MANAGEMEMT OF SUBARACHNOID HAEMORRHAGE

ANAESTHESIA TUTORIAL OF THE WEEK 163

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QUESTIONS

1. The following are risk factors for a poor outcome post aneurysmal subarachnoid haemorrhage (SAH):
   a. Male sex
   b. Presence of co-morbid conditions
   c. Anterior circulation aneurysm
   d. World Federation of Neurosurgeons (WFNS) grade IV

2. Proven therapies in the management of vasospasm include:
   a. Triple H therapy
   b. Oral nimodipine
   c. Antifibrinolytics
   d. Balloon angioplasty

3. The following statements are true:
   a. The ISAT study demonstrated a better outcome for coiled versus clipped aneurysms
   b. The overall case mortality for SAH is 10%
   c. Intraoperative hypothermia improves neurological outcome
   d. CT is 100% sensitive in detecting aneurysms

INTRODUCTION

Subarachnoid haemorrhage (SAH) is a type of stroke, which occurs when there is bleeding in the subarachnoid space around the brain. The incidence in the UK is approximately 8 per 100,000 population per year (0.008%). The commonest cause of SAH is rupture of a cerebral aneurysm (70-85%) - other causes include arteriovenous malformations, tumours, trauma and non-aneurysmal perimesencephalic haemorrhage. This tutorial addresses the management of spontaneous **aneurysmal** SAH.

The anaesthetist may be involved in the management of patients with SAH at several stages; initial resuscitation and stabilisation, therapeutic management in the ICU and providing anaesthesia perioperatively for aneurysmal occlusion. The prognosis is poor, with an overall case fatality of 50%, however early treatment has been shown to improve outcome, and it is therefore imperative that the anaesthetist is familiar with all aspects of the condition, and up to date with the latest evidence based management. The mainstays of treatment are occlusion of the aneurysm, by endovascular coiling or surgical clipping, and prevention of cerebral ischaemia.

EPIDEMIOLOGY/AETIOLOGY

SAH accounts for 5-15% of all strokes, and has an overall case fatality of around 50% with a trend towards gradual improvement. This includes the 10-15% of patients who die before reaching hospital, the rest dying (almost always) within the first 3 weeks, secondary to re-bleeding and/or cerebral vasospasm. Of those who survive, one third remain moderately to severely disabled. The rate of re-bleed is 2-4% within the first 24 hours, and 15-20% within the first 2 weeks if left un-occluded. The main predictors of mortality and dependence are impaired level of consciousness on presentation, advanced age and a large volume of blood on initial CT scan.

The National Study of SAH described patient characteristics and aneurysm aetiology, based on findings from 2397 cases. The median age of patients was 52 years, of which 66% were women. The majority of aneurysms (89%) were located in the anterior (carotid) circulation, the rest being in the posterior (vertebrobasilar) circulation. In 25% of cases aneurysms are multiple. Almost half of the patients had concurrent medical conditions such as hypertension and ischaemic heart disease.

PATHOPHYSIOLOGY

Intracranial aneurysms are most likely to develop over the course of an individual's life, with only 10% accountable to a genetic/familial cause. Modifiable risk factors include hypertension, smoking and excessive alcohol intake, all of which approximately double the risk of aneurysmal development. Aneurysms develop mostly where there is turbulent flow at vascular bifurcations, on or near to the Circle of Willis. The most common sites are illustrated in Figure 1. Only a small proportion is attributable to infection or trauma. The majority are small, between 5-10 mm, when they rupture.

