WESSEX NEUROLOGICAL CENTRE

NEUROSCIENCE INTENSIVE CARE UNIT PROTOCOLS

Edited by

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and

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Chapter 1

Responsibility for Patient Care on NICU

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Date: February 2006
Review Date: February 2007

1.1 Introduction

Patients on the NICU require multidisciplinary care. Without clear communication and patient care protocols there is a danger that coherent patient management may be compromised. This document seeks to outline mutually agreed practice for caring for NICU patients.

1.2 Neurosurgical and Neurological Admissions

Neurosurgical and neurological admissions who pass through the NICU remain under the care of the admitting Consultant, but, during their stay in the NICU, responsibility for their care is shared with the NICU Consultants. Some aspects of care will primarily fall within the remit of either the neurosurgical team (e.g. decisions regarding surgical management) or the ICU team (e.g. ventilatory management), but many issues may be best managed in consultation between the two teams. It is essential that admitting consultants maintain their input into patient care; such input will vary depending on the patient and the individual admitting consultant.

The specialist registrar in each consultant team will be the primary point of contact for such input, augmented by joint bedside discussions between the neurosurgical / neurology and NICU consultants whenever possible. Decisions regarding the care of individual patients will be discussed on the 07:45 ward round. While several people may usefully participate in these discussions, the
final decision on neurosurgical / neurological management will be the responsibility of the specialist registrar responsible for the patient. Clearly, in some cases, this may result in a need to briefly defer decision making until the appropriate consultant has been contacted. In the absence or unavailability of the specialist registrar from the admitting team, this responsibility will devolve to the duty SpR. Outside normal working hours, this responsibility will devolve to the on-call neurosurgical or neurology SpR, unless other arrangements are specifically made.

The NICU Consultants will be responsible for co-ordinating the multidisciplinary care of patients, including that provided by medical staff from outside neurosciences (eg orthopaedics) who may have a consultative role in the management of NICU patients. It is important that the NICU team be informed about management changes so that they may fulfil this co-ordinating role effectively.

These guidelines cannot be all-inclusive, and there will clearly be times when common sense dictates that they cannot be followed, and other instances where there is a difference of opinion regarding management decisions. I would expect that with time, working relationships between individual Consultant colleagues would be more clearly defined, and such instances would become progressively less common. It is important to recognise however, that they will still occur. In such instances, I would suggest that any differences of opinion need to be settled in discussions away from the bedside. This practice will improve team morale, and prevent nursing staff and junior doctors from being placed in a difficult position.

1.3 NICU (ICU/HDU) Daily Timetable

1.3.1 Weekdays

07.45–08.15: Ward round (neurosurgical SpRs, neurosurgical SHOs, NICU trainees, NICU consultant(s), nurse looking after patient, nurse in charge (where possible).

11.00–12.30: NICU ward round (NICU trainees, NICU consultant, nurse in charge, nurse looking after patient).

16.00–16.30: Hand-over ward round (duty neurosurgical SpR and neurosciences SHO, NICU trainees, NICU consultant(s), senior nurse).

1.3.2 Saturday/Sunday

08.00–08.30: AM ward round: (duty neurosurgical SpR, neurosciences SHO and NICU consultant. Senior nurse. The duty NICU Consultant will conduct a ward round during the course of the day.

Neurosciences patients on the GICU will be reviewed at the end of the NICU ward round when appropriate.
Chapter 2

NICU Admission and Discharge

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Director of Neurosciences Intensive Care Unit.

Date: February 2006
Review Date: February 2007

2.1 Admission Guidelines

The Consultant covering the NICU should be informed regarding all admissions.

<table>
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<th>NICU Level 3 beds</th>
<th>Need for ventilation for acute ventilatory failure or ICP control</th>
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<tbody>
<tr>
<td></td>
<td>More than 2 vasoactive agents</td>
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<tr>
<td></td>
<td>High and varying dose of vasoactive agents</td>
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<tr>
<td>NICU Level 2 Beds</td>
<td>Invasive haemodynamic monitoring</td>
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<tr>
<td></td>
<td>Non-invasive ventilation</td>
</tr>
<tr>
<td></td>
<td>$F_iO_2 &gt; 0.7$</td>
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</table>

Table 2.1: NICU admission criteria

2.2 Discharge Guidelines

Discharge to ward if:

- Haemodynamically stable.
- No requirement for inotropes.
• $F_1O_2 < 0.6$, no CPAP.

• Secure airway.

• Tracheostomy acceptable, but only if needing suction < than 2 hourly.

Transfers to the wards may be undertaken outside these guidelines only after discussion between medical and nursing staff on the NICU.

Patients will be transferred from the ICU/HDU only when the NICU Consultant of the day and the neurosurgical/neurological team caring for the patient have agreed that such a move is appropriate.

2.3 Admission Procedures

The NICU primarily services the Wessex Neurological Centre and all admitting firms within WNC have equal right of access to its beds. The running of the unit is the responsibility of the director of the NICU. Admission and discharge policies are defined in consultation with other consultants in WNC. Day-to-day organisation of the work of the unit is the responsibility of the NICU consultant in charge. Admissions to the NICU are best considered under different levels of bed availability, but the central principle of bed management is that the Consultant covering the NICU must be informed of all admissions.

2.3.1 When beds are freely available

Admission will be automatic. In practice, the admitting neurosurgical or neurology SpR will accept the patient after checking bed availability with the unit and speaking to the NICU consultant. It will be the duty of the NICU staff to inform the on-call consultant regarding admissions that are accepted after 17:00 if appropriate. This practice will ensure that any changes in planned activity that an acute admission produces can be organised as early as possible. For example, while an acute extradural haemorrhage clearly has to be admitted, the patient may occupy a bed that was meant to receive an elective postoperative admission. Clearly, early feedback regarding the unavailability of beds will minimise any disruption caused by changed plans.

2.3.2 When no beds are available but patients are thought to be fit for transfer to the ward

In many instances the decision to transfer such patients may have already been made on the ward round, but delayed because of unavailability of beds on the ward or logistical convenience. In such circumstances, if the patient’s condition is unchanged, the transfer can be effected and a new patient accepted as above. If the decision to transfer the patient has not been made, such a decision should be made by the NICU Consultant on call. In some instances this decision will require consultation with the Neurosurgical Team caring for the patient, the anaesthetic SpR on call or a bedside review of the patient’s status.
2.3.3 When no beds are available or can be freed

Under these circumstances the admitting team will approach the GICU to request admission. If these admissions are accepted by the GICU, the NICU will ensure that these patients are taken over at the earliest possible opportunity when beds are available.

2.3.4 When no ICU beds are available in the hospital

The on call Clinical Manager should be informed if this situation is imminent or has been reached, and admitting teams will be requested to tailor patient acceptance in the light of these circumstances. If emergency admissions are still felt to require transfer from other hospitals based on clinical need, consideration should be made as to urgent repatriation of any NICU patients already accepted for transfer. If none have been identified, consideration should be made to transfer out the most stable ICU patient to another hospital within the region. Identification of such patients should be done by the senior doctor and senior nurse covering the unit. Liaise with the duty neuroanaesthetist / intensivist as to who might be available to accompany the patient. This should be flagged as a major clinical incident and the NICU director informed as soon as possible on the next working day.

2.3.5 When beds are available but nursing staff are short

The default decision under these circumstances will be to accept the new admission if possible, but it is essential that arrangements for accepting the patient are made before transfer is initiated. There must be a designated plan for any patient that is accepted, and it is the responsibility of the admitting SpR to ensure that such a plan has been made. The responsibility for providing the nurses required for the duration of the patients stay cannot rest with NICU staff. If this is not possible, the senior nurse covering the NICU will discuss the situation with the NICU consultant on call. If the situation is felt to be unsafe, the admitting Neurosurgical/Neurology teams will be informed of our inability to accept admissions and take such action as is required (see above).

2.3.6 Planned Admissions

The high percentage of emergency and urgent admissions make the cancellation of planned admissions regretfully common. If such an admission has been cancelled or postponed on more than one occasion, the NICU consultant for the day will formally meet with the consultant requesting the planned admission to try and protect a bed for the next day. It is recognised however, that this may not always be possible.

2.4 Discharge Protocols

No patient will be discharged from the ICU or HDU areas without the approval of the NICU consultant covering the Unit and the patients admitting consultant and, when appropriate, after discussion with the nurse in charge of the NICU.
In practice, this decision is often delegated to resident medical staff, but the ultimate responsibility rests at consultant level. The neurosurgical/neurology teams will reassess patients on at least a daily basis with the option of discharge being explicitly considered where appropriate. In most cases, elective decisions regarding the fitness of patients to leave the unit will be made by the NICU consultant (on weekdays), following discussion with nursing staff, after ward rounds at 07:45, 11:00 and 16:00 hours.
Chapter 3

Routine Post-operative Care

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Date: February 2006
Review Date: February 2007

3.1 Introduction

Patients will be admitted to the NICU (ICU/HDU) rather than the ward following surgery or coiling for one of the following reasons:

1. Long operations
2. Large blood losses
3. Overnight ventilation to ensure stability
4. Monitoring to ensure stable postoperative haemodynamics
5. Preoperative medical problems
6. Perioperative problems

The aims are to control haemodynamics, ensure that any blood loss is replaced and rewarm the patient. If appropriate, special measures may be needed to ensure adequate analgesia, but this is generally not a problem for cranial surgery.

3.2 On Admission

1. Clerk the patient in. A brief summary will suffice: admission diagnosis, surgery, any problems with operation and expected problems.
2. Always clearly state the reason for admission to NICU.

3. Routine post-op bloods are FBC, clotting screen and U&Es. Consider osmolalities, crossmatch and whether the patient need beriplex, vitamin K, FFP or platelets.

4. CXR if ventilated, or gas exchange abnormal (i.e. needs more than 40% O$_2$, or needs O$_2$ for more than 4 hours postop to maintain saturations > 95%).

5. CXR if line insertion or tube changed.

6. ECG.

7. Arterial line (all ventilated patients, all aneurysms, vasoactive drugs, other indications).

8. CVP line should be placed if large volume losses, significant cardiac disease, vasoactive infusions, hypotensive or oliguria not readily responsive to fluid challenge.

9. ABG if arterial line in, or if $S_aO_2$ a problem.

### 3.3 Analgesia For Non-Ventilated Patients

1. Codeine 30–60mg up to 6-hourly im/po/ng (never iv).

2. Paracetamol 1g po/ng/pr (max 4g/d).


### 3.4 Ventilated Patients

#### 3.4.1 Sedation

- Use propofol 2–4mg/kg/hr (10–30 ml/hr for an average adult) if overnight ventilation planned.

- Midazolam up to 5–20mg/hr in an average adult if longer ventilation anticipated.

#### 3.4.2 Analgesia

- Use opiates sparingly: No more than 2µg/kg/hr of fentanyl (typically 1–2ml/hr for average adult).

- Use morphine infusion (1–4mg/hr) if longer ventilation anticipated.

- Use paracetamol for additional analgesia or antipyresis.
3.4.3 Paralysis

- Atracurium 0.5 mg/kg/hr if paralysis indicated (4–5 ml/hr in an average adult).

3.4.4 Ventilation

Initial settings SIMV; $F_{I\text{O}_2}$ 0.4; VT 10 ml/kg; rate 12–16 (adults); PEEP 5–10 cm H$_2$O

Targets $P_{a\text{O}_2}$ > 11 kPa; $P_{a\text{CO}_2}$ 4–4.5 kPa; pH 7.35–7.45.

Manipulation Increase or decrease rate to change $P_{a\text{CO}_2}$; increase $F_{I\text{O}_2}$, or PEEP to increase $P_{a\text{O}_2}$. Try to keep peak inspiratory pressure < 30 cm H$_2$O.

3.5 Haemodynamic Management

1. Aim for heart rate 60–100 bpm, no swing on arterial line (CVP 6–10 mmHg), normal MAP or CPP > 70 mmHg, urine output >0.5 ml/kg/hr.

2. Initial crystalloid 1 litre 0.9% saline with 3 g KCl / 8 hrs. After that depends on electrolyte results (aim for Na 135–145 mmol/l, K 4–5 mmol/l).

3. Colloid until Hb < 8 g/dl then blood. Initial colloid gelofusin: replace with FFP or other clotting factors if needed.

4. Follow up all interventions with re-assessment. Always recheck FBC and clotting if significant transfusion, electrolytes if supplemented and ABGs if ventilation changed.
Chapter 4

Craniospinal Trauma

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Date: April 2006
Review Date: April 2007

4.1 Initial Resuscitation

Initial assessment and resuscitation should follow the guidelines suggested by the ATLS protocol (Table 4.1). The identification of compromised airway, inadequate ventilation or circulatory insufficiency must take precedence over detailed assessment of the neurological state.

A Airway (with cervical spine control)
B Breathing
C Circulation (with haemorrhage control)
D Disability (neurological assessment)
E Exposure (to identify all injuries)

Table 4.1: Resuscitation Priorities in Trauma

4.1.1 Airway Management

The first priority must always be to secure, maintain and protect a clear airway. Remove secretions and foreign bodies by manual extraction or suction, giving oxygen by mask (10–12l/min). There are many threats to the patency of the airway or the adequacy of breathing, and some require immediate correction. Table 4.2 shows the indications for immediate controlled rapid sequence
induction (to avoid aspiration secondary to regurgitation), orotracheal intubation (avoid nasotracheal intubation as the skull base may be fractured), and assisted ventilation. Patients with any of the features in the second part of the table, though not in immediate danger, should also be intubated and ventilated in this way if they are to be transported. Drugs should be used for sedation, analgesia, and muscle relaxation (see Table 4.7). Take great care to protect the cervical spine from movement during intubation. Use cuffed endotracheal tubes in adults (not in children under 12) and secure them with adhesive tape that does not pass round the neck, or with loosely tied tape (to avoid reducing venous return from the head). Check repeatedly that both sides of the chest are moving in case the tube migrates down a bronchus. Pass an orogastric tube to empty the stomach, which commonly dilates after trauma.

<table>
<thead>
<tr>
<th>Immediately</th>
<th>Ventilation</th>
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<tbody>
<tr>
<td>Coma (GCS &lt; 8)</td>
<td>An intubated patient must also be ventilated.</td>
</tr>
<tr>
<td>Loss of protective laryngeal reflexes</td>
<td>Aim for $P_{aO2}$ &gt; 13 kPa, $P_{aCO2}$ 4.0–4.5 kPa. (presume raised ICP).</td>
</tr>
<tr>
<td>Ventilatory insufficiency (as judged by blood gases):</td>
<td></td>
</tr>
<tr>
<td>hypoxaemia ($P_{aO2} &lt; 13$ kPa on oxygen)</td>
<td></td>
</tr>
<tr>
<td>hypercarbia ($P_{aCO2} &gt; 6$ kPa)</td>
<td></td>
</tr>
<tr>
<td>Spontaneous hyperventilation causing $P_{aCO2} &lt; 3.5$ kPa</td>
<td></td>
</tr>
<tr>
<td>Respiratory arrhythmia</td>
<td></td>
</tr>
</tbody>
</table>

| Before the start of the journey                  |                                     |
| Deteriorating consciousness (Fall in GCS by 2 points since admission, not due to drugs, even if not in coma) |                                     |
| Bilaterally fractured mandible                   |                                     |
| Copious bleeding into mouth (for example, from skull base fracture) |                                     |
| Seizures                                         |                                     |

Table 4.2: Indications for Intubation and Ventilation after Head Injury

4.1.2 Cervical Spine Control

Patients who have sustained a head injury and present with any of the following risk factors should have full cervical spine immobilisation unless other factors prevent this:

1. GCS less than 15 at any time since the injury.
2. Neck pain or tenderness.
3. Focal neurological deficit.
4. Paraesthesia in the extremities.
5. Any other clinical suspicion of cervical spine injury.

