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Interventional neuroradiological procedures—a review for anaesthetists

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SUMMARY

Interventional neuroradiology is a rapidly expanding field, and the complexity and duration of these procedures makes anaesthetic support essential to their success. Such has been the development in this area that the American Heart Association has published a scientific statement on the indications for these procedures. A detailed understanding of patient pathology, the technical aspects of the interventions and their associated risks, and the remote location in which they are performed are important for providing expert anaesthetic care. The aim of this article is to provide a description and contemporary analysis of the common interventional neuroradiology procedures relevant to the anaesthetist. This article will cover the management of intracranial aneurysms, cerebral vasospasm following intracranial haemorrhage, intracranial and spinal arteriovenous malformations, idiopathic intracranial hypertension, carotid artery stenting, intra-arterial thrombolysis for stroke and endovascular treatment of intracranial atherosclerosis. Protection from ionising radiation and acute kidney injury are also discussed.

Key Words: anaesthesia, interventional, intracranial aneurysm, arteriovenous malformations

Interventional neuroradiology is a rapidly expanding field, and the complexity and duration of these procedures makes anaesthetic support essential to their success. Such have been the advances in this area that the American Heart Association Council on Cardiovascular Radiology and Intervention published a scientific statement on the indications for these procedures[1]. These interventions are performed in locations remote from the operating suite and the procedures present unique challenges to the anaesthetist. A detailed understanding of patient pathology and the technical aspects of these procedures, with their associated risks, is essential for providing expert anaesthetic care.

The aim of this article is to provide a description of the common interventional neuroradiology procedures relevant to the anaesthetist with a comprehensive and contemporary analysis of this topic and the anaesthetic considerations. A review of the literature revealed a paucity of evidence from randomised prospective studies relating to much of the material discussed in this article, and the design and size of the studies quoted have been made explicit throughout the paper to enable readers to critically appraise the literature. The first part covers cerebral aneurysm and arteriovenous malformation procedures and the second part covers other common interventional neuroradiology procedures and considerations including protection from ionising radiation and acute kidney injury.

METHODS

A comprehensive review of the relevant literature pertaining to neuroradiological procedures involved a search of the PubMed database using the following search terms: 'interventional neuroradiology', 'anaesthesia', 'cerebral aneurysms', 'arteriovenous malformations', 'idiopathic intracranial hypertension', 'stroke', 'carotid artery stenting', 'acute kidney injury' and 'radiation exposure'. English language papers from the past ten years were included in the search. Further articles of relevance were identified from reference lists in articles identified in the initial literature search.

PART 1: CEREBRAL ANEURYSMS AND ARTERIOVENOUS MALFORMATIONS

Percutaneous endovascular treatment of cerebral aneurysms

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Background

Cerebral aneurysm rupture is the most common cause of subarachnoid haemorrhage and is associated with a high mortality rate if untreated[2]. The prevalence of aneurysms in the adult population without comorbities is approximately 3.2%[3]. The incidence of cerebral aneurysmal rupture peaks in the sixth decade of life[4] and results in a considerable socioeconomic burden, costing an estimated £510 million in healthcare expenses and productivity loss annually in the UK[5]. The first endovascular treatment of cerebral aneurysms using balloon occlusion of carotid and vertebral arteries was performed in the 1970s by Serbinenko in Moscow[6]. Endovascular treatment accelerated following the development of detachable endovascular coils by Guglielmi in 1990[7]. Subsequently, the International Subarachnoid Aneurysm Trial[8] compared coiling versus surgical clipping for aneurysms in 2134 patients with ruptured intracranial aneurysms (World Federation of Neurosurgeons Grades I and II) and established the endovascular technique as the preferred treatment for selected cerebral aneurysms. In this trial, the majority of patients (average age 52 years) had anterior circulation aneurysms <1 cm in diameter and the absolute risk reduction in the primary outcome, death or dependency, at one year was 7.4% (P=0.0001) favouring endovascular therapy. This survival benefit was maintained for seven years with a lower rate of epilepsy in the endovascular group[9]. Further analyses demonstrated that the rate of re-rupture beyond one year was low in both the surgical and coiled patients (<1%) but the rate of re-bleeding was higher in the endovascular treatment group at a mean of nine years' follow-up (ten re-bleeds from the endovascular treated aneurysm in 8447/years of follow-up versus three patients in the surgically treated group in 8177/ years of follow-up)[10]. This trial indicated the need to select patients carefully for either surgery or coiling on the basis of the size, architecture and position of the aneurysm and an understanding of the long-term complications associated with aneurysm coiling.

The endovascular treatment of unruptured intracranial aneurysms is controversial because the natural history of unruptured intracranial aneurysms remains uncertain, and both surgical and endovascular treatment carries risk[11]. The largest cohort study exploring the natural history of unruptured aneurysms found that both the size and the site of the aneurysm influenced the five-year risk of aneurysmal rupture. In this study the majority of ruptured aneurysms were 7–9 mm in diameter, and the risk of rupture of aneurysms <7 mm in the anterior circulation was 0.1% per year. Conversely, the five-year cumulative risk of rupture of larger aneurysms in the posterior circulation was 50%. The limitations of the study included limited follow-up and the unreported variability of risk in each group[1,11,12]. Investigating the risk of endovascular treatment of unruptured aneurysms, a large multicentre prospective study demonstrated a 95.7% technical success rate and 5.4% short-term clinical complication rate (including transient or permanent neurological injury and death)[13]. In this trial thromboembolic complications occurred in 7.1% of patients, intraoperative rupture occurred in 2.6% of patients and device-related problems occurred in 2.9% of procedures. There were no differences in complication rates between aneurysms <3 mm and >3mm in size, although success rates were lower in the very small aneurysm group (86.3 vs 96.7 %, P=0.003)[14]. There are no randomised trials comparing conservative management with endovascular treatment of unruptured aneurysms. Current guidelines in the USA recommend that endovascular or surgical treatment of unruptured aneurysms should be considered[15], taking into account the factors (such as size and site of aneurysm) that may increase the risk for haemorrhage[16].

Technique

Computed tomography angiography is used to assess the size, location, neck width and relationship to parent and neighbouring vessels for planning therapy[2]. Cerebral angiography provides further clarification and may be combined with biplanar fluoroscopy in anaesthetised patients to enable real-time visualisation in two planes. After cerebral angiography, a microcatheter is advanced into the aneurysm and a framing coil that covers the neck and outlines the contour of the aneurysm is then placed. Progressively smaller coils are sequentially placed to fill the aneurysm.

A range of different coils are used to treat cerebral aneurysms. GDC Coils (Boston Scientific, Fremont, CA) are platinum coils attached to stainless steel pusher wires and are deployed using an electrical signal that serves to both promote thrombus formation and release the coil from the proximal stainless steel delivery wire[16]. These coils are soft and are designed to adopt the shape of the aneurysm to fill the sac. As recanalisation remains a problem with bare platinum coils, newer bioactive coils have been developed to reduce this complication. Polyglycolic-polyactic acid (Matrix) and hydrogel are used to produce more complete occlusion of the aneurysm. There is presently mixed evidence to support the use of these coils over bare platinum coils[17].

