Appendices

Appendix 1: The Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Glasgow coma scale</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Eye opening</strong></td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To speech</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>Nil</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best motor response</strong></td>
<td></td>
</tr>
<tr>
<td>Obey commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizes to pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdraws to pain</td>
<td>4</td>
</tr>
<tr>
<td>Abnormal flexion to pain</td>
<td>3</td>
</tr>
<tr>
<td>Extends to pain</td>
<td>2</td>
</tr>
<tr>
<td>Nil</td>
<td>1</td>
</tr>
<tr>
<td><strong>Best verbal response</strong></td>
<td></td>
</tr>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>Nil</td>
<td>1</td>
</tr>
</tbody>
</table>
## Appendix 2: Causes of Altered Consciousness

<table>
<thead>
<tr>
<th>Cause of altered conscious level</th>
<th>History and examination</th>
<th>Investigations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Traumatic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Extradural hematoma</td>
<td>History of trauma Classic lucid interval External signs of head injury e.g., skull fracture Focal neurological signs Signs of raised ICP</td>
<td>Abnormal CT</td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td>Acute type as above without lucid interval Chronic type may follow innocuous trauma and have a more insidious onset</td>
<td>Abnormal CT</td>
</tr>
<tr>
<td>Intracerebral hematoma/ contusions/ traumatic subarachnoid hemorrhage</td>
<td>As for extradural hematoma without lucid interval</td>
<td>Abnormal CT</td>
</tr>
<tr>
<td>Diffuse axonal injury</td>
<td>As for extradural hematoma without lucid interval May be few external signs of injury</td>
<td>Abnormal CT-may only become evident &gt;24 h</td>
</tr>
<tr>
<td><strong>Vascular</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>Sudden onset History of headaches Signs of meningism Consider polycystic kidney disease</td>
<td>Abnormal CT (10% are normal) Increased red blood cells/xanthochromia in CSF</td>
</tr>
<tr>
<td>Intracerebral hemorrhage</td>
<td>History of hypertension, coagulopathy or anticoagulants</td>
<td>Abnormal CT</td>
</tr>
<tr>
<td>Thrombo-embolic</td>
<td>Sudden onset Vascular disease, atrial fibrillation</td>
<td>Abnormal CT-may only become evident &gt;48–72 h Echocardiography Carotid Doppler angiography</td>
</tr>
<tr>
<td>Hypertensive crisis</td>
<td>Blood pressure Fundoscopy</td>
<td></td>
</tr>
<tr>
<td>Hypotension/ low cardiac output state</td>
<td>Low blood pressure Evidence of reduced perfusion to other tissues (skin, kidneys)</td>
<td>Cardiac output monitoring Echocardiography</td>
</tr>
<tr>
<td><strong>Infective</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain abscess</td>
<td>Slow onset May be systemically unwell Consider ENT or dental source of infection</td>
<td>Abnormal CT (contrast) Blood cultures</td>
</tr>
<tr>
<td>Meningitis</td>
<td>Hours to days onset Signs of meningism Purpuric meningococcal rash</td>
<td>Abnormal CSF Blood cultures CT may be abnormal</td>
</tr>
<tr>
<td>Encephalitis</td>
<td>Hours to days onset Viral-like illness</td>
<td>CT/ MRI may be abnormal EEG</td>
</tr>
<tr>
<td>Generalized sepsis</td>
<td>May be obvious infective source Signs of SIRS</td>
<td>Raised white cell count/ C-reactive protein</td>
</tr>
<tr>
<td><strong>Metabolic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypo/ hypernatremia</td>
<td>History may be useful Consider medications</td>
<td>Abnormal venous/ arterial blood results ECG changes</td>
</tr>
<tr>
<td>Hypo/ hyperglycemia</td>
<td>Specific features on examination e.g., features of chronic liver disease, breath odor, fistulae</td>
<td></td>
</tr>
<tr>
<td>Hypoxia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypercapnia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypo/ hyperthermia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hepatic failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Endocrine</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Myxedema</td>
<td>Usually apparent from history</td>
<td>Abnormal blood results e.g., thyroid function tests, short synacthen test, more complex endocrine tests</td>
</tr>
<tr>
<td>Adrenal insufficiency</td>
<td>Features of a specific condition e.g., myxoedemic facies, hypothermia, abnormal pigmentation</td>
<td></td>
</tr>
<tr>
<td>Hypopituitarism</td>
<td>Neurological signs of pituitary tumor</td>
<td></td>
</tr>
</tbody>
</table>

