Anaesthesia for interventional neuroradiology

IL Dorairaj MBBS MD FRCA
SM Hancock MB ChB FRCA

Key points

Evidence of a better outcome with coiling of cerebral aneurysms compared with craniotomy and clipping is increasing the requirement for dedicated neuroradiological anaesthetic services in the UK.

The unfamiliar remote environment, patient transfers, use of contrast, anticoagulation, and radiation exposure pose unique challenges.

Thorough assessment and optimization of patients' systemic conditions needs to be performed before the procedure.

Rapid and short-acting anaesthetic agents are essential to allow rapid recovery and assessment.

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.[1] Evidence that coiling is associated with a better outcome than craniotomy and clipping[2] is moving more procedures out of theatres into the neuroradiology suite.[3]

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,[4,5] 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.[6]

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.[6] 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.

IL Dorairaj MBBS MD FRCA
Specialist Registrar
Queen's Medical Centre
Derby Road
Nottingham NG7 2UH
UK

SM Hancock MB ChB FRCA
Consultant Anaesthetist
Queen's Medical Centre
Derby Road
Nottingham NG7 2UH
UK
Tel: +44 01159249924
Fax: +44 01159783891
E-mail: sally.hancock@nuh.nhs.uk
(for correspondence)

doi:10.1093/bjaceaccp/mkn013

Table 1 Neuroradiological procedures that may require anaesthetic input

Diagnostic
CT, MRI, angiography, myelography
Therapeutic
Embolization: cerebral aneurysm, AVM (intracranial, dural, and spinal)
Stereotactic-guided neurosurgery: tumour, movement disorders, biopsy, and radiotherapy
Embolization: tumours, carotid cavernous fistulae, and epistaxis
Sclerotherapy: venous angiomas
Balloon angioplasty: carotid stenosis and vasospasm
Intra-arterial chemotherapy: head and neck tumours
Thrombolysis: acute thromboembolic stroke
Superselective angiography: aneurysms and AVM
Carotid occlusion for aneurysm and tumours: therapeutic and test occlusion

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. H_2-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[5] 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. Procedures may be long, the neuroradiology suite cold and large volumes of flush, and contrast can be used making a urinary catheter, temperature monitoring, and the availability of warming devices necessary. Cerebral venous obstruction should be avoided; due attention must be paid to the position of the head and neck, tracheal tube ties, monitoring cables, ECG leads, and gown ties. Care should be taken to avoid impairment of the radiologist's view from trailing radio-opaque lines. 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 ($CMRO_2$) and produce a persistent post-anaesthetic hyperaemia for up to 1 h after use.[4] The latter increases risk for intracranial haemorrhage, particularly if systolic BP is more than 160 mm Hg for two or more consecutive measurements. Sevoflurane is the volatile anaesthetic agent of choice because of its low potential for increasing CBF–ICP and its rapid offset. Up to 1 MAC, there is preservation of the reactivity of cerebral blood vessels to carbon dioxide and coupling of CBF and $CMRO_2$. Sevoflurane also provides faster recovery and postoperative neurological assessment than isoflurane. Nitrous oxide (N_2O) elevates CBF and ICP and increases the consequences of air embolism; it should not be used.

The use of a TIVA technique incorporating propofol and a short-acting opioid reduces CBF, ICP, and $CMRO_2$. However, there is evidence that sevoflurane is associated with more rapid early recovery than propofol, for long duration interventional neuroradiology procedures (Table 2).[8]

Of the short-acting opioids, remifentanil is frequently used; it provides stable haemodynamics and allows more rapid recovery from anaesthesia than alfentanil or fentanyl.[9] Rebound hypertension

Table 2 Special considerations for interventional neuroradiology (INR)

Special considerations	Response
Unfamiliar environment, dim lighting, radiology staff unfamiliar with anaesthetic practices, help not easily available	Ensure skilled assistance, dedicated recovery area and staff, resuscitation, and difficult airway equipment in INR suite
Transfers between CT, MRI, radiology, theatres, and hospitals	Closely observe clinical status, body temperature, lines, tubes, ventriculostomy, and equipment
Radiation	Radiation shielding, minimize total time, keep distance from source (inverse square law)
Radiology equipment	C-arm, injector, console, table kept unimpeded by lines and cables Secure connections, enough slack in lines and tubing, mount transducers and pumps on INR table
Closed skull	Control intracranial pressure by manipulating Pa_{co_2}, blood pressure, and intravascular volume
Contrast and flush	Fewer adverse reactions with non-ionic low osmolar agents Watch for hyperosmolarity, hypervolaemia followed by dehydration
Use of heparin, antiplatelet drugs, and thrombolytics	Monitor ACT, be prepared to use protamine, platelets, FFP, or plasmapheresis
Sedation, neurolept anaesthesia, and repeated neurological testing	Use short-acting agents, careful positioning. Watch for airway obstruction, vomiting, vagal reactions, loss of cooperation, shivering
Image degradation, interference with 'road mapping'	GA to facilitate respiratory immobility, high-frequency jet ventilation; treat shivering

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 sevoflurane[7] have all been described.

Ventilation aims for mild hypocapnia to normocapnia (Pa_{co_2} 4–4.5 kPa) to help control ICP.[5] End-tidal CO_2 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 normal[5] 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 microcatheter, 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.[10] 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.[10] 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

1. Brilstra E, Rinkel G. Treatment of ruptured intracranial aneurysms by embolization with controlled detachable coils. *Neurologist* 2002; **8**: 35–40
2. Van der Schaff I, Algra A, Weremer M *et al.* Endovascular coiling versus neurosurgical clipping for patients with aneurysmal subarachnoid haemorrhage. *Cochrane Database Syst Rev* 2005; **4**: CD0030853
3. Webb ST, Farling PA. Survey of arrangements for anaesthesia for interventional neuroradiology for aneurysmal subarachnoid haemorrhage. *Anaesthesia* 2005; **60**: 560–4
4. Armonda RA, Vo AH, Dunford J, Bell RS. Anaesthesia for endovascular neurosurgery. *Neurosurgery* 2006; **59** (Suppl. 3): S66–76
5. Jones M, Leslie K, Mitchell P. Anaesthesia for endovascular treatment of cerebral aneurysms. *J Clin Neurosci* 2004; **11**: 468–470
6. Solenski NJ, Haley EC, Jr, Kassell NF, *et al.* Medical complications of aneurysmal subarachnoid haemorrhage: a report of the multicenter, cooperative aneurysm study. *Crit Care Med* 1995; **23**: 1007–17
7. Levy DM, Nowicki RWA. Anaesthesia for treatment of cerebral aneurysms. *CPD Anaesth* 2002; **4**: 106–14
8. Castagnini HE, van Eijs F, Salevsky FC, Nathanson MH. Sevoflurane for interventional neuroradiology procedures is associated with more rapid early recovery than propofol. *Can J Anaesth* 2004; **51**: 486–91
9. Coles JP, Leary TS, Monteiro JN *et al.* Propofol anaesthesia for craniotomy: a double-blind comparison of remifentanil, alfentanil and fentanyl. *J Neurosurg Anaesth* 2000; **12**: 15–20
10. Dodson B. Interventional neuroradiology and the anaesthetic management of patients with arteriovenous malformations. In: Cottrell J, Smith D, eds. *Anaesthesia and Neurosurgery.* St Louis: Mosby, 2001

Please see multiple choice questions 6–9