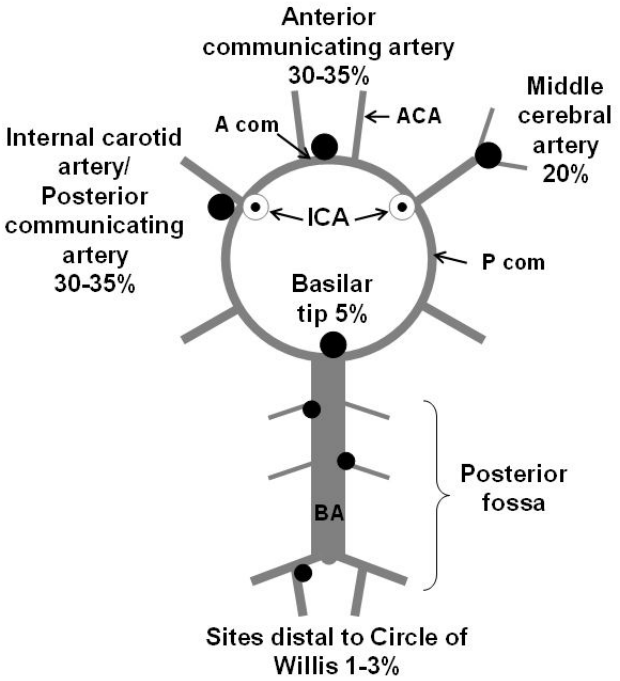

Figure 1: Common sites of aneurysms within the cerebral circulation. ACA - anterior cerebral artery; A com – anterior communicating artery; ICA – internal carotid artery; P comm. – posterior communicating artery; BA – basilar artery.

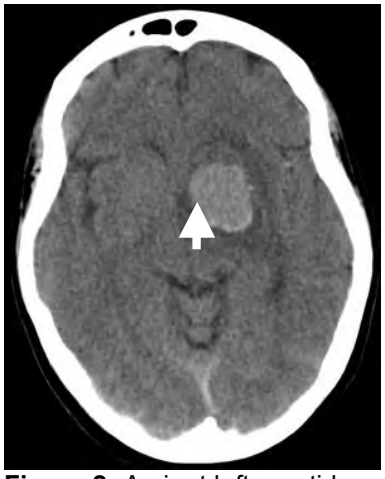

Figure 2: A giant left carotid artery aneurysm (arrow)

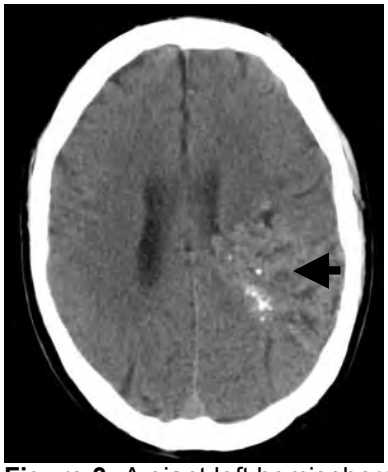

Figure 3: A giant left hemisphere arteriovenous malformation (AVM)

CLINICAL PRESENTATION

The most characteristic presenting symptom is a sudden onset severe headache, accounted for by the sudden rise in ICP which occurs at rupture. This is often accompanied by the development of meningism (meningeal irritation caused by blood in subarachnoid space), seizures, confusion/agitation and focal neurological deficits (due to focal ischaemia and/or cranial nerve involvement). By the time of admission, two thirds of patients have a depressed level of consciousness. A GCS score of less then 8 is usually associated with increased ICP. Systemic dysfunction is common in the acute phase, and may include cardiac ischaemia, and neurogenic pulmonary oedema.

There are several grading systems based on both clinical assessment and investigations. The World Federation of Neurosurgeons (WFNS) clinical grading system is the most widely used in the UK. (Table 1). The Fischer scale is described in Diagnosis.

Table 1: WFNS Grading scale for SAH. GCS, Glasgow Coma Score.

Grade	GCS Score	Motor Deficit
I	15	Absent
II	13 or 14	Absent
III	13 or 14	Present
IV	7 – 12	Either
V	3 – 6	Either

DIAGNOSIS

In the majority of cases the diagnosis of SAH is made by unenhanced (non-contrast) CT (Figure 3). Cranial CT is indicated in all patients complaining of sudden onset severe headache lasting for longer than one hour and with no alternative explanation. It should be performed as soon as possible after the patient presents, since SAH may not be evident on delayed CT if blood reabsorption has begun. CT may miss 2-7% of subtle SAH and in the case of a negative CT, further investigations are required. These include lumbar puncture, angiography (Figure 5B and 5C) and MRI or MRA (magnetic resonance angiography).

