

Current Concepts in Anaesthesia for Aneurysmal Subarachnoid Haemorrhage

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INTRODUCTION

Subarachnoid haemorrhage (SAH) is a neurological emergency, which occurs as a result of extravasation of blood into the subarachnoid space(1, 2). The worldwide incidence of SAH is reported to be 6 – 7 per 100 000, accounting for 5% of all strokes, with the most common aetiology being the rupture of an intracranial aneurysm(2, 3). Aneurysmal Subarachnoid haemorrhage (aSAH) accounts for 80-85% of nontraumatic SAH episodes, while arteriovenous malformations account for a further 5%, with the rest being due to a diverse cause of intracerebral haemorrhages(1, 4, 5).

The mortality rate in patients with aSAH is very high approaching 50%(5, 6). A quarter of patients with acute SAH die before reaching the hospital, while those surviving the initial event suffer significant morbidity due to neurocognitive impairment resulting in dependence for care and an impaired quality of life(6). Therefore, the management of patients with SAH requires a multi-disciplinary approach aimed at securing the ruptured aneurysm as early as feasible, prevention of secondary insult, as well as treatment and prevention of the associated systemic complications(6).

The role of the anaesthetist in these patients may be at presentation in the emergency department for initial resuscitation, during the peri-operative period, as well as in the critical care setting(1). This requires an understanding of the therapeutic goals, the potential risks associated with surgical intervention – be it neurosurgical clipping or endovascular coiling of the aneurysm – as well as the challenges surrounding the chosen intervention, particularly the remote location in the case of endovascular techniques(7).

Pathophysiology

Intracranial aneurysms are acquired lesions that occur due to haemodynamic stress on the arterial wall at bifurcation points, resulting in thinning of the vasculature wall(8). The majority of aneurysms occur at the terminal portion of the internal carotid artery at the Circle of Willis close to bifurcations, accounting for 80-90%, while the remaining 10-20% are located in the vertebro-basilar circulation(9). The most common site of rupture of these aneurysms are the posterior communicating artery and the anterior cerebral artery(6).

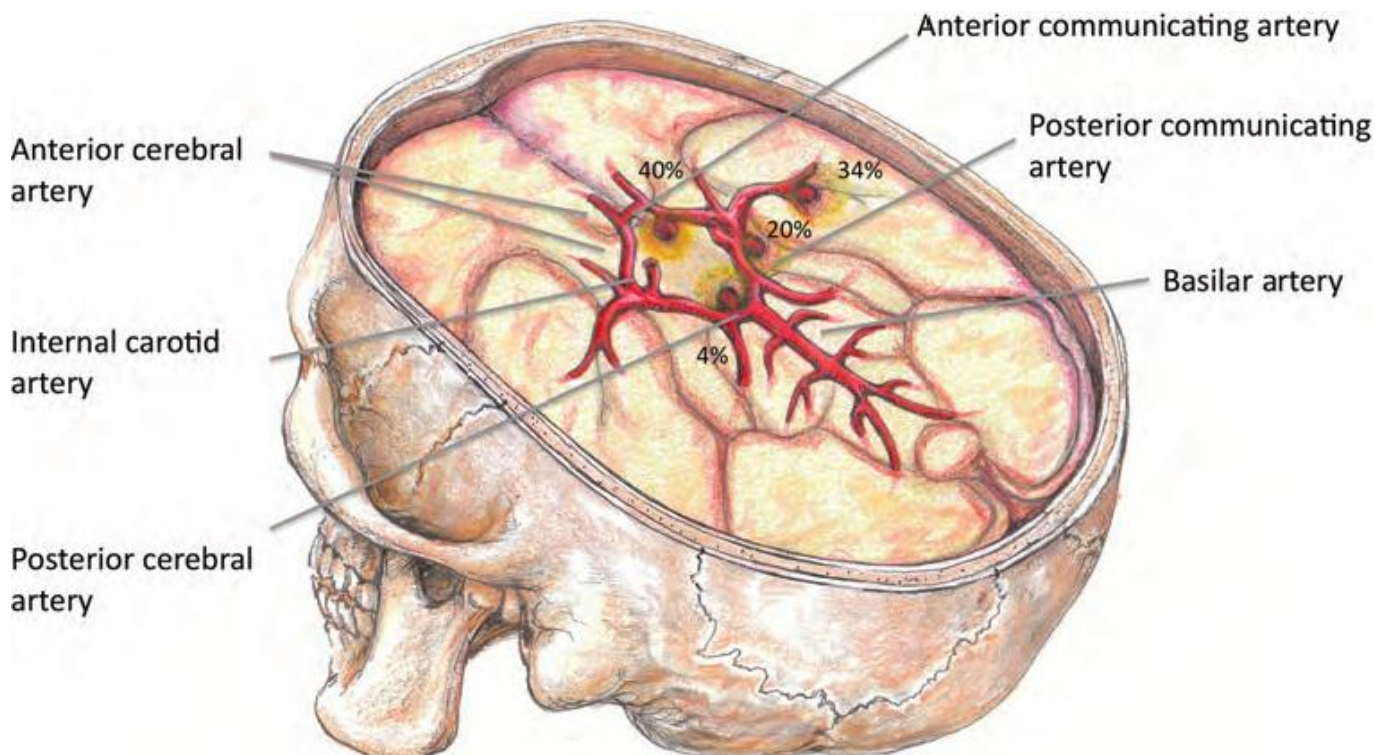


Fig 1. The intracranial circulation with the common site of aneurysms illustrated(6). *From Luoma A, Reddy U. Acute management of aneurysmal subarachnoid haemorrhage. Continuing Education in Anaesthesia, Critical Care & Pain. 2012;13(2):52-8.*