An alternative to coils is the use of a liquid embolic

material that is delivered into the aneurysm with stent or balloon assistance to promote thrombus formation. Onyx (Onyx Liquid Embolic System, ev3 Neurovascular, Irvine, CA), an ethylene-vinyl-alcohol biocompatible copolymer combined with tantalum powder (to facilitate fluoroscopic visualisation) is an example[18]. It solidifies on contact with blood to form a spongy cast. To date there are a few small case series that report the effectiveness of this technique in selected aneurysms[19].

Coils combined with balloons or stents are used to expand the range of aneurysms amenable to endovascular treatment. Temporary balloons that serve as a provisional support are used in the treatment of wide-necked aneurysms to prevent extension into the parent vessel as coils are deployed in the aneurysm. Alternatively, stents can be used to prevent prolapse of the coils into the parent artery. There is evidence to support these techniques, but they are limited by an increased risk of thromboembolic complications[20,21]. A recent review of studies involving stent-assisted coiling in acutely ruptured aneurysms reported a technical success rate of 93%, a mortality rate of 19%, clinically significant haemorrhagic complications in 8% and clinically significant thromboembolic events in 6%. This highlighted the higher risk of these procedures compared with endovascular coiling procedures without stent placement[22].

Flow diversion stents are an alternative endovascular technique for the treatment of aneurysms[23]. These stents redirect blood flow in the parent artery and induce flow disruption and stasis, and consequently thrombosis in the aneurysmal sac. They also provide a scaffold for neo-intimal growth in the parent artery[24,25]. Early studies demonstrated the feasibility of this technique for complex and large aneurysms. Aneurysm occlusion is not immediate, but occurs within six months. Late aneurysm rupture, perforating vessel occlusion and stent thrombosis are complications. The long-term outcomes with this technique are currently unknown.

Complications

The complications of endovascular aneurysm treatment include stroke, aneurysmal rupture during the procedure, aneurysm recurrence, re-rupture after first coiling, technical failure and vascular access complications including groin haematoma, pseudoaneurysm and retroperitoneal bleeding. Table 1 summarises the procedural morbidity and mortality rates of the major endovascular therapy trials.

Anaesthetic considerations

Anaesthesia for coiling shares many similarities with anaesthesia for conventional neurosurgery but with some unique exceptions. While coiling can be performed under sedation[26], many proceduralists and anaesthetists prefer general anaesthesia because superior image quality (from immobility), cardiorespiratory control and improved patient comfort (with a prolonged procedure) can be achieved.

General principles of neuroanaesthesia are important in the management of these cases and particularly so in the challenging setting of ruptured aneurysms. A careful approach to maintain adequate cerebral perfusion, while also preventing acute surges in blood pressure or intracranial pressure (ICP), is required to prevent rupture or re-rupture. General

Table 1

Summary of the procedural morbidity and mortality rates of the major endovascular therapy trials

Trial	Mortality rate	Complication rate	Further detail
ISAT trial 2005 (n=2143, RCT)[8]	Not reported	Not reported	2.6% re-bleeding rate in endovascular Group V. 1.2% in surgical group at 1 year 7.5% cases not able to coiled
Vanninen 1999 (n=109, RCT)[26]	2%	17% (perforation, vessel occlusion, coil migration & re-bleeding)	5 patients required surgical rescue
Smith 2011(bare platinum vs coated coils, n=101, non-randomised)[18]	1% (coil migration into MCA leading to infarction and death)	4% (thrombus, rupture, embolism)	
ATENA Trial (unruptured aneurysms, RCT, n=649, 1100 aneurysms)[14]	1.4% at 1 month	1.7% at 1 month 15.4% technical complications (thromboembolic complications [7.1% per procedure], intraoperative rupture [2.6% per procedure], device-related problems [2.9% per procedure]), 5.4% transient and permanent neurological deficit.	Endovascular treatment was unable to be performed for 4.3% of aneurysms

ISAT=International Subarachnoid Aneurysm Trial, RCT=randomised controlled trial, MCA=middle cerebral artery, ATENA=Adjuvant post-Tamoxifen Exemestane versus Nothing Applied

anaesthesia with inhalational or intravenous agents, or a combination of the two, has been used with success for these procedures. A smooth and rapid emergence from anaesthesia is essential to facilitate early neurological assessment. While there is no evidence supporting a particular anaesthetic technique over any other, total intravenous anaesthesia with propofol is preferred by some anaesthetists because cerebral vasomotor tone is preserved resulting in improved flow-metabolism coupling and carbon dioxide responsiveness, less vasodilation and a lower ICP[27]. Haemodynamic stability during intubation, a smooth extubation with a rapid recovery from a relatively painless procedure is achieved with a short-acting opioid infusion in combination with a propofol infusion. Although there is no evidence to support specific blood pressure targets during these procedures, avoidance of acute hypertensive and prolonged hypotensive episodes are accepted principles in this setting. Phenylephrine or metaraminol infusions may be required to augment the patient's blood pressure because of the lack of surgical stimulation.

In our practice, routine monitoring is usually supplemented by invasive arterial monitoring. Central venous access is not routinely required but may be warranted in patients with significant cardiac comorbidities or for vasoactive drug infusions.

The patients are usually heparinised (activated clotting time—two or three times the baseline recordings) but antithrombotic regimens vary between institutions. In the event of intraoperative aneurysm rupture urgent reversal of heparin with protamine may be required after discussion with the proceduralist. Antiplatelet drugs are usually required when stent-assisted coiling is undertaken.

Care with intravenous fluid administration is necessary as it is not uncommon for several litres of intravenous fluid to be used as a catheter flush by the proceduralist. In addition, most patients require a urinary catheter because the use of hyper-osmotic contrast media commonly results in a significant diuresis. The patients frequently become hypothermic from exposure and cold intravenous fluids unless active warming measures are undertaken.

Cerebral arteriovenous malformation embolisation

Background

Cerebral arteriovenous malformations (AVM) are abnormal labyrinths of vessels comprising one or more arteries that drain directly into one or more veins via a nidus without an intervening capillary bed. The flow in the arteriovenous shunt can be high, resulting in a high pressure that is transmitted to the draining veins. The location, size and architecture of AVM are highly variable and complex. AVM that occur within the brain parenchyma or within the dura are referred to as arteriovenous fistulae. Published estimates of the prevalence of brain AVM vary widely. The most reliable estimate is from a population-based study in the USA, which estimated an age and sex adjusted prevalence rate of identified intracranial vascular malformations, including AVM, of 19 per 100,000 person years[28]. Brain parenchymal AVM are a rare but an important cause of intracranial haemorrhage (ICH) in young adults. While they account for 1–2% of strokes overall, they account for 3% of strokes and up to one third of all primary ICHs in young adults[29]. Approximately 50% of patients with an AVM present with an ICH[30,31]. Other presenting symptoms include focal neurological signs, seizures, migraine-like headaches and bruits. For patients presenting without a preceding bleed, the annual risk of ICH is estimated to be 0.9–4% per year[30,31] and between 6–34% in the year following a bleed[30,32]. The factors associated with an increased risk of ICH include increased age, deep brain location and deep venous drainage[30]. Early treatment is recommended because of the high risk of re-bleeding in patients who have already had an ICH. In patients who do not have an ICH at diagnosis, the decision as to when and how to best treat these patients remains uncertain. The current treatment options include surgical resection, embolisation and stereotactic radiosurgery individually or in combination, although no randomised trials comparing options are available. Where indicated, pre-surgical embolisation is performed over one or more treatment sessions to reduce the nidal size and flow in the AVM to reduce the risk and difficulty of surgical resection or improve the effectiveness of radiosurgery. The success rates of complete embolisation of AVM vary from 40–60% of attempted embolisations, but this is associated with significant mortality (~10%)[33–36]. The indications for treatment of patients who have not bled may be clarified by the ARUBA trial (A randomized trial of unruptured brain arteriovenous malformations). This trial compares medical to invasive therapy—any combination of neurosurgery, endovascular treatment or radiosurgery—for unruptured AVM (www.clinicaltrials.gov NCT00389181).