The amount of blood on CT can be described by the Fischer grading scale, which is scored from 1 to 4, 4 being the most extensive. It may be the best predictor of cerebral vasospasm complicating SAH and overall patient outcome. The location and distribution of blood on a positive CT aids the identification of the cause of the SAH, be it an aneurysm or other pathology. There are three methods to delineate this; contrast CT angiography, magnetic resonance angiography and catheter angiography, which is the gold standard. Angiography not only indentifies one (or more) aneurysm, it details the anatomical configuration, which allows optimum selection of occlusion management (coiling or clipping). The most recent development is in 3-D rotational angiography, which gives more detailed assessment of the aneurysm (Figure 5C).

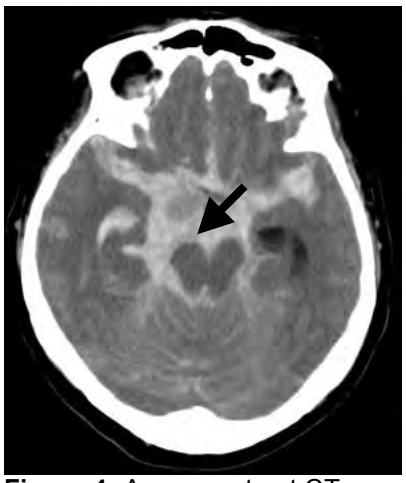

Figure 4: A non-contrast CT scan showing a large amount of subarachnoid blood

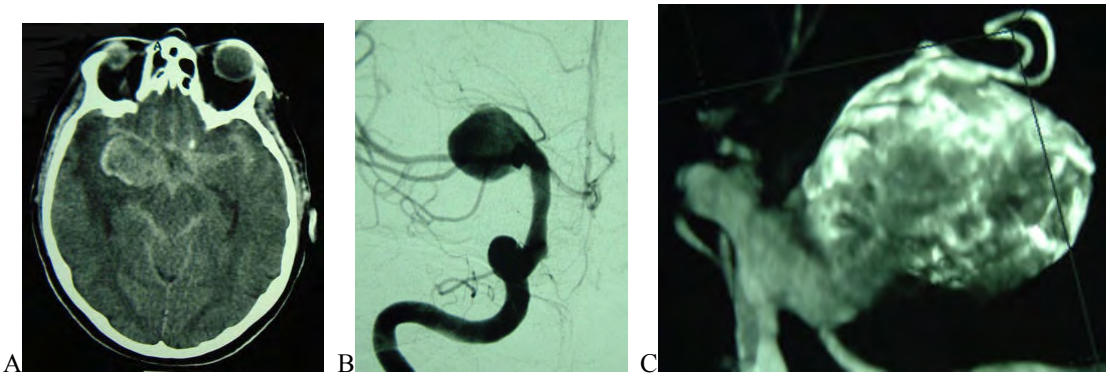

Figure 5: Imaging of a right 3cm diameter posterior communicating artery aneurysm. A, non-contrast CT; B, angiogram via right carotid artery; C, digital reconstruction of aneurysm to assess suitability for coiling

MANAGEMENT

There are 3 major neurological complications post ruptured cerebral aneurysm:

- Re-bleeding (Figure 6A),
- cerebral vasospasm leading to ischaemia (Figure 6B), and
- hydrocephalus (Figure 6C).

