

## Cerebral challenge

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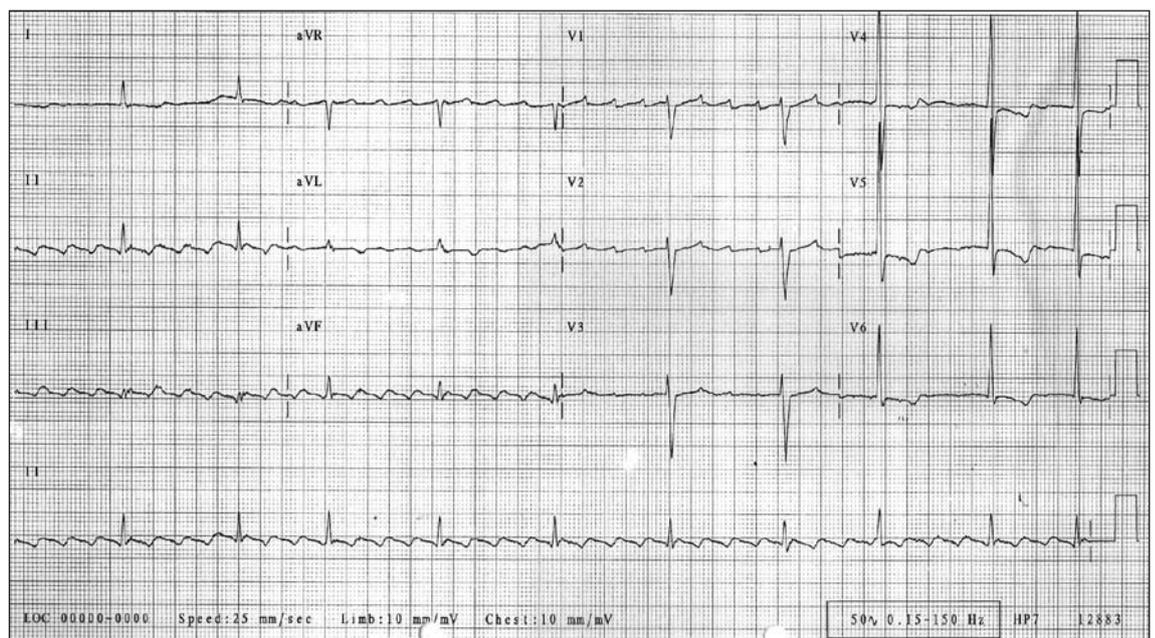
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### Case 1

A 61-year-old woman is due to undergo laparotomy for small bowel obstruction. She describes occasional palpitations over the last

six months and takes digoxin 62.5mcg daily. A 12-lead ECG is performed during an episode of palpitations (Figure 1).

Figure 1.



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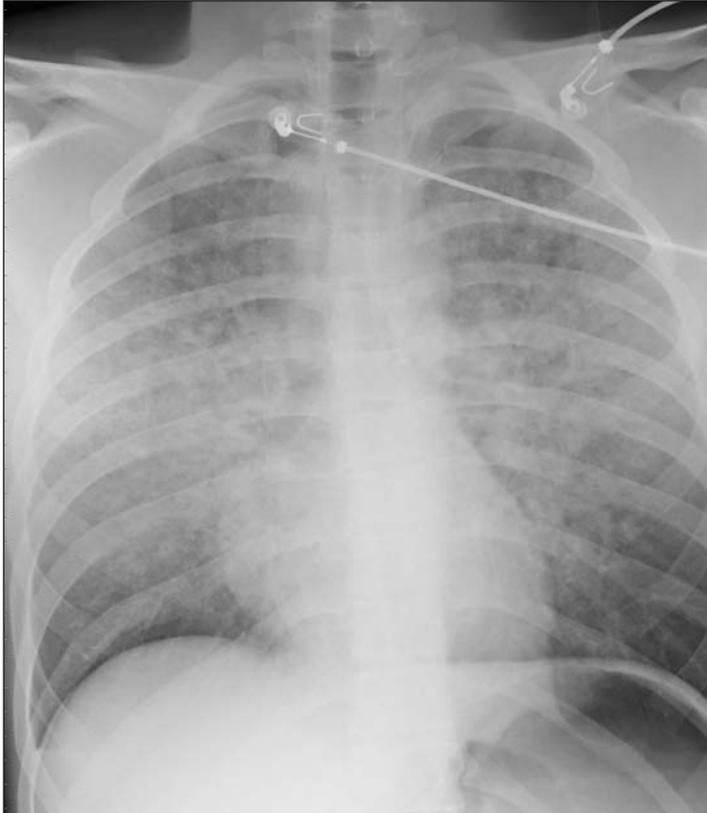
- What abnormalities does this ECG show?
- What are your options regarding her anaesthetic management?

### Case 2

You take over a case from a colleague - an 18-year-old who is undergoing a manipulation under anaesthesia for a forearm fracture. On removal of the laryngeal mask the patient develops fairly severe laryngospasm as she emerges from anaesthesia. Despite applying continuous positive airway pressure (CPAP) using an anaesthetic mask and breathing circuit, and administering 100% oxygen, her oxygen saturations dip down to 84%. The laryngospasm

settles when you administer propofol 30mg IV and you take her to recovery. 15 minutes later the recovery nurse asks you to review your patient, who is now short of breath, with a respiratory rate 32 breaths.min<sup>-1</sup> and SaO<sub>2</sub> 88% on air, rising to 90% receiving oxygen at 6l.min<sup>-1</sup> via a Hudson mask. On auscultation of her chest she has bibasal crackles and you request an urgent chest X-ray (CXR).

**Figure 2.**



- What does the CXR show?
- What is the most likely cause in this case?
- Name other possible causes of these CXR findings.
- Describe how you would manage this patient.

### Case 3

A 36-year-old woman, gravida 7 para 6, has been brought to labour ward at 36 weeks gestation, with a history of headache and neck pain. Shortly after arrival she vomited and lost consciousness. When last seen in ante-natal clinic two months previously her BP was 150/100 and methyldopa was started.

## DISCUSSION

### Case 1

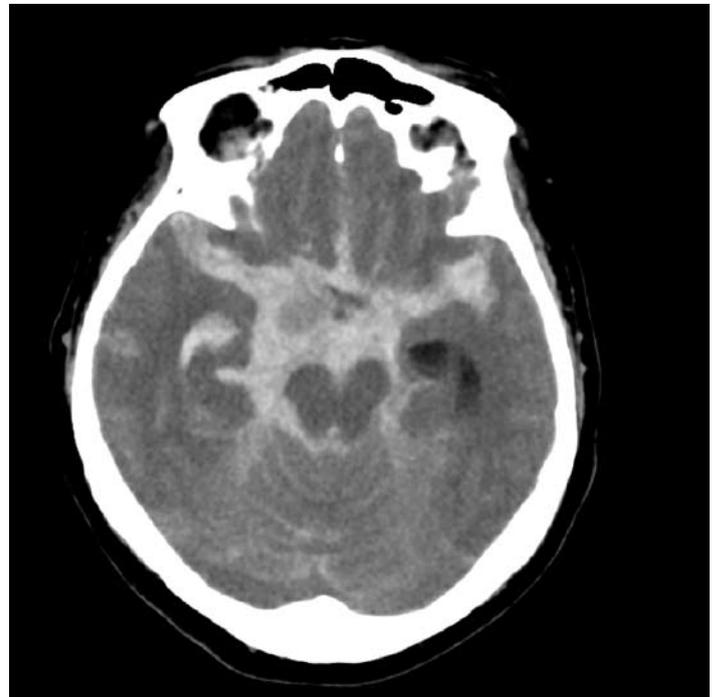
Analysis of the 12-lead ECG should be done methodically and in the same manner from patient to patient, to prevent missing abnormalities. The ECG shows atrial flutter with predominantly 4:1 block (see Figure 4). There is also ST depression in the lateral chest leads (V4-V6), which has the characteristic 'reverse tick' appearance seen with digoxin use.

On examination she is apyrexial, her neck is stiff, Glasgow Coma Score (GCS) is 11 (Eyes 3, Motor 5, Vocal 3), pupils are 4mm and equal and reactive. Her reflexes are bilaterally brisk and her blood pressure is 197/119mmHg, heart rate 96min<sup>-1</sup> and regular, and respiratory rate 20min<sup>-1</sup>. Capillary venous glucose is 8.3mmol.l<sup>-1</sup>. She is not in labour and the foetal heart rate is 136min<sup>-1</sup>.

- What is your differential diagnosis?

The patient has a grand mal seizure, after which her GCS deteriorates to 5 (E1, V1, M3), BP increases to 238/139 and she requires intubation and ventilation. A CT head is performed (Figure 3).

**Figure 3.** CT head without contrast



- What does the CT show?
- How would you management this patient?

### Atrial Flutter – associated conditions

- Ischaemic heart disease
- Cardiomyopathy
- Valvular disease
- Post-cardiac surgery
- Rheumatic heart disease

Atrial flutter may be idiopathic or associated with cardiac abnormalities:

Management involves consideration of three factors: ventricular rate, rhythm and anticoagulation.

#### Ventricular rate

- Ventricular rate in atrial flutter depends on the degree of AV blockade.
- Patients with low degrees of blockade will have high ventricular rates with poor ventricular filling, poor coronary perfusion and reduced cardiac output.
- Ventricular rate can be controlled with digoxin, beta blockers or calcium channel antagonists.

#### Rhythm

- Reversion to sinus rhythm may be spontaneous or chemically or electrically mediated.
- It is not clear whether rhythm control decreases mortality. If tolerable to the patient, rate control,

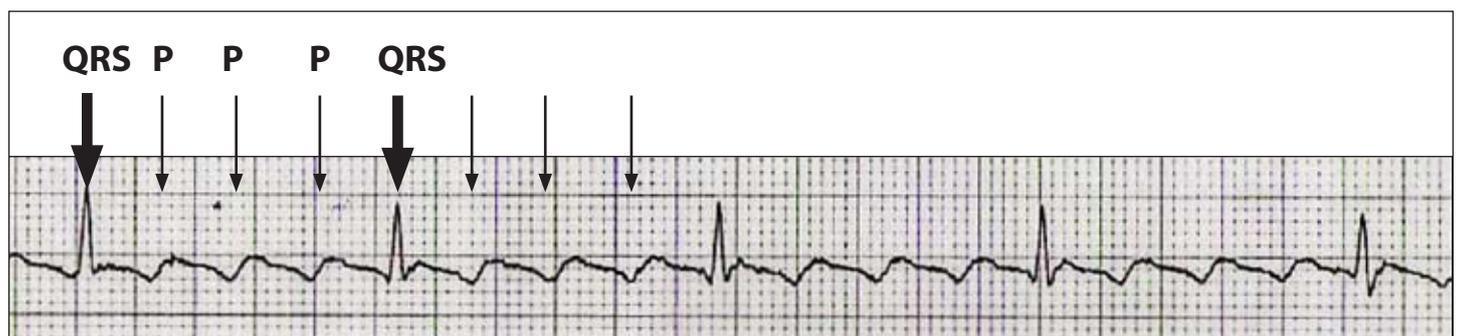
in combination with anticoagulation, is a satisfactory alternative to cardioversion.

- Electrical cardioversion involves the inherent risks of sedation or general anaesthesia, but shock energies are lower than those used in atrial fibrillation – typically an initial synchronised shock of 50J is used.
- Chemical cardioversion is most commonly achieved with flecainide or amiodarone.

