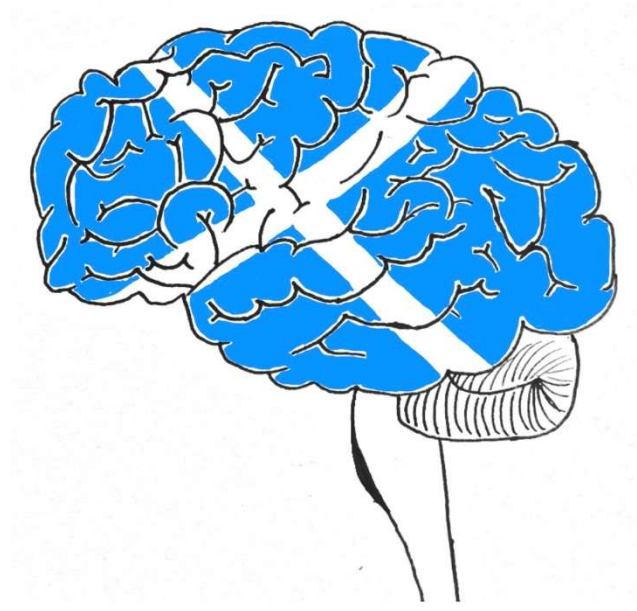




One Brain - Scotland

Course Manual

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IMPORTANT: This manual provides general advice to trained anaesthetists who should at all times exercise their clinical judgement about the most appropriate clinical management for an individual patient. Local variation in practice does occur and it is always best practice to discuss any concerns with senior help.

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Principles of Neuroanaesthesia

General aims of neuroanaesthesia should include:

1. Avoiding secondary cerebral insult

- Avoid obstructing venous return
- Maintain $\text{PaO}_2 > 13\text{kPa}$
- Maintain PaCO_2 at 4.5 – 5.0 kPa
- Maintain CPP at 60 – 70 mmHg
- Normoglycaemia (6 – 10 mmol/L)
- Maintain normothermia ($36^\circ - 37^\circ$)

2. Maintenance of CPP

3. Avoidance of rises in ICP

4. To facilitate swift recovery from anaesthesia to assess neurological status

Pre-operative

Take a standard anaesthetic history and examination and review relevant investigations (including CT, MRI). Particular attention should be paid to:

- **The risk of aspiration.** A raised ICP is associated with nausea and vomiting and the presence of either of these features, in addition to reflux and non-fasted state, may necessitate an RSI.
- **The presence of cervical spine instability.** Post-trauma or in cases of cervical decompression the cervical spine may be unstable. In non-trauma cases discuss with the patient their range of their neck movement and the presence of any nerve root symptoms. Review relevant imaging and discuss any concerns with the surgeon before determining a strategy for airway managements. This may involve minimal force laryngoscopy with either direct, or video, laryngoscopy or awake fiberoptic techniques. The use of **manual in-line stabilisation** (MILS) techniques is contentious, with most evidence now suggesting that it makes laryngoscopy more difficult and there is a low risk of additional damage to the cervical spine. Discuss with your local department about the consensus opinion regarding the use of MILS if you have concerns about this in practice.
- **The presence of raised ICP.** Assess for symptoms or signs of raised ICP. Imaging may also provide additional information with the appearances of “a tight brain”. If present particular care must be taken to avoid rises in ICP, for example during laryngoscopy.

Symptoms and signs of a raised ICP ¹

Early Signs

- Headache
- Nausea and vomiting
- Seizures
- Papilloedema
- Focal neurology

Late Signs

- Decreased GCS
- Hypertension
- Bradycardia
- Abnormal respiration
- Pupillary dilatation
- Decorticate posturing (leg extension, arm flexion)
- Decerebrate posturing (leg and arm hyperextension)

- **Neurological status.** Assess and document neurological status pre-operatively. This should include GCS, pupillary reflexes, the presence of any cranial nerve or sensory deficit and any motor weakness.

Finally:

- **Discuss with the surgeon.** Discuss the patient position intra-operatively, the need for dexamethasone or anti-epileptic administration, the estimated blood loss and likely duration of the operation.
- **Determine post-operative destination.** Determine post-operative destination (ward, HDU, ITU) based on co-morbidities, pre-morbid status and anticipated post-operative status.
- **Discuss with the patient.** Discuss the anaesthetic plan, the use of invasive monitoring where intended, positioning (if prone, park-bench), post-operative analgesia and anticipated post-operative destination with the patient. Answer all questions where possible.

Peri-operative

- Determine the need for invasive monitoring and whether this needs to be sited pre-induction, for example significant cardiac co-morbidity. “Surgical” indications for invasive blood pressure monitoring include risk of significant blood loss and requirement for intra-operative ABG analysis.
- Factors such as a decreased GCS, the risk of aspiration, the need to control PaCO₂, a remote airway, prolonged operating time and patient positioning often mean that intubation with an endotracheal tube is the norm.
- Whether RSI or not, aims of induction should be to optimise intubation conditions, whilst producing minimal haemodynamic disturbance and avoiding a rise in ICP caused by laryngoscopy.
- Whilst there are numerous described methods of obtunding the pressor response to laryngoscopy, e.g. lidocaine, 1mg/ kg, the most common method is to use propofol together with an appropriate dose of opiate ²:
 - Fentanyl 2 - 3 mcg / kg
 - Alfentanil 7.5 – 10mcg / kg
 - Remifentanil 0.5mcg / kg / min
- Having confirmed intubation secure the airway carefully (tape and two “trouser-legs”). Avoid obstructing venous return.
- Avoid hypotension post-induction. In cases of known or suspected raised ICP classic teaching is to maintain MAP > 80mm Hg. The recently updated Brain Trauma Foundation Guidelines suggest maintaining SBP ≥ 100mm Hg (for patients aged 50 – 69) or at ≥