(continued)
## Cause of altered conscious level

<table>
<thead>
<tr>
<th>Cause of altered conscious level</th>
<th>History and examination</th>
<th>Investigations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pharmacological</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sedatives</td>
<td>May be apparent from history</td>
<td>Drug/substance levels in blood/urine</td>
</tr>
<tr>
<td>Pupil signs</td>
<td></td>
<td>Improvement after antidote administered</td>
</tr>
<tr>
<td>Needle marks</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breath odor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Narcotics</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pupil signs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Depressed respiration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alcohols</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac arrhythmias</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychotropic drugs</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seizures</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poisons (e.g., carbon monoxide)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Changes in skin color</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Substance withdrawal</td>
<td>Agitation rather than coma with acute withdrawal</td>
<td></td>
</tr>
<tr>
<td>Brain tumor</td>
<td>Usually slow onset</td>
<td>Abnormal CT</td>
</tr>
<tr>
<td>Neoplastic</td>
<td>Consider metastasis</td>
<td></td>
</tr>
<tr>
<td>Focal signs</td>
<td>Raised intracranial pressure</td>
<td></td>
</tr>
<tr>
<td>Paraneoplastic syndromes</td>
<td>Can present as “aseptic meningitis”</td>
<td>Abnormal EEG</td>
</tr>
<tr>
<td>Seizures</td>
<td>Usually typical history</td>
<td>Need CT to exclude underlying lesion</td>
</tr>
<tr>
<td>Nonconvulsive are rare</td>
<td>Consider underlying cause</td>
<td>Anticonvulsant levels</td>
</tr>
<tr>
<td>Demyelination</td>
<td>Disseminated in time and place</td>
<td>Abnormal MRI</td>
</tr>
<tr>
<td>ICU psychosis</td>
<td>No specific features</td>
<td>Diagnoses of exclusion</td>
</tr>
<tr>
<td>Sleep deprivation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pseudocoma</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Catatonia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Psychological/behavioral</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix 3: Initial Stabilization of the Brain Injured Patient on ICU

Initial stabilisation of the brain injured patient on ICU

**VENTILATION**
- FiO₂ 1.0 until ABG
- V₁ 7-10ml/kg
- Freq. 10-12/min
- PEEP 2.5-5cm H₂O
- PaO₂>13kPa
- PaCO₂ 4.5-5.5kPa* (until S₁O₂ available)

**CIRCULATION**
- MAP > 80mmHg
- CPP > 60, if ICP measured*
- Hb >10g/dl
- Maintain adequate circulating volume (e.g. CVP, PCWP, Doppler)
- If hypotensive, check for bleeding
- Consider need for inotropes or vasopressors*

**SEDATION**
- Propofol 1-6mg/kg/hr
- Alfentanil 1-4mg/hr
- Midazolam if unstable
- Consider paralysis
- EEG for Thiopentone coma**

**MONITORING**
- ECG, SaO₂, ETCO₂
- IABP
- CVP/PCWP/Doppler
- Temperature
- ICP & CPP
- S₁O₂, PbtO₂
- EEG

**GENERAL MEASURES**
- 15° Head up tilt, neck neutral
- Check ETT ties, hard collar
- OGT/NGT
- Early enteral feeding
- Metoclopramide if not absorbing
- H₂ blocker/PPI
- Insulin: maintain glucose 4-8mmol/l
- VTE prophylaxis*

**INVESTIGATIONS**
- FBC, Clotting
- U&Es, LTFs
- Glucose, Ca²⁺
- Arterial blood gases
- Group & Save

**FLUIDS***
- Daily Fluid Balance
- 0.9% NaCl maintenance (unless grossly hypernatremic)
- 6% starch (up to 1.5l/day)
- Blood, Hb ~ 10g/dl
- Clotting products (INR, APTT<1.2, platelets >100)

**PYREXIA**
- Culture blood, sputum, urine
- CRP
- CXR, consider BAL
- Paracetamol 1g qds
- +/- NSAIDs**
- Active Cooling
- Consider line change
- Antibiotics*

