Endovascular Management of Cerebral Aneurysms

L van Zyl

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I) Introduction:

Interventional neurovascular procedures are part of a trend towards performing minimally invasive neurosurgery. It is an evolving clinical speciality in which neurointerventional radiologists and endovascular neurosurgeons are the key role players.

Percutaneous radiological techniques are used to gain vascular access for the purpose of delivering therapeutic drugs and devices. Neuroradiologists have extended their treatment modalities in the field of endovascular neurosurgery to such an extent, that they can now access almost any vessel of the neurosystem.

The growth in this hybrid of traditional neurosurgery and neuroradiology has been facilitated by rapidly emerging and re-engineered neuroradiological techniques and sophisticated catheter developments.

More procedures have moved out of the operating room into the neuroradiology suite. Procedures have become increasingly complex, and the neurointerventional suite has become a sub-specialized operating room, with cases being done both electively and as emergencies. Procedures are less invasive and less painful, but can be lengthy, and the patient population is ageing, many having major medical comorbidities.

The anaesthetist has a crucial role in facilitating neuroradiological procedures, and as the volume of cases done endovascularly increases, so will the need for anaesthesia assistance. Anaesthesia for these procedures can be challenging, and we need to familiarize ourselves with the technicalities of the procedures, their potential complications, and management thereof.

II) Anaesthesia Outside the Operating Room:

Optimal management of procedures being performed in the neurointerventional suite requires forethought and careful planning.

Open channels of communication should be maintained between the neuroradiologist, anaesthesiologist, nurses and radiographer at all times. This is essential for provision of routine care and crucial for management of complications.

The OR model of anaesthesia care should be extended to procedures being performed in the interventional radiology (IR) suite.

This includes adequate pre-procedural assessment, standardized monitoring and equipment, clearly marked and easily accessible resuscitation and difficult airway equipment, adequate back-up in case of emergencies or difficulties, and postoperative care in a post-anaesthesia care-unit setting.

Skilled anaesthesia assistance and dedicated recovery staff should be available.

Suboptimal conditions in the IR suite include the unfamiliar environment, dim lighting, radiology staff unfamiliar with anaesthetic practices, and the fact that swift help is not easily available.

Despite recent improvements, the problem of inadequate anaesthesia provision for interventional neuroradiology remains. Identified problems include inadequate facilities for induction and recovery.

Consideration should also be given to the design of the neuroradiology unit itself, as well as the hospital layout, to allow efficient integration and easy accessibility of the unit with recovery and critical care units, operating theatres, wards and the emergency department.
Intra-hospital transfers need to be performed safely and speedily, whilst closely observing the patient’s clinical status, lines, tubes, ventriculostomy and equipment.

A survey was conducted by Webb and Farling (36), to assess provision of peri-operative care for patients with aneurysmal subarachnoid haemorrhage (SAH) in the UK and Republic of Ireland. It identified lack of consultant neuroanaesthetists as the most common problem reported by respondents. The need for recruitment into the speciality has previously been stressed. (36) The need for better neuroscience critical care units was also emphasized. (36)

It was previously standard practice to maintain very cool temperatures in the IR suite, as these were thought to be beneficial for the fluoroscopy equipment. After consultation with manufacturers, it was determined that the temperature tolerance of the equipment was 20 degrees Celsius, so the thermostats in the IR suites were reset to this temperature. This addressed the anaesthetists’ concerns that the low ambient temperatures were unnecessarily increasing the risk of hypothermia, especially in elderly and sick patients. (25)

Due to the design of the IR suite and its required equipment, the traditional position of the anaesthetist and anaesthesia equipment at the head of the bed may be difficult to maintain. The image intensifier needs to be around the head end of the fluoroscopy table, making airway access difficult.

Options are:

- Positioning the anaesthetist opposite the neuroradiologist and towards the patient’s feet.
- Facing the patient’s side where the neuroradiologists are working.

The anaesthetic machine and equipment are placed at the anaesthetist’s side and connected to the patient by long extensions. This allows the anaesthetist to observe the patient, the interventionalist, and the information interfaces.

Unobstructed view of the patient’s head and good access to the patient are vital.

Equipment and monitors used by the neuroradiologists and anaesthetists need to be arranged in a convenient manner that does not cause hindrance to other members of the team.

**III) Radiation Safety:**

Personnel working in the IR suite risk exposure to ionizing radiation.

Sources of radiation include direct radiation from the X-ray tube, leakage radiation through collimators and protective shielding, and scatter radiation reflected from the patient and the surrounding area.

Regulatory agencies publish and regularly update annual allowable limits for maximum exposure for health-care workers.

The International Commission on Radiological Protection (ICRP) has recently released new guidelines and lowered the recommendation to 20mSv per year.

All staff should wear dosimeter badges that are checked monthly or quarterly.

Protective shielding should be worn and includes knee length aprons at least 0.5mm thick that are regularly checked for cracks, thyroid collars, moveable transparent glass lead screens, as well as protective eyewear due to the significant levels of ocular radiation.

Some IR suites have facilities for anaesthetists to monitor the patient from a distance, usually an adjoining console area.
If this facility is not available, sit as far away from the patient as possible during the procedure, at least 4 feet away from the radiation source, as ionizing radiation follows the inverse square law. This means that the amount of radiation exposure drops off proportional to the square of the distance from the source. Activity at the patient’s head should thus be minimized during fluoroscopy.

Another protective measure is to minimize the total time that ionizing radiation is used during the procedure.

**IV) Neuroradiological Imaging:**

High-speed digital subtraction angiography (DSA) and fluoroscopic technology are utilized. DSA delivers considerably more radiation than fluoroscopy.

The vascular anatomy is visualized by injecting a bolus of contrast agent into the circulation through a guide catheter in the internal carotid or vertebral artery and “road-mapping” the vasculature. Road-mapping facilitates the placement of superselective catheters into the distal circulation.

A scout film is taken before each sequence of fluoroscopy to remove bone shadows and other non-vascular structures from images. The scout film serves as a mask, which is subtracted by the computer from all subsequent images, so that only vessels opacified by contrast are visible.

The computer then superimposes the road-mapped image onto real-time high-resolution bone subtracted fluoroscopy. This enables the radiologist to follow the progress of the radiopaque microcatheter tip through the vascular circulation.

Patient movement severely degrades the road-mapped image and risks aneurysm rupture if the microcatheter tip is in the aneurysm at the time of movement. Patient immobility is thus preferable to decrease movement artefact.

**V) Radiological Vascular Access:**

The aim of obtaining vascular access is to facilitate percutaneous introduction of catheters into the arterial circulation of the head, neck or spine. The transfemoral arterial approach is usually used, but one can use direct carotid or brachial puncture.

To perform diagnostic angiography, the femoral puncture site is infiltrated with local anaesthetic. A large introducer sheath (6 French) is then inserted into the femoral artery. An end-hole catheter (4-6 F) is then manipulated through the introducer under fluoroscopic guidance, into the ipsilateral internal carotid or vertebral arteries. A superselective microcatheter (1.2- 2.8 F) is introduced through the guide catheter into the cerebral circulation.

The flexible microcatheter allows navigation to the distal intracranial and spinal circulation and is used to deliver drugs or embolic agents. Modified microcatheters are employed during balloon angioplasty.

**VI) Cerebral Aneurysms:**

An aneurysm is a localized dilatation of a blood vessel. Cerebral aneurysms are most frequently localized in the anterior communicating artery (34%), the origin of the posterior communicating artery (23%), and the middle cerebral artery bifurcation (20%).

The incidence of cerebral aneurysms in the general population is 1.5- 8%. There is an increased incidence in patients who have a first-degree relative with an aneurysm. This represents a lifetime risk of 2- 5%. Multiple aneurysms occur in 20% of patients.
Aneurysms are classified into 3 categories:

- unruptured asymptomatic
- unruptured giant asymptomatic
- ruptured with or without concomitant vasospasm.

Treatment of unruptured aneurysms is largely restricted to those discovered incidentally, or those causing mass effects on adjacent structures eg the optic nerve. Screening policies have led to a significant increase in the number of diagnosed asymptomatic aneurysms.

Aneurysm rupture can be disabling and is lethal in 10-15% of cases. Sudden death is attributable to systemic derangements, direct brain damage, vasospasm, and complication of clipping and coiling. The incidence of rebleed in treated aneurysms is reduced, from 30% in untreated aneurysms, to 4% during the first 6 months following initial rupture. In a study of 100 patients followed over 2-6 years, the rate of rebleed was 0% for small, 4% for large, and 33% for giant aneurysms.