110mm Hg for patients 15 – 49 or over 70. It is likely these values will be similar – the key remains to avoid hypotension.⁵

- In certain cases, (e.g. blown pupil) it may be more appropriate, where the ICP is unknown, to maintain MAP at pre-induction values if there was significant hypertension. If and when the ICP is known maintain CPP at 60 – 70mmHg.
- Either volatile (isoflurane, sevoflurane, desflurane) or propofol infusion can be used to maintain anaesthesia. Nitrous oxide and halothane should be avoided as they can significantly increase CBF and ICP.
- Remifentanyl infusion can be used together with either volatile or TIVA technique. The advantages are a reduced anaesthetic requirement, a more rapid recovery from anaesthesia and the suppression of the cough reflex.
- The latter effect means that, with care, the patient in Mayfield pins or in the prone position can be anaesthetised without further neuromuscular blockade. If remifentanyl infusion is not employed these patients should receive subsequent doses of neuromuscular agent as guided by peripheral nerve stimulator monitoring.
- Ensure large-bore IV access.
- Site oropharyngeal temperature probe and urinary catheter (if required).
- Transfer the patient to the operating theatre and position them on the operating table. Ensure circuit connections are secure before draping.
- Continue maintenance and begin IPPV - either pressure or volume control can be used.
- If an arterial line is present obtain an ABG sample and use it to calibrate the ETCO₂ to the PaCO₂. Target a PaCO₂ of 4.5 - 5kPa and a PaO₂ > 13 kPa.

- Ensure the patient is wearing TEDS and apply Flowtrons. Connect the fluids via a fluid warmer and apply a patient-warming blanket.
- If the patient is to be placed in Mayfield pins, ensure adequate depth prior to pinning (e.g. increasing remifentanyl to 0.5 mcg / kg / min or TCI 5 – 6 ng/ml for duration of pinning).
- Intravenous fluids should be used with the aim of maintaining normovolaemia and ensuring adequate end-organ perfusion. Excessive fluid administration can worsen cerebral oedema. Simple markers such as urine output, base deficit and serum lactate are useful in determining adequate end-organ perfusion.
- If remifentanyl infusion has been used, additional morphine, fentanyl or oxycodone can be administered towards the end of the procedure.
- Analgesic requirements are often not significant. Following closure, the surgeon will administer local anaesthetic to the incision site and this, together with intravenous paracetamol and a small amount of strong opiate is often all that is required. Avoid NSAIDs as these may interfere with platelet function.
- If extubation is planned extubate when the usual criteria for extubation are met – the presence of airway reflexes, spontaneous respiration and obeying commands. The latter criteria are particularly important if pre-operative GCS was reduced or post-operative GCS is expected to be reduced.

Post-operative

- Transfer the patient to recovery. Include pre-operative GCS and the presence of any pre-existing focal neurology as part of the handover to recovery staff.
- The presence of new neurological signs or a persistently reduced GCS should prompt urgent neurosurgical review to assess for post-operative bleeding.
- Post-operative analgesic requirements are often not significant. Prescribing regular paracetamol, regular codeine and PRN oramorph or oxycodone is often sufficient. Occasionally patients may require a PCA.
- Continue fluid strategy as before – aiming to restrict excessive fluid administration whilst preserving end-organ perfusion.

Airway Management in Traumatic Brain Injury

- Indications for intubation post head-injury are listed in the text box below.

Indications for intubation Post-Brain Injury^{1, 3, 27}

Airway

- Loss of airway reflexes
- Significant bleeding into the airway

Breathing

- Hypoxia – $\text{PaO}_2 < 13 \text{ kPa}$
- Hypercarbia – $\text{PaCO}_2 > 6 \text{ kPa}$
- Spontaneous hyperventilation ($\text{PaCO}_2 < 4 \text{ kPa}$)

Disability

- $\text{GCS} \leq 8$
- Seizures

Other

- To facilitate CT scanning
- Before transfer to a Neurosurgical Unit AND:
 - Significantly deteriorating conscious level (e.g. fall in GCS of ≥ 2 points, or a fall in motor score of ≥ 1 point)
 - Bilateral fractured mandible

- If the cervical spine has not been cleared or spinal injury is suspected, minimal force laryngoscopy should be utilised. The recommendation from the Difficult Airway Society is that MILS makes intubation more difficult, and the risk of cervical injury is low (although the evidence is low grade)³⁰.

- The dose and type of induction agent(s) chosen should be selected with the aims of rapidly securing the airway with minimal haemodynamic disturbance and minimal rise in ICP.
- Where possible siting an arterial line pre-induction can allow more rapid detection and treatment of hypotension.
- In the uncomplicated airway a modified RSI using a reduced dose of thiopentone or propofol together with an opiate (alfentanil, fentanyl) and neuromuscular blocking agent (suxamethonium or rocuronium) can be used.
- A vasopressor (e.g. metaraminol) should be drawn up to treat any hypotension.
- The use of ketamine in Traumatic Brain Injury is contentious. Ketamine has been thought to cause a rise in ICP and previously avoided in the head injured patient, as well as other areas of Neuroanaesthesia. The evidence for this is weak however – there is no clear evidence that it increases ICP or causes harm in brain injury.
- Ketamine has a more favourable haemodynamic profile than propofol and has gained widespread usage in polytrauma patients, including those with head injuries, both in a Pre-Hospital and Emergency Department setting.
- The decision whether or not to use ketamine should be determined considering several factors including haemodynamic state, operator familiarity with the drug and the presence or absence of extra-cranial injuries.
- Secure tube well (two trouser-leg tapes as a minimum) and **ensure venous return is not obstructed by this.**

