Anaesthesia for interventional neuroradiology

IL Dorairaj MBBS MD FRCA
SM Hancock MB ChB FRCA

Anaesthesia is required for neuroradiological diagnostic procedures such as angiograms, computerized tomography (CT), and magnetic resonance imaging (MRI) or for therapeutic intervention (Table 1). Interventional neurovascular procedures are part of a trend towards minimally invasive neurosurgery, an important development in which has been the introduction of the Guglielmi detachable coil (GDC) for endovascular aneurysm coiling. Evidence that coiling is associated with a better outcome than craniotomy and clipping is moving more procedures out of theatres into the neuroradiology suite.

Prolonged procedures, improved patient safety, and optimal conditions for imaging have resulted in a trend towards a greater use of general anaesthesia (GA), especially in aneurysm and arteriovenous malformation (AVM) treatments, while conscious sedation is preferred for cerebral ischaemic disease (carotid stents, angioplasty, and thrombolysis). Though many of the risks encountered in this newer arena are conceptually similar to traditional neurosurgery, important differences in the working environment and practice exist.

Endovascular treatment of cerebral aneurysms

Aneurysmal subarachnoid haemorrhage (SAH) remains a disabling and frequently lethal disease. Approximately 10–15% of patients experience sudden death which may be attributable to several factors: systemic derangement, direct damage to the brain, complications of surgical clipping and coiling, and vasospasm. Treatment of unruptured intracranial aneurysms is largely restricted to those discovered incidentally or those causing a mass effect on adjacent structures such as the optic nerves.

Using a femoral arterial guide catheter, a series of GDCs (radio-opaque, MRI-compatible, platinum coils) are deployed through a microcatheter into an aneurysm until occlusion is achieved. Coiling may carry a greater incidence of re-bleeding and does not seem to decrease the incidence of cerebral vasospasm. There may also be the potential for the aneurysm to reform postoperatively.

Preoperative assessment

Patients who have had an SAH can have marked derangement of various organ systems; the proportion of deaths after SAH from medical complications equals deaths from direct effects, re-bleeding, or vasospasm individually. Pulmonary complications are the most common non-neurological cause of death.

Neurology

A brief history and short neurological examination should be carried out to establish Glasgow Coma Scale, grade of SAH and any cranial nerve, visual field, and motor and sensory deficits. The nature, location, and size of the lesion and previous treatment must also be ascertained.

Cardiovascular system

Massive catecholamine release is the most likely cause of cardiac dysfunction seen after SAH. This may include dysrhythmias, abnormal ECG morphology (T inversion, ST depression, Q waves, U waves, and prolonged QT), elevated cardiac enzymes, and frequent left ventricular dysfunction and pulmonary oedema. Therapy with oral anticoagulants should be stopped and, if necessary, converted to heparin, which can be stopped or reversed, if required.

Respiratory system

In addition to the strong causal relationship between SAH and cigarette smoking, reduced levels of consciousness and prolonged bed rest predispose to atelectasis and pneumonia.
A baseline measurement of activated clotting time (ACT) should be made from an arterial sample before commencement of the procedure.

**Vascular access**

At least two wide bore i.v. cannulae should be inserted. Connections, extensions, and infusion devices should be checked before and during the anaesthetic, particularly if total i.v. anaesthesia (TIVA) is the chosen technique. Attempts at arterial and i.v. cannulation should be minimal and peripheral, in view of systemic anticoagulation during the procedure.

**Induction**

The overriding priority is to maintain cardiovascular stability, avoiding surges in arterial pressure that might cause aneurysm rupture while maintaining adequate perfusion of a possibly ischaemic cerebral circulation. \(^7\) To this end, propofol is usually used to induce anaesthesia combined with remifentanil, alfentanil, or fentanyl; thiopentone and etomidate are alternatives. \(^4\) Pressor responses can also be obtunded with i.v. lidocaine or rapid, short-acting β-blockers (e.g. esmolol). Before tracheal intubation, it is important to ensure that neuromuscular block is profound; before administration of neuromuscular blocking agent, the correct placement of electrodes for peripheral nerve stimulation should be verified. \(^7\) Rocuronium, atracurium, or vecuronium are suitable for neuromuscular block. It is useful to use a cut endotracheal tube so that the image intensifier does not push in or kink the tube; an armoured tube is preferred in some centres. The laryngeal mask airway has also been used in this setting; there is insufficient evidence to recommend its routine use.

