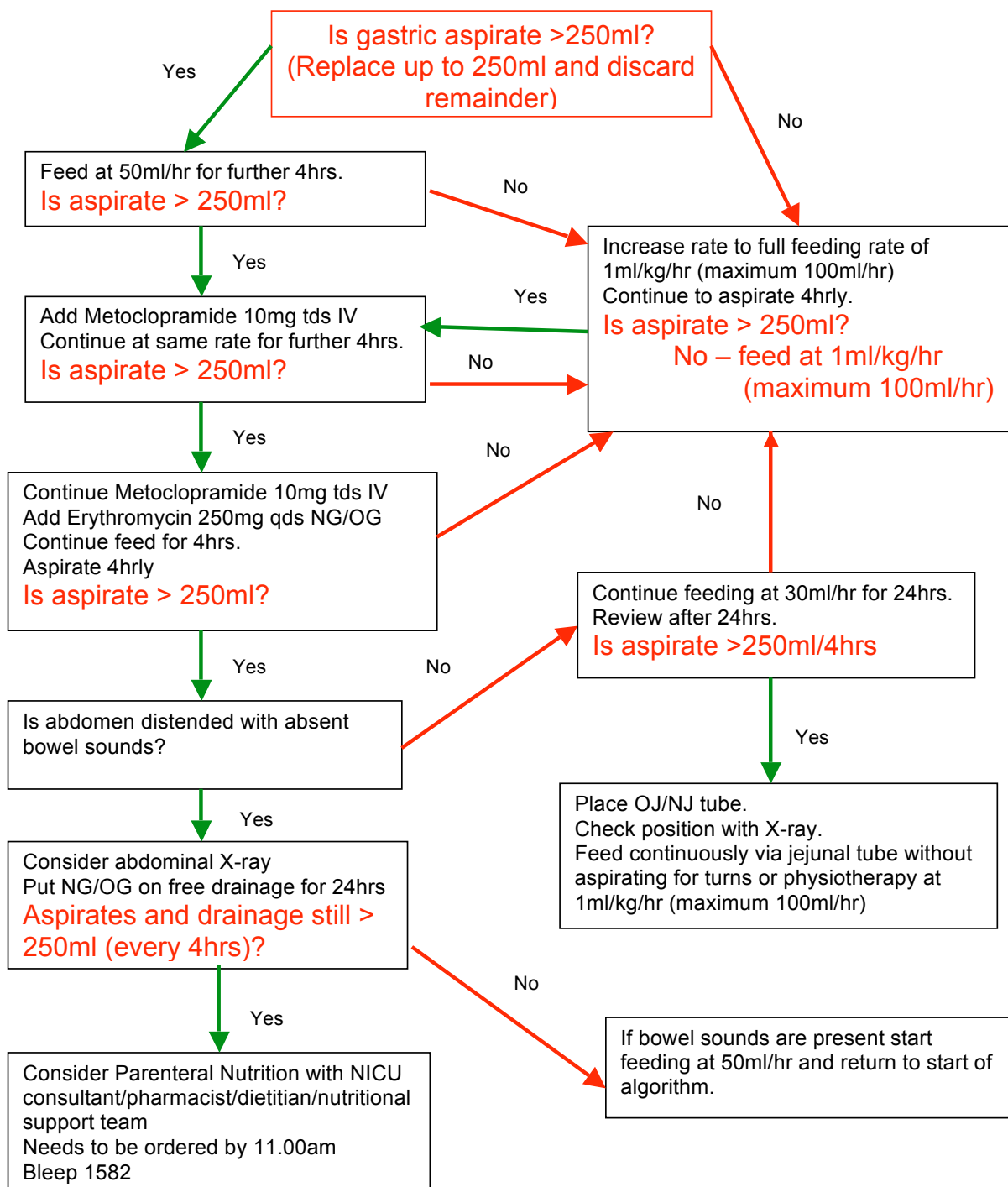
Feeding tube placement must be confirmed by aspiration of gastric fluid pH≤3. If this is not possible, a chest x-ray must be performed to confirm the tip of the tube is positioned below the hemidiaphragm prior to starting enteral feed.

- 3 Tube length must be recorded at time of insertion, and checked with each nursing shift (recorded on main obs chart.) If the patient vomits or a change of length is noted, tube position must be reconfirmed as above.
- 4 Aim to start enteral feeding as soon as patient is haemodynamically stable, (inadequate gut perfusion prior to this limits its usefulness) to provide appropriate caloric, protein and fluid intake. Enteral feeding also promotes the integrity of the GI mucosa.
- 5 Start enteral feed at 50ml/hr nutrison standard (energy feed in TBI patients). Increase to full feeding rate of 1ml/kg/hr (max 100ml/hr) as guided by enteral feeding algorithm.
- 6 Ensure patient receives at least 500ml **multifibre** feed per 24hours. Prescribe 1 bag of nutrison multifibre (energy multifibre for TBI patient)
- 7 If gastric aspirates are >250ml per 4 hours,
  - a start prokinetic drugs
    - metoclopramide 10mg IV tds
    - erythromycin 250mg NG qds, review after 72 hours

- b** Consider NJ tube insertion using Cortrak
  - c** If unable to establish adequate enteral nutrition in first 3 days, consider starting TPN but aim to wean it as soon as enteral feeding established
- 8** Patients may require separate supplementation of Na<sup>+</sup> and K<sup>+</sup>.
- 9** Stop enteral feed for 2 hrs before and after administration of enteral phenytoin. Where feed is stopped for phenytoin administration, or any procedure, the rate over the remainder 24h period should be augmented to ensure full feeding.
- 10** Any patient suspected of suffering from alcohol abuse, needs vitamin B supplementation; starting with Pabrinex parenterally ([Pabrinex 1 pair IV bd](#) for 3 days). Vitamin B supplementation may be continued when fully enterally fed & able to absorb as vitamin B compound, strong
- 11** If serum osmolality excessive (>320mosm/kg), enteral feed may be supplemented with water via the feeding tube
- 12** If feed stopped for any reason and patient on insulin infusion, check blood glucose 1 hourly until glucose stable and feed restarted, adjusting insulin dose appropriately, according to sliding scale
- 13** Drugs may be given via the gastric tube if aspirates are low, otherwise give all drugs parenterally
- 14** All patients should have blood taken for serum magnesium & phosphate levels, once they have been resuscitated and stable>24hrs
- 15** Any patient with low serum phosphate that is suspected to be at risk from refeeding syndrome (eg anorexia nervosa or cachexia), must receive enteral phosphate and potassium, and IV magnesium supplementation with a gradual increase in rate of feeding from 30ml/hr of standard feed, whilst serum phosphate levels remain low.

### Wessex Neurosciences Intensive Care Unit Enteral Feeding Algorithm

- Aim to start enteral nutrition as soon as patient is haemodynamically stable
- Check position of NG/OG enteral feeding tube as per Adult Enteral Feeding Guidelines
- **Start feeding Nutrison Standard (Energy in Traumatic Brain Injury patients) at 50ml/hr**
- Monitor gastric feeding tube aspirate 4 hourly
- Aspirate gastric feeding tube prior to chest physiotherapy and turning.



P.T.O

- **Complete MUST score on admission and review weekly**
- **Record height and weight of patient (measure if necessary)**
- **Ensure patient receives at least 500ml multifibre feed/day**
- **Flush tube with minimal amount of water or saline before and after drugs (especially Nimodipine) and after stoppages to prevent blocking.**
- **If stopped for a procedure, restart feed at previous rate.**
- **Stop feed 2hrs before and 2hrs after giving enteral Phenytoin.**
- **Make up any feed intake lost due to Phenytoin administration or procedures by increasing the feeding rate for the remaining hours.**
- **Only add water to feeding regimen if patient is dehydrated with a high serum sodium.**
- **Consider insertion of fine bore NG tube after 10 days – unless there are high gastric aspirates.**
- **Follow SLT Flow Chart when introducing oral fluids.**
- **Start food record charts and monitor daily protein and calorie intake, if patient starts to take oral diet.**
- **Ensure adequate protein and calorie intake in oral diet before removal of NG feeding tube**

**Refer all trauma patients to trauma dietitian on admission**

- **telephone ext: 3456**
- **e-mail: [majortraumadietitian@uhs.nhs.uk](mailto:majortraumadietitian@uhs.nhs.uk)**



## Insertion and management of jejunal feeding tubes

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- 1** A wide bore gastric feeding tube should already have been inserted, and its position checked. This should be maintained to assess gastric aspirates every 4 hours and to allow the administration of enteral drugs when appropriate.
- 2** Instructions for insertion of Cortrak feeding tubes are included with the tube and are easy to follow. In general, leaving time for tube migration may be better than repeated manipulation, once end of tube is at the pylorus.
- 3** Start [erythromycin 500mg IV infusion](#) prior to attempting insertion and consider adding [metoclopramide 10mg IV](#) to aid tube migration
- 4** Check the position of the tube on an upper abdominal X-ray prior to use
- 5** Flush jejunal tubes with 20ml water or saline at each feeding bag change
- 6** Never use a jejunal tube to give drugs – it is only for continuous feeding
- 7** Do not stop jejunal feed for physiotherapy or turns, just aspirate gastric tube to ensure that stomach is empty
- 8** Should insertion of a jejunal tube prove impossible, refer patient for endoscopic tube insertion urgently, unless absorbing well via gastric tube

# Deep vein thrombosis prophylaxis

# 9

All patients on Neuro ICU are at high risk of venous thromboembolism (VTE). Consequently a VTE assessment form must be completed on admission and at 24 hours for all patients admitted.

Many patients will be at high risk of bleeding, or rebleeding into the CNS, in the first few days after admission. This risk may exceed the risk of VTE, so careful consideration must be given to the use of low molecular weight heparin (eg enoxaparin).

## Anti-embolic stockings (AES)

All patients should have full length anti-embolic stockings fitted on admission, unless specifically contra-indicated:

- Severe peripheral arterial disease
- Severe peripheral neuropathy
- Major leg deformity
- Local leg conditions with which stockings would interfere e.g. dermatitis, vein ligation, gangrene, recent skin grafts, or presence of acute deep vein thrombosis (DVT)

## Intermittent pneumatic compression (IPC) devices

All patients should **also** have full length intermittent pneumatic compression devices applied and functioning at all times, unless specifically contra-indicated:

- Tissue-paper thin skin (eg elderly, or as a result of prolonged steroid use)
- Presence of acute DVT
- Allergy to the material of manufacture

Any patient where fitting of full length AES or IPC is not possible, should have calf AES and IPCs applied or, where this is impossible, IPC foot pumps unless contra-indicated.

## Low molecular weight heparin (LMWH)

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Consideration of LMWH as prophylaxis for VTE:

- A daily assessment of the relative risk for the use of LMWH must be performed and documented in the notes.
- Specific surgical instructions regarding the timing of commencement of LMWH should be followed
- Aim to start LMWH (**enoxaparin 40mg SC od**) with neurosurgical agreement:
  - 24 hrs after any neurosurgical procedure, or as documented in neurosurgical operation notes. This may be delayed until performance of a post operative CT scan rules out significant post-operative haematoma.
  - 24 hours after securing of an aneurysm, unless presence of hydrocephalus suggests that patient may require insertion of EVD acutely.
  - 24 hours after acute spinal cord injury, unless presence of epidural or intradural haematoma is present. In these cases, delaying LMWH until 48-72hrs may be more appropriate
  - 48 hours after traumatic brain injury, if there is no evidence of significant intracranial haemorrhage/haemorrhagic contusions, and the patient is unlikely to require an imminent neurosurgical procedure (eg EVD placement)
  - 48 hours following surgical evacuation of acute subdural/extradural haematoma
  - LMWH should be considered at 5 days post injury in all other cases
- Spontaneous intracerebral haemorrhage and spontaneous acute subdural haematoma, as well as TBI patients that have undergone decompressive craniectomy with significant intracranial haemorrhage, must be considered on a case by case basis with their neurosurgical team.
- Any patient that is not prescribed LMWH must have a plan documented in the notes with a timing for review / commencement.



Any patient with significant lower limb trauma that precludes the use of mechanical VTE prophylaxis, whose traumatic brain injury makes the use of LMWH inadvisable for the first 72 hours, should be considered for insertion of IVC filter within the first 36 hours of admission.

### Timing of procedures

- **LMWH following EVD, spinal drain or ICP bolt placement** - LMWH (eg enoxaparin) should be started no sooner than **6 hours** after insertion or removal of EVDs, spinal drains or ICP bolts.
- **Procedures following LMWH** - Placement or removal of EVD's, spinal drains or ICP bolt should be delayed for **12 hours** after administration of DVT prophylactic doses of LMWH (eg [enoxaparin 40mg SC od](#))

## MRSA screening & prophylaxis

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- All patients must be fully screened for MRSA on admission and weekly thereafter
- All patients must have daily chlorhexidine washes and nasal bactroban, for the first five days after admission
- Any patient that has been previously noted to be MRSA positive must:
  - Be treated as still being MRSA positive
  - Be isolated
  - Continue chlorhexidine washes and nasal bactroban for 5 days initially, and for 48 hours prior to elective surgery
  - Be documented as MRSA positive on cover of hospital notes and in medical, nursing, and physiotherapy notes
  - Be notified to the infection control team if they would be otherwise unaware of the patient, eg patient was found to be MRSA positive while at a hospital other than University Hospital Southampton

## Diarrhoea

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- Any patient with diarrhoea (Bristol stool type 5 to 7) must be isolated within 4 hours if an infective cause is suspected for that diarrhoea.
  - Risk factors for infective cause:
    - Patient admitted with diarrhoea
    - Other family members or carers affected
    - Recent history of foreign travel
    - Recently eaten high risk food (eg BBQ, mass catered event or takeaway)

- Previous hospital admission within 28 days
- Previous positive *C. difficile* result
- Currently or recently completed course of antibiotics
- Patient on proton pump inhibitor
- Non-infective causes for diarrhoea include:
  - Patient's normal bowel pattern eg diverticulitis / inflammatory bowel disease / malignancy
  - Patient receiving aperients ( eg senna / lactulose)
  - Patient receiving medication or treatment that may cause diarrhoea eg omeprazole / ferrous fumarate / radiotherapy
  - Patient receiving enteral or supplementary feeds eg fortisips
  - Severe constipation with overflow diarrhoea
- Any patient that is considered to have an infective cause for the diarrhoea must have a sample sent for *C. difficile* analysis and be isolated within 4 hours
- Only essential staff should enter the room of an isolated patient and they must wash their hands with soap and water on exit. Alcohol hand rub must not be used because it may increase the risk of spreading *C. difficile*.

## Prophylactic antibiotics

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- Most neurosurgical operations will require antibiotic prophylaxis prior to skin incision.
- Cefuroxime 1.5g IV is indicated for most neurosurgical operations unless the patient has a severe allergy to beta-lactam antibiotics (eg penicillin), in which case alternative antibiotic prophylaxis will be indicated (eg vancomycin 1g IV & gentamicin 120mg IV)
- A further dose of cefuroxime 750mg IV should be given for all operations that last longer than 6 hours (unless severe allergy as above.)
- Any operation that involves the air filled spaces of the skull (eg frontal sinus or mastoid air cells) will also require metronidazole 500mg IV prior to skin incision.
- Pituitary surgery performed via the sphenoid sinus will require co-amoxiclav 1.2g IV, unless penicillin allergic, instead of cefuroxime.

- Any patient that is known to be MRSA positive or has been resident in a healthcare institution for more than 7 days, should receive, instead of cefuroxime, both:
  - **vancomycin 1g IV over 1 hour**, prior to skin incision. When possible this should be given on the ward prior to surgery.
  - **gentamicin 120mg IV**, prior to skin incision
- Vancomycin and gentamicin should be considered in all high risk procedures (eg cranioplasties)
- Neurosurgical consultants may request that the prophylactic antibiotics be continued for two further doses post-operatively or in high risk cases for two days.
- All CSF shunt procedures are very high risk for infection. In addition to the prophylactic antibiotics outlined above:
  - Patients should be first on the operation list
  - All entrances to the operating theatre must be labelled with signs indicating that a shunt insertion is in progress, and entry is restricted.
  - The procedure should be performed by two operators
  - Theatre personnel and movement should be minimised
  - A CSF sample must be sent to microbiology for microscopy, C&S
  - All revision surgery should have intrathecal vancomycin (usually 20mg, pre-prepared syringe) +/- gentamicin (usually 5mg of the intrathecal preparation) given at operation

## Infection and sepsis

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- The vast majority of patients admitted to Neuro ICU intubated and ventilated will have aspirated at the time of deterioration in conscious level.
  - This will cause aspiration pneumonitis
  - This may become infected over the following days
  - Antibiotics are not indicated unless the overall clinical assessment is of lower respiratory tract infection, which should be suspected in a patient with a combination of:
    - Fever
    - Rise in CRP
    - Purulent sputum

- Abnormal signs on respiratory examination of spontaneously breathing patients.
- Increased respiratory support to achieve the target oxygenation
- Chest x-ray changes suggestive of consolidation
- Lower respiratory tract infection occurring in the first 4 days of admission is likely to respond to antibiotics for community acquired infection eg co-amoxiclav, chloramphenicol, or cefuroxime
  - Patients who have a history of rash to penicillin should receive cefuroxime +/- metronidazole instead of co-amoxiclav. Patients who have a history of anaphylaxis to penicillin should be discussed with the microbiologist.
- Thereafter, pneumonia should be treated with antibiotics for hospital acquired (eg ventilator associated) pneumonia, e.g. **tazocin 4.5g IV tds** (provided patient is not penicillin allergic) +/- single dose **gentamicin IV 5mg/kg**. Other options include meropenem or the combination of vancomycin and ciprofloxacin. These should be discussed with the Neuro ICU consultant before commencement.
  - Vancomycin should be started at higher doses in septic patients. In a patient with normal renal function, a good urine output and MAP>65mmHg, vancomycin should be initially prescribed at 1.5g iv tds.
  - Target vancomycin trough level of 15-20, taken after 3rd dose. Further levels are only required if dose changed or renal function deteriorates.
  - Any patient who is treated with ciprofloxacin should have IV preparation changed to oral/ng as soon as they are absorbing. If ciprofloxacin is given NG in a patient receiving enteral feed, the dose should be increased to 750mg NG bd. (The proportion of drug absorbed is reduced by binding to divalent cations in enteral feed. Avoid giving ferrous fumarate near time of ciprofloxacin administration.)
  - Any patient treated with rifampacin should be given the PO/NG preparation provided they are absorbing feed. Rifampacin has 100% oral bioavailability.
- Antibiotic treatment should be guided by microbiological results and sensitivities. This will usually involve discussion on a microbiological ward round.
- All patients that develop a fever must be considered to have an infection and undergo a septic screen, which includes:
  - Chest x-ray
  - Sputum sample or endotracheal aspirate for culture and sensitivity



- Urine dipstick followed by M,C&S. State 'fever' or 'septic screen' on the request form to ensure the sample is processed.
- Blood cultures: both peripheral and via any lines present e.g. central/arterial/long lines, which should be labelled as line samples appropriately (central/arterial/long line sample)
- CSF sample from an external ventricular drain or ventriculoperitoneal shunt or consideration for a lumbar puncture
- Swab from any wound or vascular access site that looks clinically infected
- Central/long/arterial catheters are not routinely replaced after any set length of time on Neuro ICU. All such catheters should be removed as soon as they are no longer required. (However, femoral vascular access catheters must be removed within 48 hours of insertion)
- Femoral cooling lines may need to be kept in situ for many days to control raised ICP in traumatic brain injury. Consequently, the vascular access ports on the cooling line should be flushed on insertion, then capped off and not used except for taking specific blood cultures from the line (as part of a septic screen) or for emergency resuscitation.
- New onset of sepsis, particularly if associated with a requirement for a vaso-pressor or increase in the dose, must be treated as a medical emergency:
  - A full septic screen must be performed immediately
  - The Neuro ICU consultant must be informed immediately
  - Appropriate broad spectrum antibiotics must be given within one hour of diagnosis, e.g. **tazocin 4.5g IV tds** and **gentamicin IV 5mg/kg**
    - In any patient with normal renal function, gentamicin can be given at a dose of 5mg/kg daily without delaying administration by waiting for levels. In many patients with normal renal function it may be considered unnecessary to perform levels.
    - Any vascular access device that is considered to be the most likely source of infection must be removed within one hour
  - Where possible, no new vascular access device should be inserted until after infusion of the antimicrobials (eg antibiotics or antifungals) to minimise the risk of colonisation of the new catheter. This is particularly important if a fungal infection is suspected.

## Ventriculitis & cerebral abscess

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- Whilst meningitis will usually be associated with a significant elevation of CRP and peripheral WBC count, the development of an intracerebral abscess or ventriculitis will frequently not be associated with either of these.
- Fever, deterioration in conscious level or neurology, or unexplained tachycardia, particularly in the presence of an external ventricular drain (EVD), must always prompt:
  - Examination of the patient for signs of meningism eg neck stiffness
  - Immediate sending of a CSF sample for microscopy, culture and sensitivity
  - Consider using antibiotics at doses appropriate for CNS infection if the CSF WBC count is significantly increased from previous values (usually with predominance of neutrophils) e.g. [cefotaxime 2g IV 4 hourly](#)
    - [Meropenem 2g IV 8 hourly](#) (CNS dose), is reserved as 2nd line antibiotic management of CNS infection. It should only be started after discussion with the duty microbiologist.
  - Informing the neurosurgical team of the deterioration and potential presence of ventriculitis
- Significant positive CSF cultures must always be treated immediately with CNS dose IV antibiotics and may also require intrathecal antibiotics to be instilled daily via the EVD, according to microbiological advice
- If CSF gram stain microscopy is +ve for organisms, repeat CSF sample should be sent to check the result before starting antimicrobials. If patient appears seriously unwell, start antimicrobials immediately after taking that sample. Consider instilling IT vancomycin or gentamicin at that time.
- If the CSF or EVD continues to be infected despite antibiotic treatment, then consideration must be given, in conjunction with the microbiologist and neurosurgical team, to the removal of the EVD and its replacement via a new access site or management of communicating hydrocephalus by serial lumbar punctures
- If the patient is EVD dependent and will require a VP shunt insertion:
  - Appropriate antibiotics must be continued until CSF shows no organisms on microscopy nor growth on culture for >48 hours
  - Microbiological advice should be sought to agree antibiotic prophylaxis for insertion of the VP shunt
  - CSF should be sent for a protein level as well as for microscopy to help determine the timing of any VP shunt insertion.

### Requesting microbiological advice

- These guidelines should be used in conjunction with Trust guidelines on infection control and treatment
- Most microbiological decisions should be taken during daylight hours, preferably as part of the microbiological ward round.
- Plans should be made for appropriate antimicrobial treatment in case of patient deterioration
- If a patient deteriorates out of hours with sepsis, the Neuro ICU consultant must be informed.
- The on-call consultant microbiologist should not be called for advice out of hours without first discussing the patient with the Neuro ICU consultant.

### HIV Testing

- HIV testing is indicated in any patient that has, or is suspected to have, the following conditions:
  - Cerebral toxoplasmosis
  - Cryptococcal meningitis
  - Primary cerebral lymphoma
  - Leucoencephalopathy
  - Aseptic meningitis / encephalitis
  - Cerebral abscess
  - Other AIDS defining conditions (eg pneumocystis)
- In patients that **have capacity to consent**, a pre-test discussion to establish informed consent is recommended. Written consent is unnecessary, as is lengthy pretest counselling. The benefits of testing to the individual, the reason that the test is recommended and details of how the result will be given, must be discussed with the patient. This should be documented in the patients notes.
- In patients that are **unconscious or lack capacity** to consent, in the absence of any valid advance statement, testing should be performed where this is in the best interests of the patient. The result should be communicated to the patient at an appropriate time if/when capacity has been regained. Following the death of any patient that has been tested, onward disclosure to others at risk may be appropriate.

# Seizure management

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Patients with known epilepsy must continue all of their usual drugs at their prescribed doses and times. Insert a NG tube if necessary for drugs that have no parenteral preparation.

## Diagnosing seizures

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Beware labelling all abnormal patient movement as seizures - '*All that twitches is not epilepsy!*'

Dissociative seizures (pseudoseizures) need to be considered in treatment resistant status.

## Factors that support a diagnosis of seizures

- Tonic clonic movements that are stereotypical of grand mal epilepsy and are usually associated with:
  - loss of consciousness
  - incontinence
  - tongue biting
  - abnormal pupils
  - post ictal phase
- Focal seizures affecting one side of the body, a single limb, or confined to the face. These may progress to generalised seizures. Focal seizures cause motor, sensory or alterations of level of consciousness with automatisms
- Supratentorial pathological process of the brain eg tumour, infection, encephalitis, haematoma, contusion or depressed skull fracture.
- Recent neurosurgery above the tentorium cerebelli
- EEG confirmation of previous seizures

- Previous history of epilepsy

### Factors that make a diagnosis of seizures unlikely

- Atypical movements that do not follow any of the normal patterns of epilepsy.
  - Opisthotonus: Generalised extension of the body with arching of the back. This may occur during a hydrocephalic attack.
  - Parkinsonian tremor may occur as a result of the side-effects of anti-psychotics (NB these may be given for other actions eg as antiemetics metoclopramide, prochlorperazine), particularly in young patients.
  - Rigors: These will usually be associated with severe infection
  - Acute withdrawal of opioid or sedative agents, particularly in patients that were previously dependent, and had developed tolerance to them
  - Tremor resulting from anxiety in the awake patient
- Posterior fossa disease and surgery is very unlikely to cause seizures because any abnormal electrical activity would be unable to generalise




Any significant neurological deterioration (eg reduction in GCS) after posterior fossa surgery or in a patient with significant pathology of the posterior fossa, **MUST** be assumed to be from hydrocephalus and **NOT** from seizures. The patient should be given hyperosmolar therapy (mannitol / hypertonic saline) and taken for an emergency CT scan. This may indicate requirement for EVD insertion +/- posterior fossa decompression. Failure to identify a hydrocephalic crisis may result in the patient coning.

## Management of generalised seizures

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- 1 Assessment of patient
  - A Airway** : ensure that airway is clear and maintained
  - B Breathing**: Ensure adequate oxygenation and ventilation. Monitor SpO<sub>2</sub> and capnography where possible. Consider arterial blood gas.
  - C Circulation**: Monitor heart rate & blood pressure. Treat hypotension or arrhythmia as indicated. Ensure adequate IV access.
  - D Disability**: Monitor neurology and pupils.