Keep the neck immobile, in line with the body, apply a rigid or semirigid cervical collar, and (unless the patient is very restless) secure the head to the trolley
with sandbags and tape. Cervical spine injury can be difficult to diagnose in the unconscious patient and should be assumed to be present until it can confidently be excluded. Normal cervical spine films do not exclude the possibility of important cord injury. Maintain aligned immobilisation of the neck when turning the patient during examination and treatment.

### 4.1.3 Breathing

Whenever possible, use a ventilator rather than hand ventilation, and assess the adequacy of ventilation clinically and by arterial blood gas analysis. If possible, an indwelling cannula should be inserted when the initial arterial sample for blood gas analysis is obtained, thus allowing serial blood gas measurement and continuous recording of blood pressure (see section 4.5). Pulse oximetry is valuable for indirect measurement of how well the patient is being oxygenated, but it can mislead if peripheral perfusion is poor, the probe is dirty or incorrectly applied, or the patient is particularly restless, and it gives no information about arterial carbon dioxide tension. Target $P_{a}O_2$ and $P_{a}CO_2$ values are specified in Table 4.2.

### 4.1.4 Circulation

Hypotension is a late sign of hypovolaemic shock, especially in children and fit young adults. Pulse rate, respiratory rate and capillary refill time are more useful ways of assessing the circulation after injury. An isolated head injury is almost never the cause of shock, especially in adults.

Maintenance of cerebral perfusion pressures ($CPP = MAP - ICP$) above 60 mmHg is the single best intervention that improves outcome. The ICP will not be measured at this stage but is certain to be high if the CT scan suggests raised intracranial pressure. Assume that the ICP will be > 20–30 mmHg, and maintain MAP above 90 mmHg. Avoid severe hypertension (MAP > 120 mmHg).

Set up two large bore, peripheral intravenous infusions and rapidly infuse an electrolyte solution like normal saline or Hartmann’s solution (not dextrose). Underinfusion is a more common error than overinfusion and loss of more than 15% of blood volume needs correction by blood transfusion. In patients with obvious hypovolaemia, early direct monitoring of arterial pressure and central venous pressure is helpful for assessing the adequacy of resuscitation. In such cases, if an internal jugular line is required, if possible, avoid the right internal jugular vein. Do not compromise resuscitation for this requirement.

Identify and control sources of blood loss; have a high index of suspicion for thoracoabdominal injuries and for major pelvic and limb fractures. Delay in recognising such injuries is common and is associated with a high incidence of hypovolaemic shock and a poor outcome. A patient in persistent clinical shock despite fluid resuscitation must not be transported for computed tomography or to the Neurosurgical Unit until the source of continuing major blood loss has been identified and controlled as part of resuscitation—in theatre if necessary.
CHAPTER 4. CRANIOSPINAL TRAUMA

4.1.5 Disability
The patient’s conscious level should be measured using the Glasgow Coma Scale, modified in the case of young children (Table 4.4 and 4.5. Repeat and document the measurements often—every 10 minutes at least during the first hour in hospital. Never assume that altered conscious level is due simply to alcohol.

Record any asymmetry of limb movements, and compare the pupils repeatedly for size and reaction to light. Any deterioration in conscious level or the development of focal neurological signs must be recorded and acted on: the patient may have become hypoxaemic or shocked, or have an expanding intracranial haematoma.

Burrhole exploration of a presumed extradural haematoma in a General Hospital seldom saves life. A single burrhole will not decompress a life-threatening extradural haematoma, and often the haematoma proves to be intradural or the burrholes miss it altogether. Time is wasted that would be better spent getting the patient to the Neurosurgical Unit. A mannitol infusion (0.25–1.0 g/kg) can buy time during transfer of a patient with clear signs, clinically or on computed tomography, of an expanding intracranial haematoma.

Conscious level and limb movements cannot be measured in a patient who has been pharmacologically paralysed and ventilated. Repeated assessment of pupil size and reaction (which is unaffected by pharmacological paralysis) is especially important in such patients.

4.1.6 Exposure: Other Injuries
Remove all clothing and check the patient for injuries from head to toe, front and back.

Some injuries do not need treatment at once but will do later, and all should be documented. Consider the need for neck, abdominal and thoracic CT scans when the head is being scanned. CT of the torso is best used in patients who are not so unstable as to require exploration, but would benefit from having such injuries excluded before transfer.

4.1.7 Monitoring
The minimum standards of monitoring for anaesthesia recommended by the Association of Anaesthetists apply equally to a seriously injured patient during resuscitation and transfer: Continuous monitoring of blood pressure, electrocardiography, oxygen saturation, and urine output (by catheter).

In ventilated patients, monitor end-tidal carbon dioxide concentrations and check arterial blood gas concentrations repeatedly (keep $P_aO_2 > 13$ kPa and $P_aCO_2 4–4.5$ kPa). Avoid excessive hyperventilation, which can depress the myocardium and induce cerebral ischaemia.

4.2 Indications for CT Head
Patients who have sustained a head injury and present with any one of the following risk factors should have CT scanning of the head immediately requested.

1. GCS less than 13 at any point since the injury.
4.2. **INDICATIONS FOR CT HEAD**

2. GCS equal to 13 or 14 at 2 hours after the injury.

3. Suspected open or depressed skull fracture.


5. Post-traumatic seizure.

6. Focal neurological deficit.

7. More than one episode of vomiting (clinical judgement should be used regarding the cause of vomiting in those aged 12 years or younger, and whether imaging is necessary).

8. Amnesia for greater than 30 minutes of events before impact. The assessment of amnesia will not be possible in pre-verbal children and is unlikely to be possible in any child aged under 5 years.

CT should also be immediately requested in patients with any of the following risk factors, provided they have experienced some loss of consciousness or amnesia since the injury:

1. Age greater than or equal to 65 years.

2. Coagulopathy (history of bleeding, clotting disorder, current treatment with Warfarin).

3. Dangerous mechanism of injury (a pedestrian struck by a motor vehicle, an occupant ejected from a motor vehicle or a fall from a height of greater than 1 metre or five stairs). A lower threshold for height of falls should be used when dealing with infants and young children (that is, aged under 5 years).
| **Suspect hypovolaemia** | Even in the absence of hypotension 
Tachycardia, arterial respiratory swing, capillary refill, source of bleeding, oliguria |
| **Confirm hypovolaemia** | Measure CVP (avoid RIJ cannulation if possible) |
| **Find a cause for hypovolaemia** | Multiple fractures, thoracic or abdominal haemorrhage |
| **Correct hypovolaemia** | Targets: CVP = 8–10 mm Hg, Hct = 30–45%, normal clotting 
There is no need to “run head injured patients dry”. Use 0.9% saline, Hartmann’s solution, Gelofusin, Haemaccel, blood, FFP. Avoid 5% dextrose, Hetastarch, and hypotonic solutions (unless being used to treat hypernatraemia or osmolality). |
| **Catheterise bladder and measure urine output** | Polyuria is usually the consequence of mannitol. If a profuse diuresis results in hypovolaemic hypotension, replace urinary losses with colloid. If polyuria is disproportionate or persistent consider diabetes insipidus. |
| **Assume elevated ICP** | (> 20 mmHg) unless proven otherwise in patients with GCS < 12. |
| **Maintain MAP** | To ensure CPP > 60 mmHg 
Use vasopressors / inotropes (ephedrine 3 mg as boluses, noradrenaline infusion) to maintain MAP if needed. |
| **If resistant hypotension consider** | Myocardial contusion, tamponade, tension pneumothorax, high spinal cord injury, coning |

Table 4.3: Systemic and Cerebral Haemodynamic Management in Acute Head Injury
4.2. INDICATIONS FOR CT HEAD

**Eye opening response:**
- Spontaneously: 4
- To speech: 3
- To pain: 2
- None: 1
- Eye closed due to swelling: C

**Best motor response:**
- Obeys commands: 6
- Localisation to painful stimuli: 5
- Normal flexion to painful stimuli: 4
- Spastic flexion to painful stimuli: 3
- Extension to painful stimuli: 2
- None: 1

**Best verbal response:**
- Oriented: 5
- Confused: 4
- Inappropriate words: 3
- Incomprehensible sounds: 2
- None: 1
- Intubated: T

Table 4.4: The Glasgow Coma Scale in Adults. Some units (including Southampton and Glasgow) score out of 14 due to inter-observer variability in classifying flexion and spastic flexion.

<table>
<thead>
<tr>
<th>Age</th>
<th>Best motor response</th>
<th>Best verbal response</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 6 months</td>
<td>Flexion</td>
<td>Smiles and cries</td>
</tr>
<tr>
<td>6–12 months</td>
<td>Localisation</td>
<td>Smiles and cries</td>
</tr>
<tr>
<td>1–2 years</td>
<td>Localisation</td>
<td>Sounds and words</td>
</tr>
<tr>
<td>2–5 years</td>
<td>Obeys commands</td>
<td>Words and phrases</td>
</tr>
</tbody>
</table>

Table 4.5: Modification of Normal Responses in Children under 5
CHAPTER 4. CRANIOSPINAL TRAUMA

4.2.1 Investigations for Injuries to the Cervical Spine

See also section 4.8

1. The current investigations of choice for the detection of injuries to the cervical spine are three-view plain radiographs of good technical quality.

2. Where it is not possible to achieve the cervical spine views desired with X-ray, CT imaging is indicated.

3. CT is also indicated if the plain film series is technically inadequate (for example, desired view unavailable), suspicious or definitely abnormal or if there is continued clinical suspicion of injury despite a normal study.

4.3 Indications for Neurosurgical Referral

Criteria for referring head injured adults and children are shown in below, but it is important to discuss any case that you are unsure about with the Neurosurgical SpR on call. Much time may be saved if the items of information outlined in Table 4.6 are available when speaking to the Neurosurgeon.

4.3.1 Criteria For Neurosurgical referral of Head Injured Patients

Without Preliminary Head CT

1. Coma (not obeying commands) even after resuscitation — even if no skull fracture.

2. Deterioration in level of consciousness ($\geq 2$ GCS points) or progressive neurological deficit.

3. Open injury, depressed fracture, penetrating injury or suspected basal skull fracture.

4. Tense fontanelle in a child.

5. Patient fulfils criteria for CT, but this cannot be performed within a reasonable time (3–4hrs).

6. Unexplained confusion persisting for $>4$ hours.

7. Seizure without full recovery.

After CT in a General Hospital

1. Abnormal CT (preferably after neurosurgical opinion on electronically transferred images).

2. CT normal, but patient’s progress unsatisfactory.
4.3.2 What the Neurosurgeon needs to know

Referrals to the Wessex Neurological Centre should be by phone to the on call neurosurgical SpR, contactable through Southampton General Hospital switchboard on 023 8077 7222, bleep number 2877. The information in table 4.6 will be extremely helpful.

<table>
<thead>
<tr>
<th>Patient's age</th>
<th>(if known)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PMH</td>
<td></td>
</tr>
<tr>
<td>History of Injury</td>
<td>Time of Injury</td>
</tr>
<tr>
<td></td>
<td>Cause and mechanism (e.g height of fall, approximate velocity of impact)</td>
</tr>
<tr>
<td>Neurological State</td>
<td>Neurological status at scene</td>
</tr>
<tr>
<td></td>
<td>Conscious level on arrival at A&amp;E</td>
</tr>
<tr>
<td></td>
<td>Trend in conscious level after arrival (sequential GCS)</td>
</tr>
<tr>
<td></td>
<td>Pupil and limb responses</td>
</tr>
<tr>
<td>Cardiorespiratory State</td>
<td>Blood pressure and pulse rate</td>
</tr>
<tr>
<td></td>
<td>Arterial blood gases, respiratory rate pattern.</td>
</tr>
<tr>
<td>Injuries</td>
<td>Skull fracture</td>
</tr>
<tr>
<td></td>
<td>Extracranial Injuries</td>
</tr>
<tr>
<td>Imaging findings</td>
<td></td>
</tr>
<tr>
<td>Management</td>
<td>Airway protection, ventilatory status</td>
</tr>
<tr>
<td></td>
<td>Circulatory status and fluid therapy</td>
</tr>
<tr>
<td></td>
<td>Treatment of associated injuries</td>
</tr>
<tr>
<td></td>
<td>Monitoring</td>
</tr>
<tr>
<td></td>
<td>Drug doses and times of administration</td>
</tr>
</tbody>
</table>

Table 4.6: What the neurosurgeon needs to know

4.4 Drugs and Dosages

4.4.1 Drugs and Dosages for Intubation

The choice of anaesthetic drugs and neuromuscular blockers used for intubation is essentially an individual decision, which will be modified by drug availability and clinical circumstance. We would encourage the use of an intravenous anaesthetic agent prior to intubation even in the patient who is neurologically obtunded, since these agents can markedly attenuate the rises in intracranial pressure associated with intubation. We tend to use etomidate (up to 2 mg/kg) in the hypovolaemic or hypotensive patient, and either thiopentone or propofol in others. We do not, at present, recommend the use of ketamine in this clinical
situation.

The majority of such patients will require a rapid sequence intubation with cricoid pressure with or without in-line cervical traction. In such circumstances the rapid onset of action of suxamethonium clearly outweighs any theoretical risk of increase in intracranial pressure caused by this agent. There is no contraindication to the use of opiates in a patient who is intubated and ventilated, but we would recommend the use of synthetic opiates, since morphine may be associated with histamine release, reductions in blood pressure and increases in ICP. It is essential that large doses of fentanyl or alfentanil are not administered as a rapid bolus, since these may cause reductions in MAP and reflex rises in ICP, even in the ventilated patient. We tend to restrict individual boluses of fentanyl to 100µg.

4.4.2 Maintenance of Anaesthesia

Anaesthesia may be maintained with any intravenous agent except ketamine, but we would recommend propofol infusion at a dose of 2–4 mg/kg/hr, titrated to individual patient response. It is essential to avoid hypotension during transfer, and if adequate anaesthesia cannot be maintained without falls in MAP below 90 mmHg, we would suggest further volume loading, supplementation with opiates, or the substitution of another intravenous agent (e.g. a benzodiazepine). In such patients it is essential to exclude inadequate fluid resuscitation, tension pneumothorax, pericardial tamponade, or concealed or continuing haemorrhage prior to transfer. Three additional causes of persistent hypotension need to be considered: the coexistence of a myocardial contusion injury, high spinal cord injury with sympathetic paralysis, or coning associated with extreme intracranial hypertension. These may require the concurrent use of inotropes or vasopressors.

4.4.3 Treatment of seizures

Fits may be treated with phenytoin 15 mg/kg by slow iv bolus with blood pressure monitoring (at a rate not exceeding 50 mg/minute), or with intravenous diazepam.

4.4.4 Antimicrobial Prophylaxis

We use a short course of broad-spectrum antibiotic (e.g. cefuroxime) for patients with significant open injuries. Available evidence suggests that specific antimicrobial prophylaxis is ineffective and possibly harmful in the situation of a CSF leak or basal skull fracture; we do not therefore recommend specific prophylaxis in this situation. Check tetanus status.

