The annual incidence of subarachnoid haemorrhage (SAH) due to rupture of a cerebral aneurysm is 10 per 100,000 per year. About 15% of patients who suffer a SAH will die before reaching hospital. Of the remainder, 25% will die in hospital and only 50% will make a full recovery. The risk of dying is highest during the first 24 h after haemorrhage, but remains significant for a further 2 months.

Rupture can occur at any age but is most common in 50–60-year-olds. This is the age group where other co-morbid conditions may make anaesthesia more complex. The commonest co-morbid condition is essential hypertension which may be present in as many as 40% of patients prior to rupture. Polycystic kidney disease and coarctation of the aorta are also associated with cerebral aneurysm.

Symptoms and signs
The following symptoms and signs may be present:

- Headache: severe with sudden onset
- Loss of consciousness: transient or prolonged
- Vomiting
- Seizures
- Focal neurological signs: due to pressure effect from haematoma or a large aneurysm
- Neck stiffness: develops 3–12 h after SAH
- Positive Kernig’s sign
- Papilloedema
- Vitreous haemorrhage: caused by sudden rise in ICP
- Reactive hypertension
- Pyrexia

Loss of consciousness, particularly if prolonged, is related to the size of the haemorrhage which is itself positively correlated with a poor outcome. Various scales have been suggested to quantify the severity of haemorrhage and to predict outcome. The World Federation of Neurosurgeons has approved the scale shown in Table 1.

### Complications of subarachnoid haemorrhage
It is important to understand the pathophysiology of these complications because their presence will complicate the administration of anaesthesia.

#### Rebleeding
Rebleeding is one of the most feared complications and is usually manifest as a deterioration in neurological state. If rupture occurs during anaesthesia, the signs are usually dysrhythmias and massive brain swelling. If a ruptured aneurysm is untreated, there is a 30% chance of rebleeding in the first 28 days. Approximately 70% of patients who suffer a second bleed will die. This explains why early treatment is favoured. Rupture is thought to occur when an excessive gradient develops between the pressure in the aneurysm and that of the surrounding brain tissue. The transmural pressure gradient is actually the same as the cerebral perfusion pressure (CPP), i.e., mean arterial pressure (MAP) minus intracranial pressure (ICP).

### Table 1 Severity scale for subarachnoid haemorrhage

<table>
<thead>
<tr>
<th>World Federation of Neurosurgeons’ grade</th>
<th>Glasgow Coma Score</th>
<th>Motor deficit</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>Unruptured aneurysm</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>15</td>
<td>Absent</td>
</tr>
<tr>
<td>II</td>
<td>14–13</td>
<td>Absent</td>
</tr>
<tr>
<td>III</td>
<td>14–13</td>
<td>Present</td>
</tr>
<tr>
<td>IV</td>
<td>12–7</td>
<td>Present or absent</td>
</tr>
<tr>
<td>V</td>
<td>6–3</td>
<td>Present or absent</td>
</tr>
</tbody>
</table>
Raised intracranial pressure
Raised intracranial pressure occurs at the time of SAH and may be responsible for stimulating the sympathetic discharge which also occurs. It usually falls rapidly, but there may be a secondary rise caused by blockage of subarachnoid villi, occlusion of cerebrospinal fluid outflow, arterial spasm or intracerebral haematoma. Patients with a reduced conscious level are most likely to have a secondary rise in ICP.

Cerebral ischaemia or infarction
Cerebral ischaemia or infarction can occur immediately following SAH but is most common 4–12 days after the haemorrhage. It occurs in approximately one-quarter of patients. Contributing factors include vasospasm and inflammatory changes in the vascular endothelium. Hypovolaemia and reduced CPP caused by haematoma, cerebral oedema or hydrocephalus can increase the severity of ischaemia.

Some patients with angiographic vasospasm are asymptomatic. Similarly, although not all patients have radiological or clinical evidence of ischaemia, all patients have reduced cerebral blood flow following SAH and this may persist for 3 weeks. Additionally, cerebral metabolic rate for oxygen is reduced in excess of cerebral blood flow indicating metabolic uncoupling. Loss of autoregulation sometimes occurs, most commonly in poorer grade patients and those who develop clinical signs of ischaemia. The prophylactic use of nimodipine also causes loss of autoregulation. Vascular carbon dioxide reactivity is impaired in some, but not all, patients.

Hydrocephalus
Hydrocephalus occurs in one-fifth of patients and, if severe, may produce unconsciousness. Extraventricular drainage of cerebrospinal fluid through a burr hole is the usual treatment and is often performed under local anaesthesia. The anaesthetist may be asked to assess the patient to confirm that the airway will not be at risk during the procedure.

‘Expanding’ intracerebral haematoma
An ‘expanding’ intracerebral haematoma describes an increase in ICP caused by secondary swelling around a haematoma. It may cause a reduction in conscious level or progression of focal signs.