#### Anticoagulation

- Thromboembolism is a risk although less so than in atrial fibrillation.
- Intra-cardiac clot formation may occur after 48 hours of onset of the arrhythmia - cardioversion after this period carries a risk of migration of thromboembolism to distant sites.
- Patients with paroxysmal atrial flutter or prolonged atrial flutter should be anticoagulated with warfarin, aiming to achieve an INR of 2-3.

This patient requires an urgent laparotomy. Her rate is well controlled by the digoxin she is taking. Further examination reveals that she is cardiovascularly stable with a good blood pressure. It is reasonable to proceed with a cautious general anaesthetic, knowing that you can perform a synchronised DC cardioversion if she deteriorates during the procedure. Postoperatively it would be advisable to ask a cardiologist to recommend an alternative anti-arrhythmic drug to treat her paroxysmal flutter. If this is ineffective, she should consider long-term anticoagulation.



**Figure 4.** The lowest strip on the ECG (the 'rhythm strip') shows continuous monitoring of lead II for the duration of the ECG. This is best used to calculate the rate and rhythm. P waves occur at a rate of 300 beats.min<sup>-1</sup>, with a characteristic 'saw-tooth' appearance. The AV node fails to conduct all the P waves because of its refractory period. The QRS complexes, representing ventricular depolarisation, appear every 4th P wave. In atrial flutter, the ratio of P waves to QRS complexes is most commonly 2, 3 or 4 to 1. A supraventricular tachycardia with a ventricular rate of 150 beats.min<sup>-1</sup> is commonly atrial flutter with 2:1 block

## Case 2

The chest X-ray shows bilateral fine ‘alveolar’ shadowing, predominantly in the mid and upper zones, that is characteristic of pulmonary oedema. In this previously fit patient, with the history of laryngospasm causing upper airway obstruction during emergence, the most likely cause is **negative pressure pulmonary oedema**. Ventilatory effort against a closed glottis generates high negative intrathoracic pressures and consequent pulmonary oedema. Patients who are septic and have sub-clinical acute lung injury may be more prone to developing this complication of anaesthesia.

Pulmonary oedema can be either cardiogenic or non-

cardiogenic in aetiology.

An ‘ABC’ approach should be adopted, with administration of high flow oxygen (15 l.min<sup>-1</sup>) via a mask with a reservoir bag. Treatment is supportive - this normally fit patient is likely to recover within a few hours. If life-threatening hypoxia or respiratory distress occurs, CPAP via a tight-fitting mask may help. Intubation and ventilation may be necessary in severe cases, if CPAP fails to help, or if the patient has significant underlying cardiopulmonary disease. Although a trial of a diuretic such as frusemide seems logical, there is no evidence that a diuresis alters the course of the illness.

**Table 1.** Causes of pulmonary oedema

Cardiogenic	Non-cardiogenic
<ul style="list-style-type: none"><li>• Left ventricular failure due to ischaemic heart disease, valvular disease or cardiomyopathy</li></ul>	<ul style="list-style-type: none"><li>• Adult respiratory distress syndrome</li><li>• Neurogenic pulmonary oedema (following a major neurological insult such as subarachnoid haemorrhage)</li><li>• Transfusion-related acute lung injury (TRALI)</li><li>• Negative pressure pulmonary oedema</li></ul>

## Case 3

The main differential diagnosis based on the patient’s presentation and subsequent deterioration can be divided into diagnoses related to or unrelated to her pregnancy. The latter will depend on the endemic diseases of the region.

Related to pregnancy	Unrelated to pregnancy
<ul style="list-style-type: none"><li>• Preeclampsia / eclampsia</li></ul>	<ul style="list-style-type: none"><li>• Subarachnoid haemorrhage</li><li>• Cerebral malaria</li><li>• Meningitis</li></ul>

If CT scanning is unavailable, it can be very difficult to distinguish between subarachnoid haemorrhage (SAH) and preeclampsia/eclampsia. Three sets of blood films should be viewed to exclude malaria. The presence of proteinuria, hypertension during pregnancy and oedema would suggest preeclampsia/eclampsia. Treatment with magnesium and urgent caesarean section should be considered, since this is indicated as the primary treatment for this condition. In the absence of clinical features suggesting other diagnoses, and if there are focal neurological signs, SAH must be considered. If the mother’s condition is felt to unrecoverable, again urgent caesarean section should be considered with a view to delivering the baby. This decision should be taken jointly between the

obstetrician and other medical staff, after discussion with and agreement from the patient’s family.