4.5 Transfer

Both intra- and inter-hospital transfer can expose head injured patients to substantial risks, if not optimally managed. The distance travelled is not the main factor; hypoxaemia and hypercarbia can develop insidiously within a few minutes because of an obstructed airway or inadequate ventilation, and even transfer
### 4.5. TRANSFER

<table>
<thead>
<tr>
<th>Induction</th>
<th>Thiopentone 3–4 mg/kg or propofol 1–2 mg/kg&lt;br&gt;Etomidate up to 2 mg/kg in hypovolaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neuromuscular blockade</td>
<td>Succinylcholine 1.5 mg/kg for intubation&lt;br&gt;Atracurium, vecuronium or pancuronium for continuing paralysis</td>
</tr>
<tr>
<td>Maintenance of anaesthesia</td>
<td>Propofol 2–4 mg/kg/hr&lt;br&gt;Supplement with up to total fentanyl 5 µg/kg (avoid boluses &gt; 100 µg).&lt;br&gt;Midazolam 2–20 mg/hr instead of propofol if hypotension a problem.</td>
</tr>
<tr>
<td>Fits</td>
<td>Phenytoin 15 mg/kg slow iv bolus (&lt; 50 mg/min)&lt;br&gt;Diazepam 5–10 mg</td>
</tr>
<tr>
<td>Raised ICP</td>
<td>0.5–1.0 g/kg mannitol (CT, unequal pupils or focal deficit)</td>
</tr>
<tr>
<td>CPP maintenance</td>
<td>Ephedrine boluses (3 mg), Noradrenaline infusion</td>
</tr>
</tbody>
</table>

Table 4.7: Drugs and dosages

within a hospital needs to be taken seriously. Adequate resuscitation and the optimisation of systemic haemodynamics can substantially reduce such risks and allow the maintenance of physiological homeostasis during transfer. In our experience with moving ICP monitored patients, major reductions in CPP can occur with head movement despite seemingly adequate sedation. When the ICP is not monitored these will remain undetected, but can probably be minimised by small doses of iv anaesthetic before movement, as long as these do not compromise MAP. Undesirable changes in the patient’s homeostasis may go unnoticed or untreated because of inadequate monitoring, lack of equipment and drugs, or inexperienced escorts. The good work done in the resuscitation room is then undone during the journey, and the patient is no longer stable when presented to the Neurosurgical Team. Serious failures of communication can compound the problem and it is important for us to know if our protocols and admission procedures make it difficult to ensure a comprehensive handover.

#### 4.5.1 Timing

The decision that a patient has been rendered stable for transfer (Table 4.8 requires experience and follows a period of monitoring in the resuscitation room, which continues during the journey. This can legitimately delay the actual transfer, and it is helpful to telephone the Neurosurgical Unit as the journey starts to give the estimated time of arrival. Never start a journey with an unstable patient because of the high risk of complications during the journey.
4.5.2 Escort

As a bare minimum the severely head injured patient must be escorted by a doctor and a trained nurse or paramedic. They are professionally responsible for the patient until they hand over after reaching the Neurosurgical Unit. They must be well informed about the patient before the journey starts, and ideally should have been involved in the resuscitation room. They must know about what can go wrong during the journey and must have the skills and equipment needed to identify and deal with these problems. Anaesthetic skills are particularly valuable for airway care and for sophisticated monitoring during the journey. An anaesthetist (or at least a doctor with anaesthetic training and experience) must escort every intubated and ventilated patient.

Clinical judgement must be employed regarding the choice of medical staff who accompany the unintubated patient; if there appears to be a substantial risk that deterioration during transfer may result in a need for airway protection, the patient is best intubated before leaving the referring hospital. When the risk of deterioration is less, the presence of an appropriately equipped and skilled doctor during transfer will ensure airway protection should the patient deteriorate during transfer. If in doubt, we would recommend intubation prior to transfer.

4.5.3 Monitoring

It is unwise to rely solely on eyes and ears when monitoring a patient in the back of a noisy, dark and moving ambulance. During transfer continue the monitoring started in the resuscitation room, as described above. There must be reliable intravenous access. The electrocardiogram should be monitored continuously as should the blood pressure (as sphygmomanometry is unreliable in a noisy moving ambulance). Invasive blood pressure monitoring is ideal, failing this an automated NIBP machine is mandatory, but may be inaccurate due to artefact in the back of a fast moving ambulance. All lines must be secured, and arterial and central venous lines should be clearly labelled to prevent inadvertent drug injection through them. A pulse oximeter is mandatory. While capnography during transport represents the ideal, this is rarely available. An injured patient quickly becomes cold in an ambulance. It is not essential to correct mild hypothermia ($< 36.0^\circ$C) since this may contribute to cerebral protection. The ventilator itself should have a pressure dial to indicate inflation and a blow off valve to avoid barotrauma. There should be a system to deliver high flow oxygen to the patient, and two oxygen cylinders should be carried, both full and checked before use.

4.5.4 Other Equipment and Drugs

Even if the patient has been intubated before transfer it may be necessary to reposition or replace a tube which obstructs or falls out, and the ambulance must carry a range of endotracheal tubes and two working laryngoscopes. The oxygen supply can fail, and it is always advisable to carry a self-inflating bag (for example an ambu bag) to let ventilation of the patient continue. Intravenous lines inserted before transfer may block or fall out or may have to be supplemented for rapid fluid infusion during transfer. A range of cannulas (including wide bore)
4.5. TRANSFER

| Respiration: | Is the $P_aO_2 > 13$ kPa? |
|             | Is the $P_aCO_2 < 5$ kPa? |
|             | Is the airway adequately protected for the journey? |

| Circulation: | Is the systolic BP > 120 mmHg? |
|             | Is the pulse rate < 100/min? |
|             | Is the peripheral perfusion adequate? |
|             | Is there reliable and adequate venous access? |
|             | Has enough volume/blood been given to replace losses? |
|             | Is there suggestion of continuing bleeding? |
|             | Is the patient catheterised (essential if mannitol has been administered)? |

| Head injury: | What is the GCS score? |
|             | What is the trend in GCS score? |
|             | Is there any focal neurological deficit? |
|             | Is there a skull fracture? |

| Other injuries: | Has a cervical spine injury been excluded? |
|                | Have broken ribs/pneumothorax been excluded or dealt with (chest drain)? |
|                | Could there be an intrathoracic or abdominal bleed? |
|                | Are there pelvic or long bone fractures? |
|                | Have extracranial injuries been splinted (cervical collar, limb splints)? |

| Imaging: | If the patient is sufficiently stable, have you considered the need for CT of thorax/abdomen/cervical spine at the time of head scan? |
|          | Ensure notes and hard copies of imaging sent with patient |

Table 4.8: Checklist before transfer of head injured patients to Neurosurgical Unit

should be carried, as well as crystalloid (for example, Hartmann’s solution) for infusion. If blood has been cross matched before the journey it should travel in the ambulance, not in a separate taxi. The escorts should carry drugs for cardiac resuscitation, non-depolarising muscle relaxants and short acting analgesics and sedatives for controlling ventilation, anticonvulsants, and mannitol.

4.5.5 Ambulance and Trolley

Modern ambulances do not always offer a smooth, quiet ride but usually provide reasonable space and lighting. The problems of working in any moving vehicle make it vital to stabilise the patient and set up monitoring before transfer. The ambulance rarely needs to travel at great speed, which can worsen cardiovascular instability; a smooth ride at constant speed is safer. The patient should be placed head first in the ambulance, so as better to tolerate any sudden deceleration during the journey. A head up tilt of 10–15° should be maintained.
throughout the ambulance journey and when moving the patient through the hospital (including manoeuvres up and down ramps). If life saving procedures like endotracheal intubation have to be carried out along the way it is more sensible to stop the ambulance briefly than to attempt heroics under poor conditions, exposing the patient to the risks of failure or delay in carrying out the procedure. The patient should be moved between trolleys the minimum number of times. Ideally a single trolley should be used from the resuscitation room into the ambulance and out again at the Neurosurgical Unit.

4.5.6 Handover
The escorts must be able to give the Neurosurgical Staff an accurate description of all injuries, the trends in conscious level and neurological signs since injury or admission, and the drugs and intravenous fluids given. They should not leave until this has been done. The Neurosurgical Team may need further information later and must know who to contact. All medical and nursing clinical notes, observation charts, drug prescription sheets, and X-ray films and scans should be left with the Neurosurgical Team.

4.5.7 Summary of Transfer of the Ventilated Head Injury Patient
1. Ensure patient stable: consider items on transfer checklist (Table 4.8).
2. Neurosurgical Registrar “on call” knows expected time of arrival.
3. Adequate escort: Airway competent doctor (usually Anaesthetist) + appropriately trained paramedic/nurse.
4. Minimise number of trolley transfers, 15° head up tilt.
5. Boluses of iv anaesthetic (eg propofol 30–50mg) before manipulation or movement if ICP high.
6. Mannitol 0.5–1.0 g/kg if raised ICP on CT or pupillary inequality.
7. Adequate monitoring: ECG, $S_{o2}$, invasive BP (or automatic NIBP), airway pressure (? capnograph)
8. ABG and Hb prior to transfer. Catheterise before transfer. Always catheterise if mannitol given.
9. Spare intubation equipment, check oxygen cylinders, self-inflating resuscitation bag. Emergency drugs, iv fluids, iv access equipment

4.5.8 Further reading


4.6 NICU Management of TBI

4.6.1 On admission

1. Ensure patient is adequately resuscitated:
   A  Airway secured? Is cervical spine protected or cleared?
   B  Breathing and ventilation adequate?
   C  Circulation. Is volume resuscitation complete? Is the patient still bleeding?

2. Clerk the patient in:
   - History of injury.
   - Presenting GCS, post-resuscitation GCS, pupillary and any lateralising signs.
   - CT findings.
   - Other injuries.
   - Past medical history.
   - Management prior to arrival on NICU. Has tetanus booster been given?

3. Examination:
   - Ensure all other injuries assessed and prioritised.
   - Is referral to other specialties indicated?
   - Are other imaging studies indicated (CT chest and abdomen, ECHO, arch aortogram)?

4. Baseline investigations:
   - FBC, clotting screen.
   - U&Es, LFTs, CRP, glucose.
   - Arterial blood gases.
   - CXR (+ other X-rays or CT as indicated).
   - ECG.
   - Cardiac enzymes (if chest trauma).
   - Group and save.

4.6.2 Monitoring

1. ECG.

2. Pulse oximetry.

3. End tidal CO₂.
4. Invasive arterial pressure monitoring. Perform Allen’s test. Preference in
this order: nondominant radial artery (RA) > dominant RA > dorsalis
pedis > brachial > femoral > axillary.

5. Urinary catheter: hourly urine output and fluid balance recording.

6. Core and peripheral temperature.

7. Triple lumen line for CVP measurement. Preferred site is subclavian vein
(on side of chest drain if present). Avoid internal jugular puncture unless
essential. (Try external jugular first if clotting abnormal).

8. ICP monitoring.

9. (Jugular bulb oximetry (retrograde RIJ).)

10. (MCA velocities (transcranial Doppler).)

4.6.3 Additional monitoring

1. LiDCO or PA catheter if moderate to severe lung injury, cardiac contusion
or cardiac disease.

2. Cerebral function monitor for suspected fits or need to achieve burst sup-
pression.

4.6.4 Daily Investigations

1. ABGs as indicated.

2. FBC, clotting screen.

3. U&Es, LFTs, glucose, CRP.

4. CXR if chest problems.

5. Sputum cultures or non-directed bronchoalveolar lavage, MSU C&S.

6. Plasma and urine osmolality if possible DI or multiple mannitol doses.

7. Consider the need for drug levels (eg digoxin, phenytoin or antibiotics),
Mg, TFTs, Short Synacthen test or lipid screen if on high dose propofol.

4.6.5 Summary of Initial Management Goals

1. Ensure well sedated and analgesed (propofol and fentanyl if plan is to wake
up within 24 hours, midazolam and morphine if likely to require sedation
for longer than 24 hours). See section 4.6.6.

2. SIMV: minimal PEEP ($\leq 5$ cm H$_2$O), $F_{1\text{O}_2} \leq 0.5$; Target $P_aO_2 \geq 11$ kPa
(or $SpO_2 \geq 95\%$); Target $P_aCO_2 = 4.0$–4.5 kPa (avoid $P_aCO_2 < 3.8$ kPa).
See section 4.6.7.

3. ICP $\leq 25$ mmHg; CPP $\geq 70$ mmHg. See section 4.6.8 and ICP manage-
ment algorithm in section 4.7
4. Core temperature 35–37°C (treat \( \uparrow \) temperature vigorously).

5. Optimal haemodynamic and volume status (define in terms of target MAP, CVP, HR etc. for each patient).

6. Tight control of seizures. Prophylactic phenytoin if significant head injury on CT, patient sedated and / or paralysed.

7. Early enteral feeding (within 24 hrs). Ranitidine.

8. Tight blood sugar control (5–7 mmol/l, start insulin sliding scale if BM >7)

9. Conservative antibiotic policy, but treat established or clinically significant infection vigorously.

10. No routine line changes.

11. (Maintain SjO\(_2\) \( \geq \) 55% by increasing \( P_aCO_2 \) and elevating CPP).

### 4.6.6 Sedation and Analgesia

The choice of drugs used for sedation and analgesia lies mainly between the combination of Propofol and Fentanyl, if relatively rapid reversal is likely to be useful, or Midazolam and Morphine, where longer term ventilation is anticipated.

**Propofol and fentanyl:** *If rapid reversal of sedation required (weaning, brain death criteria, neurological reassessment etc.), or only overnight sedation anticipated.* Propofol 15–30ml/hr of 1\% in average sized male, fentanyl 1–4 ml/hr (50 \( \mu \)g/ml). *Check plasma lipids and acid base status regularly on high doses of propofol.*

**Midazolam and morphine:** *If likely to remain sedated for > 24 hours.* Midazolam 10–20ml/hr in average sized male, morphine 2–10mg/hr.

### 4.6.7 Ventilation

**Initial settings**

SIMV; \( V_T \): 6–10 ml/kg (if high plateau or peak airway pressures consider decreasing); rate — appropriate to age (12–16 breaths/min in adult); \( FIO2 \) 0.3–0.4; I:E ratio 1:2.

Use rate to control \( P_aCO_2 \).

Use \( FIO2 \) or PEEP to control \( P_aO2 \).
Hypoxia

If $P_{a}O_2 \leq 10$ kPa despite $F_{i}O_2 \gg 0.5$ and PEEP $\geq 5$ cm H$_2$O:

- ensure adequate humidification and physiotherapy.
- obtain new CXR for to look for complication and check position of ET tube.
- and consider:
  - treatment of bronchospasm.
  - fibreoptic bronchoscopy if segmental or lobar collapse.
  - drainage of marginal pneumothorax or haemothorax.
  - ↓ lung water by ↓ CVP or PCWP (diuretics, inotropes etc.).
  - look for infection and treat vigorously.
  - consider pressure control ventilation.

4.6.8 ICP/CPP Control

See also ICP management algorithm in section 4.7. Maintain MAP to achieve target CPP with adequate hydration and avoid excessive sedation. Switch to midazolam if propofol causing hypotension.

Maintain ICP $< 25$ mmHg. Nurse $30^\circ$ head up.