**Maintenance**

Under clinical conditions, all volatile agents have the potential to increase cerebral blood flow (CBF), cerebral blood volume (CBV), and intracranial pressure (ICP), uncouple CBF and metabolic demand (CMRO2) and produce a persistent post-anesthetic hypercarbia. \(^7\) To this end, propofol is usually used to induce anaesthesia combined with remifentanil, alfentanil, or fentanyl; thiopentone and etomidate are alternatives. \(^4\) Pressor responses can also be obtunded with i.v. lidocaine or rapid, short-acting β-blockers (e.g. esmolol). Before tracheal intubation, it is important to ensure that neuromuscular block is profound; before administration of neuromuscular blocking agent, the correct placement of electrodes for peripheral nerve stimulation should be verified. \(^7\) Rocuronium, atracurium, or vecuronium are suitable for neuromuscular block. It is useful to use a cut endotracheal tube so that the image intensifier does not push in or kink the tube; an armoured tube is preferred in some centres. The laryngeal mask airway has also been used in this setting; there is insufficient evidence to recommend its routine use.

**Metabolic considerations**

Tight control of blood glucose is essential since hyper- and hypoglycaemia are associated with poor outcomes, particularly in the presence of cerebral ischaemia. Patients are often dehydrated with electrolyte disturbances such as hypomagnesaemia, hypernatraemia, hyponatraemia associated with the syndrome of inappropriate ADH secretion, hypokalaemia, and hypocalcaemia.

**Conduct of anaesthesia**

**Premedication**

Premedication should be individualized. A titrated dose of a benzodiazepine (e.g. midazolam) provides anxiolysis, sedation of short duration, and amnesia, although it can impair assessment of neurological status and worsen confusion. \(^6\) Narcotics are best avoided because of potential respiratory depression and hypercarbia. H2-receptor antagonists, alone or with metoclopramide, may be used to reduce the risks of gastric aspiration. Nimodipine is frequently used to reduce cerebral ischaemia consequent to cerebral vasospasm.

**Monitoring and equipment**

Monitoring for clipping and coiling is similar \(^4\) and includes ECG, pulse oximetry, a nerve stimulator, inspired and expired gas, and invasive arterial pressure monitoring. It is preferable to monitor the latter before induction of anaesthesia. In cases where an arterial cannulation fails, the femoral artery introducer sheath can be transduced, providing reliable mean but overestimating diastolic and underestimating systolic BP. Central venous pressure may be monitored if fluid/electrolyte requirement or medical status warrants it. A peripherally inserted long line is preferred at our institution. The laryngeal mask airway has also been used in this setting; there is insufficient evidence to recommend its routine use.

**Table 1** Neuroradiological procedures that may require anaesthetic input

<table>
<thead>
<tr>
<th>Diagnostic</th>
<th>Therapeutic</th>
</tr>
</thead>
<tbody>
<tr>
<td>CT, MRI, angiography, myelography</td>
<td>Embolization: cerebral aneurysm, AVM (intracranial, dural, and spinal)</td>
</tr>
<tr>
<td>Stereotactic-guided neurosurgery: tumour, movement disorders, biopsy, and radiotherapy</td>
<td>Embolization: tumours, carotid cavernous fistulae, and epistaxis</td>
</tr>
<tr>
<td>Sclerotherapy: venous angiomas</td>
<td>Balloon angioplasty: carotid stenosis and vasospasm</td>
</tr>
<tr>
<td>Intra-arterial chemotherapy: head and neck tumours</td>
<td>Intra-arterial chemotherapy: head and neck tumours</td>
</tr>
<tr>
<td>Thrombolysis: acute thromboembolic stroke</td>
<td>Superselective angiography: aneurysms and AVM</td>
</tr>
<tr>
<td>Carotid occlusion for aneurysm and tumours: therapeutic and test occlusion</td>
<td></td>
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</tbody>
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Table 2 Special considerations for interventional neuroradiology (INR)