- E Measure blood glucose**, and treat hypoglycaemia with 50% glucose IV (usually 20-50ml depending on severity of hypoglycaemia). If this may be associated with alcohol abuse or malnutrition, pabrinex 1 pair IV must be given at the same time.
- 2** Ensure the patient is safe from harming themselves (eg where possible ensure a clear space around them).
  - 3** Observe for up to 5 minutes in case the seizure self terminates (>90% self-terminate in ≤5min)
  - 4** If seizure continues >5minutes without clear signs of self termination:
    - a** Administer a benzodiazepine:
      - Lorazepam titrated 0.5 mg IV every minute, against response, up to a maximum of 0.1mg/kg in the first 10 minutes
      - Alternatively diazemuls 1mg every minute against response, up to 20mg, or midazolam 0.5mg every minute against response up to a maximum of 10mg
      - In patients without IV access diazepam may be given rectally in a dose of 10-20mg or midazolam 10mg buccally or IM
    - b** Administer phenytoin, unless already in therapeutic range
      - In a patient not already receiving regular phenytoin, a loading dose of 20mg/kg (this is usually given as a 1g infusion over 1 hour)
      - In a patient who is on regular phenytoin, but that is known or suspected to be sub-therapeutic, a further loading dose of 1g over 1 hour should be given
      - In a patient who is known to have phenytoin at a therapeutic level, they should be loaded with either levetiracetam (load with 1g IV, followed by 500mg bd) or phenobarbitone (load with 10-15mg/kg at rate of 100mg/min) or sodium valproate (load with 1g, followed by 500mg bd.)
- 

Phenobarbitone can be given most rapidly, and may avert the need for intubation. Levetiracetam & sodium valproate require a longer loading time, and will normally be given following intubation. There is greater evidence supporting the use of sodium valproate in the treatment of status, but it may interfere with platelet function, and should not be given to neurosurgical patients.
- 5** A patient that remains in status despite a benzodiazepine and phenytoin loading, should have general anaesthesia induced by an anaesthetist / intensivist.
    - a** Induction of anaesthesia:

- This should be with thiopentone 3-5mg/kg
  - Alternatively a combination of propofol and midazolam may be used
  - b** Maintenance of sedation should be with an infusion of propofol initially. If more than 4mg/kg/hr then consider adding in midazolam infusion
  - c** Consider emergency CT Brain scan in all patients with new onset seizures, particularly post-operative patients
  - d** If the patient is not already on Neuro ICU, they should be transferred there, preferably after their CT scan
- 6** Upon transfer to Neuro ICU, inform the neurologist or neurosurgeon responsible for the patient of their admission and any new abnormality seen on CT. Perform the following investigations:
- a** Standard admission blood tests
  - b** Consider LP or obtaining CSF from EVD/VP shunt if present
  - c** Consider use of Cerebral Function Monitor (CFM) or BIS monitor set to show raw EEG
  - d** Perform a formal EEG during daylight hours to confirm that no seizure activity is present. This initial EEG should be performed prior to reducing sedation.
- 7** Where a CNS infection is suspected as the cause for the seizures, then appropriate antimicrobial agents should be started. (eg [cefotaxime 2g IV 4hrly](#), [acyclovir 10mg/kg IV 8hrly](#))
- 8** Commence a 'fit chart', recording all episodes of seizure activity
- 9** Further anti-epileptic drugs and doses should be guided by a neurologist.
- 10** In general:
- a** Continue regular phenytoin (eg 300mg nocte IV/NG/PO) and perform daily phenytoin levels, which should be corrected for albumin level (g/l)



$$\text{corrected phenytoin} = \frac{\text{measured phenytoin level}}{(0.02 \times \text{albumin}) + 0.1}$$

- b** Sedation should be continued without neuromuscular blockade to assess for ongoing seizure activity. A formal EEG should be gained as soon as possible to confirm abolition of seizure activity.
- c** If seizures continuing, abolish with adequate sedation and add in an additional agent. Options include:
  - Levetiracetam, load with 1g IV followed by 500mg IV bd. (Preferred agent for neurosurgical patients)

- Sodium valproate, load with 1g, followed by 500mg bd. (Preferred agent for neurological patients. Should not to be given to neurosurgical patients because it interferes with platelet function.)
  - Phenobarbitone, load with 10-15mg/kg at rate of 100mg/min.
  - Lamotrigine starting at 25mg NG od may be used in patients that develop seizures from cerebral tumours. It is usually started at a low dose, and titrated slowly up over weeks, so may not be appropriate for the rapid control of new onset seizures on Neuro ICU.
- d** If any evidence of ongoing seizure activity despite propofol infusion at 4mg/kg/hr, add in a midazolam infusion.
- e** Consideration of establishing thiopentone coma should occur once:
- Status epilepticus has been confirmed on formal EEG
  - There is concern of ongoing seizure activity despite propofol & midazolam infusion
  - Phenytoin is at a therapeutic level
  - Another anti-epileptic drug has been commenced (eg levetiracetam, sodium valproate or phenobarbitone)

## Investigation of new onset status epilepticus

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- 1** CT scan with IV contrast as soon as patient stabilised
  - If clear structural abnormality eg tumour / abcess, discuss with neurosurgery and agree management plan
- 2** MRI scan if CT scan inconclusive
- 3** If imaging is non-diagnostic:
  - a** Perform lumbar puncture (provided no contra-indication eg on imaging / coagulation screen). Opening and closing pressure should be measured. Samples should be collected separately for:
    - Microscopy, culture & sensitivities (M,C&S)
    - Cytology
    - Immunology
    - Virology
    - Protein
    - Glucose (fluoride bottle)
    - Sample for storage



- b** Blood samples should be taken for:
  - Ammonia level (alert lab technician and transport on ice)
  - Vasculitis screen (eg ANCA, ESR, CRP)
  - Auto-antibody screen (eg ANA, dsDNA, NMDA receptor antibodies)
- c** Urine should be dipstick tested and sent for M,C&S

## Management of focal seizures

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Assessment of cause of seizures

- 1** Clinical assessment of patient looking for any new neurological deficit
- 2** Investigations:
  - a** Check / perform routine blood tests
  - b** CT brain scan with IV contrast
  - c** Consider lumbar puncture or obtaining CSF from EVD / VP shunt
  - d** Formal EEG during daylight hours
- 3** Discuss treatment options with the patient's neurosurgeon or neurologist
- 4** Commence a 'fit chart', recording all episodes of seizure activity

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Patients may be admitted to Neuro ICU post operatively for:

- Premorbid medical conditions (eg obstructive sleep apnoea)
- Neurosurgical considerations (eg unstable neurology)
- Requirement for continued ventilation
- Vasoactive agent for blood pressure control
- Major haemorrhage

## Admission process

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- Routine admission procedure (see earlier chapter)
- Ensure post-operative surgical and anaesthetic instructions are documented and followed including:
  - Antibiotics / steroids / anticonvulsants
  - Wound drain management
  - Timing of post operative CT scan
  - Timing of administration of low-molecular weight heparin
  - External ventricular / lumbar spinal drain management (eg EVD height / CSF draining volume)
  - Haemodynamic & ventilator parameters
- Patients will commonly have cannulae placed in the lower limbs for neurosurgery. These will generally have been removed in recovery. However, if cannulae are still present in the lower limbs on arrival to Neuro ICU, do not remove them until adequate IV access has been achieved in the upper limbs and the patient is no longer at high risk of returning to theatre as an emergency.

## Analgesia

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- All patients should have their pain controlled at all times.
- All patients must be able to have their GCS and neurology assessed at all times.
- No drug should be prescribed that may increase the risk of haemorrhage from the site of surgery or the GI tract, without agreement from their consultant.

### Awake patients:

- Prescribe **paracetamol 1g PO/IV qds** to all patients unless contra-indicated.
- Prescribe **morphine 1mg IV PRN (15 mins)** to all patients unless contra-indicated.
- Prescribe a **morphine infusion 0–3 mg/hr IV** if 1mg boluses are inadequate.
- PCA morphine 1 mg / 5 min (prescribe as 2 mg/ml, 0.5 ml bolus, lockout 5 mins) may be prescribed for patients with GCS 15/15. The bolus dose may need to be increased to 2 mg in patients with severe pain.
- NSAIDs should only be prescribed with agreement from the consultant surgeon and Neuro ICU consultant
- Patients with neuropathic pain may benefit from novel opioids (e.g. oxycodone) and adjuncts e.g. **amitriptyline 10mg PO/NG nocte** and **gabapentin 300mg PO/NG od** (300mg on Day 1)
- Avoid codeine and compound analgesic preparations.



Tramadol lowers the seizure threshold (i.e. makes it more likely for a patient to fit) and should be avoided in any patient post craniotomy or who is at risk of seizures.

### Sedated patients:

- Prescribe **paracetamol 1 g PO/IV qds** to all patients unless contra-indicated.
- Prescribe **fentanyl 50–200 micrograms/hr IV**, in addition to propofol.
- **or** Prescribe **morphine infusion 1–20 mg/hr IV**, in addition to midazolam or propofol.

## Sedation & ventilation settings

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Patients may be admitted to Neuro ICU for ventilation overnight, following a long or otherwise complicated procedure.

### Sedation

- Use propofol 2–4 mg/kg/hr (10–30 ml/hr for an average adult) if overnight ventilation planned.
- Midazolam up to 20 mg/hr in an average adult if longer ventilation anticipated.

### Paralysis

- Atracurium 0.5 mg/kg/hr if paralysis indicated (4–5 ml/hr in an average adult).
- Check train of four, using a peripheral nerve stimulator, aiming for 1 twitch

### Ventilation

Initial settings :

- PCV/VG
- FiO<sub>2</sub> 0.5
- Tidal volume 7 ml/kg
- Rate 12–18 /min
- PEEP 5–10 cm H<sub>2</sub>O
- Targets
  - PaO<sub>2</sub> > 12 kPa
  - PaCO<sub>2</sub> 4.5–5.5 kPa
  - pH 7.35–7.45



Patients who are post op transphenoidal pituitary surgery **must not** receive non-invasive ventilation. This will force gas intracranially through the surgical approach in the nasopharynx. Any such patient must be considered for early intubation if failing to ventilate adequately on a face mask. ('Birding' by the physio may also be possible.)

## Haemodynamic & haematological management

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Initial targets:

- Heart rate 50–100 bpm
- Mean arterial blood pressure >60mmHg, unless:
  - Alternative set by surgeon / anaesthetist
  - Cerebral perfusion pressure target set (eg >60mmHg)
- Urine output > 0.5 ml/kg/hr on average over 4 hours
- Hb ≥ 8g/dl (unless chronically anaemic)
- APTR & INR < 1.4
- Platelet count > 150 x10<sup>9</sup>/ml

### Fluid administration

- Start enteral feeding as soon as haemodynamically stable and neurologically appropriate



Patients who are post op transphenoidal surgery **must not** have NG tubes inserted, unless they are placed under direct vision endoscopically.

- Initial intravenous fluid should be 0.9% saline with 0.3% KCl at 125ml/hr (100ml/hr in patients >70 years)



Hypotonic fluids (including hartmanns) **must** be avoided, to reduce risk of cerebral oedema.

- Further fluid according to electrolyte results (aim for Na 135–145 mmol/l, K 4–5 mmol/l).

### Tranexamic acid

Patients with a history of alcohol abuse or antiplatelet agents that have received tranexamic acid per-operatively should be considered for continuation at 1g tds for 4-7 days. NB this is contra-indicated in patients with subarachnoid haemorrhage.

# Traumatic brain injury

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## Introduction

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The initial stabilization of patients should have followed ATLS guidelines and patients should satisfy the admission criteria for trauma patients (see above).

All patients require adequate imaging of their spine with reformats and a consultant radiological report in order to allow appropriate nursing care. Manage as per spinal algorithm (see Spinal Management Chapter).

Patients will generally fall into one of two groups:

- 1** Intubated and ventilated patient who will either require:
  - Titration of sedation to a level that allows regular assessment of GCS and neurology. (Eg propofol & fentanyl to “tube tolerance” i.e. minimal sedation required to allow the patient to tolerate an endotracheal tube.)
  - Full sedation and intracranial pressure monitoring.
- 2** Self ventilating patient with a GCS 8-13 requiring careful observation post head injury. These patients may be agitated.



As a general rule, **no** patient should be ventilated and sedated to a level that prevents regular assessment of their underlying neurology without intracranial pressure monitoring. (The patient is at risk of rising ICP from cerebral oedema or further haemorrhage, which may not be recognised until the patient's pupils dilate)

## Management of intubated and ventilated patients

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- 1** Rapid assessment of patient including pupils and monitoring parameters
  - A Airway** : COET tube type & length
  - B Breathing**: FiO<sub>2</sub>, respiratory rate, tidal volume. Bilateral air-entry with SpO<sub>2</sub>>97%
  - C Circulation**: HR, BP (MAP>90mmHg), presence and position of arterial line
  - D Disability**: pupils, sedation
  - E Environment**: temperature, glucose
- 2** All patients should be assumed to have unstable spinal injury unless spinal algorithm has been completed and consultant radiology report confirms the absence of any acute spinal injury. Transfer patient onto Neuro ICU bed maintaining spinal alignment.
  - a** Patient should be placed in hard collar
  - b** Transfer of patient will require spinal turns or use of scoop.
  - c** 30° head up tilt to whole bed
- 3** Neurosurgical team should be alerted to arrival of patient (as an emergency if either pupil is fixed)
- 4** Transfer to Neuro ICU ventilator and monitoring (settings as per transfer)
- 5** Targets
  - a** SpO<sub>2</sub> >97%
  - b** PEEP 5cm H<sub>2</sub>O
  - c** EtCO<sub>2</sub> 4.0-4.5
    - Measured EtCO<sub>2</sub> is generally lower than PaCO<sub>2</sub>
  - d** MAP > 90mmHg
    - Ensures cerebral perfusion pressure (CPP)>60mmHg in patients with ICP<30mmHg



Suspect hypovolaemia in all trauma patients where MAP target is not achieved with low dose phenylephrine to offset the vasodilatory effect of propofol. Beware patients that may have bled from scalp or facial fractures (blood loss at scene, or concealed eg in stomach) prior to admission, and resuscitate all hypotensive patients as if hypovolaemic. NB In patients with small extradural haematoma not causing midline shift, raising the BP may expand extradural, therefore, discuss with Neuro ICU consultant.

- 6** Sedation & analgesia
  - a** Adequate sedation : propofol 2-4mg/kg/hr
  - b** Appropriate analgesia according to injuries : fentanyl 50-200mcg/hr
- 7** Take full handover from anaesthetic team, including:
  - a** Mechanism of injury
  - b** Extraction time
  - c** Initial and post resuscitation GCS scores, with breakdown of score (Eyes, Voice & Motor). Pupils & lateralising signs.
  - d** Episodes of hypoxia / hypotension
  - e** Grade / difficulty with intubation
  - f** Management prior to arrival on Neuro ICU, including all medication and fluids given.
  - g** CT Findings
  - h** Premorbid history, drug & allergy history
  - i** Antibiotics / tetanus / pregnancy test
  - j** Details of patient and relatives if known.
- 8** Full examination of patient to ensure that all injuries are assessed and prioritized. Back of patient, including scalp, must be examined during spinal turn. Consider referral to other specialties.
- 9** Complete the admission proforma in detail.
- 10** Check arterial blood gas, inserting arterial cannula if not already present
- 11** Target:
  - a**  $\text{PaO}_2 \geq 13 \text{ kPa}$
  - b**  $\text{PaCO}_2$  4.5-5 kPa
  - c**  $\text{Hb} > 10 \text{ g/dl}$



**12** Baseline investigations:

- a** FBC & Clotting screen
- b** Group & Save
- c** U&Es, LFT, CRP
- d** Chest X-Ray
- e** ECG
- f** Cardiac enzymes & echo if severe chest trauma.

NB check any blood tests performed already. (Any patient having urgent neurosurgical procedures should have a platelet count and coagulation screen prior to procedure.)

**13** Prescription of drugs to include:

- a** Gut protection (**ranitidine 50mg IV tds** initially, unless on a proton pump inhibitor already)



Gut protection is continued for entire Neuro ICU stay in brain or spinal cord injured patients, as this patient group is associated with a high risk of peptic ulceration - Cushing ulcers

- b** Laxatives
- c** Phenytoin (1g loading dose, IV over 1 hour, if not already given. Followed by 300mg IV/NG daily for 7 days in total) unless patient sedated with high dose midazolam. Set review date at 1 week and on discharge.
- d** Tetanus if open wound (Revaxis 0.5ml IM)
- e** Pneumovax should be given to all patients with a base of skull fracture or with the presence of intracranial air.
- f** Antibiotic treatment of any open skull fracture (skull fracture with scalp injury that is either overlying or that may be communicating. The presence of intracranial air should alert you to this possibility, although if derived from mastoid air cells antibiotics would not necessarily be required.) Treat with **cefuroxime 1.5g IV tds** for 5 days.



Base of skull fracture or rhinorrhoea are not automatic indications for antibiotics.

- g** **Pabrinex 1 pair BD** for 72 hrs for all suspected alcohol abusers
- h** Start insulin sliding scale if appropriate. Target blood glucose 4-8mmol/l.

**14** Review all imaging, and ensure consultant radiology report complete.

- a** Chest x-ray: eg check COET tube position (between clavicular heads and carina), presence of pneumothorax
- b** Trauma CT : eg abdominal viscus injury, fractures of spine / ribs, pneumothorax
- c** Head CT: eg presence of blood, midline shift, fractures
- d** CT angiogram is required for all patients with a base of skull fracture that crosses the course of the internal carotid artery, or where basal subarachnoid blood is present, to exclude any vascular injury. This should be delayed in patients:
  - That have already had a trauma CT with contrast, to reduce risk of contrast injury to kidneys.
  - That have cardiovascular instability, until patient is stable and with normalized renal function.

**15** Ensure patient has urinary catheter

**16** Insertion of ICP monitor by neurosurgeon/technician

- Ensure adequate analgesia and sedation for procedure
- Procedure checklist should be completed by any member of the team
- Ensure adequate asepsis and draping of the patient to exclude risk of contamination

### Management following ICP monitor insertion

- Maintain cerebral perfusion pressure (CPP) >60mmHg.
  - (CPP=MAP-ICP)
- Ensure adequate volume expansion using boluses of 0.9% Saline. Suspect hypovolaemia in all trauma patients.
- An isolated head injury is rarely the cause of hypotension, however, haemorrhage from scalp wounds and facial fractures may cause significant blood loss. This may not be evident at the time of patient's admission.
  - 1st line vasopressor : phenylephrine infusion (can be given via peripheral line)
  - 2nd line vasopressor : noradrenaline infusion (via central line only)



Subclavian lines are the preferred site for central venous access on Neuro ICU, in view of use of hard collars or possible angiograms. However, if a subclavian line cannot be easily inserted, or there is abnormal clotting or platelets, then internal jugular or femoral access should be used. Ultrasound should always be used to confirm the position of the vein, but need not be used for insertion.

- Once noradrenaline infusion >10ml/hr
  - Start hydrocortisone 100mg IV tds
  - Consider fludrocortisone 100mcg ng tds
  - Set up LiDCO
  - Ensure ST segment monitoring is operational on monitor
- If ICP>25mmHg, check pupils and consider repeat CT head to exclude further haemorrhage /haematoma expansion
- Follow ICP management algorithm



#### Beware ICP monitor drift:

- If ICP is considered to be inappropriately high for the CT scan appearance (eg ICP>20 with gyri and sulci visible over the surface of the brain) it may be appropriate to check the zeroing of the ICP bolt. This will involve removing the monitor in an aseptic manner and verifying that it reads zero.
- Drift can also occur to read an inaccurately low ICP, so if a pupil dilates despite the monitor reading a normal ICP value, a CT scan must be performed immediately and consideration given to changing the monitor.

## Levels of ICP management

Level	Intervention
1	Sedation & optimisation
2	EVD placement
3	Cool to 35°
4	Cool to 34°
5	Thiopentone coma or decompressive craniectomy

At all stages consider evacuation of intracranial haematoma.

## Level 1 : Sedation & optimisation

- Positioning
  - Nurse 30° head up. Initially this may require head up tilt of whole bed, until spinal management algorithm complete.
  - Ensure head and neck position and alignment. Flexion of neck/ twisting of head may compromise venous drainage via internal jugular veins, increasing ICP.
  - Tube ties should be secure but not constricting.
  - Ensure cervical collar is not so tight fitting that it raises ICP.
- Adequate sedation with midazolam
  - High dose midazolam is the preferred sedative on Neuro ICU for patients undergoing ICP control. Midazolam reduces cerebral metabolic rate and raises seizure threshold.



When changing the sedation from propofol to midazolam, as part of ICP control, the propofol must be continued for 1 hour after starting the midazolam infusion. Stopping the propofol immediately may result in the patient becoming inadequately sedated, and their ICP increasing.

- For patients <60yrs
  - Start at 20mg/hr (10ml/hr of double strength midazolam)
  - Escalate as necessary to obtund response to interventions
  - Up to a maximal dose of 120mg/hr (30ml/hr of quad strength midazolam [4mg/ml]).
- For patients ≥60yrs
  - Start at 10mg/hr
  - Ensure that dose does not escalate excessively.
- Appropriate analgesia with morphine
  - Morphine is the preferred intravenous analgesic on Neuro ICU and should be titrated to injuries and response to interventions.
  - For patients <60 years:
    - Start at 5mg/hr
    - Escalate to 10mg/hr if responding to interventions
    - Only escalate further ( up to a maximum of 25mg/hr) if injuries expected to cause severe pain (eg unstable rib/pelvic fractures)
  - For patients ≥60 years:

- Start at 2mg/hr
- Ensure that dose does not escalate excessively.
- Maintain  $\text{PaO}_2 \geq 13\text{kPa}$ 
  - $\text{PaO}_2$  is related to mean airway pressure and  $\text{FiO}_2$
  - Set PEEP appropriate to lung pathology (eg collapse / consolidation on CXR) and body habitus.
    - Eg start with PEEP at 5cm in patients with a BMI<30 and a normal CXR
  - Increase PEEP incrementally ensuring ICP is not affected adversely to a maximum of 15cmH<sub>2</sub>O. (Up to 20cmH<sub>2</sub>O in extreme cases)
  - Set  $\text{FiO}_2$  to achieve  $\text{PaO}_2 \geq 13\text{kPa}$
  - As oxygenation improves, initially reduce  $\text{FiO}_2$  sequentially down to 28%, maintaining PEEP. Only reduce PEEP once  $\text{FiO}_2 < 0.3$  with  $\text{PaO}_2 > 13\text{kPa}$ .
  - Consider
    - Treatment of bronchospasm with salbutamol nebulisers
    - Fibreoptic bronchoscopy if segmental or lobar collapse
    - Drainage of pneumothorax / haemothorax
- Target  $\text{PaCO}_2$  4.5-5.0kPa
  - $\text{PaCO}_2$  is inversely related to minute ventilation (minute ventilation=tidal volume x respiratory rate)
  - Set tidal volume appropriate to lung pathology (eg collapse / consolidation on CXR) and body habitus.
    - Patients with lung pathology require lower tidal volumes to prevent exacerbating lung injury. Aim for 6ml/kg
    - In patients with no evidence of lung injury, aim for 7-8ml/kg
  - Set respiratory rate to control  $\text{PaCO}_2$ . Start at 12 breaths/minute (range 10-24 breaths/min)



Cerebral blood flow is usually halved during the first 24 hours of traumatic brain injury (TBI). Therefore, patients whose ICP remains <15mmHg for the first 24-48 hours, may be ventilated to normocapnia ( $\text{PaCO}_2$  5-6 kPa). This will maximise cerebral blood flow during this critical period, minimise ventilator damage to injured lungs, and allow moderate hyperventilation as a therapeutic option to help control ICP if it should rise >20mmHg thereafter, when hyperaemia of the brain would be expected.

- Ventilation will normally start with PCV-VG. This mode targets the set tidal volume while minimising peak airway pressures.
- In any patient with ICP>20mmHg aim for Na>140mmol/l, except in cases of chronic hyponatraemia, where a gradual increase towards normal range may be appropriate.
  - 0.9% saline is the fluid of choice for fluid challenges
  - 0.9% saline with 0.3% KCl is the fluid of choice for maintenance (125ml/hr)
  - 1.8% saline may be infused peripherally up to 70ml/hr
  - 5% saline may only be infused centrally and only on Neuro ICU consultant instruction



Avoid hypotonic solutions (dextrose, dextrose saline and hartmanns) which will result in worsening cerebral oedema.

- Changes in sodium should not occur faster than 1mmol every 2 hours
  - These patients may develop diabetes insipidus, with resulting rapid rise in sodium. Regular checking of sodium on blood gases is essential, with stopping of hypertonic saline if rising too fast. These patients may require [DDAVP 0.5mcg IV stat](#).
- Target CPP of 60mmHg
  - Evidence suggests that a CPP<60mmHg and / or ICP>25mmHg is associated with a worse outcome in patients with traumatic head injury
  - In patients that are globally autoregulating, modest elevations in CPP may be associated with a decrease in ICP. However a higher CPP may worsen cerebral oedema, and increased use of vasopressors may be detrimental.
- Avoid pyrexia
  - Pyrexia is associated with raised cerebral metabolic rate and worse outcome
  - Use regular [paracetamol 1g NG/IV qds](#) (if patient <50kg consider 750mg qds)
  - Consider use of cooling line if unable to control temperature with surface cooling
  - Treatment of any underlying infection essential
- Early enteral feeding

- Start enteral feeding as soon as haemodynamically stable
- Aim to provide 30kCal/kg/24hrs initially. NB Patients with traumatic brain injury require 140% of normal expected intake, due to a rise in metabolic rate. Recommended normal calorie intake (ie not TBI):
  - Males : 2500 kCal/24hrs (In TBI aim for 3500 kCal/24hrs)
  - Females : 2000 kCal/24hrs (In TBI aim for 2500 kCal / 24hrs)
- Feeding should be continuous over 24hrs. (Rest periods may increase risk of peptic ulceration, reduce calorie intake and worsen control of blood glucose.)
- **Never** reduce calorie intake for fluid balance reasons. Change to concentrated feed (energy feed, if not already on it)
- Prescribe:
  - 2 bags of energy feed at 1ml/kg/hr (Energy feed contains 1.5kCal/ml)
  - 1 bag of multifibre (500ml) per 24 hours, at 1ml/kg/hr. (ie 70ml/hr for 70 kg patient)
  - Adjust prescription according to dietitian advice
- If failure to absorb:
  - 1st line : metoclopramide 10mg IV tds
  - 2nd line : erythromycin 250mg NG/IV qds (review at 72 hours)
  - 3rd line : Consider placement of jejunal feeding tube using Cortrak system
- Ensure tight seizure control. Start prophylactic phenytoin on admission (see above). However, high dose midazolam increases seizure threshold (ie patient less likely to fit). Seizure activity causes a rise in cerebral metabolic rate, which is likely to worsen outcome
- Neuromuscular blockade (pharmacological paralysis.)
  - Start once patient fully sedated, to avoid venous hypertension associated with coughing and straining
  - Infusion of atracurium at 0-10ml/hr after bolus of 1mg/kg
  - Target 1 twitch on train of four (TOF)
  - Place electrodes along line of ulnar nerve (medial aspect of palmar side of wrist.)
  - Observe thumb movement (Opponens pollicis)
- Spikes of ICP>25mmHg for >5 minutes should be managed:
  - a Check position of head and neck, and patient head up 30°

- b** Examine the patient to exclude the presence of pneumothorax, migration of the endotracheal tube causing one lung ventilation, etc
- c** Bolus of midazolam (3ml of current infusion), and consider increasing rate if ICP improves
- d** Check ABG to ensure no hypoxia / hypercarbia –adjusting ventilator settings as appropriate
- e** Give :
  - If **no** central access: **100 ml mannitol 20% IV**
  - Repeated use of mannitol is an indication for the insertion of a central line.
  - If central access : **50-100ml 5% saline IV**
  - 5% saline can be given provided serum sodium <150mmol/l, and serum osmolality < 320mosmol/kg
- f** Consider CT scan of head and escalation to Level 2 of algorithm
- g** Check with neurosurgeons whether CT head should be stereotactic scan to guide EVD placement.

## LEVEL 2 – EVD placement

Liaise with neurosurgeons about insertion of external ventricular drain at this stage

- An EVD allows drainage of CSF, aiding ICP control
- Drainage of even small volumes of CSF can have a significant effect on ICP when the brain is poorly compliant
- Delay in insertion risks increased technical difficulty in placement of drain, as ventricles may become slit like.
- If EVD placement is delayed until patient cooled, there may be an increased risk of bleeding.
- An EVD provides gold standard ICP measurement.
- Manage spikes in ICP as above
- A repeat CT head must be performed if ICPs continue to spike after insertion of EVD, to check position and rule out haematoma.



**LEVEL 3 – Cool to 35°**

Cooling reduces cerebral metabolic rate by 6% for each 1°C and will usually reduce ICP.