If ICP $> 25$ mmHg

- ensure optimal head and neck positioning.
- ensure no venous constriction (collar, ET tube tapes).
- ensure optimal sedation, analgesia and neuromuscular blockade.
- ensure optimal blood gases.
- treat any pyrexia vigorously.
- check for inadvertent PEEP or ↑ venous pressures.
- Is seizure activity the cause? Consider phenytoin or other anticonvulsant.
- CONSIDER REPEAT CT: Is there a surgical lesion? Will an EVD help?
If no surgically treatable cause consider:

- further reduction in $P_aCO_2$ (if $> 4.0 \text{kPa}$).

- bolus of $2 \text{ml/kg}$ 20% mannitol up to plasma osmolality of $320 \text{mosm/l}$ ($\times 3$ doses blind).

- reduction in core temperature to $34^\circ\text{C}$ (use cooling line). *Stop propofol and change to midazolam.*

- CSF drainage.

- surgical decompression (may include removing a surgical mass lesion or bone flap, frontal or temporal lobectomy, bifrontal or unilateral craniectomy).

- bolus (1 mg/kg) of lidocaine before suction or physiotherapy if this provokes ↑ ICP (1% lidocaine contains 10 mg/ml).

- thiopentone bolus followed by infusion — see section 4.6.16

Maintain CPP $\geq 70 \text{mmHg}$

- Optimal volume status (Hct 30–35%, CVP 5–10 cm H$_2$O).

- Vasoactive agents: noradrenaline (1–4 µg/kg/min).

- LiDCO or PA catheter, and ST segment monitoring if noradrenaline dose $> 4 \mu\text{g/kg/min}$, age $> 50$ or cardiac disease. Default targets: $\text{CI} \simeq 3.5$–4.0 L/min/m$^2$; $\text{SVRI} 1400$–2000 dynes/sec/cm$^{-5}$.

4.6.9 Neuromuscular blockade

- Maintain 1–2 post-tetanic twitches.

- Atracurium 0.5–1 mg/kg/hr.

- *No long-term vecuronium infusions.*

4.6.10 Temperature Control: 35–36°C

If ↑ temperature:

- Paracetamol 1 g qds NG/PR.

- Fan or cooling blanket.

- Cold sponging.

- Cooling line.
4.6. NICU MANAGEMENT OF TBI

4.6.11 Optimal Haemodynamic Control
If otherwise well use hydration and feeding to achieve:

- MAP ≃ 90 mmHg (CPP ≥ 70 mmHg).
- CVP 5–10 cm H2O.
- HR 50–100/min (if outside range, look for cause and consider treatment).
- Urine output ≃ 0.5–1 ml/kg/hr.

Consider LiDCO or PA catheter if:

- Hypotension.
- Oliguria.
- Systemic sepsis.
- Pulmonary oedema.
- Cardiac contusion.
- Acute or pre-existing cardiac disease.
- Inotrope therapy, especially if high doses, cardiac disease or age > 50.

Volume: 2.5–3.0 L per day, unless otherwise indicated

- Maintain hydration with early enteral feeding.
- Supplement with iv 0.9% NaCl + KCl if enteral feeding not established.
- Treat low filling pressures with RBCs, FFP or gelofusin to maintain:
  - Hct 30–35% (Hb 10–11 g/dl).
  - Normal or near normal clotting, especially postoperatively.

4.6.12 Tight Seizure Control
Prophylactic treatment should be given if there is a compound skull fracture. A patient known to have epilepsy should continue on their current medication. If a patient fits whilst on phenytoin then check levels and ensure an adequate dose has been given. An alternative or secondary agent can be given if witnessed fits or EEG evidence of seizure activity.

- Load with iv Phenytoin 15 mg/kg (at ≤ 50 mg/min).
- Maintenance 3–4 mg/kg per day. If given NG, feed should be off for 2 hrs before and after dose to facilitate absorption. Liaise with SHO to ensure correct timing.
- If persistent fits consider: valproate, carbamazepine, lorazepam, clonazepam, and chlormethiazole.
- If seizures still uncontrolled: thiopentone.
4.6.13 **Early Enteral Feeding**

*High dose thiamine IV should be given on admission if there is any suspicion of chronic alcohol abuse.*

Enteral feeding should be started within 24 hours of admission if there is no contraindication. Build up to 100 ml/hr for 20 / 24 hrs. A 4–6 hr rest at night will allow acidification. In addition consider the following options:

- high fibre feed for diarrhoea.
- low sodium feed for hypernatraemia.
- IV or enteral supplementation of:
  - thiamine.
  - vitamins, minerals and trace elements.
  - potassium.
  - magnesium.
  - phosphate.

**If failure to absorb consider:**

- metoclopramide 10 mg tds IV.
- erythromycin 125 mg qds NG.

**If diarrhoea consider:**

- feed intolerance.
- *Clostridium difficile* (x 3 stool for toxin).

*If enteral feeding not established by 48–72 hours consider nasojejunal feeding tube or TPN.*

4.6.14 **Tight Blood Sugar Control**

Monitor blood sugar with at least 4 hourly BM stix on admission, and continue if blood sugar high. If well controlled, reduce frequency. Use IV insulin sliding scale to maintain blood sugar 5–7 mmol/l.

4.6.15 **Infection Control**

- No white coats.
- Individualised stethoscopes.
- Hand washing, alcohol gel.
- Gowns for central line insertion.
- No blind antibiotics unless rising CRP, WBC or pyrexia with:
  - worsening organ function (eg worsening gas exchange).
4.6. NICU MANAGEMENT OF TBI

- systemic sepsis.

- Close liaison with microbiology.

- *No routine central line changes. Change line only if pyrexial with no clear cause, puncture site infected, or line blocked.*

4.6.16 Use of thiopentone for control of raised ICP

**Indications**

Thiopentone should only be used to control ICP when other medical and surgical measures have failed. It should be started only after discussion with the consultant in charge to ensure that other treatment options have been exhausted. Check that the following are already in place:

1. Full sedation and analgesia.
2. Paralysis.
3. Cooling to 34–35°C.
4. $P_{CO_2}$ 4–4.5 kPa.
5. Head up 30° and no venous obstruction from collar or ET tapes.
6. No surgically amenable lesion (EVD or decompression).

**Action**

Sodium thiopental is a potent cerebral vasoconstrictor and reduces cerebral blood flow, cerebral blood volume and ICP. The primary mechanism of protection is a reduction in the cerebral metabolic requirement for oxygen of <60%. It may also act as a GABA agonist and free radical scavenger, and cause membrane stabilisation, NDMA antagonism and calcium channel blockade. Although an isoelectric EEG (flatline trace) can easily be produced, this does not allow titration of the thiopentone dose. Therefore, the amount of thiopentone is adjusted to produce burst suppression (long periods of electrical silence on the EEG interspersed with short periods of some electrical activity).

Thiopentone can cause an inverse steal phenomenon whereby vasoconstriction in normal tissue improves perfusion of ischaemic areas that are unable to vasoconstrict.

**Administration**

1. A bolus of IV anaesthetic agent (eg propofol 5–10 ml of 1%, or thiopentone 2–3 mg/kg) should be tried to see if there is a beneficial effect on ICP. ICP should fall by >10 mmHg. Cerebral perfusion pressure must be maintained using fluids and vasoactive agents.

2. If there is a beneficial effect, the cerebral function monitor should be applied to the patient. If there is no beneficial effect, thiopentone coma should not be attempted.
3. Small boluses of thiopentone (250 mg) should be administered whilst watching the EEG monitor and systolic blood pressure. Owing to the distribution characteristics of thiopentone, 3–5 g over a period of 30 minutes or more may be needed to get sustained burst suppression.

4. Once sustained burst suppression is achieved, an infusion of thiopentone should be started (10–30 ml/hr of 25 mg/ml). This should be titrated according to the EEG — if no electrical activity is seen on the EEG, the infusion should be stopped. Once activity returns, the infusion should be restarted at half the previous rate and the rate increased by 2–5 ml/hr every 30 minutes until burst suppression is achieved.

5. The lowest possible infusion rate to achieve burst suppression should be used. The pharmacokinetics of thiopentone are such that once hepatic enzyme activity is saturated, thiopentone undergoes zero order kinetics with plasma levels rising rapidly.

6. CPP should be maintained throughout by the use of fluids and vasopressors.

7. Once the patient is burst suppressed, other measures to reduce ICP may be slowly withdrawn — monitor the EEG and increase the thiopentone infusion rate if necessary. In order, the other sedation and analgesia should be withdrawn, followed by the paralysis, followed by rewarming.

Withdrawal of thiopentone

1. The normal terminal half-life of thiopentone is about 12 hours. After 5 half-lives it is effectively removed from plasma. If the patient has received large amounts of thiopentone over a prolonged period, zero-order kinetics are encountered initially and the effects will be further prolonged.

2. On withdrawal of thiopentone, it may be necessary to initiate other measures to control ICP — these should be used in a step-wise manner as normally with sedation and analgesia initially.

Unsuccessful therapy

1. Brainstem tests cannot be carried out in the presence of sedative agents. Because of the prolonged half-life of thiopentone and its metabolism to the active drug pentobarbitone, plasma levels of both must be measured before brainstem tests can be performed.

2. There are no absolute values as to when brainstem tests can be done; the biochemistry department will advise when levels are sufficiently low as to be ignored.

3. Brainstem tests are only required if the patient is a candidate for organ donation. The decision to cease supportive therapy in other patients is difficult, especially in the presence of thiopentone, and expert advice should always be sought.
Complications of thiopentone coma

1. Hypotension — correct with fluid, inotropes and vasopressors as necessary.


3. Rhabdomyolysis can complicate thiopentone, propofol and phenytoin administration. Usually occurs in conjunction with high vasopressor use and relative hypotension. Need to stop causative agent and aggressively rehydrate.

4. Hypokalaemia — at very high doses, thiopentone can cause a relative hypokalaemia, probably due to ion pump inhibition. On stopping the infusion, rebound increases in K\(^+\) can occur.

5. Polyuria — thiopentone contains a significant sodium load. Administration commonly results in polyuria with appropriately high sodium content. Fluid replacement should be with 0.9% saline (occasionally 0.45% saline). Do not confuse this with diabetes insipidus.
4.7 ICP / CPP Management Algorithm

- All patients at risk of intracranial hypertension must have arterial monitoring, CVP line and ICP monitor on admission to NICU.
- Check whether patient is in, or may be a candidate for, research protocols.
- Treatment levels 3 and 4 should only be initiated with express approval of consultant in charge.

**Level 1**

- 10-30° head up, no venous obstruction, check collar and ET tape.
- CPP ≥ 70, CVP 5–10.
- $S_pO_2 \geq 97\%; P_aO_2 \geq 11\,kPa$; $P_aCO_2 \leq 4.5\,kPa$
- Temp $\leq 37^\circ C$.
- Propofol 2–4 mg/kg/hr and fentanyl 1–4 µg/kg/hr or midazolam 10–20 mg/hr and morphine 2–10 mg/hr.
- Phenytoin 15 mg/kg if indicated (fits, depressed #).

**Level 2**

- Volume replacement, noradrenaline to increase MAP (CPP > 70). Consider LiDCO.
- Switch Propofol to midazolam.
- Atracurium 0.5 mg/kg/hr (ensure 1–2 post-tetanic twitches).
- Reduce $P_aCO_2$ to 4.0 kPa.
- Ensure temp 35-36°.

**Level 3**

- Reduce temp to 34°C.
- (ensure on midazolam).

**Level 4**

- Trial of bolus IV anaesthetic (eg Propofol 50-200 mg), maintain CPP with fluids and vasoactive agents.
- If favourable effect on ICP and CPP start thiopentone. 250 mg boluses up to 3–5 g, with infusion 4–8 mg/kg/hr to achieve and maintain burst suppression (needs CFM).
4.7. ICP / CPP MANAGEMENT ALGORITHM

Patient at risk of raised ICP

A-line, CVP line, ICP monitor

Yes

ICP < 20
CPP > 70

Level 1 treatment

Level 2 treatment

No

Recent CT
Low risk of surgical lesion

Surgical Mass Lesion

CT scan

No recent CT

EVD ?
Surgical lesion ?

Yes

Surgery / EVD

Failure
ICP > 25, CPP < 60

Repeat CT ?
Check probe
Decompressive surgery ?
EVD ?

No

Level 3 treatment

Failure
ICP > 25, CPP < 60

Level 4 treatment

Failure
ICP > 25, CPP < 60

Figure 4.1: NICU ICP management algorithm.
4.8 Spinal Clearance Protocol

4.8.1 To exclude bony cervical injury

In a conscious patient, a full cervical spine series is required which will include a lateral view, AP and AP view of the peg. If clinically appropriate a swimmer’s view may be added to demonstrate the cervico-thoracic junction.

In the unconscious, uncooperative or intubated patient a lateral film of the cervical spine is supplemented with 2 mm contiguous sections covering the foramen magnum to C2/3 obtained at the time of the head scan. If C7/T1 has not been seen on the lateral film, further scanning of this region should be performed in spiral mode with sagittal reformats. Further CT imaging of the cervical spine will be determined on the results of these investigations. These should be acquired at the time of the initial CT assessment. Alternatively, spiral CT of the entire cervical spine may be performed.

The films must be reported by a Radiologist.

4.8.2 Ligamentous cervical injury

A normal bony investigation will not exclude ligamentous injury. A hard collar must remain on and the neck adequately immobilised until symptoms can be assessed and the neck examined clinically. If this is not possible and it becomes imperative to exclude a ligamentous injury, MRI of the cervical spine is indicated. Discuss with a Consultant Radiologist.

Flexion extension views are only considered safe in a conscious and cooperative patient and should only be carried out following discussion with the Consultant who has clinical responsibility for the patient and a Consultant Radiologist.

4.8.3 To exclude lumbar and thoracic spine injury

AP and lateral films of thoracic and lumbar spine should be performed on all multiple trauma patients and reported by a radiologist. These X-rays are mandatory if:

1. Pedestrian RTA.
2. Passenger in vehicle if another occupant killed.
3. High speed impact.
4. Fall > 2 m.
5. Clinical suspicion e.g. unexplained hypotension, priapism, vasodilation of lower limbs.
# ICU SPINAL CLEARANCE CHECKLIST

Name: 

Hospital No: 

<table>
<thead>
<tr>
<th>Type of investigation</th>
<th>Date performed</th>
<th>Date reported by radiologist</th>
<th>Was it cleared Y/N</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervical Spine (C1 – C7/T1 junction)</td>
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<tr>
<td>Lateral view</td>
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<tr>
<td>CT scan</td>
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<tr>
<td>Other</td>
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<tr>
<td>Thoraco-Lumbar Spine</td>
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<tr>
<td>Lateral view</td>
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<tr>
<td>AP view</td>
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<tr>
<td>CT scan (if needed)</td>
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<tr>
<td>Other</td>
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<tr>
<td>Ligaments</td>
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<tr>
<td>Clinical examination</td>
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<tr>
<td>MR scan (if needed)</td>
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</tbody>
</table>

**Notes:** This chart is for information only. Please check documentation in the patient’s notes.
4.9 Management of Spinal Injury

4.9.1 History

On admission / transfer, ensure that these points are made clear by the referring and /or transferring team:

2. Complete / incomplete / absent spinal cord injury at referring hospital.
3. Methylprednisolone given / not given. If spinal cord injury present but methylprednisolone not given, check with duty consultant. The window of clinical effectiveness is 8 hours. However see 4.9.12.
4. Other injuries.