Aneurysms can present with symptoms of subarachnoid haemorrhage (SAH), cranial nerve palsies, seizures, cerebral compression, or hydrocephalus. 77% of acute, non-traumatic spontaneous SAH is attributable to aneurysms. SAH is a multisystem disease, and patients whose aneurysms present with SAH are the most unstable, with the highest risk of morbidity (50%) due to re-rupture, hydrocephalus, vasospasm and comorbid disease. If the initial bleed is survived, the risk of re-rupture during the first 24 hours is 4%, and 1% per day thereafter.

Definitive management involves excluding the aneurysm from the circulation to prevent re-bleeding. This is done after the initial resuscitation of the patient.

Surgical clipping had been the established treatment modality for both ruptured and unruptured aneurysms until the introduction of GDC embolization in 1991.

Choice of technique is dependent on anatomical location, dimensions and size (small: <12mm, large: 12-24mm, giant: >24mm), aneurysm neck, and configuration/geometry of the aneurysm. Advanced imaging includes a 3-D view of the lesion and its surrounding vessels. Once all the measurements have been made, a decision is made regarding the feasibility of safely coiling the aneurysm, and which approach to take. Aneurysm stenting technology continues to evolve, implying that aneurysms with complex vascular architecture can now be considered for endovascular management.

In patients with very wide aneurysm necks, surgical clipping is preferable, attaining a total occlusion rate of 15-35%. In necks of <4mm, endovascular occlusion is especially successful, achieving complete thrombosis in 57-85%.

Surgical clipping may be preferable for aneurysms with very difficult angiographic anatomy eg vessels arising from, or in close relation to aneurysmal neck, patients with difficult vascular access, or where there is an associated large haematoma.

In poor WFNS grade patients not suitable for clipping, endovascular occlusion may be an option, but randomized evidence is lacking and management thus has to be individualized.

Clipping might be preferable for poor grade patients where brain swelling and vasospasm might deter surgeons from craniotomy.

Indications for coiling further include medically unstable patients, those with poor neurological grade, early vasospasm, unruptured aneurysms and multiple aneurysms in different arterial territories.

Even for complex aneurysms, efficacy is at worst equal to clipping.
Morbidity and mortality (3%) rates related to embolization of acute aneurysms are lower than those associated with untreated ruptured aneurysms. Selection of aneurysms appropriate for coiling has also improved. Coiling can safely be done within hours of rupture, with low probability of aneurysm perforation (40).

The International Subarachnoid Aneurysm Trial (ISAT)(10), was a prospective randomized multicentre clinical trial comparing safety and efficacy of endovascular coil treatment and surgical clipping for the treatment of acute ruptured aneurysms. 2143 patients were randomized. Inclusion criteria were low-grade SAH scores (WFNS 1 and 2) and small anterior cerebral circulation aneurysms. Posterior circulation aneurysms have traditionally preferentially been managed with coiling. (28)

Patients in the coiling group had better outcomes, if measured as survival and development of seizure states, at 1 year. At 1 year, 30.6% of surgical subjects had died or were dependent, compared to 23.7% of the coiling subjects. This means that the relative risk of death or significant disability at 1 year was 22.6% lower in the coiling group, with an absolute risk reduction of 6.9%. This represents 69 patients avoiding death or dependency at 1 year for every 1000 patients treated. Follow-up data indicated that this advantage was still evident after 7 years. The risk of re-bleeding during the first month was higher in the endovascularly treated group. Case mortality rates were similar in the 2 groups, 10.1% in the clipping, and 8.1% in the coiling group. No significant cost differences were present at 1 or 2 year follow-up.

Criticisms of the study are that mostly good grade SAH patients with small anterior cerebral artery aneurysms were included, and that there was no long-term follow-up.

The better results after endovascular treatment reported in ISAT have led to more patients undergoing coiling of their aneurysms, thereby increasing the anaesthetist’s role in the IR suite.

An ISAT sub-study, N-ISAT, evaluating neuropsychological assessments at 1 year follow-up, has shown that cognitive impairment was more prevalent in the clipping group.

Analysis of a further subgroup, the elderly, suggests that patient age is a very important consideration when deciding on suitability for coiling or clipping.

The Society of British Neurological Surgeons (SBNS) and the British Society of Neuroradiologists (BSNR) have produced a report following publication of ISAT results stating that “where both neurosurgeon and neuroradiologist agree that either (neurosurgical or endovascular) treatment are equally feasible, in the light of currently available data, coiling is the treatment of choice”. (36)

This concurs with the Cochrane Collaboration’s current statement that, in patients with good grade SAH, presenting with aneurysms that could be treated either by clipping or coiling, coiling results in a better outcome.

Koivisto (3) studied 109 patients with acute SAH and observed comparable survival and recovery rates, as well as neuropsychological test scores. However, there was a statistically significant increase in superficial brain retraction injuries and ischaemic lesions among the surgical patients.

Hadjivassiliou (3) also observed less structural damage in the endovascular group.

Johnston (3) noted a significant increase in adverse outcomes, length of hospital stay and hospital charges in the surgical group of patients with unruptured aneurysms. In another study he found that surgically treated patients were more likely to experience residual neurological symptoms and disability, as well as having longer recovery periods.

Major advantages of coiling are avoidance of a craniotomy and quicker recovery.
Disadvantages include the potential for the aneurysm to reform, the observed possibility of greater incidence of re-bleeding, and the fact that the incidence of vasospasm is not reduced.

The presence of both endovascular and surgical treatment options has been shown to create better patient outcomes (3).

Endovascular procedures have a steep learning curve and operator experience is crucial in achieving optimal therapeutic results. Procedural outcomes are better at high volume institutions, where physician experience and practice is greater.

Cross (3) noted that odds of patient in-hospital death were 40% greater at hospitals with low-volume aneurysmal SAH treatment procedures.

Singh (3) noted that the risk of complications with coil embolization of unruptured aneurysms decreased dramatically with increasing physician experience. 53% of the first five cases in his study experienced complications, as opposed to only 10% in later cases.

1. Aneurysm Stent Technology:

Endovascular treatment of cerebral aneurysms dates back to the mid 1970s when Serbinenko introduced endovascular parent vessel occlusion (16). It became more appealing with the introduction of Guglielmi Detachable Coils (GDCs), which allowed occlusion of the aneurysm without interruption of distal blood flow (16).

Coils are attached to a pusher wire, which is used to advance the coil into position through the microcatheter that has been inserted through the femoral arterial guide catheter. The coils are not detached from the pusher wire until the operator is satisfied with their placement. If the position is suboptimal, the coils can be retrieved and redeployed, or removed and replaced by a more appropriate coil.

The aim of placing the coils is long term occlusion of the aneurysm and prevention of rebleeding. Occlusion is achieved by filling the aneurysm sac with a series of coils, where they act to initiate thrombus formation. The initial coil should be of a diameter that matches that of the aneurysm. It forms a basket that will be packed with progressively smaller coils.

Common deployment methods are:

- Electrical: GDC: the coil itself is made of platinum, which is malleable, does not conduct electricity, is MRI compatible and radiopaque. The coil is fused to a small, insulated stainless steel conducting wire. The detachable zone undergoes electrolysis and takes several minutes to detach fully. The detachment time becomes progressively prolonged as more coils are deployed.
- Mechanical: Cook coil. The disadvantage of this coil, is that one is unable to retract the coil once it has been completely advanced beyond the catheter tip
- Thermal: Micrus coil.

Recent advances include bioactive coils that are coated with materials such as polyglycolic acid, that promote thrombus formation and endothelial growth (Matrix coils).

Matrix coils, or coils with a PGA/PGLA coating are platinum coils coated with an absorbable polyglycolic-polylactic acid (PGLA) copolymer. 70% of the coil volume is made up of the PGLA coating, which is absorbed by the body within 90 days. The additional coating is responsible for the larger diameter of the coils.
Advantages are the increased packing density that is achieved, as well as the accelerated conversion of thrombus.

A disadvantage is, that it is not radiopaque, thus radiolucent gaps appear between adjacent coils, making it difficult to assess how tightly packed the aneurysm is.

Murayama (3) found that aneurysms treated with Matrix were 18% smaller on the 3 month angiogram vs the unchanged size of GDC treated aneurysms. Although the neck tissue thickness was found to be greater at 3 months, no difference was observed at 6 months. They concluded that aneurysm fibrosis and neointima formation was accelerated by Matrix coils, without causing parent artery stenosis.

Recent advances in gene therapy may allow for enhancement of direct gene transfer in situ by coated coils (3).

Not only coils are used for embolization, but cyanoacrylates, onyx embolic material, CAP and polyvinyl alcohol.

Cyanoacrylates: Histoacryl. This is a rapidly polymerizing adhesive that causes liberation of heat into surrounding tissues through an exothermic process. To avoid the catheter sticking, it has to be withdrawn immediately after the cyanoacrylate is injected.