- 1** Insert femoral cooling line
  - Standard Seldinger technique under US guidance
  - Aspirate and flush infusion ports with saline and clamp off. These should only be used in emergency to reduce the risk of infection.
- 2** Cool patient to 35°
- 3** Reduce rate of atracurium infusion as elimination is temperature dependent
- 4** Check PaCO<sub>2</sub>, and reduce minute volume as necessary (metabolic rate is temperature dependent.)
- 5** If patient is receiving propofol then this should be replaced by midazolam (increased risk of propofol infusion syndrome)
- 6** Enteral feed should only be reduced if the patient has already achieved full enteral feeding for several days
- 7** Any signs of infection must be treated aggressively when the patient is cooled.
- 8** Manage spikes in ICP as above

**LEVEL 4 – Cool to 34°**

Cooling to 34° should lower cerebral metabolic rate further, however may be associated with more complications.

- Manage spikes in ICP as above

**LEVEL 5 – Thiopentone coma or decompressive craniectomy**

Currently this is part of an international research trial – Rescue ICP. Where there is equipoise, the patient should be randomised after assent from relatives.

**THIOPENTONE COMA**

Commencing a patient on thiopentone coma must only happen after discussion with the Neuro ICU consultant.



Thiopentone coma will reduce the cerebral metabolic requirement for oxygen by approximately 50%. Associated cerebral vasoconstriction reduces cerebral blood volume and ICP.

- 1** Patient must have cerebral function monitor (CFM, modified EEG) monitoring started prior to bolus of thiopentone
- 2** Make up a 25mg/ml solution of thiopentone with 3 ampoules (500mg per ampoule) of thiopentone, in 60mls of water for solution.
- 3** Bolus thiopentone in 10ml aliquots (250mg).
- 4** Boluses will reduce EEG activity, eventually producing short periods of isoelectric EEG (flat line) interspersed with bursts of activity.
- 5** Target 3-4 bursts per minute (this equates to 1-2 bursts per screen on the CFM monitor)
- 6** At this stage, start infusion of thiopentone at 12ml/hr.
- 7** Reassess EEG every 5 minutes and, if patient not adequately burst suppressed, give further boluses (rather than increasing infusion rate), until patient remains burst suppressed on re-assessment.
  - Once burst suppression maintained, gradually reduce infusion rate.



Once hepatic enzyme activity is saturated, thiopentone undergoes zero order kinetics (constant rate of metabolism independent of plasma concentration), with consequent rapid rise in plasma levels.

- Boluses of thiopentone may significantly lower blood pressure. Ensure CPP maintained by use of fluids and vasopressors.
- Once burst suppression maintained, stop midazolam, morphine and atracurium infusions. Consider slow rewarming.

**COMPLICATIONS OF THIOPENTONE**

- **Hypotension** – correct with fluids and vasopressors
- **Sepsis** – high dose thiopentone suppresses immunity. Treat any evidence of infection early and aggressively.

- **Hypokalaemia** – a relative hypokalaemia can be caused by high dose thiopentone, possibly due to ion pump inhibition. Beware rebound hyperkalaemia on stopping the infusion.
- **Hypernatraemia** – thiopentone has a high sodium content and may cause an appropriate polyuria with normal urine osmolality. (Use urine dipstick assessment of specific gravity to differentiate from diabetes insipidus.)

#### DISCONTINUATION OF THIOPENTONE

- Once ICP controlled for 24 hours, consider slow wean of thiopentone infusion.
- Watch for rebound hyperkalaemia – stop potassium supplementation temporarily
- Once thiopentone infusion stopped, consider restarting morphine and midazolam infusions at low dose.

#### DECOMPRESSION

- Check clotting, platelets and group & save.
- Decompressive craniectomy can result in significant blood loss – check whether crossmatch of blood products necessary.
- Neurosurgeon and anaesthetist must be contacted
- Noradrenaline should be infused via a syringe driver (4mg in 50mls) rather than from an infusion pump.
- Following return from theatre
  - Full set of bloods should be sent
  - Continue current level of ICP management for at least 24 hours, then gradual de-escalation required.
  - Patient should be assessed, and assessment documented in the notes. NB Pupils, ICP (this may be a subdural catheter or EVD)
- ICP should be significantly lower following decompression. Whilst an ICP<10 is acceptable, any ICP>15 is an indication to assess the tension in the flap, alert the neurosurgeon, and consider performance of a CT scan. Any patient with an ICP>20mmHg for greater than 30 minutes should be immediately rescanned.

## Management of self ventilating patient with head injury

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- 1** Rapid assessment of patient including:
  - A Airway** : Maintained and clear. No signs of upper airway obstruction
  - B Breathing**: Adequate rate and depth of respiration with SpO<sub>2</sub>>97%
  - C Circulation**: Assess HR & BP
  - D Disability**: GCS, pupils, lateralising neurology. (Including dysphasia)
- 2** All patients should be assumed to have unstable spinal injury unless spinal algorithm has been completed.
  - Patient should be placed in hard collar
  - Transfer of patient will require spinal turn or use of scoop, maintaining spine alignment
  - 30° head up tilt to whole bed
- 3** Patients should not be triple-immobilised once on the bed. (Triple immobilisation keeps the patient's head still, but the rest of their body may move resulting in malalignment of their C-spine.)
- 4** Neurosurgical team should be alerted to arrival of the patient (as an emergency if either pupil is fixed)
- 5** Take full handover, including:
  - a** Mechanism of injury
  - b** Extraction time
  - c** Initial and subsequent GCS scores, with breakdown of score
  - d** Episodes of hypoxia / hypotension
  - e** Grade / difficulty with intubation, if performed for CT scan
  - f** Medication and fluids given
  - g** Premorbid history, drug & allergy history
  - h** Antibiotics / tetanus / pregnancy test
  - i** Details of patient and relatives if known.
- 6** Full examination of patient, completing admission proforma in detail (see admission section). Back of patient, including scalp & PR, must be examined during spinal turn.
- 7** Take blood for venous blood gas, FBC, U&E, Coag and group & save. NB check any blood tests performed already. (Any patient having urgent neu-

rosurgical procedures should have a platelet count and coagulation screen prior to procedure).

**8** Ensure the prescription of:

- Gut protection ([ranitidine 50mg IV tds](#) initially, unless on a proton pump inhibitor already)
- Laxatives
- Consider phenytoin (1g loading dose if not already given, followed by 300mg IV/NG daily) in patients with:
  - History of seizures
  - Skull fracture
  - Extradural haematoma managed conservatively
  - Temporal lobe damage (eg contusions)
- Tetanus, if open wound (Revaxis 0.5ml IM)
- Pneumovax, if presence of intracranial air
- [Pabrinex 1 pair IV bd](#) for 72 hrs for all suspected alcohol abusers
- Antibiotic treatment of any open skull fracture
- Insulin sliding scale if appropriate

**9** Review all imaging, and ensure consultant radiology report complete

- Chest X ray: eg ng tube position, presence of pneumothorax
- Trauma CT : eg abdominal viscus injury, fractures of spine / ribs, pneumothorax
- Head CT: eg presence of blood, midline shift, fractures

**10** Consider urinary catheter / convene

**11** Nutrition of these patients must be considered as soon as patient is haemodynamically stable and has consistent neurology. This may require assessment of swallow or insertion of NG tube.

**12** Manage post head injury agitation as per algorithm. See Chapter on management of the agitated patient



Any patient with a head injury who is sedated to a level that they cannot be clinically assessed should be discussed with the neurosurgeons for consideration of ICP monitoring.

## General principles

- Safety of patients and staff are paramount
- Aim to minimise pharmacological sedation by careful nursing care in a quiet environment
- Reduce stimulation. Eg remove urinary catheter if causing irritation, but ensure bladder empty regularly with bladder scan
- Suppositories to ensure bowels open regularly
- Identification and treatment of any underlying cause should ideally precede pharmacological control of the agitated patient, unless the patient presents a danger to themselves or staff. (Eg hypoxia, hypoglycaemia, sepsis) Some medications (eg high dose dexamethasone) have been associated with agitation in patients. It may be appropriate to consider weaning the dose or even temporarily suspending such medication. However, in the case of steroids and anticonvulsants, this must be discussed and agreed first with both the Neuro ICU consultant and the patient's neurosurgical/neurological consultant.
- Whilst haloperidol is considered to prolong post-traumatic amnesia in traumatic brain injured patients, it may be used to rapidly gain control of severely agitated patients, whilst introducing other agents
- Hourly assessment of the patient using the Richmond Agitation Sedation Score (RASS) should be documented on the observation chart
- Any patient that is aggressive, scoring +3 or +4 on RASS, must be brought to the attention of the Neuro ICU consultant and have agitation controlled urgently
- Any patient that remains aggressively agitated and uncontrollable may require intubation and ventilation for their safety and the safety of staff



Any patient with a head injury who is sedated to a level that they cannot be clinically assessed should be discussed with the neurosurgeons for consideration of ICP monitoring.

## Richmond agitation-sedation score (RASS)

Score	Term	Description
<b>+4</b>	Combative	Overtly combative, violent, immediate danger to staff
<b>+3</b>	Very Agitated	Pulls or removes tubes or catheters; aggressive
<b>+2</b>	Agitated	Frequent non-purposeful movement, fights ventilator
<b>+1</b>	Restless	Anxious, but movements not aggressive or vigorous
<b>0</b>	Alert and Calm	
<b>-1</b>	Drowsy	Not fully alert, but has sustained awakening
<b>-2</b>	Light Sedation	Briefly awakens with eye contact to voice
<b>-3</b>	Moderate Sedation	Movement or eye opening to voice
<b>-4</b>	Deep Sedation	No response to voice, but movement or eye opening to physical stimulation
<b>-5</b>	Unroutable	No response to voice or physical stimulation

- 1** Observe patient
- 2** Patient is alert, restless, or agitated: Score 0 to +4
- 3** If not alert, state patient's name and say to open eyes and look at speaker. If patient:
  - Awakens with sustained eye opening and eye contact: Score -1
  - Awakens with eye opening and eye contact, but not sustained: Score -2
  - Has any movement in response to voice but no eye contact: Score -3
- 4** When no response to verbal stimulation, physically stimulate patient by shaking shoulder and / or applying painful stimulus.
  - Patient has any movement to physical stimulation: Score -4
  - Patient has no response to any stimulation: Score -5

## Management of agitation

---

- Adequate analgesia
  - Regular paracetamol
  - Morphine 1mg IV prn (Max 1mg per 15mins)
- If alcohol withdrawal likely, start:
  - Chlordiazepoxide if oral route available
    - Initial dose of 10mg NG/PO to assess response
    - Escalate as necessary up to 30mg 4hrly
  - Diazepam (diazemuls preparation) for IV route
    - Initial dose of 5mg IV to assess response
    - Escalate as necessary up to 20mg tds
- If known to be smoker, start nicotine patch
- If known IV drug abuser, may need opioid replacement

## Patients with a traumatic brain injury

Patients with traumatic brain injury are at particular risk from side-effects of medication used to control agitation. Eg prolongation of post traumatic amnesia with haloperidol.

### WEANING FROM HIGH DOSE SEDATION

Prior to extubation

- 1** If known or suspected problems with anger management, start **carbamazepine 200mg NG bd** prior to reducing sedation. Beware the risk of SIADH with carbamazepine - monitor serum sodium closely. Ensure indication and review date is documented in notes and discharge/transfer summary.
- 2** If sedation prolonged greater than 5 days, or patient is hypertensive & tachycardic, start clonidine 0-3ml/hr as sedation is reduced, and continue post extubation. Aim to wean off within the first few days following extubation.
- 3** Once midazolam and morphine have been stopped, it may be necessary to start an infusion of propofol for tube tolerance, prior to extubation.
- 4** Consider commencing olanzapine if patient remains agitated prior to extubation, to facilitate reduction of propofol.



Following extubation, if agitation remains a problem manage as below.

#### SELF-VENTILATING TBI PATIENT

- 1** 1st line: **olanzapine 2.5mg NG**, repeated after 2hrs up to max 20mg per 24hrs. (Consider insertion of NG under sedation by consultant anaesthetist/intensivist)
- 2** 2nd line: If patient is hypertensive and tachycardic, consider using **propranolol 40mg NG 8 hourly** or a **clonidine IV infusion at 0-3ml/hr**
- 3** 3rd line: If known or suspected problems with anger management, start **carbamazepine 200mg NG bd** prior to reducing sedation. Beware the risk of SIADH with carbamazepine - monitor serum sodium closely. Ensure indication and review date is documented in notes and discharge/transfer summary.
- 4** 4th line: Low dose propofol infusion (for short term control under medical supervision. This requires Neuro ICU consultant approval)
- 5** Aim to avoid haloperidol (prolongs post traumatic amnesia), unless it is necessary to gain rapid control of a severely agitated patient RASS +3 or +4
- 6** Consider diazepam (beware of prolonged duration of action and active metabolites in the elderly) at a dose of 5mg NG tds initially
- 7** If patient deteriorates or agitation cannot be controlled, then sedation, intubation and ventilation may be necessary to ensure safety of patient and staff.

## **Management of the Agitated Brain Injured Patient on the Neurointensive Care Unit.**

### **Assessment / considerations**

This guidance applies to any level 1, 2 or 3 patient in the Neuro ICU.

Before commencing pharmacological measures ensure the following are excluded:

1. Pain
2. Constipation
3. Urinary retention
4. Hunger
5. Poorly fitting cervical collar or thoraco-lumbar brace
6. Sepsis
7. Hypoxia and/or Hypocarbica or hypercarbia
8. Withdrawal from: alcohol, normal psychiatric medication, nicotine, Illicit drugs

### **General measures**

The following should be considered:

1. Appropriate analgesia is prescribed and administered as per Neuro ICU protocol
2. Ensure bowels open recently (ideally should be open daily – especially spinal cord injury patients)
3. Make sure urinary catheter is free flowing. Use bladder scan to confirm empty bladder. Consider removal of catheter and use of convene/urine bottles.
4. Ensure patient has access to appropriate food and drink or is being adequately fed by a gastric tube.
5. Check cervical collar or thoraco-lumbar brace is fitted correctly. Remove cervical collar if not required.
6. Exclude sepsis or start appropriate treatment (Including central line removal and antibiotics).
7. Check adequate oxygenation with saturation monitor. Arterial blood gas to exclude inadequate ventilation
8. Make sure a full history of dosing regimes for normal medications is noted and prescribed appropriately.
9. Remove all unnecessary monitoring equipment.
10. Avoid excessive audible and visual stimulation
11. Consider gentle tactile reassurance with hand holding instead of active restraint.
12. Provide suitable “toy” for patient to hold/play with. E.g. circle of ventilator tubing.
13. Identify the most appropriate mattress to use to allow movement of patient but not allowing harm to occur.
14. In case of severe psychotic reaction, contact neuropsychiatrist to explore assessing mental capacity and utilising Deprivation of Liberty Safeguard, before considering allowing patient to self discharge from hospital.

### **Pharmacological management**

For drug withdrawal consider the following:

1. Alcohol: Adequate Chlordiazepoxide dosing or other benzodiazepine
2. Psychiatric medication: Reinstate normal regime as soon as possible.
3. Nicotine: Use nicotine patches and wean over 3 weeks.
4. Illicit drugs: Obtain accurate history and replace with adequate doses of methadone +/- benzodiazepines.

If these measures are not adequate and the patient is at risk of harming themselves or staff, consider:

1. Olanzapine 2.5mg -10mg PO/NG. Repeat after 2 hours if necessary. Max 20mg combined daily dose
2. If hypertensive & tachycardic: Propranolol at 40mgPO/NG 8hrly or Clonidine 0-3 ml/hr (750mcg in 50mls 0.9% Saline [15mcg/ml])
3. Carbamazepine 200mg NG bd
4. Propofol infusion. Only following discussion with Neuro ICU consultant.
5. Propofol bolus (1 – 2 ml). This can only be given by charge nurse after prescription by Neuro ICU Consultant.
6. Consider diazepam (beware of prolonged duration of action and active metabolites in the elderly) at a dose of 5mg NG tds initially.

If the above measures have not been successful, sedate, intubate and ventilate the patient.

## Non-traumatic brain injury patient

- 1** Give **olanzapine 2.5-5mg NG stat**, and assess response to guide dosage for regular treatment. Continue a suitable dose of olanzapine regularly bd up to max 20mg per 24hrs. (Consider insertion of NG under sedation by consultant anaesthetist/intensivist)
- 2** **Haloperidol 2mg-4mg IV prn** up to a total dose of 18mg per 24 hours, may be used for acute control
- 3** If patient is hypertensive and tachycardic, consider using clonidine infusion at 0-3ml/hr (Beware hypotension, particularly if agitation may be secondary to vasospasm.)
- 4** Consider diazepam (beware of prolonged duration of action and active metabolites in the elderly) at a dose of 5mg NG tds initially.
- 5** Low dose propofol infusion (for short term control under medical supervision. This requires Neuro ICU consultant approval)
- 6** If patient deteriorates or agitation cannot be controlled, then sedation, intubation and ventilation may be necessary

## RASS & Wessex modified RASS

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### Richmond Agitation-Sedation Score (RASS)

Use this for agitated patients, and those that are **pharmacologically** sedated.

Score	Term	Description
+4	Combative	Overtly combative, violent, immediate danger to staff
+3	Very Agitated	Pulls or removes tubes or catheters; aggressive
+2	Agitated	Frequent non-purposeful movement, fights ventilator
+1	Restless	Anxious, but movements not aggressive or vigorous
0	Alert and Calm	
-1	Drowsy	Not fully alert, but has sustained awakening
-2	Light Sedation	Briefly awakens with eye contact to voice
-3	Moderate Sedation	Movement or eye opening to voice
-4	Deep Sedation	No response to voice, but movement or eye opening to physical stimulation
-5	Unroutable	No response to voice or physical stimulation

### Wessex modified RASS for stroke & brain injured patients

Use this for patients who have a reduced level of consciousness for **pathological** reasons.

Score	Term	Description
0	Alert and Calm	
-1	Sustained interaction	Able to sustain meaningful interaction, but if left alone may become less alert
-2	Stimulated interaction	Requires repeated verbal stimulation to maintain interaction
-3	No meaningful interaction	Verbal or motor response to voice, but no meaningful interaction
-4	No response to voice	No response to voice, but verbal or motor response to physical stimulation
-5	Unroutable	No response to voice or physical stimulation

# Spinal precautions for the trauma patient

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All patients with any traumatic brain injury (including acute or chronic subdural haematoma) **must** have full spinal precautions, and be managed as per spinal algorithm, unless:

- Patient was receiving any antiplatelet / anticoagulant agent (including aspirin) and had no history of significant trauma (eg fall less than patient's own height / low energy injury to head).
- Patient had witnessed isolated head injury

These patients must still have C-Spine immobilisation with a hard collar, until CT cervical spine has been reported by a consultant radiologist and does not show signs of acute bony injury. However, provided that the patient does not complain of pain or have tenderness of their thoracolumbar spine, they may be sat up and turned normally (with head hold if ventilated).



There are 3 main components to spinal column precautions:

1. **Hard collar or no hard collar** : Initially all trauma patients should be managed in a hard collar until it has been demonstrated that there is no evidence of bony injury to C-spine or occipital condyles on CT
2. **Sat up or bed flat** : Initially the bed should be flat (i.e. not 'broken') and tilted 30° head end up. Sitting the patient up may cause further harm in a patient with thoracolumbar injury
3. **How the patient is turned** : Initial management is full spinal turns, to maintain spinal alignment

## Transfer of patients with spinal precautions

The transfer of patients with full spinal precautions requires the use of a spinal board or scoop with triple immobilisation of the cervical spine. Once the patient is stable in bed, the patient can be managed with a hard collar and supports either side of the head to maintain cervical spine alignment.



Patients with unstable cervical column spinal injuries should be placed in a Miami J collar. This should only be removed as part of washing & pressure area care.

Patients with stable cervical column spinal injuries may have the front of their hard collars removed whilst they are sedated, paralysed and not being turned or undergoing physio, after agreement with the spinal surgeon.

## Clinical assessment to exclude bony cervical injury

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This is unlikely to be able to be performed on the admission of any patient with a traumatic brain injury.

The cervical spine may only be assessed clinically in adults if:

- The patient is fully conscious (GCS 15/15)
- There is no central neck tenderness
- There is no neurological deficit
- There are no distracting injuries e.g. limb or rib fractures
- There are no confounding factors such as drugs or alcohol that may mask injury

If none of the above is present, the collar can be removed and the patient asked to rotate their neck 45° to the left and then to the right. If full movement is possible without pain, the spine can be cleared clinically without the need for radiographs.

## Sedated & ventilated patients

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This should be read in conjunction with the “Guidelines of initial spinal management of sedated and ventilated trauma patients” form, which must be completed. A copy of this form is at the end of this chapter.



All patients should have full spinal precautions, until spinal management algorithm completed.

- All trauma patients require CT head & cervical spine and thoracolumbar imaging, where there has been:
  - An unknown mechanism of injury
  - Or fall greater than patients own height
  - Or high energy impact
  - Or age >50 (increased risk of degenerative spine)
  - This should take the form of a full trauma CT scan when performed as the initial imaging
- Trauma patients who do not fulfil the above list may have just CT head and cervical spine. However, where acute cervical spine injury is then demonstrated, they MUST be managed with full spinal precautions until thoracolumbar imaging has been performed and reported to show no acute injury
- Patients who have had only a CT head and cervical spine (eg in their referring hospital), but fulfil the above criteria MUST also be managed with full spinal precautions until thoracolumbar imaging has been performed and reported to show no acute bony injury
- The radiological imaging must be reported by a consultant radiologist. The report of spinal imaging should include :
  - Any inadequacy of imaging
  - Fractures
  - Alignment
  - Soft tissue swelling indicative of ligamentous injury
  - The name of the consultant radiologist who has reported this imaging. This must be documented on the guideline form.
- Where there is no injury reported on the imaging of the spine, no occipital condyle fracture, and while the patient is sedated:
  - The hard collar can be removed
  - The patient may be sat up (NB except in patients with unstable pelvic injury)
  - The patient can be turned normally
- Once the sedation is reduced, allowing patient to move:
  - Where there is evidence of vertebral fusion due to a disease process or operation, or where there is evidence of extremely high velocity injury, the patient must be placed back into a hard collar until they can be clinically assessed or an MRI can be performed demonstrating stability.

- Otherwise, the patient does not require a hard collar unless they complain of neck pain or develop neurological symptoms eg paraesthesia, or signs eg weakness.
- If any spinal injury is reported by the consultant radiologist, the patient must be reviewed by a consultant spinal surgeon to determine a management plan and decide what nursing care is necessary to ensure alignment is maintained. See guidelines.
- All patients may be managed 30° head up, unless there is a spinal cord injury above T6 and the patient is weaning / self ventilating. If there is unstable thoracolumbar injury then the bed must be tilted rather than the patient sat up.
- Patients with unstable thoracolumbar injury, but no cervical spine injury, do not need a head hold during turns once they are extubated and able to control their own head.
- Patients with a spinal cord injury above T6 should be nursed flat once spontaneously ventilating. These patients may have no intercostal muscle function, so are reliant on diaphragmatic excursion. When flat the process of expiration is aided by abdominal pressure. Sitting or tilting these patients up reduces this effect, and will reduce their tidal volumes. This may cause a rise in PaCO<sub>2</sub>, despite normal oxygen saturations.
- Side lie all patients with or without spinal injury to prevent pressure sores, unless specifically contra-indicated (eg unstable pelvis).



## Guidelines for initial spinal management of sedated and ventilated trauma patients

University Hospital Southampton **NHS**  
NHS Foundation Trust

**Full spinal precautions**

↓

Unknown mechanism of injury or fall greater than patient's own height or high energy impact or age >50

Yes ☐ No ☐

**1**

**Full Trauma CT or CT Head & Cervical spine & thoracolumbar imaging**

**CT Head & Cervical spine**

Bony injury of C-Spine → **Thoracolumbar spinal imaging**

Hospital No: \_\_\_\_\_

DOB: \_\_\_\_\_

Surname: \_\_\_\_\_

First name: \_\_\_\_\_

ADDRESSOGRAPH LABEL

Signed: \_\_\_\_\_

Print: \_\_\_\_\_

Date: \_\_\_\_\_

Time: \_\_\_\_\_

**Consultant radiologist report**

Report of spinal imaging to include:  
?any inadequacy of imaging  
?fracture ?alignment (dislocation)  
?soft tissue swelling indicative of spinal injury

Cervical (C-spine) imaging report  
Consultant name: \_\_\_\_\_

Thoracolumbar (T&L spine) imaging report  
Consultant name: \_\_\_\_\_

**2**

Injury reported on imaging of spine

No ☐ Yes ☐

Signed: \_\_\_\_\_

Print: \_\_\_\_\_

Date: \_\_\_\_\_

Time: \_\_\_\_\_

A normal CT does not exclude ligamentous injury.

**Consultant spinal Surgeon**

Name: \_\_\_\_\_

Management plan / nursing care to ensure alignment maintained

Stable C-spine  
Stable T&L spine

Stable C-spine  
Unstable T&L spine

C-spine stable in hard collar  
Stable T&L spine

Unstable C-spine +/-  
Unstable T&L spine

**A**

No hard collar  
Patient sat up  
Normal turns

☐

**B**

No hard collar  
Bed tilted head up  
Full spinal turns

☐

**C**

Hard collar  
Patient sat up  
Normal turns with head hold

☐

**D**

Hard collar  
Bed tilted head up  
Full spinal turns

☐

**3**

ICU Consultant signature: \_\_\_\_\_

Print: \_\_\_\_\_

Date: \_\_\_\_\_

Time: \_\_\_\_\_

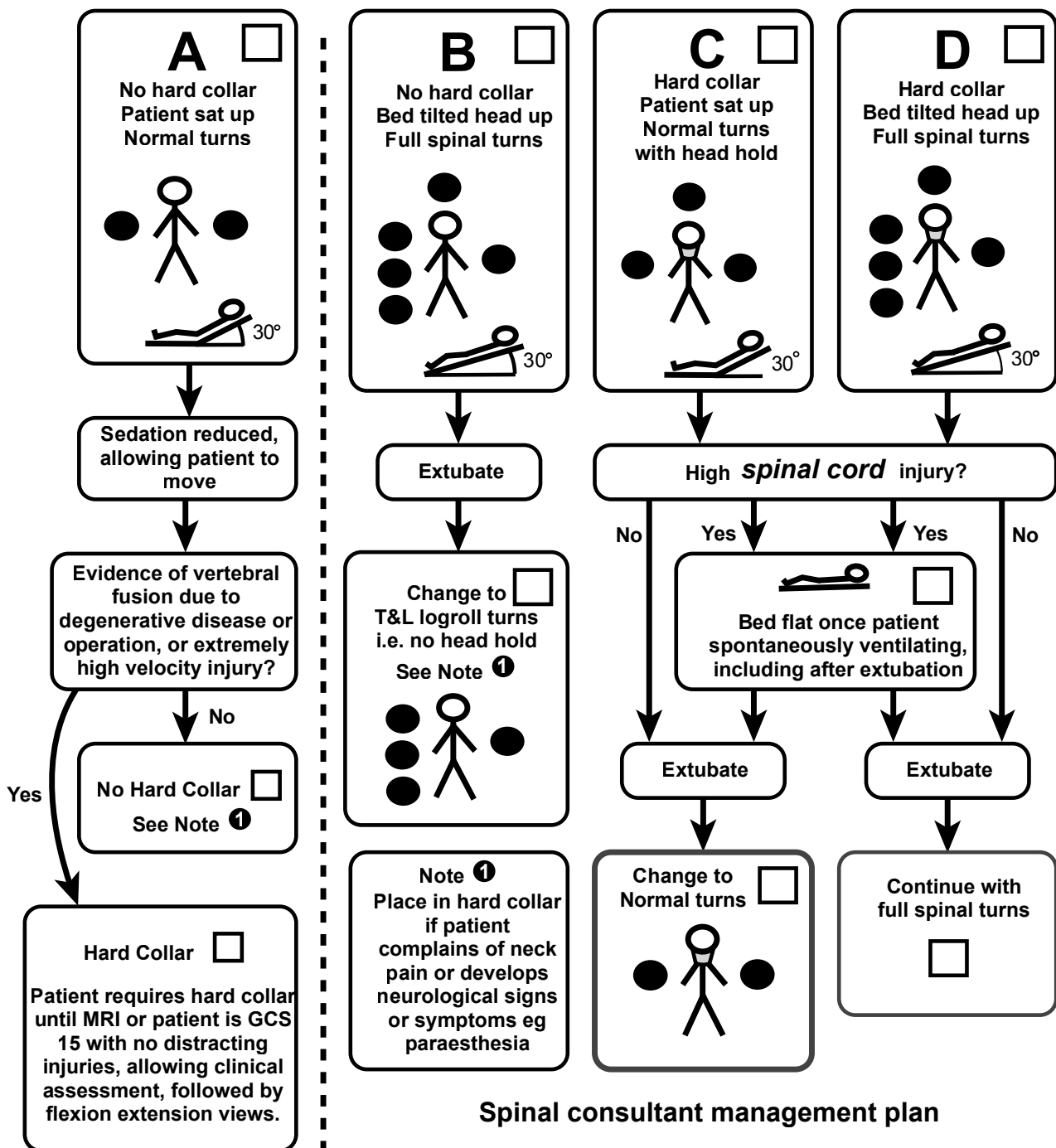
**Trauma Patient Spinal Management**

Sept 2012, Version 4.2

Mark identical box A-D over the page →

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### Management plan for when sedation is reduced



### Spinal consultant management plan

Name of spinal consultant:

Comments

- ☐ Hard collar for ..... weeks
- ☐ Halo jacket
- ☐ Extension brace

#### ALL PATIENTS

Side lie ALL patients to prevent pressure sores, unless specifically contra-indicated. Eg unstable pelvis

☐ Contra-indicated  
Reason .....