4.9.2 Examination

1. ABCD
   A Airway and Cervical spine immobilisation.
   B Adequacy of ventilation.
   C Fluid resuscitation (presume hypotension secondary to blood loss, not spinal shock).
   D GCS, pupil size.
2. Associated injuries including pressure areas.
3. Level of spinal injury
   • radiologically
   • clinically
   • complete / incomplete / absent

4.9.3 Investigations

• Routine bloods.
• CXR.
• Spinal imaging (see protocol).
  – Is further imaging needed at the level of injury?
  – Has the entire spine been adequately imaged (10-15% have another injury at another level)?
• ABGs.
• Non-ventilated patients require regular measurement of vital capacity.
4.9. MANAGEMENT OF SPINAL INJURY

4.9.4 Monitoring

- Pulse oximetry.
- ECG.
- NIBP / IABP.
- Twice daily VC in non-ventilated patients.
- Urinary catheter.

4.9.5 Management

1. **Oxygen.** Most patients with lesions below C4 are able to make sufficient respiratory effort to avoid ventilation, but do need supplemental humidified oxygen.

2. **Analgesia.**
   - Paracetamol 1 g 6 hourly.
   - DF118 6 hourly.
   - ± Voltarol < 150 mg/d.
   - Careful titration of IV opiates (some patients may be able to operate a PCA).

3. **Ventilation.**
   - Look for signs of fatigue.
   - BD Vital capacity.
   - Tetraparetic patients find it easier to breathe when supine.
   - Consider elective ventilation in high cervical injuries with RR > 30 /min, worsening ABGs, vital capacity < 1200 ml.
   - Where longer term ventilation is likely to be required, consider early placement of tracheostomy (beware of spinal instability if performing percutaneous placement).
   - Suxamethonium can be used up to 48 hours post-injury, **but after that time may cause a sudden onset of profound hyperkalaemia which may result in cardiac arrest.**
   - When weaning patients with ventilatory failure (with normal lung physiology) consider using tidal volumes greater than 6 ml/kg (up to 12 ml/kg may be required). BIPAP via an uncuffed tracheostomy tube is often used medium term. Application of expiratory positive airway pressure (EPAP) reduces risk of aspiration and allows phonation.
4.9.6 Haemodynamic management

- Spinal shock may produce hypotension, bradycardia and poikilothermia. This results especially from injuries above T6 (base level of sympathetic outflow).

- Presume hypotension due to blood loss until proven otherwise (consider placing CVP line).

- Increased vagal activity may cause cardiac syncope (often triggered by airway manipulation such as intubation or changing tracheostomy). Responds to $\text{O}_2$ and atropine.

4.9.7 GI management

- Paralytic ileus is common with spinal cord injury. Patients are commonly kept NBM for first 48 hours.

- Thereafter, establish early enteral feeding ± prokinetics.

- H2 blockers to prevent stress ulceration.

- Where nasogastric feeding has failed, consider placement of nasojejunal tube or tiding the patient over with TPN until enteral feeding can be established.

- If prolonged nasogastric feeding is likely to be required, consider early placement of a PEG.

- Early commencement of bowel care.

4.9.8 DVT prophylaxis

- Flowtron boots.

- S/C heparin 5000 iu tds, or enoxaparin 40 mg/d, dalteparin 5,000 iu od (if risk of intraspinal haemorrhage is low).

4.9.9 Pressure areas and contractures

- Need suitable pressure-relieving mattress.

- Two hourly turns.

- Minimise hypotension, hypoxia, oedema.

- Ensure adequate nutrition.

- Orthopaedic casts should be removed regularly to prevent development of plaster sores.

- Joint contractures develop rapidly following spinal cord injury. Liaise with the physiotherapists regarding passive movements and splinting.
4.9. MANAGEMENT OF SPINAL INJURY

4.9.10 Immobilisation

• Patients should be removed from spinal boards as soon as possible.
• A hard cervical collar and a firm mattress are the standard means of immobilisation before the application of traction or definitive stabilisation.
• Cervical collars
  – Two-piece collars such as the Philadelphia or Aspen models are used for longer-term use as they are more comfortable and cause less skin problems. Soft collars have no role.
  – Once immobilised, the decision to remove protection should only be made after appropriate investigations have been completed and reported. *The decision to remove immobilisation must be clearly documented in the notes, signed and dated.*
  – Collars may be removed or loosened in cases of raised ICP. The decision should be clearly documented in the notes.
• Patients being transferred for short periods such as for imaging may be moved on a spinal board for convenience. Ideally a pressure-relieving gel mattress should be placed between the patient and the board.
  – Most spinal boards and scoop stretchers cause minimal artefact for CT and MRI.
  – Modern scoop stretchers are non-magnetic and can be used to transfer patients on to MRI tables.
  – Seven staff are necessary if a sliding board transfer is required. Four are required to perform a log-roll with a fifth to position the board.
  – Skull callipers, unless MRI compatible, will have to be removed and a hard cervical collar should be in place before traction is released.
• It is inappropriate for patients to be left on spinal boards for prolonged periods (e.g. operative procedures or for hospital transfer).

4.9.11 Autonomic dysreflexia

Autonomic dysreflexia occurs in response to painful stimuli perceived below the level of the lesion. Causes include:

1. Bladder distension. *Commonest cause is blocked catheter.* Latex catheters often get blocked and should be changed weekly. Consider suprapubic catheter placement.
2. Bowel distension; manual evacuation may be required on alternate days.
3. Fracture below level of lesion.
4. Pressure sore.
5. UTI / bladder spasm.
6. DVT.
7. Surgery on area below level of spinal injury.
CHAPTER 4. CRANIOSPINAL TRAUMA

Symptoms
1. Hypertension. *May be severe.*
2. Bradycardia.
3. Headache.
4. Sweating above level of lesion.
5. Pallor below level of lesion.
6. Flushing / blotchiness above level of lesion.
8. Bronchospasm, dyspnoca.

Treatment
1. Remove cause.
2. Analgesia (IV opiates).
3. Local anaesthetic blockade.
4. Vasodilators (clonidine, GTN, nifedipine etc.).

4.9.12 Steroid Treatment in Acute Spinal Cord Injury
There is ongoing debate regarding the efficacy of steroids in acute spinal cord injury. The consensus opinion from the Wessex Neurological Centre is that steroids should *not* be given.

4.9.13 Further reading
5.1 Introduction

Patients with aneurysmal subarachnoid haemorrhage may deteriorate rapidly due to a number of potentially reversible causes, including:

Rebleeding, which is usually only a problem in unsecured aneurysms.

Hydrocephalus, which can be treated by placement of an external ventricular drain.

Vasospasm, causing cerebral ischaemia. This may present as neurological deficit in the vascular territory of the treated aneurysm’s parent vessel, or a completely different and distant vascular territory.

Intra-arterial thrombosis or embolism, which we think is more commonly seen after coiling, especially if a coil has prolapsed into the parent vessel. This may require Reopro, aspirin, heparin or clopidogrel, either singly or in combination.

Neurogenic pulmonary oedema and cardiac stunning may cause low cardiac output, hypoxia and further compromise oxygen delivery to a brain under pressure.
Seizures may also occur, more commonly with middle cerebral artery aneurysms. Be aware, however, of re-bleeding and A-waves of raised ICP masquerading as seizures.

Electrolyte disturbances may occur due to cerebral salt wasting syndrome, or less commonly SIADH, or a mixture of both.

5.2 Management Protocol

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

5.2.1 On admission

1. Clerk the patient (noting the success or otherwise of the procedure, complications, previously noted neurological deficit, time of SAH).

2. Full examination, including groin site if coiled.

3. Routine post-operative bloods, ± ECG, ± CXR, ± ABGs.

4. Patients should be nursed flat for at least 24 hrs.

5.2.2 Monitoring

- Pulse oximetry.
- ECG.
- NIBP or IABP.
- ± CVP.
- Urinary catheter.
- Daily U&E.
- Transcranial Doppler (Mon/Wed/Fri) — liaise with medical physics (bleep 1250).

5.2.3 Fluid management

1. Three litres per day of 0.9% saline ± KCl.

2. Maintain MAP 80–90mmHg (CVP 5–10mmHg) with colloid (gelofusin / haesteril / dextran).

3. Establish early enteral feeding (NG feeding should stop at 06.00 if extubation is planned).
5.2. MANAGEMENT PROTOCOL

5.2.4 Nimodipine

1. Continue 60 mg 4-hourly PO/NG (for a total of 3 weeks).
2. If MAP falls, try 30 mg 2-hourly.
3. Do not use IV unless discussed at consultant level (as it is associated with hypotension).

5.2.5 Avoid

- Prolonged hypotension (MAP ≤ 70 mmHg).
- Prolonged hypoxia (\(S_\text{a}O_2 < 97\%\), \(P_\text{a}O_2 < 10\) kPa).
- Prolonged hypocapnia (\(P_\text{a}CO_2 < 4\) kPa).
- Fluid overload.

5.2.6 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 100% oxygen.
2. Discuss with neurosurgical SpR the need for CT (to exclude a further bleed, hydrocephalus etc).
3. If vasospasm thought to be the cause, institute Triple H protocol.

5.2.7 Triple H protocol

This three-pronged attack on cerebral ischaemia consists of hypervolaemia, hypotension, haemodilution.

1. Make a presumptive diagnosis of vasospasm from the clinical picture, CT and TCD.
2. Continue maintenance fluids. Replace IV fluids with enteral feeding if possible.
3. Ensure patient has arterial and CVP lines in situ.

4. Hypervolaemia.

- Volume expansion with gelofusin (250 ml every 6 hours).
- Aim to maximise cardiac output (CVP 10–15 mmHg).
- If volume expansion not maintained:
  - Consider switching from gelofusin to haesteril.
  - Enteral fludrocortisone (0.1–0.2 mg/day) if Na normal or low.
  - If high the urine output is high, with a high plasma Na, consider diabetes insipidus (check osmolarities, treat with DDAVP 0.5–1 µg IV).
5. **Hypertension.**

- Aim for MAP 110–120 mmHg (may be taken higher if aneurysm secured).
- If volume loading insufficient to maintain MAP, start noradrenaline infusion.
- Consider LiDCO / PA catheter and ST segment monitoring if ↑ NA requirement, cardiac disease, age > 50 yrs, or pulmonary oedema.

6. **Haemodilution**

- Aim to keep haematocrit 30–35%.
- If haematocrit remains above 35%, consider venesection of 500 ml and replacement of volume with colloid.

Triple H therapy is generally continued for a minimum of 5 days and should be weaned off in a stepwise manner.

### 5.3 Deterioration Following Coiling

Acute neurological deterioration following coiling of a cerebral aneurysm will usually be due to thrombo-embolism related to the coils. This can often be treated effectively by the prompt administration of Reopro (Abciximab). Reopro is a potent anti-platelet antagonist which acts on platelet aggregation, the cause of coil related thrombo-embolism. Rarely, acute neurological deterioration is due to re-haemorrhage despite an apparently successful coiling procedure. 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 they are an Interventional Neuroradiologist JM, SB, AD).

3. Obtain Reopro (10 mg vial for reconstitution) from the fridge in NICU and have this ready in the CT scan room to be drawn up and administered by slow (2 minutes) intravenous injection if the scan reveals no evidence of new haemorrhage.

4. 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).

5. Discuss subsequent Heparinisation and/or Aspirin therapy with the Interventional Neuroradiologist.

6. 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.

7. Continue close observation of femoral artery puncture sites, arterial lines, ventricular access devices etc., following administration of Reopro.
Chapter 6

Fluid Management

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Date: February 2006
Review Date: February 2007

6.1 Enteral Feeding

1. Aim to start enteral feeding as soon as possible to provide appropriate caloric and fluid intake. Enteral feeding also promotes the integrity of the GI mucosa.

2. If gastric aspirates are high, start prokinetic drugs — metoclopramide (10 mg tds IV/NG) ± erythromycin (125 mg qds NG).

3. Patients may require separate supplementation of Na\(^+\) and K\(^+\).

4. Stop enteral feed for 2 hrs before and after administration of enteral phenytoin.

5. If possible, start 4 hr rest period at 6am.

6. A rest period is not indicated if feed has been stopped for any significant period of time in previous 24 hrs, or if the patient is not achieving full calorie intake.

7. If gastric aspirates remain high despite prokinetic agents, the gastroenterology team should be contacted for consideration of NJ tube placement.
6.2 Intravenous Fluid Therapy

1. Check to ensure that renal function is normal.

2. If renal function is normal, prescribe 0.9% saline 1000 ml with KCl 3 g (40 mmol) 8 hourly.

3. Liaise with nursing staff to slow rate of IV fluids as enteral feeding becomes established.

6.3 Hypernatraemia

The commonest causes of hypernatraemia on NICU 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 elevations hypernatraemia 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 is not recommended for this reason.

6.3.1 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 in any intracranial condition.

- Diagnosis suggested by urine output > 200 ml/hr (3 ml/kg/hr in children) for ≥ 2 hrs (with urine specific gravity < 1.005) and serum sodium > 145 mmol/l. Plasma volume state will be reduced. Check fluid balance chart.

- 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.

- Treatment: DDAVP 0.5–1 µg IV repeated PRN.

- Any patient with urine output > 200 ml/hr for ≥ 2 hrs should have ABGs 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, careful IV titration of hypotonic saline (0.45%) may be necessary to slowly correct serum sodium (by < 3 mmol/hr).

- Never give DDAVP and hypotonic IV fluids concurrently.
6.4. HYTONATRAEMIA

- 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.

6.3.2 Hypernatraemia due to administration of hypertonic fluids

This is not usually associated with volume depletion. Correction should be done by slow administration of isotonic saline.

6.4 Hyponatraemia

Hyponatraemia in NICU 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.

6.4.1 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 depends on:
  1. urine osmolality > serum osmolality.
  2. urinary Na\(^+\) > 20 mmol/l.
  3. serum Na\(^+\) < 130 mmol/l.
  4. normal renal, hepatic, cardiac, pituitary, adrenal and thyroid function.
  5. absence of hypovolaemia, hypotension, oedema, drugs affecting ADH secretion.
- Management consists of fluid restriction (800–1000 ml/day). Use caution as patients may become intravascularly depleted. In severe cases demeclocycline, which impairs action of ADH on the kidney, can be used.
- If Na\(^+\) < 120 mmol/l and patient symptomatic, hypertonic saline (1.8%) can be used cautiously aiming to increase Na\(^+\) by 2 mmol/l every 4 hours. More rapid correction risks central pontine myelinolysis. Hypertonic saline should be given centrally.
- In SIADH associated with volume expansion, giving hypertonic saline alone is ineffective as the excess sodium is excreted. In this situation, frusemide should be given with the hypertonic saline to produce a hypotonic urine.
6.4.2 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 by:
  
  1. presence of hypovolaemia, hypotension.
  
  2. absence of aldosterone deficiency, diuretics, renal tubular damage or Bartter’s syndrome.

- Management is by replacement with isotonic or hypertonic saline depending on severity (see comments above).
Chapter 7

Deep Vein Thrombosis Prophylaxis

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Date: February 2006
Review Date: February 2007

7.1 Patient Characteristics

These instructions cover all patients over 10 years of age admitted to the Neurosurgical wards, Wessex Neurological Centre.

7.2 Risk Factors

Deep vein thrombosis (DVT) is caused by reduction in deep venous blood flow, changes in blood composition, trauma to walls of deep veins, or more often to a combination of these mechanisms.

A number of clinical factors causing such changes increase the risk of DVT. These include: increasing age, prolonged immobility, stroke or paralysis, previous DVT, cancer and cancer treatment, major surgery and trauma, obesity, varicose veins, cardiac dysfunction, indwelling central venous catheters, inflammatory bowel disease, nephrotic syndrome and pregnancy or oestrogen use.

