Key points
A plain PA or AP radiograph generally underestimates the size of a pneumothorax.
Simple aspiration is recommended as a first-line treatment for all primary pneumothoraces requiring intervention in the spontaneously breathing patient without CPAP.
Patients who develop a pneumothorax while on positive pressure ventilation or CPAP should be treated with a chest drain unless immediate weaning from positive pressure ventilation is possible.
A thoracic surgical opinion should be sought in cases of persistent large volume air leaks or failure of the lung to re-expand significantly within 4 days.
Except in rare cases of tension, air emphysema and pneumomediastinum are not life-threatening and need observation only.
Tension pneumothorax is an unusual but a recognized cause of respiratory and cardiovascular compromise during anaesthesia and surgery.

Pathophysiology
Although patients with PSP do not have clinically apparent lung disease, sub-pleural bullae are found in 76–100% of cases during video-assisted thoracoscopic surgery.\(^1\) The mechanism of bulla formation is unclear. Even among non-smokers with a history of pneumothorax, 81% have bullae.\(^2\) In smokers, a likely explanation is that degradation of elastic fibres in the lung occurs, induced by the smoking-related influx of neutrophils and macrophages. This degradation causes an imbalance in the protease–antiprotease and oxidant–antioxidant systems. After bullae have formed, inflammation-induced obstruction of the small airways increases alveolar pressure, resulting in an air leak into the lung interstitium. The air then moves to the hilum, causing pneumomediastinum and, when the mediastinal pressure increases, rupture of the mediastinal pleura occurs causing pneumothorax.\(^3\)

In patients requiring intensive care, a pneumothorax is often caused by barotrauma associated with mechanical ventilation in the presence of reduced lung compliance. The initial process in barotrauma is the production of perivascular interstitial emphysema. When the pressure gradient between the alveoli and the interstitium exceeds a critical level, alveoli rupture occurs and air enters into the interstitium. The pressure at which this occurs varies with severity of lung injury and is associated with the use of excessive tidal volumes during ventilation.\(^4\) The over-distension of the non-dependent areas of the lung and rupture of the mediastinal pleura explain why anterior, medial, and sub-pulmonary pneumothoraces are more common in ARDS. Restrictive ventilatory strategies are helpful in preventing such air leaks.

A pre-existing pneumothorax is likely to enlarge and tension with the application of positive pressure ventilation, the use of nitrous oxide anaesthesia, and any reduction in barometric pressure (e.g. high altitude/air transport). Similarly, such changes in pressure may cause the development of a pneumothorax in patients with a pre-existing air leak.

Causes of pneumothorax
Primary spontaneous pneumothorax occurs more often in smokers and patients with Marfanoid habitus. The common causes of secondary pneumothorax are summarized in Table 1.
Clinical features

Typically, most awake spontaneously breathing patients have pleuritic chest pain and dyspnoea. In patients with underlying lung disease, dyspnoea is severe and significant hypoxaemia can occur, even with a small pneumothorax. Arterial blood gas measurements typically show an increase in the alveolar–arterial oxygen gradient and acute respiratory alkalosis.

The diagnosis of pneumothorax in critical illness or in the ventilated patient is suggested by deterioration in the patient’s respiratory and cardiovascular variables, and physical examination. It is confirmed by radiological investigation or ultrasound. Patients with a small pneumothorax (<15% of the hemithorax) often have a normal physical finding on examination. Tachycardia is the most common physical finding. In patients with a larger pneumothorax, examination shows decreased movement of the chest wall, a hyper-resonant percussion note, tracheal shift, and decreased or absent breath sounds on the affected side. The physical findings are often subtle and may be masked by the underlying lung disease, particularly in patients with chronic obstructive pulmonary disease. Systemic hypotension and central cyanosis should raise the suspicion of a tension pneumothorax. Ventilator parameters will show decreased tidal volumes and compliance.

Chronic lung conditions give rise to adhesions between parietal and visceral pleura restricting lung collapse. In such situations, a pneumothorax may be loculated and localized rather than spreading throughout the pleural space. In established adult respiratory distress syndrome (ARDS), a pneumothorax is often present without the lung completely collapsing as a result of the stiff, non-compliant nature of the lungs that are filled with fluid and cellular debris and associated pleural inflammation. Similarly, such affected lungs may be slow to re-expand (Fig. 1). Therefore, a tension pneumothorax may exist without total lung collapse or mediastinal shift. Once a pneumothorax has occurred, the high pressures generated during mechanical ventilation cause the pneumothorax to tension producing respiratory and haemodynamic effects. If suspected, it should be confirmed with a chest X-ray or other investigations without delay.

Subcutaneous emphysema, pneumomediastinum, and pneumopericardium

Subcutaneous emphysema is characterized by painless swelling of the tissues because of air tracking along tissue planes. It is commonly seen over the chest wall around drain sites, in the head and neck. Palpation elicits a characteristic tissue paper feeling beneath the fingers. Air may track deeper into the mediastinum, retroperitoneum, scrotum, and down into the limbs. It is rarely a problem clinically but may herald the presence of a pneumothorax or other visceral damage. If very tense and causing distress to the patient, it can be drained by skin incision with a needle or pointed blade. Radiologically, it is seen as translucent areas along the line of anatomical structures (Fig. 1).