Epilepsy
Epilepsy occurs at the time of the SAH or later if a haematoma has caused cortical damage. Prolonged seizure activity can increase the risk of cerebral ischaemic damage.

Catecholamine release
Catecholamine release at the time of SAH can be massive and is thought to be responsible for the reactive hypertension, electrocardiographic (ECG) changes, dysrhythmias and pulmonary oedema that commonly occur. Plasma norepinephrine concentrations are raised for 2 weeks following SAH.

The significance of ECG changes is uncertain. Although some patients have echocardiographic evidence of reduced motion at the ventricular apex, the coronary arteries are usually normal. It is believed that ST segment and T wave changes represent myocardial dysfunction rather than injury.

Hyponatraemia
Hyponatraemia frequently accompanies SAH. Its aetiology is uncertain and may be due to renal sodium loss or inappropriate secretion of antidiuretic hormone.

Gastric aspiration
Aspiration of stomach contents is a risk for any patient with a significant reduction in conscious level and/or obtunded airway reflexes. The SAH and many of its complications cause loss of consciousness which, if likely to be prolonged, should be managed by intubation and ventilation.

Specific measures for cerebral protection following SAH

Nimodipine
Nimodipine is a calcium antagonist and has been shown to reduce the incidence of cerebral infarction by one-third if given prophylactically after SAH. Usually, it is given by the oral route in a dose of 60 mg every 4 h. This method of administration is rarely associated with significant side effects. However, in the presence of vasospasm confirmed by angiography or transcranial Doppler, some surgeons prefer the intravenous route. Systemic hypotension can occur and is treated with colloids and dopamine or dobutamine. The choice of inotrope seems to be based on surgical tradition. Norepinephrine is equally effective and is preferred by many anaesthetists.

Despite the fact that nimodipine can cause systemic vasodilatation, the outcome improvements associated with its use do not correlate with angiographic improvements in vasospasm and the reasons for its beneficial effect are unclear.

Maintenance of cerebral perfusion pressure and cerebral blood flow
Maintenance of cerebral perfusion pressure and cerebral
blood flow is of crucial importance. Circulating blood volume is frequently reduced after SAH and should be restored with colloid. If there is any doubt about the volume required, central venous pressure measurement is a useful guide. It is usually recommended that patients be given at least 3 litres of maintenance fluid daily. Serum sodium should be measured at least daily. If the concentration begins to fall, oral sodium chloride tablets may be sufficient replacement. If the serum sodium falls below 130 mmol litre⁻¹, fludrocortisone or hypertonic saline should be given. Fluid restriction is not recommended because of the risk of ischaemia.

Control of mean arterial pressure
In the event of either essential or reactive hypertension, antihypertensive therapy is not usually recommended even though there is a risk of rebleeding. This is because, if autoregulation is compromised, reducing the blood pressure may compromise cerebral blood flow and cause cerebral ischaemia.

In the presence of cerebral ischaemia that has caused a deterioration in the neurological state, colloid challenges are usually given to ensure that hypovolaemia has been corrected. Because the reduction in blood volume associated with SAH includes all components, the haematocrit remains normal until additional volume is given. Blood transfusion is considered only if the haematocrit falls below 33%. A degree of haemodilution reduces blood viscosity and improves cerebral blood flow. If volume replacement fails to bring about an improvement, the blood pressure should be raised with norepinephrine. Some surgeons set a target systolic blood pressure, but it is better to raise the systolic blood pressure in a controlled manner by 10 mmHg every 20 min until clinical improvement has occurred. The rationale is that, in the absence of autoregulation, increased systemic pressure will increase cerebral blood flow. If titrated hypertensive therapy does not reverse the neurological deficit, angioplasty may be indicated.

Caution is advised if the aneurysm is untreated and, therefore, may rebleed. In this situation, the maximum systolic blood pressure tolerated is limited to 160 mmHg.

The combination of hypervolaemia, haemodilution and hypertensive therapy is sometimes called ‘Triple H’ therapy.

Definitive treatment of ruptured cerebral aneurysm
Surgery
Traditionally, aneurysms have been clipped and those that have a wide neck or are impossible to clip have been re-inforced with muscle or artificial material. The advantages of open surgery include the fact that haematoma (which may cause vasospasm) can be removed and, if the aneurysm ruptures during the procedure, clipping can still be achieved.

Interventional radiology
Interventional radiology using coils or balloons to occlude the aneurysm is a more recent development. The techniques are currently being evaluated in comparison with surgery. At present, there appear to be benefits because aneurysms that are difficult to access by surgery can be easily treated and the patient is not subjected to a craniotomy. However, if the aneurysm ruptures during the procedure, the outcome does not appear to be as good as when an aneurysm ruptures during an open surgical procedure.

Goals of anaesthetic management
These are to: (i) provide optimum operating conditions for the surgeon; (ii) prevent an increase in transmural pressure which may cause rupture of the aneurysm; and (iii) maintain adequate cerebral perfusion pressure and cerebral oxygenation.