Surgical patients have additional risk factors specific to the type of procedure and anaesthesia. Neurosurgical procedures carry a high risk, mainly due to the duration of surgery and the prolonged postoperative immobilisation.

Thrombophilic disorders also increase the risk of DVT. These disorders are: activated protein C resistance; prothrombin variant 20210A; antiphospholipid antibodies; deficiency or dysfunction of antithrombin, protein C, protein S, or heparin co-factor II; dysfibrinogenemia; decreased levels of plasminogen and
plasminogen activators; heparin induced thrombocytopenia; hyperhomocysteinemia; and myeloproliferative disorders such as polycythemia vera and primary thrombocytosis.

### 7.3 Risk Assessment

When a patient is admitted, the admission nurse (elective patients) or the ward nurse (acute admissions) complete a modified Autar DVT risk assessment score [1] (reproduced in figure 7.1). Body mass index is used to give an objective assessment of the patient’s build—see table 7.1. Body mass index can be calculated or read from the reckoner reproduced as figure 7.9. The sum of points will place the patient in one of these risk categories: insignificant risk (no fatal PE), low risk (fatal PE 0.002%), moderate risk (fatal PE 0.1–0.4%), or high risk (fatal PE 0.4–5%).

The SHO will then complete the form by ticking the appropriate prophylaxis at the bottom of the form in accordance with the table on the next page.

<table>
<thead>
<tr>
<th>Build</th>
<th>Body mass index (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt; 19</td>
</tr>
<tr>
<td>Average</td>
<td>20–25</td>
</tr>
<tr>
<td>Overweight</td>
<td>26–30</td>
</tr>
<tr>
<td>Obese</td>
<td>31–40</td>
</tr>
<tr>
<td>Very Obese</td>
<td>&gt; 41</td>
</tr>
</tbody>
</table>

Table 7.1: Assessment of patient’s build

### 7.4 Prophylaxis

In the majority of cases prophylaxis starts in theatre with intermittent pneumatic compression during surgery. *Prophylaxis never starts postoperatively.* In a few cases of high risk, for example, spinal trauma with paralysis, the prophylaxis is initiated or continued on admission. Prophylaxis is continued until the patient is fully mobilized (= spending more than half the day out of bed).

The surgeon will make sure that heparin is prescribed on the drug chart before the patient leaves theatre.

The recovery staff are responsible for the immediate continuation of any prophylaxis post surgery, and this includes fitting of TED stockings. In the rare event where patients need to keep a Venflon in the foot, the recovery staff fit the TED stocking on the available leg and send the other stocking with the patient to the ward. This routine will further reduce DVT rate as early removal of foot venflons prevent phlebitis on the foot.

There are a number of contra-indications to TEDs and heparin (see below). In many cases heparin can substitute TEDs or vice versa.

Table 7.2 summarises the prophylaxis needed in the different risk groups.
7.5 HIGH DOSE HEPARIN AND WARFARIN TREATMENT

This is very rarely indicated for patients as prophylaxis preoperatively and should always be discussed with the registrar. This is also the case for patients admitted on warfarin treatment. Warfarin may be indicated in some patients with paralysis. If used, high dose heparin should always precede warfarin to avoid transient protein C and S deficiency and relative hypercoagulopathy (day 1–3) caused by warfarin.

7.6 Paralysed Patients

Patients with limb paralysis from spinal trauma, cancer, or degenerative diseases and ventilated patients paralysed as part of their treatment need special attention. They come into the high-risk group automatically, and in addition to heparin 5000 IU bd and TED stockings when they arrive, they get intensive physiotherapy with passive movements of the paralysed limb(s). They need intermittent pneumatic compression on the ward whenever available. Frequent passive movement therapy is also an integrated part of the care provided by the nurses. In paralysed patients with additional risk factors high dose heparin may be indicated, but this should always be an individual registrar or consultant prescription.

7.7 Contra-indications to TEDs

- Lower extremity arterial insufficiency (check foot pulse).
- Wounds or bruises on lower extremity.
- Correct fit not possible.

7.8 Relative contra-indications to heparin

- Intra-axial pathology for surgery (due to increased risk of postoperative haematoma).
- Conditions with haemorrhagic diathesis.
• SAH patients before clipping / coiling of aneurysm (risk of rebleed).

These contra-indications are relative and it may be justified to use heparin in these patient groups. This however should always be discussed with the Registrar.

7.9 Absolute contra-indications to heparin

• Allergy to heparin.

• Heparin-induced thrombocytopenia can occur. This should be suspected in any patient on heparin who has a falling platelet count. Heparin should be stopped immediately and the patient discussed with a haematologist as, paradoxically, this is a very thrombogenic condition.
References

Figure 7.1: Modified Autar risk assessment tool for DVT.
Figure 7.2: BMI reckoner
Chapter 8

Patients with Abnormal Clotting

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Date: February 2006
Review Date: February 2007

8.1 Introduction

Two groups of patients on NICU may present with abnormal clotting results: those on long-term anticoagulation who require neurosurgery, and those who develop clotting abnormalities as a result of their underlying illness. The first group is discussed in section 8.2 and the second in section 8.3.

8.2 Patients on long-term anticoagulation

The problems of managing neurosurgical patients on long-term anticoagulation are significant. A balance has to be found between the risks of bleeding and the risk of thromboembolism. The mortality of intracranial haemorrhage associated with oral anticoagulation is about 60%. Thus, in most neurosurgical patients, the risk of bleeding outweighs the risk of thromboembolism. Common indications for anticoagulation to prevent arterial thromboembolism are:-

- Atrial fibrillation (AF).
- Presence of mechanical heart valves.
• Patients with carotid stenoses or other peripheral vascular disease with or without arterial grafts.

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.

8.2.1 Risk of thromboembolism

Risk of Arterial Thromboembolism

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 or advanced age (≥75 years).

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 again increased in presence of AF with a history of previous thromboembolism. The greatest risk appears to be within 90 days of placement of mechanical valve.

Risk of Venous Thromboembolism

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

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

8.2.2 Management

Elective Neurosurgery

Elective neurosurgery should be delayed:

• For at least 90 days after placement of a mechanical heart valve.

• For a minimum of at least 3 months in patients who have had a venous thromboembolic event.

Patients with Atrial Fibrillation

• Stop Warfarin 5 days pre-operatively.

• Check INR on the day before surgery aiming for a ratio of ≤1.4.

• Admit 24 hours pre-operatively and give 40 mg Enoxaparin.

• Restart Warfarin (at usual maintenance dose) 48–72 hours post surgery or when full haemostasis has been secured.
8.2. **PATIENTS ON LONG-TERM ANTICOAGULATION**

- Antithrombotic measures, e.g. compression stockings, should be used if the patient is going to be immobile for a prolonged period.

**Elective Surgery on Venous Thromboembolic patients who are taking Warfarin**

- Stop Warfarin 5 days pre-operatively.
- Admit 24 hours pre-operatively and give 40 mg Enoxaparin.
- Use Elastic Stockings. In very high risk patients consider use of retrievable IVC filter (e.g. Gunther - Tulip).
- Restart Warfarin 24–48 hours after surgery, or when full haemostasis has been secured.
- Continue Enoxaparin until INR ≥ 2.0.
- For patients with acute DVT use low molecular weight heparin (LMWH) as initial IV boluses of unfractionated heparin (UH) are more likely to promote bleeding than initial subcutaneous doses of LMWH. A meta-analysis of 13 clinical trials has concluded that LMWH is more effective, and is associated with less bleeding, than UH when used for treatment of DVT.

**Elective Surgery on Patients with Prosthetic Heart Valves**

- Stop Warfarin 4–5 days pre-operatively.
- Admit 48 hours pre-operatively and start full dose intravenous heparin (unfractionated).
- Stop heparin infusion 6 hours before surgery and check APTT 2 hours before surgery. Check INR as well.
- Restart heparin 24–48 hours after surgery and continue until INR ≥ 2.
- Restart Warfarin 24–48 hours after surgery as guided by post-operative haemostatic integrity.

**Semi-Emergency Surgery on Patients taking Warfarin**

In this group of patients a 6 hour delay is acceptable.

- Intravenous vitamin K will reverse anticoagulation in 6 hours.
- Give 2–10 mg depending on how quickly patient needs to be re-anticoagulated post-operatively.
- NB. Fresh frozen plasma (FFP) is rarely of any value unless volume expansion is required.
Emergency Surgery on Patients Taking Warfarin

In this group of patients immediate correction is required to allow surgical intervention - e.g. intra-cerebral bleed on Warfarin.

- Give Beriplex (Prothrombin Complex Concentrate (PCC) containing Factors II, VII, IX and X) which will normalise INR (i.e. INR $\leq 1.2$) in 10 minutes. Dose 50iu/kg.
- FFP has virtually no role, (unless PCC is unavailable).
- Also give 5 mg intravenous vitamin K.
- Consider use of compression stockings and / or temporary IVC filters if patient is likely to be immobilised for a long period.

Emergency Surgery on Patients on Prophylactic LMWH

- Maximum anticoagulation is at 3–4 hours post injection.
- In practice not a problem.
- Go ahead with surgery without waiting for reversal (if reversal is deemed absolutely necessary please see reversal guidelines below).

Emergency Surgery on patients on Full Dose LMWH

- Difficult and of major concern.
- Protamine only partially reverses LMWH.
- Anti-Xa assays are of no value other than initially to assess level of anticoagulation.
- In practice give 50 mg Protamine Sulphate IV at a rate of 5 mg/min. Check blood pressure after each administration.

NB: For Clexane (ENOXAPARIN) the manufacturers (Rhone-Poulenc Rorer) recommend a Protamine dose of 1 mg Protamine per 1 mg Clexane if last dose given less than 8 hours before and 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 site epidural/spinal drain if LMWH given within 12 hours.
- Remove catheter 4 hours before the next dose.
8.3. SECONDARY CLOTTING FAILURE

8.2.3 Recommencing Anticoagulation

- The time frame for recommencing anticoagulation is unclear.
- Suggested guidelines include waiting for a period of 4–6 weeks after intracranial bleed, with a check CT scan first, in patients with a mechanical heart valve.
- Similar time frames would be appropriate for patients in AF.∗
- Patients with a recent venous thromboembolic event should probably be started sooner, but each individual will need to be assessed in conjunction with neurosurgeon and cardiologist.†

8.3 Secondary clotting failure

Patients admitted to intensive care units frequently develop mild, sometimes progressive, abnormalities of INR. There is usually no history of anticoagulant therapy or of malnutrition. Patients may also develop abnormal clotting as a result of severe blood loss, massive transfusion, disseminated intravascular coagulation (DIC) or severe sepsis.

8.3.1 Management

Prophylaxis

- All ventilated patients on NICU with a history of alcohol abuse should receive routine vitamin K (5 mg PO bd).
- Patients receiving total parenteral nutrition (TPN) should receive routine vitamin K (if PO therapy not possible give 0.5–1 mg slowly IV od, or 5 mg IM od).

Treatment

- INR ≥ 1.2. If the patient has had any form of intracranial haemorrhage then start PO vitamin K (5 mg bd).
- INR ≥ 1.4 and recent intracranial haemorrhage, give vitamin K (10 mg IV). If there has been no intracranial haemorrhage, do not treat unless there are clinical signs of bleeding.
- In life-threatening haemorrhage, discuss with haematologist regarding use of factor concentrates e.g. Beriplex. **FFP has no place in the treatment of life threatening intracranial haemorrhage where rapid and reliable correction of abnormal clotting is required.** Beriplex 50iu/kg is the treatment of choice.

NB As Beriplex is thrombogenic, antithrombotic measures e.g. elastic compression stockings etc. may be required to minimise risk of DVT.

∗Warfarin may be delayed until judged to be haemostatically safe.
†If systemic anticoagulation with Warfarin is judged to be too risky with regard to re-bleeding then use LMWH and consider insertion of temporary IVC filter. Warfarin should be re-started when judged to be safe by neurosurgeon.
In DIC, cryoprecipitate and/or FFP may be required.

8.4 References


Chapter 9

External Ventricular Drains

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Date: February 2006
Review Date: February 2007

9.1 CSF Sampling and Intrathecal Vancomycin Administration Via an External Ventricular Drain

9.1.1 Equipment

- *Always* use a dressing trolley whenever possible.
- *Always* use aseptic technique.
- *Always* use a new vial of vancomycin for each patient.

<table>
<thead>
<tr>
<th>Dressing pack</th>
</tr>
</thead>
<tbody>
<tr>
<td>Two 2-ml syringes</td>
</tr>
<tr>
<td>Sterile universal container</td>
</tr>
<tr>
<td>Sterile gloves (if not in dressing pack)</td>
</tr>
<tr>
<td>Chlorhexidine spray</td>
</tr>
</tbody>
</table>

Table 9.1: Equipment required for CSF sampling from an external ventricular drain

9.1.2 How to take a CSF sample

1. Ensure procedure is explained in full to the patient, wash hands and prepare equipment for use.

77
Table 9.2: **Additional equipment required for administration of IT vancomycin via an external ventricular drain**

2. Turn off the drain at the distal three-way tap.

3. Identify sample port along tubing, closest to the patient, *not* the distal port.

4. Place sterile towel under sample port.

5. Spray sample port and tubing with chlorhexidine. *Wait* for it to evaporate.

6. Put gloves on/change gloves.

7. Carefully draw off 2 ml of CSF (over 2 minutes) and discard. *Do not use needles.* If CSF cannot be aspirated with ease then abandon procedure (see 9) and discuss with Specialist Registrar.

8. Carefully draw off another 1–2 ml of CSF (1–2 minutes), send this in a sterile universal container to Microbiology for MC&S.

9. Ensure the zero point external drainage system is level with the external auditory meatus and that the reservoir chamber draining at the correct height. Open the clamps.

10. *All* CSF samples should be sent urgently to the labs.

### 9.1.3 How to give intrathecal vancomycin

1. Ensure vancomycin has been ordered from the pharmacy.

2. Take sample as above, omitting No. 9.

3. Give the Vancomycin via the sample port, over 2 minutes.

4. Draw up at least 2 ml of normal saline and flush the tube slowly (over 1–2 minutes), to clear the dead space

5. Turn off the proximal three-way tap. *(The drain should now be completely off).*

6. Remove gloves and wash hands

7. Inform the nursing staff of the time that the drain was turned off and ask them to open the drain in one hour.

8. Sign on the drug charge that the drug has been given.
Chapter 10

Withdrawal of Therapy and Related Issues

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Date: February 2006
Review Date: February 2007

10.1 Withdrawal of Therapy

In situations when continuing treatment is not thought to be in the interests of the patient an ethical decision may be made to withdraw active therapy that does not relieve symptoms. While decisions of this sort are made in all specialties, they have a particular significance in the intensive care setting, since cessation of life-supporting therapy often leads to death quickly. The aims of these guidelines are to ensure that such terminally ill patients die with dignity and without suffering.

10.2 Guidelines for withdrawal of therapy

1. The patient’s condition is reviewed by a Senior Member of the admitting clinical team and the Consultant in charge of the NICU, both of whom must agree that further treatment is not in the patient’s best interest. If the admitting Consultant does not take part in this discussion, he/she must be aware of it since he or she shares responsibility for the decisions that are taken.

2. The nursing staff caring for the patient should be involved in the discussion and given an opportunity to express their views.