Onyx: Microtherapeutics. This is a biocompatible non-adhesive liquid embolic agent. It consists of an ethylene vinyl alcohol copolymer dissolved in dimethyl sulphoxide (DMSO). A micronized tantalum powder is added to provide contrast for fluoroscopy. On contact with blood, it solidifies through precipitation. This process begins immediately after injection, forming a soft, spongy embolus that solidifies from the outside in. No heat is produced during the precipitation process. Because it is non-adhesive, injection and filling of the vascular abnormality can take place over several minutes, and concurrent angiography can be performed with the catheter left in place.

Perceived advantages are the ability to reach difficult anatomic locations, ability to penetrate a large number of feeding vessels in one injection, and more precise control when delivering material. Thus, better access, handling, delivery and visibility. Aneurysm occlusion rates with Onyx are superior to coil occlusion.

Limitations are the poor control over migration into the parent artery, occurrence of transient and permanent neurological deficits, SAH, massive reflux into afferent artery peduncle, incomplete filling of aneurysm sac, and imaging difficulties. It appears hypointense on MRI, interferes with MRA in patients with stents, and creates artefacts that hinder CT evaluation.

CAP: cellulose acetate polymer. This is also a liquid embolic material dissolved in DMSO.

Complications associated with its use are aneurysm rupture, parent artery stenosis, and strong chemical erosion with acute damage of the aneurysm wall and an inflammatory cell infiltrate. (3)

Polyvinyl alcohol particles: Contour. This facilitates temporary occlusion of vessels lasting days to weeks. It is mainly used for preoperative embolization of tumours.

Other advances in technology include coils with more complex shapes, multidimensional coils, softer coils, remodelling aneurysm necks using balloon catheters, and stent-assisted coil embolization. (3).

Stent assistance is useful in wide-necked and fusiform aneurysms, which involve entire vessels or are irregular in shape. In these aneurysms coils often migrate after deployment, with the potential to occlude parent vessels, or embolize. Use of stents has lead to improved aneurysm occlusion rates and fewer cases of parent vessel occlusion. Stents are also useful in basilar apex and other bifurcating aneurysms.
Self-expanding stents are used in a Y-configuration double stent-assisted technique. The microcatheter is manipulated through the stent mesh deployed across the aneurysm, into the aneurysm remnant, and coils then deployed sequentially, to occlude the remnant. This preserves arterial flow and maintains selective occlusion of the aneurysmal pouch.

Complications include stent malposition in large aneurysms, dislodgement during microcatheterization, stent movement after deployment or during coiling, vasospasm during stenting, and rupture of the artery.

Early versions of the Neoform stent were difficult to pass through tortuous vessels and risked migration because of their softness.

**Balloon assistance** is used to manage broad-necked aneurysms or aneurysms with a neck-to-body ratio close to 1. It is used to temporarily occlude parent vessels to determine adequacy of collateral flow, eg in carotid ophthalmic artery aneurysms.

A non-detachable balloon catheter is deployed across the aneurysm neck and the balloon is inflated when the coils are deployed. The balloon provides a temporary barrier across the aneurysmal neck to prevent coil migration or protrusion into the parent vessel. The coil is forced to assume the 3D shape of the aneurysm without impinging on the parent artery, and also stabilizes the microcatheter in the aneurysm during coil delivery. Furthermore, it allows denser intra-aneurysmal coil packing without compromising the parent artery.

Complications include vasospasm followed by haemorrhagic infarction, intra-arterial thrombus, subtotal or incomplete aneurysm occlusion, increased technical complexity, and temporary rise in intra-aneurysm pressure with possible resultant rupture.

**The Cerebral Rupture After Aneurysm Trial** (CARAT) \(^{(27)}\) reported a re-rupture rate of 2.2% at 1 year, and 0.2% at 2 years following endovascular intervention.

Potential future issues that need to be addressed are in-stent thrombosis (especially in clopidogrel resistant patients), stent migration and delayed recanalization. \(^{(27)}\)

The largest study on aneurysm stent technology to date showed, a 2% mortality and a 2.8% morbidity rate following stenting of 124 patients. \(^{(27)}\)

Previously discussed advances in stent devices and grafts could lead to a future trend of fewer aneurysms being embolized through coiling, with a move towards vessel preservation, flow diversion, and reconstruction of parent vasculature. \(^{(78)}\). Preliminary results appear promising, but efficacy and safety outcomes need further prospective randomized controlled trials, and this technique can currently only be recommended for unruptured aneurysms.

2. Anaesthetic Considerations:

As mentioned previously, the volume of patients being treated endovascularly is increasing, and the procedures themselves are gaining complexity, as those with more intricate aneurysm anatomy are presenting for embolization. The proportion of higher medical risk patients is also increasing. Anaesthesia thus requires careful planning, and should take the needs of the neuroradiologist, and the technical aspects of the procedure into consideration.

Patients may be relatively healthy with an incidentally discovered unruptured aneurysm, or may have marked derangements of various organ systems secondary to a devastating aneurysm rupture, and be dramatically unstable.
Following aneurysmal SAH, the proportion of deaths from medical complications equals that of deaths from direct effects, re-bleeding or vasospasm individually.

Pulmonary complications are the most common non-neurological cause of death.\(^{(5)}\)

The main principles of neuroanaesthesia apply for the endovascular repair of cerebral aneurysms. The choice of technique is controversial and differs between various centres. No comparative studies have been done, and very little data to support any specific technique, is available.\(^{(20)}\)

There is, however, a trend towards greater use of general anaesthesia with endotracheal intubation and neuromuscular blockade.\(^{(20)}\) The following are factors:

- Procedures can be lengthy, though most are completed within 2 hours.\(^{(20)}\)
- Optimal imaging conditions exist, as the patient is motionless.
- Reduced incidence of aneurysm rupture due to immobility
- Induced hypo- and hypertension are easier to facilitate.
- Better airway control in the supine position, with improved patient safety
- Improved control of elevated intracranial pressure (ICP)
- Better management of intra-operative neurological emergencies, with easier facilitation of rescue operations, including ventriculostomy and craniotomy

Disadvantages include inability to perform neurological assessments intra-operatively, as well as the consequences of intubation and extubation, such as hypertension, coughing or straining, which can all lead to a rise in ICP.

Some endovascular aneurysm repairs are performed under local anaesthesia or conscious sedation, primarily to facilitate assessment of neurological function during the procedure. One needs to carefully assess whether the patient will be able to remain supine for protracted periods and maintain a patent airway.

The primary goals during provision of conscious sedation are:

- Analgesia. Ensure administration of adequate local anaesthesia where indicated.
- Anxiolysis
- Relief of discomfort from having to lie still for prolonged periods. Ensure that the patient is comfortable before starting the sedation, with specific attention to padding of pressure points.
- Immobility.

Avoidance of the haemodynamic changes associated with intubation and extubation are perceived advantages.

Disadvantages include the following:

- The airway is unprotected, risking aspiration.
- High incidence of upper airway obstruction, complicating airway control, with the possibility of hypoxaemia and hypercapnoea.
- Sudden patient movements.
- Delayed management of neurological emergencies.
- Problematic behavioural disinhibition with potential loss of cooperation from patients, especially in patients with pre-existing neurological impairment.

Due to the above mentioned disadvantages, sedation is generally considered a less optimal technique.
3. Pre-operative Evaluation:

i) General:

- Pregnancy needs to be excluded in all females of childbearing age, due to the significant radiation exposure. Additional radiation protection is needed in pregnant patients.
- Recreational drug abuse, specifically cocaine, needs to be enquired about.
- Arthritic conditions may influence the patient’s ability to lay supine for prolonged periods.
- Note the patient’s previous experience with angiography.

ii) Central Nervous System:

- The focus is on careful neurological evaluation to identify signs of CNS injury.
- Various SAH grading scales exist. These are prognostic classifications that allow a more accurate risk and outcome prediction and are of use to anaesthetists in predicting the need for post-operative ventilation.
- WFNS (World Federation of Neurological Surgeons) grading is the most widely used and accepted. (16)

Table 2 World Federation of Neurological Surgeons Grading Scale for aneurysmal SAH. *Excludes cranial neuropathies, but includes dysphasia

<table>
<thead>
<tr>
<th>Grade</th>
<th>GCS Score</th>
<th>Motor Deficit*</th>
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<tbody>
<tr>
<td>I</td>
<td>15</td>
<td>Absent</td>
</tr>
<tr>
<td>II</td>
<td>13 or 14</td>
<td>Absent</td>
</tr>
<tr>
<td>III</td>
<td>13 or 14</td>
<td>Present</td>
</tr>
<tr>
<td>IV</td>
<td>7-12</td>
<td>Present or Absent</td>
</tr>
<tr>
<td>V</td>
<td>3-6</td>
<td>Present or Absent</td>
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- Hunt and Hess grading scale:

<table>
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<th>Grade</th>
<th>Clinical description</th>
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<tbody>
<tr>
<td>I</td>
<td>Asymptomatic or minimal headache and slight nuchal rigidity</td>
</tr>
<tr>
<td>II</td>
<td>Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy</td>
</tr>
<tr>
<td>III</td>
<td>Drowsiness, confusion, or mild focal deficit</td>
</tr>
<tr>
<td>IV</td>
<td>Stupor, moderate to severe hemiparesis, and possibly early decerebrate rigidity and vegetative disturbances</td>
</tr>
<tr>
<td>V</td>
<td>Deep coma, decerebrate rigidity, and moribund appearance.</td>
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• Ascertain the nature, location and size of the aneurysm and previous treatment.
• Fisher four-point grading scale describes the amount of blood on unenhanced CT and is the best predictor of cerebral vasospasm and overall patient outcome.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Findings on CCT</th>
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<tbody>
<tr>
<td>1</td>
<td>No subarachnoid blood detected</td>
</tr>
<tr>
<td>2</td>
<td>Diffuse or vertical layers &lt; 1mm</td>
</tr>
<tr>
<td>3</td>
<td>Localized clot and / or vertical layer &gt; 1mm</td>
</tr>
<tr>
<td>4</td>
<td>Intracerebral or intraventricular clot with diffuse or no subarachnoid haemorrhage</td>
</tr>
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• Glasgow Coma Score
• Pupil size and reactivity
• Cranial nerve, visual field, motor and sensory deficits
• Monitor for increased ICP, cerebral ischaemia and hydrocephalus and be on the lookout for subtle neurological deficits. (20)
• Pre-existing neurological deficits should alert the anaesthetist to the increased risk of excacerbating cerebral ischaemia should the mean arterial pressures fall.

iii) Respiratory System:

Patients frequently have a reduced level of consciousness with impaired airway reflexes and have been bedridden for prolonged periods. Therefore they are prone to aspiration, pneumonia and atelectasis.

Smoking has a strong causal relationship with aneurysmal SAH, both in the formation and rupture of the aneurysm. Smokers have five times the risk of SAH compared to non-smokers. (16)

Patients thus frequently have smoking associated comorbidities such as COAD, respiratory infections, hyperreactive airways, impaired ciliary motility, increased carboxyhaemoglobin levels and increased oxygen requirements.

A CXR should be obtained in all patients.

Pre-operative blood gas analysis or pulmonary function tests (if feasible), are indicated for patients who desaturate significantly on room air or fail to respond adequately to supplemental oxygen. (16)

The strongest predictor of death or severe disability at 3 months is an alveolar-arterial gradient >125mmHg within 24 hours of admission. (16)

Pulmonary oedema of cardiac and non-cardiac mechanisms is prevalent. Its incidence in survivors is 23% vs more than 90% in patients who died. (16)

Hypothalamic ischaemic stress causes sympathetic stimulation, resulting in hydrostatic pressure injury to pulmonary capillaries, thereby causing pulmonary oedema.
Increased capillary permeability can lead to pulmonary oedema on its own, without accompanying increase in pulmonary capillary hydrostatic pressure.

It is unknown whether the capillary leak is produced by pressure induced mechanical injury due to elevated capillary hydrostatic pressure, or by direct nervous system control over pulmonary capillary permeability. (16)

Neurogenic pulmonary oedema is also seen and can develop within seconds or up to 2 weeks following SAH. This period of onset correlates with the duration of catecholamine hypersecretion, which lasts 10 days or more. It also has a significant association with increasing age (>30 years) and poorer WFNS grades. (16)

iv) Airway Assessment:

Routine airway assessment should be performed to anticipate potential difficulties with intubation, and the potential for airway compromise with conscious sedation.

v) Cardiovascular system:

SAH is associated with hyperactivity of the sympathetic system with massive release of catecholamines, especially noradrenaline.

Baseline blood pressure, preferably arterial pressure, should be determined and cardiovascular reserve assessed.

Significant ECG changes are prevalent after SAH, with abnormalities in both rhythm and morphology of ECG in up to 100% of patients with SAH. (16)

Arrhythmias can be benign, such as sinus bradycardia or tachycardia and atrioventricular dissociation, to potentially life threatening rhythms such as ventricular tachycardia and fibrillation.

Morphological changes include T-wave inversion, ST depression, Q-waves, U-waves and a prolonged QT- interval that is associated with dangerous ventricular dysrhythmias in one third of patients.

ECG changes usually appear within the first 48 hours and may return to normal from 10 days to 6 weeks. T-wave changes can persist for months. (16)

The importance of ECG changes in patients with known coronary artery disease is not fully understood. Patients on oral anticoagulants for cardiac indications should be converted to heparin if necessary.

ECG changes should not influence the decision to operate, as cardiac changes have not been shown to contribute significantly to perioperative mortality or morbidity. They may, however, influence decisions regarding the choice of invasive monitoring. (16)

Left ventricular systolic dysfunction is demonstrated on echo in 10% of patients with SAH. (16)

In a very small series of 5 patients with SAH, all 5 had left ventricular wall motion abnormalities with a reduced ejection fraction on 2-D echocardiography at 1-4 days. At follow-up 14-42 days later, left ventricular ejection fraction was normal in all 3 patients that had survived. The dysfunction was thus assumed to be transient.

Elevated serum levels of cardiac enzymes, including troponin I, are a highly sensitive and specific indicator of myocardial dysfunction in aneurysmal SAH.

However, ECG changes, LV dysfunction and elevated cardiac enzymes are all present in these patients without angiographic evidence of CAD or vasospasm. (16).
The enzyme changes are most likely caused by focal myocytolysis (16), secondary to excessive catecholamine release from intramyocardial nerve endings.

Enzyme changes are not influenced by surgery or endovascular management.

It is advisable to optimize patients presenting with hypertension, ECG changes and dysrhythmias prior to the procedure.

vi) Electrolytes:

Hyponatraemia occurs in 10-34% of patients with SAH. (16) It is either due to SIADH (syndrome of inappropriate secretion of antidiuretic hormone) or cerebral salt wasting syndrome.

Cerebral salt wasting syndrome is the result of intense neurohumeral activation secondary to SAH. Neurohumeral activation results in increased levels of both ANP (atrial natriuretic peptide) and BNP (brain natriuretic peptide). (16)

Patients are hyponatraemic with a high urinary sodium and reduced extracellular volume. They require fluid loading with normal saline. Rarely, hypertonic saline is needed, but it needs to be administered cautiously due to the risk of central pontine myelination, cerebral oedema and seizures if the serum sodium is corrected too rapidly. (16)

SIADH is characterized by hyponatraemia with a low plasma osmolality and an inappropriately high urine osmolality.

Patients should be maintained normovolaemic with isotonic saline, and not fluid restricted as is usually done, as fluid restriction can worsen vasospasm in SAH patients.

Diabetes insipidus is rare, and causes hyponatraemia due to inadequate secretion of antidiuretic hormone.

DDAVP (desmopressin acetate) is used for treatment of diabetes insipidus by inhibiting free water clearance. Tight glucose control is essential, as hyper- and hypoglycaemia are associated with poor neurological outcome, especially in the presence of cerebral ischaemia.

The incidence of hypomagnesaemia in patients with SAH is more than 50%. It is associated with the occurrence of DCI (delayed cerebral ischaemia) and poor outcome after 3 months. (16)

The MASH trial (16) evaluated the use of magnesium and acetylsalicylic acid in SAH. The authors suggest that magnesium reduces DCI and associated poor outcome, and proposed that magnesium reverses vasoconstriction caused by endothelin-1, which is thought to contribute to the development of vasospasm. Magnesium also inhibits release of excitatory amino acids and blocks the N-methyl-aspartate-glutamate receptor which contributes to DCI. (16)

Patients are frequently dehydrated with electrolyte disturbances, including hypokalaemia and hypocalcaemia. These should be corrected pre-operatively.

36%- 100% of patients have been found to have an abnormally low intravascular volume. This is attributed to bedrest, supine diuresis, a negative nitrogen balance and fluid shift into the interstitial compartment as part of the stress response (16), and could result in renal dysfunction.

Baseline renal functions also need to obtained due to the large amounts of potentially nephrotoxic contrast materials that are used. (20).

Calculating glomerular filtration rates sould be considered.
After administration of contrast and flush, patients should be monitored for hyperosmolarity and hypervolaemia, followed by dehydration.

**vii) Coagulation:**

Enquire about a history of pre-existing coagulation disorders and prior administration of anticoagulants.

Baseline **coagulation studies** need to be done. 4% of SAH patients have reported to be thrombocytopaenic during the first 14 days.\(^{16}\)

**viii) Allergies:**

Iodine or shellfish allergies are particularly important to ask about, and warrant careful attention and planning.\(^{40}\) Pre-treatment with steroids and anti-histamines should be considered\(^{35}\).