<table>
<thead>
<tr>
<th>Special considerations</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfamiliar environment, dim lighting, radiology staff unfamiliar with anaesthetic practices, help not easily available</td>
<td>Ensure skilled assistance, dedicated recovery area and staff, resuscitation, and difficult airway equipment in INR suite</td>
</tr>
<tr>
<td>Transfers between CT, MRI, radiology, theatres, and hospitals</td>
<td>Closely observe clinical status, body temperature, lines, tubes, ventriculostomy, and equipment</td>
</tr>
<tr>
<td>Radiation</td>
<td>Radiation shielding, minimize total time, keep distance from source (inverse square law)</td>
</tr>
<tr>
<td>Radiology equipment</td>
<td>C-arm, injector, console, table kept unimpeded by lines and cables</td>
</tr>
<tr>
<td>Closed skull</td>
<td>Control intracranial pressure by manipulating PaCO₂, blood pressure, and intravascular volume</td>
</tr>
<tr>
<td>Contrast and flush</td>
<td>Fewer adverse reactions with non-ionic low osmolar agents</td>
</tr>
<tr>
<td>Use of heparin, antiplatelet drugs, and thrombolytics</td>
<td>Monitor ACT, be prepared to use protamine, platelets, FFP, or plasmapheresis</td>
</tr>
<tr>
<td>Sedation, neurolept anaesthesia, and repeated neurological testing</td>
<td>Use short-acting agents, careful positioning. Watch for airway obstruction, vomiting, vagal reactions, loss of cooperation, shivering</td>
</tr>
<tr>
<td>Image degradation, interference with ‘road mapping’</td>
<td>GA to facilitate respiratory immobility, high-frequency jet ventilation; treat shivering</td>
</tr>
</tbody>
</table>

may develop on sudden discontinuation of the infusion, necessitating a slow decrease in rate before emergence. Propofol and remifentanil, sevoflurane and remifentanil, and a combination of propofol and remifentanil supplemented with sevoﬂurane⁷ have all been described.

Ventilation aims for mild hypocapnia to normocapnia (PaCO₂, 4–4.5 kPa) to help control ICP.⁵ End-tidal CO₂ may be allowed to run slightly higher than in surgical patients, since the reduction in brain bulk to facilitate surgical exposure is not required. The reduced CBF due to the vasoconstriction associated with mild hyperventilation reduces contrast transit time and allows contrast to fill to the edges of the arterial lumen, thus improving the quality of the vascular image. Tailored blood pressure control is important.

In patients with SAH, it is vital to avoid hypotension as cerebral autoregulation is impaired. Noxious stimulation during coiling is usually minimal and maintenance of cardiovascular stability is sometimes difficult. This problem is managed by minimizing anaesthesia and supporting the circulation with fluids and vasopressors.⁵ Controlled hypertension is used in cases of iatrogenic vascular occlusion and acute thromboembolic stroke. The goal is to increase cerebral perfusion pressure to ischaemic areas via collateral circulation. With all pressors, it is important to know the patient’s volume status.

After femoral cannulation, heparin is administered as an initial i.v. bolus (5000 IU) followed by intermittent boluses or an infusion to keep ACT 2–3 times baseline; ACT is monitored hourly. For reversal of heparin anticoagulation, protamine is used in a dose of 1 mg per 100 units of heparin or dosed according to the heparin dose–response curve. Control of body temperature is important; hyperthermia is associated with poor outcome, and mild hypothermia has not shown to improve neurological outcome.

Recovery
A rapid and smooth recovery is desirable to facilitate early neurological assessment and safe transfer to recovery areas. Blood pressure is allowed to return to normal⁵ or up to a systolic pressure of 160 mm Hg. An unsecured or incompletely secured aneurysm may call for induced hypotension.⁵ Postoperative shivering needs treatment. Patients who have had neurological complications may need to be transferred to neurointensive care for continued sedation and ventilation.

Postoperative analgesia
Paracetamol, and either codeine or morphine administered parenterally, may be used.

Complications
Vascular complications are either haemorrhagic or occlusive. The management of these requires emergency management and the radiologist should lose no time in informing the anaesthetist of the suspected event and vice versa. Vascular rupture or perforation may be: (i) spontaneous; (ii) due to hypertension during laryngoscopy, emergence, inadequate depth of anaesthesia, or associated with the use of vasoactive drugs; or (iii) brought about by the microcathether, guide wire, coil, or injection of contrast.

Clinical signs of a rise in ICP or a sudden rise in blood pressure with or without a fall in heart rate should alert the anaesthetist to this possibility. Extravasation of contrast may also be seen. The goals are to increase coagulability by reversing heparin, decrease bleeding by lowering blood pressure (to the level before the bleed), control ICP with hyperventilation, head elevation, steroids and osmotic agents, control seizures, and initiate cerebral protection. Once the bleeding is controlled, the pressure may be raised to check for leaks. Usually, the coiling continues; rarely, a ventriculostomy may be required. If the coiling is unsuccessful, a rescue craniotomy and clipping will be required. Management may also involve performance of CT scans and subsequent transfer to ICU.