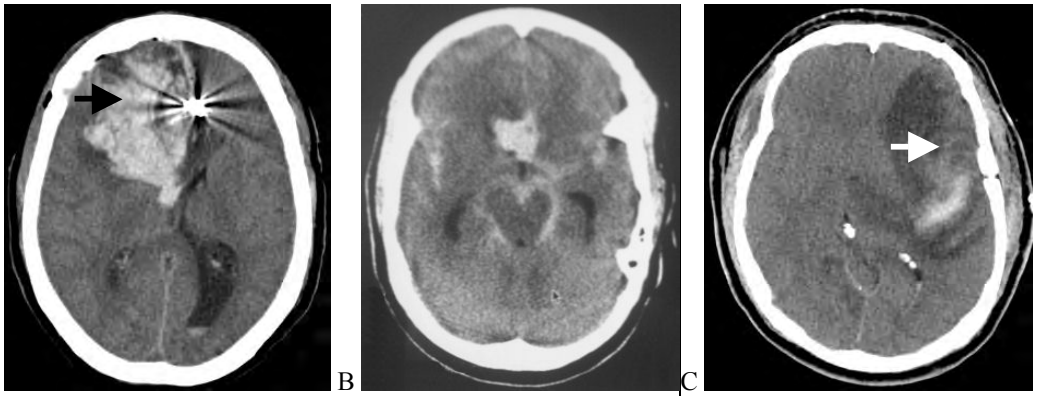

Figure 6: A, Right hemisphere bleed after clipping of an aneurysm; B,Left hemisphere infarction complicating vasospasm after SAH; C, Hydrocephalus post-SAH

Additionally, there are several systemic manifestations, most importantly, cardiopulmonary dysfunction and electrolyte disturbances. Management is therefore targeted at prevention of rebleeding by means of aneurysm occlusion, and management of complications. Depending on the neurological state of the patient, they will need to be managed on either the High Dependency Unit, or intubated and ventilated on Intensive Care. General neuroprotective strategies should be employed on ICU, particularly; adequate sedation, control of oxygenation and ventilation (CO_2 levels), avoidance of hypotension, prevention of hyperthermia, and normoglycaemia.

Occlusion Therapy

This can be done either surgically (clipping at craniotomy) or using a radiological endovascular technique with detachable coils (coiling). Early occlusion prevents re-bleeding. Evacuation of subarachnoid blood (at clipping) may also reduce the incidence of vasospasm. Early clipping or coiling, within 72 hours, is now the goal for all grades of SAH (WFNS I -V), replacing the out-dated approach of delayed aneurysm occlusion (until after the period of vasospasm) for poor grade SAH.

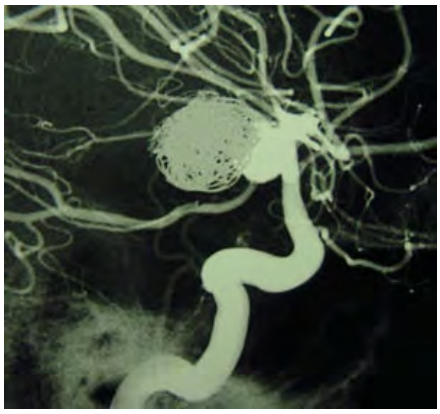

Figure 7: Angiogram showing aneurysm post-coiling

To clip or to coil?

Neurosurgical aneurysm clipping requires a craniotomy, performed under general anaesthesia. It takes 4 – 8 hours, and has a procedural mortality rate of 1-3%.

Coiling is a minimally invasive percutaneous endovascular treatment, which has proved to be a safe alternative to traditional surgical clipping of the aneurysm, and may be associated with a better outcome in selected patients. The technique consists of packing the aneurysm with detachable coils, and is performed under general anaesthesia. It avoids craniotomy, and recovery after the procedure is more rapid. It is not indicated in 5-15% of cases, due to morphological or positional aneurysm characteristics.

The largest randomised controlled trial comparing the two treatments is the ISAT trial. This multicentre randomised trial compared the efficacy and safety of endovascular coiling with surgical clipping in patients with aneurysmal SAH. It enrolled 2143 patients between 1994 and 2002, at 43 neurosurgical centres, and randomly assigned them to clipping or coiling. Patients had to be deemed suitable for either treatment to be included into the trial. The primary outcome measure was the proportion of patients either dead or dependent (by means of a modified Rankin scale, 3 – 6) at one year. Recruitment to the trial was stopped prematurely on the basis of an interim analysis in 2002. At one year, poor outcome (death or dependency) for the coiled group was 24%, compared with 31% for the clipped group. This was calculated to be an absolute risk reduction for coiling versus clipping of 7%, which means that for every 14 patients coiled instead of clipped, one poor outcome was prevented. ISAT has continued follow-up of the patients to look at long-term outcomes (mean follow-up 9 years), and the survival benefit of coiling has continued. The major concern regarding coiling is the potential increased risk of late rebleeding. ISAT has demonstrated that despite rebleeding being more common in the coiled group, the risk is very small, and does not cancel out the survival benefit of coiling over clipping. There are limitations to the ISAT study; it only included patients with the best prognosis – that is those that were neurologically capable of consenting, and the majority with aneurysms in the anterior circulation.

Prevention of Cerebral Ischaemia

Cerebral vasospasm is most likely to develop 3-12 days post SAH, lasting approximately 2 weeks. It occurs radiologically in about two thirds of SAH patients, of which half will develop symptomatic cerebral ischaemia causing neurological deficit of varying severity. It may also lead to increased ICP, secondary to infarction. It is diagnosed both clinically and radiologically, by means of angiography, CT angiography, and transcranial Doppler (TCD). A large amount of subarachnoid blood on CT is a predictor of the development of vasospasm.

Prophylaxis of vasospasm includes oral nimodipine, positive fluid balance, and prevention of hyponatraemia. Symptomatic treatment consists of triple-H therapy, balloon angioplasty, and intra-arterial papaverine.

Nimodipine

There is level 1 evidence to show that administration of oral nimodipine (a calcium channel blocker) improves outcome after SAH. It should be started at admission in all patients, at a dose of 60mg orally every 4 hours, and continued for 21 days. The oral (or NG) route has been shown to be just as effective as intravenous administration, but is associated with less hypotension.

Triple-H Therapy

The triple H refers to hypertension, hypervolaemia, and haemodilution. It is based on the concept that cerebral blood flow becomes pressure dependent once autoregulation is impaired, therefore an increase in blood pressure, and decrease in blood viscosity, may reverse vasospasm. The goal is to achieve a systolic blood pressure of 120-150mmHg in untreated aneurysms and up to 200mmHg in aneurysms that have been clipped or coiled, using hypervolaemia with vasoactive drugs where necessary. It is an accepted technique, despite being unproven. Serious complications are recognised, including pulmonary oedema, respiratory insufficiency, myocardial ischaemia, and it's use in unclipped aneurysms is controversial due to its potential to cause a re-bleed.

Balloon Angioplasty/Papaverine

This is indicated for patients with deteriorating neurological function, unresponsive to medical treatment. Angiography may be done with or without concomitant intra-arterial papaverine, used as a locally-administered arterial dilator. It does have serious complications including arterial dissection, rupture and thrombosis. Patients must not have infarction distant to the spastic vessel. Papaverine can also be used alone, for smaller vessel spasm, but is associated with seizures and irreversible brain injury.

Antifibrinolytic Therapy

There is no evidence that antifibrinolytic drugs, such as tranexamic acid, or anti platelets drugs, will improve outcome. The potential protection offered against rebleeding is offset by the increased risk of thrombo-embolism and cerebral ischaemia.

Hydrocephalus

This occurs when ventricular drainage is obstructed by blood clot, and causes a raised ICP. Although this may resolve spontaneously, some patients may require surgery for placement of a ventricular drain. It should be ruled out by CT scan before attributing neurological deterioration to vasospasm.

Other neurological complications include seizures and cerebral oedema.

Systemic Complications of SAH

Non-neurological complications occur in over half of SAH cases, and adversely affect outcome.

Cardiac dysfunction

SAH triggers the sympathetic nervous system, leading to excessive release of catecholamines and cardiac dysfunction. This is manifest as systemic and pulmonary hypertension, cardiac arrhythmias, myocardial infarction, and neurogenic pulmonary oedema. In severest form, patients may develop cardiogenic shock. Investigations may reveal an abnormal ECG and elevated cardiac troponin levels. Management is mainly supportive (inotropes, ventilation), and does not lead to long-term cardiac

dysfunction in most cases. The benefit of triple H therapy against vasospasm, may need to be balanced against the detrimental effects this may have on cardiac function.

Electrolyte derangements
Hyponatraemia is the commonest electrolyte abnormality, secondary to either cerebral salt wasting syndrome or the syndrome of inappropriate secretion of antidiuretic homone (SIADH). The former is caused by excessive secretion of brain and atrial natriuretic peptide, leading to hyponatraemia with volume depletion. It is managed with normal saline and IV fludrocortisone (or hydrocortisone). SIADH is associated with excess free water, however is managed with normal/hypertonic saline rather than water restriction, which assists with a hypervolaemic state, and thus may limit vasospasm development.

Potassium, calcium and magnesium levels should also be controlled by appropriate supplementation. Hyperglycaemia may occur secondary to sympathetic stimulation.

PERIOPERATIVE ANAESTHETIC MANAGEMENT

The principles of anaesthetic conduct should be applied whether the patient requires craniotomy for clipping, endovascular coiling, or another neurosurgical procedure to treat a complication of SAH (such as insertion of a ventricular drain).

Understanding neurophysiology is an essential part of giving a good 'neuro-'anaesthetic. The key is to maintain cerebral perfusion pressure (CPP), avoid sudden swings in intracranial pressure (ICP), control the transmural pressure gradient of the aneurysm (TMPG), provide optimal brain relaxation for surgery and enable rapid awakening for assessment in good clinical grade patients.

Remember that CPP = MAP – ICP, thus an adequate mean arterial pressure for brain perfusion and oxygenation is essential, especially in the presence of impaired autoregulation and elevated ICP. It is advisable to maintain the BP to within 20% of preoperative values. However, TMPG = MAP – ICP also, therefore there is a compromise to be met between maintaining the arterial pressure and minimising the TMPG to avoid sudden aneurysm rupture. This balance may differ depending on the clinical grade of the patient. Patients with no neurological deficit and normal ICP may tolerate a lower MAP to avoid increasing the risk of aneurysm rupture, however, patients in poor clinical condition may be very dependent on a high MAP for cerebral perfusion.

There are critical events, in the procedure, such as intubation and head pin insertion which the anaesthetist must be familiar with and anticipate, in order to deliver a smooth anaesthetic avoiding swings in MAP and ICP. In contrast to this, there are also times of minimal stimulation, when the blood pressure may need to be supported.

Optimal brain relaxation (thus ICP control), which enables surgical exposure, is achieved by giving a balanced anaesthetic with attention to maintaining oxygenation and normoventilation, controlling blood pressure and ensuring unobstructed venous return. The conduct of anaesthesia, and discussion of specific therapies will be discussed below.

Preoperative assessment

Most cases will be done on an urgent basis, in fasted patients. Remember that not all patients will be capable of participating in an assessment or consenting to procedures.

Aside from a thorough preoperative assessment, key areas to identify are the patient's neurological status and estimation of ICP. Attention to systemic complications, as above, is required with optimisation of any disturbances. Of course, achievement of full preparation for surgery must be balanced against the urgency of surgery/endovascular therapy.

Monitoring

Invasive monitoring of arterial blood pressure and CVP, by means of central or long line, is indicated where available. The arterial line should be inserted prior to induction to enable fine control of blood pressure at this crucial time. Central access will also allow for administration of vasopressor agents,

electrolytes and mannitol. All patients should have urinary output and core temperature measured. Some patients will have ICP monitoring in place, which maybe useful during induction.

The use of neurophysiological monitoring, such as evoked potentials, has not been shown to improve outcome. It has a poor predictive value, and is affected by the use of volatile anaesthetic agents. Jugular venous bulb monitoring has also not been established and may interfere with cerebral venous drainage. Neither are routinely used in the UK for aneurysm clipping.

Brain relaxation

ICP monitoring is indicated for some patients with poor clinical grade or hydrocephalus. Knowledge of the ICP, and the ability to drain CSF to control ICP, is particularly useful in the postoperative period for ventilated patients. Ventricular drains provide an effective means of reducing brain bulk by drainage of CSF perioperatively. Care should be taken not to drain too big volumes of CSF such as to cause brain sagging secondary to an acute drop in ICP. This can cause cardiovascular instability, and will increase the transmural pressure gradient across the aneurysm wall, thus increasing the probability of aneurysm rupture with torrential bleeding.

Pharmacological manipulation of ICP is also used. Mannitol, an osmotic diuretic, is usually the first choice, and is often administered post induction of anaesthesia for surgical clipping. It increases plasma osmolality, thus creating an osmotic pressure gradient across an intact blood brain barrier, resulting in water moving out of the cell. If the BBB is damaged, as occurs with brain damage, then mannitol may follow its pressure gradient and worsen cerebral oedema. Its full mechanism of action is complex, and is outside the discussion of this tutorial, but users must be aware that it can cause electrolyte derangements and metabolic acidosis. Mannitol is usually given as a 20% solution, at a dose of 0.5-1.0g.kg^{-1} over 20 minutes. If administered too quickly, it can cause an abrupt drop in ICP, which may increase the risk of aneurysm rupture. Furosemide (5-20mg) is sometimes given in combination with mannitol (or used alone at higher doses) to enhance its effect, but caution should be employed to avoid over-diuresis and hypovolaemia. The primary mechanism of action of furosemide in decreasing ICP is not fully understood.

Thiopentone is sometimes used for its cerebroprotective effect, particularly if temporary proximal clips are required in order to occlude the aneurysm. It is usually given as a bolus of 500mg, and may need to be accompanied by the use of vasopressors to counteract the hypotension caused. If given as an infusion, delayed emergence from anaesthesia should be expected.

The use of drugs and CSF drainage should not replace the delivery of a balanced anaesthetic, with adequate oxygenation, ventilation, cerebral perfusion pressure and unobstructed venous drainage (head position and head-up tilt, avoid tube ties and internal jugular central lines). Avoid hyperthermia, hyperglycaemia and acid-base imbalance. Temporary mild hyperventilation (aiming for $PaCO_2$ of 4.0kPa) can reduce brain bulk in critical situations, but should be balanced against the risk of cerebral ischaemia.

Anaesthetic Conduct

The principles of administering a smooth anaesthetic are discussed above. The emphasis of this, with regard to induction is to administer an adequate depth of anaesthesia, whilst maintaining an arterial pressure within 20% of baseline, and avoiding sudden increases which may increase the TMPG and thus cause aneurysm rupture.

Induction of anaesthesia is usually intravenous, with the use of nondepolarising muscle relaxation and an opiate. Propofol is the most commonly used agent, however etomidate or thiopentone are a suitable alternative if this is not available. Ketamine has been avoided in the past due to its effects on the ICP, however recent evidence of its safe use in head injury is emerging. Remifentanil, given as an infusion, can be titrated against the blood pressure and used to obtund the pressor response at intubation. Alternative drugs which can be given to blunt this response are lignocaine and esmolol. The endotracheal tube should be taped rather than tied, and the eyes protected.

Anaesthesia can be maintained with either volatile or total intravenous anaesthesia. Sevoflurane is the first choice volatile agent in the UK, as it causes the least change in cerebral blood flow (and thus ICP)

at concentrations of 1 MAC. At deeper levels of anaesthesia, cerebral vasodilatation will occur. Intravenous agents have the advantage of reducing cerebral blood flow and metabolic rate, thus conferring some cerebroprotective effect, which may be of significance in patients with raised ICP.

Mechanical ventilation is indicated, with attention to maintaining normoventilation. The benefits of positive end expiratory pressure on respiratory function need to be balanced against the risk of impaired cerebral venous drainage caused by increased central venous pressure.

There are several critical stimulating points perioperatively, including insertion of head-pins and raising the bone flap which may require boluses of remifentanil to counteract the pressor response. Similarly, there will be considerable time when surgical stimulation is minimal, especially after opening the dura during surgical dissection, when it may be necessary to administer a vasopressor such as phenylephrine or norepinephrine to maintain the MAP. This is preferable to reducing the depth of anaesthesia, which could result in the patient coughing.

Induced mild (33°C) hypothermia has not been shown to improve neurological outcome after craniotomy for aneurysm clipping, and leads to a higher incidence of bacteraemia. This was shown in the randomised prospective International Hypothermia Aneurysm Trial (IHAST), which included 1001 patients, with WFNS grade I – III, in 30 centres.

The risk of aneurysm rupture, particularly at clipping, may be reduced by local hypotension. This is best achieved by temporary clipping of the proximal artery, with maintenance of arterial blood pressure slightly above baseline to improve collateral flow. The clip should be on for no more than 20 minutes, and often the surgeon will request a bolus of thiopentone for cerebral protection, although there is no strong evidence for this. This technique may also be employed if there is sudden rupture of the aneurysm. Temporary clipping is preferred to induced hypotension, which impairs overall cerebral perfusion and is associated with a higher risk of cerebral vasospasm postoperatively.

Consideration to fluid balance is important. Normovolaemia is the goal until the aneurysm is clipped, after which careful volume loading may help reduce the incidence of postoperative cerebral vasospasm. This can be guided by invasive monitoring.

The intention is to wake up all good clinical grade patients postoperatively to assess neurological function, therefore the use of short acting analgesics, such as remifentanyl, perioperatively is ideal. Adequate analgesia is achieved with intravenous paracetamol, a non-steroidal (if no contraindication) and a small amount or morphine (2 - 5mg). All patients should receive antiemetics.

Intraoperative aneurysm rupture

This may occur at any time during the procedure, associated with a sudden rise in the TMPG (increased MAP or decreased ICP) across the aneurysm wall. It makes sense that if it occurs with the dura open, as opposed to at induction, for example, then the chance of occluding it will be better. It can lead to massive blood loss and carries a high mortality. The preferred acute management is temporary occlusion of the arteries proximal and distal to the bleeding, which allows surgical access. The blood pressure should be maintained and blood loss replaced. However, occasionally, surgical access may be so difficult that a transient decrease in MAP may be required to facilitate orientation and clipping. This will compromise cerebral perfusion and should be used as a last resort technique.

Recovery

Where available all patients will require High Dependency or Intensive Care postoperatively.

The goal is a smooth and rapid emergence, to allow early assessment of neurological function. If emergence is unexpectedly delayed, or there is a new neurological deficit, then CT or angiography is indicated. Often patients will be relatively hypertensive, and will require further morphine (or an antihypertensive drug) after assessment of neurology. Shivering and vomiting should be treated. Some degree of hypertension, above preoperative levels, may be beneficial in preventing vasospasm, but this should not be to the extent of risking intracranial haemorrhage.

Patients with WFNS grade III – V, or with perioperative complications, should not be extubated after the procedure, and will require management on Intensive Care.

Anaesthesia for Coiling

Endovascular coiling is undertaken in the angiography suite by neuroradiologists. The same monitoring facilities and anaesthetic assistance should be available. A general anaesthetic is usually required, and the principles of administering a 'neuro-'anaesthetic are the same. It is less likely to be complicated by procedural blood loss and the need for brain relaxation, but if rupture of the aneurysm does occur, then this is invariably fatal (no surgical access). The benefits of coiling over clipping, for suitable patients, have already been discussed.

SUMMARY

Mortality from SAH is high. Poor clinical condition on admission is a predictor of a poor outcome. Management is aimed at early occlusion of the aneurysm, prevention of vasospasm leading to cerebral ischaemia and supportive management of systemic complications. An anaesthetist who understands the pathophysiology of the condition, and can manage the patient on both ICU and perioperatively, will contribute to an improved outcome.

ANSWERS TO QUESTIONS

1. FTFT

   Aneurysms are more common in females, sex does not alter survival outcome. Risk factors for an unfavourable outcome are; age, poor neurological condition on admission, posterior circulation/>10mm size, comordity especially hypertension, blood in subarachnoid space on CT.

2. FTFF

   Triple H therapy is often used for symptomatic vasospasm, but has not been proven to be effective. There is no evidence that balloon angioplasty is better than medical treatment. There is level 1 evidence that commencing oral nimodipine at admission in all cases of SAH will decrease development of vasospasm. Antifibrinolytics (potential to decreases rebleeding) do not improve outcome and may increase thromboembolic risk.

3. TFFF

   The outcome advantage for coiling comes from level 1 evidence, note it included patients in mostly good neurological condition who had to be deemed suitable for both treatments. The overall fatality is nearer 50%. Mild intraoperative hypothermia did not improve outcome in the IHAST trial. CT may miss small/delayed presentation SAH, and therefore lumbar puncture may be indicated in some patients before the diagnosis can be excluded.

WEBLINKS

For links to ISAT trial full reports:
http://www.surgery.ox.ac.uk/nvru/isat

National Study of Subarachnoid Haemorrhage Final Report, The Royal College of Surgeons 2006
http://www.rcseng.ac.uk

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