Update in
Anaesthesia

Cerebral challenge

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CASE 1

A 73-year-old man presents to the emergency department feeling generally unwell. He is short of breath and dizzy when he tries to stand. He denies chest pain, but does have a history of ischaemic heart disease and hypertension. On examination he is clammy and looks grey, and he has a raised jugular venous pressure with visible 'cannon' waves. On auscultation there are bibasal crackles. His initial observations are blood pressure 75/34 mmHg heart rate 38 per minute, respiratory rate 28 per minute and oxygen saturation 91% in room air. Figure 1 shows his ECG.

- What does this ECG show?
- What are the causes of the ECG findings?
- What is the treatment for this condition?

CASE 2

A 28-year-old woman is brought in to the emergency department having dived into the shallow end of a swimming pool whilst under the influence of alcohol. Friends immediately recovered her from the swimming pool, with no requirement for cardiopulmonary resuscitation at the scene. She has complained of neck pain since the injury, and paraesthesiae in her legs and fingers, but the paramedics were not able to immobilise her neck at the scene as she was agitated and non-compliant.

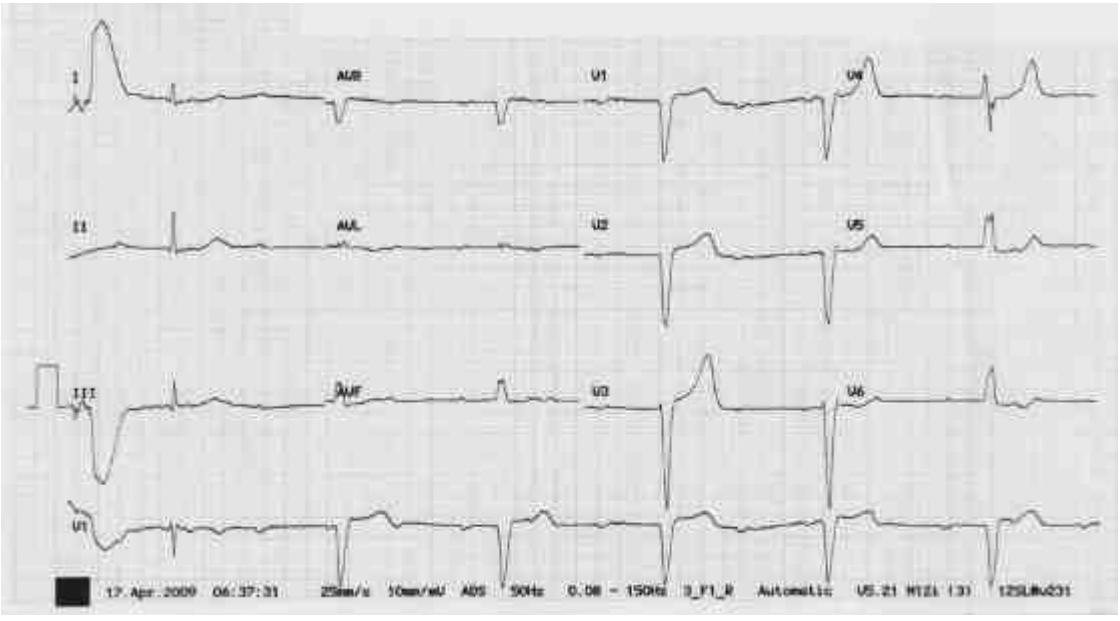

Figure 1. *ECG of patient 1*

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- What are the key priorities for management of this patient on arrival in the emergency department?
- What does this cervical spine X-ray (Figure 2) demonstrate?
- How should this patient be managed?