Acute contrast reactions occur within 20 min of exposure to iodinated contrast and are caused by hypertonicity, direct cardiac depression, or idiosyncratic anaphylactoid reactions.

Delayed reactions present as a rash within 1 week.

The most commonly used contrast medium used for INR procedures is iohexol, which has an osmolality of 672mOsm/kg.

Fewer adverse reactions occur with non-ionic low osmolar agents. Fatal reactions occur at the same frequency as ionic agents (1:10 000), but mild and moderate reactions have a reduced incidence\(^{35}\).

A history of known heparin or protamine allergy also needs to be elicited.

Protamine can cause anaphylaxis and acute pulmonary hypertension.

**ix) Contrast –induced Nephropathies:**

This is the third most common cause of hospital-acquired renal failure, and occurs in 12% of patients.\(^{35}\)

A 25% increase in serum creatinine within 3 days is highly suggestive.

High risk patients are the elderly, and those with pre-existing renal dysfunction and volume depletion. Administration of large volumes of high osmolar contrast and co-administration of nephrotoxic medication are contributory.\(^{35}\)

A direct correlation exists between the osmolality of the contrast medium and nephrotoxicity.

Non-ionic contrast media are less likely to cause contrast-induced nephropathy in patients with pre-existing renal dysfunction.\(^{35}\)

Risk of nephropathy can be reduced by using small dilute volumes of contrast, avoiding nephrotoxic drugs, and ensuring adequate hydration. Pre-operative normovolaemia, obtained by administration of 0.9% NaCl offsets the diuretic effect of contrast.

Use of N-acetylcysteine, a free radical scavenger, in doses of 600-1200mg twice daily, has shown to cause significant reduction in the incidence of nephropathy.\(^{35}\) Two doses before, and two doses after the procedure are administered. It is acceptable for use in high-risk patients, but evidence to support its routine use is lacking.

N-acetylcysteine, together with an isotonic bicarbonate infusion, leads to alkalinization of the renal tubular fluid, minimizing tubular damage.\(^{35}\) It is potentially beneficial.
Vasodilators (dopamine and fenoldopam), theophylline, calcium channel blockers and anti-oxidants (ascorbic acid) have all been tried without conclusive results.

4. Monitoring and Equipment:

Monitoring standards for anaesthesia in the IR suite should be no different from those in the operating room. Most neuroradiology suits have full monitoring capabilities. Ensure that adequate equipment and supplies are readily available prior to starting a procedure.

Minimum monitoring includes:

- ECG.
- Blood pressure.
- Oximetry. The probe should be placed on the big toe of the leg that will be receiving the femoral introducer sheath, to provide early warning of femoral artery obstruction or distal thromboembolism.
- Capnography with inspired and expired gas analysis. In spontaneously breathing patients the sampling port can be placed into the face mask.
- Nerve stimulator.
- Temperature probe.

The threshold to monitor arterial pressures invasively should be low, especially if the aneurysm is ruptured. It is also helpful if frequent blood samplings are indicated.

It is preferable to monitor the arterial pressure before induction. In patients with unruptured aneurysms and those with a good WFNS grade, it may be possible to monitor the blood pressure with a rapidly cycling cuff until the patient is asleep.

If arterial cannulation is fails, the side port of the femoral artery introducer sheath can easily be transduced. Using a coaxial or triaxial catheter system, the arterial pressure at the carotid artery, vertebral artery and distal cerebral circulation can be measured. Coaxial catheter measurements provide reliable means, but the diastolic blood pressure is overestimated, and the systolic underestimated.

A CVP is less indicated than during clipping, as marked fluid shifts, blood loss or air embolism are not likely to occur. It can be inserted by taking advantage of fluoroscopy and may be monitored if the patient's medical or fluid/electrolyte status warrants it. This may be the case in patients that need close assessment of their fluid status and meticulous postoperative haemodynamic maintenance eg patients with pre-existing medical problems, focal neurological deficits, significant cerebral vasospasm and a low GCS.

A peripherally inserted long line is preferred at some institutions.

Temperature needs to be monitored and active warming devices have to be available to keep the body temperature near normal and to measure the core temperature.

The Intra-operative Hypothermia for Aneurysm Surgery Trial (IHAST) has shown no benefit to mild hypothermia.

No conclusive human data exists to support the use of mild hypothermia as being neuroprotective in the clinical setting of SAH.

Hyperthermia is detrimental and augments brain injury, and should therefore be avoided.

Glucose needs to be monitored and hyper- and hypoglycaemia avoided.
A urinary catheter is essential to measure and frequently assess output, in order to assist in fluid management, manage diuresis secondary to administration of mannitol and furosemide, and aid in patient comfort during the lengthy procedures.

Large volumes of heparinized flush and radiographic contrast are used. Contrast produces an osmotic load and often leads to vigorous diuresis.

All monitoring cables have to be placed out of the radiologist's view to avoid image impairment from trailing radio-opaque lines. Transducers and pumps are mounted on the INR table.

Extra long tubing and lines should be used. Enough slack should be present in all monitoring lines, IV lines and tubing, as well as airway connections, to facilitate unobstructed movement of the table and the screening equipment during imaging and coiling.

Avoid cerebral venous obstruction and pay specific attention to head and neck positioning.

5. Neurological Monitoring:

ICP monitoring via the ventriculostomy drain has proven useful in patients with SAH. (40)

Patients with a ventriculostomy drain are, however, more prone to transmural pressure changes and are at an increased risk of rebleed during periods of raised arterial pressure.

Bispectral index monitoring is useful not only to assess and manage anaesthetic depth, but also in demonstrating the condition of the neurological state and its response to the procedure. (53)

Transcranial Doppler (TCD) is widely used to assess vasospasm after SAH. It is more sensitive than neurophysiological monitoring, which relies on changes in CBF.

The primary goal of TCD use, is to identify early development of significant vasospasm, so that appropriate treatment can be initiated. It is also used to assess the haemodynamic effects of interventions during the procedure.

Advantages are, that it instantly reflects cerebral perfusion and detects emboli peri-procedurally.

Disadvantages are, that it is technically inconvenient and practically difficult, interfering with imaging. It also has a limited ability to detect distal branch vasospasm, has poor sensitivity for mild (<20mmHg) increases in ICP, makes grading of spasm severity in arteries other than MCA difficult, appearance of high resistance flow patterns due to increased cardiac output rather than increased ICP, and finally, the accuracy of data interpretation depends on the technician and interpreter's knowledge, skill and experience. (16)

The role of neurophysiological monitoring during endovascular procedures still needs to be validated. It is certainly desirable, but difficult to perform routinely.

It has been used in attempts to minimize neurological morbidity from operative manipulations.

The goal is to identify changes in brain function prior to irreversible brain damage, as endovascular aneurysm treatment can alter regional CBF in the arterial distribution of the aneurysm, and cause ischaemic complications. (16) Thromboembolic complications or partial impedance of blood flow in parent vessels may also be caused.

NPM directly assesses the functional state of specific cerebral regions and provides indirect measurement of regional ischaemia.
Limitations are its relative insensitivity in detecting ischaemic changes in certain vascular territories such as the posterior cerebral artery territory.

Anaesthetic agents cause confounding effects which mimic cerebral ischaemia.

NPM includes EEG (electroencephalogram), SSEP (Somatosensory evoked potentials) and BAEP (brainstem auditory evoked potentials).

Which technique is used depends on the aneurysm location and the vascular territories at risk.

Use of EEG and SSEP monitoring has become standard practice during aneurysm clipping. The same is not true for coiling, as perfusion to all parts of the brain should remain intact during the embolisation procedure. (20)

It should be considered in complex cases eg giant and wide-neck aneurysms, aneurysms with necks in close proximity to other vessels, significantly increased ICP, large SAH, and patients otherwise at risk for ischaemia due to concomitant pathology.

Constraints include lack of space, the need for trained personnel (technician or neurophysiologist), additional cost and time, and difficulty in obtaining good quality signals in the radiology suite.

When monitoring evoked potentials, the anaesthetic technique, including the use of TIVA, may need to be modified. (28)

Liu et al (28) analized the usefulness of intra-procedural NPM during endovascular aneurysm therapy in 35 patients. Changes were observed in 9, resulting in altered management in 5, including abandonment of coiling in 1. They concluded that NPM use may reduce ischaemic complications, and may be used to guide therapeutic decisions.

6. Intravenous Access:

Attempts at IV and arterial cannulation should be minimal and peripheral in light of systemic anticoagulation during the procedure.

At least two wide bore IV cannulae should be inserted. Infusions of drugs should be given through a dedicated infusion cannula. Connections, extensions, three-way taps and infusion devices should be diligently checked, especially when using TIVA. The injection port should be easily accessible.