Anaesthetic management of mean arterial pressure
The prevention of a rise in MAP is essential at all times but especially at laryngoscopy, during the placement of head pins and after the skull is open when ICP is atmospheric. Various methods of prevention are described and are summarised in Table 2. None of these measures have been shown to be clearly superior to any other.

Uncontrolled hypotension should also be avoided because this may cause cerebral ischaemia. Controlled hypotension used to be a popular method of reducing transmural tension in the aneurysm during manipulation prior to clipping. A safe

Table 2 Methods of minimising hypertension during induction and anaesthesia

| Pre-operative β-blockade |
| Captopril 2–3 mg kg⁻¹ as premedication |
| Adequate dose of alfentanil or remifentanil |
| Adequate induction dose of thiopental or propofol |
| Bolus dose of induction agent immediately prior to stimulus |
| Intravenous lidocaine (1.5 mg kg⁻¹) |
| Hyperventilation with isoflurane through a face mask |
| Vasoactive drugs to reduce MAP (trimetaphan, phenolamine, PGE₁) |
| Use of nerve stimulator to confirm muscle paralysis |
| Careful intubation technique |
lower limit for mean arterial pressure has never been established and the value of the technique for preventing rupture has also not been proved. Many surgeons now prefer to place a temporary clip onto vessels feeding the aneurysm. If this is done, most anaesthetists maintain a normal MAP but some prefer to increase MAP by 10–15% to improve collateral flow.

In addition to active manipulation of the blood pressure, it is important to ensure that the patient is normovolaemic prior to induction of anaesthesia and that fluid and electrolyte balance are maintained during anaesthesia.

**Anaesthetic management of intracranial pressure**

The World Federation of Neurosurgeons’ grades I and II patients normally do not have raised ICP, but those with higher grades do. Usually, a goal of neuroanaesthesia is to provide a slack brain in order to improve surgical access. During anaesthesia surgery, the ICP must not be lowered unless the MAP is also lowered or the transmural pressure of the aneurysm will increase. It is usual to maintain the arterial carbon dioxide tension at the lower end of the normal range. Particular caution is needed when mannitol is used before the skull is open. Mannitol causes an increase in circulating blood volume and reduces ICP. Therefore, there is a theoretical risk that the aneurysm could rupture. Mannitol is most safely given after the skull has been opened.

Lumbar drainage of cerebrospinal fluid is also requested by some surgeons. The drain should only be opened when the skull is open and drainage should be limited to a maximum rate of 5 ml min⁻¹.

**Cerebral protection using hypothermia**

Intra-operative mild hypothermia is believed by many neurosurgeons to offer cerebral protection. A recent study has shown that the balance between cerebral oxygen supply and demand may worsen under mild hypothermic conditions. Therefore, hypothermia should not be used unless a method of monitoring cerebral oxygenation is available.

**Anaesthetic technique for surgery**

Anaesthesia for aneurysm surgery is a challenge for all the reasons already discussed. Careful planning is essential and should be guided by the patient’s pre-operative condition. The key features relating to induction of anaesthesia have already been described. Maintenance of anaesthesia may be with total intravenous anaesthesia (e.g. propofol infusion plus remifentanil or alfentanil infusion) or with inhalational agents (such as isoflurane or desflurane). The inhalational agents are usually supplemented with boluses of fentanyl and muscle relaxation is maintained with atracurium or vecuronium. There is limited evidence to suggest that, although the eventual outcome is the same for both techniques, total intravenous anaesthesia is associated with shorter operation time and shorter length of hospital stay. The reasons for this finding are unclear, but may indicate that operating conditions are better.

Routine monitors should include electrocardiogram, pulse oximetry, capnography, intra-arterial blood pressure, central venous pressure, urinary output, temperature and arterial blood gases and electrolytes. Pulmonary artery pressure, ICP, brain stem evoked potentials and venous jugular bulb oximetry should be considered in patients at high risk of cardiac complications or cerebral ischaemia.

**Anaesthetic technique for interventional radiology**

The principles and practice of anaesthesia for interventional radiology are the same as for surgery. Rupture of the aneurysm can be catastrophic. The anaesthetist should be prepared to reverse heparin anticoagulation with protamine and to take urgent measures, which may include hyperventilation and mannitol, to reverse extreme rises in intracranial pressure.

**Postoperative analgesia**

Traditionally, codeine phosphate has been used for postoperative analgesia because it is thought to be ‘safe’. It is, however, metabolised to morphine, and may not provide adequate analgesia in approximately 20% of patients. Adequate pain relief should always be given, using morphine if necessary. When using morphine, it is important to ensure that the neurosurgical team and the nursing staff understand that a deterioration in the patient’s conscious level should be taken seriously and is highly unlikely to be due to the morphine.

**Key references**

Archer DP, Leblanc RL. Haemodynamic considerations in the management of patients with subarachnoid haemorrhage. Can J Anaesth 1991; 38: 454–70


See multiple choice questions 32–35.