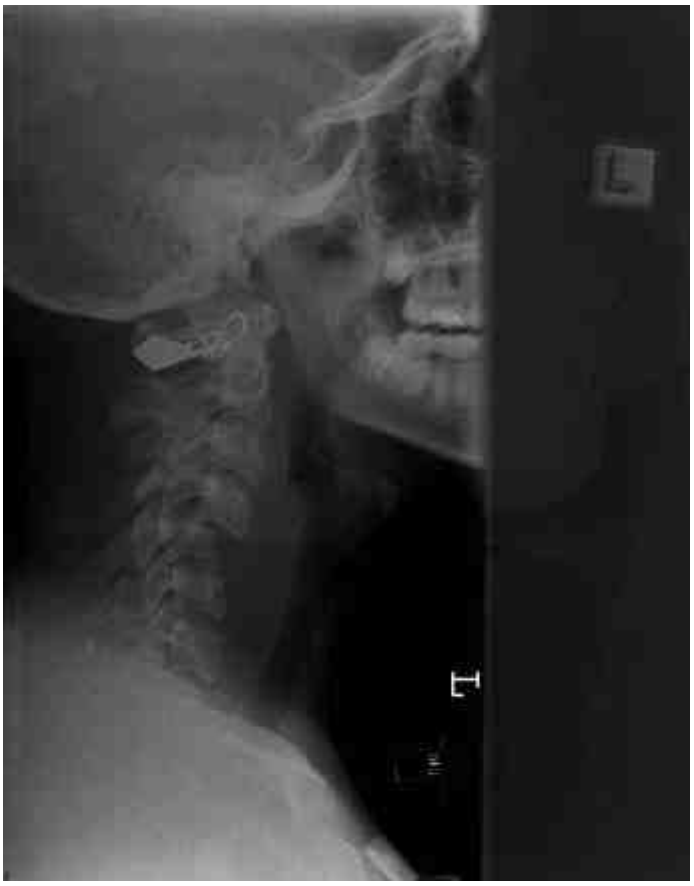

Figure 2. *Lateral cervical spine X-ray of patient 2*

CASE 3

You are asked to review a 33-year-old man in the high-dependency unit. He is a known asthmatic, admitted via the emergency department with an acute severe asthma attack and has been receiving appropriate medical therapy.

The nurse looking after him is concerned as he is complaining of increased chest pain and shortness of breath. You immediately attend to assess him. On examination you find him in respiratory distress, unable to speak a full sentence. He is swollen around the face and neck, tachypnoeic and tachycardic. There is a crackling under your fingertips on palpation of the swollen tissues in his neck, the trachea is central and there is decreased chest expansion on the left. His chest is hyper-resonant to percussion bilaterally and there is widespread expiratory wheeze on auscultation except for in the left upper zone, where you cannot hear any breath sounds. His oxygen saturations are 94% on supplemental oxygen. You order a chest X-ray (Figure 3).

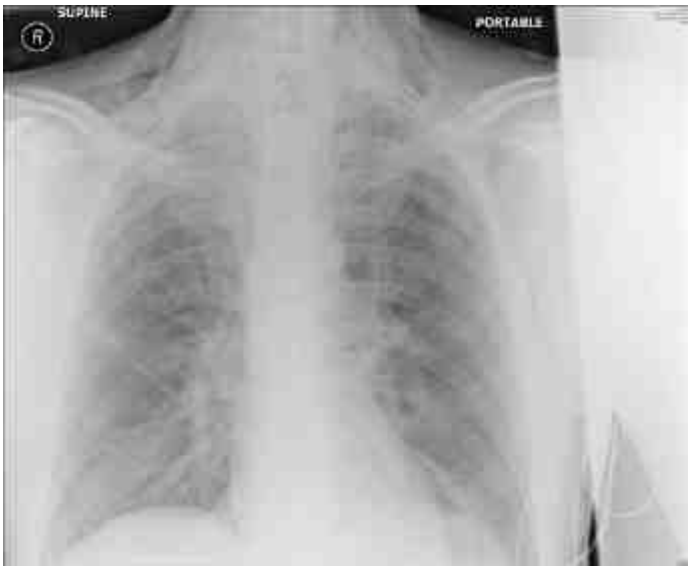

Figure 3. *Chest X-ray of patient 3*

- What does the chest X-ray show?
- What is the diagnosis?
- How would you manage this patient?