It should be kept in mind that drug effects may be delayed due to extension tubings. One should try to minimize infusion dead space where possible.

7. Premedication:

The use of premedication should be individualized.

A pre-operative visit alone may alleviate anxiety in grade 1 and 2 patients.

A small dose of a benzodiazepine such as midazolam, titrated in small increments, provides anxiolysis, sedation of short duration and amnesia.

It can, however, impair assessment of the neurological status and worsen confusion in patients who already have an altered sensorium. (5)
Generally, sedatives are avoided (20) to allow accurate assessment of immediate pre-operative neurological condition and grade. This has to be weighed against the risk of an anxious patient becoming hypertensive, thereby increasing his risk of rebleeding.

**Narcotics** are also best avoided due to their potential for causing respiratory depression and hypercarbia due to $CO_2$ retention, which will increase CBF and CBV, which in turn will cause an increase in ICP.

**H2 receptor antagonists**, alone or in combination with metoclopramide, reduce the risk of gastric aspiration.

**Nimodipine**, orally or intravenously, has usually already been commenced for cerebral ischaemia secondary to cerebral vasospasm. It should be continued, as it lessens the incidence of traumatic vessel spasm in the neck and in intracranial arteries. (16)

**8. Induction:**

The aim during induction is to facilitate haemodynamic stability and avoid blood pressure surges which could lead to rupture, while maintaining adequate perfusion of a possibly ischaemic cerebral circulation. (5)

The table should be turned as closely as possible to the anaesthesia machine to facilitate effective airway management.

Transmural pressure (TMP) determines the integrity of the aneurysm. TMP is determined by the difference between pressure within the aneurysm (MAP), and pressure surrounding the aneurysm (ICP). Thus $\text{TMP} = \text{MAP} - \text{ICP}$. Thus, to reduce the risk of rupture, acute changes in TMP need to be minimized.

The incidence of rupture during induction is 2% (16), and is associated with a high mortality rate.

The risk of rupture is present until the aneurysm is successfully coiled, as there is no skull decompression during endovascular treatment.

With anaesthetic agents such as propofol, desflurane and sevoflurane, anaesthesia can rapidly be induced with minimal haemodynamic changes.

A sleep dose of propofol is usually combined with an opioid. Remifentanil 0.3-0.5mcg/kg/min, alfentanil up to 10mcg/kg, or fentanyl can be used.

Thiopentone and etomidate are alternatives, although the myoclonic movements produced by etomidate are not desirable. (5)

Gastric distension should actively be avoided by limiting ventilatory pressures prior to intubation.

Pressor responses are obtunded with IV or tracheal lignocaine, or rapid-and short-acting beta-blockers such as esmolol.

Magnesium sulphate should be considered, as it aids in haemodynamic control and offers cerebral protection.

One should ensure that the neuromuscular block is profound prior to tracheal intubation. Correct placement of electrodes for peripheral nerve stimulation should be verified prior to the administration of a neuromuscular blocking agent. Atracurium 0.5mg/kg, rocuronium or vecuronium are suitable.
It is essential to confirm tube depth, and to secure the tube well. Cutting the endotracheal tube to the correct length helps to prevent it kinking or being pushed in by the image intensifier. Some centres prefer the use of armoured tubes.

Antibiotic prophylaxis should be administered.

9. Maintenance:

The aim is to provide a cardiovascularly stable anaesthetic with manipulation of systemic and regional blood flow. CPP needs to be optimized, with avoidance of decreases in CPP, as well as further increases in ICP. Responses to painful stimuli need to be ablated. It is important that the patient has to be motionless, especially at critical times of coil positioning and deployment. A smooth emergence and a rapid recovery, to allow neurological evaluation, needs to be facilitated.

Procedures are generally less stimulating and painful, thus less anaesthetic is required to achieve tolerance of the endotracheal tube. However, the risk of awareness due to insufficient anaesthetic depth exists, especially as muscle relaxants are used for prevention of movement and coughing.

Ideal anaesthetic agents should not impair cerebral autoregulation, $CO_2$ reactivity or cerebral metabolism.

Cerebral autoregulation includes flow-metabolism coupling, pressure-flow coupling and $CO_2$ reactivity of regional blood flow. The balance between physiological autoregulation and the direct vasodilatory effect of the agent determine what effect the volatile will exert on CBF.

Cerebral autoregulation remains intact at low inspiratory concentrations, and thus the vasodilatory effect is negligible. Inconsistent changes in CBF may be produced by the combined effect of the impaired autoregulation and the direct cerebral vasodilatation produced by the volatile, at higher concentrations. Metabolism of the volatile, perfusion pressure and arterial $CO_2$ tension also play a role in the uncoupling. \(^{(14)}\)

Increased CBF, CBV, ICP and uncoupling of CBF and metabolic demand $CMRO_2$ can potentially be caused by all volatiles for as long as 1 hour after use. \(^{(5)}\) They can also lead to persistent post-anaesthetic hyperaemia, which increases the risk of intracranial haemorrhage, especially if the systolic blood pressure is >160mmHg on 2 or more consecutive measurements.

Sevoflurane is considered the choice volatile. CBF and $CMRO_2$ coupling are maintained up to 1 MAC. Cerebral blood vessel reactivity (CBF) to changes in arterial $CO_2$ tension, are also preserved. It has a low potential for increasing CBF-ICP.

Sevoflurane’s rapid offset and faster recovery, facilitating earlier postoperative neurological assessment, make it preferable to isoflurane.

In a study of 103 patients, comparing speed of recovery after sevoflurane and propofol for maintenance of anaesthesia in neuroradiology procedures, Castagnini et al \(^{(28)}\) showed that sevoflurane is associated with a more rapid recovery. There was no demonstrated difference in the time to discharge between the two groups.

A limitation of the study is, that intraoperative depth of anaesthesia was not controlled, and may therefore not have been the same.

Isoflurane and desflurane have also been used safely in concentrations of less than 1 MAC. Higher concentrations cause increased CBF with loss of autoregulation. \(^{(28)}\)
The beneficial effects of propofol include the reduction of CMR and CBF in a parallel and dose related manner, while leaving autoregulation intact. It either decreases or does not alter ICP and may help to prevent delayed neuronal death after transient cerebral ischaemia. It is viewed as the hypnotic of choice.

The combination of propofol and a short-acting opioid, such as remifentanil, in a TCI or TIVA technique, reduces CBF, ICP and $CMRO_2$. It avoids pollution caused by vapours and facilitates a fast recovery.

Another technique that is used, is the combination of low-dose propofol and remifentanil infusions, supplemented with sevoflurane in air. This technique minimizes individual drug side-effects and allows “fine-tuning” of the depth of anaesthesia, by varying the vapour concentration.

Nitrous oxide has adverse effects on intracranial dynamics and should be avoided. It elevates CBF and ICP and may expand micro-air emboli during injection of contrast or irrigation fluid.

Dexmedetomidine is a selective alpha-2-adrenoceptor agonist with centrally mediated sympatholytic effects. It significantly reduces intra-and post-operative anaesthetic requirements.

Patients who are sedated with dexmedetomidine remain rousable and able to cooperate when stimulated. Another distinct advantage, is that it lacks respiratory depressive effects. It has successfully been used in awake craniotomy procedures requiring neuropsychological tests. Its use in the neuroradiology suite, and specifically its ideal dose, require further investigation.

As mentioned previously, endovascular procedures are generally not very painful. The most stimulating part of the procedure is usually cannulation of the femoral artery. Patients experience a burning sensation on injection of contrast into the cerebral arteries. During microcatheter manipulation, distension and traction of cerebral arteries may produce a headache. Pain is also caused by stretching of the arteries against the pial covering in the skull base when attempting distal access.

Opioids are devoid of intrinsic cerebrovascular effects. The increase in ICP that is produced by administration of opioids is presumably independent of cerebral autoregulation, implicating mechanisms other than direct vasodilatation.

All fentanyl analogues produce a similar increase in ICP (7mmHg) and decrease in MAP (24mmHg). This effect is short-lasting and measurements return to baseline after 15min.

As the overall analgesic requirements are low, a relative opioid overdose can lead to difficulty in maintaining cardiovascular stability.

Remifentanil has favourable haemodynamic properties, and its administration as a continuous infusion has the advantage of combining a rapid onset of action with a short elimination half-life, allowing a more rapid recovery than alfentanil or fentanyl.

Rebound hypertension may, however, develop on sudden discontinuation of the infusion. A slow decrease in rate is thus advised before emergence.

Post-operative analgesia should be administered prior to extubation to blunt any post-operative stress and excessive blood pressure increases. Perfadgan is frequently used.