4

ICU Consultant signature:  
Print:

Date:  
Time:

## Patient assessment

---

### History

On admission to Neuro ICU of any patient with a known or potential spinal cord injury, the following points should be determined:

- Nature and mechanism of injury
- Complete / incomplete / absent spinal cord injury at the scene or in the referring hospital
- Methylprednisolone given / not given. The consensus opinion from the Wessex Neurological Centre is that steroids should not be given
- Other injuries

### Examination

- 1 Initial assessment:
  - A **Airway** and Cervical spine immobilisation
  - B **Breathing**: Adequacy of oxygenation & ventilation
  - C **Circulation**: Fluid resuscitation (in trauma patients hypotension should always be considered to be secondary to blood loss, not spinal shock)
  - D **Disability**: GCS, pupil size
- 2 Associated injuries including pressure areas
- 3 Level of spinal injury
  - radiologically
  - clinically
  - complete / incomplete / absent

- ## American Spinal Injury Association (ASIA) Score

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**ASIA IMPAIRMENT SCALE**

- ☐ **A = Complete:** No motor or sensory function is preserved in the sacral segments S4-S5.
- ☐ **B = Incomplete:** Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4-S5.
- ☐ **C = Incomplete:** Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade less than 3.
- ☐ **D = Incomplete:** Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3 or more.
- ☐ **E = Normal:** motor and sensory function are normal

**CLINICAL SYNDROMES**

- ☐ Central Cord
- ☐ Brown-Sequard
- ☐ Anterior Cord
- ☐ Conus Medullaris
- ☐ Cauda Equina

**Investigations**

- Routine bloods and arterial blood gas
- Trauma CT (or equivalent CT imaging of spine), 10-15% of patients with a spinal injury, have an injury at another level.
- Magnetic resonance imaging (MRI) of spine

## Monitoring

- Pulse oximetry
- ECG
- NIBP
- Twice daily Vital Capacity in non-ventilated patients
- Monitor urine output with a urinary catheter

## Management

---

### Oxygenation and ventilation

- All patients should receive supplemental oxygen to maintain  $\text{SpO}_2 \geq 98\%$
- Patients with cord injury at C3 or above will require intubation and ventilation, and early consideration for tracheostomy, as there will be inadequate phrenic nerve innervation of diaphragm.
- All other patients with cervical or high thoracic (T6 and above) cord injury are at risk of inadequate ventilation.
  - Non-invasive ventilation (NIV) should be instituted after admission to Neuro ICU, as prophylaxis against the development of atelectasis and infection.
  - Use of the 'Bird respirator' with the physio
    - Allows resting from the facemask, washing of the face, communication etc
    - Facilitates secretion removal as part of chest physio
  - The level of ventilatory function can be expected to further deteriorate over the subsequent days due to:
    - spinal cord oedema. This should improve as oedema resolves
    - lack of intercostal tone and diaphragm fatigue
    - lack of sympathetic innervation causing bronchoconstriction and increased secretions.
  - During this deterioration patients will require more frequent turning (to facilitate secretion removal), increased non-invasive ventilation (NIV) pressures (increased IPAP) and the use of facial NIV rather than nasal NIV.

- Commence all patients on salbutamol nebs (lack of sympathetic tone to lungs will result in bronchoconstriction) and consider Otrivine nasal spray for self ventilating patients to offset nasal stuffiness.
- Secretions commonly settle before spinal shock has worn off. With an improvement in secretions the patient may tolerate nasal NIV rather than facial NIV
- Ventilation will improve as intercostal and abdominal muscle tone increases, with cessation of the initial flaccid paralysis (passing of spinal shock). This should result in an improvement in the patients vital capacity.
- When non-invasive ventilation is to be instituted, the nasal route is preferred initially for patient tolerance and comfort (provided vital capacity (VC) >1.5L) . Alternating nasal cushions with nasal masks may help avoid the development of pressure sores. Patients may require a nasogastric tube to be inserted to allow decompression of the stomach, and later supplementation of nutrition. (Nasal NIV remains effective in the presence of a nasogastric tube.)
- Patients should be changed to a face mask for NIV when they worsen with increased secretions (3-5 days post injury) or if they have a VC<1.5L
- Patients should be nursed in a horizontal position (flat), with turns to side lie ≤ 3hrly. The frequency of turns should be increased when the patient worsens with increased secretions
- Once physiotherapy assessment of respiratory function and vital capacity indicates that ventilation is not made worse by head up tilt, then this may be started slowly with frequent re-assessment.



When the patient is flat the abdominal contents help push the diaphragm cranially during expiration, improving tidal volumes in patients that have no intercostal function that are solely reliant on diaphragmatic excursion for ventilation. This is most marked in the spinal shock stage.

- A maintained  $\text{SpO}_2 \geq 98\%$  does not indicate adequate minute ventilation. Any patient that appears fatigued, has a raised respiratory rate, has deteriorating vital capacity, or a  $\text{PaCO}_2 > 6.4 \text{ kPa}$ , despite non-invasive ventilation, should be considered for early intubation and ventilation.
- Chest X-ray changes suggestive of progressive atelectasis, or with pulmonary contusions, should prompt consideration of increasing ventilator support.
- Patients with spinal cord injuries higher than T6, that have required intubation and ventilation should:
  - be managed with the bed flat (rather than head up) once they are on a spontaneous mode of ventilation and weaning.

- only be considered for extubation once secretions have settled
- be extubated to NIV on a weekday morning to maximise physiotherapy involvement. They are likely to require use of the Bird respirator with the physio to aid secretion removal.
- When considering patients for tracheostomy:
  - Careful consideration will need to be given to issues of spinal instability, which may dictate a surgical tracheostomy rather than percutaneous placement. Percutaneous tracheostomy can be associated with greater posterior force during dilation of the tract.
  - Patients that may require an anterior approach to cervical stabilization, should not have a tracheostomy placed prior to operation or within 10 days of operation.
  - The presence of a hard collar can make the physical management of a tracheostomy extremely difficult. It may be preferable to delay tracheostomy until a hard collar is no longer required.
- When intubating and ventilating a patient, rocuronium would usually be the muscle relaxant of choice, although suxamethonium can be used up to 48 hours post-injury, if felt indicated.



Use of suxamethonium >48 hours post injury may cause a sudden onset of profound hyperkalaemia that will result in cardiac arrest.

## Analgesia

- Paracetamol 1g IV/NG 6 hourly
- Consider use of **ibuprofen 600mg PO/NG tds** at 48 hours after injury (Avoid earlier use because of risk of exacerbating any haemorrhage at site of trauma, and risk of causing peptic ulceration in patients that are not receiving enteral nutrition. These patients may have an ileus and poor gastric emptying for up to 48 hours.) **Check with spinal surgeons prior to starting NSAID.**
- Careful titration of IV opiates. **Morphine 1mg IV PRN 15 mins**, unless patient is able to operate a PCA. (Morphine 1mg/ml, with 1mg bolus and 5 minute lockout)
- Patients with neuropathic pain should start **amitriptyline 10mg PO nocte**, and may require gabapentin escalated slowly from 300mg PO daily. Tramadol may be a better opioid than morphine for neuropathic pain, because of its adjunctive actions on noradrenaline and serotonin receptors. (NB tramadol lowers the seizure threshold, ie makes it more likely that a patient may fit, so should be avoided in any patient with intracranial pathology.)



### Cardiovascular management

- Ensure haemorrhage controlled and patient adequately fluid resuscitated (target urine output  $>0.5\text{ml/kg/hr}$ ). Presume hypotension due to blood loss until proven otherwise.
- Ensure blood pressure is adequate to allow normal mentation (in non-TBI patients) and urine output  $>0.5\text{ml/kg/hr}$ . Where there is concern about spinal cord perfusion (eg evidence on imaging of compression of spinal cord) consider maintaining a higher MAP eg  $\geq 85\text{mmHg}$ .
- Spinal shock may produce hypotension, bradycardia and poikilothermia. This results especially from injuries above T6.
- Unopposed vagal activity, due to loss of sympathetic outflow (thoracolumbar), may cause profound bradycardia and syncope. This may be triggered by airway manipulation such as intubation, suctioning, or changing tracheostomy tube. Acute treatment may require atropine, but this may be avoided through prophylactic use of glycopyrrolate prior to procedures .

### Gastro-intestinal management

- All patients with high spinal cord injury (above T6) should be started on full dose proton pump inhibitors (eg **pantoprazole 40mg IV od**, followed by **lansoprazole 30mg NG od** after 48 hours) on admission, to help prevent development of peptic ulceration. This is a particularly high risk group because of unopposed vagal nerve activity.
- Paralytic ileus is common after spinal cord injury. Patients with spinal cord injury T6 or higher should be kept **nil by mouth** for the first 48 hours. This includes omitting oral medication.
- Thereafter, establish early enteral feeding with **metoclopramide 10mg IV tds** if necessary. (Avoid erythromycin in unintubated patients because of nausea and vomiting, which is a very common side-effect of this drug).
- Where high gastric aspirates persist with nasogastric feeding, despite prokinetics, a nasojejunal tube should be placed.
- Ensure bowels are evacuated daily. Commence glycerine suppositories and regular aperients (lactulose 10ml BD, senna 2 tabs nocte) once absorbing. Daily manual evacuation may be necessary.

### Venous thromboembolism (VTE) prophylaxis

- Full length Anti-Embolic Stockings (AES)

- Full length Intermittent Pneumatic Compression (IPC) devices.
- Start **enoxaparin 40 mg SC daily** following agreement with spinal surgeon that risk of further intra-spinal haemorrhage is low.

### Avoidance of pressure sores

- Patients must be managed on a spinal bed with a pressure-relieving mattress.
- Three hourly turns to side-lying is mandatory, with observation and documentation of pressure areas at each turn.
- Avoid hypoxia and hypotension (target  $\text{PaO}_2 \geq 12\text{kPa}$  and MAP adequate to ensure urine output  $>0.5\text{ml/kg/hr}$ ), which may contribute to inadequate tissue perfusion.)
- Ensure adequate protein and calorie nutrition (target  $30\text{kCal/kg/24hrs}$ )
- Orthopaedic casts should be avoided in this group of patients to prevent development of plaster sores. This patient group is at high risk because of lack of sensation and complete immobility. Alternative stabilization of fractures may need to be considered (eg external fixation).
- Joint contractures may develop rapidly following spinal cord injury. Liaise with the physiotherapists regarding passive movements and splinting. The two main areas spinal patients develop contractures are:
  - Elbows : this is most likely to occur in patients with biceps power (C5) but no triceps power (C6). These patients may need 'pillow splints' to keep elbows straight
  - Feet: patients with weakness of the lower limbs should have their feet propped up at  $90^\circ$  (dorsiflexed) with a pillow. These patients may require splints in the longer term.

### Immobilisation

- Remove patient from spinal board or scoop as soon as possible.
- Spinal management as per spinal algorithm (see spinal management chapter)
- A hard cervical collar and a firm mattress are the standard means of immobilisation before the application of traction or definitive stabilisation.
- Cervical collars
  - Replace the temporary hard collar with which patients are initially immobilised in the Emergency Room, by a two piece collar, such as the Philadelphia collar.

- For longer term use consider a Miami J or Aspen collar as they may be more comfortable and less likely to cause pressure areas. The Miami J collar has a separate back section that is padded to reduce occipital sores.
- Soft collars have no role in stabilising spinal injury.
- The decision to remove spinal protection should only be made after appropriate investigations have been completed and reported, as per spinal management algorithm.
- Patients with stable cervical spine injuries may have the front section of their hard collars removed whilst they are sedated, paralysed and not being turned or undergoing physio, after agreement with the spinal surgeon.
- Patients with unstable cervical spine injuries and raised ICP may have the front of their collar loosened whilst they are sedated, paralysed and not being turned or undergoing physio.
- For transfer of patients:
  - To CT: Patients with full spinal precautions should be placed on a scoop or a spinal board with a pressure relieving gel mattress.
  - To MRI: Patients should be transferred with full spinal precautions on to the MRI table from their bed.
  - Patients in traction will require placement in a hard collar prior to the release of traction. In the case of MRI this must be followed by removal of skull callipers if these are not MRI compatible.
- It is inappropriate for patients to be left on spinal boards for prolonged periods (e.g. operative procedures or for hospital transfer).

## Autonomic dysreflexia

Autonomic dysreflexia is associated with lesions above C6 and does not occur until spinal shock has passed. When it does occur, it is in response to noxious stimuli perceived below the level of the lesion. Sympathetic discharge causes sweating, vasoconstriction with hypertension and a reflex bradycardia. Causes include:

- Bladder distension. Commonest cause is blocked catheter. All patients should have silicone catheters inserted. Consider suprapubic catheter placement if long term use anticipated.
- Bowel distension; ensure daily bowel evacuation, manual evacuation may be required.
- Fracture below the level of the lesion

- Pressure sore
- Urinary tract infection / bladder spasm
- Deep Vein Thrombosis
- Surgery on area below level of spinal injury

### SIGNS AND SYMPTOMS

- Hypertension. May be severe
- Bradycardia
- Headache
- Sweating above level of lesion
- Pallor below level of lesion
- Flushing / blotchiness above level of lesion
- Nasal congestion
- Bronchospasm, dyspnoea
- Diplopia
- Nausea, vomiting

### TREATMENT

- 1 Remove cause
- 2 Analgesia if appropriate (IV opiates)
- 3 Local anaesthetic blockade eg femoral nerve block for femoral fracture.
- 4 Treatment of hypertension with vasodilator drugs
  - a nifedipine 10mg PO
  - b hydralazine 10-20mg IV titrated to response

## **Steroid treatment in acute spinal cord injury**

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Following publication of NASCIS I, II, III, and the Cochrane Review, there is on-going debate as to the usefulness and risks associated with high dose methyl prednisolone in acute traumatic spinal cord injury. The consensus opinion of the spinal surgeons in University Hospital Southampton, is that methyl prednisolone is not indicated in this situation.

However if the spinal cord injury has been caused by surgical removal of a tumour, then treatment with dexamethasone may be appropriate.

# Spontaneous subarachnoid haemorrhage

# 17

## Introduction

Spontaneous subarachnoid haemorrhage (SAH) typically presents with either a history of severe headache or a patient in coma, sometimes following a seizure. Although confirmation of SAH may require a lumbar puncture, most patients admitted to the Neuro ICU as a result of SAH have evidence of a bleed on CT scan.

Patients with SAH are commonly classified by two grading systems. World Federation of Neurosurgeons (WFNS) score grades patients according to best initial neurology post SAH bleed. Fisher scale classifies according to CT Scan appearance of blood load.

## World Federation of Neurosurgeons (WFNS)

I	GCS 15	No motor deficit
II	GCS 13-14	No motor deficit
III	GCS 13-14	Motor deficit present
IV	GCS 7-12	Motor deficit present/absent
V	GCS 3-6	Motor deficit present/absent

## Fisher scale

1	No clot seen on CT scan
2	Diffuse thin layer of subarachnoid clot (<1mm thickness)
3	Localised clot or thicker layer of subarachnoid clot (>1mm thickness)
4	Intracerebral or intraventricular clot with diffuse or no subarachnoid clot

## Complications of SAH

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- **Death** from raised intracranial pressure resulting in coning, or as a result of myocardial infarction/arrhythmia
- **Myocardial ischaemia/infarction/stunning**: Intense sympathetic discharge causing increased myocardial contraction against a vasoconstricted circulation may result in myocardial ischaemia/infarction. Takotsubo cardiomyopathy may result in severe cardiac failure.
- **Neurogenic pulmonary oedema**: this is multifactorial. Myocardial contraction against increased afterload results in functional mitral regurgitation, acutely increasing left atrial pressure and resulting in pulmonary oedema. This is compounded by myocardial ischaemia and subsequent impairment of left ventricular function. In addition to this, there may be increased permeability of pulmonary vasculature.
- **Re-bleed**: This is most common in the first 24hrs post SAH (>2% of patients will rebleed in first 24 hours), but may occur at any time (1% of patients will rebleed per day thereafter). This risk is dramatically reduced by coiling or clipping. Re-bleed increases risk of complications and may cause death.
- **Seizures**: These may occur at presentation or at any time thereafter. Treatment is with phenytoin 1g IV loading over 1 hour, followed by 300mg nocte.



A rebleed may present in a similar fashion to a seizure. Any patient with a history of SAH who has a drop in GCS, must have an urgent CT Brain to exclude rebleed.

- **Hydrocephalus**: These patients are at risk of communicating hydrocephalus due to blockage of arachnoid granulations by breakdown products of blood. They may occasionally have obstructive hydrocephalus due to compression of the CSF outflow tract. Blood within the ventricular system rarely causes complete obstruction to CSF flow. Hydrocephalus will usually present as a low GCS in a patient with a heavy blood load. Treatment involves drainage of CSF either by lumbar puncture or external ventricular drain (EVD). Patients with obstructive hydrocephalus must have drainage by EVD.
- **Delayed ischaemic neurological deficit (DIND) / vasospasm**: This is most likely to occur from day 4 to day 10, but may occur at any time from 72 hours to 3 weeks. Vasospasm is more common in poor WFNS grade SAH with high blood load and occurs earlier in young patients. The resultant cerebral ischaemia typically produces focal neurological deficits (eg dysphasia/hemiplegia). Any new neurological deficit should prompt the performance of a CT scan. If this does not demonstrate rebleed or hydrocephalus, then the patient should be actively managed with hypertensive therapy for presumed vasospasm. Transcranial dopplers, and CT Perfusion may help in determining this

diagnosis, although positive response to hypertensive therapy is often more useful.

- **Electrolyte disturbances:**

- **Cerebral salt wasting** – These patients are at risk of natriuresis (Excess loss of sodium and water in the urine) which is thought to be secondary to release of hormones (eg brain natriuretic peptide) from the CNS. Watch out for low serum sodium with low serum potassium and a raised urea, with a negative fluid balance. Notoriously difficult to diagnose in the presence of adequate fluid resuscitation.
- **Diabetes insipidus** – This is more common in patients with a heavy blood load and raised intracranial pressure, which affects the release of antidiuretic hormone from the posterior pituitary. Watch out for excess volume of dilute urine (>200ml/hr for 2 hours consecutively, or >400mls in a single hour) with a urine specific gravity <1.01 (Checked with a dipstick) and a rise in serum sodium on blood gases. If serum sodium >145mmol/l, treat with DDAVP 0.5mcg IV, which may be repeated one hourly if necessary.
- **SIADH** – These patients may develop hyponatremia secondary to inappropriate ADH release. Watch out for low serum sodium with a positive fluid balance. (A low or normal urea and a low or normal urine output may be present.) This is difficult to diagnose and use of paired serum and urine osmolality and sodium may be helpful. Treatment is with hypertonic saline, and avoiding excess hypotonic fluid ingestion. Fluid restriction carries the risk of hypovolaemia and impaired cerebral perfusion, which may exacerbate delayed ischaemic neurological deficit. **Avoid** V2 receptor antagonists(eg tolvaptan, covivaptan) which may cause excessively rapid increase in serum sodium.

## Management protocol

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All patients with acute SAH who have had aneurysm clipping or coiling should be admitted to ICU or HDU, for the above reasons.

### On admission

- 1 Clerk the patient (noting the success or otherwise of the procedure, complications, previously noted neurological deficit, timing and history of SAH).
- 2 Full examination, including groin site and foot pulses, if coiled.





The arterial territory injured affects the clinical signs:

**ACA/ACom:** more likely to cause weakness in lower limbs

**MCA:** may affect upper limbs and speech

**Basilar tip:** may cause homonymous hemianopia and in severe cases multiple cranial nerve deficits.



Check patient for presence of femoral artery catheters. If these have not have been removed at the end of coiling, they will require removal usually 12 hours post procedure. This must be done at the beginning of the next day shift, rather than overnight. Prior to removal check that platelets >100, INR <1.4 & APTR <1.4. Only remove one sheath at a time, and apply secure pressure for a minimum of 15 minutes with the patient flat. Only remove the second sheath once there is no bleeding or swelling at the site of the first sheath.

- 3** Investigations on admission:
  - a** Routine bloods and arterial blood gas
  - b** ECG – note any territorial ischaemia, evidence of left ventricular hypertrophy. ECG changes are almost universal following SAH. Any patients with ischaemic changes on ECG should have:
    - Repeated 12 lead ECGs every 6 hours, for the first 24 hours, and then daily
    - Troponin in all patients with abnormal ECGs or difficulty oxygenating
    - Echo to assess ventricular function and regional wall abnormalities
  - c** CXR – These patients are at high risk of having aspirated following SAH, may have neurogenic pulmonary oedema, or may have developed significant atelectasis following prolonged anaesthesia.
- 4** Traditionally these patients have been nursed flat for at least 24 hrs, to improve cerebral perfusion. However, they may be sat up if necessary to improve respiratory function.
- 5** Prescribe :
  - a** Senna 2 tabs PO/NG nocte and lactulose 10ml PO/NG bd
  - b** Paracetamol 1g PO/IV qds so long as patient >50kg
  - c** Nimodipine 60mg PO/NG 4hrly for 21 days from last SAH
  - d** Ranitidine 50mg IV tds or 150mg PO/NG bd if absorbing
  - e** Morphine 1mg IV PRN every 15mins
  - f** Phenytoin 300mg NG/IV nocte if patient has had a seizure.

- g** Aspirin MAY be required post coiling – check post procedure instructions.

## Monitoring

- Pulse oximetry.
- ECG.
- NIBP in addition to IABP.
- Urinary output.
- Full set of daily bloods
  - These patients are at risk of impaired renal function secondary to high dose radio-contrast media. May develop cerebral salt wasting, diabetes insipidus, or SIADH, or a combination.
- Transcranial doppler : Transcranial dopplers assess the velocity of blood through the middle cerebral artery on both sides, and compare it with the velocity in the internal carotid arteries of both sides. An increase in velocity above normal for that age group, a significant difference in velocity between the two sides, or a Lindegaard Ratio (MCA/ICA velocity) >3 is indicative of cerebral artery constriction suggestive of vasospasm. Anterior cerebral artery and basilar artery may also be assessed. TCDs are routinely performed Monday, Wednesday & Friday, but may be performed at any time that a suitably qualified medical technician is available, provided that a request card has been filled out. Aim to obtain baseline TCDs within first 3 days of ictus, rather than waiting for neurological deficit to occur.

## Fluid management

- 1** Start IV fluid supplementation with 0.9% saline  $\pm$  0.3% KCl at 125ml/hr and reduce according to oral/enteral fluid intake.
- 2** Reduce IV fluid administration in the elderly to ensure total fluid administration, including nutrition, of approximately 1ml/kg/hr.
- 3** In first 3 days following SAH, the risk of vasospasm is minimal, so mean arterial pressure only needs to be adequate to maintain urine output >0.5ml/kg/hr over 4 hours.
- 4** Where neurology is clinically assessable, the MAP target can be adjusted to maintain stable neurology.
- 5** Establish early enteral feeding

## Targets

- $\text{SpO}_2 > 97\%$  or  $\text{PaO}_2 > 13\text{kPa}$
- Normal  $\text{PaCO}_2$  (4.3-6.4kPa)
- Mean arterial pressure (MAP):
  - Prior to the securing of a ruptured aneurysm, the treatment of extreme hypertension may be considered:
    - Particular care must be taken when reducing blood pressure in patients who have had a SAH. They tolerate relative hypotension poorly and may suffer cerebral infarction, particularly if they have any vasospasm, hydrocephalus, raised intracranial pressure or distortion of blood vessels around ICH.
    - It is vital to be sure about the date of the 1st bleed, and beware any sentinel bleed. The patient may have a degree of vasospasm at the time of admission to Neuro ICU, and require a high MAP for cerebral perfusion.
    - Any limits to blood pressure must be agreed by both the Neuro ICU consultant and the consultant neurosurgeon, and this be documented in the notes.
    - There are risks associated with severe hypertension, eg rebleed, expansion of ICH & cardiac ischaemia. Hypertension with a  $\text{MAP} > 140\text{mmHg}$  is an indication for gradual control of the blood pressure.
    - Short duration agents should be used. The preferred method on Neuro ICU is with a labetalol infusion.
    - Any limits to the blood pressure MUST be stopped once the aneurysm has been protected.
  - In the presence of vasospasm, 'MAP targets' may be set after discussion between the Neuro ICU consultant and the neurosurgical team. This target is likely to be higher than the patient's untreated MAP, particularly if the patient has developed a neurological deficit with the vasospasm.
  - In first 3 days, a MAP target may not necessarily be set provided patient is passing urine and/or mentating normally (see above)

## Neurological deterioration

If the patient deteriorates neurologically post-op:

- 1** Ensure airway protected (if GCS < 8 call the anaesthetist and discuss the need for intubation), give oxygen via non-rebreathe mask.
- 2** Inform Neuro ICU consultant and Neurosurgical SpR of any deterioration. Discuss with them the need for CT (to exclude a further bleed, hydrocephalus etc), and whether it is indicated to perform a CT perfusion urgently at this time.
- 3** If there is no evidence of rebleed or increased hydrocephalus to explain the neurological deterioration, vasospasm is likely to be the cause. Start hypertensive treatment and discuss with neurosurgical team to agree MAP target, and assess need for LP/CSF drainage and/or interventional angiography.

## Hypertensive guideline

Following adequate fluid resuscitation, ensure an appropriate hypertensive MAP to reverse any neurological deficit

- 1** Vasospasm is the likely diagnosis in a SAH patient >72hrs after bleed with worsening neurology (usually lateralising) who has no evidence of rebleed or worsening hydrocephalus on CT. TCDs and/or CT perfusion scanning may help confirm the clinical suspicion.



When CT perfusion is performed, it is essential to document the MAP at the time of the scan. This MAP is vital to the interpretation of the scan. A scan demonstrating normal perfusion at a high MAP does NOT exclude significant vasospasm. It may be that if the scan was performed at a lower MAP, ischaemia would be evident.

## **2** Fluid management

- a** Adequate fluid resuscitation with saline boluses to maximise MAP
- b** Aim for 3 litres total fluid input/24hrs, ensuring appropriate nutrition/calorie intake (minimum 25kCal/kg daily)
- c** If serum sodium low (<140mMol/l), start fludrocortisone 100mcg PO/NG tds

## **3** Hypertension



Patients with unsecured aneurysms must not receive vasopressor therapy to achieve therapeutic hypertension without prior discussion with their consultant neurosurgeon and the Neuro ICU consultant.