DISCUSSION

Case 1

The ECG shows P-waves (atrial contraction) and QRS complexes (ventricular contraction) that have no relationship to each other – in normal cardiac conduction, each P-wave should be followed by a QRS complex. This is complete atrioventricular (AV) dissociation, also called third-degree heart block (or complete heart block). The QRS complexes are widened (142 ms, normal < 120 ms) and the rate is slow – this is termed a 'ventricular escape rhythm', here with a rate of 38 beats per minute.

Complete heart block occurs when there is no conduction of electrical activity from the atria to the ventricles. This block in electrical conductance can occur at the AV node, the bundle of His or the Purkinje system. Both the rate of the escape rhythm and the width of the QRS complexes are determined by the site of origin of the escape beats. If the pacemaker cells of the heart causing the escape rhythm are above the bundle of His, a narrow QRS complex will be produced; pacemaker cells in the bundle of His or Purkinje system will lead to a broad QRS complex.

Table 1. *Causes of third-degree heart block*

Congenital	Autoimmune (maternal SLE), structural heart disease (transposition of the great vessels)
Idiopathic fibrosis of the His Purkinje system	Lev's disease, Lenegre's disease
Ischaemic heart disease	Acute myocardial infarction (particularly of the right coronary circulation), ischaemic cardiomyopathy
Non-ischaemic heart disease	Calcific aortic stenosis, idiopathic dilated cardiomyopathy
Cardiac surgery	Valve replacement, coronary artery bypass graft, ventricular septal defect repair
Iatrogenic	Radiofrequency AV node ablation, pacemaker implantation
Drug induced	Beta blockers, amiodarone, digoxin, calcium channel antagonists (diltiazem and verapamil); tricyclic antidepressants, clonidine
Infective	Endocarditis, Lyme's disease, rheumatic fever, Chagas' disease, diphtheria
Connective tissue	SLE, rheumatoid arthritis
Neuromuscular	Duchenne muscular dystrophy

AV dissociation is not only seen in complete heart block; it can occur when the atrial rate is slower than the ventricular rate, such as in ventricular tachycardia.

The most likely cause in this man is ischaemic heart disease (IHD). Both IHD and hypertension can lead to left ventricular hypertrophy and myocardial damage, eventually causing third-degree heart block. Other causes include infectious diseases, for example diphtheria and rheumatic fever (group A *Streptococcus*) and congenital and autoimmune diseases, such as systemic lupus erythematosus (see Table 1).

Heart block can occur after cardiac surgery. Cardiac medications aimed at treating tachyarrhythmias, such as amiodarone, beta-blockers, digoxin and calcium channel antagonists, can also predispose to complete heart block, as can clonidine and tricyclic antidepressants.

Complete heart block can present with a broad range of symptoms. These can range from fatigue, confusion, dizziness and blackouts, known as Stokes–Adams attacks, to chest pain, severe dyspnoea, cardiogenic shock and cardiac arrest.

Treatment of third-degree heart block

Initial management follows an ABCDE approach; with application of high-flow oxygen, intravenous (IV) access and initial attempts to increase heart rate by administration of atropine 500 µg, up to 3 mg in total. However, this may only work temporarily, and will only work if the block is at the AV node. It can also be dangerous to administer to a patient with an on-going myocardial infarction.

Some patients suffer sudden asystolic cardiac arrest and so this patient should receive continuous cardiac monitoring and, if available, the application of transcutaneous pacing pads. This man requires cardiac pacing, and a cardiologist should be sought as a matter of urgency to insert a temporary pacing wire. Transcutaneous pacing is uncomfortable for the patient, and so, if it is instigated due to haemodynamic compromise, a transvenous pacing wire should then be inserted as soon as possible. Ultimately, a permanent pacemaker may be required, if no reversible cause is identified.

Medications should be reviewed, and any potential causative agents stopped immediately. If overdose of medication is suspected, these should be treated accordingly. It is important to identify if the patient is suffering from an acute myocardial infarction at the time of presentation, as a patient with an evolving infarct is at greater risk of cardiac arrest. Reversal of acute ischaemia may reverse the heart block.

Case 2

What are the key priorities for management of this patient on arrival?

As with all trauma cases, the management of this patient begins with a 'C-ABCDE' assessment.