* see Chapter 3
** discuss with RNC
Appendix 4: Acute Brain Herniation

Warning signs:
- Reduction in conscious level
- Unilateral third Nerve palsy
- Lateralizing motor signs e.g., hemiparesis, extensor posturing
- Hypertension, bradycardia or respiratory irregularity (Cushing’s Triad)

Management
- **Rapid intubation and ventilation** (great care needed to avoid exaggerated pressor response to laryngoscopy and intubation – experienced anesthetist essential. Invasive blood pressure monitoring ideal, but do not delay establishing ventilation)
- **Hyperventilate** to $P_{\text{a}CO_2}$ 3.5–4.0 kPa as a temporary measure
- **Mannitol** 20% 0.5 g/kg over 10 min
- **Sedation** to reduce cerebral metabolic rate (e.g., propofol, thiopentone) supplemented with opioid analgesic (e.g., fentanyl, alfentanil)
- **Head up** position and good neck position to encourage venous drainage
- Maintain adequate MAP (ideally 90–100 mmHg) with pressor. Do not treat hypertension (may reduce cerebral perfusion)
- 100% $O_2$ (*hyperoxia*) may reduce cerebral blood volume and ICP (especially in younger patients) and can be utilized whilst more definitive treatment is sought
- A **CT scan** will be required as soon as the patient is stable enough to be moved to exclude surgical lesions such as hydrocephalus or a hematoma
- Contact RNC for further advice
Appendix 5: Drug Doses

<table>
<thead>
<tr>
<th>Drug</th>
<th>Suggested dose range</th>
<th>Infusion concentration</th>
<th>Dose range (mL/h) for 70 kg patient</th>
<th>Notes</th>
<th>Route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adrenaline (epinephrine)</td>
<td>0.05–1.0 mcg/kg/min</td>
<td>5 mg in 50 mL 0.9% NaCl</td>
<td>2.1–42.0 mL/h (0.05–1.0 mcg/kg/min)</td>
<td>Use for refractory hypotension in low cardiac output states where other agents e.g., dobutamine have been ineffective. Severe tachycardia may be seen.</td>
<td>CVC</td>
</tr>
<tr>
<td>Noradrenaline (norepinephrine)</td>
<td>0.05–1.0 mcg/kg/min</td>
<td>4 mg in 50 mL 5% glucose</td>
<td>2.6–52 mL/h (0.05–1.0 mcg/kg/min)</td>
<td>Predominantly α agonist with some β₁ agonism. More potent than phenylephrine. May mask hypovolemia. Reflex bradycardia may occur.</td>
<td>CVC</td>
</tr>
<tr>
<td>Phenylephrine (Neosynephrine)</td>
<td>1.0–10 mcg/kg/min</td>
<td>50 mg in 50 mL 0.9% NaCl</td>
<td>4.2–42 mL/h (1.0–10 mcg/kg/min)</td>
<td>Useful agent that can be given peripherally whilst central venous access is being established.</td>
<td>Large vein</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>2.5–20 mcg/kg/min</td>
<td>250 mg in 50 mL 0.9% NaCl</td>
<td>2.1–16.8 mL/h (2.5–20 mcg/kg/min)</td>
<td>β₁ agonist used to augment cardiac output. Can be given peripherally whilst central venous access is being established. Tachycardia frequently seen.</td>
<td>Large vein</td>
</tr>
<tr>
<td>Dopamine</td>
<td>2–15 mcg/kg/min</td>
<td>400 mg in 50 mL 0.9% NaCl</td>
<td>1.0–7.5 mL/h (2–15 mcg/kg/min)</td>
<td>Predominant effect depends on dosage. At lower doses acts as β₁ agonist. More α effects at higher doses (&gt;10 mcg/kg/min).</td>
<td>CVC</td>
</tr>
<tr>
<td>Vasopressin</td>
<td>0.01–0.04 units/min</td>
<td>50 units in 50 mL 0.9% NaCl</td>
<td>0.6–2.4 mL/h (0.01–0.04 units/min)</td>
<td>Side effects include reduced CO and hepatosplanchnic blood flow. Doses &gt;0.04 units/min may lead to cardiac arrest.</td>
<td>CVC</td>
</tr>
</tbody>
</table>