- a** Target an initial MAP of 110mmHg, or 20mmHg above current MAP, if higher.
- b** Increase stepwise to a MAP of 140mmHg to fully reverse neurological deficit. All MAP targets should be agreed with consultants.

- c** If volume loading insufficient to maintain MAP, start phenylephrine infusion (10mg in 500ml saline titrated to a maximum of 180ml/hr)
  - d** All patients that require hypertensive treatment should have an arterial line.
  - e** Change nimodipine 60mg 4hrly to 30mg 2hrly if administration is associated with decreased MAP.
  - f** Patients where MAP is not adequately maintained with phenylephrine infusion should have CVP line inserted and be started on Noradrenaline. CVP line placement should be confirmed with pressure transduction and CXR. Noradrenaline infusion (20mg in 250ml 5% Glucose) should be titrated to MAP target
  - g** If Noradrenaline requirement >10ml/hr
    - ensure ST monitoring in place
    - start **hydrocortisone 100mg IV tds** and convert ranitidine to a proton pump inhibitor (eg **lansoprazole 30mg PO/NG od**)
    - start **fludrocortisone 100 micrograms PO/NG tds**
    - consider LiDCO
  - h** Stopping nimodipine should be considered if its administration causes a decrease in MAP with neurological deterioration, or if noradrenaline infusion greater than 15ml/hr to achieve MAP target. (The stopping of nimodipine should be discussed with the Neuro ICU consultant.)
  - i** If MAP target not achieved with above measures and noradrenaline infusion at 40ml/hr, consider adding vasopressin infusion. (Max 3ml/hr). No patient is to be started on vasopressin without the express agreement of the Neuro ICU consultant.
  - j** Hypertensive therapy is generally continued for a minimum of 5 days or until neurology and TCDs have normalised.
  - k** Hypertensive treatment should be weaned off in a stepwise manner.
- 4** CSF drainage
- a** consider when maximal previous therapies (eg MAP target of 140mmHg) have failed to reverse neurological deficit
  - b** Liaise with neurosurgeons regarding lumbar vs EVD drainage
  - c** If lumbar drainage appropriate, perform LP and check opening pressure. If opening pressure >20cmCSF:
    - drain to halve initial pressure, or to 20cmCSF, whichever is lower. No more than 50mls should be drained at one time.
    - consider insertion of lumbar drain, ensuring drainage of 10ml/hr with EVD collection system.

## Deterioration following coiling

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If a patient deteriorates neurologically in the first 24 hours post-coiling:

- 1** The on-call radiology SpR and on-call neuroradiographer should be contacted to arrange an urgent CT scan.
- 2** Discuss the case immediately with the duty neurosurgical SpR, Bleep 2877, who should then contact the interventional neuroradiologist who performed the procedure or the on-call neuroradiologist if that person is not available.
- 3** If there is no other obvious cause for the deterioration on CT scan, it should be considered whether a coil has caused thrombosis or embolism. Where this is the case, the decision may be made to give Reopro.
  - a** Reopro (abciximab) is a potent antagonist of platelet aggregation.
  - b** Reopro (10 mg vial for reconstitution) may be obtained from the fridge in Neuro ICU or angio suite.
  - c** Administration is by slow (2 minutes) intravenous injection.
  - d** If no neurological improvement is recorded within 15 minutes, repeat the dose of Reopro to a maximum of 20 mg. (Check whether abciximab was administered during the interventional procedure earlier in the day).
  - e** Discuss subsequent heparinisation and/or aspirin therapy with the interventional neuroradiologist.
- 4** If the aneurysm is large or giant (> 12 mm) consider treatment with intravenous dexamethasone to reduce possible effects of peri-aneurysmal oedema related to thrombosis.
- 5** Continue close observation of femoral artery puncture sites, arterial lines, ventricular access devices etc, following administration of Reopro.

# Lumbar puncture

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## Indications

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- As part of a septic screen for investigation of pyrexia and raised inflammatory markers, particularly in patients who have had neurosurgery or an EVD
- Investigation / treatment of communicating hydrocephalus in patients with subarachnoid haemorrhage
- Investigation of various neurological conditions
- Treatment for patients with CSF leaks eg post transphenoidal surgery

## Contra-indications

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- Non-communicating hydrocephalus, raised intracranial pressure or posterior fossa lesions
- Abnormal coagulation / platelet function. LP should only be performed if INR<1.4, APTR<1.4 and >100,000 functioning platelets
- Any patient that has taken aspirin or clopidogrel should be considered on a case by case basis. Platelet cover may be appropriate if LP essential.
- The management of patients receiving dabigatran and other new anticoagulant agents should be discussed with haematology
- Patient refusal
- Less than 12 hours after enoxaparin administration. Enoxaparin must not be given less than 6 hours after a lumbar puncture
- Infection / recent surgery at the site of the lumbar puncture

## Procedure

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- 1** Full asepsis (Hat, gown, mask, gloves)
- 2** Patient positioning - this is the most important factor in success. Where possible the patient's head should be flexed forwards, their legs drawn up towards their abdomen and their back arched
- 3** 2% chlorhexidine, allow to dry fully
- 4** Adequate draping
- 5** Identify the level for the lumbar puncture - below Tuffier's line
- 6** Inject lignocaine
- 7** For hydrocephalus or CSF leaks a large bore cutting needle (20/18G) should be used. For neurological diagnosis or septic screen consider using a 22G atraumatic needle.
- 8** The opening pressure must be measured and recorded
- 9** Sterility must be maintained whilst collecting CSF into sterile bottles. CSF should always be sent for M,C&S.
- 10** If opening pressure >20cmCSF: drain to halve initial pressure, or to 20cmCSF, whichever is lower. No more than 50mls should be drained at one time.
- 11** Closing pressure should be measured and recorded
- 12** No more than 3 attempts should be made by any trainee/fellow before seeking senior help
- 13** Following LP:
  - Diagnostic : Patient should remain flat in bed for 6 hours
  - Therapeutic CSF drainage: Patient may be sat up immediately following procedure to allow CSF to continue draining
- 14** Concordant venous blood samples should be sent for blood glucose and protein estimation
- 15** The procedure must be fully documented in the patient's notes
- 16** Ensure CSF samples have been received by the lab and processed within 2 hours
- 17** Any abnormal results must be discussed with the Neuro ICU consultant immediately



# External ventricular drains

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## Introduction

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External ventricular drains (EVDs) are commonly used for:

- Treatment of hydrocephalus
- Measurement of ICP and the drainage of CSF, in patients with raised ICP
- Instilling intrathecal antibiotics for ventriculitis
- Instilling Urokinase in patients with severe intraventricular haematoma (Currently, only as part of Clear III trial.)

## Key points about EVDs

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- Bolt EVDs
  - May be placed on the neurointensive care unit for hydrocephalus.
  - May be inappropriate in severely agitated patients, who may dislodge them more easily than a tunnelled EVD that has been adequately sutured (including the connector with wings for suturing at the end of the EVD tubing).
  - Must be placed using full asepsis and draping of the whole patient and surroundings



Tunnelled EVDs must only be inserted in the operating theatre with full asepsis and draping of the whole patient, to reduce the risk of infection.

- ICP can only be accurately measured via an EVD when the drainage tubing is clamped. This should be performed hourly, unless the EVD is clamped pending removal.
- The height of CSF drainage from the EVD must be measured in mmHg, not cmH<sub>2</sub>O, to ensure consistent management on Neuro ICU.
- The EVD collection system must be zeroed to the external auditory meatus at all times. This will require the system to be raised or lowered as the patient's head is raised or lowered.

## CSF sampling

CSF sample to be taken from the EVD and sent for microscopy, culture and sensitivity (check glucose & protein if infection suspected and at initial insertion):

- On insertion
- Mondays and Thursdays
- At any time that GCS or neurology deteriorates if >24 hours after last sample
- From any patient with new onset neck stiffness (meningism)
- As part of a septic screen for any patient with pyrexia, raised CRP +/- WBC
- Prior to administration of intrathecal (IT) antibiotics via EVD



The following abnormalities should be escalated to the consultant neurosurgeon and Neuro ICU consultant urgently:

- Presence of organisms in the CSF. IV +/- IT antibiotics should be started
- A high white cell count with predominantly polymorphs. Consider IV antibiotics eg [cefotaxime 2g IV 4 hourly](#)
- A rising ratio of WBC to RBC, particularly if WBC : RBC ratio is greater than 1:1000 should also prompt consideration of IV antibiotics

**To accurately sample CSF from an EVD:**

- 1** If CSF output low, consider clamping 1 hour prior to sampling
- 2** Use full aseptic precautions, sterile gloves & sterile field
- 3** Dressing pack, 2% chlorhexidine wipes, 2x5ml syringes, replacement bung
- 4** Need assistant to place 3 way tap in sterile field
- 5** Thoroughly clean 3 way tap and tubing away from tap for 10cm in both directions
- 6** Allow chlorhexidine solution to dry
- 7** Turn 3 way tap off to air, remove and discard bung
- 8** Insert syringe into port using no touch technique
- 9** Turn 3 way tap to connect syringe to patient limb of EVD
- 10** Gently aspirate CSF (stop if resistance, keep clamped and try again in 1 hr)
- 11** Before removing syringe, turn 3 way tap off to syringe port
- 12** Discard 1st syringe containing at least 3 ml of CSF
- 13** Insert new syringe into port, turn 3 way tap to connect syringe to patient limb
- 14** Gently aspirate >2ml CSF
- 15** Close 3 way tap to syringe port, allowing free drainage again
- 16** Remove syringe and place new bung on port
- 17** Transfer CSF to sterile sample bottle, label and send to lab immediately
- 18** If clinical concerns out-of-hours, you must ensure that lab staff are waiting to receive sample, having already been alerted by phone, following request via e-quest
- 19** If giving IT vancomycin or gentamicin (pre-prepared syringes by pharmacy), you will need to leave EVD clamped for 1 hr (inform bedside nurse) having added volume of antibiotic & 1ml saline flush, so must ensure that adequate volume of CSF has been removed at steps 10 & 14
- 20** When injecting antibiotic or saline via 3 way tap injection port, always aspirate any air bubble into syringe from port (with tapping of syringe to move air), before injecting. (Avoids injecting bubbles of air into ventricles that can accumulate)

## Ventriculitis

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Clinical evidence of ventriculitis includes:

- Unexplained deterioration in GCS or neurology
- New onset of neck stiffness (meningism)
- Pyrexia
- Raised CRP and / or raised WBC. (NB ventriculitis may occur in the absence of raised inflammatory markers.)



Any patient with clinical evidence of ventriculitis must have CSF sampled as part of a septic screen, then be discussed with the Neuro ICU consultant and escalated to the consultant neurosurgeon within 1 hour, for decision regarding starting antibiotic treatment. This may require microbiological advice +/- result of CSF microscopy.

### Antibiotic management of ventriculitis

- Usual 1st line antibiotic: **cefotaxime 2g IV 4 hourly**
  - This may be started by the Neuro ICU consultant following a marked rise in CSF white cell count, in a patient with signs consistent with ventriculitis. Prior to giving the first dose of antibiotics a fresh CSF sample should be sent for M,C&S, unless already sent within 24 hours.
  - **Meropenem 2g IV 8 hourly** is considered 2nd line, and may be given to patients that have developed ventriculitis while on high dose antibiotics
- Intra-thecal antibiotics as guided by microbiological advice
  - Intra-thecal antibiotics are usually given to patients that have organisms seen on microscopy or cultured from the CSF
  - Usual dose **vancomycin 20mg IT od** (Pre-prepared syringe)
  - Usual dose **gentamicin 5mg IT od**

- Thrombolysis should be considered for all patients presenting acutely with suspected stroke, except where contra-indicated eg primary intracranial haemorrhage, previous gastrointestinal bleed or recent surgery.
- The initial management, CT scanning, and administration of thrombolysis should be performed by the appropriate medical team (usually the stroke team or neurologists).
- These patients should usually be managed on the stroke unit, but where expertise is not available out of hours to nurse these patients there, they may be admitted to Neuro ICU.
- Where thrombolysis is to be given on Neuro ICU, it is the responsibility of the stroke team or neurologist to ensure that it is safely administered.

## Initial management

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- 1 Admission clerking with full examination.
- 2 Check admission blood results – these should have been taken prior to thrombolysis. **No** routine blood tests post thrombolysis
- 3 Nurse head up 30°
- 4 Bed rest for 24 hours
- 5 Monitor ECG, SpO<sub>2</sub>, NIBP and neurology hourly
- 6 Do not pass NG tube for 24 hours. Can eat and drink once swallow assessed as adequate.
- 7 Particular care with procedures which may cause trauma e.g. mouth care, suctioning. Do not pass nasopharyngeal airway.
- 8 Avoid catheterisation. If essential, 30 minutes after completion of thrombolysis.

- 9** No arterial puncture or central lines.
- 10** Observe for all signs of bleeding or neurological deterioration e.g. puncture wounds, gastrointestinal bleeding, signs of raised intracranial pressure.
- 11** Monitor and record temperature, pulse, blood pressure, oxygen saturation, and neurological observations:
  - a** Every 15 minutes for 2 hours
  - b** Then every 30 minutes for 6 hours
  - c** Then every 60 minutes for 16 hours
- 12** No heparin, antiplatelet agents, warfarin or NSAIDS
- 13** Book CT brain scan for 24hrs post thrombolysis (or next available time during daylight hours)
- 14** Ensure stroke unit aware of patient and plan discharge from Neuro ICU at 24 hours.

## Potential complications of thrombolysis

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### Anaphylaxis

Extremely rare event and usually mild, but if severe should be treated according to current AAGBI guidelines.

### Hypertension post thrombolysis

- Treat a systolic BP >180mmHg or diastolic BP >105mmHg
- Give **labetalol 10mg IV over 1–2 minutes**
  - May be repeated every 5 minutes. If repeated doses required consider starting infusion
  - If BP remains uncontrolled with labetalol, consider the addition of further agents eg hydralazine, nifedipine or phentolamine (Discuss with Neuro ICU consultant)
- If patient requires BP intervention then increase observations to every 15 mins for duration of intervention

## Management of suspected intracranial haemorrhage (ICH)

Symptoms suggestive of ICH following thrombolysis include:

- Increased neurological deficit, including deteriorating level of consciousness
- New or increasing headache
- Acute hypertension (two successive readings over 10 minutes of systolic BP greater than 185 mmHg following thrombolysis)
- Nausea and vomiting

### MANAGEMENT OF SUSPECTED ICH

- 1** Stop infusion of thrombolysis
- 2** Ensure airway protected (if GCS < 8 call the anaesthetist and discuss the need for intubation), give maximum oxygen via non-rebreathe mask.
- 3** Alert stroke physician / neurologist
- 4** Arrange emergency CT scan
- 5** If CT scan confirms ICH, discuss with stroke team whether:
  - a** Haematological management of coagulopathy from thrombolysis is appropriate – discuss with haematologist the requirement for blood products (eg cryoprecipitate and platelets, 2–4 units of fresh frozen plasma may be infused to replenish Factors V and VIII.)
  - b** Neurosurgical team should be involved

## Management of extra-cranial haemorrhage

### BLEEDING FROM A COMPRESSIBLE SITE

Venflon sites, venepuncture sites, nose bleeds or other superficial sites.

- 1** Direct pressure
- 2** Apply dressings
- 3** Continue thrombolysis infusion unless bleeding uncontrollable
- 4** Resuscitation as necessary

**BLEEDING FROM NON-COMPRESSIBLE SITE**

Gastrointestinal, urogenital or retroperitoneal haemorrhage, or other parenchymal haemorrhage.

- 1** Stop thrombolysis infusion
- 2** Ensure adequate IV access for prompt fluid resuscitation
- 3** Take bloods:
  - Blood gas
  - FBC
  - U+E
  - PT, aPTT, Fibrinogen
  - X-Match 6 units packed RBC
- 4** Resuscitate with rapid 0.9% Saline infusion initially
- 5** Target Hb>10g/dl during acute bleed. Use O-ve blood whilst awaiting fully crossmatched blood to be available. (Closest blood fridge to Neuro ICU is D Level). Blood should be given through a blood warmer when possible.
- 6** Warn haematologist that patient is bleeding uncontrollably following thrombolysis, and request they provide appropriate products to correct coagulopathy (eg cryoprecipitate and platelets). Target a normal coagulation profile.
- 7** May need to seek surgical or interventional radiology assistance.

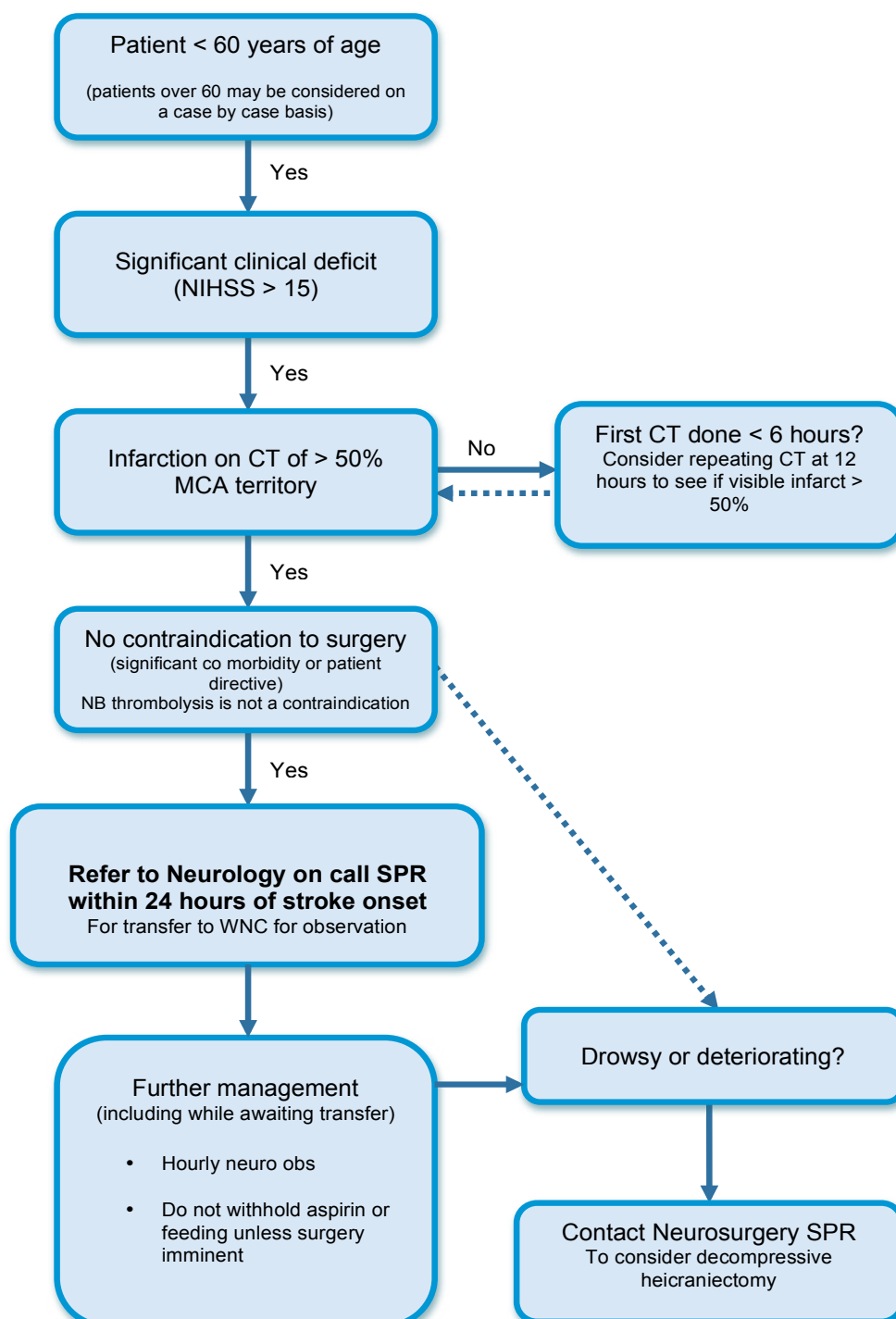


# Decompressive craniectomy for neurological conditions

# 21

## Who gets referred to Wessex Neurological Centre

### Guidance for referral of malignant MCA stroke patients to Wessex Neurological Centre



## Cerebellar infarction

Patients with cerebellar infarcts should be discussed with the neurosurgery SPR on call if :

- There is CT evidence of hydrocephalus
- **Or** there is other CT evidence of mass effect – for example brain stem displacement or effacement of the fourth ventricle or quadrigeminal cistern
- **Or** there is a reduced conscious level

Patients with cerebellar infarcts may be discussed with the neurology SPR on call to consider transfer for observation if none of the above are present if the infarct is large (for example more than 50% of one cerebellar hemisphere or a complete PICA territory infarct), especially in young patients.

## Cerebellar haemorrhage

Patients with cerebellar haemorrhage should be discussed with the neurosurgical SPR on call.

## Primary intracerebral haemorrhage

### URGENT REFERRALS

Patients with supratentorial primary intracerebral haemorrhage should be discussed urgently with the neurosurgical SPR on call if the haemorrhage is:

- Associated with significant mass effect
- Associated with hydrocephalus
- Associated with reduced conscious level
- $\leq 1\text{cm}$  from the cortical surface and  $\geq 2.5\text{cm}$  diameter

### LESS URGENT REFERRALS

Patients with smaller haemorrhages, not fulfilling the above criteria, in whom the cause is unknown, may be discussed with the neurosurgical team who will then refer the case on to the neurovascular neurosurgery team.

Note: In general, unexplained primary intracerebral haemorrhages require a minimum investigation of a follow up CT (with and without contrast) at 6 weeks and MRI at

3 months. After this, if no cause has been found, referral to the neurovascular neurosurgery team should be considered as the patient may require catheter angiography.

## Management of stroke patients prior to arrival at WNC

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- All such patients are at high risk of secondary brain injury and require formal assessment prior to transfer
- Identification of compromised airway, inadequate ventilation or circulatory insufficiency must occur
- These patients are at high risk of deterioration during transfer

### Airway management

- Priority must always be to maintain and protect a clear airway
- Give oxygen by mask (10l/min)
- Consider placing a nasopharyngeal airway if necessary
- Patients may require intubation and ventilation to ensure safety during transfer
- Indications for intubation and ventilation
  - Immediate:
    - Coma (GCS <8)
    - Loss of protective laryngeal reflexes
    - Tolerating oropharyngeal airway
    - Inadequate oxygenation:  $\text{PaO}_2 < 13 \text{ kPa}$  on oxygen via mask
    - Ventilatory insufficiency:  $\text{PaCO}_2 > 6.4 \text{ kPa}$
    - Respiratory arrhythmia eg Cheyne-Stokes respiration
  - Before transfer
    - Deteriorating consciousness (GCS fallen by 2 points from admission, even if not in coma)

### Breathing

- If intubated and ventilated, aim for:
  - $\text{PaO}_2 > 13 \text{ kPa}$

- PaCO<sub>2</sub> 4.5kPa

## Circulation

- Mean arterial pressure should be maintained >80mmHg to preserve intracranial perfusion.
- Do not acutely reduce blood pressure even in the presence of intracranial haemorrhage
- Patients require an adequate blood pressure to maintain intracerebral perfusion – acute falls can precipitate ischaemic stroke.

## Disability

- Record every 15 minutes prior to and during transfer:
  - GCS. Record as breakdown of Eyes, Verbal and Motor
  - Pupillary size and response to light
- Patients who are sedated and paralysed must still have repeated assessment of pupil size and reaction This is unaffected by neuromuscular junction blockers eg rocuronium, atracurium.
- The neurosurgeon may request the administration of mannitol prior to transfer
  - A urinary catheter should be inserted in all patients that may require mannitol, due to the diuresis provoked by this drug
  - The recommended dose is usually:
    - 0.5g/kg for one fixed dilated pupil
    - 1g/kg for bilateral fixed dilated pupils
    - 20% mannitol contains 20g per 100ml
- Hypotonic solutions eg Hartmanns, Plasmalyte and Dextrose Saline MUST be avoided in these patients as these will worsen cerebral oedema.
- 0.9% saline +/- KCl is the infusion fluid of choice.

## Transfer

- Intra and inter-hospital transfer can expose stroke patients to substantial risks.
- Hypoxia and hypercarbia can develop insidiously.

- Patients with posterior fossa strokes are at high risk of respiratory arrest, as a result of pressure on brainstem or lower cranial nerves.
- A doctor and a trained nurse must escort all patients.
- Accompanying staff must understand what can go wrong during a transfer and have the skills and equipment to be able to deal with it.
- A doctor with anaesthetic skills must escort every intubated and ventilated patient but advanced airway skills may also be required to safely transfer some non-intubated patients.
- If there is doubt regarding the risk of deterioration en route, it is better to err on the side of caution and intubate and ventilate prior to departure.
- If intubation is required, this must be communicated to Wessex Neuro ICU prior to departure.

### **Monitoring**

- All patients require:
  - 2x IV access
  - ECG
  - NIBP
  - Oxygen saturation monitoring
- Ventilated patients require :
  - Capnography
  - Arterial line

### **Prior to transfer**

- 1** Ensure patient stable
- 2** Ensure accepting ward knows time of arrival and state of patient
- 3** Adequate escort team
- 4** Adequate monitoring
- 5** Transfer documentation – the critical care network has transfer documents available on all ICUs
- 6** Ensure copies of all notes accompany patient and that all radiological images have been copied onto ExoPACS for review at WNC (not just the CT scans)

## Management at Wessex Neurological Centre

### Assessment

- Patients < 60 years old with large volume middle cerebral artery infarction will be admitted to the acute stroke unit or Neuro ICU, as soon as identified as being appropriate candidates for consideration of decompressive surgery in their referring hospitals
- Full history and examination
- Full investigations to be carried out as pre-operative assessment for surgery:
  - FBC
  - U&Es
  - Clotting
  - Blood group & save
  - ECG
  - CXR
- Ensure baseline CT head scan available on EXO-PACS
- Any patient whose NIHSS 1A (Alertness score)  $\geq 1$  or Wessex modified RASS  $\leq -1$  (see below) will be immediately referred to the neurosurgical registrar on-call and discussion held over repeating the CT brain scan. If there is a CT Scan showing large volume MCA infarction already, there is no need to repeat the CT scan. The patient should be transferred for emergency decompressive craniectomy whilst receiving 20% mannitol or hypertonic saline.

**National Institutes of Health Stroke Scale (NIHSS)** is composed of 11 items including responsiveness, eye movement, visual fields, motor & sensory function. Each element is scored, with 0 indicating normal function. The NIHSS is the sum of the 11 components. 1-4 is a minor stroke & 21-42 is a severe stroke.