Top 10 Ways to Optimise the Brain Injured Patient

1. Ventilate to maintain PaCO₂ at 4.5 – 5.0 kPa

- Cerebral blood flow (CBF) is linearly related to PaCO₂. It increases by 2 – 4% per 1 mmHg rise in PaCO₂ (15 – 30% per 1kPa rise in PaCO₂)⁴
- Hyperventilation, although reducing CBF and ICP, also reduces cerebral perfusion and may worsen ischaemia. It can be used as a temporising measure in the treatment of raised ICP. However, the optimal degree of hyperventilation remains unclear.^{3,5}
- **AAGBI guidelines recommend hyperventilation (PaCO₂ 4.0 – 4.5 kPa) if clinical or radiological evidence of raised ICP.**³
- Current Brain Trauma Foundation (BTF) guidelines recommend against prolonged prophylactic hyperventilation below a PaCO₂ of 25 mmHg (≈3.3 kPa).⁵
- Calibrate ETCO₂ to PaCO₂ by arterial blood gas analysis.

2. Avoid hypoxia

- Hypoxia, defined as SaO₂ < 90% or PaO₂ < 60 mmHg (8 kPa) increases morbidity and mortality from severe traumatic brain injury.⁵
- **Maintain PaO₂ > 13 kPa**
- Whilst use of PEEP will increase ICP to a degree, hypoxia is more likely to have a detrimental effect on patient outcome.

3. Avoid hypotension / Maintain CPP

- Hypotension increases morbidity and mortality in severe traumatic brain injury (TBI).
- In severe TBI a single episode of hypotension (SBP < 90 mmHg) was shown to double mortality versus a matched group of patients.⁶
- **Maintain CPP at 60 – 70 mmHg**
- Current Brain Trauma Foundation Guidelines recommend a Cerebral Perfusion Pressure (CPP) of 60 – 70 mmHg.⁵

- **CPP = MAP – ICP**

- Classically a MAP > 80 was targeted to achieve a CPP > 60 – unmeasured ICP in patients is assumed to be 20 mmHg.
- Current Brain Trauma Foundation Guidelines recommend: ⁵
 - **SBP ≥ 100 mmHg for patients aged 50 – 69.**
 - **SBP ≥ 110 mmHg for patients aged 15 – 49 or ≥ 70.**
- A vasopressor may be required (metaraminol, noradrenaline) to maintain MAP and offset the hypotensive effect of anaesthesia.

4. Avoid excessive fluid administration

- Consider carefully fluid strategy with an aim to maintain normovolaemia.
- Aim to achieve a balance of maintaining end-organ perfusion without worsening cerebral oedema through excessive fluid administration.
- With increased sodium content, 0.9% Normal Saline (Na⁺ 154 mmol/ l) is often used, Hartmann's solution (Na⁺ 131 mmol/ l) or Plasmalyte (Na⁺ 140 mmol/ l) are also used.
- Avoid hypotonic fluids (e.g. 5% dextrose).

5. Treat raised ICP promptly

- Without an ICP monitor the first signs of raised ICP may be “late signs” of hypertension, bradycardia, and pupillary dilatation.
- Therapeutic strategies are discussed in detail later but may include surgical treatment, osmotherapy, furosemide, cooling and paralysis.

6. Maintain normoglycaemia

- Hypoglycaemia and hyperglycaemia can worsen the injury.
- **Aim to maintain normoglycaemia. Blood glucose 6 – 10 mmol L**

7. Promptly treat any observed seizures

- Clinically observed seizures may be seen in up to 12% of patients following severe TBI. ⁵
- Seizure activity is associated with increased cerebral metabolism and can worsen secondary injury.

- Anti-epileptic drugs, phenytoin or levetiracetam, are used to reduce the incidence of early post-traumatic seizures (within 7 days of injury). ⁵

8. Maintain normothermia (36° – 37°)

- Cerebral Metabolic Rate (CMR) is related to body temperature – at 37°C for every 1°C fall in body temperature CMR decreases by 7%. ⁴
- Hyperthermia causes increased CMR and should be avoided. The evidence concerning hypothermia is more complex:
 - Hypothermia may be neuroprotective – reducing CMR and ICP. It has been used prophylactically, prior to the development of raised ICP, and therapeutically, to treat refractory raised ICP. ^{5,11}
 - A recent meta-analysis has suggested hypothermia may cause worse outcomes. Trauma patients, including those specifically with TBI, admitted with accidental hypothermia were observed to have increased mortality (OR 2.38, 95% CI 1.53 – 3.69). ²⁸
 - The BTF Guidelines state early (< 2.5 hours), and short-term (<48 hours) prophylactic hypothermia is not recommended to improve outcome. ⁵
 - The role for therapeutic hypothermia remains unclear – management algorithms often include mild hypothermia (34° – 35°C) as an option for treating refractory intracranial hypertension. ¹¹

A pragmatic view of the available evidence would be to maintain normothermia in elective neurosurgical patients and emergency neurosurgical patients on admission. Therapeutic hypothermia is likely reserved as a treatment option in selected cases with refractory intracranial hypertension on ICU.

9. Ensure optimal venous drainage

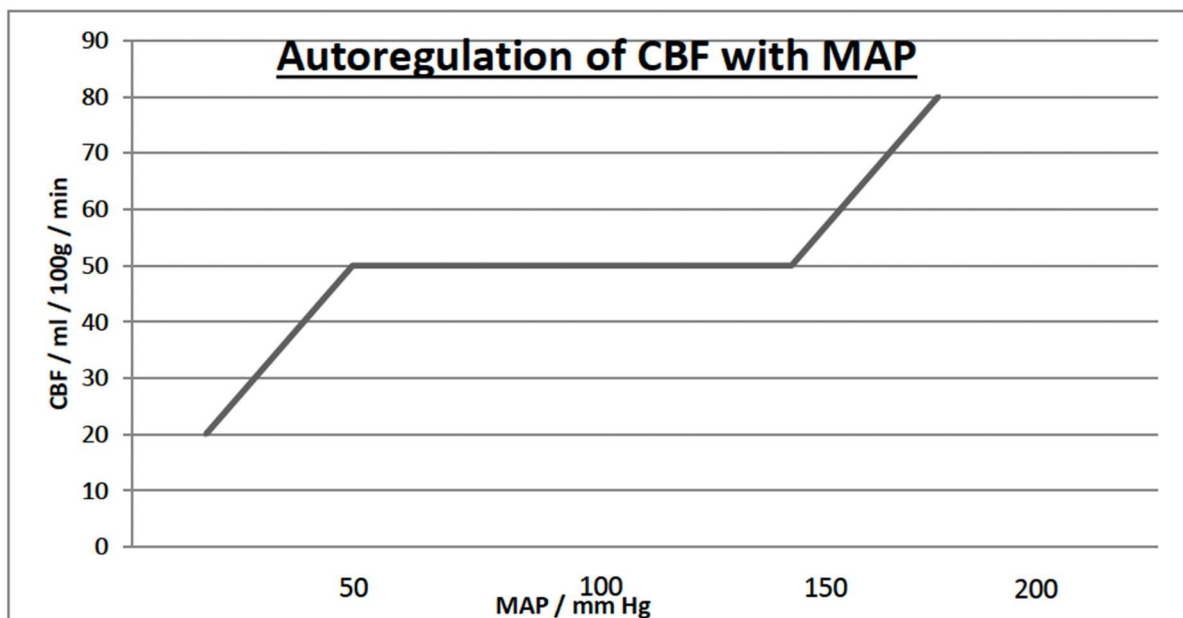
- Avoid obstruction to venous drainage (twisted neck position, tightly tying ETT tubes) and position 15° – 30° head up.

10. Identify any other serious injuries

- A systematic evaluation of the patient is essential to identify any other life-threatening injuries that may take priority over the head injury (e.g. tension pneumothorax, ruptured spleen)

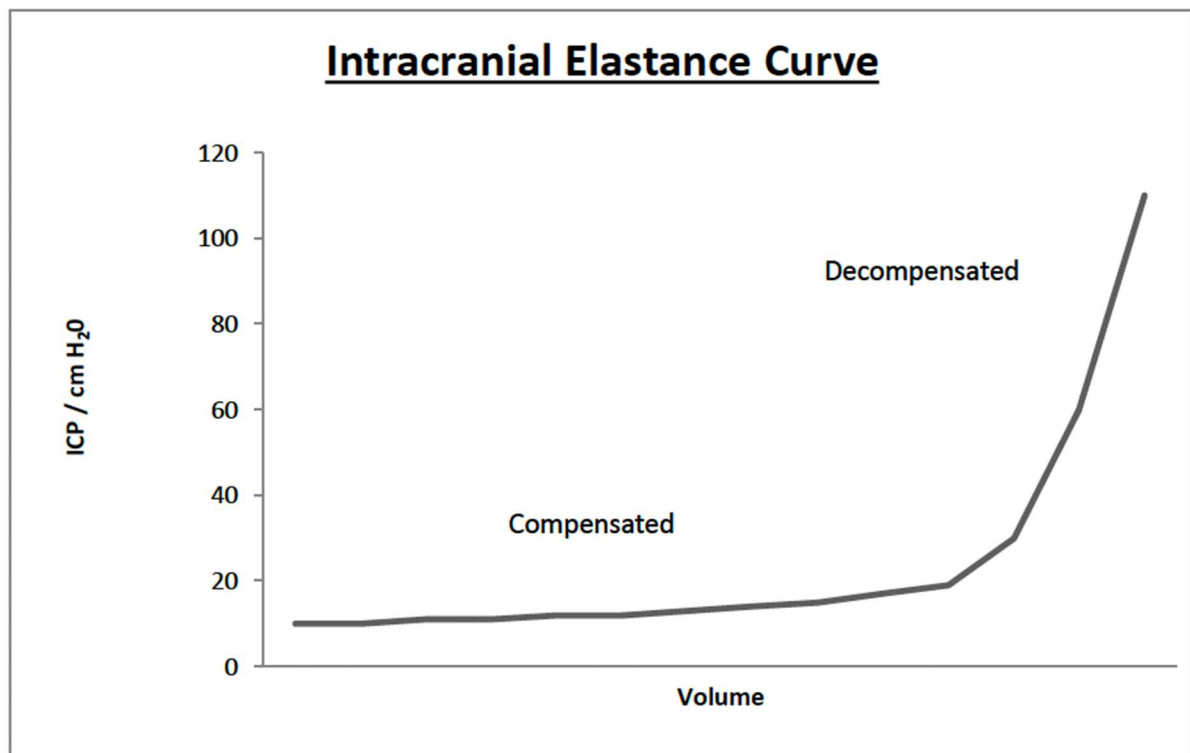
Neurophysiology

- As with all anaesthesia a key principle in neuroanaesthesia is maintaining the delivery of oxygenated blood to the target end-organ – the brain. Ischaemia occurs when the perfusion of brain tissue does not meet its metabolic demands.
- Normal CBF is $\approx 50\text{ml}/100\text{g}/\text{min}$. Autoregulation ensures this is maintained over a wide range of perfusion pressure (MAP $\approx 50 - 150\text{ mmHg}$)⁴