Technique

Both angiographic and magnetic resonance imaging are required to provide a detailed characterisation of the AVM prior to embolisation. AVM embolisation in patients is performed under general anaesthesia.

The embolic material used in these cases is either n-butyl cyanoacrylate, an adhesive glue with high thrombogenicity, which polymerises to form a hard cast, or Onyx (Onyx Liquid Embolic System, eV3 Neurovascular, Irvine, CA), which precipitates into a spongy cast on contact with blood. Super-selective catheterisation of the AVM nidus, typically from the arterial side, is performed, followed by an injection of embolic material into the nidus. During injection induced hypotension and transient cardiac asystole are sometimes employed to reduce flow through the AVM and improve the accuracy of placement of the embolic material.

Complications

Complications of this procedure include delayed (1–90 hours post-embolisation) parenchymal oedema or haemorrhage adjacent to the AVM, blood vessel perforation or rupture, microcatheter retention and embolisation of material to normal brain parenchyma resulting in stroke[3]. The morbidity rates range from 1.3–1.7% and mortality rates range from 7.1–13%[36,36]. There is also risk of embolisation of the embolic material beyond the brain, such as pulmonary embolism causing acute respiratory distress syndrome[37]. Pulmonary oedema secondary to excretion of the solvent (dimethyl sulfoxide) via the lungs may also occur.

Anaesthetic considerations

General anaesthesia using controlled ventilation reduces patient movement and enables respiratory pauses. This facilitates the mapping of the cerebral vasculature and the controlled injection of embolic material that enhances the resolution of the image used to guide embolic material injection. The use of hypotension and transient cardiac asystole to reduce flow through AVM facilitates the accurate and safe deployment of embolic material into the AVM. Adenosine is used for this purpose[38], although the dose required to induce a period of asystole and hypotension is unpredictable. A case study reported that a dose range of 6–90 mg adenosine was required in one paediatric and four adult patients[39]. In this study asystole lasted 8±3 seconds; mean arterial pressure <30 mmHg lasted 18±12 seconds. A sodium nitroprusside infusion was used pre-emptively to obtund rebound hypertension.

Avoidance of hypertension is essential in these patients to reduce the risk of bleeding both pre and post-treatment. The exact mechanism of ICH in normal brain tissue adjacent to AVM post-treatment is uncertain. The most widely accepted theory is a normal perfusion pressure breakthrough syndrome, which occurs because the dilated arteries in the surrounding brain are maximally dilated with loss of autoregulation. As a result, cerebral oedema and haemorrhage can occur when normal blood pressure is restored to this vascular bed[40]. Alternative theories include capillary instability and weakness as a consequence of neovascularisation[41] and occlusive hyperaemia due to obstruction of adjacent venous outflow and stagnant arterial flow in previous AVM feeding vessels[42]. It is recommended that the blood pressure is maintained at 15–20% below the patient's baseline blood pressure but this is not evidence-based[43,44]. Diligent avoidance of hypertension and maintenance of normotension or controlled hypotension are the aims of the perioperative care of these patients.

Cerebral vasospasm management

Background

Cerebral vasospasm is common following subarachnoid haemorrhage (SAH) of any aetiology and occurs in 30–70% of patients following aneurysmal SAH[15]. Its onset is unpredictable and is associated with a poor outcome. Anaesthetists are often required to assist in the endovascular management of these patients, either for percutaneous transluminal angioplasty and/or intra-arterial vasodilator therapy. The use of percutaneous transluminal angioplasty in the treatment of cerebral vasospasm is generally limited to large proximal vessels. One centre recently reported that the use of percutaneous transluminal angioplasty for distal vessel vasospasm reduced the need for repeat intra-arterial vasodilator treatments[45]. Numerous pharmacological agents have been delivered into the cerebral vasculature to treat vasospasm. Papaverine, calcium antagonists (nimodipine, nicardipine or verapamil) and milrinone[46,47] are effective in reversing cerebral vasospasm to varying extents[48] but repeat treatments are frequently required.

Complications

A study of 189 patients who received endovascular treatment for cerebrovascular vasospasm following SAH reported complications in 3.2 % of patients and this included three dissections, one vessel rupture, one thromboembolic event and one intractable ICP rise[49]. The haemodynamic effect of intra-arterial vasodilators can persist beyond the period of treatment. Verapamil can cause haemodynamic changes, a reduction in mean arterial pressure and cerebral perfusion pressure, which can last up to six hours and result in prolonged elevations in ICP[50]. Nimodipine has a long half-life (nine hours).

Papaverine has a short duration of effect and is less commonly used now because it is associated with a reduction in brain oxygen tension, an increase in ICP (particularly when multiple arterial segments are treated)[51] and neurological deterioration related to a permanent neurotoxic effect[52].

Anaesthetic considerations

There are currently no guidelines for blood pressure targets. Weak evidence from non-randomised trials supports the use of induced hypertension (mean arterial blood pressure increased 20–33 mmHg) to improve neurological outcome[53].

When intra-arterial nicardipine or milrinone are administered to treat SAH vasospasm, vasopressor requirements are significantly increased: approximately 60% for phenylephrine and double for noradrenaline[54]. These increases are not associated with an increase in end organ injury or systemic acidosis and are associated with an increase in the calibre of vasospastic vessels. The majority of these procedures will be performed under general anaesthesia and the duration of treatment ranges from 60–537 minutes[54].

Spinal arteriovenous malformation embolisation

Background

Spinal vascular malformations are rare vascular lesions that occur at any position along the spine and represent approximately 10% of central nervous system AVM[55]. In the USA, approximately 300 patients present annually with spinal AVM that require treatment[56]. The patients (peak age 45–64 years) may present with pain, sensorimotor changes and myelopathy secondary to mass effect, vascular steal or haemorrhage.