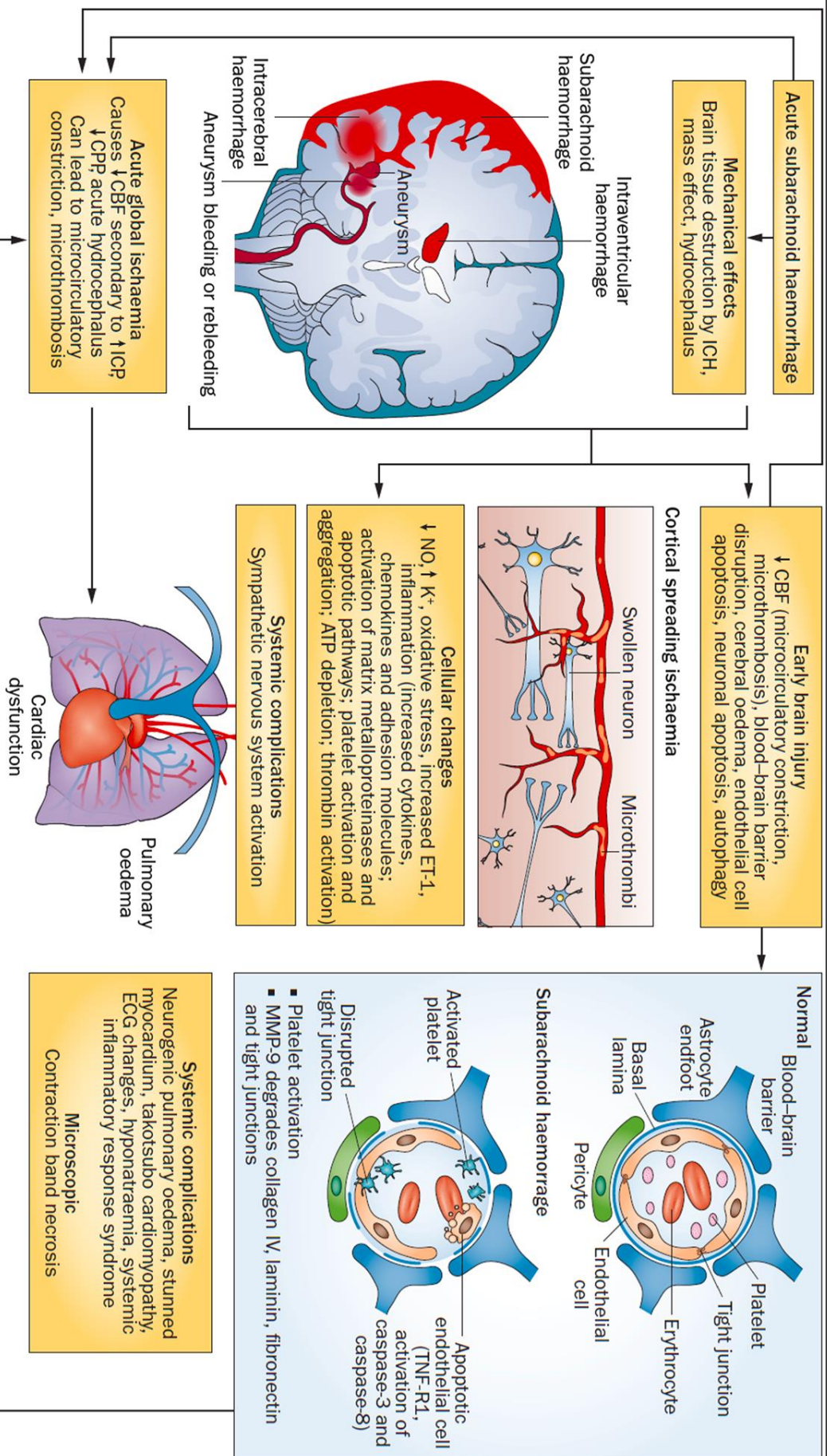
Rupture of the intracranial aneurysm occurs due to haemodynamic shear stress on the weakened arterial wall, for instance in a case of a sudden increase in the cerebrovascular arterial pressure(6). This results in free communication between the intravascular and subarachnoid space with consequent extravasation of blood into the subarachnoid space, which continues until the equalisation of the pressure gradient between the intravascular and subarachnoid space(5, 9). This process is responsible for the sudden onset severe headache and the sudden transient (or permanent) loss of consciousness(LOC)(9).

The extravasation of blood into the subarachnoid space results in the headache, meningism and hydrocephalus; while also causing a mass effect leading to raised intracranial pressure(ICP)(9). There is also loss of cerebral blood flow (CBF) autoregulation and loss of cerebrovascular reactivity to carbon dioxide CO₂, particularly in patients with poor grade SAH(9). Blood clots may

form in the subarachnoid space as well as in the ventricles, causing hydrocephalus secondary to decreased absorption and/or obstruction of flow of cerebrospinal fluid (CSF) (9).

The brain injury associated with aSAH is thought to occur in two distinct phases: early brain injury and delayed brain injury. The early brain injury (Figure 2) occurs immediately after aneurysm rupture and is due to direct mechanical damage of brain parenchyma by subarachnoid blood and the initial transient global cerebral ischaemia resulting from the acute rise in ICP with subsequent reduced CBF(10). Delayed brain injury results from delayed cerebral ischaemia (DCI) which may develop 3-14 days after the initial insult and may present with deterioration in neurological function(8).

Fig 2. Early pathophysiology of subarachnoid haemorrhage. Acute haemorrhage from an aneurysm can physically damage the brain and lead to acute transient global ischaemia. Transient global ischaemia secondary to increased intracranial pressure can also trigger sympathetic nervous system activation, leading to systemic complications. The contribution of each process to the pathophysiology is unknown, but transient global ischaemia and subarachnoid blood result in early brain injury, characterised by microcirculation constriction, microthrombosis, disruption of the blood–brain barrier, cytotoxic and vasogenic cerebral oedema, and neuronal and endothelial cell death. CBF cerebral blood flow, CPP cerebral perfusion pressure, ECG electrocardiographic, ET-1 endothelin-1, ICH intracranial haemorrhage, ICP intracranial pressure, MMP-9 matrix metalloproteinase-9, NO nitric oxide, TNF-R1 tumour necrosis factor receptor 1. *From de Oliveira Manoel et al. Critical Care (2016) 20:21(10).*



Risk Factors

Risk factors for the development and rupture of intracranial aneurysms are multifactorial, with a combination of both modifiable and non-modifiable risk factors(8). Peak incidence is seen between the 5th and 6th decade of life, with a female to male ratio of 3:2(6). Smoking, hypertension and alcohol abuse have been reported to double the risk of SAH(8). Several inherited conditions (Table 1) have also been associated with an increased risk of formation of intracranial aneurysms(6).

Table 1. Risk factors for the development and rupture of intracranial aneurysms(4, 6, 8)

Formation of aneurysm	Modifiable	Non-modifiable
	Hypertension Atherosclerosis Smoking Alcohol abuse Cocaine use	Age Female Family history Inherited diseases: Polycystic kidney disease, Marfan, Ehlers Danlos, Neurofibromatosis T1, Fibromuscular dysplasia
Rupture of aneurysm	Modifiable	Non-Modifiable
	Smoking Hypertension Cocaine	Female Posterior circulation aneurysm Giant aneurysm

Clinical Assessment

Clinical presentation

Patients may present with the classic “worst headache ever”, with associated nausea and vomiting, meningism, photophobia, seizures, and neurological deficits (which may range from a mild cranial nerve deficit to hemiparesis and a depressed level of consciousness)(4, 6, 8, 9).

Diagnosis

The initial investigation of choice in patients suspected to have a SAH is a non-contrast CT scan which has a sensitivity close to 100% within 6 hours of headache onset, declining with increasing time post-ictus(6). Other additional investigations which may be required include a lumbar puncture, CTA, four-vessel catheter angiography, and MRI(4, 6, 8, 9).

Assessment of Severity

Several grading scales (Tables 2 – 4) have been developed for the standardized clinical assessment of severity of SAH and to estimate prognosis, the most widely used of which are the Hunt and Hess, the World Federation of Neurological Surgeons, and the Fisher grading scale(1, 4, 9). A good working knowledge and understanding of these grading scales allows for effective communication between clinicians, as well as appropriate planning for the perioperative anaesthetic management of these patients(9). Prognosis worsens with an increase in the score(1). The SAH grade also allows the clinician to consider the underlying pathophysiological abnormalities that may be associated with that particular grade of SAH, for instance, the higher the clinical grade, the more likely are complications such as cerebral vasospasm, raised intracranial pressure, impaired cerebral autoregulation, impaired cerebrovascular reactivity to CO₂, as well as neurogenic cardiac dysfunction(9).

Table 2. World Federation of Neurological Surgeons (WFNS) grading scale for SAH(1)

Grade	GCS Score	Motor deficit
I	15	Absent
II	13-14	Absent
III	13-14	Present
IV	7-12	Present
V	3-6	Present

Table 3. Hunt and Hess grading scale for SAH(1)

Grade	Clinical description
I	Asymptomatic or minimal headache and slight nuchal rigidity
II	Moderate to severe headache, nuchal rigidity, no neurological deficit other than cranial nerve palsy
III	Drowsiness, confusion, or mild focal deficit
IV	Stupor, moderate to severe hemiparesis, and possibly early decerebrate rigidity and vegetative disturbances
V	Deep coma, decerebrate rigidity, and moribund appearance

Table 4. Fisher grading scale of cranial computerized tomography (CT)(1)

Grade	Findings of cranial CT
1	No subarachnoid blood detected
2	Diffuse vertical layers ≤ 1 mm
3	Localized clot and/or vertical layer > 1 mm
4	Intracerebral or intraventricular clot with diffuse or no SAH

These grading scales have several limitations due to inter- and intra-observer variability, furthermore, there is no confirmed prognostic advantage of using one scale over the other(4). The WFNS and Hunt and Hess are both clinical grading scales, while the Fisher scale is purely based on CT scan findings(9). The most important predictor of death and disability is the patient's LOC (and therefore GCS score) at presentation(5). The amount of blood present in the subarachnoid space, as assessed using the Fisher score, is arguably the best predictor of vasospasm, delayed cerebral ischaemia (DCI), and possibly even overall patient outcome(4, 6).

Complications of SAH

Neurological complications

Rebleeding

Rebleeding prior to aneurysm repair is one of the main causes of early death due to SAH, along with brain damage due to initial SAH(8). Death in the subsequent 2 weeks is usually attributed to rebleeding, DCI and medical complications of SAH(8). Up to a quarter of patients rebleed in the first 3 days after SAH, with the majority of these patients bleeding within 6 hours following SAH(8). Rebleeding carries a 20-60% mortality(8). Guidelines recommend that early intervention in order to improve outcome – securing the aneurysm within the first 72 hour(8). However this may not always be possible in our setting where patients spend hours to days between diagnosis and referral to a specialist centre.

While the aneurysm is not secured, strategies that can be employed to reduce rebleeding include preventing large changes in blood pressure and avoiding large fluctuations in intracranial pressure(6). Studies have shown a benefit in the use of tranexamic acid with reduction in the rate of rebleeding, however this did not translate to improved neurological outcomes(8). Also, the use of tranexamic acid increases the risk of thrombo-embolic complications(8). Consequently, routine use of tranexamic acid has not been included in guidelines until further compelling evidence for its benefit can be produced, however when definitive management of SAH is delayed, short term therapy with antifibrinolytic is considered appropriate(11).

Cerebral vasospasm and delayed cerebral ischaemia

Cerebral vasospasm

Cerebral vasospasm is one of the most devastating complications of SAH, described as the presence of macro- and microvascular spasms of intracranial vessels(12). Vasospasm typically occurs at 3 to 14 days post-ictus, and lasts for up to 2 weeks(9). It occurs in up to 70% of patients with SAH, 70% of these patients will be asymptomatic, while 30% can experience significant neurological deficit which is associated with significant morbidity(1, 6).

Many theories for the pathophysiology of vasospasm have been postulated(9). It is thought that following SAH, there is reduced expression of vasodilators with an associated increase in the expression of vasoconstrictors, such as endothelin-1, along with associated stimulation of calcium(12). Other factors contribute to the development of vasospasm: the imbalance in cerebral blood flow and increase in intracranial pressure results in disturbed cerebral autoregulation and consequently, cerebral ischaemia(8, 12).

The only proven predictor of cerebral vasospasm after SAH is presence of large volume of subarachnoid blood or large blood clot size(9). Diagnosis of cerebral vasospasm is by angiography (CTA, MRA, and/or DSA), transcranial doppler, or clinically – ischaemic consequences of vasospasm presenting with new or worsening neurological status(6). The only proven beneficial treatment of cerebral vasospasm is administration of oral nimodipine(6). Other interventions such as Triple-H therapy (hypertension, hypervolemia and haemodilution) are controversial and are no longer recommended as there has been no proven benefit in clinical trials(6, 8, 13).

Delayed cerebral ischaemia (DCI)

DCI has historically been linked to cerebral vasospasm, with the terms being used interchangeably(14). DCI is thought to occur between day 4 – 10 post-ictus, which overlaps with the time frame for development of cerebral vasospasm(14). However, it is important to understand that DCI can occur in the absence of vasospasm, and may affect more than one vascular territory, i.e. DCI can be present outside the territory of arterial narrowing in cases where cerebral vasospasm is present as well(6). Furthermore, not all patients with vasospasm develop DCI, and not all patients with DCI have vasospasm(14).

The Neurocritical Care Society recommends the following definitions for DCI and cerebral vasospasm(6):

- DCI is neurological deterioration related to ischaemia (unrelated to treatment of aneurysm) that persists for more than 1 hour and has no other cause (e.g. hydrocephalus, seizures, or metabolic).
- Vasospasm is arterial narrowing demonstrated angiographically or with Doppler ultrasonography, with corresponding neurological symptoms and signs.

Management of DCI

Pharmacological management

Nimodipine

Oral nimodipine, a dihydropyridine calcium channel blocker given at dose of 60mg every 4 hours as prophylactic therapy for vasospasm and DCI, is an established practice with strong evidence to support its use. It is a Class I recommendation with Level A evidence as per American Heart Association/American Stroke Foundation (AHA/ASA)(8, 15, 16). Nimodipine should be started at diagnosis of SAH and continued for 21 days(6). In the event of systemic hypotension, the dose can be halved and given every 2 hours(6).

Magnesium

The Intravenous magnesium sulphate in Aneurysmal Subarachnoid Haemorrhage (MASH) trial, and other subsequent meta-analyses, showed no outcome benefit in administration of magnesium.

Statins

Patients already receiving statins for hypercholesterolaemia are to be continued, but the introduction of statins in all patients with SAH is not recommended(6). Statins may however be considered in selected patients who are at high risk of DCI(17).

Non-pharmacological Management

Triple H therapy

This refers to the use of hypertension, hypervolaemia and haemodilution in order to improve CBF and oxygen delivery in the treatment of DCI and vasospasm(6). This approach is no longer recommended as standard practice due to the lack of evidence to support its use(17). Hypervolaemia and haemodilution is no longer recommended due to the associated non-neurological complications of hypervolaemia, such as pulmonary oedema, and the cerebral ischaemia associated with low haematocrit levels(15); treatment should aim at euvolaemia with careful assessment of the patient's fluid balance(17).

Induced hypertension is still recommended in the active management of DCI and vasospasm with the aim of optimizing cerebral perfusion pressure, unless blood pressure is elevated at baseline or cardiac status precludes it (ASA/AHA Class I recommendation, Level of Evidence Class B)(15).

Endovascular therapy

Balloon angioplasty with or without intra-arterial vasodilator therapy is indicated for patients with worsening neurological function despite optimal medical therapy (AHA/ASA Class IIa Recommendation, Level of Evidence Class B)(15).

Hydrocephalus

Hydrocephalus is a complication that may occur as early as the first three days, while it may present later in some patients(6). It occurs due to impairment of the cerebrospinal fluid circulatory pathway(12). In the presence of acute clinical deterioration, a high index of suspicion is necessary and an urgent CT scan can identify the cause(6). Patients with a large subarachnoid blood volume, or those with intraventricular clots are at particularly increased risk(6). In acute hydrocephalus, an urgent EVD insertion may be warranted to relieve the intracranial pressure(6).

Seizures

Seizures may occur in patients with SAH with an incidence of up to 26%, often occurring within the first 24 hours post ictus(12), and may be a sign of rebleeding in the unsecured aneurysm(6). Even though seizures need to be treated aggressively, prophylactic anti-epileptic treatment is not recommended as it has been shown to be associated with a worse cognitive outcome(6, 12). Current recommendations support treating clinically apparent seizures only(16, 18).

Non-neurological complications

Cardiac dysfunction

Patients may present with a wide range of cardiac manifestations following subarachnoid haemorrhage, occurring in 49 – 100% of patients, in particular those with poor neurological grade of SAH(4-6). Following brain injury, a catecholamine surge occurs with increased sympathetic outflow and autonomic dysfunction(4-6). This elevated sympathetic tone and increase in circulating catecholamines results in calcium overload in cardiac myocytes, and consequently myocyte necrosis(4-6). The cardiac changes seen reflect the severity of neurological injury in these patients(4-6).

Electrocardiogram (ECG) changes that may be seen include ST segment abnormalities, T wave changes, prolonged QT interval and presence of U waves(4-6). Tachyarrhythmias are a common occurrence, troponin leak and myocardial dysfunction in the absence of coronary vasospasm may also be seen(4-6). The degree of troponin leak is associated with increased severity of neurological damage, vasospasm and DCI, and increased risk of cardiovascular complications which all translates to poor neurological outcome(4-6).

Although these changes are thought to be reversible in most cases, they pose a particular challenge for the anaesthetist in that it may be difficult to differentiate between neurogenic cardiac injury and actual occurrence of an acute ischaemic coronary event(4-6). However, it is important to note that the peak troponin levels in neurogenic cardiac injury are usually lower than after an acute coronary event(4-6). The associated ventricular dysfunction is usually transient, however patients may develop Takotsubo cardiomyopathy which is associated with poor outcome(4-6). The management of cardiac abnormalities is supportive with continued management of the underlying neurological insult(4-6).

Hypertension

Blood pressure control is one of the important aspects of SAH management with an effect on outcome(16, 18). However there is a lack of consensus on blood pressure management targets as there is no robust evidence to guide clinical practice(16, 18). The causes of hypertension in patients with SAH in the pre-aneurysmal treatment is usually an interplay of the following: (i) sympathetic surge associated with SAH, (ii) in the case of chronic hypertensive patients, the

reduction or even complete cessation of regular anti-hypertensive medication, and (iii) iatrogenic blood pressure augmentation during institution of HHH therapy to treat cerebral vasospasm(16, 18). Pain is also an important and yet often neglected contributing factor to the hypertension, which may be amenable to simple analgesic therapy(16, 18).

The challenge in managing these patients is balancing the risk of rebleeding due to elevated blood pressures, against the risk of cerebral ischaemia(16, 18). Current European guidelines recommend keeping systolic blood pressures (SBP) below 180mmHg and a mean arterial pressure (MAP) above 90mmHg, while the AHA/ASA guidelines recommend SBP below 160mmHg and MAP below 110mmHg(16, 18). Due to the lack of evidence, there is great variation in clinical practice(16, 18).

Pulmonary complications

These include neurogenic or cardiogenic pulmonary oedema, aspiration pneumonitis, hospital acquired pneumonia, pulmonary embolism, and acute lung injury or adult respiratory distress syndrome(4, 6, 12). The incidence of pulmonary complications in patients with SAH haemorrhage has been reported to range from 30 – 80%(4, 6, 12). They are associated with poor grade SAH, and are associated with poor outcomes(4, 6, 12).

Neurogenic pulmonary oedema is thought to be caused by the sympathetic overactivity that occurs in patients with SAH. Cardiogenic pulmonary oedema in patients with SAH can occur in patients with pre-existing cardiac disease, or can be associated with reduced left ventricular systolic function associated with SAH-related cardiac dysfunction described above. Overlap in these two entities can occur, where neurogenic pulmonary oedema can in turn result in cardiac failure due to accompanying afterload increase. Treatment is supportive, aimed at maintaining cerebral oxygenation and avoiding secondary brain injury(4, 6, 12, 19).

Electrolyte disturbances: Hypo- and Hypernatraemia

Disturbances in sodium balance are common after SAH, along with other electrolyte abnormalities such as hypokalaemia, hypocalcaemia and hypomagnesaemia(5, 9). Therefore, appropriate monitoring and treatment is essential(5, 9).

The most common sodium disturbance in SAH is hyponatraemia which can be due to syndrome of inappropriate antidiuretic hormone secretion (SIADH), cerebral salt wasting syndrome (CSW), acute adrenocorticotrophic hormone or glucocorticoid insufficiency, and iatrogenic due to excessive intravenous fluids or diuretic therapy(12, 20). It is essential to differentiate between SIADH and CSW as the management of these two entities is different with fluid restriction for the former, and replacement for the latter(12, 20). Cautious correction is indicated to avoid the complication of central pontine myelolysis(12, 20).

Hypernatraemia may be due to diabetes insipidus (DI) resulting in fluid loss, or excessive sodium administration during management of these patients(12, 20). As in the case with hyponatraemia, careful correction must be undertaken in order to avoid complications including seizures and cerebral oedema(12, 20).

Fever

Fever in patients with SAH is an independent risk factor for poor outcome(18) and is associated with increased mortality(12). Older patients, poor SAH grade and those with intraventricular haemorrhage are prone to develop fever(12, 18). Active pharmacological and physical management of fever is recommended with a target of normothermia(16, 18).

Hyperglycaemia

Hyperglycaemia is common and has been shown to be associated with an increased risk of vasospasm and poorer outcome(12, 16). Strict avoidance of hypoglycaemia is recommended(16), and hyperglycaemia over 10mmol/l should be treated(18).

Anaemia.

Anaemia in patients with SAH will exacerbate delayed cerebral ischaemia, however red blood cell transfusion has been shown to be associated with poorer outcome and an increased risk of vasospasm(12). The optimal haemoglobin target in patients with SAH has not yet been identified(16).

Management of SAH

Clipping vs Coiling

Aneurysms are secured by one of two methods: craniotomy for neurosurgical clipping of the aneurysm, or endovascular coiling(6, 9). The International Subarachnoid Aneurysm Trial (ISAT), a landmark trial which compared the two interventions for aneurysmal SAH, published its results in favour of endovascular coiling(21). The findings of this trial showed that endovascular treatment conferred benefit over neurosurgical clipping: patients that underwent endovascular treatment were more likely to be independent at 1 year(21). However, due to the several limitations in the recruitment process, the results of this trial can only be extrapolated to patients with smaller aneurysms in the anterior circulation with a good neurological grade (WFNS I and II)(4).

Nevertheless, endovascular treatment has been increasing over the years with further developments in the application of interventional radiology technology(4, 5). Current recommendations are for early treatment of aneurysmal SAH, within 72 hours post-ictus regardless of neurological grade, in order to reduce risk of rebleeding. In aneurysms amenable to either clipping or coiling, coiling is recommended(16, 18).

Endovascular management of aneurysmal SAH

Treatment of aneurysmal SAH can be achieved either through occlusion of the parent artery or obliteration of the aneurysm sac(7). During coiling, a platinum coil is deployed into the aneurysm sac via femoral arterial access(7, 22). Placement of the coil in the sac initiates a clotting process that results in occlusion of the aneurysm(7).

Remote location anaesthesia

Anaesthesia for procedures in the neuroradiology suite requires unique considerations from the anaesthesiologist. These are summarized in the table below, adapted from a review by Dorairaj and Hancock(22).

Table 5. Special considerations for anaesthesia in interventional neuroradiology suite

Special consideration	Response
Unfamiliar environment, dim lighting, help not easily available	Ensure skilled assistance, resuscitation and difficult airway equipment should be available
Transfer between ward, theatres and radiology suite	Closely observe clinical status, lines, and equipment
Radiation	Shielding, minimize total exposure, safe distance from radiation source (inverse square law)
Radiology equipment	C-arm, injector, console, table kept unimpeded by lines and cables
Closed skull	Control ICP by manipulating ventilation, blood pressure, and intravascular volume
Contrast and flush	Monitor intravenous fluid administration, consider renal function
Anticoagulation	Monitor ACT, protamine available
Anaesthetic technique	Short acting agents, careful positioning, carefully secure airway

Peri-operative management for endovascular coiling

Regardless of the anaesthetic technique of choice, and taking to account the clinical picture of each patient, there are certain key principles and goals that one needs to aim to achieve when anaesthetizing patients for coiling of cerebral aneurysm(5). These are listed below in no particular order.

- Optimize/maintain adequate cerebral oxygenation;
- Maintain adequate cerebral perfusion;
- Avoid/prevent sudden and large changes in intracranial pressure;
- Control of the transmural pressure gradient across the aneurysm wall;
- Maintain normal metabolic parameters: glucose, temperature;
- Meticulous fluid administration;
- Adequate preparation for possible intra-operative emergent complications;
- And allow rapid emergence for early post-operative neurological assessment(5).

Preoperative assessment

As discussed earlier, patients with SAH can have considerable physiological derangements affecting multiple organ system(3). Accordingly, a thorough pre-operative assessment is essential, with particular attention to the neurological and cardiorespiratory status of the patient(5).

Neurological status

A thorough neurological history and examination is essential(7, 22). Collateral from family may be required in patients who are unable to give a history. Establishment of the patient's pre-operative GCS, neurological and radiological grade of the aneurysm, as well as precise documentation of the degree of neurological fallout is essential(22).

Cardiovascular evaluation

Particular attention needs to be paid in evaluation of the patient's cardiovascular status(5). A thorough history of pre-morbid cardiovascular diseases and functional status must be obtained(5). The catecholamine surge and sympathetic activation associated with SAH are responsible for a wide variety of cardiovascular changes seen in these patients(22). Patients may present with dysrhythmias, ECG changes suggestive of myocardial ischaemia, elevated cardiac enzymes, ventricular dysfunction with associated pulmonary oedema(9, 22). Baseline blood pressure must be established, as blood pressure manipulation and fluctuation are common during the procedure(7).

Respiratory evaluation

Smoking is one of the risk factors for SAH(22), therefore these patients are likely to have some degree of chronic obstructive pulmonary disease. Reduced level of consciousness predisposes to aspiration pneumonias, while the bedridden patient may develop atelectasis and pneumonia(22)

Metabolic considerations

Perioperative glucose control is essential, with hypo- and hyperglycaemia being associated with poorer outcome(22). Electrolyte disturbances are common, including hyponatraemia, hypomagnesaemia, hypernatraemia, hypokalaemia and hypocalcaemia(22). Sodium disturbances may be associated with an underlying problem such as SIADH, CSW or DI(5, 22). Careful evaluation of the patient's hydration status as dehydration of a common finding in these patients(22).

Premedication

Premedication should be considered on an individualized basis, guided by the patient's clinical grade, respiratory condition, co-morbid illnesses and chronic medication(9, 22). A benzodiazepine premedication may benefit an anxious patient who may become hypertensive thus predisposing them to rebleeding, however this needs to be balanced against the risk of deleterious effects of an increasing arterial partial pressure of carbon dioxide secondary to respiratory depression and hypoventilation(9, 22). In general, premedication should be avoided in poor neurological grade patients, whereas good neurological grade patients may benefit from pre-operative reassurance and a mild benzodiazepine premedication for anxiolysis(5). Nimodipine, and any other vasoactive drugs, should not be interrupted(5).

Choice of anaesthetic technique

Whichever the chosen anaesthetic technique, the goals are to maintain physiological and haemodynamic stability, and to keep the patient immobile during the procedure(7). In our unit, general anaesthesia is the technique of choice, however, sedation has been described as an option in good grade aneurysm(7, 16).

Sedation

Sedation may be considered a viable option due to the added advantage of real time assessment of neurological evolution during the procedure(4, 5, 16). Another advantage of sedation is the avoidance of the haemodynamic changes associated with intubation and emergence(7). However, the disadvantage of an unprotected airway, the possible need for emergent airway management during the procedure should complications arise, the risk of hypoxia, hypercapnia and sudden patient movement during the procedure makes this technique less appealing(5, 7). There is currently no evidence to in the form of randomised controlled trials to make recommendations for sedation over general anaesthesia(7, 16).

General anaesthesia

General anaesthesia for endovascular management of ruptured SAH is a well-established technique and has the benefit of ensuring an immobile patient during this complicated and often long procedure(5, 7, 16). Some clinicians opt for supraglottic airway management in selected patients in order to mitigate the haemodynamic changes associated with intubation(5). In general, endotracheal intubation with muscle relaxation is often preferred(7, 16).

Induction of anaesthesia

The ultimate goal at induction of anaesthesia is to maintain haemodynamic stability(5). Surges in blood pressure during intubation need to be avoided, while maintaining an adequate cerebral perfusion pressure to avoid cerebral ischaemia(7, 22). Propofol in combination with remifentanyl is a widely used combination to achieve adequate anaesthesia depth at intubation although etomidate or thiopentone in combination with any other short acting opioid can be used as alternatives(22). Use of lignocaine, or esmolol may also be used to blunt the intubation response, as is ensuring adequate neuromuscular blockade at time of intubation(22).

Maintenance of anaesthesia

Endovascular coiling procedures are generally painless, with the most stimulating point being femoral arterial access often done with local anaesthetic infiltration(3). The most important aspect is keeping the patient immobile for the duration of the procedure, which can be achieved with either total intravenous anaesthesia or inhalational techniques(3).

All volatile anaesthetic agents can cause an increase in cerebral blood flow, cerebral blood volume, and consequently an increase in intracranial pressure(22). Uncoupling of CBF and cerebral metabolic demand are also a concern(22). Sevoflurane has the least impact on CBF and ICP(22). Below 1MAC of sevoflurane, cerebrovascular reactivity to carbon dioxide, as well as coupling of CBF and cerebral metabolic demand, is maintained(22). This, together with the rapid offset allowing for postoperative neurological assessment, makes sevoflurane the volatile agent of choice in neuroanaesthesia(22).

Total intravenous anaesthesia with propofol and a short acting opioid such as remifentanyl is also associated with reduced intracranial pressure, cerebral blood flow, and cerebral metabolic demand(22). However, sevoflurane is associated with a quicker recovery after prolonged procedures when compared to propofol(22).

Intra-operative monitoring

In addition to standard monitoring which includes ECG, pulse oximetry, capnography, temperature and non-invasive blood pressure monitoring, invasive blood pressure monitoring which should be initiated preferably before induction of anaesthesia is considered mandatory during this procedure(22). Depth of neuromuscular blockade can additionally be used to ensure adequate muscle relaxation as any movement or coughing during a critical point in the procedure may be catastrophic.

Blood pressure management

There are currently no clear guidelines on the blood pressure management during aneurysm coiling, however haemodynamic stability and avoidance of abrupt changes in blood pressure are essential(7). Acute rises in blood pressure, such as may occur during laryngoscopy and intubation, should be avoided by appropriate use of agents to blunt the sympathetic response in order to mitigate the risk of rebleeding(7). On the other hand, hypotension at any point during anaesthesia may result in cerebral ischaemia due to hypoperfusion and should therefore be avoided(7). Current guidelines propose that the anaesthesiologist should maintain a delicate balance between the risk of aneurysm rupture and cerebral ischaemia by optimizing intraoperative blood pressure management(16, 23).

Anticoagulation

During endovascular coiling, the anaesthetist is frequently required to administer heparin(3). An intravenous dose of heparin, 50-70IU/kg is given to achieve an activated clotting time(ACT) of 2-3 times baseline during the procedure(7). ACT is monitored hourly and additional doses of heparin is administered as necessary(5). Protamine 1mg:100IU can be administered to reverse anticoagulation if warranted(24). These patients are also often prescribed dual antiplatelet therapy, aspirin and clopidogrel 3-7days before the procedure or alternatively receive a high loading dose during the procedure(7).Antiplatelet medication is continued post operatively based on an individualised approach, depending on whether coiling alone, or coiling plus stenting, or flow-diverting stents were used intraoperatively(24).

Contrast

Neurointerventional procedures require the use of contrast and patients can receive a significant amount of contrast, up to 300ml(3, 24). All patients undergoing embolization are at risk of developing contrast induced nephropathy, the risk factors for contrast induced nephropathy include the following:

- Age >75 years;
- Pre-existing renal impairment;
- Hypertension;
- Diabetes mellitus, especially with metformin use;
- Dehydration;
- And co-administration of nephrotoxic drugs(24).

The risk can be reduced by ensuring that patients are well hydrated, limiting the amount of contrast used, avoiding nephrotoxic drugs(3, 24). Patients must have their renal function monitored for 72 hours, an increase in creatinine more than 25% suggests contrast induced nephropathy(7). N-acetylcysteine has been suggested to reduce the incidence of contrast induced nephropathy however there is insufficient evidence to support its preventative use(24).

Complications

Intraoperative aneurysmal rupture / Vessel dissection

Rupture of the aneurysm during the manipulation of the aneurysm sac can present as sudden hypertension and bradycardia due to the sudden raise in intracranial pressure. Extravasation of contrast may also be seen on imaging(24). Risk factors for intraoperative aneurysmal rupture include small aneurysm size less than 3mm, atherosclerosis, worse Fisher grade SAH, and the presence of vasospasm(23)

The anaesthetist needs to control blood pressure, either by increasing the depth of anaesthesia or administering antihypertensive drugs to a systolic of less than 120mmhg or a mean arterial pressure of less than 80mmhg(23). Reversal of anticoagulation with protamine should be administered, and prepare for immediate transfer of the patient to theatre for craniotomy for surgical clipping of the aneurysm(24).

Intraoperative vascular occlusion

Intraoperative vascular occlusion can occur either one of the following; arterial thrombus, emboli, vasospasm, or coil displacement(24). Management involves blood pressure augmentation to 30-40% above baseline in order to increase collateral blood flow, with or without direct intra-arterial thrombolysis with abciximab(24). Mislaced catheter are removed and thrombectomy is performed if indicated(24)

Emergence and recovery

A smooth emergence is desirable to avoid coughing with consequent rise in intracranial pressure during extubation(3, 5). Rapid recovery from anaesthesia is essential to ensure early neurological assessment post intervention and safe transfer to the post-anaesthesia care unit(3, 22). Blood pressure should be allowed to return to normal, or up to a systolic of 160mmHg in secured aneurysms, while lower blood pressures may be preferred in those patients with unsecured or partially secured aneurysm in order to avoid the risk of rebleeding(22).

Post-operative management

Post-operative care for patients with SAH usually occurs in a high dependency unit, and those who suffered intra-operative complications will require care in an intensive care unit with post-operative sedation and ventilation(22). Ongoing neurological assessment and haemodynamic monitoring needs to be carried out during the post-operative period, with active observations for potential post-operative complications(3).

CONCLUSION

Subarachnoid haemorrhage is a serious disease with potentially devastating neurological outcome. Thorough assessment and careful perioperative management of these patients according to current recommendations is essential in order to improve outcomes, with particular attention to prevention of rebleeding and aggressive treatment of DCI and other related complications.

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