3. A member of the medical staff and a nurse will discuss the patient’s prognosis with the relatives or next of kin. Depending on the patient, the
medical staff involved may be a member of the admitting team, the NICU team or both. Normally the fact that the patient is hopelessly ill is intro-
duced to them over one or more days. This gives relatives time to come to
terms with the situation. The Consultant and the nurse will also explain
the way in which treatment will be withdrawn.

4. In the course of the discussion it must be pointed out to the family that
some patients do not die quickly following withdrawal of therapy. It is also
pointed out that every effort will be made to keep the patient comfortable
with appropriate medication, and that such symptomatic therapy may
incidentally hasten the patient’s demise.

5. The NICU Consultant or member of the admitting team documents the
decision to withdraw treatment in the patient’s notes. He/She also docu-
ments:
   - the discussion with the family
   - specific instructions for terminal care

6. The patient will be prescribed opioids (morphine 2.5–5 mg i.v prn or mor-
phine infusion at 1–10 mg/hr) in order to ensure comfort and freedom
from respiratory distress (a respiratory rate greater than 20/min or in-
creased accessory muscle activity are useful triggers for escalating opioid
dose). The patient can also be prescribed hyoscine 0.2–0.4 mg iv prn to
cope with noisy or distressing respiratory secretions.

7. Infusions of vasoactive drugs are stopped. All other prescriptions (e.g
antibiotics) are cancelled. Intravenous or enteral hydration is continued
where appropriate.

8. Respiratory support is withdrawn:
   - If the patient is able to breathe spontaneously the trachea is extu-
bated.
   - If the patient is unable to breathe spontaneously the patient may
   satisfy criteria for brain stem death, in which case the tests for BSD
   can be performed.
   - Rarely patients may not breath spontaneously, but performance of
   BSD criteria is inappropriate. Under these circumstances the NICU
   Consultant responsible will make a decision as to whether the patient
   should be extubated. It is not essential that BSD criteria be fulfilled
   if withdrawal of therapy is agreed to be the course of action that is
   in the patient’s best interests.

9. Monitoring of the patient will be minimised or stopped.

10. The patient’s family is given as much privacy as possible. It may be
appropriate to transfer the dying patient to a side room or possibly to
a general ward if they are likely to survive for more than 24 hours. It
is usually preferable to allow a patient to die in the NICU, where the
family can be offered support by familiar members of staff. The support
of religious and/or cultural organisations may be sought.
11. All nursing and medical actions are appropriately documented.

12. 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.

10.3 Do not resuscitate (DNR) decisions/non-escalation of therapy

For patients who are on intensive or maximal therapy but show little response, it may be perfectly ethical to continue with full therapy as prescribed, but to take a decision not to resuscitate in the event of a cardiac arrest, or re-institute or increase ventilatory support in the event of acute respiratory deterioration. Such decisions must be made after the discussions described in points 1–3 above have taken place. DNR orders must be documented and reviewed regularly (at least every 24 hours).

In other instances a decision will be taken not to escalate therapy beyond certain predetermined endpoints. These must be explicitly documented in the notes by the NICU Consultant or after discussion with him/her. Specific points that may need to be addressed include:

- DNR status.
- Institution of renal support (haemofiltration or dialysis) for hyperkalaemia, acidosis or volume overload.
- Re-intubation and/or re-ventilation.
- Change of or restarting antibiotics.
- Institution of or escalation of vasoactive therapy (specify agent and dose limits).
- Intensification of monitoring
- Symptomatic therapy.
Chapter 11

Guidelines for the Diagnosis of Brain Death and Brain Stem Testing

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Date: February 2006
Review Date: February 2007

11.1 Introduction

For the majority of us, our death will be confirmed when our heart stops and our respiration ceases. In a very small percentage of cases, modern medicine allows continued support of cardiorespiratory function after the brain has ceased to function. How then do we diagnose death? Currently, there is no statutory definition of death in English Law. In 1976, the Conference of Medical Royal Colleges and their Faculties issued a statement on “brain death”, and the proposals contained in the statement have been recognised by the courts in England and Northern Ireland for the diagnosis of death. Confusion has arisen because of the differences that exist between countries as to what criteria are required to diagnose death using neurological examination and also the relationship with solid-organ transplantation.

In the Conference of Royal Colleges and their Faculties statement on brain death in 1976, brain death was defined as “permanent functional death of the brain stem”. Once this occurred, further artificial support was deemed fruitless and should be withdrawn. This definition discounts the relevance of any residual activity in the upper brain and has proved reliable and robust. In a follow-up series of over 1300 patients diagnosed as brain dead on the basis of the UK criteria, all patients suffered loss of cardiorespiratory function even if supportive measures were continued.
CHAPTER 11. BRAINSTEM DEATH

In 1983, the Health Department published “Cadaveric organs for transplantation: A code of practice including the diagnosis of brain death.” This code of practice was refined in 1998 to “A code of practice for the diagnosis of brainstem death including guidelines for the identification and management of potential organ and tissue donors.” It is this latest document that provides the basis for current UK requirements for the diagnosis of brainstem death: In 2004, the Intensive Care Society published practical guidelines for the performance of brainstem tests.

11.2 Preconditions

1. The patient’s condition is due to irremediable brain damage of known aetiology. In some cases such as massive head trauma or subarachnoid haemorrhage this may be apparent immediately. In others, such as global hypoxia following cardiac arrest, the fact that it is irremediable may take some time to establish.

2. The patient is deeply unconscious.

3. The patient is being maintained on the ventilator because spontaneous respiration has been inadequate or ceased altogether. The effects of neuromuscular blockers or other respiratory depressants must be excluded. The presence of neuromuscular blockade can be easily assessed using a peripheral nerve stimulator.

11.3 Exclusions

1. There should be no evidence that the state is due to depressant drugs (narcotics, hypnotics, and tranquillisers). There are no specific directives as to whether drugs should be cleared completely or reached a level incompatible with the clinical picture of brain stem death. The criteria used to determine whether depressant drugs may invalidate the tests, vary considerably. These included an empirical wait of varying time following cessation of these drugs, to the use of specific antidotes (flumazenil, naloxone). The performance of brain stem tests after the administration of barbiturates is particularly difficult as the half-life is long and there is no specific antidote. Analysis of blood levels is recommended to aid management.

2. Primary hypothermia must have been excluded ( < 35°C)

3. Potentially reversible circulatory, metabolic and endocrine disturbances must have been excluded as the cause of the continuation of unconsciousness. The circulatory and metabolic effects of brain stem death such as bradycardia, hypertension, hypernatraemia and diabetes insipidus are recognised as being secondary rather than the cause of the condition and do not preclude subsequent testing of brainstem function.
11.4 Clinical assessment of brain stem function

1. **No pupillary response to light.** The pupils are fixed and do not respond directly or consensually to sudden changes in light intensity. This tests cranial nerves II (afferent) and III (efferent).

2. **No corneal reflex.** Direct stimulation of the cornea using a cotton bud or tissue elicits no response: cranial nerves V (afferent) and VII (efferent).

3. **No vestibulo-ocular reflex.** No eye movements are seen during or following slow injection of > 50 ml of ice-cold water over one minute into each external auditory meatus in turn. The ears should be checked before to ensure that the passage is clear and the tympanic membrane intact. An intact response is indicated by eye movement towards the stimulus. This tests cranial nerves VIII (afferent) and III, IV and VI (efferent).

4. **No motor response to central stimulation.** No motor response is elicited following adequate stimulation of any somatic area (usually performed using painful stimuli to the supraorbital ridges). This tests cranial nerves V (afferent) and VII (efferent). Peripheral painful stimulation may not result in a central motor response in the presence of a cord injury and therefore is not recommended. Importantly, spinal reflexes may be preserved and can produce complex responses—the Lazarus reflex involves back arching and extension of the arms. This can be dramatic and upsetting but is indicative of intact pathways below the brain stem and does not invalidate a diagnosis of brain stem death.

5. **No gag and cough reflexes.** There is no gag reflex on stimulation of the soft palate using a spatula and no cough reflex on bronchial stimulation by placing a suction catheter in the trachea (cranial nerves IX and X).

6. **Apnoea.** No respiratory movements are seen during apnoea testing. No respiratory movements are seen when the patient is disconnected from the ventilator. During the test, the arterial \( P_{\text{CO}_2} \) should reach at least 6.65 kPa. In practice, the patient should be preoxygenated for ten minutes with 100% oxygen whilst reducing the minute ventilation such that the \( P_{\text{CO}_2} \) reaches approximately 5 kPa. Hypoxia during disconnection of the ventilator is avoided by delivering oxygen at 6 litres/minute through a catheter placed in the trachea. Once the \( P_{\text{CO}_2} \) has reached 6.65 kPa and the absence of spontaneous respiration confirmed by clinical observation, the patient is reconnected to the ventilator.

11.5 Performance and repetition of testing

1. The performance of brain stem tests should be done by at least two medical practitioners who have been registered by the General Medical Council for at least five years. One of these should be a consultant. Both should be competent in the field and neither should be a member of the transplant team.

2. 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
one should do a full set of tests whilst being observed, this counting as
one set of tests. The time interval between the tests is not stipulated.

3. The legal time of death is when the first set of tests has been completed.
Death is certified on completion of the second set of tests irrespective of
when active support is discontinued.

11.6 Special circumstances

11.6.1 Neonates and small children
A report of a working party of the British Paediatric Association that was
supported by the Council of the Royal College of Physicians has suggested that
in children older than 2 months, the criteria for brain stem death should be
the same as for adults. Between 37 weeks gestation and 2 months of age, it is
rare to be able to confidently diagnose brain stem death. Below 37 weeks, the
criteria should not be applied.

11.6.2 Pre-existing respiratory disease
Some patients with pre-existing lung disease may respond only to hypoxic drive
and need supra-normal levels of CO₂. Advice should be taken as to appropriate
targets—this is commonly taken to be a $P_{CO_2}$ at least 2 kPa above the patient’s
normal baseline value. The oxygen level administered may also need to be
reduced.

11.6.3 Local pathology that precludes clinical testing
Local facial trauma may preclude one or more of the tests for brain stem reflexes.
All possible tests should be completed and consideration given to additional tests
such as cerebral angiography, radionuclide scanning, transcranial doppler or
electroencephalogram. Although these additional tests have not been validated
in the UK this is not because they are insensitive, but rather that they are not
widely available.

11.7 Other clinical conditions that may present
difficulties
Published reports have identified other conditions that may mimic brain death.
These include severe Guillain-Barré syndrome, rabies encephalitis, brainstem
encephalitis, amitriptyline overdose, beryllium overdose, and severe hypophos-
phataemia.

The “locked-in” syndrome consists of quadriplegia and anarthria with preser-
vation of consciousness. Vertical eye movements are retained. The syndrome is
usually caused by damage affecting the ventral pons or rarely by extensive de-
struction of corticobulbar and corticospinal tracts. The condition is extremely
rare and is often first recognised by family members who report that the patient
appears aware. It may be possible to establish non-verbal communication using
the retained vertical eye movements. Ten year survival for these patients has been reported to be as high as 80%.

Patients in a “persistent vegetative state” appear to be awake at times but show no signs of awareness. Generally patients are able to breathe unaided. The syndrome usually arises as a result of severe hypoxic injury or extensive traumatic brain injury. It is arbitrarily defined if a vegetative state persists for longer than four weeks. Criteria for the diagnosis of a persistent vegetative state have recently been clarified.
Chapter 12

Organ and Tissue Transplantation

Andy Eynon BSc, MBBS, MD, MRCP, FFAEM, EDIC.
Director of Neurosciences Intensive Care Unit.

Trish Collins, Transplant Co-ordinator.

Date: February 2006
Review Date: February 2007

12.1 Introduction

All patients with severe, irreversible brain injury are potential solid organ and tissue donors. Even if they do not satisfy brainstem criteria, they may be potential non heart beating donors or tissue donors. It is worth discussing all potential donors with the transplant coordinator.

12.2 Criteria for organ and tissue donation

1. Family consent.

2. Absence of malignancy with metastatic potential (primary brain tumours should have histological confirmation).

3. Absence of overwhelming sepsis.

4. Absence of life-threatening communicable disease (HIV and CJD are absolute contraindications, as is a family history of CJD).

A referral can then be made to Transplant Coordinator who will then establish if the patient is suitable.
12.2.1 Consent (Lack of Objection)

Relatives may be approached by senior staff or one of the transplant coordinators. Wessex regional coordinators would like to be involved, if possible, at the time of brain stem death discussion. It is important that the family understand the diagnosis and implications, and that the request for organ or tissue donation is seen as separate from any decision making regarding the futility of continued medical treatment. Although discussion of end of life decisions and organ/tissue donation is stressful for all concerned, many families find solace in some good arising from an inevitable tragedy.

Cases that require a coroner’s post-mortem are not automatically excluded from organ/tissue donation. Each case needs to be discussed individually with the coroner’s office.

12.3 Types of organ/tissue donation

12.3.1 Patients who are diagnosed as brain dead using formal brain stem testing

Patients are potentially donors of multiple solid organs, including heart, lungs, kidneys, liver, pancreas, small bowel (and can also be tissue donors following solid organ donation).

Age restrictions are in place for some organs (e.g. heart donors should be < 65 years) but other organs may be donated at virtually any age.

12.3.2 Non heart-beating donors

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.

Patients should be 16–65 years of age.

There is potential for donating liver (when time between withdrawal of treatment and death is < 1 hour) and / or kidneys (< 4 hours), in addition to tissues.

12.3.3 Tissue donation

- heart valves: birth to 65 years
- skin: 17 years upwards
- corneas: > 2 year (no upper age limit)
- tendon, ligaments 17–50 years
- bone: 17 years upwards
12.4 Medical and nursing care of the organ donor

This is a very difficult time for relatives. Patients must continue to be treated with respect and dignity. If possible, it is preferable to move the patient into a side-room for privacy.

12.4.1 Patients who are diagnosed as brain dead using formal brain stem testing

Brain death is usually associated with autonomic dysfunction. Alteration in cardiovascular and respiratory function occurs, often with diabetes insipidus and hypothermia. The principle of management is to maintain the organs in the best functional state prior to transplantation.

General care

- Continued active nursing care and physiotherapy
- Keep normothermic
- Investigations
- ABGs 2–4 hourly
- ECG
- CXR
- Echocardiogram if possible
- Minimum of daily FBC, U&E, clotting

Cardiovascular

- Arterial line (preferably left arm) and CVP line (preferably on right)
- Target MAP $\geq$ 60 mmHg, urine output > 30 ml/hr, CVP 8–12 mmHg
- Crystalloid / colloid if not achieving targets
- Add dopamine < 10 $\mu$g/kg/min if not achieving target
- If still not achieving target, use CO monitoring (LiDCO or SG catheter) and add adrenaline / noradrenaline / vasopressin

Respiratory

- Minimum $F_{O_2}$ to achieve $P_{O_2}$ of 12–14 kPa
- Ventilate to $P_{CO_2}$ of 3.8–6 kPa (keep peak airway pressure < 35 cmH$_2$O)
- PEEP < 5–10 cmH$_2$O
Endocrine

- methylprednisolone; 15 mg/kg bolus on declaration of brain death
- T3; bolus of 4 µg then 3 µg/hr infusion
- insulin infusion to keep BM 5–8
- vasopressin; initial bolus of 2 units followed by an infusion of 1–10 u/hr to maintain MAP > 70 mmHg. Wean down / off other inotropic agents. If blood pressure rises following administration of vasopressin, discontinue infusion.
- Treat DI with DDAVP

Patients may deteriorate despite support and it may prove impossible to donate organs. This is explained to the relatives during the initial discussions with the transplant coordinator. It is inappropriate to attempt heroic measures (including CPR) if the patient deteriorates purely in the hope of successful donation.