Blood pressure control has to be individually tailored. Tight control is vital, and, as during induction, wide swings should be avoided as they increase the risk of rebleeding by increasing the transmural pressure gradient. Controlled hypertension is desired in many cases, especially if the patient is at risk for vasospasm. Sustained elevation of pressure, with systolic measurements > 160mmHg may lead to aneurysm rupture or cerebral oedema and should be controlled.
BP control in SAH patients is problematic. It is crucial to avoid hypotension. In areas with loss of autoregulation, ischaemia can occur with aggressive treatment of BP surges. Therefore, it is best to reserve anti-hypertensives for extreme BP elevations, as well as clinical or laboratory evidence of rapidly progressive end-organ damage. One should generally target the same BP as prior to the bleed.

The following **anti-hypertensives** have been used:

- **Labetalol.** It has minimal effects on the cerebral circulation and ICP.
- **Esmolol.** This ultra short-acting beta blocker, titrated to desired effect, does not increase ICP and is used in the management of tachycardia and/or hypertension. Unless contra-indicated, beta-blockers are the agents of choice.
- **Calcium channel blockers.** They have a fast onset and reduce existing vasospasm, but can lead to increased ICP. (16)
- **Vasodilators** such as hydralazine, glyceryl trinitrate and sodium nitroprusside can cause increased ICP and CBV and may worsen cerebral perfusion and ischaemia.

**Adenosine** can be used to induce a partial flow arrest to enable deposition of embolic material. (14) A reversible atrioventricular block, with a functional cardiac arrest of about 15s, is produced by giving an IV bolus. The optimal dose is found by administering preceding boluses in a dose-escalation fashion, though clinical experience in the INR suite is lacking. Alternatively, boluses of 1mg/kg can be given. External pads for defibrillation or pacing should always be placed. With increasing skill levels of neuroradiologists, extreme bradycardias and short arrest periods should no longer be necessary.

**Ventilation** during endovascular aneurysm repair should aim for normocapnoea to mild hypocapnoea (PaCO₂ 4-4.5kPa) to help control ICP. (5) As brain bulk reduction, to facilitate surgical exposure, is not necessary, end-tidal CO₂ may be allowed to run slightly higher than in patients undergoing clipping. The reduced CBF due to the vasoconstriction associated with mild hypocapnoea, reduces the radiological transit time and allows contrast to fill the arterial lumen, improving the quality of the vascular image. (16)

Low normocapnoea is appropriate in the attempt to divert flow from normal brain tissue, towards a lesion that is about to receive an occlusive device or material. (16)

The **laryngeal mask airway** (LMA) has been used as an alternative to endotracheal intubation for airway management. It causes less haemodynamic stress and facilitates smooth emergence. Muscle relaxation and controlled ventilation can be achieved with the LMA. Appropriate patient selection is crucial. (20)

Attempts to decrease a raised ICP include:

- Administration of mannitol and furosemide.
- Moderate hyperventilation.
- Head elevation to improve venous drainage if systolic BP permits. This can be challenging on the flat radiology table.
- CSF drainage is used in refractory cases.

No single anaesthetic technique has proven superior, and many combinations have been used. (20) With raised ICP, high-dose volatile is a poor technique, given the increase in CBF that is produced. A propofol based technique is preferred, even if it delays emergence. (20)

**Temperature** regulation is important. Hyperthermia is not encountered frequently, but is associated with a poor outcome due to the risk of increasing the cerebral metabolic demand, with potential worsening of the injury. Hypothermia occurs easily due to the cold environment. As mild hypothermia has not shown to improve neurological outcome, hypothermia should be averted with the use of warming devices.
Peri-operative **blood transfusion** has been associated with angiographically confirmed vasospasm after SAH and subsequent worse outcome \(^{(16)}\), and is thus avoided in some centres, unless strongly indicated.

During **emergence**, coughing, straining, hypercarbia and wide blood pressure fluctuations need to be avoided. The use of intravenous lignocaine at the end of the procedure should be considered.

**10. Anticoagulation:**

**Heparin** is used routinely to prevent thromboembolic complications during and after the procedure. Thrombus formation occurs on intravascular catheters and on endothelial damage in small intracranial vessels.

It is administered at different times by different centres, usually between femoral cannulation and insertion of the first coil. In some centres its administration is delayed until the first coil has been deployed in acutely ruptured aneurysms, even though rupture during the endovascular procedure is rare.

An IV bolus of 3000-5000 IU (70-100 IU/kg) is administered after femoral artery cannulation and the initial angiogram. The bolus is followed either by intermittent boluses or an infusion of 1000 IU/hour.

The activated clotting time (ACT) is the preferred method of monitoring the effect of heparin. It is measured hourly, with the aim of keeping it at 2-3 times the baseline value. Additional doses of heparin are given if indicated.

The heparin infusion may be continued after the procedure to protect against the thrombogenic effects of endothelial trauma and the thrombogenic nature of the instilled materials, which can cause retrograde thrombosis in embolized vessels. \(^{(20)}\)

**Protamine sulphate** is used for reversal. It is given in a dose of 1mg/100 IU heparin, or is dosed according to a heparin dose-response curve.

**Argatroban**, a direct thrombin inhibitor, has been approved as an anticoagulant in patients undergoing percutaneous coronary interventions with, or at risk of heparin-induced thrombocytopaenia. Its potential advantages include a more predictable anticoagulant response and its minimal effects on platelets. Its potential use in neuroradiology is being investigated.

Local intra-arterial fibrinolysis using **recombinant plasminogen activator** has been described by Haehnel et al \(^{(28)}\) and achieved a recanalization rate of 44%.

**11. Antiplatelet Agents:**

Another strategy to reduce the risk of intraprocedural thrombosis is pre-treatment with antiplatelet agents in the treatment of unruptured aneurysms. \(^{(28)}\)

Agents used include aspirin, clopidogrel, ticlopidine and abciximab. Promising results have been demonstrated by Fiorella et al \(^{(28)}\) with intravenous and intra-arterial **abciximab**, a glycoprotein iib/iiia receptor antagonist.

A sustained reduction in morbidity and mortality by these agents in coronary thrombosis patients undergoing angioplasty/ stenting or thrombolysis, has led to great interest in their potential use for neuroradiology procedures. \(^{(20)}\)
They are currently used in patients with thromboembolic vascular occlusion leading to reduced blood supply.

Problems associated with their use are mainly related to their effects on platelet aggregation. It is difficult to monitor these effects clinically, as bedside tests are not readily available. It might also be problematic if the aneurysm ruptures after administration of these agents.

Their effects can rapidly be reversed with a platelet transfusion.

If is unknown if there is an increased risk of haemorrhage if they are used together with heparin. (16)

12. Potential Complications:

i) Aneurysm Rupture or Perforation (Haemorrhagic):

The incidence of intra-procedural rupture is 2.3-3% and may be higher in patients with already ruptured aneurysms. (28) The risk of perforation of a previously unruptured aneurysm is <0.5%. (20) The incidence of intra-procedural rupture should decrease with increasing operator experience.

The mortality after intra-procedural rupture is as high as 20%.

Rupture can occur:

- Spontaneously.
- Secondary to microcatheter or guide wire manipulation, coil delivery, or injection of contrast.
- Due to blood pressure surges during laryngoscopy or emergence, or due to inadequate depth of anaesthesia or the use of vasoactive drugs.

Bleeding can also occur due to perforation of proximal vessels or intracranial vessel injury or dissection.

There can either be a slight leak or a massive SAH.

Clinically patients under general anaesthesia present with cardiovascular instability. A Cushing response, characterized by hypertension with

an abrupt rise in MAP, with or without bradycardia, occurs secondary to the rise in ICP.

Awake or lightly sedated patients present symptomatically with signs of raised ICP, including headache, nausea, vomiting, altered mental state or even unconsciousness.

Extravasation of contrast on imaging can also be seen.

Rupture should immediately be communicated between the neuroradiologist, radiographer and anaesthetist to mitigate the extent of patient sequelae.

The primary responsibility of the anaesthetist is for the airway and gas exchange.

Management depends on the severity of the bleed. Very small leaks are often surprisingly well tolerated.

If the procedure is being done under sedation, general anaesthesia should immediately be induced and the airway secured without any further rises in blood pressure. As this may prove difficult, sedation is not recommended for endovascular repair of aneurysms.
The following is recommended:

- Initiate immediate cerebral protection strategies by maintaining CPP, controlling or lowering ICP with hyperventilation (maintaining the PaCO₂ between 4.5-5 kPa)
- Head elevation.
- Give steroids and osmotically active agents, such as mannitol 0.25-0.5g/kg, or hypertonic saline, to reduce cerebral oedema.
- Treat convulsions.
- Immediately reverse anti-coagulation with protamine 1mg/100 IU heparin.
- Decrease bleeding by lowering the blood pressure to the level before the bleed.
- Control the leak/defect by rapidly delivering more coils to seal the breach.
- Once bleeding is controlled, the blood pressure may be raised to check for leaks.
- Rarely, ventriculostomy may be required to decrease and monitor ICP, as acute hydrocephalus may develop secondary to the new SAH.
- Urgent rescue craniotomy with clipping may be needed if coiling fails, is incomplete or unsuccessful.