The C prior to the usual ABCDE encourages immediate assessment and management of:

- **C**atastrophic haemorrhage
- **A**irway with inline **C**-spine immobilisation
- **B**reathing
- **C**irculation
- **D**isability (neurological assessment)
- **E**xposure/environment.

C

In this case, there is no obvious catastrophic haemorrhage, his only complaints being pain in his neck and paraesthesiae.

A with cervical spine immobilisation

This woman is likely to have sustained a cervical spine injury – the mechanism of injury, diving into a shallow pool has caused an axial load to her head. This is a factor associated with high risk of cervical spine injury according to the Canadian C-spine risk factors. Other risk factors are listed in Table 2.

She has been drinking and, although she is able to communicate and her airway is patent at the moment, her agitation *may* be due to an

intracranial injury. She tolerates manual in-line stabilisation of her neck, but not a hard collar. In these situations, it is best to accept what the patient will allow, as opposed to trying to enforce a hard collar, potentially leading to further patient agitation and movement creating further injuries. If definitive airway control is indicated, a rapid sequence induction is recommended, as these patients are likely to have gastric paresis and a full stomach.

B

The location of a spinal cord injury will determine its impact on breathing. As well as excluding life-threatening complications, such as pneumothorax or haemothorax, attention needs to be paid to the ability to deep breathe and cough. Repeat assessments need to be made, as the neurological level of a spinal cord injury can ascend in the hours following injury. Thoracic spinal injuries will affect the innervation to the intercostal muscles, whereas spinal injuries to C3–C5 will affect the phrenic nerve and diaphragmatic innervation. Respiratory rate and saturations need to be recorded; any oxygen requirement should raise concerns. You notice that this woman has 'see-saw' breathing – when she breathes in her abdomen bulges and her chest is drawn in. This is also called 'paradoxical' breathing; the movements of the chest and abdomen are opposite to what we would usually see in spontaneous breathing. This occurs when a high thoracic or cervical cord injury has paralysed the intercostal muscles and the patient is reliant solely on diaphragmatic breathing.

Table 2. *Canadian C-spine risk factors*

High risk factors
Age > 65 years
Paraesthesiae in upper/lower limbs
Dangerous mechanism of injury
Fall from height > 1 m or five steps
Axial load to head (diving)
High speed motor vehicle collision
Ejection from/rollover vehicle accident
Bicycle collision
Horse-riding accident
Low risk factors
Involved in minor rear-end collision
Not comfortable in sitting position
Not ambulatory since time of accident
Midline cervical spine tenderness
Delayed onset neck pain
and
Unable to actively rotate neck to 45° to left and right

C

Secure IV access, and record heart rate and blood pressure regularly. In any trauma patient, hypotension needs to be investigated thoroughly, but patients with a spinal cord injury may present with bradycardia and hypotension but be warm and vasodilated due to interruption of the sympathetic nervous system pathways. This is neurogenic shock. The hypotension can be resistant to fluid resuscitation, and vasopressor support may be required, in order to prevent secondary spinal cord ischaemic injury.

D

Thorough and systematic repeated full neurological assessments need to be accurately documented. The American Spinal Injuries Association (ASIA) has produced sheets (ASIA charts) to accurately and repeatedly document neurological findings in patients with spinal cord injuries. This allows identification of deterioration and improvement in neurological findings. Any symptoms of spinal pain, weakness in the arms or legs, altered sensation or priapism requires that spinal protective measures be taken. If there are any distracting injuries, reduced consciousness or the patient is under the influence of alcohol or drugs, then spinal protective measures should be initiated. A high index of suspicion needs to be maintained for associated head injury, and usual guidelines for urgent computerised tomography (CT) of the head should be followed.

E

The patient should be fully exposed, whilst maintaining dignity and warmth as much as possible to look for further injuries. This often occurs as part of the log roll, and the spine is palpated for tenderness, and a digital rectal examination should be performed to assess anal tone. Monitor temperature and keep the patient warm, providing warm fluids, forced air blankets and warm mattresses if possible.

What does this cervical spine X-ray demonstrate?