Spinal vascular malformations are classified into two groups based on the vascular anatomy: fistulae and AVM with a nidus (see aforementioned AVM section). They may also be classified according to the arterial supply of the lesion: a) those that are supplied by radiculomeningeal arteries that supply the meninges and nerve roots, and b) those that are supplied by intrinsic arteries of the spinal cord. Spinal dural arteriovenous fistulae are the most common spinal vascular malformations, accounting for 70% of these lesions. Spinal dural arteriovenous fistulae are characterised by radiculomeningeal arteries feeding the shunt[57]. These lesions cause an increase in spinal venous pressure thereby reducing the arteriovenous pressure gradient resulting in reduced venous drainage and venous congestion with intramedullary congestion. Spinal AVM fed by arteries that supply the spinal cord neural tissue can be classified into glomerular AVM or fistulous AVM. Glomerular AVM are usually intra-medullary with multiple arterial feeders and drainage into dilated spinal cord vessels, whereas fistulous AVM have superficial reticulomedullary feeding vessels and superficial peri-medullary venous drainage and only rarely contain intra-medullary components[57]. The pathophysiological features of these lesions include venous congestion, haemorrhage and vascular steal phenomena. This may result in acute back pain due to haemorrhage, or progressive weakness secondary to venous congestion or steal-related ischaemia.

Treatment of these variable AVM may include surgical resection, endovascular embolisation, stereotactic radiosurgery or a combination of these. Embolisation of spinal AVM is increasingly used as a component of multimodal therapy or as definitive therapy alone. While surgery remains a component of treatment in selected cases, particularly for dural arteriovenous fistulae, the number of patients managed surgically has been declining in the USA[56].

Technique

Magnetic resonance imaging is the imaging modality of choice in the diagnosis of spinal AVM. Selective spinal digital subtraction angiography is then used to plan therapy. Deposition of glue or coils to occlude the nidus or venous receptacle is then performed after super selective catheterisation of the lesion. Recent small studies reported the effective use of Onyx glue for spinal dural arteriovenous fistulae embolisation[58], extradural AVM[59] and intramedullary AVM[60]. The goal of therapy in these patients is to restore haemodynamic balance and reduce venous congestion rather than achieve angiographic obliteration of these lesions[57]. In a series of 11 patients with fistulous AVM, Krings et al reported that ten AVM were completely occluded, four requiring multiple procedures and there were no procedure related complications[57].

Complications

Because of the rarity of the condition and the evolution of embolisation techniques, complication rates are only available from small case studies. Acute haemorrhage related to catheter disruption of feeding vessels and vascular occlusion with catheters and embolisation of embolic material resulting in neurological sequelae have been reported. In one study of 24 patients with glomerular AVM treated in 43 sessions, one patient had a new sensory deficit due to glue reflux and four patients had temporary pain or sensory disturbance symptoms[57]. In another small case series, a 45-year-old patient with a subarachnoid bleed associated from an intramedullary AVM died

after acute embolisation treatment with Onyx glue caused cervical anterior artery occlusion[60].

Anaesthetic considerations

General anaesthesia is indicated to ensure immobility for the accuracy of imaging and catheter placement in these small lesions. Respiratory pauses are required to reduce movement artefact during digital angiography and when selectively catheterising the lesion. Controlled hypotension to reduce flow through the malformation during deposition of embolic material improves the accuracy of deposition and reduces the potential to embolise material beyond the target lesion. A theoretical model evaluating the use of controlled hypotension during embolisation of intracranial AVM suggested that it may reduce the chance of nidus rupture in these procedures[61], although evidence for this in spinal AVM is not available.

Neuro-physiological monitoring of the spinal cord with somatosensory (SSEP) and motor evoked potential monitoring has been used in this setting. This may be combined with pharmacologic provocative testing, using lignocaine or barbiturate injections through the microcatheter placed at the site of the planned embolisation. This is used to identify the functional supply of the catheterised vessel and guide the embolisation of these anatomically complex lesions. A retrospective analysis of 84 angiographies demonstrated that this technique has a high negative predictive value[62]. There were 19 positive results. One false negative result occurred with an increase in postoperative spasticity after embolisation. Whether or not pharmacologic provocative testing is used, the use of SSEP and motor evoked potential monitoring may identify spinal cord pathway ischaemia in these cases as has been demonstrated in a few case reports[63–65].

Systemic heparinisation is typically required during these cases and in some centres is continued for 24 hours post-procedure with an APTT target of 50–60 seconds[66], although there are currently no published guidelines for anticoagulation and heparinisation.

PART 2: INTERVENTIONAL NEURORADIOLOGY PROCEDURES BEYOND ANEURYSMS AND ARTERIOVENOUS MALFORMATIONS

Idiopathic intracranial hypertension and cerebral venous stenting

Background

Idiopathic Intracranial Hypertension (IIH), also known as pseudotumour cerebri, is a rare (~11/100,000) condition characterised by raised intracranial pressure without any identified intracranial pathology[67]. The condition is most common in overweight women of childbearing age in whom the prevalence is increased eight-fold[68]. The aetiology of IIH is uncertain but may be related to abnormal cerebrospinal fluid re-absorption[69], possibly mediated by alterations in glucocorticoid metabolism[70]. It is also associated with transverse venous sinus stenosis[71]. Headaches are the most common symptom and papilloedema the major clinical sign. Up to 25% of patients ultimately develop visual impairment due to optic atrophy[67]. The goals of treatment are to reduce headache symptoms and preserve visual function. Unfortunately, the management remains controversial[69] with no randomised trials available and limited evidence to guide the treatment of these patients[72]. Medical therapies include diuretics, commonly acetazolamide, and corticosteroids. Weight loss is also associated with an improvement in symptoms[73]. Surgical treatments for patients developing visual impairment or unrelieved symptoms include ventriculo-peritoneal shunts, optic nerve sheath fenestration and, more recently, venous sinus stenting[74]. Limited improvement in headache symptoms and the need for repeat shunt revisions, despite an improvement in visual acuity and reduction in visual deterioration[75], has prompted the interest in venous stenting as an alternative therapeutic option in patients unresponsive to medical therapy. The rationale for venous sinus stent placement in this condition is based on a model of the collapsible venous sinuses functioning as Starling resistors that, while not necessarily the primary cause of the condition, contribute to IIH through disruptions to normal cerebrospinal fluid physiology[76].

Data from non-randomised case studies support the efficacy of venous stenting. In a large retrospective analysis of 52 patients with IIH, symptoms unresponsive to maximal medical therapy and associated with transverse sinus stenosis who underwent stent placement, 49 patients reported resolution of symptoms[77]. The mean transverse sinus stenosis gradient before stent placement was 20 mmHg. In all patients the stent eliminated the pressure gradient, improved IIH symptoms and abolished papilloedema. Similar improvements have been reported in other studies[78-81].

Technique

Venous stenting is typically performed under general anaesthesia to limit patient movement and because dural stretching during deployment of the stents causes pain. Patients are treated with dual

antiplatelet therapy preoperatively and some units use platelet function analysers to establish the response to this therapy[77]. Intraoperative anticoagulation with heparin is used but protocols differ: some report targeting an activated clotting time of 250 seconds[78], while others aim for a doubling of baseline activated clotting time[77,80]. After a venous roadmap is obtained via femoral venous access, the catheter is advanced across the stenosis and a stent (sized according the normal sinus diameter) deployed. Venous sinus manometry is performed before and after stent placement to confirm appropriate reduction of the pressure gradient. Finally, angiography is performed to assess patency of the arterial branch and venous flow through the stented segment.