#### 1A Level of Consciousness: Responsiveness

0	Alert; responsive
1	Not alert; Verbally arousable or aroused by minor stimulation to obey, answer, or respond
2	Not alert; Only responsive to repeated or strong and painful stimuli
3	Totally unresponsive; Responds only with reflexes or is areflexic

- Neurology registrar will review patient:
  - GCS, pupils, lateralising neurology
  - CT brain scans – assessing extent of mass effect
  - Considering the impact of co-morbidities on surgical procedure & potential outcome
  - Ensure during discussion with patient &/or relatives that they understand aims of hemicraniectomy and would be prepared to accept potential survival with severe disability (including risk of surviving wheel-chair bound, and unable to speak if dominant hemisphere affected)
- Hemi-craniectomy principally prevents death from brain herniation; most survivors will be left with some degree of disability. As a rule of thumb, for every 10 operations, 5 deaths are prevented. Of these 5 survivors:
  - 3 will have moderate-severe disability (unable to walk and need help with all bodily needs)
  - 1 will have moderate disability (able to walk but needing help with some bodily needs)
  - 1 will have mild disability (able to walk and independent in basic needs, but unable to do all their previous activities)
- Neurology registrar will discuss their findings with the on-call consultant
- Neurology consultant will decide whether decompressive craniectomy is appropriate for that patient and indicated at that time or only if patient deteriorates further

### **Observation under neurology or stroke team**

- Start hourly neuro observations with special attention to Wessex modified RASS score (See below)
- The Wessex modified RASS 6 hours after stroke, should be taken as the baseline level
- Ensure vascular neurosurgical team are aware of patient in normal working hours, or on-call neurosurgical team have been warned for all acute admissions
- Continue feeding unless surgery imminent
- Discuss with the neurosurgical team about withholding aspirin
- If IV fluids required prescribe Normal Saline infusion +/- KCl

**Guidance for management of malignant MCA stroke patients in WNC****Referral Criteria**

- Patient < 60 years of age  
(patients over 60 may be considered on a case by case basis)
- Significant clinical deficit (NIHSS > 15)
- Infarction on CT of > 50% MCA territory
- No clear contraindication to surgery (e.g. significant co morbidity or patient directive. N.B. thrombolysis is not a contraindication)

**Neurology on call SPR accepts transfer to WNC for observation under the care of Neurology**

- Aim to transfer within 24 hours of onset
- Stanley Graveson Ward (or NHDU / NITU / neurosurgery wards according to patient status or bed availability)

**Further management**

- Hourly neuro obs
- **All team members to observe closely for any deterioration** - taking GCS and neurological status at 6 hours from onset as baseline
- Do not withhold aspirin or feeding unless surgery imminent
- All usual acute stroke care, including any urgently required investigations for causes of stroke
- Inform neurosurgery SPR (see below)

**Patient stable**

- If in working hours: inform vascular neurosurgery team
- If out of working hours: inform neurosurgery on call SPR who will inform the vascular neurosurgery team at 8am meeting the following day
- Repatriate patient after 5 days from onset

**Patient drowsy or deteriorating**

- Assess whether there could be an immediately obvious systemic cause (e.g. sepsis, hyponatraemia, hypoxia)
- Contact on call neurosurgery team (or vascular neurosurgical team, if available) to consider immediate decompressive hemicraniectomy
- Do not delay surgery for CT or other investigations
- Ideally surgery < 48 hours of onset but consider surgery > 48 hours on a case by case basis



- Avoid sedative medications. Use paracetamol analgesia. If severe headache consider rising ICP - reassess and repeat CT head scan; call neurosurgeon if significant mass effect.
- Avoid lowering BP unless strong indication. If BP > 180/110 routinely, or steadily rising, suspect raised ICP - reassess and repeat CT head scan; call neurosurgeon if significant mass effect.
- Call neurological team to review patient if Wessex modified RASS drops a single level from the baseline.
- Contact on-call neurosurgical team (or vascular neurosurgical team, if available) to consider immediate decompressive hemicraniectomy in any drowsy or deteriorating patient
- Referral to neuroanaesthetist if decision for surgery
- The patient should be observed for at least 4 days in the Wessex Neurological Centre to cover the full period that they are at risk of cerebral oedema following their stroke.
- A final CT scan should be performed and reviewed prior to consideration of their repatriation

### Wessex modified RASS for stroke & brain injured patients

Score	Term	Description
0	Alert and Calm	
-1	Sustained interaction	Able to sustain meaningful interaction, but if left alone may become less alert
-2	Stimulated interaction	Requires repeated verbal stimulation to maintain interaction
-3	No meaningful interaction	Verbal or motor response to voice, but no meaningful interaction
-4	No response to voice	No response to voice, but verbal or motor response to physical stimulation
-5	Unroutable	No response to voice or physical stimulation

## Surgery

- Decompressive surgery is based on a hemicraniectomy in combination with a duraplasty.
- All patients that have received aspirin / clopidogrel in the previous week **must** have 2 pools of platelets infused prior to surgery, irrespective of their platelet count.
- After incision of the skin in the shape of a question mark, a bone flap that has a diameter of at least 12 cm is removed, including parts of the frontal, parietal, temporal, and occipital squama.
- The removed bone flap must be of a sufficient size to prevent additional ischaemic lesions.
- After opening of the dura, a dural patch may be inserted. Autologous tissue or artificial dura substitute may be used.
- Ischaemic brain tissue is not resected.
- Insertion of an intracranial pressure probe or external ventricular drain may be done at the consultant surgeons discretion (NB patient will be on aspirin)
- Skull removed to be implanted subcutaneously into abdominal wall.

## Post-op Care

- Transfer to Neuro ICU for routine post-operative care
- These patients **MUST NOT** be lain on their decompressed side, but should be nursed from supine to the other side only. Pressure followed by release of pressure on the herniated brain will increase the risk of post operative haemorrhage.
- Consideration may be given to continuing sedation and ventilation in the immediate post operative period.
- To minimise herniation, consideration may also be given to the use of hypertonic saline, with the aim of increasing the serum sodium by a maximum of 1mmol/l per 2 hours.
- No routine ICP monitor to be inserted on Neuro ICU – risk of haemorrhage into normal brain
- Assess the extent and tenseness of herniation site. If this becomes tense the Neuro ICU consultant and neurosurgical registrar must be immediately informed. The patient may require an emergent CT Scan.
- Post-op CT brain scan should be performed at 24 hours after surgery in daylight hours.

- The timing or the post operative dose of aspirin 75mg OD should be agreed with the neurosurgical team.



When aspirin is given following decompressive craniectomy, it should be at the dose of 75mg daily, and only once the neurosurgical consultant agrees. The use of 300mg daily should be avoided following neurosurgery. If it is considered necessary to give this dose, the consultant neurosurgeon must be aware of, and in agreement with, the higher dose.

- Arrange transfer back to host hospital using the WNC repatriation policy

### Reversal cranioplasty post hemicraniectomy

- Hemicraniectomy should be reversed between 6 weeks and 6 months. It does not always need to be reversed in patients who have had a poor neurological outcome and who do not wish to have the surgery performed.
- Autologous cranioplasty will be the first line treatment in all patients using the skull flap implanted in the abdominal wall.
- For each patient, the discharge document will name the surgeon who performed the surgery and state:
  - a Wait for the hemicraniectomy site to become sunken and a minimum of 6 weeks after the surgery.
  - b Then assess the patient's recovery and perform a CT brain scan.
  - c Put the scan on EXO-PACS and inform the surgeon who performed the surgery.
  - d They will then make arrangements for an elective admission for reversal cranioplasty or outpatient review if necessary.

### Decompressive craniectomy for other neurological conditions

Neurosurgical decompressive craniectomy has been successfully used in other neurological conditions eg Acute Disseminated Encephalomyelitis (ADEM) and subdural empyema.

## Introduction

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Acute neuromuscular weakness is most commonly caused by Guillain-Barré syndrome or myasthenia gravis. Less commonly it may be due to motor neurone disease, myotonic dystrophy, polymyositis, muscular dystrophy or acute brainstem / upper cervical spinal cord disease. Always consider botulism and tetanus, particularly if there is a history of intravenous drug abuse. Poisoning with organophosphates, lead, arsenic and certain shellfish can also cause neuromuscular weakness.

## Guillain-Barré syndrome (GBS)

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- GBS is an acute inflammatory polyneuropathy characterised by progressive weakness and areflexia. It usually presents with symmetrical weakness and sensory impairment. Weakness commonly ascends from the legs with progression over < 4 weeks.
- Patients may develop bulbar or respiratory weakness requiring airway management and ventilator support.
- Autonomic dysfunction frequently occurs. This ranges from sinus tachycardia to malignant arrhythmias or rapid extremes of blood pressure.
- The Miller-Fisher variant causes areflexia, ataxia and ophthalmoplegia.
- LP commonly shows raised protein with normal WBC in CSF. Diagnosis is confirmed by EMG demonstrating demyelination. (5% also show axonal degeneration).

## Management

Patients are usually admitted to Neuro ICU for ventilator support or management of bulbar dysfunction.

- 1** Assess airway, bulbar and ventilatory function:
  - a** Beware choking, coughing or cyanosis on swallowing. Check the swallow, gag and look for pooling of saliva.
  - b** Check cough.
  - c** Check for breathlessness or orthopnoea, speaking in short sentences or words only.
  - d** Measurement of vital capacity may be helpful. Generally a **VC less than 1.5L** suggests inadequate ventilatory function which may require support.
  - e** Oxygen saturations may be maintained and falsely reassuring in ventilatory failure. Arterial blood gas measurement should be performed, to assess PaCO<sub>2</sub>. (Raised/rising PaCO<sub>2</sub> are markers for ventilatory insufficiency.)
  - f** Patients with GBS may require ventilation for a period of weeks or months. Ideally, patients should be warned of this and the likelihood of a tracheostomy, before ventilation is started.



If acute concern regarding airway maintenance or ventilatory function, patient should be urgently assessed by anaesthetist/intensivist to determine requirement for intervention.



Avoid suxamethonium because of risk of severe hyperkalaemia (Hang 'No suxamethonium' warning sign above bed.)

- 2** Take a careful history, looking for any underlying cause. Perform full examination, including detailed neurological examination.
- 3** Assess for signs of autonomic dysfunction, eg tachycardia, urinary retention, postural hypotension, sweating or ileus. Management of cardiovascular instability may require:
  - a** Anticholinergics for bradycardia (pacing in severe cases)
  - b** Beta-blockers for tachycardia, or hypertension
  - c** Vasopressors (eg phenylephrine) for hypotension
- 4** Monitoring: ECG/SpO<sub>2</sub>/NIBP/Vital capacity

- 5 Swallow assessment and nasogastric feeding if bulbar function is poor.
- 6 VTE prophylaxis. Full length AES, IPCs and [enoxaparin 40mg SC od](#)
- 7 Eye care (may have inadequate eyelid closure especially at night).
- 8 Communication: patients may be unable to speak or to use their limbs to operate a language board. Careful nursing and physiotherapy assessment should determine the most appropriate method for summoning help and communicating wishes.
- 9 Patients may develop severe neuropathic pain. Start [amitriptyline 10mg NG/PO nocte](#) and consider gabapentin (300mg PO/NG od on first day, then escalated as required).

## Specific therapy—IVIg or plasma exchange

### IVIg

- IVIg is easier to administer but expensive.
- Patients with autonomic dysfunction or cardiovascular disease may tolerate IVIg better.
- Anaphylaxis, aseptic meningitis and renal dysfunction have been reported.

### PLASMA EXCHANGE

- Plasma exchange is most effective if given within two weeks of diagnosis.
- Generally twice the plasma volume is exchanged against 4.5% albumin solution. Exchange is repeated up to 5 times.
- The biggest risks of plasma exchange are:
  - Severe uncontrollable [sepsis](#) secondary to loss of circulating antibodies. The source is likely to be from the respiratory tract, but may also be from urinary tract or lines used for exchange.
  - [Haemorrhage](#) secondary to washing out of coagulation factors and destruction of platelets. Caution with urinary catheters and NG tubes. Avoid intramuscular injections.

## Myasthenia gravis (MG)

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- MG is rare (incidence 1/200,000). It has two peaks in incidence, affecting females more than males in the third decade of life and males more than females in the fifth decade of life. It results from an autoantibody against acetylcholine receptors in the post-synaptic membrane resulting in increasing muscle fatigue.
- Another autoimmune disease may be present in <5% of cases.
- Diagnosis is generally made through history and examination
  - Fluctuating weakness of the muscles of the head, face and neck. Classically it presents with diplopia and ptosis but may cause dysarthria, dysphagia or respiratory insufficiency.
  - Fatigue is increased by exercise and improved by rest.
- Confirmation of diagnosis :
  - Serum acetylcholine receptor antibodies (80–90% cases are positive).
  - Among patients who are seronegative for acetylcholine receptor antibodies, a subgroup with antibodies to muscle-specific tyrosine kinase has been found.
  - Decreasing response on repetitive nerve stimulation. Fade on tetanic stimulation and post-tetanic facilitation.
  - Positive edrophonium test. This should not be carried out by the inexperienced. Cardiorespiratory collapse can occur. Patients who respond will generally show a dramatic improvement in strength within 1 minute (patients with a cholinergic crisis will get a worsening of symptoms—this lasts roughly 10 minutes).

## Management

Patients are usually admitted to Neuro ICU for plasma exchange, or for ventilatory support, which may require intubation and ventilation if bulbar dysfunction is present.

- 1** Assess airway, bulbar and ventilatory function.
  - a** Beware choking, coughing or cyanosis on swallowing. Check the swallow, gag and look for pooling of saliva.
  - b** Check cough
  - c** Check for breathlessness or orthopnoea, speaking in short sentences or words only

- d** Measurement of vital capacity may be helpful. Generally a **VC less than 1.5L** suggests inadequate ventilatory function which may require support.
- e** Oxygen saturations may be maintained and falsely reassuring in ventilatory failure. Arterial blood gas measurement should be performed, to assess PaCO<sub>2</sub>. (Raised/rising PaCO<sub>2</sub> are markers for ventilatory insufficiency.)



If acute concern regarding airway maintenance or ventilatory function, patient should be urgently assessed by anaesthetist/intensivist to determine requirement for intervention.

## 2 Ventilation

- a** Relatively resistant to small doses of suxamethonium.
- b** Extremely sensitive to non-depolarising neuromuscular blocking agents—these should be used at 1/10 their usual dose.
- c** May require ventilation for a period of weeks or months. Ideally, patients should be warned of this and the likelihood of a tracheostomy, before ventilation is started.

## 3 Take a careful history, looking for any underlying cause for deterioration in muscle function. Full examination, including detailed neurological examination.

## 4 Monitoring: ECG/SpO<sub>2</sub>/NIBP/Vital capacity

## 5 Swallow assessment and nasogastric feeding if bulbar function is poor.

## 6 VTE prophylaxis. Full length AES, IPCs and **enoxaparin 40mg SC od**

## 7 Communication. Patients may be unable to speak or to use their limbs to operate a language board. Careful nursing and physiotherapy assessment should determine most appropriate method for summoning help and communicating wishes.

## 8 Certain drugs can exacerbate symptoms

- a** Antibiotics: neomycin, streptomycin, gentamicin, tetracyclines, erythromycin, polymixin, ampicillin, kanamycin
- b** Cardiovascular:  $\beta$ -blockers, quinidine, procainamide, verapamil
- c** CNS drugs: chlorpromazine, lithium, morphine
- d** Drugs for RA: penicillamine, chloroquine, quinine
- e** Others: lignocaine, steroids, procaine, gadolinium





It is usually considered safe to give a single dose of gentamicin 120mg IV to patients with myasthenia gravis, while on the Neuro ICU. Any exacerbation of symptoms will generally be mild and short lived.

### Specific therapy

- Anticholinesterases (pyridostigmine)
- Immunosuppression (steroids, azathioprine, cyclosporin)
- Thymectomy for patients with a thymoma
- IVIg
- Plasma exchange. Generally 1.5× plasma volume is exchanged against 4.5% albumin for 5 exchanges. The biggest risks of plasma exchange are:
  - Severe uncontrollable **sepsis** secondary to loss of circulating antibodies. The source is likely to be from the respiratory tract, but may also be from urinary tract or lines used for exchange.
  - **Haemorrhage** secondary to washing out of coagulation factors and destruction of platelets. Caution with urinary catheters and NG tubes. Avoid intramuscular injections.

### Myasthenic crisis

Sudden worsening and spreading weakness. May be provoked by drug omission or infection. Management should follow the principles above.

### Cholinergic crisis

- May result from relative overdose of acetylcholinesterase inhibitors eg pyridostigmine.
- Excess stimulation by acetyl choline will result in nicotinic effects (muscle weakness, fasciculation) and muscarinic effects (sweating, lacrimation, miosis, abdominal colic etc).
- Management requires stopping acetylcholinesterase inhibitor, giving anticholinergic (eg atropine) and providing respiratory support if necessary.

## Motor neurone disease (MND)

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MND is a group of disorders characterised by progressive deterioration in motor function (sensory and higher functions remain normal). Males are affected more frequently than females. Treatment is supportive with death occurring within 3–5 years from diagnosis.

Diagnosis is generally made from history and clinical features:

- Progressive weakness and wasting of muscle groups with fasciculation. Combination of upper motor neurone signs (increased reflexes) and lower motor neurone signs (weakness with fasciculations).
- Cardiac and smooth muscle are affected as well as striated muscle
- Progressive bulbar palsy with dysarthria and dysphagia may cause recurrent aspiration / pneumonia
- Daytime sleepiness (due to sleep apnoea type syndrome)
- Confirmation of diagnosis is through EMG studies

## Management

Patients are usually admitted to Neuro ICU for ventilatory support, which may require intubation and ventilation if bulbar dysfunction is present.

- 1** Assess airway, bulbar and ventilatory function.
  - a** Beware choking, coughing or cyanosis on swallowing. Check the swallow, gag and look for pooling of saliva.
  - b** Check cough
  - c** Check for breathlessness or orthopnoea, speaking in short sentences or words only
  - d** Measurement of vital capacity may be helpful. Generally a **VC less than 1.5L** suggests inadequate ventilatory function which may require support.
  - e** Oxygen saturations may be maintained and falsely reassuring in ventilatory failure. Arterial blood gas measurement should be performed, to assess PaCO<sub>2</sub>. (Raised/rising PaCO<sub>2</sub> are markers for ventilatory insufficiency.)



If acute concern regarding airway maintenance or ventilatory function, patient should be urgently assessed by anaesthetist/intensivist to determine requirement for intervention.

- 2** Take a careful history, looking for progressive deterioration in muscle function. Full examination, including detailed neurological examination.
- 3** Monitoring: ECG/SpO<sub>2</sub>/NIBP/Vital capacity
- 4** Swallow assessment and nasogastric feeding if bulbar function is poor.
- 5** VTE prophylaxis. Full length AES, IPCs and **enoxaparin 40mg SC od**
- 6** Communication. Patients may be unable to speak or to use their limbs to operate a language board. Careful nursing and physiotherapy assessment should determine most appropriate method for summoning help and communicating wishes.

#### DECISIONS TO VENTILATE OR PERFORM RESUSCITATION

- An assessment must be made as to whether there is a reversible condition (eg infection) that has caused their respiratory function to deteriorate. The decision to ventilate a patient for an acute deterioration must be discussed with an ICU consultant and patient's consultant neurologist
- Patients with MND that is progressing with deteriorating ventilatory function may need supportive (non-invasive) ventilation especially overnight. This should be assessed and managed whilst the patient is in a stable phase of their illness. Long-term invasive ventilation is possible, but will require tracheostomy, PEG and community support. This will require involvement of the respiratory consultants that run this service.
- Respiratory deterioration is the most common mode of death for patients with MND and this may be a terminal episode. It is important to avoid instigating invasive ventilation inappropriately in patients that would not be suitable for longterm community ventilation.
- Most of these patient will have advanced directives, which should be readily available and considered when deciding management. In the absence of an advanced directive, the MND care co-ordinator / neurologist may be contacted for further guidance.

DNACPR decisions. All patients should be offered the chance to discuss their wishes regarding end-of-life decisions. These should be formally documented in the notes.

Therapeutic plasma exchange allows removal of plasma constituents associated with disease eg antibodies, antigens, immune complexes or drugs. To be effective, the rate of removal of these substances must exceed their rate of production.

It differs from renal replacement haemofiltration in that the pore size is set to allow removal of substances with a high molecular weight (eg antibodies) rather than low molecular weight molecules (eg urea, creatinine, electrolytes). Therefore, plasma exchange requires replacement of plasma proteins (eg albumin, clotting factors) rather than just crystalloid.

It differs from plasmapheresis in the volume of plasma that is removed.

- Plasmapheresis is rarely used, and involves the removal of up to 600ml of plasma, which may not require replacement with IV fluids.
- Plasma exchange will always require replacement of human albumin and may also require fresh frozen plasma to control the anticoagulant effect of removing clotting factors.

Plasma exchange requires a vascath attached to a haemofiltration pump that has a large pore filter to allow separation of cellular components of blood from plasma. Plasma is removed in exchange for 4.5% human albumin solution. The patient and filter are anticoagulated with heparin during the procedure.

Plasma exchange is most commonly used on Neuro ICU for the treatment of:

- Myasthenia gravis
- Guillain-Barré
- Neuromyelitis optica (NMO)
- Chronic inflammatory demyelinating polyneuropathy (CIDP)
- Paraneoplastic neurological deterioration
- Antibody mediated encephalitis eg anti-NMDA

- It may also be considered in multiple sclerosis that is rapidly progressive and unresponsive to immunotherapy.

Patients may be transferred from the ward on a regular basis for exchange, or may require admission to the unit.

## Management

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- 1** Careful assessment to exclude contra-indications:
  - a** Sepsis – plasma exchange will result in worsening of sepsis due to the removal of circulating antibodies.
  - b** Cardiovascular instability – care required in patients on vasopressor support.
  - c** Recent ACE inhibitor administration (within 24hrs)
- 2** Vascular access, with a vascath, should be obtained immediately prior to the first exchange. This will limit the duration of time that large bore venous access is present, limiting risk of systemic infection.
- 3** Prescription of appropriate volume of replacement fluid
  - a** Calculate patient plasma volume: Estimated plasma volume (l) =  $0.07 \times \text{weight (kg)} \times (1 - \text{haematocrit})$ 
    - This is usually about 3 litres
  - b** The volume of replacement fluid may be reduced by up to 500ml per exchange if the patient is fluid overloaded.
  - c** For Guillain-Barré:
    - 2 plasma volumes exchanged against 4.5% human albumin
    - Plasma exchange 4 times in one week
  - d** For myasthenia gravis:
    - 1.5 plasma volumes exchanged against 4.5% human albumin
    - Plasma exchange 5 times in 10 days
- 4** Patients require daily bloods taken on the morning prior to exchange.
  - a** U&Es : monitor potassium and calcium, and replace as necessary. Initially add  $K^+$  at 4 mmol/l and  $Ca^{2+}$  at 2.0 mmol/l, adjust as required for later sessions.

- b** FBC: watch platelets and white cell count (consider withholding exchange or platelet transfusion)
- c** Clotting: abnormal coagulation may prompt infusion of FFP as part of exchange

## Complications

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- **Hypotension:**
  - Usually vaso-vagal or hypovolaemia.
  - Treatment with anticholinergic or IV fluids as required.
- **Respiratory distress:**
  - Fluid overload or bronchospasm.
  - Occasionally anaphylactoid with FFP.
- **Hypocalcaemia:**
  - Tetany relatively common
  - Give prophylactic calcium chloride 10 ml 10%
- **Coagulopathy:**
  - Clotting factors are removed
  - Daily treatments may need FFP at last exchange
- **Sepsis**
  - Immunosuppression as a result of antibody removal
  - Rapid antimicrobial treatment
- **Hypokalaemia:**
  - May be caused by dilution.
  - Replace as required—best 4 mmol KCl per litre of fluid
- **Drug removal:**
  - Protein-bound drugs are removed.
  - Best given after rather than before plasma exchange

## Pre-operative care

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Ensure that the patient has:

- 2 UHS patient labels, one on the arm and one on the leg
- A consent form
- A completed theatre checklist with appropriate additional forms (eg CJD & VTE)
- Current group and save. The antibody status must be checked and, if antibodies are present, blood should be ordered if appropriate
- Their name on an operating list that day
- Had enteral feed stopped 6 hours before surgery and the gastric tube has been aspirated. It is not necessary to stop enteral feed if the patient is being fed via a jejunal tube, but the gastric tube should still be aspirated
- Adequate sedation and vasopressor infusions via syringe drivers, with spare syringes available

### Drugs on day of surgery

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When a patient is admitted for surgery, all their normal medication should be prescribed. However, some medication should be omitted on the day of surgery. If you are unsure as to the class of a medication, look in the current edition of the BNF. Drugs may be taken with a small amount of water at any time during the 'Nil By Mouth' period.

#### Give:

- All "cardiac" or blood pressure drugs **except** ACE inhibitors, angiotensin-II receptor antagonists and diuretics
- All antibiotics (including oral)

- All epilepsy or Parkinson's drugs
- All asthma drugs or inhalers
- All tablets which reduce gastric acid (e.g. omeprazole, lansoprazole, ranitidine)
- All thyroid drugs
- All hypnotics, anxiolytics, barbiturates, antipsychotic and antidepressant medications taken regularly at home
- All drugs used in substance dependence e.g. nicotine replacement patches
- All steroids taken regularly, including inhalers
- All immunosuppressants and cancer drugs (eg. azathioprine, tamoxifen)
- All analgesics can be given before surgery **except** non-steroidal anti-inflammatory drugs (NSAIDS)

### Do NOT give:

- ACE inhibitors and angiotensin-II receptor antagonists. These drugs may drop the blood pressure during an anaesthetic. Some anaesthetists may request that these drugs are given before surgery but this will be requested on an individual basis.
- All diuretics. The anaesthetist may request that these are given, this will be on an individual basis.
- Diabetic treatment. The patient must be written up for alternative diabetic treatment by the surgical team.
- Aspirin, clopidogrel, dipyridamole or warfarin



Any patient taking aspirin, clopidogrel, dipyridamole or warfarin **must** be brought to the attention of both the consultant neurosurgeon and consultant anaesthetist. Further discussion may also be required with a cardiologist, as stopping these drugs in patients with coronary stents risks stent thrombosis.

- Drugs which are not essential in the short term eg vitamins, iron, laxatives, osteoporosis treatment, liquid antacid medicines (eg gaviscon), HRT, antihistamines, herbal remedies or homeopathic medicines.
- Lithium should not be given for 24 hours before surgery.



- Non-steroidal anti-inflammatory drugs (eg. diclofenac (Voltarol), ibuprofen, indometacin, naproxen), unless prescribed by an anaesthetist as a pre-med.



Any patient taking NSAIDs preoperatively **must** be brought to the attention of both the consultant neurosurgeon and consultant anaesthetist. For intracranial or intradural / multi-level spinal procedures the NSAIDs may provide an unacceptable risk of perioperative bleeding.

Patients on Neuro ICU may have abnormal coagulation or platelet function as a result of:

- **Medication:** Prophylactic anticoagulation or antiplatelet agents e.g. warfarin, aspirin, clopidogrel, rivaroxaban.
- **Disease:** As a result of an underlying disease present on admission, eg myeloproliferative disorders and alcoholism, or developed during admission as a result of traumatic brain injury or sepsis.

## Patients on long-term anticoagulation

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The mortality of intracranial haemorrhage associated with oral anticoagulation is about 60%. Thus, in most neurosurgical patients, the risk of bleeding far outweighs the risk of thromboembolism.

## Arterial thromboembolism

Common indications for anticoagulation to prevent arterial thromboembolism are:

- Atrial fibrillation (AF)
  - Annual risk of thromboembolism in untreated AF is about 4.5%.
  - Risk is increased if there is associated
    - Valvular disease (particularly mitral)
    - Left ventricular dysfunction
    - Left atrial enlargement
    - Ischaemic heart disease
    - Advanced age ( $\geq 75$  years).
- Presence of mechanical heart valves.

- With no anticoagulation, the average annual risk for thromboembolism in patients with mechanical heart valves is 8%. Other studies quote a risk of 0.016% per day of valve thrombosis.
  - Higher risk is conferred by mitral position and different types of mechanical valve, with caged ball (Starr-Edwards) being highest, followed by tilting disc (Medtronic and Carbomedics).
  - Risk is increased further by the presence of AF with a history of previous thromboembolism.
  - The greatest risk appears to be within 90 days of placement of mechanical valve.
- Patients with carotid stenoses or other peripheral vascular disease with or without arterial grafts.

### Venous thromboembolism

The most common indication for anticoagulation to prevent venous thromboembolism is a history of deep venous thrombosis or pulmonary embolism, with or without a familial or acquired prothrombotic state.

Risk of recurrence is 40% if anticoagulation is stopped within one month of the acute event, which decreases to 10–15% within 1–3 months and stabilises thereafter to 5% per annum.

This risk increases further with major surgery, morbid obesity, or malignant disease.

### Management of patients on anti-coagulation

The following plans are to act as a framework only. These patients should ideally be pre-assessed where possible and latest guidance followed. Complex cases may require discussion with haematology team.

#### ELECTIVE NEUROSURGERY

Elective neurosurgery should be delayed for:

- At least 90 days after placement of a mechanical heart valve.
- A minimum of 3 months in patients who have had a venous thromboembolic event.

**PATIENTS WITH UNCOMPLICATED ATRIAL FIBRILLATION (AF)**

These patients should be pre-assessed, and a plan made for their care.

- 1** No warfarin for the 5 days pre-operatively.
- 2** Check INR on the day of surgery ensuring an  $\text{INR} \leq 1.4$ .
- 3** Commence 40 mg enoxaparin 24 hours post operatively unless advised otherwise by neurosurgeon. All patients should have full length AES and IPCs at all times.
- 4** On the ward warfarin is usually restarted at the previous maintenance dose 48–72 hours post surgery at neurosurgical discretion

**ELECTIVE SURGERY ON PATIENTS WHO ARE TAKING WARFARIN FOR PREVIOUS PE OR DVT OR WITH AF COMPLICATED BY FURTHER RISK FACTORS**

These patients usually require bridging with LMWH. This involves starting LMWH once the INR is sub-therapeutic after stopping warfarin. Other conditions requiring bridging with LMWH include:

- VTE within 12 months of planned procedure date
- Recurrent VTE
- AF with rheumatic heart disease
- AF with stroke or TIA within 3 months of planned procedure date
- CHADS<sub>2</sub> score of 3 or more

These patients should be pre-assessed, and a plan made for their care.

- 1** No warfarin for 5 days pre-operatively.
- 2** Usually these patients will require bridging with LMWH once INR is subtherapeutic
- 3** In very high risk patients, consider use of retrievable IVC filter
- 4** Commence enoxaparin 40mg SC od 24 hours post operatively unless advised otherwise by neurosurgeon. All patients should have full length AES and IPCs at all times.
- 5** Enoxaparin bridging should continue post-operatively until fully anticoagulated with warfarin.
- 6** On the ward warfarin is usually restarted at the previous maintenance dose 48–72 hours post surgery at neurosurgical discretion

**ELECTIVE SURGERY ON PATIENTS WITH PROSTHETIC HEART VALVES**

These patients should be pre-assessed, and a plan made for their care.

- 1** Stop Warfarin 5 days pre-operatively.
- 2** Admit 48 hours pre-operatively and start full dose intravenous unfractionated heparin according to guideline.
- 3** All patients should have full length AES and IPCs at all times.
- 4** Stop heparin infusion 6 hours before surgery and check APTT and INR 2 hours before surgery.
- 5** Restart heparin 24–48 hours after surgery at neurosurgical discretion. Heparin is continued until warfarin therapeutic.
- 6** On the ward warfarin is usually restarted at the usual maintenance dose 48–72 hours post surgery at neurosurgical discretion.

**EMERGENCY SURGERY ON PATIENTS TAKING WARFARIN**

- 1** Give Vitamin K 10mg IV stat
- 2** Give Prothrombin Complex Concentrate (PCC), containing Factors II, VII, IX and X, which should normalise INR (i.e  $INR \leq 1.2$ ) in 10 minutes.
  - eg octaplex (dose based on weight & INR, discuss with haematology)
- 3** If PCC is unavailable, FFP may be considered in a dose of 15ml/kg.



Octaplas should be used rather than FFP for any patient born after 1st January 1996

- 4** All patients should have full length AES and IPCs at all times
- 5** An IVC filter may be considered in patients with previous venous-thromboembolism

**EMERGENCY SURGERY ON PATIENTS ON THERAPEUTIC DOSE LMWH**

- 1** These patients should be discussed with the haematologist
- 2** Protamine is commonly advised, although it only partially reverses LMWH
  - For Clexane (enoxaparin) the manufacturers recommend a protamine dose of :

- 1 mg protamine per 1 mg Clexane if last dose given less than 8 hours before
- 0.5 mg protamine per 1 mg Clexane for a dose given 8–12 hours previously
- Reversal is usually not required for doses given more than 12 hours previously.

#### LMWH AND EPIDURALS / SPINAL DRAINS

- Do not perform lumbar puncture or insert/remove spinal drain, EVD, bolt or subdural catheter **less than 12 hours** after prophylactic dose enoxaparin, except in emergency.
- Prophylactic dose enoxaparin should not be given **less than 6 hours** after performing lumbar puncture or insertion/removal of spinal drain, EVD, bolt or subdural catheter

#### RECOMMENCING LONG TERM ANTICOAGULATION FOLLOWING INTRACRANIAL OR INTRASPINAL HEMORRHAGE

- There is no consensus between neurosurgeons and cardiologists. Therefore there should be discussion between the consultants on a case by case basis.
- Most neurosurgeons would recommend waiting at least 6 weeks after CNS haemorrhage, checking for resolution of haemorrhage with appropriate imaging.
- Balance of risks and benefits would suggest that patients with mechanical heart valves (especially mitral with AF) should be anticoagulated sooner than patients with uncomplicated AF.
- Patients on anticoagulation for venous-thromboembolic disease may be considered for IVC filter

## Patients on antiplatelet agents

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### Intracranial and cervical spine surgery

#### ELECTIVE SURGERY

- Patient should have been pre-assessed and management planned

- Patients are advised to stop aspirin or clopidogrel 10 days prior to surgery, and avoid non-steroidal anti-inflammatory drugs in the week preceding surgery.



Any patient found to be on aspirin or clopidogrel at the time of surgery MUST have this brought to the attention of BOTH the operating neurosurgeon and anaesthetist, with a view to postponing surgery.

### URGENT SURGERY

Where neurosurgery can be delayed by 7-10 days, patients may be observed whilst awaiting offset of antiplatelet agents.



An elderly patient, previously on aspirin, with an acute subdural haematoma that is not causing acute neurological deficit. This patient may be observed whilst awaiting liquefaction of the haematoma, prior to burr hole drainage.

Any patient that deteriorates neurologically during this period, should be considered for emergency surgery as below.

### EMERGENCY SURGERY

- 1 Stop all antiplatelet agents
- 2 Crossmatch 2 pools of platelets
- 3 Discuss with haematologist the requirement for adequate number of circulating, functioning platelets for the neurosurgical procedure.



To reduce the likelihood of post operative haemorrhage, patients require  $150 \times 10^9/\text{ml}$  functioning platelets.



Any patient that has evidence of inadequate platelet function, eg excessive bleeding from a venepuncture site or haematuria, MUST immediately have further platelet transfusion if post operative haemorrhage is to be avoided.

## Lumbar and thoracic spinal surgery

The operating neurosurgeon and anaesthetist **MUST** be informed about any patient found to be taking antiplatelet medication. For elective surgery, this should have been identified at pre-assessment and a management plan formulated.

- Many surgeons are prepared to operate on patients taking Aspirin 75mg od, or non-steroidal anti-inflammatory agents, for decompressive thoracolumbar surgery (where they do not anticipate breaching the dura).
- Clopidogrel carries a higher risk than aspirin, and all patients should be considered on a case by case basis.

## Coronary stent thrombosis

- Discontinuation of antiplatelet agents can provoke stent thrombosis, with a mortality rate of 20%.
- Dual antiplatelet therapy with aspirin and clopidogrel is often given for four weeks after placement of a bare metal stent, and for 12 months following a drug eluting stent (which have a lower restenosis rate but a higher incidence of late stent thrombosis).
- Elective surgery should be delayed for **six weeks** following placement of a bare metal stent and **six to twelve months** following placement of a drug eluting stent.
- A cardiologist should be involved if there is a need to discontinue antiplatelet therapy in a patient with a recently implanted coronary stent.

## Abnormal coagulation or platelet function from disease

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### Alcoholism

Frequently associated with abnormal platelet function and inadequate platelet numbers. May be further complicated by abnormal coagulation (particularly prolonged INR secondary to alcoholic liver disease.)



These patients frequently re-bleed following neurosurgery, often with catastrophic consequences.



- Aggressive correction of coagulation abnormalities perioperatively is essential.
- Platelets should be transfused to ensure:
  - A minimum of  $150 \times 10^9/\text{ml}$ , prior to surgical haemostasis
  - Platelet number & function is maintained for at least the first 72 hours post-operatively.
  - Any evidence of inadequate platelet function (eg excessive bleeding from venepuncture site or haematuria) is an immediate indication for platelet transfusion
- Patients with  $\text{INR} > 1.3$ 
  - Vitamin K 10mg IV stat should be given immediately, and continued daily if INR abnormal.
  - FFP 15ml/kg should be given prior to surgical haemostasis. (Consider PPC in patients that are unable to tolerate the volume of FFP.)
  - Further FFP may be required postoperatively targeting  $\text{INR} < 1.4$
  - Octaplas should be used rather than FFP for any patient born after 1st January 1996
- All alcoholic patients with abnormal platelet number, or likely to have abnormal platelet function, that require neurosurgery should be given tranexamic acid:
  - At time of surgery tranexamic acid 1g IV
  - tranexamic acid 1g IV tds post operatively for 4 days.

## Myeloproliferative disorders

Frequently associated with abnormal platelet function and inadequate platelet numbers.



These patients frequently re-bleed following neurosurgery, often with catastrophic consequences.

- Platelets should be transfused to ensure:
  - A minimum of  $150 \times 10^9/\text{ml}$ , prior to surgical haemostasis
  - Platelet number & function is maintained for at least the first 72 hours post-operatively.

- Any evidence of inadequate platelet function (eg excessive bleeding from venepuncture site or haematuria) is an immediate indication for platelet transfusion
- Early haematological advice should be sought regarding treatment of the underlying disorder, adequate platelet transfusion to prevent rebleed and underlying prognosis

## Traumatic brain injury

This is a multi-system disease process that frequently affects coagulation, particularly INR.

- Mild derangement (INR<1.4) is frequently seen and may require no treatment
- INR>1.3 should prompt consideration of:
  - Vitamin K 10mg IV daily
  - FFP if within 48 hours of neurosurgery or traumatic intracranial haematoma.
  - Octaplas should be used rather than FFP for any patient born after 1st January 1996
- In patients with major haemorrhage, tranexamic acid should be administered early in the resuscitation of the patient with blood products.

## Sepsis

Sepsis can produce abnormalities in both platelet function and coagulation.

- Platelets should be transfused to ensure:
  - A minimum of  $150 \times 10^9/\text{ml}$ , prior to surgical haemostasis
  - Platelet number & function is maintained for at least the first 72 hours post-operatively.
  - Any evidence of inadequate platelet function (eg excessive bleeding from venepuncture site or haematuria) is an immediate indication for platelet transfusion
- Patients with INR>1.3
  - Vitamin K 10mg IV should be given immediately, and continued daily if INR abnormal.
  - FFP 15ml/kg should be given prior to surgical haemostasis. (Consider PPC in patients that are unable to tolerate the volume of FFP.)

- Further FFP may be required postoperatively targeting INR<1.4
- Octaplas should be used rather than FFP for any patient born after 1st January 1996

## Emergency neurosurgery

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Patients with abnormal coagulation or platelet function that require emergency neurosurgery should have surgery commenced immediately after receiving products (eg 15ml/kg FFP and/or 2 pools platelets). Do **NOT** delay surgery to check for correction of clotting.

## Patients born after January 1996

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As a result of concerns over CJD:

- Octaplas should be used rather than FFP for any patient born after 1st January 1996
- Any patient born after 1st January 1996 who requires cryoprecipitate, must be given Methylene Blue treated cryoprecipitate

# Epidural blood patch for intracranial hypotension

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The patient must have been reviewed by a consultant neurologist and it must be documented in the notes that the neurologist is convinced that the patient has intracranial hypotension from a spinal CSF leak, preferably confirmed on imaging, that has not settled with conservative management.

A doctor from that team should speak to the anaesthetist running the CEPOD theatre and the theatre coordinator to plan the best time to perform the epidural blood patch and on which list.

## Patient preparation

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- 1** The patient can eat and drink and should not be starved before the procedure.
- 2** They should have an up to date (not more than a day old) full blood count, CRP and a coagulation profile and have a dipstick test of a urine sample.
- 3** They should have their vital signs recorded regularly up to the time of the procedure i.e. temperature, pulse, blood pressure and oxygen saturation on air.
- 4** Blood culture bottles and request should be provided by the admitting ward.
- 5** The anaesthetist performing the procedure must obtain written consent from the patient. Consent must include:
  - a** The expected benefits:
    - Relief of headache
    - Prevention of development/expansion of intracranial subdural collections
  - b** The following complications:
    - Failure to locate the epidural space
    - Risk of a dural puncture and the consequences

- Risk of nerve damage
- Risk of infection leading to epidural abscess and the consequences, including paralysis
- Potential to cause a radiculopathy with persistent neuropathic pain
- Risk of arachnoiditis
- Risk of epidural haematoma/ subdural haematoma
- Risk of paralysis

## Contraindications

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- Fever
- Raised white cell count and CRP
- Any possibility of ongoing infection in CNS & especially CSF
- Evidence of urinary tract infection
- Infection at site of epidural injection
- Coagulopathy
- Technical difficulty

## Procedure

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- 1** This procedure requires an anaesthetist, who must be a consultant or a senior registrar, and another doctor able to take blood aseptically.
- 2** Both doctors should scrub and wear gowns, gloves and masks.
- 3** One performs the epidural and the other does the venesection.
- 4** An aseptic technique is used to locate the epidural space in the lower lumbar spine and in the lateral position with loss of resistance to saline.
- 5** The antecubital fossa of the dependant arm is cleaned with 2% chlorhexidine and the area is draped. Blood is drawn and handed aseptically to the anaesthetist performing the epidural. A further 20 ml of blood is taken and injected aseptically into the blood culture bottles.
- 6** 20-30 ml of blood is injected into the epidural space slowly and injection should be stopped if any of the following occur:

- Neck pain
- Radicular pain in the leg
- Worsening headache
- Resistance to injection
- Complaint of back pain

There is no consensus as to the optimum amount of blood to be injected but 20-30ml is the generally accepted figure. Once all of the blood has been injected, the Tuohy needle should be cleared with a small amount of saline before removal with the introducer replaced, to minimise leakage of blood into the subcutaneous tissues.

## Post epidural blood patch instructions

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- 1** The patient should remain in the supine position for at least 2 hours after the procedure. After this time, they can gradually sit up but aim to keep the lumbar spine straight or lordotic.
- 2** All patients should be prescribed aperients to ensure easy bowel motion and advised to avoid opiates for their headache.
- 3** All straining and heavy lifting should be avoided for at least 2 weeks.
- 4** Patients should be told to seek medical care immediately if they experience severe back pain, fever or any new neurological symptoms.

## Withdrawal of therapy

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If the treating team are in agreement that a patient would not recover to a level of functioning that the patient would consider acceptable, it may be appropriate to withdraw active therapy. This decision may already have been taken by the patient in the form of an advanced directive.

- Continuing active treatment of a patient becomes futile, or against their best interests if the clinical and radiological features support the diagnosis of an unsurvivable brain injury. These features include:
  - Clinical
    - Persistent motor score of 1 or 2 (ie extends at best)
    - Persistent Eye score of 1
    - Bilaterally fixed pupils in the absence of local injury or blindness
    - Absence of brainstem reflexes
  - Imaging
    - Tonsillar herniation
    - Complete loss of grey white matter differentiation
    - Severe midline shift with sub-uncal herniation
    - Complete effacement of sulci, gyri & basal cisterns
    - Inadequate arterial blood flow on CT angiogram
- The decision to withdraw active treatment on a patient requires documented agreement by:
  - The neurointensive care consultant. This would normally be in discussion with colleagues, however this may be unnecessary depending on the clinical context
  - The responsible neurosciences consultant (neurosurgeon, neurology or stroke)

- The patient's next of kin
- The entire neurointensive care team involved with that patient should be in agreement prior to withdrawal of active treatment
- Where the next of kin disagrees with the decision to withdraw active treatment, even after detailed explanation of radiological imaging and investigations:
  - Confirm brainstem death, if thought to be present, as this may help with next of kin acceptance
  - Whilst legally it is not necessary to have next of kin agreement for withdrawal of active treatment, the hospital legal team should be involved if agreement cannot be reached

### Guidelines for withdrawal of therapy

- 1** Ensure full documentation of decision making process as above
- 2** Ensure the next of kin understands:
  - a** The reasons for the decision made
  - b** That withdrawal of active treatment means that the management will change to palliative care with preservation of comfort and dignity
  - c** That the patient may not die rapidly following withdrawal of active treatment, in which case they will start a compassionate care pathway
- 3** Where the patient is likely to die rapidly following withdrawal of care, it is appropriate to consider organ donation. (See separate chapter). In these cases it is vital that the relatives understand that the decision to withdraw care is entirely separate from the process of organ donation.
- 4** Where organ donation is considered possible, the timing and process of withdrawal of active treatment should be guided by advice from the organ donation team. These cases must be discussed with the coroner prior to withdrawal of care.
- 5** Ensure adequate symptom control to maintain comfort and dignity. Stop all non-palliative care medication and minimise monitoring. See Compassionate Care Pathway (below)
- 6** Move the patient into a side room, if possible, or a more appropriate bed space
- 7** Insert NG tube, if not already present, prior to extubation. This is to allow feeding / hydration to continue for symptom relief



- 8** Withdrawal of respiratory support
  - a** Patients who have been diagnosed as brainstem dead, and who are awaiting organ donation, will remain ventilated on Neuro ICU. Ventilation will be withdrawn in theatre.
  - b** Patients who are potential non-heart beating donors, should be extubated at a time guided by the organ donation process
  - c** All other patients should be extubated at a time that is appropriate for patient, relatives and Neuro ICU team
- 9** The support of religious and/or cultural organisations may be sought
- 10** Staff may find terminal care patients with whom they have been closely associated stressful and distressing. It is important to seek the support and guidance of colleagues and other professionals such as the Hospital Chaplain

## Compassionate care pathway

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- 1** Ensure that the patient's family are fully informed that the care of the patient is to become palliative, prioritising dignity and comfort. This discussion must be documented in the patient's notes
- 2** Move the patient into a side room, if possible, or a more appropriate bed space
- 3** Stop all non-palliative care medication. This includes antibiotics, gut protection and LMWH injections
- 4** **Analgesia** prescribe:
  - a** Morphine 1mg IV PRN every 15 mins
  - b** Morphine infusion 1-10mg/hr IV
  - c** Diamorphine 1-5mg/hr S/C (If no IV cannula)
- 5** **Shortness of breath:** opiates as above. Oral / nasal airways should be avoided, as these can cause bleeding & pressure areas
- 6** **Anxiety / agitation:** prescribe midazolam 1mg IV PRN every 15 mins
- 7** **Secretion control** prescribe:
  - a** Hyoscine 200-400mcg IV PRN
  - b** Hyoscine patch (if no IV cannula)
- 8** **Hydration & feeding:** continue NG feed for symptom relief

- a** Insert a NG tube prior to extubation of patient, if not already present
  - b** Consider re-inserting NG tube into patient if required, to continue hydration & feed
- 9** **Pressure areas:** The frequency of turns should be assessed on an individual patient basis, but must be a minimum of 3 hourly
- 10** Continue all usual nursing care of eyes, mouth, bladder, bowels etc
- 11** Stop all monitoring, except those required to observe for symptom control. Disable all alarms
- 12** In the event of loss of IV access, consider replacing IV cannula if it is required for symptom control. However, alternative methods should be used where possible. Eg diamorphine S/C, hyoscine patch
- 13** Following the death of the patient, inform:
  - a** The family
  - b** The patient's GP, both by telephone & typed summary
  - c** The primary consultant (neurosurgical, neurological or stroke)

## **Do not attempt CPR (DNACPR)**

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- Patients with severe brain injury may be successfully resuscitated from a cardio-respiratory perspective, but be left severely brain damaged with a quality of life they would have considered unacceptable
- On Neuro ICU, DNACPR decisions must be made with agreement between:
  - The Neuro ICU consultant
  - The neurosurgical / neurological / stroke consultant
  - The Next of Kin
- All active treatment should be continued despite any DNACPR order, up to the point of CPR
- All patients that survive to Neuro ICU discharge, who are not on a palliative care pathway, should have the DNACPR order reconsidered

## Do not escalate therapy

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It may not be in patients best interest to escalate therapy if :

- 1** They have an irreversible neurological injury, which would prevent them from recovering to a quality of life that they would have wished for
- 2** They have a progressive underlying terminal condition that is no longer responding to maximal therapy
- 3** The new therapy would be likely to worsen the patients underlying condition

Treatments limitations include:

- DNACPR
- Withholding the institution of renal support (haemofiltration or dialysis) for hyperkalaemia, acidosis or volume overload
- Not re-intubating and/or re-ventilating
- Not changing or restarting antibiotics
- Not escalating vasoactive therapy (specify agent and dose limits)

Death is the irreversible loss of the essential characteristics which are necessary to the existence of a living human person and thus the definition of death should be regarded as the irreversible loss of the capacity for consciousness, combined with the irreversible capacity to breathe.

The irreversible cessation of brain-stem function whether induced by intracranial events or the result of extra-cranial phenomena, such as hypoxia, will produce this clinical state and therefore irreversible cessation of the integrative function of the brain-stem equates with the death of the individual and allows the medical practitioner to diagnose death. (*Academy of Medical Royal Colleges (2008) "A Code of Practice for the Diagnosis And Confirmation Of Death". <http://www.aomrc.org.uk>*)

In the legal system in the UK there is no statutory definition of death but the courts have adopted the use of neurological testing as a criteria for diagnosis of death. This has been the case since 1976 (*Conference of Medical Royal Colleges and their Faculties in the UK. BMJ 1976;2:1187*)

## Preconditions

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- 1 The patient's condition is due to irreversible brain damage of known aetiology.
- 2 Exclusion of reversible causes of coma and apnoea.

## Exclusion of reversible causes of coma and apnoea

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The following should be excluded before commencement of testing:

- 1 Coma and apnoea secondary to depressant drugs: The patient must not have had any drugs that may have contributed to unconsciousness, apnoea or loss of brain stem reflexes.

- a** These include the following drugs:
- Hypnotics / Sedatives/ tranquilisers
  - Narcotics
  - Muscle relaxants
- b** Consideration must be taken if the patient has hepatic or renal failure as this may prolong metabolism or excretion of the drugs. Measuring drug levels for midazolam (recommendation <10mcg/ml) or thiopentone (<5mg/ml) can be considered although this is not widespread practice within the UK.
- c** Consider specific antidotes to benzodiazepines (eg flumazenil 200mcg IV every minute up to max of 1mg) and/or opiates (eg naloxone 400mcg IV every 2 minutes up to maximum of 10mg) as necessary.

## 2 Coma and apnoea secondary to temperature, circulatory, metabolic or endocrine disturbances

- a** The following criteria should be achieved before testing:

<b>Core temperature</b>	> 34°C
<b>Mean Arterial Pressure</b>	> 60mmHg
<b>pH</b>	7.35-7.45
<b>PaO<sub>2</sub></b>	>10kPa
<b>PaCO<sub>2</sub></b>	>6kPa
<b>Serum Na</b>	115-160mmol/l
<b>Serum K</b>	>2.0mmol/l
<b>Serum Mg</b>	0.5-3.0mmol/l
<b>Serum PO<sub>4</sub></b>	0.5-3.0mmol/l
<b>Serum glucose</b>	3-20mmol/l

- b** It is recognised that some disturbances may be secondary to brain-stem death and do not preclude determination of brainstem death. Furthermore it may be detrimental to rapidly correct such abnormalities
- c** If endocrine disturbance is suggested this should be excluded with the appropriate assays

## Clinical assessment of brain stem function

All the following tests must be fulfilled for the diagnosis of Brain Stem Death (BSD) to be made.

Clinical test	Cranial nerves tested		Technique
	Afferent	Efferent	
No pupillary response to light	II	III	Fixed pupils that do not respond to sharp changes in the intensity of light
No corneal reflex	V	VII	No blink response when cornea is touched
No vestibulo-ocular reflex	VIII	III, VI	No eye movement when 50mls of ice-water is irrigated to each ear over a 1 minute period. (Head tilt at 30°) <i>Normal response in an individual with a functioning brainstem is for the eyes to turn towards the side cold water is irrigated, with nystagmus away from that side.</i>
No motor response to central stimulation	V	VII	No somatic or cranial nerve response to pressure exerted on the supraorbital ridge
No gag and cough reflexes	IX, X	IX, X	Visualisation of the posterior oropharynx and stimulation with Yankauer sucker. Passing of suction tubing to carina to stimulate response
Apnoea (ICS Guidance 2013)			Starting PaCO <sub>2</sub> is greater than 6.0 kPa Starting pH is less than 7.4 Allow PaCO <sub>2</sub> to increase by at least 0.5kPa Observe for any respiratory effort for at least 5 minutes

## Performance and repetition of testing

- The performance of brain stem tests should be done by at least **two medical practitioners** who have been fully registered by the General Medical Council for at least **five years**. One of these must be a consultant. Both should be competent in the field and neither should have a conflict with performing the tests.
- **Two sets of tests** are performed to remove the risk of observer error. The two practitioners may do these together or separately. If together, then **each practitioner has to perform a full set of tests**. One practitioner observing

the other practitioner does not count as a second set of tests. The time interval between the tests is not stipulated.

- The legal time of death is when the first set of tests has been completed.
- Death is confirmed on completion of the second set of tests irrespective of when active support is discontinued. Confirmation of death still needs to be performed if there is a circulatory arrest between tests.

## Miscellaneous considerations

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- **Pre-existing respiratory disease** Patients with chronic carbon dioxide retention should have a starting  $\text{PaCO}_2$  of greater than 6.5kPa. The  $\text{PaCO}_2$  should increase over the duration of testing to >0.5kPa above the starting value.
- **Local pathology that precludes clinical testing** Facial trauma can cause difficulties in performing the cranial nerve tests. In this situation ancillary testing such as EEG or cerebral angiogram can be considered although not mandatory.
- **Medical conditions that may mimic brain death.** Medical conditions such as severe Guillain-Barré syndrome, rabies encephalitis, brainstem encephalitis, amitriptyline overdose and bretyllium overdose could mimic brain death.

This chapter considers the management of the patient for organ donation following either brain stem death or circulatory death.

The General Medical Council (GMC) guidance 'Treatment and care towards the end of life: good practice in decision making' requires that consultant staff who have clinical responsibility for patients who are potential donors exercise a duty to consider organ donation as part of end-of life care. The legal framework that allows donation after death is described in the Human Tissue Act 2004.

## Donor identification and referral

Early identification of patients for donation is advised. NICE have published guidance. University Hospital Southampton is still in the process of developing its own guidelines. There should be early liaison with the Specialist Nurse in Organ Donation (SNOD).

When organ donation occurs, it follows either cardiac or brainstem death of the donor:

- **Donor after cardiac death (DCD):** When the medical team and family agree that continuing medical treatment is not in the best interests of the patient, eg poor neurological outcome, then the decision may be made to withdraw active medical treatment, eg stop invasive ventilation. If the patient dies within a few hours of being extubated, then organ donation may be possible.
- **Donor after brain death (DBD):** This refers to organ donation after brainstem testing has demonstrated that the patient is brainstem dead.



## Contraindication to organ donation

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### Absolute contraindications to organ donation

- Age >85 years
- Any cancer with evidence of spread outside affected organ (including lymph nodes) within 3 years of donation (however, localised prostate, thyroid, in situ cervical cancer and non-melanotic skin cancer are acceptable)
- Melanoma (except completely excised Stage 1 cancers)
- Choriocarcinoma
- Active haematological malignancy (myeloma, lymphoma, leukaemia)
- Definite, probable or possible case of human transmissible spongiform encephalopathies, including CJD and vCJD, individuals whose blood relatives have had familial CJD, other neurodegenerative diseases associated with infectious agents
- TB: active and untreated
- HIV disease (but not HIV infection)

### Organ specific contraindications

In addition to the absolute contraindications a number of organ specific contraindications have been developed by each NHSBT Advisory Group to assist in the assessment of a potential organ donor; these are listed below. Each contraindication is specific to the organ listed and does not preclude the donation of any other organ. In some cases, individual transplant units have developed further contra-indications.