A post-procedural CT may be required prior to transfer to OR for open craniotomy.

The anaesthetist must always be prepared to transport the patient to alternate locations, and to tailor the anaesthetic technique with regard to haemodynamic and ICP management.

**ii) Cerebral Occlusion:**

Cerebral obstruction leading to ischaemia and infarction can occur due to thromboembolic events, catheters, misplaced/ displaced coils with unintentional occlusion of the parent artery, coil unravelling and fracture, and arterial dissection.

The risk of occlusive complications is 2.5-5%.

Thrombus can form on the catheter, guide wire or coil, during or after coil placement.

The embolism incidence is closely related to vascular risk factors. The appearance of new lesions correlates with difficulty in probing vessels, amount of contrast medium needed, fluoroscopy time, and use of additional catheters. (14)

Once again, communication with the interventional specialist is crucial. The goal of management is to increase collateral flow by increasing MAP by using controlled deliberate hypertension to ensure adequate perfusion to ischaemic areas.

- Normocarbia needs to be maintained.
- Initiate anti-coagulation with heparin, aspirin, or intra-arterial/ intravenous abciximab. Antiplatelet agents such as abciximab, a GP iib/iiiia inhibitor, have shown promising results. (35)
- Recanalization rates of up to 44% have been achieved by using intra-arterial tissue plasminogen activator.
- Thrombolytic therapy may even be indicated in some cases, but results have been mixed.
- Attempt to remove misplaced coils that are compromising the parent artery either by endovascular retrieval, or rarely, craniotomy
- Mechanical lysis using a guide wire or local infusion of saline may be used for angiographically visible thrombus.

Thromboembolic complications may be prevented by adequate hydration and heparinization of patients. (40)

Post-operative observation and neurological evaluation in an acute care setting are essential.
iii) Vasospasm:

With an incidence of 25%, vasospasm continues to be most common complication after SAH. It poses the greatest risk to functional brain recovery and life.

Proposed mechanisms are;

- The presence of oxyhaemoglobin and by-products in the subarachnoid space. The amount and location of blood correlate with the incidence of vasospasm.
- Biochemical changes induced by SAH lead to contraction and relaxation of cerebral arterial smooth muscle cells.
- Structural changes in blood vessels.
- Immune-mediated vasoconstriction.

Vasospasm is diagnosed by TCD, which estimates the velocity of blood flow in the middle cerebral artery. A velocity of >120cm/sec correlates with angiographically visible vasospasm.

Four-vessel angiography has traditionally been used for imaging, although CT and CTA perfusion techniques are increasingly employed.

Vasospasm causes reduced CBF to cerebral tissue, leading to ischaemia and delayed ischaemic neurodeficits (DIND). It usually manifests locally at the site of the bleed, with symptoms relating to the territory of the bleed. Severe diffuse spasm is present in patients with very severe SAH.

The effect of clipping or coiling on the incidence of vasospasm remains unclear. Clipping offers the theoretical advantage of surgical ligation and irrigating the blood out of the subarachnoid space, whereas coiling is less invasive.

The goal of management of vasospasm is to improve distal perfusion.

**Medical management** comprises HHH therapy and aims at augmenting CBF by increasing blood pressure, expanding blood volume and reducing blood viscosity. Parameters that should be aimed for vary slightly between the different studies.

- Haemodilution: Hct 30-35%.
- Hypervolaemia: CVP 8-12mmHg.
- Hypertension: MAP >20mmHg higher than pre-operatively. Increasing the MAP to maintain CPP can be accomplished by phenylephrine, noradrenaline, or other inotropes.

HHH therapy is commonly used in ICU. It carries the risks of pulmonary oedema, acute cardiovascular events such as myocardial ischaemia, and cerebral oedema. (20,35)

There is emerging evidence that increasing the cardiac output may be better than increasing MAP. (16) An increase in the CO, without an increase in MAP causes elevation of CBF in patients with vasospasm. An aimed for cardiac index of 6L/m² has been suggested.

HHH therapy cannot be recommended for prophylaxis of vasospasm (35). There is also no evidence that outcomes with HHH therapy for treatment of vasospasm are better than the natural course of the condition would have resulted in. (35)

**Nimodipine**, a calcium channel blocker, is given intravenously at the time of diagnosis of aneurysmal SAH at some institutions, to protect against cerebral ischaemia from vasospasm. (20) It is highly
lipophilic and effectively crosses the blood-brain barrier. It has been shown to significantly improve outcome in aneurysmal SAH. Intra-arterial administration is also safe and effective in the treatment of SAH. It can also be given orally in a dose of 60mg 4 hourly.

A side-effect is systemic hypotension and one needs to be ready to support the blood pressure should this occur.

Preliminary experiences with intra-arterial nicardipine have also been reported to be favourable. Mechanical opening is achieved with balloon angioplasty. Compliant balloons are used to conform the vessel wall of the constricted vasospastic blood vessels. Vasospasm responds to low dilatation pressures from balloons, decreasing the risk of perforation and subsequent haemorrhage encountered with higher dilatation pressures.

It is most effective to prevent transformation of an ischaemic infarct to a haemorrhagic infarct, when used within 2 hours of symptomatic ischaemia. It is effective in 98-100% of patients, being the most effective treatment method of vasospasm. Clinical improvement is seen in 79-80% of patients.

Recanalization remains a significant problem, with a greater risk thereof existing in larger aneurysms. Follow-up angiography is recommended for all coiled aneurysms at 6 months and 2 years. MRI will probably increasingly be used for this purpose in future.

The reported benefit of surface active and bioactive coils on reducing the incidence of recanalization has yet to be proven.
v) General:

- Pulmonary embolism.
- Temporary hair loss.
- Haemorrhage at puncture sites and groin haematomas.

13. Recovery and Post-op Care:

A rapid and smooth recovery is desirable to facilitate early neurological assessment and safe transfer to recovery areas.

Blood pressure control post-operatively is influenced by the presence or absence of vasospasm, and whether the aneurysm was secured or not.

- Unsecured or incompletely secured aneurysms may call for induced hypotension. The safe upper limit for unsecured aneurysms is unclear.
- If they were secured, allow the blood pressure to return to normal or up to a systolic pressure of 160mmHg. (5)
- MAP 20-30% above normal may be required to maintain the cerebral perfusion pressure in patients with vasospasm or occlusive conditions. This can be achieved with phenylephrine or noradrenaline. A target MAP needs to be agreed upon by the radiologist, neurosurgeon and anaesthetist.

Nimodipine intravenously or nasogastrically is used in aneurysmal SAH until the patient can tolerate oral medication, and is continued for 3 weeks.

Heparinization is continued post-operatively if embolic complications occurred, or if a large surface area of the coil is exposed in the parent vessel.

Most patients receive aspirin 75mg daily for 3 months.

Paracetamol, codeine or morphine are used parenterally for analgesia.

Shivering requires treatment.

5-HT3 receptor antagonists are given to avoid nausea and vomiting caused by contrast and anaesthetic agents.

Hydration needs to be maintained as there is a large osmotic diuresis secondary to the hyperosmolar contrast.

Patients who were lucid prior to the procedure are expected to wake up and undergo neurological evaluation, and should be admitted to a high-care unit, where they will undergo continued neurological evaluation to identify and manage new neurological deficits, as well as medical problems that develop during the post-procedure period.

Patients should remain supine until the femoral sheath is removed.

Patients who have had complications, or presented in poor condition with a poor grade aneurysm, need to be transferred to a neurointensive care unit for sedation and ventilation. They may also require a CT prior to admission to intensive care.
14. Conclusion:

Endovascular management of cerebral aneurysms is increasing in frequency, and the procedures being performed are becoming increasingly complex. The anaesthetist is challenged by additional anaesthetic aspects previously not encountered in neuroanaesthesia.

It is important to remember, that although an invasive operative procedure is not performed, the patient’s disease physiology, and the anaesthetic impact on it, are not altered by the change in venue from the operating room to the neurointerventional suite. The same anaesthetic goals and close attention to patient management should not be overlooked in the interventional arena.

Safe anaesthetic management is based on a broad understanding of pathophysiological and technical issues that arise in the endovascular treatment of cerebral aneurysms. To be able to anticipate and manage complications, the anaesthetist needs to understand the anaesthetic implications of current and future developments in endovascular neuroradiology, such as new embolic materials and techniques like intravascular sonography.

Provision of safe care by the anaesthetist has a great influence on the best possible patient outcome.

Anaesthetists meeting the challenges posed by interventional neuroradiology will be critical to the success of these procedures.
15. Bibliography:


