

## Cerebral Challenge

Andrew Macalister Hall and Liz Turner

Correspondence: andrewmhall@doctors.org.uk

### CASE 1

A 70-year-old man in the intensive care unit has a feeding nasogastric tube inserted. The nursing staff cannot aspirate anything from the tube in order to test for acidity and confirm gastric placement. A chest radiograph is requested to confirm its placement and you are asked if it can be used for feeding (Figure 1).

- Where does the tip of the NG tube lie?
- What would be the next most appropriate course of action?
- What tests can be used to confirm correct placement of NG tubes?



Figure 1. Chest radiograph of patient 1

### CASE 2

You are asked to anaesthetise a 65-year-old man as a day case for inguinal hernia repair. He is otherwise well, and takes ramipril 5 mg for essential hypertension. You have asked for an ECG prior to anaesthetising him (Figure 2).

- What does the ECG show?
- What implications does this have on anaesthetising this patient?
- What is the longer term management of this condition?

### CASE 3

A previously fit and well 42-year-old woman presents to the emergency department complaining of sudden-onset severe headache that occurred while she was out shopping. She describes the headache as the worst headache she has ever had. The headache started 30 minutes ago and has not improved. Her daughter reports that she appears confused. While in the emergency department her consciousness level deteriorates and she is taken for urgent computed tomography (CT) of the head (Figure 3).

- What does this CT head show?
- How would you manage this patient?

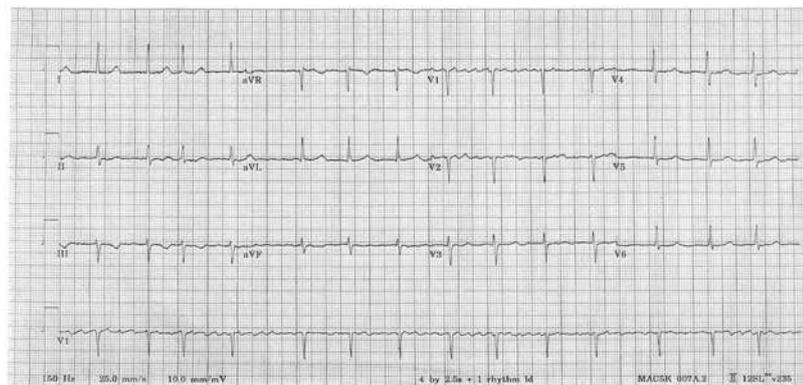


Figure 2. ECG of patient 2

**Andrew Macalister Hall**  
Specialist Trainee in  
Anaesthesia

**Liz Turner**  
Core Trainee in Anaesthesia  
Royal Devon and Exeter NHS  
Foundation Trust  
Exeter EX2 5DW  
UK

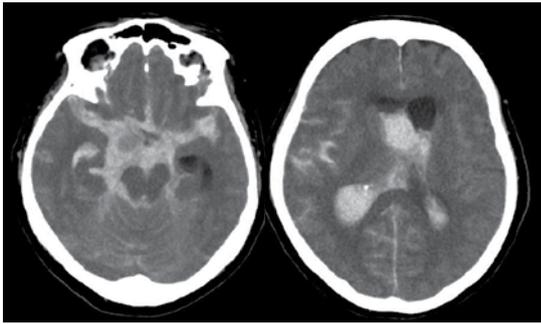


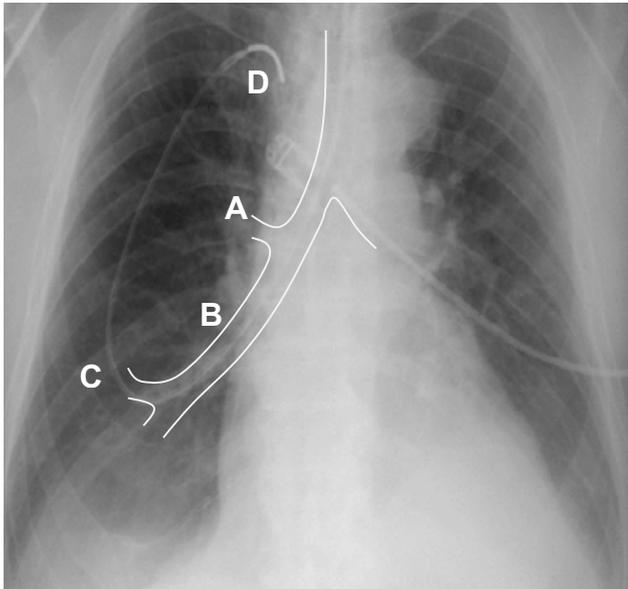
Figure 3. CT head of patient 3

## DISCUSSION

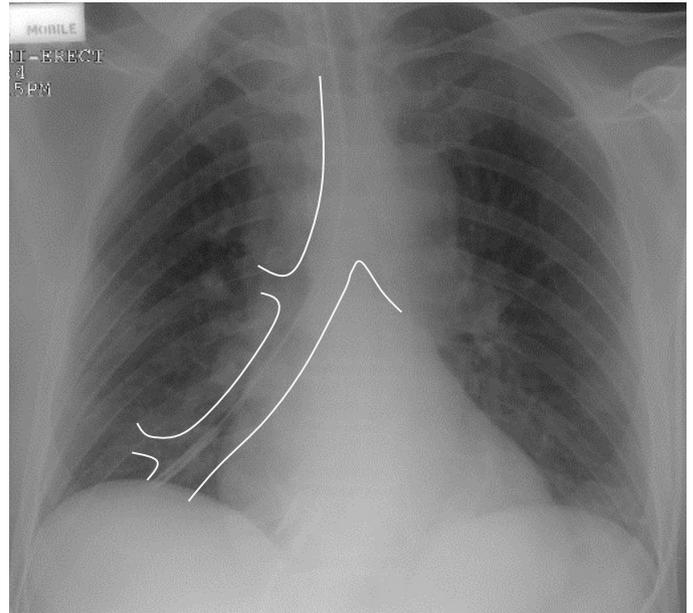
### Case 1

#### Guidance on confirmation of correct nasogastric tube placement

It is important to recognise correct nasogastric placement, as it is



**Figure 4.** Chest radiograph of patient 1. It shows that the nasogastric tube has entered the larynx rather than the oesophagus, passing down the trachea and into the right main bronchus. The right upper lobe aperture is usually within 1.5–2.5 cm from the carina (A) and the nasogastric tube has not entered this bronchus, but has continued down the bronchus intermedius (B). The nasogastric tube has probably been passed into the right middle lobe bronchus (C). After this its passage does not follow the anatomy of the bronchial tree, and to have reached its final position it must have been pushed through the bronchial wall and has entered the right pleural cavity (D). Feeding through this tube would result in accumulation of nasogastric feed within the pleural cavity ('feed-o-thorax') with respiratory compromise and other life-threatening consequences. The tube should be completely removed from the patient. As the visceral pleura has been breached there is a risk that the patient may develop a pneumothorax after the tube is withdrawn; the patient must be monitored carefully for 6 hours and the chest radiography repeated to exclude this



**Figure 5.** Chest radiograph of a different patient. This time the nasogastric tube is still in the lungs, but has not been advanced too far and so is still within the bronchial tree, probably lying within the right lower lobe bronchus

a commonly used piece of equipment with the potential to cause serious harm. In the UK alone around 170 000 tubes are supplied to the NHS each year. There is the risk of significant harm to the patient if drugs or feed are passed down a nasogastric tube that has been placed within the lungs (Figures 4 and 5). Between 2005 and 2011 the National Patient Safety Agency (NPSA) was notified of 21 deaths and 79 cases of harm due to misplaced nasogastric tubes.<sup>1</sup> The single greatest cause of harm was due to misinterpretation of radiographs, accounting for about half of all incidents and deaths. Misplaced nasogastric tubes have been confirmed by the Department of Health in England as a 'never event', one of a restricted list of serious avoidable events.<sup>2</sup> Box 1 shows an example of guidance to guide safe NG tube placement.

### References

1. National Patient Safety Agency, UK. *Reducing Harm by the Placement of Nasogastric Feeding Tubes* (February 2005). Available at <http://www.nrls.npsa.nhs.uk/resources/?EntryId45=59794>
2. Mayor S. NHS extends never events list and introduces cost penalties. *BMJ* (2011); **342**: d1263. Available at <http://www.medicalprotection.org/uk/casebook/casebook-september-2012/nasogastric-tube-errors>
3. Black R. *Confirmation of NG placement in ICU*. Royal Devon and Exeter Hospital, 2016.
4. National Patient Safety Agency, UK. *Harm from Flushing of Nasogastric Tubes Before Confirmation of Placement* (March 2012). Available at <http://www.nrls.npsa.nhs.uk/resources/type/alerts/?entryid45=133441>

### **Box 1. Example of local ICU guidance on safe nasogastric tube placement<sup>3</sup>**

#### **A. Initial placement – pH testing of aspirate**

- A pH < 5.5 indicates gastric placement.\* Document tube length at nose and date and time of insertion.
- If unable to obtain an aspirate try:
  - rolling the patient onto his or her side
  - advancing the tube 10 cm
  - injecting 10–20 mL of air and re-aspirating after 10 minutes.
- If pH > 5.5, consider retesting in 1 hour provided the patient is not on acid-suppressing medication (in which case, proceed to radiological confirmation).
- If pH testing cannot confirm the correct position of the tube, placement should be confirmed by chest radiography. The only exception to this is where the nasogastric tube has been felt in the stomach by the operating surgeon at laparotomy, or the tube has been placed during upper gastrointestinal endoscopy.

#### **B. Radiological confirmation of placement**

- Radiological confirmation should be performed by suitably competent individuals – this will involve training for new staff.
- The nasogastric tube should:
  - adhere to the midline
  - bisect the carina
  - be visible throughout its length, and
  - pass beneath the diaphragm in the midline.
- The person confirming position should document this in the patient's notes (including the date and time the confirming radiograph was obtained).
- If possible, a single chest radiograph should be taken to confirm the position of an endotracheal tube, invasive lines, drains, etc.

#### **C. Confirmation following interruptions in feeding**

- Patients in the ICU experience multiple interruptions in nasogastric feeding, for example to facilitate imaging, transfer between hospital departments, administration of certain drugs, etc. In addition, there is a theoretical risk of tube migration into the oesophagus, particularly in patients who vomit or retch.
- Reconfirmation of nasogastric tube position using the methods described above leads to unnecessary delays in reintroducing feed.

#### **Prior to interruption of feed**

- Document the tube marking at the nose

#### **Prior to restarting feed**

- If the nasogastric tube position is unchanged, there has been no retching/vomiting and no evidence of coiling at the back of the throat and there is no clinical suspicion the tube has dislodged, feed can be restarted without pH testing/chest radiography. This should be documented in notes.
- If these conditions are not met, confirmation needs to be as for 'Initial placement'

\*According to the NPSA (UK) two patients have died as a result of staff flushing nasogastric tubes with water before aspirating fluid to measure pH. The mix of water and lubricant caused the pH reading to fall below 5.5, leading the staff to assume that the nasogastric tubes were correctly placed, when they were not.<sup>4</sup>

<sup>†</sup>In 2005 the UK's NPSA issued guidance highlighting the unreliability of the 'whoosh test' (using a stethoscope to listen for bubbling sounds over the stomach after blowing air through the nasogastric tube with a syringe). This technique should not be used.

**Case 2**

This 12-lead ECG shows narrow QRS complexes that are irregularly irregular that are not preceded by P waves. This patient has atrial fibrillation (AF) with a ventricular rate of 85 bpm.

Atrial fibrillation is the commonest arrhythmia and has an estimated prevalence of 1–2%.<sup>1</sup> It is associated with significant morbidity and mortality, including cerebrovascular events and heart failure. Atrial fibrillation is due to uncoordinated atrial electrical activity which intermittently progresses to atrioventricular node conduction, causing irregularity in ventricular depolarisation. When in sinus rhythm, atrial contraction delivers between 10% and 40% of ventricular filling;<sup>1,2</sup> however, this is reduced in AF due to uncoordinated atrial contractions.

**Implications of AF for anaesthesia and surgery**

There are many causes of AF (see Table 1). As this patient was previously unknown to have AF, further investigation may be indicated prior to proceeding with general anaesthesia. The Association of Anaesthetists of Great Britain and Ireland (AAGBI) suggests that, prior to elective surgery, routine electrocardiography (ECG) should be carried out in all patients over the age of 40 who are classified as ASA (American Society of Anesthesiologists) 2 and those who have cardiovascular disease.

Almost all anaesthetic medications are negatively ino- and chronotropic, meaning that on induction of anaesthesia the patient's cardiac output will fall as a result of a reduction in both heart rate and cardiac muscle contractility. A reduction in ventricular filling due to uncoordinated atrial contraction will cause a more profound fall in cardiac output and therefore hypotension when the patient is anaesthetised. It is essential to maintain a good cardiac output with a controlled rate, monitoring for any rate related ischaemic changes or hypotension. However, AF with a normal ventricular rate does not normally cause any major anaesthetic problems.

Assuming that further history and examination shows the patient to be otherwise fit and healthy, with no evidence of any of the conditions listed in Table 1 that would preclude safe and anaesthesia and surgery, it is reasonable to proceed. His heart rate is currently controlled with no need for rate controlling medication.

**Longer-term management of AF**

The long-term management of atrial fibrillation is complex and multimodal. The following factors should be considered in the management of AF.

**Was the onset of AF within the last 48 hours or is it pre-existing?**

It is important to establish the time of onset of atrial fibrillation because of the risk of formation of atrial thrombus due to the turbulent blood flow. This could potentially lead to clot embolism, causing a cerebrovascular event (CVE) – this is a particular risk when the rhythm changes between sinus rhythm and AF. The risk of embolism is greatly reduced if cardioversion takes place in the first 48 hours. After 48 hours, transthoracic or, preferably, transoesophageal echocardiography should be performed to look for thrombus and, if necessary, anticoagulant treatment administered for a minimum of 3 weeks prior to cardioversion.<sup>3</sup> Remember to look for and treat any reversible causes of AF such as electrolyte abnormalities and hyperthyroidism.

**Is there evidence of cardiovascular compromise?**

If the patient has adverse features related to atrial fibrillation, then urgent management is needed. Adverse features include shock (hypotension, tachycardia), myocardial ischaemia, syncope and heart failure.<sup>4</sup> The treatment would be assessment using the ABCDE approach and synchronised DC cardioversion with an initial energy of 120–150 J<sup>4</sup> to try to restore sinus rhythm and treatment of any reversible causes. DC cardioversion should not be performed on a conscious patient and so, unless the patient is unconscious, anaesthetic support should be gained in order to perform the procedure under general anaesthesia. There is no time in this situation to arrange for echocardiography or to anticoagulate the patient.

**Is the aim to achieve rate control or rhythm control?**

Longer-term pharmacological management includes either rate or rhythm control. Rate control is defined as a heart rate of 60–80 bpm at rest and between 90 and 115 bpm during exercise. This could be achieved by a number of drugs, including beta-blockers or cardiac glycosides (e.g. digoxin). Rhythm control (chemical cardioversion)

**Table 1.** Risk factors for developing atrial fibrillation<sup>1</sup>

System	Aetiology	Assessment
Cardiovascular	Underlying heart disease, valvular heart disease, cardiomyopathy, pre-existing excitatory syndromes (e.g. Wolff–Parkinson–White), sinus node disease	12-lead ECG, echocardiography, stress testing, R-tests (72-hour ECG)
Respiratory	Hypoxia, pneumonia, effusions, thromboembolic disease	Pulse oximetry, arterial blood gas, chest radiography
Electrolyte	Low or high potassium, low magnesium, low calcium	Serum biochemistry
Metabolic	Acidosis, alcohol excess, thyrotoxicosis, diabetes mellitus	Arterial blood gases, thyroid function tests, liver function tests, blood glucose levels, toxicology screen
Others	Increasing age, hypovolaemia	Fluid status assessment

can be achieved by medications such as sotalol, flecainide or amiodarone, but should be attempted only after anticoagulation because of the risk of embolism.

### Does the patient need anticoagulation?

Patients who have persistent, permanent or paroxysmal AF need to be considered for anticoagulation therapy because of the increased risk of clot formation and stroke. Patients can be risk stratified using the CHADS2-VASc score (Table 2) to estimate their risk of stroke and decide if they need anticoagulation therapy.

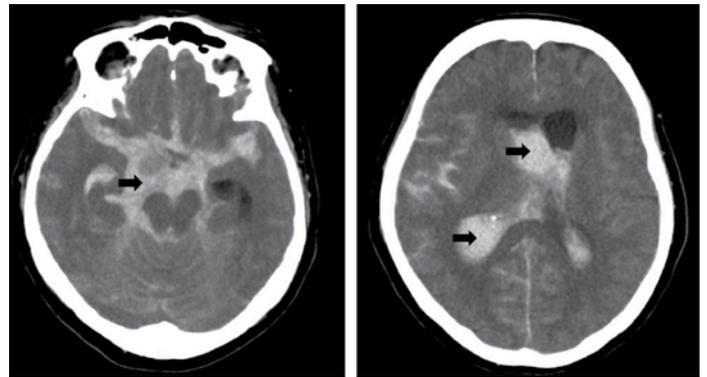
### References

1. Sokhi J, Kinnear J. Atrial fibrillation: peri-operative management in non-cardiac surgery. *Anaesthesia Tutorial of the Week* 307 (2014). Available at [http://www.wfsahq.org/components/com\\_virtual\\_library/media/e12b5079331ce4d1108a2e9f38294fd0-48ca68dafce920f3e467801da9d65b06-307-Atrial-Fibrillation--AF-Peri-operative-management-for-non-cardiac-surgery.pdf](http://www.wfsahq.org/components/com_virtual_library/media/e12b5079331ce4d1108a2e9f38294fd0-48ca68dafce920f3e467801da9d65b06-307-Atrial-Fibrillation--AF-Peri-operative-management-for-non-cardiac-surgery.pdf).
2. Hutchins D. Peri-operative cardiac arrhythmias. Part 1: Supraventricular arrhythmias. *Anaesthesia Tutorial of the Week* 279 (2013). Available at [http://www.wfsahq.org/components/com\\_virtual\\_library/media/35c9e1ade52a269fd099ffe2b2ab694c-d4034c3630abc0a887ecd5ff9087854c-279-Peri-operative-Cardiac-Arrhythmias---Part-1.pdf](http://www.wfsahq.org/components/com_virtual_library/media/35c9e1ade52a269fd099ffe2b2ab694c-d4034c3630abc0a887ecd5ff9087854c-279-Peri-operative-Cardiac-Arrhythmias---Part-1.pdf).
3. National Institute for Health and Care Excellence. *Atrial Fibrillation: Management*. Clinical guideline 180. 2014. <https://www.nice.org.uk/guidance/cg180/resources/atrial-fibrillation-management-35109805981381>.
4. Resuscitation Council UK. *Peri-arrest Arrhythmias: Tachycardia* (2015). Available at <https://www.resus.org.uk/resuscitation-guidelines/peri-arrest-arrhythmias/#tachycardia>.

**Table 2. CHA2DS2-VASc score**

Congestive heart failure	1
Hypertension	1
Age (years)	
< 65	0
65–74	1
≥ 75	2
Diabetes	1
Stroke/transient ischaemic attack/ thromboembolism	2
Vascular disease	1
Sex	
Male	0
Female	1
Score of 0 is low risk	No anticoagulation
Score of 1 is low–moderate risk	Consider anticoagulation
Score ≥ 2 is moderate–high risk	Advise anticoagulation

### Case 3



**Figure 6.** Head of patient 3. The left-hand image shows a large amount of blood around the base of the brain (arrow). The right-hand image is several slices further up, showing blood within the lateral ventricles (arrow). This is a massive subarachnoid haemorrhage (SAH), with intraventricular haemorrhage.

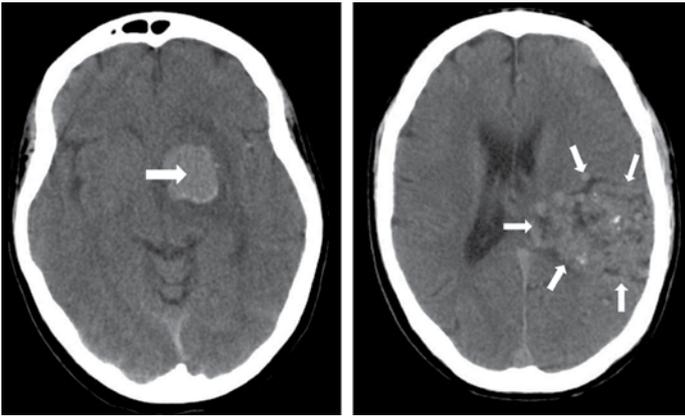
### Epidemiology of SAH

The incidence of SAH is 6–9 per 100 000 per year and it accounts for 6% of first strokes. Approximately 85% of patients with non-traumatic SAH bleed from intracranial arterial aneurysms, with the remainder due to arteriovenous malformations (AVMs). The mean age at presentation is 50 years, and women have a higher risk than men. Traumatic brain injury is another more common cause of blood in the subarachnoid space.

The hallmark of an SAH in an alert patient is the complaint that this is ‘the worst headache of my life’, which is reported by approximately 80% of patients who can give a history. It is typically a ‘thunder-clap’ headache, describing a sudden onset, severe, ‘pressure’-type headache starting in the occipital region. Other symptoms and signs include vomiting, confusion and decreased level of consciousness or seizures.

On examination patients may have normal neurology or have signs of meningism such as neck stiffness and photophobia. They may also have signs of increased intracranial pressure (ICP) or focal neurology such as third cranial nerve palsy (indicating a possible posterior communicating artery aneurysm).

Investigations include urgent CT of the head (Figure 6) to make the diagnosis and CT angiography to investigate the underlying cause (Figure 7). CT has a sensitivity of greater than 90% for SAH within the first 48 hours. If it is negative and there are no contraindications, then a lumbar puncture is performed looking for xanthochromia (degraded haem molecules). Importantly, lumbar puncture should not be done within 12 hours of symptom onset as it takes this length of time for the red blood cells to break down in the cerebrospinal fluid.



**Figure 7.** (A) A large left cerebral artery aneurysm (arrow). (B) A giant left cerebral artery AVM (arrows).

### Grading of SAH

SAH is classified into five grades (Table 3).<sup>3</sup>

Patients with SAH grades I and II will initially require diagnosis and supportive therapy, including close monitoring of vital signs and neurological status, whilst seeking advice from a neurosurgical team.

SAH grade III, IV or V results in altered neurology and so necessitates more extensive initial therapy.

### Management of confirmed subarachnoid haemorrhage

The initial management of SAH is aimed at patient stabilisation with a focus on maintaining cerebral perfusion and oxygenation.

#### Initial management

- In patients with a decreased level of consciousness or who are unable to maintain their own airway or show signs of raised ICP, elective intubation should be performed. This will help protect the patient from aspiration caused by reduced airway reflexes. Ideally, rapid sequence induction should be performed, avoiding hypotension and employing agents to blunt any increase in ICP.
- The patient should then be ventilated with the aim of achieving normocapnia.

**Table 3.** Features of the five grades of SAH

Grade	Clinical features
I	Mild headache with or without meningeal irritation
II	Severe headache and a non-focal examination, with or without mydriasis
III	Mild alteration in neurological examination, including mental status
IV	Obviously depressed level of consciousness or focal deficit
V	Patient either posturing or comatose

- IV access including central and arterial line, for invasive blood pressure monitoring, should be established.

#### Monitoring

- Intensive continuous observation is required until several days after occlusion of the aneurysm.
- Implement continuous ECG monitoring.
- Measure Glasgow Coma Scale (GCS) score, focal deficits, blood pressure and temperature at least every hour

#### Blood pressure

- Stop any of the patient's own anti-hypertensive medication.
- Treat hypotension with isotonic IV fluids aiming for euvolaemia and maintenance of sufficient mean arterial pressure to achieve cerebral perfusion pressure of > 70 mmHg. Aggressive treatment of hypotension is crucial in the management of SAH to maintain cerebral perfusion pressure.
- The use of vasopressors may be indicated to keep the systolic blood pressure > 120 mmHg, which would avoid the central nervous system damage as a result of the reactive vasospasm seen in SAH.
- Do not treat hypertension unless it is extreme; limits for extreme blood pressures should be set on an individual basis, taking into account age of the patient, pre-SAH blood pressures and cardiac history.
- Systolic blood pressure should be kept below 180 mmHg only until coiling or clipping of ruptured aneurysm, to reduce the risk of rebleeding.
- Start nimodipine 60 mg 4 hourly orally or 1 mg h<sup>-1</sup> IV infusion to reduce vasospasm. Nimodipine substantially decreases the risk of cerebral infarction and poor outcome after SAH.
- Insert an indwelling urethral catheter and monitor fluid balance.

#### Seizures

- Treat seizures with benzodiazepines if required and load with phenytoin. Use only if required as the prophylactic use of anticonvulsants has been associated with a worse outcome.

#### Temperature

- Increased temperature should be treated both medically and physically. There is currently no evidence for therapeutic hypothermia in SAH patients.

#### Blood sugar management

- Routinely monitor serum blood glucose as one-third of SAH patients develop hyperglycaemia. A level over 10 mmol L<sup>-1</sup> should be treated.

#### Pain/anxiety

- Treat pain and anxiety. Start with regular paracetamol and for severe pain use codeine or tramadol.

#### Intracranial pressure

- Avoid situations that will increase intracranial pressure.
- Keep the patient in bed with head elevated to 30°.

- Use laxatives and antiemetics as required.
  - If the patient is intubated, ensure that method of securing the endotracheal tube does not obstruct venous drainage (endotracheal tubes are normally taped rather than tied).
  - If possible, avoid positive end-expiratory pressure (PEEP) to minimise intrathoracic pressures.
  - Hypertonic solutions and mannitol will increase plasma osmolality and so decrease the water content of the brain tissue (the blood–brain barrier will act as a semipermeable membrane), thereby decreasing ICP.

#### Thromboprophylaxis

- Patients with SAH may be given thromboprophylaxis with pneumatic devices and/or compression stockings before occlusion of the aneurysm.
- If deep vein thrombosis prevention is indicated, low-molecular-weight heparin should be applied not earlier than 12 hours after surgical occlusion of the aneurysm and immediately after coiling.

#### Steroids

- There is no proof that steroids are effective in the management of SAH.

#### Definitive treatment

- This patient should be discussed with the tertiary neurosurgical referral centre regarding transfer for monitoring of ICP and/or insertion of external ventricular drain.
- Definitive management, in order to prevent re-bleeding, is by radiological coil insertion (Figure 8) or clipping of the underlying pathology at craniotomy.

#### Ongoing management

- The patient should be cared for in a high-dependency environment with ongoing monitoring for the common sequelae of SAH (see Figure 9). These include:
  - hydrocephalus
  - vasospasm
  - ischaemia
  - infarction
  - rebleeding.

#### Further reading

- van Gijn J, Rinkel GJ. Subarachnoid haemorrhage: diagnosis, causes and management. *Brain* 2001; **124**: 249–78.

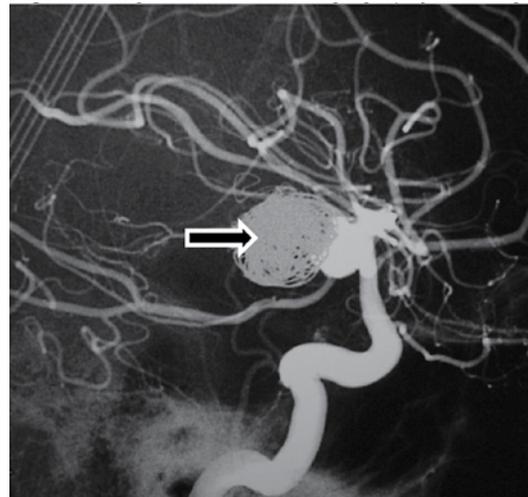


Figure 8. Digital subtraction angiography of a large intracerebral AVM containing coils (arrow).

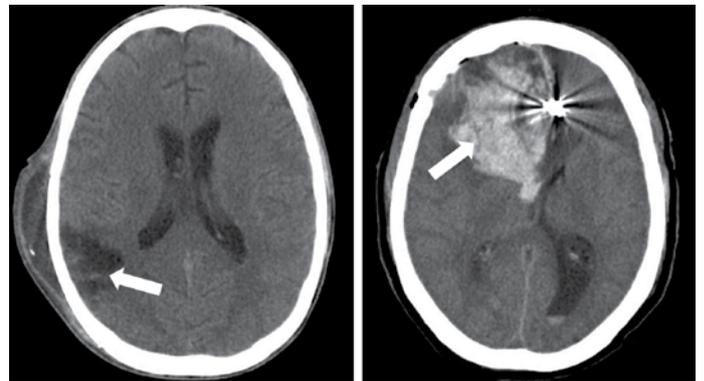


Figure 9. Complications of clipping of aneurysms following SAH. (A) Right occipito-parietal infarction. (B) Bleeding after aneurysm clipping.

#### References

1. Connolly ES Jr, et al. Guidelines for the management of aneurysmal subarachnoid haemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke* 2012; **43**: 1711–37.
2. European Stroke Organisation. Guidelines for the management of intracranial aneurysms and subarachnoid haemorrhage. *Cerebrovasc Dis* 2013; **35**: 93–112.
3. Rosen DS, Macdonald RL. Subarachnoid haemorrhage grading scales: a systematic review. *Neurocrit Care* 2005; **2**: 110.