Cerebral occlusion leading to ischaemia and infarction occurs and may be due to: thromboembolism; arterial dissection; catheters; coil misplacement; or vasospasm. The goal is to increase collateral flow by increasing MAP using controlled hypertension. Therapy with heparin, antiplatelet drugs, or even thrombolytic therapy may be indicated in some cases.

Cerebral vasospasm is one of the most serious consequences after aneurysmal SAH. Medical treatment consists of oral
nimodipine and haemodilutional, hypervolaemic, hypertensive therapy (triple-H) aiming for a haematocrit of 30%, CVP of 8–12 mm Hg and an increase in blood pressure to reverse or prevent neurological deficits. Nimodipine is given intra-arterially to treat spasm during coiling.

**Arteriovenous malformations: cerebral, dural, and spinal**

Embolization may be used to obliterate an AVM or, more commonly, to reduce its size before surgery (or radiotherapy) in order to minimize intraoperative bleeding while preserving the arteries supplying the blood flow to the brain. Rapid blood flow, multiple fistulae, feeding and draining vessels, and associated aneurysms make for prolonged and staged procedures. The materials used include polyvinyl alcohol particles, N-butyl cyanoacrylate, detachable coils, balloons, collagen, silicon, oxycel, silk, gelfoam, silastic pellets and a liquid polymer Onyx.

Embolizations may be performed under GA, awake, or under sedation. Repeated testing (Superselective Anaesthesia Functional Examination) with contrast and sodium amylobarbital or lidocaine may be required to confirm that the microcatheter feeds only the abnormal mass.

Blood pressure needs close attention; hypotension can worsen intracerebral steal; and raised ICP from recent intracranial haemorrhage may worsen with hypertension. Controlled hypotension using potent, short-acting agents may be employed for short periods to produce ‘flow arrest’ through the AVM and enable embolic glue to set rather than be carried straight through.

Complications include neurological deficits from inadvertent occlusion of normal vessels, pulmonary embolism from systemic shunting of particulate material and seizures. Severe bleeding can arise due to incomplete embolization or perforation of arterial feeders or from rupture of an associated aneurysm and may result in death from exsanguination or uncontrolled cerebral hypertension. The sudden exclusion of the AVM shunt can result in cerebral hyperperfusion (due to dysfunctional autoregulation) or occlusive hyperaemia (if the AVM and normal brain share venous drainage).

**Stereotactic neurosurgery**

Stereotactic neurosurgery allows three-dimensional localization of specific sites within the brain using CT and, more recently, MRI scanning. The initial step to stereotactic localization is the application of the base ring to the patient’s skull, using pins. This may be done under sedation and local anaesthesia, nerve blocks, or GA. The localizing ring is then attached to the base ring and a CT or MRI is performed allowing the neurosurgeon to compute the exact three-dimensional position of the region of interest. The patient is then transferred to the operating room. The points of interest are mapped onto a ‘phantom’ which, in conjunction with a computer, determines the final trajectory to guide the neurosurgeon.

The procedure involves transfers to and from neurosurgical theatres, CT, or MRI scan and anaesthesia in the scanner. The frame limits access to the airway and the key to the frame should be available to the anaesthetist at all times in case of an emergency. The surgical procedure may entail waking up the patient intraoperatively or a rapid wake-up after operation and a technique using infusions of propofol and remifentanil may be appropriate. Other precautions relevant to anaesthesia for minimally invasive neurosurgery should be followed.

**Interventional magnetic resonance imaging**

MRI systems provide high-resolution, detailed images. Only a few institutions around the world currently have intraoperative MRI capabilities. Patients may need to be transferred to an adjacent scanner or treated in a radiofrequency-shielded operating room specifically designed for MRI use. Intraoperative MRI systems contain a permanent magnet, which means that even when the machine is off, the magnet is still active. Anaesthesia care providers should be aware of special MRI-compatible equipment, allergic reactions to gadolinium contrast, and the positioning of magnets in order to preclude inadvertent movement to the patient’s head, pressure on the patient’s shoulders, or contamination of the sterile field.

**References**


Please see multiple choice questions 6–9