Complications

The complications of this procedure include acute stent thrombosis, venous sinus rupture, stent migration and the development of stenoses proximal to the stent. Other perioperative complications include transient hearing loss[77] and retroperitoneal haematoma[78]. In the largest series, two patients required urgent craniotomy: one for subdural haematoma evacuation following guidewire perforation and the other for intracerebral haemorrhage contralateral to the stent placement. These complications highlight the need to have immediate access to neurosurgical services and to be prepared for rapid transfer to a surgical operating room. Postoperatively patients often have ipsilateral headaches due to dural stretch and these typically resolve over the course of one week[77].

Anaesthetic considerations

The type of general anaesthesia used has not been reported in any of the studies of venous stenting. The presence of known elevated ICP in these patients behoves the exercise of diligence in the avoidance of factors that may further increase ICP and compromise cerebral perfusion (i.e. avoid hypercarbia, hypoxia, hypotension and acidosis). Moreover, the maintenance of the patient's preoperative blood pressure to ensure an adequate cerebral perfusion pressure is important to avoid cerebral hypoperfusion and causing a reflex vasodilatory response. Standard intraoperative care of the patient with a raised ICP is also recommended. While there are no data to support the use of an intravenous anaesthetic technique for this condition, it is preferred to a volatile anaesthetic technique to avoid cerebral vasodilatation and to optimise flow-metabolism coupling. There is limited experience with electrophysiological monitoring for this condition[78]. Invasive arterial monitoring in addition to routine monitoring is also recommended.

Carotid artery stenting

Background

Carotid artery stenting (CAS) involves the placement of an endovascular expandable stent into a stenosed carotid artery as a less invasive means of carotid revascularisation than carotid endarterectomy (CEA). Originally approved by the US Food and Drug Administration in 2004 for symptomatic patients with carotid artery disease and considered at high risk for surgery, recent evidence suggests that CAS is associated with higher stroke and death rates than CEA but lower myocardial infarction rates. The recent carotid revascularization endarterectomy versus stenting trial (CREST) is the largest randomised controlled trial to date comparing CEA and CAS and found no difference in the estimated four-year rates of the composite endpoint of death, stroke and myocardial infarction between CEA and CAS in patients with symptomatic and asymptomatic carotid artery disease[82]. Peri-procedural stroke rates were higher in the CAS Group than the CEA Group (4.1 vs 2.3%, $P=0.01$) but myocardial infarction rates were lower in the CAS Group (1.1 vs 2.3%, $P=0.03$). The International Carotid Stenting Study, a large prospective multicentre randomised controlled trial, compared CAS with CEA with a primary outcome measure of three-year rate of fatal or disabling stroke[83]. Interim results at 120 days showed higher rates of any stroke and all cause death in the CAS group. The incidence of stroke, death or peri-procedural myocardial infarction was higher in the CAS group than the CEA group (8.5 vs 5.2%, hazard ratio 1.69, $P=0.006$). A recent meta-analysis that included the aforementioned trials found that CAS is associated with a higher risk of any stroke (relative risk 1.45, 95% confidence interval 1.06–1.99), a decreased risk of peri-procedural myocardial infarction (relative risk 0.43, 95% confidence interval 0.26–0.71) and a non-significant increase in mortality (relative risk 1.40, 95% confidence interval 0.85–2.33) compared to CEA[84,85]. Similar conclusions were reached independently in another recent meta-analysis[95]. It is likely that CAS will continue to have a place in patients who are at high risk of perioperative myocardial infarction or with anatomical contraindications to CEA (contralateral vocal cord paralysis, previous radiation or ablative neck surgery or common carotid artery stenosis below the level of the clavicle)[86]. The implication for anaesthetists is that patients presenting for CAS are high-risk patients who require diligent perioperative management.

Thromboembolic complications during CAS arise as a result of intimal injury that stimulates platelet

activation and aggregation, and precipitates thrombus formation. Although there is currently no consensus[87], dual antiplatelet therapy with aspirin and clopidogrel is supported by extensive experience in coronary stent patients and is typically started preoperatively and continued for at least 3–6 months postoperatively. The optimal duration of this treatment has yet to be determined.

The use of point of care platelet function analysis preoperatively to assess response to aspirin and adenosine diphosphate receptor inhibition by clopidogrel is becoming more commonplace. Awareness of clopidogrel resistance is emerging as a significant problem in a subset of patients who lack the enzyme required to metabolise clopidogrel to its active metabolite[88]. Use of this technology enables identification of a group of patients who are at high risk of stent thrombosis. Glycoprotein IIb/IIIa receptor antagonists and prasugrel, a new and more potent adenosine diphosphate receptor antagonist with lower resistance rates, have both been used in patients who demonstrate absent or reduced adenosine diphosphate receptor blockade despite clopidogrel use.

Technique

The following techniques are commonly employed[91]. Carotid, and often cerebral, angiography is performed prior to CAS to plan treatment[89]. A guidewire with a filter, or embolic protection device, is placed beyond the carotid stenosis to capture embolic plaque material that is released when the stent and balloon are deployed (Figure 1). A balloon inflatable stent is then advanced over the wire into position at the level of the stenosis. Once the position of the stent is confirmed radiologically, the balloon is inflated to dilate the stenotic segment of carotid artery and deploy the stent. A repeat angiogram is performed to confirm the stent position and the adequacy of the dilation of the artery. Occasionally a second balloon inflation is performed to achieve greater dilation. Finally, the embolic capture device is removed and the femoral artery puncture site is closed. Intraoperatively, patients are heparinised to achieve an activated clotting time of two to three times the baseline levels.

Complications

Other than periprocedural stroke and myocardial infarction, minor complications include femoral insertion site haemorrhage, bradycardia and hypertension. Stent fracture and vocal cord paralysis, possibly from plaque embolus or direct pressure effect, have also been reported[90].

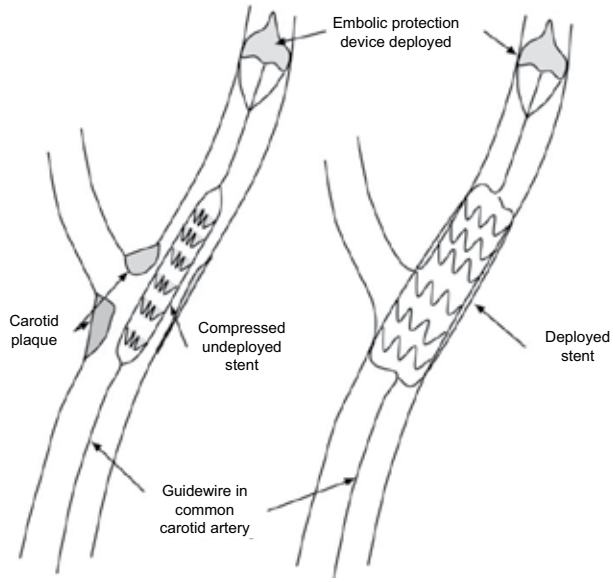

Figure 1: Carotid artery stenting technique. The guidewire with a filter (embolic protection device) to capture embolic plaque material, which is released when the stent and balloon are deployed.

Anaesthetic considerations

Performing CAS under conscious sedation allows continuous neurological monitoring and avoids the risks of general anaesthesia in this high-risk patient group. This must be balanced against the favourable operating conditions and improved patient comfort provided by the latter technique. Monitored anaesthesia care using midazolam, fentanyl and propofol have all been used with success. Dexmedetomidine, an alpha-2-agonist that provides titratable sedation, sympathetic modulation and improved maintenance of upper airway tone offers a potentially appealing alternative for these cases[91].

Bradycardia and hypotension occurring from carotid body stimulation during balloon inflation is well-described and occurs in one-third of patients. The prophylactic use of atropine prior to angioplasty can reduce bradycardia and perioperative cardiac morbidity, although the ability of the patient to tolerate tachycardia is an important consideration[92]. Placement of temporary cardiac pacing wires is an alternative option in patients with pre-existing cardiac conduction defects and with poor baseline cardiac function[93,94]. An external defibrillator with pacing functionality should be immediately available.

Stroke thrombolysis

Background

Stroke remains a leading cause of death and adult disability with an estimated prevalence of 3% for adults in the USA over 20 years of age[95]. Intravenous

thrombolysis with recombinant tissue plasminogen activator (r-TPA) is an established treatment for acute ischaemic stroke. It was first approved in the USA by the Food and Drug Administration in 1996 following the National Institute of Neurological Disorders r-TPA trial. This trial demonstrated that patients with acute ischaemic stroke treated with thrombolysis within three hours of symptom onset were 30% more likely to have minimal or no disability at three months than those treated with a placebo[96]. In 2008 the window for treatment was extended to 4.5 hours from time of symptom onset based on a further randomised controlled trial[97] and an updated analysis of the original Safe Implementation of Treatments in Stroke audit[98]. Pooled analysis of these studies showed an improved outcome the earlier thrombolysis was performed[99]. Nevertheless, the outcomes of thrombolysis remain modest with approximately half of patients either dying or not recovering completely[100].

Intra-arterial thrombolysis is an alternative to intravenous thrombolysis for patients with major stroke of less than six hours duration due to occlusion of the middle cerebral artery who are not otherwise candidates for intravenous r-TPA (e.g. patients who have had recent surgery)[101]. A study comparing these two modalities has been completed (www.clinicaltrials.gov NCT00640367) and should clarify the role of the two treatments.

Mechanical thrombectomy is an emerging option developed as an adjunct to thrombolysis or as first line stand-alone treatment for large vessel occlusive stroke[102]. Small studies have demonstrated the technical feasibility and success of these devices. Mechanical retrieval devices that are designed to capture and retrieve clot (Merci Retrieval Device, Concentric Medical, CA) or to aspirate and retrieve residual clot (Penumbra System, Penumbra Inc, CA) are available. More recently, retrievable stents, such as the self-expanding Solitaire stent (ev3 Inc, Plymouth, MN), have been developed to provide a way to recanalise the occluded segment. A stent is deployed to allow more rapid restoration of flow and this is then withdrawn minutes later with the clot attached, thus leaving no mechanical device in situ. While early results have demonstrated the technical feasibility and procedural safety of these devices, the clinical safety and outcomes of these techniques require further study[103-106].

Technique

Intravenous thrombolysis is delivered by peripheral intravenous infusion of r-TPA (dose 0.9 mg/kg). Ten percent of this dose is administered as a bolus, followed by infusion over 60 minutes. Intra-arterial thrombolysis requires selective catheterisation of the target vessel and the delivery of a more concentrated dose of thrombolytic medication.

Complications

Symptomatic intracerebral haemorrhage rates of 1.6 and 2.2% and three-month mortality of 12.2 and 12.7% of patients treated within three and 4.5 hours, respectively, were reported in the Safe Implementation of Treatments in Stroke-International Stroke Thrombolysis Register[98]. Mechanical thrombectomy is associated with ICH rates of 10–28% in the small series published to date[104,105,107]. Complications associated with femoral arterial cannulation can also occur.

Anaesthetic considerations

Blood pressure management in acute ischaemic stroke remains controversial due to mixed results from numerous observational and interventional studies[108]. The principle of maintaining adequate cerebral perfusion, particularly in the area of the penumbra of an evolving stroke to limit infarct size, has led the American Stroke Council to recommend avoiding hypotension, tolerating elevated systolic and diastolic blood pressure, and only treating hypertension if systolic blood pressure is >220 mmHg and diastolic is >120 mmHg, or in the setting of thrombolysis if systolic blood pressure is >180 mmHg and diastolic blood pressure is >105 mmHg[101]. If the patient is hypotensive, cerebral perfusion in ischaemic areas, which may have impaired autoregulation, can be improved by induced hypertension. The importance of avoiding hypotension in these patients was shown in a recent retrospective series that found that a systolic blood pressure <140 mmHg was associated with a worse neurological outcome in acute stroke patients undergoing endovascular therapy[109]. This is further supported by the finding that induced hypertension can increase cerebral perfusion pressure and ipsilateral cerebral artery flow velocities (implying loss of autoregulation) in patients with large hemispheric strokes[110]. Following thrombolysis, the active treatment of hypertension is supported by evidence from a retrospective analysis of the large Safe Implementation of Treatments in Stroke register that showed a strong association between high systolic blood pressure from 2–24 hours post-thrombolysis and poor outcome[111]. In this study, systolic blood pressure 141–150 mmHg was associated with the best outcomes. Consequently active treatment should be considered and commenced promptly and continued postoperatively[111].

There is no clinical evidence to support any particular anaesthetic agent or technique in the

management of acute stroke patients. Although data from mice suggested that ketamine is associated with a reduction in infarct volume[112], clinical evidence for effective pharmacological neuroprotective strategies remains elusive. Extrapolating from evidence in traumatic brain injury, hypoglycaemia, hyperglycaemia and hypocapnia must be avoided. Hyperthermia (>37.2°C) is associated with poor outcome in patients with stroke and is correlated with severity of stroke and inflammation[113]. Therefore, temperature monitoring should be routine.

Intracranial stenting and angioplasty

Background

Intracranial stenosis is an important risk factor in stroke and may account for 5–10% of ischaemic strokes in mixed populations[86], and up to 30–50% African American, Hispanic[114] and Asian[115] populations. Intracranial stenoses are present in over 40% of patients who have had fatal strokes[116] and are associated with a recurrent stroke risk in the territory of the stenotic vessel of up to 38% over two years despite medical therapy[117]. The management of these patients thus remains a challenge and intracranial angioplasty and stenting has emerged as a feasible option. The SAMMPRIS (stenting and aggressive medical management for preventing stroke in intracranial stenosis) study was terminated early after the randomisation of 451 patients due to a higher rate of stroke or death in the stent group versus the medical management group (14.7 vs 5.8%, $P=0.002$)[118]. The study was a large prospective randomised trial of patients who had a recent transient ischemic attack or stroke attributed to a 70–99% stenosis of a major intracranial artery. Patients were randomised either to aggressive medical management alone (comprising dual antiplatelet therapy, statins and antihypertensive therapy where indicated) or aggressive medical management plus percutaneous transluminal angioplasty and stenting. The probability of stroke or death at one year was 20% in the stent group and 12.2% in the medical management group. Based on this finding it seems likely that the current guidelines recommending that stenting be considered for patients with symptomatic severe (>70% luminal narrowing) intracranial stenosis despite maximal medical therapy[1] need further evaluation and revision. Whether a subset of these patients may benefit from this treatment remains to be elucidated.