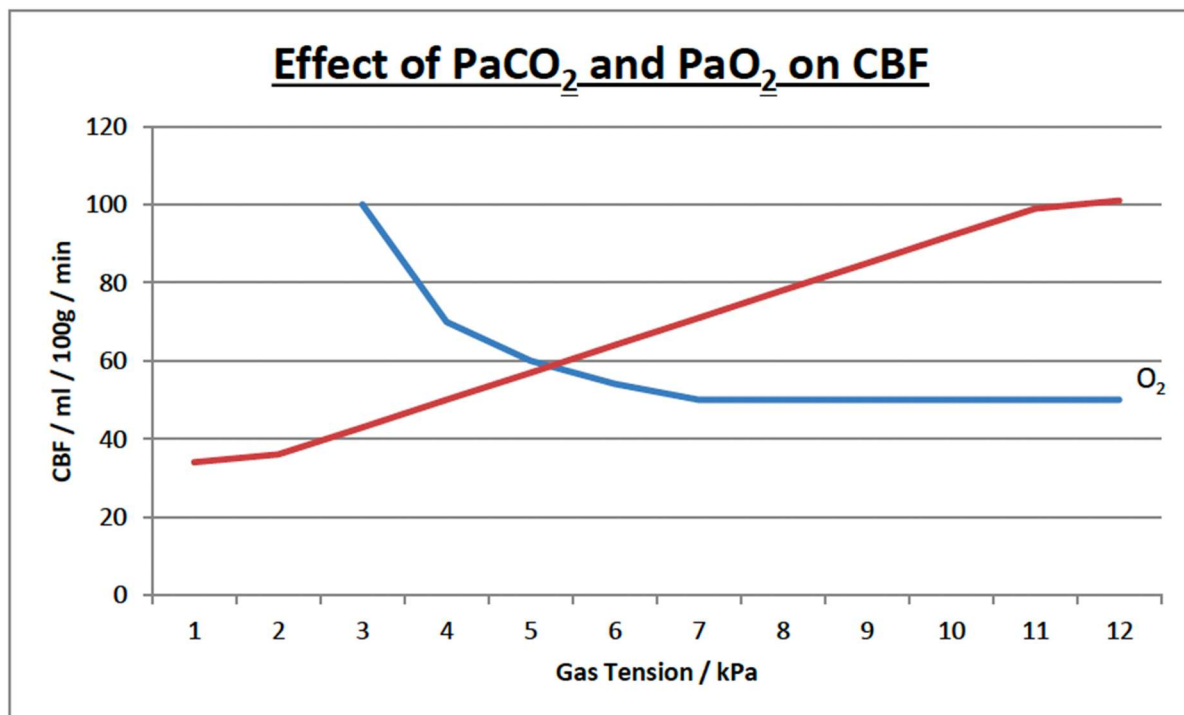


- $CPP = (MAP - JVP) - ICP$. (Often simplified to: $CPP = MAP - ICP$)
- Factors that impair CPP can be divided into those that lower the MAP and those that raise the ICP. Those lowering the MAP include:
 - Hypovolaemia
 - Vasodilatation
 - Impaired contractility

- Normal ICP is 5 – 15 mmHg. Classically treatment was initiated when ICP exceeds 20 mmHg. The recent Brain Trauma Foundation Guidelines state an ICP of 22 mmHg as being associated with increased mortality.⁵
- The contents of the normal cranium include: brain tissue ($\approx 1500\text{g}$), blood (100 – 150ml) and CSF ($\approx 120\text{ml}$), extracellular fluid ($<100\text{ml}$).⁷
- The Monroe-Kellie doctrine states that the rigid cranium has a fixed volume. If ICP is to be maintained, an increase in volume of one of the contents of the cranium must lead to an opposite decrease in the volume of another of the cranial contents.^{4,8}
- This compensation is limited – only 100 – 150ml of additional volume can be tolerated before the ICP cannot be maintained⁹. The intracranial pressure-volume curve is illustrated below.^{4,8,9}



- Factors that can cause a rise in ICP include:
 1. Increased tissue volume – e.g. brain tumour.
 2. Increased blood volume – e.g. intracranial haemorrhage
 3. Increased CSF volume – e.g. congenital hydrocephalus
 4. Increased extracellular fluid volume – e.g. cerebral oedema.
- Changes in CBF can cause significant increases in blood volume and therefore ICP.
- CO₂ can affect CBF significantly – a change of 1 kPa in PaCO₂ can cause a rise of 15 – 30% in CBF. O₂ affects CBF to a lesser degree – only causing an increase when PaO₂ falls below 8 kPa. ⁴



- Cerebral oedema also contributes to a raised ICP. Excessive fluid administration, especially of hypotonic fluid, worsens ICP.
- Fluid strategies should be balanced to ensure maintenance of adequate end-organ perfusion without significantly worsening cerebral oedema.

- A raised serum osmolality encourages the movement of water via osmosis from cerebral tissues to the circulation, thereby reducing cerebral oedema and ICP.
- Hypotonic fluids should therefore be avoided. Isotonic and even hypertonic fluids should be used with the aim of achieving mild hypernatraemia (Na^+ 145 – 150).
- As stated above ischaemia occurs when the perfusion of brain tissue does not meet its metabolic demands. As well as factors that impair perfusion, factors that increase cerebral metabolism can worsen ischaemic injury.
- Factors increasing cerebral metabolism include hyperthermia and seizure activity. Both require prompt treatment.
- CMR is exponentially related to body temperature – at 37°C for every 1°C fall in body temperature the CMR decreases by 7% ⁴. Conversely hyperthermia increases CMR and thereby worsens ischaemia.
- Although not directly affecting CMRO_2 , both hypo- and hyperglycaemia worsen cerebral ischaemic injury.