12.4.2 Non heart-beating donors

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 NIBP. These can be monitored from the nurses’ station if relatives are distressed at watching the monitor.
- Keep normothermic.
- Analgesia: morphine sulphate (60 mg in 60 ml normal saline) at a rate sufficient to alleviate pain.
- Hyoscine: 200–400 µg PRN to reduce secretions.

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 coordinator. If death has not occurred within 1 hour of treatment withdrawal, liver donation is not possible. There is a cut off of 4 hours for kidney donation. The transplant coordinator 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 < 5 minutes at the bedside. If the relatives wish for more time, donation will not go ahead.
12.4.3 Tissue donation
Tissues are retrieved in the mortuary, generally within 24 hours of death.

12.4.4 How to contact the transplant coordinator
Transplant Coordinators can be contacted 24 hrs a day on:

08700 555 500 pager no. 839261
Chapter 13

Common Drug Infusions

Andy Eynon BSc, MBBS, MD, MRCP, FFAEM, EDIC.
Director of Neurosciences Intensive Care Unit.

Date: February 2006
Review Date: February 2007
13.1 Noradrenaline

<table>
<thead>
<tr>
<th>Rate (ml/hr)</th>
<th>Noradrenaline (mg/hr)</th>
<th>Concentration (µg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.08</td>
<td>1.3</td>
</tr>
<tr>
<td>2</td>
<td>0.16</td>
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<td>3</td>
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<tr>
<td>4</td>
<td>0.32</td>
<td>5.3</td>
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<tr>
<td>5</td>
<td>0.4</td>
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<tr>
<td>6</td>
<td>0.48</td>
<td>8</td>
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<tr>
<td>9</td>
<td>0.72</td>
<td>12</td>
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<tr>
<td>10</td>
<td>0.8</td>
<td>13.33</td>
</tr>
</tbody>
</table>

Table 13.1: Noradrenaline infusion. 20 mg in 250 ml (or 4 mg in 50 ml).

13.2 Adrenaline

<table>
<thead>
<tr>
<th>Rate (ml/hr)</th>
<th>Adrenaline (mg/hr)</th>
<th>Concentration (µg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>0.1</td>
<td>1.66</td>
</tr>
<tr>
<td>2</td>
<td>0.2</td>
<td>3.33</td>
</tr>
<tr>
<td>3</td>
<td>0.3</td>
<td>5</td>
</tr>
<tr>
<td>4</td>
<td>0.4</td>
<td>6.66</td>
</tr>
<tr>
<td>5</td>
<td>0.5</td>
<td>8.3</td>
</tr>
<tr>
<td>6</td>
<td>0.6</td>
<td>10</td>
</tr>
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<td>7</td>
<td>0.7</td>
<td>11.66</td>
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<tr>
<td>8</td>
<td>0.8</td>
<td>13.33</td>
</tr>
<tr>
<td>9</td>
<td>0.9</td>
<td>15</td>
</tr>
<tr>
<td>10</td>
<td>1.0</td>
<td>16.66</td>
</tr>
</tbody>
</table>

Table 13.2: Adrenaline infusion. 5 mg in 50 ml.
### 13.3 Other Drugs

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose Volume</th>
<th>Final Vol.</th>
<th>Diluent</th>
<th>Final conc.</th>
<th>Starting dose range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline</td>
<td>5 mg 5 ml</td>
<td>50 ml</td>
<td>5% Dextrose</td>
<td>100 µg/ml</td>
<td>0–10 ml/hr</td>
</tr>
<tr>
<td>Atracurium</td>
<td>500 mg 50 ml</td>
<td>50 ml</td>
<td>Neat</td>
<td>10 mg/ml</td>
<td>0–6 ml/hr</td>
</tr>
<tr>
<td>Clonidine</td>
<td>750 µg 50 ml</td>
<td>50 ml</td>
<td>0.9% Saline</td>
<td>15 µg/ml</td>
<td>0–3 ml/hr</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>250 mg 20 ml</td>
<td>50 ml</td>
<td>5% Dextrose or 0.9% Saline</td>
<td>5 mg/ml</td>
<td>0–20 ml/hr</td>
</tr>
<tr>
<td>Dopamine</td>
<td>200 mg 5 ml</td>
<td>50 ml</td>
<td>5% Dextrose or 0.9% Saline</td>
<td>4 mg/ml</td>
<td>0–5 ml/hr</td>
</tr>
<tr>
<td>Fentanyl</td>
<td>2.5 mg 50 ml</td>
<td>50 ml</td>
<td>Neat</td>
<td>50 µg/ml</td>
<td>0–4 ml/hr</td>
</tr>
<tr>
<td>GTN</td>
<td>50 mg 50 ml</td>
<td>50 ml</td>
<td>Neat</td>
<td>1 mg/ml</td>
<td>0–10 ml/hr</td>
</tr>
<tr>
<td>Insulin</td>
<td>50 units 0.5 ml</td>
<td>50 ml</td>
<td>0.9% Saline</td>
<td>1 unit/ml</td>
<td>sliding scale</td>
</tr>
<tr>
<td>Midazolam</td>
<td>50 mg 50 ml</td>
<td>50 ml</td>
<td>0.9% Saline</td>
<td>1 mg/ml</td>
<td>0.5 ml/hr</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>4 mg 4 ml</td>
<td>50 ml</td>
<td>5% Dextrose</td>
<td>80 µg/ml</td>
<td>0–10 ml/hr</td>
</tr>
<tr>
<td>Noradrenaline</td>
<td>20 mg 20 ml</td>
<td>250 ml</td>
<td>5% Dextrose</td>
<td>80 µg/ml</td>
<td>0–10 ml/hr</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>loading dose</td>
<td>1000 mg 20 ml</td>
<td>100 ml</td>
<td>0.9% Saline</td>
<td>10 mg/ml</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>maintenance</td>
<td>300 mg 6 ml</td>
<td>0.9% Saline</td>
<td>10 mg/ml</td>
<td>over 30 min</td>
</tr>
<tr>
<td>Propofol</td>
<td>1% 50 ml</td>
<td>50 ml</td>
<td>Neat</td>
<td>10 mg/ml</td>
<td>0–20 ml/hr</td>
</tr>
<tr>
<td>Thiopentone</td>
<td>2.5 g 100 ml</td>
<td>100 ml</td>
<td>Water for injection</td>
<td>25 mg/ml</td>
<td>3–5 mg/kg/hr</td>
</tr>
</tbody>
</table>

Table 13.3: NICU common drug infusions. NB The use of vasoactive agents should be guided by haemodynamic parameters.
Chapter 14

IV Drug Compatibility Chart

Andy Eynon BSc, MBBS, MD, MRCP, FFAEM, EDIC.
Director of Neurosciences Intensive Care Unit.

Caroline Cole
Directorate Pharmacist

Date: February 2006
Review Date: February 2007

14.1 Y-site Compatibility of IV Infusions

Read before referring to compatibility chart.

• 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.
**14.1.1 Drugs which must *always* be infused separately**

<table>
<thead>
<tr>
<th>Category</th>
<th>Drug</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antibiotics</td>
<td>Normal human immunoglobulin</td>
</tr>
<tr>
<td>Antifungals</td>
<td>Omperazole</td>
</tr>
<tr>
<td>Antivirals</td>
<td>Phenobarbital</td>
</tr>
<tr>
<td>Acetazolamide</td>
<td>Phenytoin</td>
</tr>
<tr>
<td>Alteplase</td>
<td>Sodium valproate</td>
</tr>
<tr>
<td>Aprotinin</td>
<td>Thiopeptone</td>
</tr>
<tr>
<td>Mannitol</td>
<td></td>
</tr>
</tbody>
</table>

**14.1.2 For further information**

Contact critical care pharmacists: Mark Tomlin, bleep 2221; Emma Bertram, bleep 9179; Caroline Cole, bleep 2088; or medicines information, ext 6908.
14.2 Y-site Compatibility of IV Infusions

<table>
<thead>
<tr>
<th></th>
<th>Vvecuronium</th>
<th>Sodium Nitroprusside</th>
<th>Propofol</th>
<th>Norepinephrine</th>
<th>Morphine</th>
<th>Milrinone</th>
<th>Midazolam</th>
<th>Labetalol</th>
<th>Insulin</th>
<th>Heparin</th>
<th>GTN</th>
<th>Furosemide</th>
<th>Fentanyl</th>
<th>Epinephrine</th>
<th>Dobutamine</th>
<th>Calcium</th>
<th>Atracurium</th>
<th>Amiodarone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aminophylline</td>
<td>C</td>
<td>N</td>
<td>N</td>
<td>C</td>
<td>I</td>
<td>I</td>
<td>N</td>
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<td>Amiodarone</td>
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**Key.**
- **C**: Compatible at Y-site under certain conditions
- **V**: Conflicting information of compatibility / incompatibility. **Avoid** combination where possible—use together only with *extreme caution.*
- **N**: No information available. **Do not mix.**
- **I**: Known to be incompatible. **Do not mix.**

Information given in this table must be interpreted with caution—please refer to notes on previous page.
Chapter 15

Haemodynamics Algorithm

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Director of Neurosciences Intensive Care Unit.

Date: February 2006
Review Date: February 2007
Figure 15.1: NICU haemodynamics algorithm
Chapter 16

Cardiac Haemodynamic Formulae

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Director of Neurosciences Intensive Care Unit.

Date: February 2006
Review Date: February 2007

Cardiac Output (CO)

\[ CO = HR \times SV \]

Where HR is heart rate and SV is stroke volume. Normal values of 4 to 8 l/min.

Cardiac Index (CI)

\[ CI = \frac{CO}{BSA} \]

Where BSA is body surface area in m\(^2\). Normal values of 2.5 to 4 l/min/m\(^2\).

Stroke Volume (SV)

\[ SV = \frac{CO}{HR} \]

Normal values of 60 to 100 ml/beat.

Stroke Volume Index (SVI)

\[ SVI = \frac{CI}{HR} \]

Normal values of 33 to 47 ml/m\(^2\)/beat.
Left Ventricular Stroke Work (LVSW)

\[ \text{LVSW} = \text{SV} \times (\text{MAP} - \text{PAWP}) \times 0.0136 \]

Where MAP is mean arterial pressure and PAWP is pulmonary artery wedge pressure. Normal values of 58 to 104 gm·m/beat.

Right Ventricular Stroke Work (RVSW)

\[ \text{RVSW} = \text{SV} \times (\text{MPAP} - \text{RAP}) \times 0.0136 \]

Where MPAP is mean pulmonary artery pressure and RAP is right atrial pressure. Normal values of 8 to 16 gm·m/beat.

Systemic Vascular Resistance (SVR)

\[ \text{SVR} = \frac{\text{MAP} - \text{CVP}}{\text{CO}} \times 80 \]

Where CVP is central venous pressure in mmHg. Normal values of 800 to 1200 dynes-sec/cm\(^5\).

Systemic Vascular Resistance (Indexed) (SVRI)

\[ \text{SVRI} = \frac{\text{MAP} - \text{CVP}}{\text{CI}} \times 80 \]

Where CVP is central venous pressure in mmHg. Normal values of 1970 to 2390 dynes-sec/cm\(^5\)/m\(^2\).

Pulmonary Vascular Resistance (PVR)

\[ \text{PVR} = \frac{\text{MPAP} - \text{PCWP}}{\text{CO}} \times 80 \]

Where MPAP is mean pulmonary artery pressure and PCWP is pulmonary capillary wedge pressure in mmHg. Normal value is less than 250 dynes-sec/cm\(^5\).

Pulmonary Vascular Resistance (Indexed) (PVRI)

\[ \text{PVRI} = \frac{\text{MPAP} - \text{PCWP}}{\text{CI}} \times 80 \]

Normal values of 255 to 285 dynes-sec/cm\(^5\)/m\(^2\).

Arterial Oxygen Saturation

\[ S_{a, O_2} \]

Normal values of 95 to 100%.
Alveolar gas equation

\[ P_{A\text{O}_2} = P_{I\text{O}_2} - \frac{P_{A\text{CO}_2}}{R} \]

Where \( P_{A\text{O}_2} \) is the alveolar partial pressure of oxygen, \( P_{I\text{O}_2} \) is the partial pressure of oxygen in the inspired gas mixture, \( P_{A\text{CO}_2} \) is the alveolar partial pressure of carbon dioxide, and \( R \) is the respiratory quotient (normally assumed to be 0.6 to 0.8).

Carbon dioxide is considered to be very soluble and so the alveolar and arterial partial pressures of carbon dioxide are taken to be the same.

Partial pressure of oxygen in the inspired gas mixture is calculated by multiplying the measured \( F_{I\text{O}_2} \) (0.6 for 60%) by the barometric pressure (atmospheric pressure is 101.25 kPa, leading to the convenient approximation of 21 kPa on air, 35 kPa on 35% oxygen).

A clinically useful form of the alveolar gas equation therefore becomes:

\[ P_{A\text{O}_2} = (F_{I\text{O}_2} \times PB) - \frac{P_{a\text{CO}_2}}{0.6} \]

The main use of this equation is in calculating the alveolar to arterial oxygen gradient, described in the next section.

Alveolar-arterial oxygen gradient

Alveolar gas does not completely equilibrate with arterial blood, even in healthy lungs. There is always a small alveolar to arterial oxygen gradient. The gradient \( (P_{A-a\text{O}_2}) \) is calculated by using the alveolar gas equation, above, to calculate the alveolar partial pressure of oxygen, and then subtracting the measured arterial partial pressure of oxygen.

\[ P_{A-a\text{O}_2} = (F_{I\text{O}_2} \times PB) - \frac{P_{a\text{CO}_2}}{0.6} - P_{a\text{O}_2} \]

In normal individuals the gradient should be less than 2 kPa. In the elderly or patients with COPD the difference can be up to 3 kPa. There are three main causes of increased \( P_{A-a\text{O}_2} \):

1. right-to-left shunts
2. ventilation-perfusion inequalities (including pulmonary emboli)
3. diffusion defects

Pulmonary oedema also causes a large gradient, probably by a mixture of the second and third mechanisms.

Mixed Venous Oxygen Saturation

\( S_{v\text{O}_2} \). Normal values of 60 to 80%.

Arterial Oxygen Content

\[ C_{a\text{O}_2} = Hb \times \frac{S_{a\text{O}_2}}{100} \times 1.34 \]

Normal values of 17 to 20 ml/dl.
CHAPTER 16. CARDIAC HAEMODYNAMIC FORMULAE

Mixed Venous Oxygen Content

\[ C_{vO_2} = Hb \times \frac{S_{vO_2}}{100} \times 1.34 \]

Normal values of 12 to 15 ml/dl.

A-V Oxygen Content Difference

\[ C_{(a-v)O_2} = C_{aO_2} - C_{vO_2} \]

Normal values of 4 to 6 ml/dl.

Oxygen Extraction Ratio

\[ O_2ER = \frac{C_{aO_2} - C_{vO_2}}{C_{aO_2}} \]

Normal values of 22 to 30%.

Oxygen Delivery Index

\[ DO_2I = CI \times C_{aO_2} \times 10 \]

Normal values of 500 to 600 ml/min/m\(^2\).

Oxygen Consumption Index

\[ DO_2I = CI \times (C_{aO_2} - C_{vO_2}) \times 10 \]

Normal values of 200 to 250 ml/min/m\(^2\).
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