Technique

All patients in the SAMMPRIS trial were treated with dual antiplatelet therapy for a period of 90 days after enrolment. Blood pressure was actively managed preoperatively to achieve a target of <140/90 mmHg, or <130/80 mmHg in diabetics. Heparin was administered intraoperatively to maintain an activated clotting time of 250–300 seconds, and reversed with protamine at the proceduralist's discretion. All procedures were performed under general anaesthesia to ensure immobility. The procedure involved balloon dilatation of the lesion and then deployment of a stent at least 6 mm longer than the lesion. Intra-procedural blood pressure targets were a systolic blood pressure of <150 mmHg and diastolic blood pressure <95 mmHg. Technical success was considered a residual stenosis of <50%.

Complications

Acute complications of this procedure included intracranial haemorrhage due to vessel perforation, acute thrombosis and groin haematoma. The SAMPRISS trial reported a 30-day death or stroke rate of 14.7% among patients randomised to intracranial stenting but did not report acute periprocedural events[118]. The complication rates from two recent studies provide an estimate of the frequency of peri-procedural events. In one study, complications occurred in eight of 66 patients, including acute distal thrombosis resulting in stroke in two patients, two parent vessel dissections without neurological deficit plus one resulting in death, a vessel perforation resulting in death and groin haematomas in two patients[119]. A second study of 40 patients reported seven neurological complications, five (10.5%) suffered permanent morbidity (four strokes) or mortality (one death)[120].

Anaesthetic considerations

In the SAMMPRIS trial stenting was performed under general anaesthesia. In the earlier Stenting of Symptomatic Atherosclerotic Lesions in the Vertebral or Intracranial Arteries trial[121] the procedure was performed under general or local anaesthesia. While most centres employed general anaesthesia, the inability to detect early clinical neurological deficit is a major disadvantage as it delays management to prevent progression to a permanent deficit. The benefit of general anaesthesia is that it provides patient immobility and facilitates more accurate mapping of the vasculature. However, a few centres have demonstrated the feasibility of avoiding general anaesthesia. A retrospective review of 66 patients treated for elective intracranial stenting reported the use of mild sedation with midazolam and fentanyl titrated to a Ramsay sedation score of 2–3. Conversion from sedation to general anaesthesia was required in two patients[119]. The conversions to general

anaesthesia were due to an acute vessel perforation and excessive movement. The authors reported that 4.9% of patients developed neurological deficits requiring alteration of the endovascular technique or postoperative management to avoid permanent sequelae. Two patients had permanent neurological deficits and the mortality rate was 3.2%. In another prospective series, thirty-seven patients were treated under local anaesthesia[120]. Intraprocedural symptoms leading to an alteration of interventional technique occurred in 61.4% of patients. Headaches were the most common symptom, which, when persistent, heralded the occurrence of subarachnoid haemorrhage. Focal deficits suggestive of cerebral ischaemia occurred in three patients and were treated with blood pressure augmentation, and in one case, thrombolysis.

Blood pressure management to avoid inadequate perfusion in these patients with known symptomatic stenoses is critical. Just as important is the avoidance of excessive hypertension that increases the risk of peri-procedural haemorrhage and reperfusion injury. In the SAMPRISS trial intra-procedural blood pressure was treated if systolic blood pressure was >150 mmHg or diastolic blood pressure was >95 mmHg.

General issues

Cerebral monitoring

The use of neuro-physiological monitoring including electroencephalography, SSEP and/or brainstem auditory evoked potentials was assessed in a series of 35 patients who underwent endovascular cerebral aneurysm treatment[122]. Neuromonitoring changes were observed in 26% of the patients, altered treatment in 14% of patients and provided false negative results in at least two (6%) of the patients. In this study no patients in whom management was altered based on monitoring abnormalities suffered neurological deficits during the procedure but six patients subsequently developed neurologic deficits 15 hours or more post procedure. Neither this study, nor a more recent study of 63 patients undergoing endovascular coiling of aneurysms, which showed a high false positive rate for clinically and angiographically confirmed ischaemia[123], allowed determination of the sensitivity or specificity of monitoring for the detection of cerebral ischaemia. This is because the physiological monitoring changes were always acted upon and there was no control arm. Therefore, the evaluation of the angiographic findings should guide decision-making during the procedure.

There are anecdotal reports of the use of SSEP and motor evoked potentials together with provocative barbiturate injection to guide embolisation treatment of AVM, tumours and aneurysms, but there is insufficient evidence to make firm recommendations[124]. There is also little evidence currently to support the routine use of transcranial cerebral oxygenation monitoring[125].

Ionising radiation protection

Ionising radiation in interventional neuroradiology suites is a hazard to both patients and staff. Cancer and genetic injury are unpredictable side-effects of ionising radiation because there is no known safe lower limit of exposure[126]. In contrast, non-mutagenic radiation injury—including somatic effects such as cataracts, erythema and desquamation—only occurs above a threshold level, above which the risk is dose-dependent.

The occupational limits are based on the International Commission on Radiological Protection guidelines[127]. In the European Union, this is implemented as an effective dose limit of 20 mSv/year (averaged over five years), with an annual maximum limit of 50 mSv. In the USA it is implemented as an annual occupational limit of 50 mSv and a lifetime limit of 10 mSv multiplied by age in years[128].

There are several case reports of radiation-induced skin injury in patients associated with interventional neuroradiology procedures[129]. Systems to monitor skin doses, using a fitted dosimetry cap, to prevent radiation induced skin injury to patients who may undergo prolonged and possibly multiple procedures have been developed and described[130].

Three principles should be applied to protect staff from radiation: maximise distance from the radiation source, limit the exposure time and utilise adequate radiation protection. First, the intensity of radiation is proportional to the square root of distance from the source, hence doubling the distance from the source will reduce the exposure by a factor of four. The establishment of a remote anaesthetic monitor in the control room will limit radiation exposure to anaesthesia staff by increasing the distance from the source. Second, limiting exposure time for all staff is important. While the duration of imaging is under the control of the proceduralist, imaging should be stopped when physical attendance to the patient is required. Third, protective radiation barriers must be used. Lead aprons and thyroid shields must be worn in the procedure rooms. However, they do not provide total body coverage or complete protection from radiation scatter. As personnel should never be in the direct line of the X-ray beam, the majority of exposure to radiation occurs due to scatter. Most of the scattered radiation comes from the surface of the