This cervical spine X-ray (Figure 4) shows subluxation of C4 on C5, probably representing a fracture dislocation at this level. This is likely to have caused a major cord lesion at this level.

Cervical spine X-ray imaging is usually performed using three views.

Lateral view

- Ensure that the top of T1 vertebral body is included. To fully assess the images, three lines should be traced to assess vertebral alignment (Figure 5). These should be smooth unbroken lines:
  - along the anterior margins of the vertebral bodies
  - along the posterior margins of the vertebral bodies
  - joining the bases of the spinous processes.
- The vertebral body anterior and posterior heights should be equal, and the intervertebral discs should be the same height.
- Prevertebral soft tissues should be a maximum of 7 mm C1–C4 and 22 mm C5–C7. Any increase or swelling should highlight the potential presence of injury.

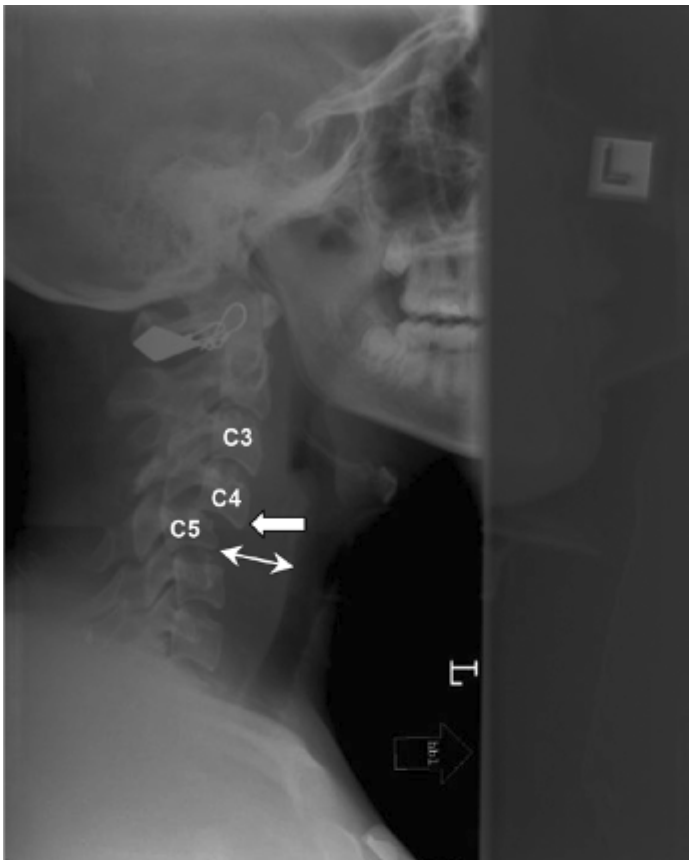

Figure 4. *Lateral cervical spine X-ray of patient 2, showing anterior subluxation (shift) of C4 on C5 (arrow). There is considerable soft tissue swelling anterior to the fracture-dislocation (double arrow). The metallic object at the top of the cervical spine is an overlying ear ring.*