NB. These drug infusion regimes are for illustrative purposes. All drug concentrations and infusion rates should be independently verified and, where possible, locally policy established. In all circumstances the circulating volume should be optimized; invasive cardiovascular monitoring may be required.
Appendix 6: Neuro-Surgical Referral of Traumatic Brain Injuries

NEURO-SURGICAL REFERRAL OF TRAUMATIC BRAIN INJURIES

Patient identified in A&E for referral to NSU at LGI or Hull

Fill in patient check list (PTO)

Anaesthetic/A&E doctor to make telephone call to nearest on-call registrar for Neurosurgery

LGI: 0113 243 2799
Mobile: xxxxxxxx

Enter Dialogue — Time: __ : __ hrs

Send scan via image link
Time: __ : __ hrs

NSU will call referring hospital back as soon as possible
Time __ : __ hrs

Patient Declined
Reason

Patient Accepted
NSU is responsible for finding a bed even if none available

Admit locally
Time: __ : __ hrs

Mass lesion requiring urgent surgery

Urgent surgery not required

Refer after 24 hrs if concerned, deteriorates or fails to improve

Call 999, transfer without delay

Optimise for transfer

Leave for NSU __ : __ hrs

Call NSU with ETA

Take patient to ______________________ at LGI

Time of arrival: __ : __ hrs

Name of Person Completing Form ______________________

Signature ______________________ Date ______________________

Time of CT Request
Time of CT Scan
Time of CT Results

Avoid secondary cerebral insult
Maintaining cerebral oxygen delivery
Controlling cerebral oxygen consumption
Avoid increases in intracranial pressure
See Neuro Care Bundle

Indications for Manitol
Unilateral pupillary dilatation
Unilateral progressing to bilateral dilatation (primary bilateral dilatation may represent fitting, drug intoxication or overdose, or overwhelming brain injury).

Dose: 0.5 gm/kg (approximately 200 mls of 20% solution in adults) over five minutes
Must be catheterised

Monitoring during ventilated transfers

a. ECG
b. Direct arterial and NIBP
c. SaO2
d. EtCO2 (calibrated against PaCO2)
e. Temperature
f. Urinary catheter
g. Pupillary size and reaction

Call WYMAS/TENYAS
Critical Care Transfers Dedicated Call Line for WYMAS-01924 834515
TENYAS – Phone 999

REFERRING HOSPITAL SHOULD ONLY NEED TO MAKE ONE PHONE CALL TO NSU
# Appendix 7: Referral Checklist – for Traumatic Brain Injured Patients to Neuro Centre

**REFERRAL CHECKLIST – FOR TRAUMATIC BRAIN INJURED PATIENTS TO NEURO CENTRE**

<table>
<thead>
<tr>
<th></th>
<th>Referring Doctor:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Referring Consultant:</td>
</tr>
<tr>
<td></td>
<td>Referring Hospital:</td>
</tr>
<tr>
<td>2</td>
<td>Time of Call:</td>
</tr>
<tr>
<td>3</td>
<td>Patients Details:</td>
</tr>
<tr>
<td></td>
<td>Name</td>
</tr>
<tr>
<td></td>
<td>DOB</td>
</tr>
<tr>
<td></td>
<td>Sex</td>
</tr>
<tr>
<td></td>
<td>Age</td>
</tr>
<tr>
<td>4</td>
<td>Injury mechanism e.g. RTA / Assault etc:</td>
</tr>
<tr>
<td>5</td>
<td>Time of Injury:</td>
</tr>
<tr>
<td>6</td>
<td>GCS, pupils and time of arrival on scene:</td>
</tr>
<tr>
<td></td>
<td>Time</td>
</tr>
<tr>
<td></td>
<td>Pupils</td>
</tr>
<tr>
<td></td>
<td>Eye Opening</td>
</tr>
<tr>
<td></td>
<td>Motor Response</td>
</tr>
<tr>
<td></td>
<td>Verbal Response</td>
</tr>
<tr>
<td>7</td>
<td>GCS, pupils and time of arrival at A&amp;E:</td>
</tr>
<tr>
<td></td>
<td>Time</td>
</tr>
<tr>
<td></td>
<td>Pupils</td>
</tr>
<tr>
<td></td>
<td>Eye Opening</td>
</tr>
<tr>
<td></td>
<td>Motor Response</td>
</tr>
<tr>
<td></td>
<td>Verbal Response</td>
</tr>
<tr>
<td>8</td>
<td>GCS, pupils at time of call:</td>
</tr>
<tr>
<td></td>
<td>Time</td>
</tr>
<tr>
<td></td>
<td>Pupils</td>
</tr>
<tr>
<td></td>
<td>Eye Opening</td>
</tr>
<tr>
<td></td>
<td>Motor Response</td>
</tr>
<tr>
<td></td>
<td>Verbal Response</td>
</tr>
<tr>
<td>9</td>
<td>Any treatment given? e.g. intubated/ ventilated</td>
</tr>
<tr>
<td>10</td>
<td>Current vital signs: P, BP, SaO₂:</td>
</tr>
<tr>
<td>11</td>
<td>Other significant injuries and past medical history:</td>
</tr>
<tr>
<td>12</td>
<td>Is patient on Warfarin, Asprin or Clopidogrel?</td>
</tr>
<tr>
<td>13</td>
<td>Referral Clinician Tel No &amp; Ext No:</td>
</tr>
<tr>
<td>14</td>
<td>Name of Neuro SpR spoken to:</td>
</tr>
<tr>
<td>15</td>
<td>Time of first contact with Neuro SpR:</td>
</tr>
<tr>
<td>16</td>
<td>Outcome of call - comments:</td>
</tr>
</tbody>
</table>

**USEFUL TELEPHONE NUMBERS:**

| LGI Switchboard: | 0113 243 2799 |
|                 | LGI Neuro ITU Tel: | 0113 392 7106 |
| (Ask to page Neuro-Sciences Registrar) | LGI Neuro ITU Fax: | 0113 392 7306 |

**Prior to transfer fax both sides of form to lgi neuro icu on 0113 392 7306 even if patient is declined**
Appendix 8a

A patient in cardiogenic or septic shock is resuscitated and subsequently managed on an Intensive Care Unit to attain a blood pressure at the lower threshold of autoregulation for that individual's organs. A systolic blood pressure of 80–90 mmHg is often accepted, provided the cerebral, renal, and myocardial perfusions are adequate, as demonstrated by the patient remaining lucid (if conscious), having an adequate urine output and no evidence of myocardial ischemia. The notion of striving to attain supra-physiological values for cardiac output and oxygen delivery has long been discarded. Similarly, for a patient with ARDS, we “permit” a degree of hypercapnia and hypoxia with a strategy of lung-protective ventilation rather than striving to normalize physiological parameters.

This is where Neurocritical Care differs from General Intensive Care. It is implicit, when employing an ICP directed protocol for the management of intracranial hypertension, that mean arterial pressure is maintained at values which exceed what would normally be considered adequate in a general ICU patient because cerebral perfusion must be maintained. To use an analogy from ATLS, dysfunction is given primacy over breathing and circulation. Therefore, patients are often overdosed on sedatives in order to achieve burst suppression, whilst being aggressively ventilated to achieve ‘normal’ arterial tensions of CO₂, and driven (often in the face of a relatively depleted intravascular compartment secondary to loop and osmotic diuretic use) to achieve a blood pressure that exceeds normal renal and cerebral autoregulation. CPP goal directed protocols have been shown to have higher incidences of lung related complications (Robertson 1999; Contant 2001) in their treatment arms. We have witnessed young patients suffer myocardial ischemia, cardiac arrests and myocardial deaths with aggressive ICP targeted therapies.

Therefore, although we present schematics for the management of raised intracranial pressure and inadequate cerebral perfusion pressure, caution must be exercised in slavishly following such protocols. Increasingly, management is becoming more tailored to the individual patient; measuring adequacy of cerebral oxygenation may allow lower threshold cerebral perfusion pressures and more rationally set PₐCO₂ levels to be targeted, thereby avoiding iatrogenic morbidity. Eventually, treatment protocols may become sophisticated enough to distinguish between subsets of patients who will benefit from a CPP directed approach (i.e., those who are autoregulating) verses a Lund approach (i.e., failure of autoregulation where primacy must be given to minimizing vasogenic edema).