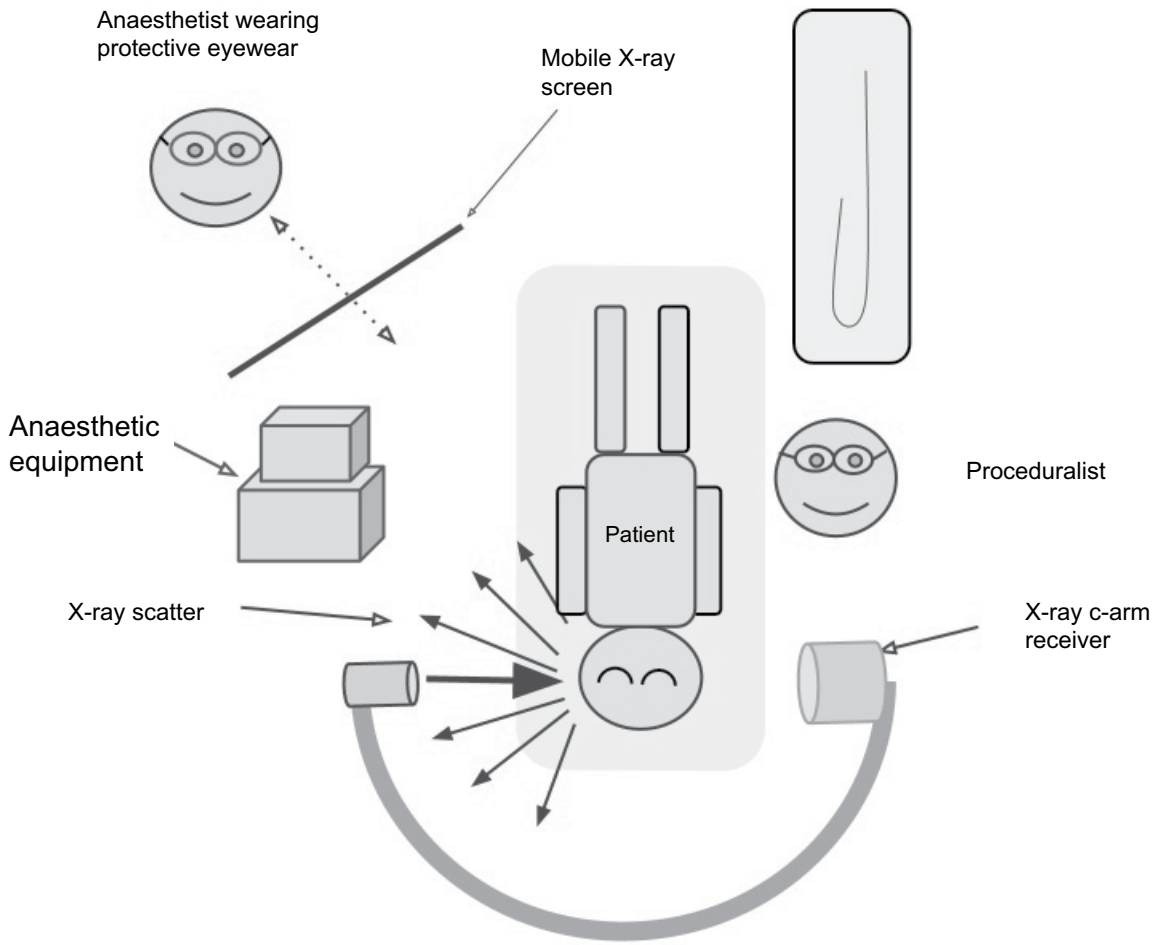

Figure 2: Suggested layout of interventional radiology procedural room showing the positions of the anaesthetist, proceduralist, patient and X-ray source.

patient nearest the X-ray source. To minimise this exposure, it is advisable to remain on the receiver side of the X-ray arm. This means that when horizontal views are used, staff should, if possible, remain on the image intensifier side of the arm or remain behind fixed or mobile radiation barriers (Figure 2). When vertical planes are used, the X-ray source should be below the patient so that most scattered radiation will be reflected on to the floor. Mobile shields on rollers provide excellent radiation protection and should be available in these locations.

The current recommended annual equivalent dose limit for eyes is 150 mSv/year[128]. Protective lead eyewear is routinely recommended for interventional radiologists but is not yet recommended for anaesthesia staff. This is despite significant ocular radiation exposure in anaesthetists during interventional neuroradiology procedures[131]. The use of protective eyewear by anaesthetists who regularly work in interventional neuroradiology suites would seem to be prudent.

The guiding principle for radiation exposure is the 'as low as reasonably achievable' principle: i.e. minimising exposure time, maximising distance from the X-ray source and scatter, utilisation of shields and monitoring of personal exposure through dosimeter use. These, along with education and training, are essential for the safe practice of anaesthesia in these procedural areas[128]. The International Atomic Energy Agency provides online information and training material on radiation protection for medical personnel, available at https://rpop.iaea.org.

Other issues

Temperature management is important to consider in the interventional neuroradiology suite as these procedures can often be long, and active measures to maintain normothermia should be routine in these patients. Shivering during and after sedation for interventional neuroradiology can cause problems with movement, patient discomfort and noncooperation, and an increase in oxygen consumption. Clonidine and nefopam (an non-opioid analgesic)

have been demonstrated to be effective in reducing the incidence of intraoperative shivering compared to placebo (6, 29 and 77%, respectively)[132]. Diligent positioning of patients, particularly when prone or lateral positioning is required, is important to prevent nerve injury. Urinary catheterisation should be routine in procedures likely to be lengthy. Detailed attention to deep vein thrombosis prophylaxis with use of intermittent calf compressors and appropriate pharmaco-prophylaxis should also be considered.

Contrast-induced acute kidney injury and allergic reactions

As there are no data for the prevention of contrast-induced acute kidney injury (AKI) in interventional neuroradiology, data extrapolated from coronary interventional procedures may be used as a guide. Contrast-induced AKI is a common cause of hospital-acquired kidney injury and is associated with increased hospital morbidity and mortality in patients following coronary angiography[133,134]. Renal toxicity from iodinated contrast media is multifactorial comprising cell toxicity due to iodine, osmolality and ionic strength; vascular dysfunction resulting in vasoconstriction and ischaemia; oxidative stress due to nitric oxide deficiency; and renal tubular dysfunction[135]. It occurs in over 10% of patients undergoing coronary angiography[136] and is more common with pre-existing renal impairment, >75 years of age, diabetes mellitus, increased contrast volume, hypotension and cardiac failure. Increases in serum creatinine are detectable 24–48 hours after contrast exposure and peak at five to seven days post-exposure[137]. Current evidence supports the use of hydration, minimising contrast media volume and the use of lower osmolar contrast media in the prevention of contrast-induced AKI[138,139]. The use of N-acetylcysteine and isotonic bicarbonate combined may reduce the occurrence of contrast induced AKI overall by 35% but not the rate of dialysis-dependent renal failure[140]. These are low risk interventions that may prevent AKI and should be considered in patients at high risk of AKI.

CONCLUSION

As the scope and complexity of interventional neuroradiology procedures continues to advance, anaesthetists are assuming an increasingly important role in the multidisciplinary management of these cases. The remote location, risk of radiation exposure, limited space, complexity of patient, and novel relationship between radiologist and anaesthetist make anaesthetising in the interventional neuroradiology suite both challenging and stimulating. A comprehensive understanding of the underlying pathology in patients, and the technical aspects of the procedures is imperative to the provision of quality anaesthetic care and maintenance patient safety.

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