Pneumomediastinum is caused by similar mechanisms to pneumothorax. Radiologically, it is seen as black lucent air collections along the line of tissue planes and visceral structures. In addition, it may indicate perforation of the GI tract (e.g. after oesophageal instrumentation). It is also seen with perforation of pharynx, duodenum, colon, and rectum with tracking of air into mediastinum. Pneumothorax is associated with but not generally caused by, pneumomediastinum. Except in rare cases of tension, pneumomediastinum is not a life-threatening condition. It is a predictable effect of positive pressure ventilation through a misplaced tracheostomy tube in the paratracheal tissues. If ventilation is continued, the latter is typically followed by a unilateral or bilateral pneumothoraces.

The Macklin effect is a pathophysiological process summed up in three steps: alveolar rupture, air dissection along bronchovascular sheaths, and spreading of this pulmonary interstitial emphysema...
into the mediastinum. This leads to pneummediastinum in conditions such as neonatal respiratory distress syndromes, acute asthma, positive-pressure mechanical ventilation, and Valsalva manoeuvres (e.g. parturition, Boerhaave’s syndrome, and epileptic seizures). Venous air embolism from such mechanisms is well described as a terminal event in neonates with neonatal respiratory distress syndrome (RDS).

Pneumopericardium is an uncommon but potentially life-threatening condition. It is often caused by penetrating chest trauma, invasive procedures (e.g. laparoscopy and tracheostomy), and infections (e.g. lung abscess), but it can also occur during coughing, the Heimlich manoeuvre, the Valsalva manoeuvre, and mechanical ventilation. In tension pneumopericardium, rapid resuscitation and emergency pericardiocentesis may be required.

**Barotrauma and mechanical ventilation**

An estimated 4–15% of critically ill patients on ventilators develop barotrauma, manifesting as abnormal air collections in the chest. Underlying lung disease, such as pneumonia and especially ARDS, increases the risk significantly. The major factors associated with development of barotrauma include a peak inspiratory pressures $>40$ cm H₂O, the use of positive end-expiratory pressure (PEEP), and an inappropriately large tidal volume. Restrictive ventilation strategies should help in prevention.

**Distinction of pneumothorax from emphysematous bullae**

The bullae of emphysema can be very large and, when situated in the periphery of the lung, can mimic a loculated pneumothorax. A chest drain inserted into a bulla in the mistaken belief that it is a pneumothorax is not uncommon. The lack of a lung edge, the round nature of the bulla, and the presence of multiple bullae elsewhere in the lung are all clues to the diagnosis. In difficult cases, computed tomography (CT) is helpful in distinguishing between the two. Another classical differential diagnosis not to be missed is air-filled stomach or bowel in the chest secondary to diaphragmatic hernia.

**Investigations**

**Chest X-ray**

The classical appearance in the upright position is the presence of radiolucent air and the absence of lung markings between the shrunken lung and the parietal pleura (Fig. 2). In the supine ventilated patient, gravity and the effects lung disease often give rise to a different appearance of the so-called ‘supine pneumothorax’. The pneumothorax is usually anteromedial or sub-pulmonic causing lucent upper quadrants of the abdomen, sharp superior surfaces of the diaphragm, the deep sulcus sign (Fig. 3), and visualization of the inferior surface of consolidated lung. Less often, the pneumothorax is apical, lateral (displaces the minor fissure from the chest wall), or posteromedial. False-positive appearances may occur from skin folds, overlying tubing/dressing/lines, and prior chest tube tracks.

Small pneumothoraces are significant in the ventilated patient as they can acutely enlarge and tension. Signs of tension include mediastinal shift, loss of compliance, cardiovascular system (CVS) instability, high central venous pressure (CVP), displacement of the anterior junction line, azygoesophageal recess, and flattening of heart and vascular shadows.
Symptomatic patients should not be left without intervention regardless of the size of the pneumothorax on a chest radiograph. Other considerations include the need for positive pressure ventilation, impending anaesthesia and surgery (nitrous oxide diffuses into air collections and increases pressure/volume), transport in or outside the hospital, and altitude changes (including air transport).

**Aspiration**

Simple aspiration is recommended as first-line treatment for all primary pneumothoraces requiring intervention but is less likely to succeed in secondary pneumothoraces. In the latter situation, it is only recommended as an initial treatment in small (<2 cm) pneumothoraces in minimally breathless patients.

Pleural aspiration is performed after strict aseptic precautions with the patient in the supine position. The aspiration is carried out in the fourth inter-costal space in the anterior axillary line. The site is infiltrated with lidocaine and an 18-G i.v. cannula is inserted into the pleural cavity. The needle is withdrawn and three-way stopcock connected to the i.v. cannula. A 50 ml syringe and i.v. tubing with its end under a water seal are connected to the cannula through the three-way tap. Air is aspirated and expelled by means of the tubing and its volume noted. The end point of the procedure is a feeling of resistance to aspiration or if the patient begins to cough excessively. The patient will usually get a sensation that the lung has expanded. The i.v. cannula is withdrawn and entry site sealed. Avoid using a needle alone, as the lung will become lacerated as it expands towards the needle.

Advanced Trauma Life Support guidelines recommend the use of a cannula of 3–6 cm long to perform needle thoracocentesis for life-threatening tension pneumothorax. However, in 57% of patients with tension pneumothorax, the thickness of the chest wall has been found to be >3 cm. Therefore, it is recommended that a cannula length of at least 4.5 cm should be used in needle thoracocentesis of tension pneumothoraces. The cannula should be left in place until bubbling is confirmed in a formal chest drain with underwater seal to indicate proper function of the intercostal tube. We encourage clinicians, wherever possible, to ascertain by imaging that a pneumothorax is present before inserting such cannulae. Otherwise, it is inevitable that the needle will enter the lung and produce an air leak.

**Chest drains and closed underwater systems**

If simple aspiration of any pneumothorax is unsuccessful in controlling symptoms, an intercostal tube should be inserted. Intercostal tube drainage is recommended in secondary pneumothorax except in patients who are not breathless and have a very small (<1 cm or apical) pneumothorax.

Small bore drains are as effective for air drainage as large bore drains and are more comfortable for patients. If there is associated blood, a large bore drain will be required. There are no large
Air leaks, pneumothorax, and chest drains

randomized, controlled trials directly comparing small and large bore drains.

The most common position for chest tube insertion is in the mid-axillary line, through the ‘safe triangle’ illustrated in Figure 4. This position minimizes risk to underlying structures such as the viscera and internal mammary artery and avoids damage to muscle and breast tissue resulting in unsightly scarring. A more posterior position may be chosen if suggested by the presence of a loculated collection. While this is relatively safe, it is not the preferred site as it is more uncomfortable for the patient to lie on after insertion and there is more risk of the drain kinking.

For apical pneumothoraces, the second intercostal space in the mid-clavicular line is sometimes chosen; it is not recommended routinely, as it may be uncomfortable for the patient, may leave an unsightly scar, and internal mammary vessels are at risk. If the drain is to be inserted into a loculated pleural collection, the position of insertion will be dictated by the site of the locule as determined by imaging. A common mistake is to insert drains too low in the chest risking damage to the diaphragm, liver, spleen, and heart.

The drain should not be removed until bubbling has ceased, and chest radiography demonstrates lung re-inflation. There is no evidence that clamping a chest drain at the time of its removal is beneficial.

The use of high-volume/low-pressure suction pumps has been advocated in cases of non-resolving pneumothorax or after chemical pleurodesis; however, there is no evidence to support its routine use in the initial treatment of spontaneous pneumothorax. If suction is required, this should be performed through the underwater seal at a level of 10–20 cm H₂O. A high-volume pump is required to cope with a large leak.

Prevention strategies

There are two methods used to prevent recurrence of pneumothoraces: (i) medical pleurodesis (installing talc/bleomycin by a chest drain) and (ii) surgical pleurodesis (parietal pleura is mechanically abraded/stripped). Both techniques produce fibrosis and scarring so that the visceral pleura adheres to the chest wall obliterating the pleural space and prevents further pneumothorax.

Bronchopleural fistula

A bronchopleural fistula is a communication between the bronchial tree and pleural space. Clinically, it may be best described as a persistent air leak or a failure to re-inflate the lung despite chest tube drainage for 24 h. Causes include chest trauma, complications of diagnostic or therapeutic procedures (e.g. thoracic surgery with a failure of suture/staple line), chest drains inserted into the lung parenchyma, and complications of mechanical ventilation. The main problems with a large fistula in a ventilated patient are the loss of delivered tidal volume, inability to apply PEEP, persistent lung collapse, and delayed weaning from assisted ventilation.

Management strategies include general conservative measures such as large bore chest drains (multiple if necessary) and the use of drainage system with adequate capabilities. In mechanically ventilated patients, the goal is to maintain adequate ventilation and oxygenation while reducing the fistula flow to allow the leak to heal. This includes reducing inspiratory pressures, tidal volumes, respiratory rate, PEEP, and inspiratory times, and accepting permissive hypercapnia and lower oxygen saturations. Most air leaks will settle spontaneously over a few days if the patient can be weaned onto spontaneous respiration without high levels of continuous positive airways pressure (CPAP). The size of the air leak is critical; small tears or punctures will heal quickly while larger structural damage to the lung or a major bronchus will not settle with conservative management, particularly if high inflation pressures are required for associated lung injury.

The use of other modes of ventilation including high-frequency ventilation, oscillation, and differential lung ventilation through double-lumen tubes has been reported. For proximal leaks, fiberoptic bronchoscopy and direct application of sealants (e.g. cyanoacrylate, fibrin agents, gelform) have been tried with limited success. Refractory cases need surgical repair of the air leak by thoracoplasty, lung resection/stapling, pleural abrasion/decortication, or other techniques.

References


Please see multiple choice questions 7–10