References


Appendix 8b: Flow Diagram for the Management of Raised Intracranial Pressure

**LEVEL 1 - SIMPLE MEASURES**
- 15-20° head up tilt
- Neutral neck position
- No venous obstruction
- Ensure reliable ICP trace
- EVD open if present
- Ensure adequate sedation
- Consider paralysis
- Ensure adequate CPP (~60)
- PaCO₂ 4.5-5.0kPa
- Exclude seizure activity
- Treat pyrexia (aim for normothermia)
- Do not stop statin
- Avoid over-hydration

**LEVEL 2 - FAILURE OF SIMPLE MEASURES**
- Hypertonic saline 20% 30ml, or
- Mannitol 0.5g/kg
- Consider furosemide 20mg IV in addition
- Repeat CT brain scan
- Contact surgeons for urgent review
- Insert S_p,O_2 catheter

**LEVEL 3 - ICP PERSISTENTLY > 25mmHg Failing to Respond to Level 1 & 2 Measures**
- Contact ICU Consultant
- Consider further osmotherapy (if Na⁺ < 160)
- Consider trial of thiopentone with EEG guidance
- Hyperventilation only as a temporary measure with S_p,O_2 in place
- Hypothermia (cool to < 35°C)
- Early surgical review and consideration of decompressive surgery (continue maximal medical therapy until time of surgery)
- Rescue ICP Trial if appropriate

**EVIDENCE OF IMPENDING BRAIN HERNIATION**
- PaCO₂ 3.5-4.0 temporarily
- Sedation boluses*
- Paralysis
- Ensure adequate CPP
- Consider 100% O₂ (hyperoxia)
- Urgent review by senior ICU and surgical staff

* sedation boluses, including thiopentone and midazolam, preferably with processed EEG guidance
Appendix 8c: Flow Diagram for Hemodynamic Management in the Context of Raised ICP in Adult Patients

** Noradrenaline (norepinephrine): 0.05-1.0 mcg/kg/min via CVC  
Phenylephrine (neosynephrine): 1.0-10 mcg/kg/min  
Dobutamine: 2.5-10 mcg/kg/min  
Dopamine: 2-15 mcg/kg/min  
Adrenaline (epinephrine): 0.05-1.0 mcg/kg/min
Appendix 9: Stages and Treatment of Tonic–Clonic Status Epilepticus

- Airway, Breathing, Circulation, Disability
- Check blood glucose
- Give thiamine or pyridoxine if appropriate

Pre-status: A phase of escalating seizures lasting hours or days.
- Buccal Midazolam (5–10 mg) or oral Clobazam (10–20 mg/day)

Early status: Seizure or serial seizures lasting up to 30 min.
Use one of the following IV benzodiazepines. 65% chance of terminating SE.
- Lorazepam (first choice): 2–4 mg; long duration of action, recurrent seizures less likely
- Midazolam: 0.05–0.2 mg/kg; short action, rapid metabolism, best choice for continuous benzodiazepine infusion

NB. Doses may need to be reduced in the elderly. Additional therapy must be started at this point to prevent further seizures.

Established status: 30–60 min
- Phenytoin 15–20 mg/kg IV @ 50 mg/min, or
- Fosphenytoin 15–20 mg/kg IV /IM @ 150 mg/min

NB Both require continuous ECG monitoring
If seizures continue, additional phenytoin or fosphenytoin 5–10 mg/kg and check levels.

Refractory status: Seizures lasting >1 h
Several options: ICU care required for ventilatory support and invasive monitoring
Use continuous EEG monitoring if available
- Propofol: 2 mg/kg bolus, 150–200 mcg/kg/min infusion, or
- Thiopental: 5–10 mg/kg bolus, 1–10 mg/kg/h infusion, or
- Midazolam: 0.2 mg/kg bolus, 0.1–0.2 mg/kg/h infusion
- Valproate: 400–800 mg/kg IV bolus may be added (if phenytoin levels ok) Levetiracetam is gaining popularity as adjunctive therapy and is available in both oral IV preparations.

NB: deep sedation is recommended for at least 12 h before reducing and looking for evidence of seizure activity, ideally using an EEG for guidance. Ensure adequate levels of anticonvulsants for chronic seizure control. Hemodialysis may be helpful in cases of drug-induced status (especially antibiotics, theophylline).
If seizures continue after a period of deep sedation despite adequate anticonvulsant drug levels, additional agents such as Phenobarbital may be added.
Appendix 10

**Glasgow Outcome Score**

GOS=1 (Good Recovery)

Capacity to resume normal occupational and social activities, although there may be minor physical or mental deficits or symptoms.

