AN INTRODUCTION TO ANAESTHESIA FOR NEUROSURGERY

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Introduction
Anaesthesia for neurosurgical procedures requires understanding of the normal anatomy and physiology of the central nervous system and the likely changes that occur in response to the presence of space occupying lesions, trauma or infection.

In addition to balanced anaesthesia with smooth induction and emergence, particular attention should be paid to the maintenance of an adequate cerebral perfusion pressure (CPP), avoidance of intracranial hypertension and the provision of optimal surgical conditions to avoid further progression of the pre-existing neurological insult.

Aims of neuroanaesthesia

- To maintain an adequate cerebral perfusion pressure (CPP)
- To maintain a stable intracranial pressure (ICP)
- To create optimal surgical conditions
- To ensure an adequately anaesthetised patient who is not coughing or straining
- To enable rapid return to consciousness to allow neurological assessment postoperatively

General considerations for craniotomy
The patients

- Acute neurological condition or injury possibly with intracranial hypertension
- Medical therapy – anticonvulsants
- Pre-existing medical problems

The procedure

- Long operation time
- Blood loss
- Surgical stimulation / brain stem manipulation

The practicalities

- Position
- Access (intravenous and access to the airway)
- Invasive monitoring

Postoperative care

- Rapid recovery and neurological assessment
- Balanced analgesia to avoid sedation

General considerations for other procedures e.g. burr holes and shunts
The patients

- Often extremes of age
- Intracranial hypertension
- Associated conditions or trauma

The procedure

- Short procedure time
- Great surgical stimulation whilst shunt is tunnelled

The practicalities

- Supine position
- Invasive monitoring for burr hole

Postoperative care

- Rapid recovery and neurological assessment

Physiological Principals
Cerebral perfusion pressure and the intracranial pressure/volume relationship
Maintenance of adequate blood flow to the brain is of fundamental importance in neuroanaesthesia. Cerebral blood flow (CBF) accounts for approximately 15% of cardiac output, or 700ml/min. This equates to approximately 50ml/100g of brain tissue per minute under normal conditions. The brain is contained within the cranial vault which is non distensible and has a fixed volume. Cerebral blood flow is therefore affected by the pressure within the cranial vault and a useful measure of this is the cerebral perfusion pressure, the effective pressure which results in blood flow to the brain. Cerebral perfusion pressure (CPP) is the difference between the mean arterial pressure (MAP) and the sum of the intracranial pressure and the central venous pressure (CVP):

CPP = MAP - (ICP + CVP)

Under normal conditions, the ICP remains at 5-12 mmHg, the venous pressure at the base of skull is zero, and CPP varies with the individual's MAP. CPP is reduced in the presence of raised ICP, raised venous pressure or low MAP. Therapeutic measures to maintain optimal CPP are therefore aimed at maintaining MAP whilst lowering ICP and avoiding venous obstruction or hypertension.

Autoregulation is the ability of the brain to maintain stable CBF in the face of a changing MAP/CPP. As shown in figure 1, this is achieved by alterations in cerebrovascular resistance. The calibre of intracranial vessels alters automatically – vessels dilate if perfusion pressure is low (or if metabolic activity in one region is high). Under normal conditions, cerebral blood flow is kept at a stable level over a range of cerebral perfusion pressures between 50 and 150mmHg.

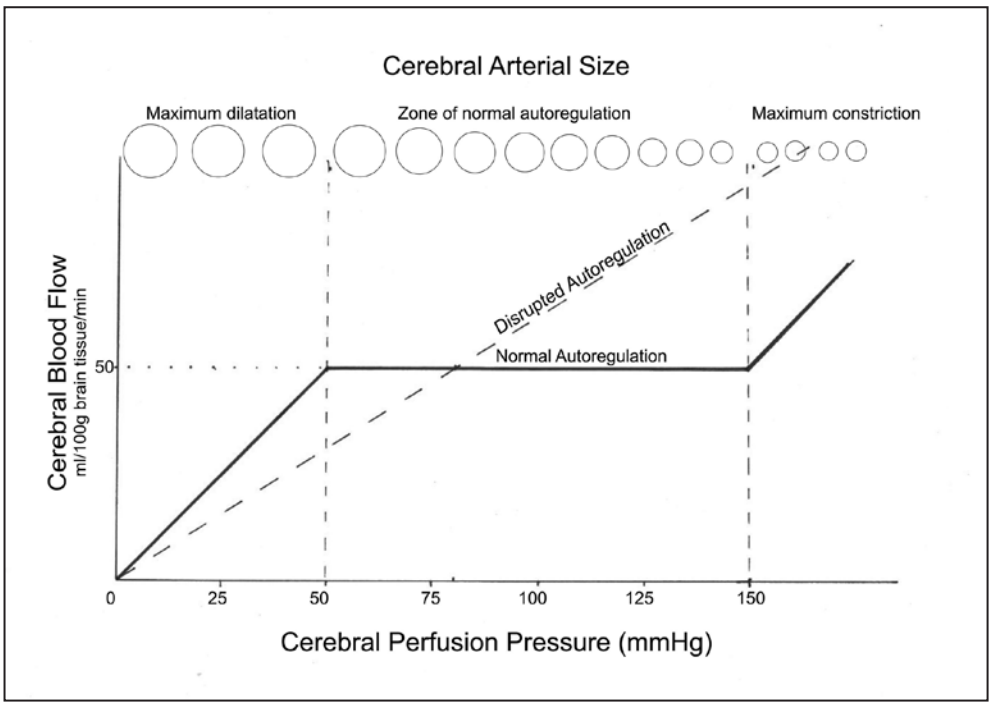

Figure 1: The relationship between cerebral blood flow and cerebral perfusion pressure. CBF is maintained at a constant value over a range of CPP (between 50 and 150mmHg) by alterations in cerebral vascular resistance. Decreased CPP causes vasodilation, increased CPP results in vasoconstriction. Conditions such as head injury disrupt autoregulation and the relationship becomes 'pressure-passive'.

In the presence of a space-occupying lesion (e.g. blood, tumour or oedema), the brain has limited compensatory ability before ICP increases. To avoid a rise in ICP, an increase in the volume of one component of the contents of the rigid cranial vault must be compensated by a reduction in the volume of another. As the volume increases in the vault (figure 2), the pressure is initially controlled by a reduction in CSF and cerebral venous blood volume. Once this mechanism is exhausted, then small increases in volume of the lesion lead to steep increases in intracranial pressure and eventually may cause displacement of brain tissue and cerebellar tonsillar herniation through the foramen magnum (coning).

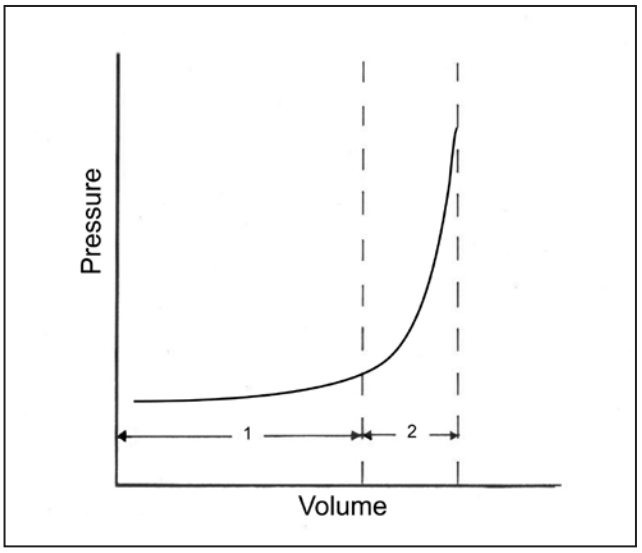

Figure 2: As the volume of a space occupying lesion within the intracranial vault rises, compensatory mechanisms allow intracranial pressure to remain stable (1). Once these mechanisms are exhausted, intracranial pressure rises very sharply (2).

Flow-metabolism coupling

Under normal circumstances, the brain is able to alter the supply of oxygen (by changing blood flow) to specific areas of the brain which have increased metabolic activity.

Arterial oxygen and carbon dioxide tensions

Carbon dioxide tension in the blood has a marked effect on the cerebral vasculature. A rise in arterial carbon dioxide ($PaCO_2$) causes cerebral vasodilation, an effect magnified in the presence of a reduced arterial oxygen tension. Cerebrovascular vasodilation causes an increase in intracranial volume that can cause coning under circumstances of raised ICP.

> In patients with raised intracranial pressure it is vital to control the $PaCO_2$ to normal and to ensure provision of adequate oxygen to avoid hypoxia.

Pathophysiological consequences of injury or disease

The pathophysiological consequences of injury depend upon the speed of onset and whether the compensatory mechanisms are overwhelmed. For example, a sudden catastrophic increase in ICP due to intracerebral haemorrhage can cause otherwise normal tissue to infarct, whereas slow increases due to hydrocephalus allow compensation, but may give rise to the symptoms of headache and nausea.

Once CBF falls below 18ml/100g/min, alterations in cellular activity occur, with intracellular acidosis and cessation of protein metabolism. At less than 12ml/100g/min electrical activity ceases and at 8ml/100g/min cell death occurs.

Autoregulation in injured parts of the brain is impaired

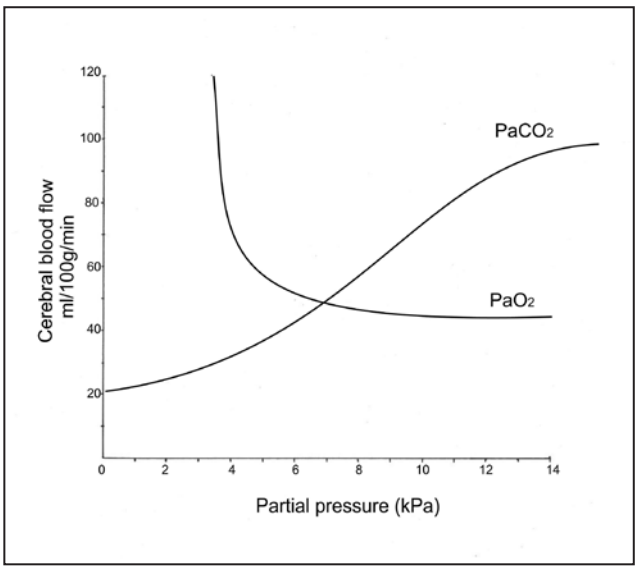

Figure 3: The effects of changes in $PaCO_2$ and PaO_2 on cerebral blood flow. A rise in $PaCO_2$ causes a marked rise in CBF. Fall in oxygen tension has a little effect on CBF until PaO_2 reaches a value of less than 8kPa.

and the threshold at which the vasculature can maintain CBF is elevated, from CPP of 50mmHg to 60–70mmHg. Hypoxia and hypercapnia cause secondary brain injury through ischaemic mechanisms. Hypercapnia causes vasodilation, an increase intracranial volume, subsequent reduction in CPP, further increase in $PaCO_2$ and a downward spiral in blood flow and worsening ischaemia.

The effects of anaesthetic agents on cerebral blood flow

Most anaesthetic agents reduce neuronal activity and so reduce the brain's cerebral metabolic requirement for oxygen ($CMR0_2$). They provide a protective mechanism when oxygen demand may outweigh supply. Unfortunately, many anaesthetic agents also reduce the mean arterial blood pressure by causing arterial vasodilatation with a potentially adverse effect on CPP.

Thiopentone

- Causes a dose-dependent fall in $CMR0_2$, also a fall in CBF. These effects are useful during craniotomy in the presence of elevated ICP.
- Anticonvulsant properties are advantageous.

Propofol

- Similar effects to thiopentone but may preserve autoregulation more effectively.
- It is useful for maintenance of anaesthesia and its amnesic and antiemetic properties make it advantageous for a smooth recovery.
- It does cause a fall in MAP which may have disadvantageous effects on CPP.

Ketamine

- This NMDA antagonist has traditionally been avoided because it increases ICP and CBF, although these effects are less marked if $PaCO_2$ is controlled to normal levels.
- It is not routinely used for elective neurosurgical anaesthesia at present, but has gained acceptance as an appropriate agent for emergency induction of anaesthesia and maintenance of sedation in head injured patients, particularly those with multiple injuries and haemodynamic compromise.

Inhalational agents

- Sevoflurane has less cerebral vasodilatory effects than other inhalational agents and preserves CO_2 reactivity. Also it allows a rapid recovery due to its lower blood:gas solubility coefficient.
- Desflurane at 1 MAC has been shown to increase ICP in patients with supra-tentorial space occupying lesions, in contrast to isoflurane.
- Most of the inhalational agents are deemed safe at concentrations of less than 1 MAC.
- Nitrous oxide increases ICP, CBF and $CMRO_2$. These effects, together with its adverse effect on closed gas spaces preclude its use, especially in trauma patients who may also have undetected chest injury.

Opioids

- Remifentanil – has a rapid onset and offset which allows titration to counter stimulating events such as the application of the Mayfield clamp (see below). A dose dependent fall in MAP and rapid offset mean that a longer acting analgesic agent is required prior to cessation of surgery.
- Fentanyl and morphine – these agents have little effect on intracranial pressure or blood flow which makes them suitable for titration to provide post-operative analgesia.

Neuromuscular blocking drugs

- The non-depolarizing agents do not have an effect on ICP.
- Suxamethonium causes a transient rise in ICP, in part due to muscle fasciculation and increased venous pressure. There may also be a slight increase in $CMR0_2$ and cerebral blood flow. These considerations must be weighed against the need for rapid airway control. Suxamethonium is usually reserved for emergency anaesthesia rather than elective cases.

Other drugs

Diuretics

- Mannitol is a large molecule that will not cross the intact blood brain barrier. It is useful in the

prevention and treatment of cerebral oedema and it causes a reduction in ICP. It can cause a transient rise in cerebral blood volume and this effect can last for up to 20 minutes. It also causes a transient rise in CVP. The usual dose is 0.5 – 1g/kg. It has the additional effect of haemodilution which is thought to improve blood flow characteristics. If the blood brain barrier is damaged it may worsen raised ICP.

- Furosemide 1mg/kg produces a reduction in ICP to the same extent as mannitol at 1g/kg. It is advantageous as it also reduces CVP.

Steroids

- Dexamethasone 8-16mg is useful to reduce cerebral oedema associated with tumours. It is less effective for reduction in global oedema.

Anticonvulsants

- For frontal and temporal surgery phenytoin can be given as a loading dose, 15mg/kg intravenously, but should be given slowly as it causes hypotension and can cause arrhythmias.

Drugs to manipulate the cardiovascular system

- α-agonists such as phenylephrine and noradrenaline are used to increase blood pressure as there are fewer alpha receptors in the cerebral vasculature. These agents have a selective effect on systemic vascular resistance and cerebral vascular resistance is relatively unaffected.
- Clonidine, an α_2-antagonist, can be used to treat hypertension at the start of surgery. It has analgesic and sedative properties which are a useful adjunct to anaesthesia but these effects must be weighed against the need for rapid emergence at the end of surgery.

Anaesthesia for craniotomy for tumour or intracranial bleed

General considerations

History and examination is important to detect signs and symptoms such as convulsions, nerve palsies and reduced levels of consciousness (assess GCS).

Posterior fossa tumours occasionally cause bulbar palsies and lower cranial nerve lesions which increase the risk of laryngeal incompetence and thus chronic or acute aspiration of gastric contents and hypoxia.

Subarachnoid haemorrhage can cause massive release of catecholamines which can cause acute heart failure and malignant arrhythmias. Non-specific T-wave and ST segment abnormalities may be seen on the ECG.

The patient's general medical condition must be stabilised, especially any respiratory or cardiovascular disease, as hypoxia, hypercarbia or failure to maintain blood pressure will be detrimental, as will uncontrolled hypertension. Preoperative medications, especially anticonvulsants, steroids and cardiac drugs, should be continued until and including the day of surgery if possible. Antiplatelet drugs should be discontinued if possible, although the risks of stopping these drugs must be considered.

Airway assessment is important because prolonged attempts at laryngoscopy are extremely stimulating and increase cerebral oxygen demand and ICP.

Patients are often anxious and a sedative premedication such as a benzodiazepine can be offered.

Conduct of anaesthesia

Induction needs to be smooth and blood pressure maintained near preoperative values to maintain cerebral blood flow. A bolus of either propofol (0.5-1mg/kg) or opiate should be given immediately prior to laryngoscopy as this is very stimulating. Adequate time should be allowed for non depolarising muscle relaxant to work before intubation is attempted.