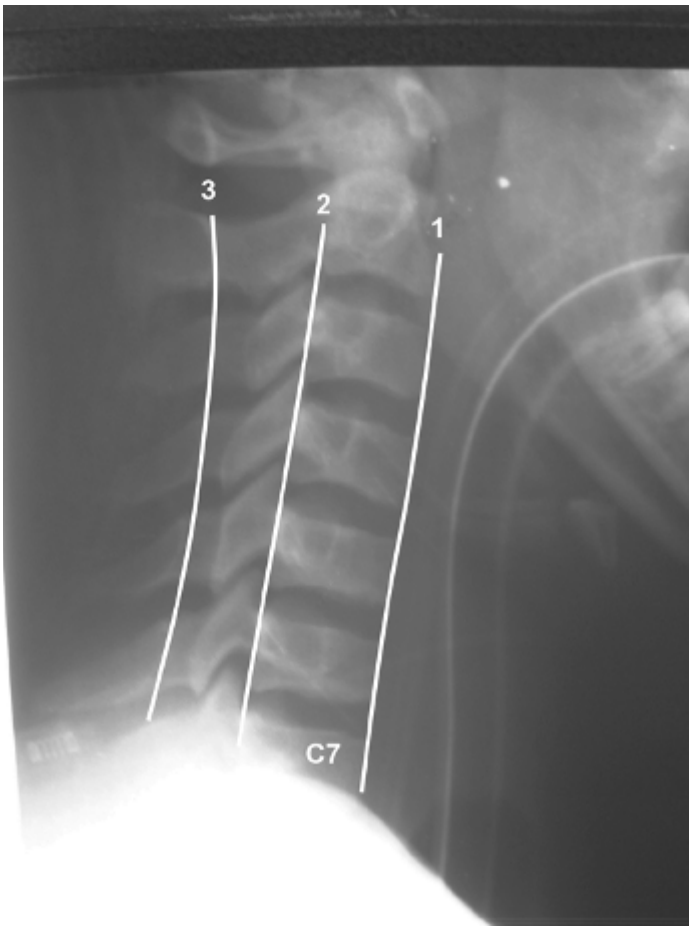

Figure 5. *Normal lateral cervical spine X-ray showing the smooth lines (1) along the anterior margins of the vertebral bodies; (2) along the posterior margins of the vertebral bodies and (3) joining the bases of the spinous processes.* ***Note that the body of C7 is not wholly seen and no part of T1 is seen and so this would not constitute an adequate lateral C-spine view***

Long anteroposterior (AP) view

- In this orientation the spinous processes should lie in a straight line and should be equidistant; an increase in distance of 50% more than the space above or below suggests the possibility of injury.

Open mouth AP view ('peg view')

- Shows C1/C2 articulation.
- The lateral margins of C1 should be in alignment with the lateral margins of C2, with equal distances between the sides of the odontoid peg and lateral mass of C2. These images can be difficult to interpret if there is any neck rotation, which can mimic subluxation.

How should this patient be managed?

Once the primary survey has been carried out and the patient has been stabilised, preparations should be made to transport the patient for CT imaging, in this case of the cervical spine and head. Any Canadian C-spine high risk factor should warrant a CT scan of the cervical spine, as should clinical suspicion but inadequate plain films. Imaging should be reviewed by a consultant radiologist, and abnormal images reviewed by a neurosurgical team for consideration or stabilisation, closed or open reduction or decompression.

Close monitoring of the patient should occur in a high-dependency unit, as there may be ascending cord oedema leading to diaphragmatic paralysis requiring intubation and ventilation. A low threshold for airway support should be maintained, as patients with cervical cord injury will be unable to cough and clear secretions; this inability to clear secretions may warrant intubation. Succinylcholine may be used within the first 72 hour, but not thereafter for 6–9 months. Vasopressors may be required to maintain a high mean arterial pressure to prevent secondary cord ischaemia. Therapies aimed at the prevention of pressure sores, venous thromboembolic disease and peptic ulcers need to be implemented.

Further reading

1. Theron A, Ford P. management of acute cervical spine injuries. *Update Anaesth* 2008; **24,1**: 30–4.

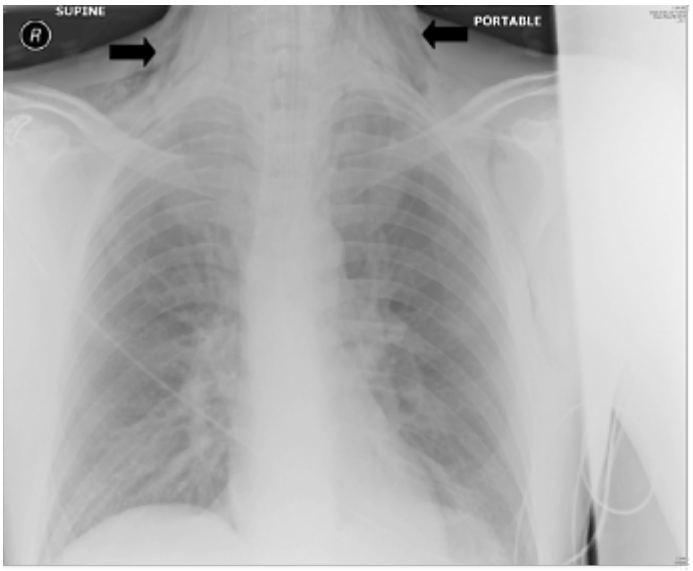

Figure 6. *Chest X-ray of patient 3 shows surgical emphysema at the root of the neck bilaterally (black arrows)*

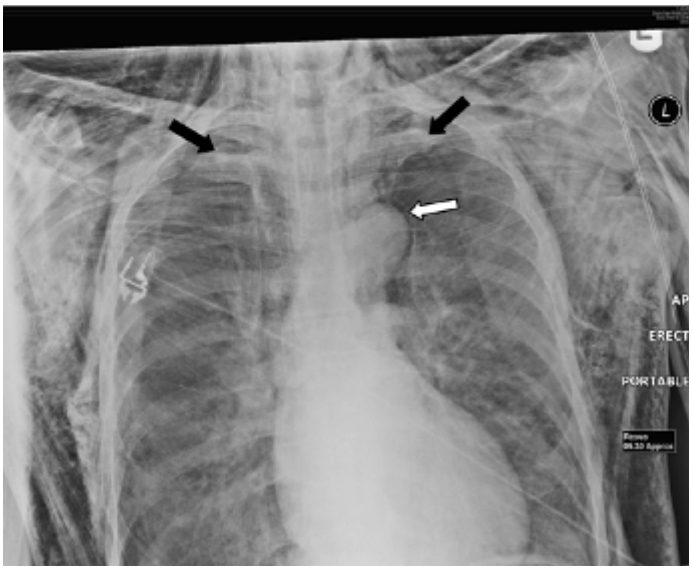

Figure 7. *Chest X-ray of a different patient, with severe surgical emphysema, throughout the X-ray. Bilateral pneumothoraces are seen (black arrows), with air in the mediastinum and around the aortic knuckle (white arrow). Radiolucent striations are seen outlining the pectoralis major when there is surgical emphysema in the anterior chest wall (Ginkgo leaf sign)*

Case 3

What does the chest X-ray show?
What is the diagnosis?

The presence of surgical emphysema suggests that this man's asthma has been complicated by pneumomediastinum and possibly pneumothorax. A pneumothorax is not seen on the chest X-ray and may not be present although that not all pneumothoraces can be seen on chest X-ray, particularly those that are small and anterior. Although rare, pneumothorax should be considered and sought in any severe acute attack of asthma, particularly when an acute deterioration occurs despite full treatment.

Pneumothorax and pneumomediastinum are rare but recognised complications of asthma. In acute severe asthma, there is overexpansion of the distal air ways due to obstruction in the bronchi and bronchioles. Excessive alveolar pressure can cause their rupture and air tracks in to the lung interstitium. The air within the interstitium follows a pressure gradient and migrates in a centripetal direction from the lung parenchyma towards the mediastinum, resulting in a pneumomediastinum. Sometimes a small double border to the left heart or aortic knuckle can be seen, representing mediastinal air (Figure 7). Air can continue to track between tissue planes and appear on the radiograph as subcutaneous emphysema in the face, neck, limbs, chest wall and abdomen. This may be very dramatic (Figure 7).

A further and more serious complication of pneumomediastinum is tension pneumothorax, which can be unilateral or bilateral. This occurs when there is a continuous leak between the airways, interstitium and tissue planes. Clinical diagnosis of a tension pneumothorax in a patient with acute asthma can be challenging but is indicated by following key signs:

- haemodynamic instability
- unequal chest movement
- tracheal deviation away from the affected side and absent breath sounds.

In acute severe asthma, tracheal deviation is the sign that best differentiates tension pneumothorax from other signs that may reasonably be attributed to asthma. Treatment is with immediate needle decompression. As the tension pneumothorax develops, cardiac output is impaired because venous return to the heart is reduced from a combination of increased intrathoracic pressure causing venous compression and kinking of major thoracic veins as the mediastinum shifts. There have been case reports describing bilateral tension pneumothoraces, and clinical detection of this is extremely difficult because tracheal deviation may be minimal or absent.