Management of Raised ICP

- Classically ICP > 20 mmHg required urgent treatment. The recent Brain Trauma Foundation Guidelines state an ICP of > 22mmHg requires urgent treatment.⁵
- If basic therapeutic targets are achieved (PaCO₂ 4.5 – 5.0 kPa, PaO₂ > 13 kPa, temperature < 37°C) and no obvious cause is identified (obstruction of venous return, lightening of sedation leading to coughing), a persistently raised ICP may require a CT scan to exclude a surgical cause.
- In addition to identifying conditions requiring surgical intervention e.g. new haemorrhage, the CT scan will also identify patients who may benefit from the insertion of an External Ventricular Drain (EVD).
- The EVD is inserted into one of the lateral ventricles which allows drainage of CSF helping reduce ICP. Not all patients with a raised ICP will benefit from an EVD – the greater the degree of ventricular enlargement the greater the potential for CSF drainage.
- Should no operative intervention be identified, therapeutic options include the use of hypertonic saline (2.7% sodium chloride) in 3ml/kg aliquots +/- Furosemide boluses (10 – 20mg).
- Together these drugs cause a rise in serum osmolality, through a rise in serum [Na⁺] and encouraging diuresis. This rise in serum osmolality lowers ICP as water moves via osmosis from cerebral tissue to the blood.
- Mannitol (0.25g – 0.5g / kg) is also used in the treatment of raised ICP, however a recent meta-analysis suggests hypertonic saline may be more effective in the treatment of raised ICP¹⁰, and work is ongoing in this area.
- Hyperventilation can be used as a temporising measure. However the optimal degree of hyperventilation remains unclear^{3, 5}.

- The AAGBI Guidelines recommend hyperventilation to a PaCO₂ 4.0 – 4.5 kPa if clinical or radiological evidence of raised ICP ⁵.
- The BTF Guidelines recommend against prolonged prophylactic hyperventilation below a PaCO₂ of 25mmHg (≈3.3 kPa). Furthermore, the guidelines recommend monitoring of cerebral oxygenation during hyperventilation – either through Jugular Venous Oximetry (S_jVO₂) or direct measurement of brain tissue oxygen levels ³.
- Hypothermia may be neuroprotective – reducing cerebral metabolism and ICP. It has been used prophylactically, prior to development of raised ICP, and therapeutically, to treat raised ICP ^{5, 11}.
- The current BTF Guidelines state early (< 2.5 hours), and short-term (< 48 hours) prophylactic hypothermia is not recommended to improve outcome ⁵.
- The role for therapeutic hypothermia remains unclear – management algorithms often include mild hypothermia (34° – 35°C) as an option for treating refractory intracranial hypertension. It should be reserved for specific cases of refractory raised ICP after exhaustion of other options.
- If raised ICP persists despite the use of the above therapies, **decompressive craniectomy** may be considered. This procedure involves the surgical removal of a large part of the skull, allowing the swollen brain to expand thereby reducing ICP ¹².
- The RESCUEicp trial, comparing Decompressive Craniectomy vs. standard medical care, showed a reduced 6-month mortality in the intervention group, but worse functional outcomes ¹².
- Barbiturates may be neuroprotective, reducing cerebral metabolic rate and oxygen consumption. They may also reduce ICP by decreasing Cerebral Blood Volume ⁵.

- Barbiturates have several side-effects, most notably haemodynamic instability, renal and hepatic impairment, immunosuppression and hypokalaemia. Dosage should be titrated to produce burst suppression on EEG monitoring ^{5, 11, 13}.
- Current BTF Guidelines recommend barbiturate use in raised ICP refractory to maximal medical and surgical treatment ⁵.
- An example dosing regimen is an induction dose of thiopentone (3 – 5 mg/kg) followed by an infusion of 3 – 5 mg/kg/hour titrated to EEG monitoring.

Neuropharmacology

- Key pharmacodynamic effects of drugs involved in neuroanaesthesia are the effects on CBF, vascular autoreactivity, ICP, CMRO₂ and MAP.
- Desired pharmacodynamic features include preservation of cerebral perfusion and cerebral vascular autoreactivity and a reduction in ICP and cerebral metabolism.
- Regarding induction agents both Propofol and Thiopentone reduce CBF, CMRO₂ and partly obtund the rise in ICP from laryngoscopy. Both also cause a dose-related fall in MAP ¹⁴.
- The use of opiates in conjunction with Propofol or Thiopentone can further obtund the pressor response to laryngoscopy as well as allowing a reduction in the dose of Propofol or Thiopentone required.
- Ketamine causes a rise in CBF, CMRO₂, but the evidence for its effect on ICP is weak. Recent meta-analysis has shown that Ketamine did not increase overall mortality in patients with TBI, and was not associated with an increase in ICP (but may lead to post-intubation hypotension). Due to its favourable haemodynamic profile it is widely used in a trauma setting ^{1, 14, 31}.
- Regarding maintenance either intravenous or volatile anaesthesia can be used and a strong debate exists about which is superior.
- One advantage of intravenous maintenance is that, unlike volatiles, there is a reduction in CBF and CMRO₂. This is of particular significance in patients with pre-existing elevated ICP.
- Regarding volatiles, the older agents are not commonly used in neuroanaesthesia. Halothane produces significant cerebral vasodilatation and enflurane is associated with seizure activity at a low PaCO₂ ¹⁴.

- Of the conventionally used agents (isoflurane, sevoflurane and desflurane) each has similar pharmacodynamic effects. Nitrous oxide produces cerebral vasodilatation and increases CMRO₂ ¹⁴ and is therefore avoided.
- Regarding CBF and ICP some studies suggest that cerebral vasodilatation occurs in a dose-dependent manner with desflurane > isoflurane > sevoflurane ^{15, 16, 17, 18}. Other studies suggest there is no statistically significant differences between the 3 agents ¹⁹.
- It is likely that, when combined with a remifentanyl infusion, allowing a MAC of < 1 to be used, the cerebral haemodynamic effects between the 3 agents are not significantly different.
- In addition, the haemodynamic effect of the volatile may play a more significant role in determining CPP ¹⁹.
- Additional considerations when comparing volatile agents in neurosurgery include speed of wake-up (DES > SEVO > ISO) and the preservation of cerebral autoregulation (preserved up to 1.5 MAC sevoflurane) and cost.

Endovascular Thrombectomy for Stroke

- Endovascular or Mechanical Thrombectomy (MT) is an interventional procedure where a thrombus is aspirated resulting in cerebral reperfusion. It is a highly effective treatment – the HERMES meta-analysis estimated a Number Needed to Treat (NNT) of 2.6 for an improved outcome vs. standard medical therapy ²⁰.
- Consideration for thrombectomy is based on ²¹:
 - Duration since onset of symptoms: (up to 24 hours as long as there is brain to save)
 - Stroke severity: Moderate to Severe stroke (NIHSS > 6).
 - Location of thrombus: large vessel occlusion of the proximal anterior or posterior cerebral circulation (basilar, M1/M2 of MCA, A1 of ACA).
 - Pre-existing functional status (mRS < 3).
- Patients presenting 6 – 24 hours after onset of symptoms may also be eligible for thrombectomy dependent on the degree of salvageable brain tissue shown on advanced imaging (CT-Perfusion Scan or MRI).
- This is a true time-critical emergency – the greater the duration before vessel recanalisation the greater the degree of cerebral infarction (2 million neurons lost each minute).
- Challenges include:
 - Acute effects from stroke such as dysphasia, hemiparesis, visual field disturbance, and nausea and vomiting.
 - Chronic co-morbidities such as AF, diabetes, and hypertension.
 - Fasting status.
 - Possible remote location of the Interventional Radiology Suite.
 - Limited time for pre-assessment and no optimisation time.

- The combination of the above factors, the emergent nature of such cases, and the more distant location of the IR suite means these are high-risk cases.
- These cases have been performed under local anaesthesia, with or without conscious sedation, or general anaesthetic. A recent meta-analysis of 3 small single-centre RCTs suggested there was an improvement in functional outcome associated with general anaesthesia vs. conscious sedation. This is likely due to the increased chance of successful recanalisation when performed under GA ²².
- Where there is significant risk of anaesthesia e.g. severe / critical aortic stenosis, discussion should take place with Neurointerventional Services as to whether or not the procedure can be performed under LA +/- sedation.
- Invasive blood pressure monitoring is recommended for cases under GA prior to induction. However, this should not delay the start of the procedure ²³. Arterial access can be utilised from the INR catheter, **so do not delay induction to site an arterial line unless it is clinically needed.**
- Irrespective of anaesthetic technique, it is essential to maintain haemodynamic stability. Excessive hypotension will reduce collateral flow to the ischaemic penumbra and worsen the infarct, whereas excessive hypertension can cause haemorrhage. Blood pressure targets are listed below.
- For GA, a Rapid Sequence Induction will be required in most cases +/- use of cricoid pressure. Use of opiates (Alfentanil 10mcg/kg, Fentanyl 2mcg/kg, Remifentanyl 0.5mcg/kg/min at intubation) allows dose-sparing of propofol and obtund the pressor response to laryngoscopy. Vasopressors should be drawn up ready to treat hypotension. Anaesthesia can be maintained with TIVA or volatile.
- Options for conscious sedation include incremental midazolam, alfentanil and fentanyl (25mcg increments). No one agent has been proven clearly superior.

- If sedation is employed, titrate sedation to patient response aiming for a patient who comfortable, easily rousable and able to follow commands. An anti-emetic such as ondansetron, is useful to pre-emptively treat any nausea. Administer supplemental oxygen and monitor exhaled CO₂ via face – avoid hyperoxia as this may worsen reperfusion injury.

- Basic targets include ²³:
 - Oxygen: SpO₂ 94 – 98%. Avoid hyperoxia.

 - Carbon Dioxide: To maintain normocapnia under anaesthesia.
Avoid hypercapnia in patients undergoing sedation.

 - Blood Pressure: Maintain BP within 15% of baselines with fluids and/or vasopressors for patients undergoing GA.
Classically an SBP 140 – 220 (**if no TPA**) or 140 – 185 (**if received TPA**) was targeted.

Recent evidence suggests a MAP of 70 – 90 mmHg may be more appropriate ²⁴. The interventionalist or stroke physician can provide further advice on the target BP.

 - Glucose: Aim normoglycaemia (BM 4 – 11 mmol/L).

 - Temperature: Avoid hyperthermia (Temp < 37.5°C).

- Post-operatively these patients can usually be woken up and extubated, before going to a hyper acute stroke unit. A small subset of patients may require HDU or ICU depending on any additional therapy, or if they arrived at the thrombectomy centre intubated.