#### LIVER

- Acute hepatitis (AST>1000 IU/L)
- Cirrhosis
- Portal vein thrombosis

#### KIDNEY

- Chronic kidney disease (CKD stage 3B and below, eGFR<45)
- Long term dialysis (that is, not acute relating to acute illness)

- Renal malignancy (prior kidney tumours of low grade and previously excised would not exclude donation)
- Previous kidney transplant (> 6 months previously)

#### PANCREAS

- Insulin dependent diabetes (excluding ICU associated insulin requirement)
- Any history of pancreatic malignancy

#### HEART

- Age >65
- Documented coronary artery disease (e.g. confirmed history of MI, CABG or percutaneous stenting)
- Median sternotomy for cardiac surgery
- LVEF≤30% on more than one occasion
- Massive inotropic or pressor support, but only if adequate circulating volume has been confirmed by monitoring

#### LUNGS

- DCD donor age >65; DBD donor age >70 years
- Previous intra-thoracic malignancy
- Significant, chronic destructive or suppurative lung disease (those with controlled asthma are suitable donors)
- Chest X-ray evidence of major pulmonary consolidation

#### Consent

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- Registration on the Organ Donor Register is now seen as a lawful consent under the Human Tissue Act 2004. If a patient is not registered the next of kin should be approached.
- The NICU Consultant and / or SNOD should make the approach for donation.



Discussion with the family about organ donation must be kept separate from the initial discussions considering withdrawal of treatment because of poor prognosis.

If these two issues are not kept separate, the relatives may end up with the impression that the withdrawal of treatment is being considered to facilitate organ donation.

## Organ donation following brainstem death

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Management of the patient being considered for organ donation following Brain Stem Death.

### Monitoring

- All patients:
  - Temperature (nasopharyngeal)
  - SpO<sub>2</sub>
  - ECG (continuous and printed 12-lead)
  - Arterial blood pressure (left radial line)
  - Urine output (urinary catheter)
  - Chest radiograph (CXR)
- The following may also be considered necessary:
  - Central venous pressure (right internal jugular triple lumen catheter)
  - Trans-thoracic or trans-oesophageal echocardiography
  - Fibre-optic bronchoscopy
  - Pulmonary artery catheter (Rarely required)

### Basic management

- Once declared brain dead the donor requires active management to maintain optimal physiology in preparation for multi-organ retrieval. Regular review by the ITU team (doctors, nurses, physiotherapists and the transplant co-ordinator) is vital, as is the communication with the transplant centre accepting the organs on offer.

- Keep the donor warm at 37°C with a warming blanket, bair hugger and warmed intra-venous fluids.
- Maintain an optimal fluid balance (neutral balance to +500 ml) with crystalloid in the form of 5% dextrose to avoid hypernatraemia (secondary to diabetes insipidus) and to maintain hepatic glucose stores. For colloid administration use packed red cells to maintain the Hb > 10g/dl.
- Hourly nasogastric tube suction to minimise the risk of aspiration. 1-2 hourly turning and endo-tracheal/bronchial suctioning using the standard aseptic technique. Two hourly chest physiotherapy for secretion clearance and recruitment manoeuvres.

### Drug therapy

- Methylprednisolone 15mg/kg bolus dose immediately after declaration of brain death.
- Vasopressin 2 unit bolus followed by 1 to 10 unit/hour infusion to maintain MAP > 70 mmHg and SVR 800 – 1200 dyn.s.cm-5. Wean down/off all existing catecholamine infusions after starting vasopressin.
- Tri-iodothyronine 4mcg bolus followed by 3mcg/hour infusion.
- Insulin infusion 1 unit/hour then adjust to maintain glucose of 4-8 mmol/l
- Supplement K<sup>+</sup> and Mg<sup>2+</sup>
- Broad spectrum antibiotics (as per local ITU protocol).

### Ventilation and airway management

- Minimise FiO<sub>2</sub> to maintain PaO<sub>2</sub> 11 – 14 kPa
- PEEP 5 – 10 cmH<sub>2</sub>O
- Tidal volume 10 ml/kg but peak airway pressures < 35cmH<sub>2</sub>O
- Repeat PaO<sub>2</sub> on 100% FiO<sub>2</sub> every 2 hours – should be >40 kPa for the lungs to be considered ideal for retrieval. If the gas exchange deteriorates acutely to < 40 kPa on 100% FiO<sub>2</sub> (despite optimal physiotherapy, endobronchial suctioning and anaesthetic review) inform the transplant centre and retrieval team. Selective pulmonary venous blood gas analysis is then considered to identify regional defects within the lungs.
- Overhydration must be avoided because of the risk of precipitating or potentiating pulmonary oedema.
- Fibre-optic bronchoscopy may be performed to check the endobronchial anatomy and to exclude the presence of aspiration and/or infection. Proximal

tracheal secretions are aspirated and a bronchoalveolar lavage (BAL) performed. The 3 samples (tracheal aspirate, left lung BAL and right lung BAL) are sent to microbiology for M,C+S to help guide the antibiotic treatment of the post-transplant recipient. The discovery of abnormal anatomy (e.g. tracheal RUL bronchus), severely inflamed mucosa and/or copious mucopurulent secretions coming from the airways are adverse features and must be reported directly to the transplant surgeon.

- Hourly turning and bronchial suction using aseptic technique and 1 to 2 hourly chest physiotherapy for secretion clearance and recruitment manoeuvres. A CXR is taken at the declaration of brain death and 3-4 hourly thereafter particularly when there has been an acute deterioration in the gas exchange.

### Optimal donor target parameters

<b>Temperature</b>	37°C
<b>pH</b>	7.36-7.44
<b>PaO<sub>2</sub> (FiO<sub>2</sub> 1.0)</b>	>40 kPa
<b>PaCO<sub>2</sub></b>	3.8-6.0 KPa
<b>PEEP</b>	5-10 cmH <sub>2</sub> O
<b>Tidal volume</b>	10 ml/Kg
<b>Peak airway pressure</b>	<35 cmH <sub>2</sub> O
<b>CXR</b>	NAD
<b>Bronchoscopy</b>	NAD
<b>HR</b>	60-90 sinus rhythm
<b>MABP</b>	65-85 mmHg
<b>ECG</b>	SR. No ST changes or Q waves. No LVH
<b>Fluid balance</b>	0 to +500 ml
<b>Maintenance crystalloid</b>	1 ml/Kg
<b>Urine output</b>	0.5 ml/Kg/hour
<b>Haemoglobin</b>	>10.0 g/dl
<b>CVP</b>	3-8 mmHg
<b>CI</b>	>2.6 l/min/m <sup>2</sup>
<b>PAWP</b>	<10 mmHg
<b>SVR</b>	800-1200 dyn.sec.cm <sup>-5</sup>
<b>TTE/TOE</b>	Normal EF. No valvular pathology. No LVH
<b>Dopamine</b>	<5 µg/Kg/min
<b>Noradrenaline</b>	<0.04 µg/Kg/min
<b>Adrenaline</b>	Nil
<b>ADH</b>	1-10 IU/hour
<b>T3</b>	3 µg/hour
<b>Insulin</b>	As per BM 4-8 mmol/l

## Non-heart beating organ donation

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- Organ donation following death by cardiorespiratory criteria. Donor after Cardiac Death (DCD).
- Patients are those who have irrecoverable brain damage but who do not satisfy formal brain stem criteria for the diagnosis of death.
- After consensus between the medical / nursing staff and the family that further medical intervention is futile and that active treatment should be withdrawn, consideration is made as to whether the patient is suitable to donate solid organs. Document consensus in notes.
- There is potential for donating liver, lungs and pancreas (when time between withdrawal of treatment and death is < 1 hour) and kidneys (< 5 hours), in addition to tissues.
- Patients should be treated in the same way as any other patient for whom active treatment is withdrawn. They should continue to receive appropriate medical and nursing care. This includes adequate analgesia and other symptom relief. Withdrawal of life-support measures may occur in the unit or in the anaesthetic room depending on the relatives.
- Continue active nursing and physiotherapy care.
- Continue monitoring of ECG, pulse oximetry and invasive blood pressure monitoring if possible. These can be monitored from the nurses' station if relatives are distressed at watching the monitor.
- Keep normothermic.
- Do not attempt to resuscitate the patient if they deteriorate unexpectedly. It may prove impossible for organs to be donated and this is explained to the relatives during the initial discussions with the transplant Co-ordinator. If death has not occurred within 1 hour of treatment withdrawal, liver, lung and pancreas donation is not possible. There is a cut off of 5 hours for kidney donation. The transplant Co-ordinator will advise if donation is still feasible.

When death does occur, it is important that the patient is certified promptly. This should be by a physician who is not part of the transplant team and should be **after 5 minutes of asystole**. Thereafter, the relatives can have a **further 5 minutes** at the bedside. If the relatives wish for more time, donation will not go ahead, and this is explained to the next of kin during initial discussions with the transplant Co-ordinator

# Unfractionated heparin IV infusion

# 30

- Check coagulation screen and platelet count before initiating intravenous heparin. Discuss patients with a low platelet count or high APTR/INR with a haematologist.
- If Enoxaparin has been administered in the previous 24 hours and management is unclear, discuss with the consultant neuro-intensivist, pharmacy or haematology.
- Caution in patients with severe renal impairment or low serum albumin.
- Daily platelet count because of risk of Heparin-induced thrombocytopenia (HIT)

## Initiation of unfractionated heparin infusions

- 1** Decide regime with the Neuro ICU consultant and Neurosurgeon/Neurologist:  
**A or B**
  - A) When risk of catastrophic haemorrhage from over-anticoagulation is high:**
    - Load with a bolus of Unfractionated HEPARIN SODIUM **2500** units intravenously
    - Start heparin infusion at dose of **15** units/kg/hr – see table below
  - B) When anticoagulating for newly diagnosed venous thrombosis or embolism:**
    - Load with a bolus of Unfractionated HEPARIN SODIUM **5000** units intravenously
    - Start heparin infusion at dose of **20** units/kg/hr – see table below
- 2** Prescribe Heparin 20,000 units in 20ml (Pump Hep). (The concentration is 1000 units /ml)

### Starting infusion rate

Patient weight (kg)	40	45	50	55	60	65	70	75	80	85	90	>90
<b>Regime A (ml/hr)</b>	0.6	0.7	0.7	0.8	0.9	1.0	1.0	1.1	1.2	1.3	1.3	1.4
<b>Regime B (ml/hr)</b>	0.8	0.9	1.0	1.1	1.2	1.3	1.4	1.4	1.4	1.4	1.4	1.4

- 3** Check APTR 4 hours after starting the heparin infusion or after any change in dose.
- 4** Once stable, APTR can be measured daily.
- 5** If initial APTR >3.5 **or** <1.39, take appropriate action and measure APTR 1 hour after starting the new infusion rate. APTR should be monitored 4-6 hourly until stable.

### Changes to infusion rate

APTR	Infusion rate change (units/hour)	Infusion rate change (ml/hour)
<b>&gt;3.5</b>	Stop infusion for 1 hour then check APTR	Stop 1 hour    ↓ 0.3ml
<b>3.01-3.5</b>	Stop infusion for 30mins then reduce rate by 300 units/hr	Stop 30 min    ↓ 0.3ml
<b>2.51-3.0</b>	Reduce rate by 200 units/hr	↓ 0.2ml
<b>2.21-2.5</b>	Reduce infusion by 100 units/hr	↓ 0.1ml
<b>1.8-2.2</b>	NO CHANGE	NO CHANGE
<b>1.40-1.79</b>	Increase infusion by 100 units/hr	↑ 0.1ml
<b>&lt;1.39</b>	Increase infusion by 200 units/hr	↑ 0.2ml

- If APTR <1.8 **or** > 2.2 ensure the nurse-in-charge and a doctor are informed
- Note if infusion has been stopped for more than 6 hours, check APTR and consider reloading the patient with a bolus of Unfractionated HEPARIN SODIUM 2500 or 5000 units intravenously.



## Bleeding whilst on IV unfractionated heparin

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- Unfractionated heparin has a short half-life; therefore stopping the infusion is usually sufficient.
- Check APTR, clotting screen and full blood count.
- If bleeding is severe contact the haematology registrar. If reversal of anticoagulation is advised intravenous protamine sulphate may be used:
- Give 1mg of protamine sulphate for every 100 units of heparin infused over the preceding 2 ½ hours (usually 25-50mg, maximum 50mg). Give by slow intravenous injection over 10 minutes.
- Recheck APTR to see if further protamine is required.
- N.B. Excess protamine will act as an anticoagulant.

## Guideline

Ward: Neuro ICU

## Adult Intravenous Unfractionated Heparin Infusion Prescription Chart (Pump Hep)

Attach patient addressograph label here or add the following detail

Patient Name: .....  
 Hospital No: ..... Date of Birth: .....  
 Consultant: .....

Loading Dose of Heparin Sodium (if required): by intravenous **bolus** administered over 5 mins

Date: .....	Dose: (units) .....	Dr's Signature: .....	Administered by & time of administration
.....	.....	.....	Time: .....
.....	.....	.....	(1).....
.....	.....	.....	(2).....

**NOTE:**  
 Prescribe  
 IV Heparin on the  
 inpatient chart and  
 state  
 "Refer to  
 intravenous heparin  
 chart"

**Initial Infusion of: Heparin 20,000 units in 20ml (1000units/ml conc<sup>n</sup>) by continuous intravenous infusion**

Date	Time Prescribed	Baseline APTT	Initial Infusion rate	Dr.'s Signature	Administered by	Time Infusion Started	Date & Time Repeat APTT due (4 - 6 hours)
.....	.....	.....	..... (ml/hr) (PTO for table)	.....	(1)..... (2).....	.....	Date: ..... Time: .....

**Subsequent Infusions of: Heparin 20,000 units in 20ml (1000units/ml conc<sup>n</sup>) by continuous intravenous infusion (Repeat APTTs to be documented sequentially in following table)**

Date	Result of repeat APTT	Using schedule overleaf – Heparin infusion rate adjusted to:	Signature of person checking APTT (Nurse/Doctor)	Signatures of Nurses who have adjusted the rate according to the schedule	Date & Time of infusion rate adjustment	Dr.'s Signature & Date (must be signed every 24 hours)	Date & Time repeat APTT due	Date & Time APTT Taken
.....	.....	<b>Either</b> a). No adjustment – maintain at ..... (ml/hr) <b>Or</b> b). Change to new infusion rate of ..... (ml/hr)	.....	(1)..... (2).....	Date: ..... Time: .....	..... (Sig) Date: ..... Time: .....	Date: ..... Time: .....	Date: ..... Time: .....
.....	.....	<b>Either</b> a). No adjustment – maintain at ..... (ml/hr) <b>Or</b> b). Change to new infusion rate of ..... (ml/hr)	.....	(1)..... (2).....	Date: ..... Time: .....	..... (Sig) Date: ..... Time: .....	Date: ..... Time: .....	Date: ..... Time: .....
.....	.....	<b>Either</b> a). No adjustment – maintain at ..... (ml/hr) <b>Or</b> b). Change to new infusion rate of ..... (ml/hr)	.....	(1)..... (2).....	Date: ..... Time: .....	..... (Sig) Date: ..... Time: .....	Date: ..... Time: .....	Date: ..... Time: .....
.....	.....	<b>Either</b> a). No adjustment – maintain at ..... (ml/hr) <b>Or</b> b). Change to new infusion rate of ..... (ml/hr)	.....	(1)..... (2).....	Date: ..... Time: .....	..... (Sig) Date: ..... Time: .....	Date: ..... Time: .....	Date: ..... Time: .....
.....	.....	<b>Either</b> a). No adjustment – maintain at ..... (ml/hr) <b>Or</b> b). Change to new infusion rate of ..... (ml/hr)	.....	(1)..... (2).....	Date: ..... Time: .....	..... (Sig) Date: ..... Time: .....	Date: ..... Time: .....	Date: ..... Time: .....
.....	.....	<b>Either</b> a). No adjustment – maintain at ..... (ml/hr) <b>Or</b> b). Change to new infusion rate of ..... (ml/hr)	.....	(1)..... (2).....	Date: ..... Time: .....	..... (Sig) Date: ..... Time: .....	Date: ..... Time: .....	Date: ..... Time: .....

Full guidelines on reverse – Please keep this chart with all other prescription charts

Developed 12/10/2011  
 Dr J. A. Hell & Dr J. Mainwaring

Guideline

**Intravenous Unfractionated Heparin in Adults  
Nursing Administration Record**

Attach patient addressograph label here or add the following details  
Patient Name.....  
Hospital number.....  
Date of birth.....  
Consultant.....

This administration record is to be used with the heparin guidelines and heparin prescription chart.  
(This chart is not a prescription)

**WARD**.....

Date	Time	Pump Setting/ Infusion rate (ml/hr)	Present volume in syringe (ml)	Volume Delivered since previous reading (ml)	Nurses' Signature for administration

## Y-site compatibility of IV infusions

- The information is provided as a guide only, since although drugs can be compatible, variations in the concentrations used may produce an incompatibility in some circumstances.
- The compatibility data is largely based on physical compatibility, i.e. no visible sign of incompatibility.
- When stated as compatible, it is assumed that drugs are being mixed via a Y-site in a line, not in an infusion bag, burette or syringe.
- Check that the drugs are compatible with the infusion fluids in use, e.g. if dopamine in NaCl 0.9% is to be infused through the same line as dobutamine in 5% glucose, check that both dopamine and dobutamine are compatible with each other and with saline and glucose.
- All drug mixtures should be checked for signs of incompatibility: cloudiness, colour change, haze, precipitate or crystal formation. Infusion sites should be checked regularly for signs of irritation that may be attributable to drug incompatibility.
- The information provided has been carefully checked. However, no responsibility can be accepted for any errors or omissions. The reader is assumed to possess the necessary knowledge to correctly interpret the information provided.

## Drugs which must always be infused separately

Antibiotics	IVIg
Antifungals	Omeperazole
Antivirals	Phenobarbital
Acetazolamide	Phenytoin
Alteplase	Sodium valproate
Mannitol	Thiopentone

## Y-site compatibility of IV infusions

	Vecuronium	SNP	Propofol	Noradrenaline	Morphine	Milrinone	Midazolam	Labetalol	Insulin	Heparin	GTN	Furosemide	Fentanyl	Epinephrine (Adrenaline)	Dopamine	Dobutamine	Calcium	Atracurium	Amiodarone
Aminophylline	C	N	C	I	I	N	N	C	I	C	C	C	N	I	V	I	V	I	I
Amiodarone	N	N	N	C	C	N	C	C	C	I	C	I	N	N	C	C	N	N	
Atracurium	N	V	I	N	C	C	C	N	N	V	C	N	C	C	C	C	N		
Calcium	N	N	I	C	C	C	I	N	N	V	N	N	N	V	V	V			
Dobutamine	C	C	C	C	C	C	V	C	I	V	C	I	C	C	C				
Dopamine	C	C	C	C	C	C	C	C	V	C	C	I	C	C					
Epinephrine (Adrenaline)	C	N	C	C	C	C	C	C	C	C	C	V	C						
Fentanyl	C	N	C	C	C	C	C	C	N	C	C	C							
Furosemide	I	N	C	I	I	I	I	I	C	C	C								
GTN	C	C	C	C	C	C	C	C	C	C									
Heparin	C	C	C	C	V	C	C	V	C										
Insulin	N	C	C	V	C	C	C	N											
Labetalol	C	C	C	C	C	C	C												
Midazolam	C	C	C	C	C	C													
Milrinone	C	C	C	C	C														
Morphine	C	C	C	C															
Noradrenaline	C	N	C																
Propofol	C	C																	
SNP	C																		



Compatible Y-site under certain conditions



Conflicting information of compatibility / incompatibility

**Avoid** combination where possible - use together with extreme cautionNo information available. **Do not mix**Known to be incompatible. **Do not mix**

Information given in this table must be interpreted with caution - please refer to notes on previous page. Further information can be obtained from the duty Neuro ICU pharmacist.

## Common drug infusions

Drug	Dose <i>Volume</i>	Final Volume	Diluent	Final conc.	Starting dose range
<b>Adrenaline</b> 1:1000	5 mg 5 ml	50 ml	5% Dextrose	100 µg/ml	0–10 ml/hr
<b>Atracurium</b>	500 mg 50 ml	50 ml	Neat	10 mg/ml	0–6 ml/hr
<b>Clonidine</b>	750 µg	50 ml	0.9% Saline	15 µg/ml	0–3 ml/hr
<b>Dobutamine</b>	250 mg 20 ml	50 ml	5% Dextrose or 0.9% Saline	5 mg/ml	0–20 ml/hr
<b>Dopamine</b>	200 mg 5 ml	50 ml	5% Dextrose or 0.9% Saline	4 mg/ml	0–5 ml/hr
<b>Fentanyl</b>	2.5 mg 50 ml	50 ml	Neat	50 µg/ml	0–4 ml/hr
<b>GTN</b>	50 mg	50 ml	Neat	1 unit/ml	0–10 ml/hr
<b>Insulin</b>	50 units 0.5 ml	50 ml	0.9% Saline	1 unit/ml	sliding scale
<b>Labetalol</b>	200 mg 40 ml	200 ml	0.9% Saline	1 mg/ml	20–160 ml/hr
<b>Midazolam</b> <i>single strength</i>	50 mg	50 ml	0.9% Saline	1 mg/ml	0–30 ml/hr
<i>double strength</i>	100mg	50 ml	0.9% Saline	2 mg/ml	0–30 ml/hr
<i>quad strength</i>	200mg	50 ml	0.9% Saline	4 mg/ml	0–30 ml/hr
<b>Morphine</b>	60 mg	60 ml	0.9% Saline	1 mg/ml	0–5 ml/hr
<b>Noradrenaline</b>	4 mg 4 ml	50 ml	5% Dextrose	80 µg/ml	0–10 ml/hr
<b>Noradrenaline</b>	20 mg 20 ml	250 ml	5% Dextrose	80 µg/ml	0–10 ml/hr
<b>Phenylephrine</b> <i>i.v. bolus</i>	10 mg 1 ml	100 ml	0.9% Saline	0.1 mg/ml	1 ml boluses
<i>infusion</i>	10mg	500ml	0.9% Saline	20mcg/ml	0-180 ml/hr
<b>Phenytoin</b> <i>loading dose</i>	1000 mg 20 ml	100 ml	0.9% Saline	10 mg/ml	over 1 hour
<i>maintenance</i>	300mg 6ml	100 ml	0.9% Saline	3 mg/ml	over 30 min
<b>Propofol</b>	1%	50 ml	Neat	10 mg/ml	0–20 ml/hr
<b>Thiopentone</b>	2.5 g	100 ml	Water for injection	25 mg/ml	3–5 mg/kg/hr

**Noradrenaline**

ml/hr	mg/hr	µg/min
1	0.08	1.3
2	0.16	2.66
3	0.24	4
4	0.32	5.3
5	0.4	6.6
6	0.48	8
7	0.56	9.33
8	0.64	10.66
9	0.72	12
10	0.8	13.33

Noradrenaline infusion 20 mg in 250 ml (or 4 mg in 50 ml)

**Adrenaline**

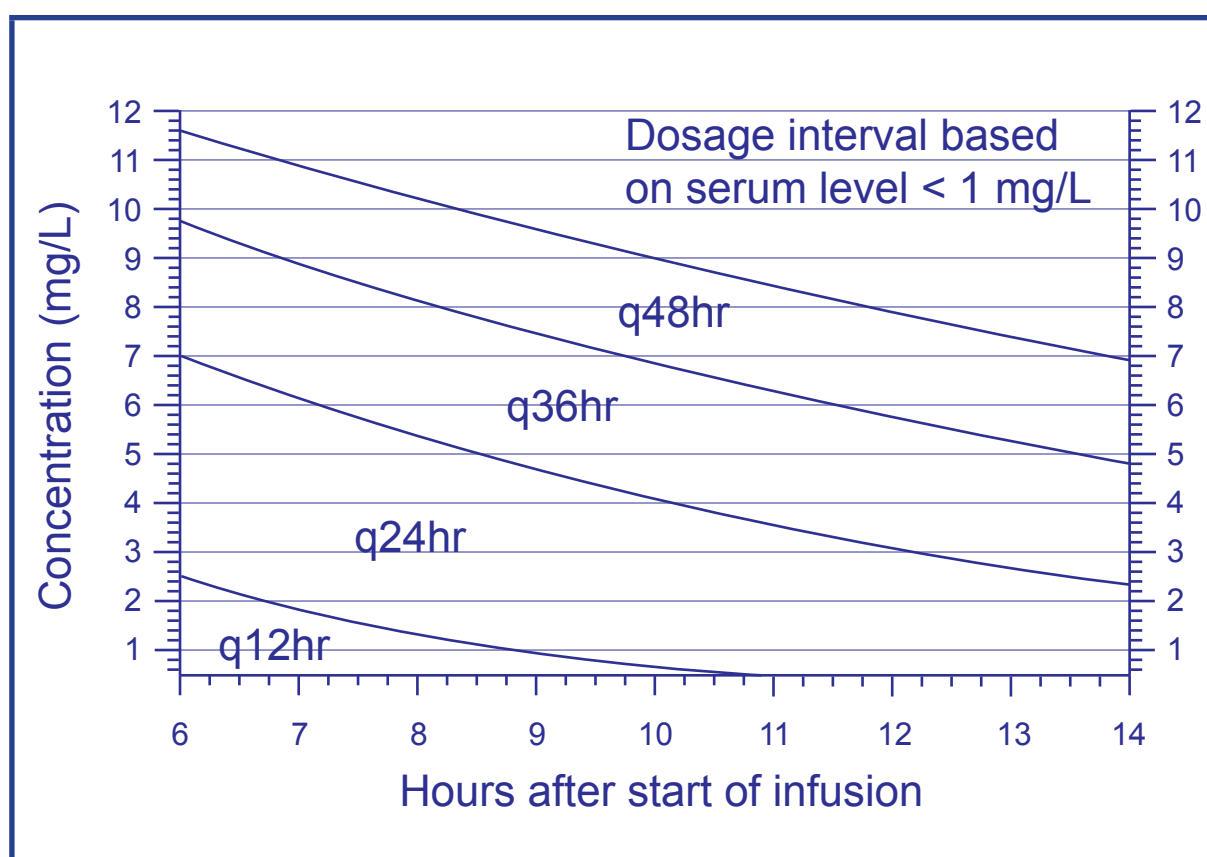
ml/hr	mg/hr	µg/min
1	0.1	1.66
2	0.2	3.33
3	0.3	5
4	0.4	6.66
5	0.5	8.3
6	0.6	10
7	0.7	11.66
8	0.8	13.33
9	0.9	15
10	1.0	16.66

Adrenaline infusion 5 mg in 50 ml

## Therapeutic drug monitoring on Neuro ICU

Drug	Range	Timing
Digoxin	0.9-2.6 nmol/L 0.5-2 mcg/L	At least 6 hours post dose
Phenytoin	5-20 mg/L	Blood must be taken pre-dose
Aminophylline	10-20mg/L	Pre-dose, or >6hrs into infusion
Vancomycin	15-20mg/L	Immediately pre-dose
Gentamicin (5mg/kg dosing)	Nomogram (See below)	6-14hrs after dose administered

### Urgan & Craig normogram for gentamicin dosing 5mg/kg



#### Urgan & Craig normogram for gentamicin dosing 5mg/kg

This nomogram is designed to ensure that gentamicin blood levels are below 1mg/L for at least 4 hours during a dosing interval. The nomogram will also ensure that the maximum duration of post-antibiotic effect does not have to be longer than 16 hours.



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