The prevalence of pneumomediastinum in asthma is thought to be 1 in 33 000, with the majority of cases reported in healthy, young asthmatic adults. There is about a 10% chance of a pneumothorax complicating a pneumomediastinum, and pneumothorax may occur in the absence of clinically detectable pneumomediastinum. Any patient with structurally abnormal lung tissue, such as chronic obstructive pulmonary disease with emphysematous bullae, bronchiectasis and asthma is at increased risk of developing pneumothoraces, and this should be considered in the differential diagnosis of a dyspnoeic patient with underlying lung disease. Positive-pressure ventilation, for anaesthesia or resuscitation, increases the chance of simple and tension pneumothorax in all patients with these lung conditions.

Pneumomediastinum is more commonly occurs as result of trauma. This can be blunt chest trauma or iatrogenic, for example after central venous cannula insertion, endotracheal tube insertion, chest drain placement or percutaneous tracheostomy. Furthermore, any mechanism that raises intramediastinal pressure can cause pneumomediastinum. Of note, oesophageal perforation following repeated vomiting should be suspected if a respiratory cause is not found and there is evidence of a pleural collection.

How would you manage this patient?

Most cases of surgical emphysema are an incidental finding on examination and resolve over time through reabsorption. Pneumomediastinum can usually be managed conservatively. The key in this patient is to maintain a high level of suspicion of pneumothorax, with frequent reassessment, particularly if the patient fails to improve or worsens with standard treatment for his asthma.

Your focus should be to optimise management of his asthma. An ABCDE assessment is sensible in any acutely unwell patient and this should be undertaken as you institute standard therapy for acute severe asthma (this is described in detail in the British Thoracic Society guidance.[1]

Rare cases of mediastinal air tracking to the hypopharynx have been reported, which means that there is potential for acute airway obstruction. This may be clear clinically with stridor or it may become apparent at laryngoscopy, when there can be gross oedema and it can be difficult to insert an appropriately sized endotracheal tube. If there is concern about airway patency, the airway should be secured. If gas exchange is severely impaired and/or the patient is tiring, P_aCO_2 will rise (Figure 8) and contribute to loss of consciousness, necessitating airway support.

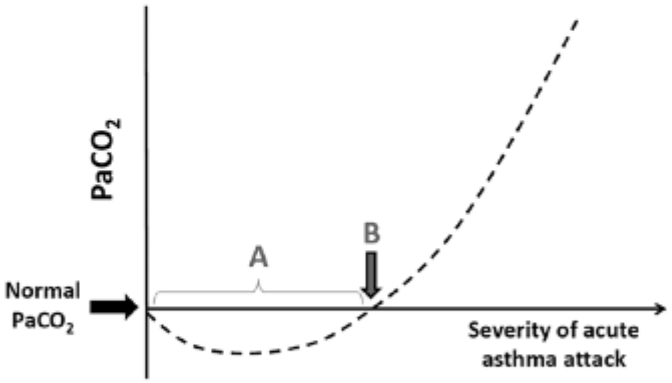

Figure 8. *Schematic graph to show change in P_aCO_2 with increasing severity of an acute asthma attack. Initially the patient will hyperventilate and tend to have a lower than normal P_aCO_2 (A). As they tire, or bronchospasm worsens, the P_aCO_2 rises. An arterial blood gas sample taken at point B may show a normal P_aCO_2, but this is not a reassuring sign (unless the patient is completely well) – the patient may be on the cusp of further deterioration to type 2 respiratory failure, CO_2 retention, loss of consciousness and collapse. Beware the normal P_aCO_2 in an acute severe asthmatic!*

REFERENCE

1. British Thoracic Society/Scottish Intercollegiate Guidelines Network. Quick reference guide. British guideline on the management of asthma 2014. Available at: https://www.brit-thoracic.org.uk/document-library/clinical-information/asthma/btssign-asthma-guideline-quick-reference-guide-2014/