GOS=2 (Moderate Disability)

Independent and can resume almost all activities of daily living. Disabled to the extent that they cannot participate in a variety of social and work activities.

GOS=3 (Severe Disability)

No longer capable of engaging in most previous personal, social or work activities. Limited communication skills and have abnormal behavioral or emotional responses. Typically are partially or totally dependent on assistance from others in daily living.

GOS=4 (Persistent Vegetative State)

Not aware of surroundings or purposely responsive to stimuli.

GOS=5 (Dead)

**Rankin Disability Score**

Rankin=0 No symptoms at all.

Rankin=1 No significant disability despite symptoms; able to carry out all usual duties and activities.

Rankin=2 Slight disability. Unable to carry out all normal activities but able to look after own affairs without assistance.

Rankin=3 Moderate disability requiring some help but able to walk without assistance.

Rankin=4 Moderately severe disability. Unable to walk without assistance, and unable to attend to own bodily needs without assistance.

Rankin=5 Severe disability. Bedridden, incontinent and requiring constant nursing care and attention.
Appendices

Appendix 11: Management of a Fall in GCS after Subarachnoid Haemorrhage

Fall in GCS more than 2 points or 1 point in the motor score

1. **Airway, Breathing, Circulation** - safety first
2. Intubate and ventilate if GCS < 8
   - Optimise oxygenation with PEEP
   - Normalise PaCO₂ (4.5-5.0kPa) unless “coning” where PaCO₂ can be reduced to 3.5-4.0kPa as a *temporary measure* only
3. Always check **blood sugar**
4. Full clinical examination - consider the possible causes:
   - **Neurological**, e.g. seizures, re-bleeding, hydrocephalus, vasospasm, cerebral oedema
   - **Non-neurological** e.g. hypoxia, MI, PE, pyrexia, ↓[Na⁺], acute abdomen
5. Investigations: ECG, CXR, FBC, U&Es, Clotting, Mg²⁺
6. Once stable, CT brain scan

**CT SCAN CHANGED**
Re-bleed, Infarction, worsening oedema, hydrocephalus

- Contact Regional Neurosurgical Centre
- Transfer to RNC
- Re-assess off sedation
- Clinical Improvement
- Poor Clinical Condition
- Ongoing DGH Management
- Definitive treatment of aneurysm at RNC after 14 days if good neurological condition

**CT SCAN UNCHANGED - LIKELY CEREBRAL VASOSPASM**

- See Figure 4.5
- Withdraw Support
  - See Editorial Note, Chapter 4
Appendix 12: Management of Vasospasm (NB Diagnosis of Exclusion)

Cerebral Vasospasm

Non-specialist Centre
Aneurysm unprotected

Specialist Neurosurgical Center

Aneurysm untreated
Aneurysm protected i.e. coiled or clipped

General Measures
- IABP, CVP monitoring
- Determine baseline SBP, CVP
- Urinary catheter, fluid balance
- NG tube
- Nimodipine PO/NG
- Treat pyrexia (paracetamol)
- Treat hyperglycemia (insulin sliding scale)
- DVT prophylaxis (e.g. compression stockings, calf compression devices)

Specific measures to mitigate vasospasm
- Close observation on ICU or HDU
- Careful fluid balance
  - 0.9% NaCl maintenance fluid (minimum 3 litres per day)
  - Maintain CVP > 8mmHg
  - 250ml colloid boluses as required
  - observe trends and clinical response
  - achieve daily 500ml positive fluid balance
  - allow for insensible losses
- Maintain modest hypertension
  - SBP < 160mmHg in previously normotensive patients
  - Vasoactive agents if normovolemic and hypotensive (but keep SBP < 160mmHg)
- Provide adequate analgesia

Triple HHH Therapy
- Positive fluid balance (may require > 6 litres/24h)
- Keep Hb ~ 10g/dl
- Increase MAP 20% above baseline levels (MAP > 120mmHg may be required)
- Cerebral salt wasting may occur. Maintain [Na⁺] with hypertonic NaCl (e.g. with 1.8% NaCl, see chapter 9) and fludrocortisone therapy.

Additional consideration should be given to endovascular options (e.g. angioplasty), magnesium infusion, lumbar CSF drainage, statin therapy and stellate ganglion blockade.
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