The CT head scan shows a cross section view with the patient supine. The image is best interpreted by imagining you are looking up towards the patient’s head from their feet. The left side of the patient is on the right side of this image. Tissues that absorb X-rays well appear white (e.g. bone, blood), lower density tissue appears darker (e.g. air, CSF) and brain tissue looks grey.

This CT head demonstrates new blood (white) within the right lateral ventricle (A) and the basal cisterns (B). The diagnosis is subarachnoid haemorrhage, with spread of blood into the ventricular system of the brain. It is unusual to see the inferior horn of the lateral ventricles at this level (C), suggesting that the ventricular system is enlarged and that hydrocephalus is present.

This woman’s prognosis is poor and by both the ‘Hunt and Hess’ and World Federation of Neurosurgeons scoring, she is has the worst grading (5 out of 5.)

The incidence of SAH in pregnancy is 10-20 per 100,000 pregnancies. Clinically it is difficult to distinguish SAH from pre-eclampsia (or cerebral malaria), however the presence of focal neurological abnormalities strongly suggests SAH.



**General management**

Management should adopt an airway, breathing, circulation (‘ABC’) approach with a low threshold for intubation and ventilation. Aim to maintain an adequate cerebral perfusion pressure (CPP).

$$CPP = (ABP - CVP) - ICP$$

This can be calculated from arterial blood pressure (ABP) and intracranial pressure (ICP) where this is known. Unless ICP monitoring is available, assume ICP is 20mmHg.

**Table 1. Aetiology of subarachnoid haemorrhage (SAH)**

Ruptured arterial (berry) aneurysm(s)	<ul style="list-style-type: none"> <li>• 80% aneurysms are in the anterior circulation</li> <li>• 15% aneurysms are bilateral</li> </ul>
Ruptured arteriovenous malformation	
Trauma	
Hypertension	
Cocaine or amphetamine abuse	

**Specific management**

Secondary brain injury is minimised by preventing and aggressively treating the major complications of SAH. These are:

• **Hydrocephalus**

This patient has evidence of hydrocephalus on her CT scan and it is possible that this is contributing to her low conscious level. Where facilities exist she should have an external ventricular drain (EVD) inserted.

• **Rebleed**

The incidence of a re-bleed is 4% in first 24 hours, then 1.5% per day. The mortality in those who re-bleed is 80%.

Where available, treatment is usually considered for all patients who are grade 3 or better. This can be either operative (a titanium clip is put across the neck of the aneurysm) or radiological (a coil or coils are inserted via an endovascular route).

• **Cerebral vasospasm**

Vasospasm results from blood in the subarachnoid space, maybe oxyhaemoglobin, free radicals and lipid peroxidases and causes ischaemia and infarction of brain tissue. The risk of vasospasm is higher if the patient is female, a smoker, hypertensive and there is more blood on CT. Peak incidence is at 7-10 days and it is rare before 3 days and after 21 days post SAH.

Treatments include:

- Nimodipine – a calcium antagonist given enterally (60mg orally or via naso-gastric tube).
- Triple-H therapy (hypervolaemia, hypertension, haemodilution).
- Angioplasty (where available).

Other complications of SAH include seizures, myocardial dysfunction, neurogenic pulmonary oedema and cerebral salt wasting.

SAH in pregnant women carries a maternal mortality between 35-83% with SAH accounting for 5% of maternal deaths in the UK. If the patient fails to improve after EVD insertion to treat hydrocephalus, her prognosis is extremely poor. Consideration should be given to proceed to urgent caesarean section to deliver the baby.