Once the patient is draped and surgery underway access to the airway is very limited, so it is absolutely vital that the airway is secured reliably, preferably with waterproof tapes. An armoured cuffed tracheal tube prevents the tube kinking when the patient's head is manipulated, particularly if the patient is to be positioned prone. Meticulous attention to securing the tube is vital. A prone patient whose skull is pinned is very difficult to manage should the tube fall out.

Intravenous access must be of large bore and reliable as sudden massive haemorrhage can occur. Invasive arterial monitoring is very useful for reliable minute to minute blood pressure assessment and also for evaluating arterial blood gasses and haemoglobin levels. Consideration should be given to site the IV access and arterial pressure monitoring before induction of anaesthesia (although not in children).

Central venous access is appropriate as a guide to venous pressure and to administer drug infusions. The sitting position is used much less frequently now, but placement of the tip of a central line in the right atrium should be considered for these patients and others at risk of air embolus, to allow aspiration of air from the heart should this occur.

Anaesthesia can be maintained using a volatile agent in air and oxygen or propofol infusion. The patent's lungs are ventilated to achieve normocapnia - normal areas of the brain vasoconstrict secondary to hypocapnia and total cerebral blood flow will be reduced inappropriately. Hypocapnia is reserved for situations where ICP is very high; it may be life saving in this situation but there can be rebound vasodilation when normal CO_2 levels are achieved subsequently.

Surgical procedures are often very lengthy and so attention must be paid to patient position, protection of pressure areas, including the eyes, and to ensuring unrestricted venous drainage from the head.

The patient must have a urinary catheter for lengthy craniotomy or if diuretics are used. Calf compressors help to reduce thromboembolic risk. Core body temperature should be measured, preferably with an oesophageal probe.

During patient positioning in theatre the skull is often pinned into a clamp (a Mayfield clamp) to maintain optimal surgical positioning. This is stimulating and so a pre-emptive bolus of propofol or an opioid should be given to prevent a sudden rise in blood pressure.

Once the skull is open a bolus of diuretic optimises operating conditions if intracranial pressure is elevated and the surgeon comments that the dura is bulging. Mannitol 0.5mg/kg or furosemide 1mg/kg are appropriate. Mannitol increases the central venous pressure so should be given slowly - especially if the patient's myocardial function is impaired.

During the procedure, maintain low normal end tidal CO_2 (around 4.0 kPa), normotension and normal oxygen levels. Mild hypotension can help improve the surgical field if necessary. Normothermia should be maintained, especially if the procedure is long. Temperature should be allowed to passively drift to about 35 degrees centigrade if the patient's cerebral blood supply is at risk – for instance during aneurysm surgery. Allowing the patient to become hypothermic has consequences of poor clotting function, impaired cardiac contractility and postoperative shivering which increases oxygen demand.

Fluid replacement with glucose-free crystalloid, such as normal saline or lactated Ringers, is appropriate. Hyperglycaemia is associated with worse neurological outcome and tight glycaemic control may help improve outcome and avoids lactate accumulation. Hypotonic intravenous solutions must never be used as they will exacerbate cerebral oedema.

If the ICP is high during surgery

- Place the patient in a slightly head up position.
- Check there is no neck vein kinking or compression and the abdomen can move freely with no diaphragmatic compression.
- Ensure the patient is paralysed.
- Ventilate without PEEP.
- Ensure the blood pressure is adequate.
- Ensure the $PaCO_2$ is not raised and consider reducing it to 4-4.5kPa.
- Ensure the PaO_2 is normal.
- Reduce the brain's metabolic activity – bolus thiopentone 3mg/kg or propofol 1mg/kg, or lidocaine 1.5mg/kg if cardiovascularly unstable .

During surgery, especially posterior fossa surgery, the brainstem may be manipulated, which can cause profound bradycardias. If this occurs, communicate with the surgeon to release traction or pressure and treat with glycopyrrolate 200–400 micrograms (atropine crosses the blood-brain barrier) and allow surgery to resume when the heart rate is normal.

Once surgery is drawing to a close the surgeon closes the dura, cranium and scalp, which can take up to 30 minutes. If anaesthesia is too light the patient may cough as the head is moved to apply the dressings at the end of surgery. Good communication between surgeon and anaesthetist will help with timing of paralysis and cessation of anaesthesia. Remifentanil infusion, if available, helps to smooth out the waking and extubation process, but remember to give a bolus of longer acting opioid to avoid postoperative agitation. Whichever agent has been used, aim for a smooth extubation with a minimum of straining and coughing.

Pain from craniotomy is described as 'mild to moderate' and paracetamol and codeine are popular analgesics. Non-steroidal anti-inflammatory drugs have an anti-platelet effect and should be used with caution. Small (1-2mg) boluses of intravenous morphine are appropriate for patients in severe discomfort. The aim at the end of surgery is to have a comfortable, co-operative and lucid patient whose neurology can be assessed.

Anaesthesia for other neurosurgical procedures

Burr holes

Burr holes are usually performed as an emergency procedure in those who have had an extradural haemorrhage (arterial, usually associated with a skull fracture) or subdural haemorrhage (venous, may be chronic especially in the elderly). Patients will usually have:

- Altered level of consciousness (measure GCS).
- Raised ICP.
- Focal neurology, such as a dilating pupil.

Other injuries may be present that need consideration. Protection of the patient's cervical spine must be considered.

Preoperative interventions include:

- Secure the airway with cervical spine control if GCS is <9/15 or ventilation is inadequate for normal oxygenation and carbon dioxide elimination (rapid sequence induction with thiopentone and suxamethonium is appropriate).
- Maintain normal blood pressure with fluids and inotropes, using non-glucose containing crystalloid and α-agonists if necessary. Aim for a MAP of 90mmHg in adults.
- Nurse the patient 15-30 degrees head up with no tube tie.

- Ventilate the patient to achieve normocapnia and normoxia.
- Sedate, for example using a propofol infusion 1-3mg/kg/hr.
- Insert invasive arterial pressure monitoring
- Monitor the CVP.

Extracranial injuries must be assessed and their management incorporated into the definitive treatment plan.

Communication between the trauma team and neurosurgical/ICU team is of paramount importance. Other life threatening injuries must be dealt with and stabilised and may occasionally take priority over evacuation of an intracranial haematoma. In this situation, intracranial pressure must be assumed to be raised and diuretics and low CO_2 may delay brain herniation. Patients are often transferred to regional neurosurgical centres, making management of raised ICP even more important. Make sure cross-matched blood is available

Patients who have a low GCS preoperatively or who have high ICP intraoperatively should be managed in an intensive care unit postoperatively and kept sedated and ventilated for 24 hours after surgery.

Ventriculoperitoneal (VP) shunt for hydrocephalus
Hydrocephalus can present acutely with a low GCS, or chronically with headaches, neurological symptoms and vomiting. It can result from the overproduction of CSF (non-obstructive and very rare), blockage of CSF absorption (obstructive, communicating resulting from arachnoiditis) or obstruction to normal CSF flow (obstructive, non-communicating).

In treatment of hydrocephalus a fine bore tube with a valve is inserted to drain CSF from the dilated ventricle into the peritoneal cavity.

- Patients may be children.
- The commonest shunt is from the lateral ventricle to the peritoneum.
- The commonest complication of VP shunts are infection and blockage.
- Patients can have an intracranial haemorrhage if CSF is drained too quickly.

The procedure is shorter than full craniotomy and invasive monitoring is not usually required unless the patient is medically unwell. The airway should be secured and attention paid to good oxygenation, normalising $PaCO_2$ and preventing coughing and straining – especially during the stimulating part of surgery when the shunt is being tunnelled subcutaneously. As with craniotomy, arrhythmias or bradycardia can occur as the catheter is placed.

Post-operatively the aim is to have an awake and comfortable patient in recovery that can co-operate with neurological assessment. Paracetamol and codeine phosphate are the mainstay of analgesia and intravenous morphine titrated to effect may also be required.

Further reading

- Matta B, Menon DK, Turner JM et al. Textbook of Neuroanaesthesia and Critical Care. Published by Cambridge University Press, Cambridge, UK
- Clayton T and Manara A. Neurosurgery in the Oxford Handbook of Anaesthesia (second edition). Published by Oxford University Press, Oxford, UK

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