Subarachnoid Haemorrhage

- Incidence of 6 – 12 per 100,000 / year with 30-day mortality rates of up to 50% ²⁵.
- 85% result from intracranial aneurysms – usually located at bifurcations with the Circle of Willis. Most affected sites are at the junction of the anterior cerebral artery and anterior communicating artery (40%) and branching of the middle cerebral artery (35%) ²⁵.
- World Federation of Neurosurgeons (WFNS) is a commonly used grading system. Higher grades are associated with worsening outcomes.
 - Grade 1 – GCS 15.
 - Grade 2 – GCS 13-14, no motor deficit.
 - Grade 3 – GCS 13-14, motor deficit.
 - Grade 4 – GCS 7-12.
 - Grade 5 – GCS 3-6.
- Treatment comprises of endovascular treatment (coiling most common) or surgical clipping. The International Subarachnoid Aneurysm Trial (2002) – ISAT, showed coiling resulted in a reduced risk of death or dependence at 1 year versus clipping (23.5% vs 30.9%)²⁵.
- Subarachnoid haemorrhage is associated with intra- and extracranial complications. Intracranial complications include:
 - Rebleeding: 2-4% within first 24 hours.
 - Hydrocephalus: Up to 33% of patients ²⁶.
 - Seizures: 1-7% of patients ²⁵.

- Extracranial complications include¹:
 - Cardiac complications ranging from ECG abnormalities (common), clinically significant arrhythmias, myocardial injury and ventricular dysfunction.
 - Disorders of sodium balance.
 - Pulmonary oedema and aspiration pneumonia.
- NCEPOD recommend all aneurysms (WFNS Grade 1-3) should be secured within 48 hours.
- Blood pressure control is important – systolic BP > 160 mmHg requires treatment to reduce risk of rebleeding²⁵.
- Conduct a standard pre-operative assessment documenting GCS, focal neurology and baseline blood pressure.
- Key aim is to maintain cardiovascular stability – hypotension is associated with cerebral ischaemia, hypertension with risk of aneurysmal rupture. Consider arterial line insertion pre-induction.
- Induce anaesthesia with propofol, opioid and muscle relaxant of choice. Fentanyl (2 – 3 mcg/kg) or remifentanyl (0.5 mcg/kg/min) obtund the pressor response to laryngoscopy.
- Intubation allows careful control of CO₂ – set the ventilator to achieve low normocapnia (ETCO₂ 4.0 – 4.5 kPa).
- Insert a temperature probe and urinary catheter – large volumes of contrast and intra-arterial fluid can be given during the procedure.
- Maintain with either TIVA or volatile agent. Remifentanyl infusion allows dose-sparing of anaesthetic agents and minimises the risk of coughing.

- The procedure is minimally stimulating – metaraminol boluses or infusion may be required to maintain pre-operative blood pressure.
- If the aneurysm is not secured avoid coughing at extubation.
- There is no consensus to the best anaesthetic technique – other centres use general anaesthesia with an LMA / spontaneous ventilation.
- Analgesic requirements are minimal – paracetamol may be all that is needed.
- Delayed Cerebral Ischaemia (DCI) is neurological deterioration related to ischaemia lasting more than 1 hour and not attributable to other causes (e.g. metabolic disturbance). Vasospasm is defined as radiologically-proven arterial narrowing with associated clinical symptoms and signs ²⁵.
- DCI, which may be associated with the presence, or absence of vasospasm, is seen in up to 60% of patients ²⁵:
 - Most commonly between 4 and 10 days post-ictus.
 - Nimodipine prophylaxis (60mg, 4-hourly for 21 days) reduces risk of cerebral infarction and poor outcome.
- Little evidence to support classic “Triple H Therapy”: Hypertension, Hypervolaemia and Haemodilution. Current management of vasospasm consists of hypertension, avoidance of hypovolaemia and maintenance of Haemoglobin between 80 – 100 g/L.

Seizures

- Seizures are common in the neurosurgical population and can be the presenting complaint for patients with many different pathologies, including brain tumour, TBI, intracranial haemorrhage or infection ³².
- Additionally, patients presenting for surgery may have an underlying seizure disorder such as epilepsy ³³.

Pre-operative assessment

- Understand the nature of lesion and surgery planned i.e. fronto-temporal tumours are higher risk for perioperative seizures ^{32, 33}.
- Confirm seizure type and triggers. Can include fatigue or stress, both of which are common in the perioperative period ³³.
- Continue current antiepileptic drugs (AEDs).

Diagnosis of perioperative seizure

- Most perioperative seizures occur at induction or emergence ³³.
- Diagnosis of intraoperative seizures can be challenging as anaesthetic agents and neuromuscular blockade can mask motor signs. Potential indicators include tachycardia, hypertension, high E_tCO_2 , and pupillary dilatation ^{32, 33}.
- EEG can identify epileptiform activity but pEEG monitors are unreliable for seizure detection.
- Intraoperative seizures can be managed with ice-cold saline flush by surgeons, this suppresses cortical activity, and/or incremental propofol boluses 0.5mg/kg ³⁴.

Management of status epilepticus

- Status epilepticus, defined as a seizure that lasts 5 minutes or longer, or recurrent seizures without recovery in between, should be managed as a medical emergency to prevent neurological injury and death.
- It can be convulsive or non-convulsive.
- Consult your local guidelines for specific drug dosing and administration.
- The following is adapted from NICE guidance ³⁴:

- Stage 1: ABCDE assessment including blood glucose and timing of seizure.
- Stage 2: Benzodiazepene administration e.g. lorazepam 2-4mg. Can be repeated at 5 – 10 min intervals.
- Stage 3: AED administration i.e. levetiracetam 60mg/kg max 4.5g; phenytoin 18mg/kg max 1800mg; sodium valproate 25mg/kg max 2500mg (reproductive risks with valproate). Can consider second stage 3 AED if ongoing seizure.
- Stage 4: Induction of general anaesthesia.

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