Pulmonary atelectasis in anaesthesia and critical care

Komal Ray MBBS FRCA
Andrew Bodenham MBBS FRCA FICM
Elankumaran Paramasivam MBBS MRCP FICM

Key points
Atelectasis during general anaesthesia (GA) is common, but usually does not cause clinically significant problems. Persistent prolonged atelectasis after GA increases perioperative respiratory complications.

Risk factors contributing to atelectasis include: obesity, chronic lung disease, thoracic or upper gastrointestinal surgery, and prolonged use of high-inspired oxygen concentration.

The aetiology and significance of atelectasis in critically ill patients is different from that seen in patients undergoing GA. The treatment and prevention of atelectasis varies according to the aetiology, severity, and clinical context.

Atelectasis is derived from the Greek words *ateles* and *ektasis*, meaning incomplete expansion. It is also referred to as collapse of the lung. Atelectasis is the loss of lung volume, either a part or all of a lung with or without mediastinal shift. This is in contrast to consolidation where the lung volume is normal. In clinical practice, there is often a combination of both. Atelectasis is common in the setting of anaesthesia and critical care. Atelectasis can be broadly classified into obstructive and non-obstructive, each having a particular radiological pattern. Obstructive atelectasis is by far the most common cause of lung collapse, in both adult and paediatric populations.

Types of atelectasis

In the context of chest medicine, several types of atelectasis can be categorized according to aetiology (Table 1).

Obstructive

Bronchial obstruction is a frequent cause of atelectasis. This is usually due to a neoplasm, mucus plug, or foreign body. Progressive airway collapse develops distal to the obstruction.

Non-obstructive

Various types exist:

- Compressive: a large peripheral tumour, bullae, or extensive air trapping (emphysema) compresses adjacent normal lung tissue.
- Passive: this is also referred to as relaxation atelectasis, and it is caused by the loss of contact between parietal and visceral pleura, for example, due to pleural effusion, pneumothorax, or pleural malignancy.
- Adhesive: this is caused by a lack of or inactivation of surfactant and can be seen in severe acute lung injury (ALI), radiation pneumonitis, or neonatal respiratory distress syndrome.
- Cicatrizating: this occurs due to scar tissue formation as a result of granulomatous disease or necrotizing pneumonia.

The above types of atelectasis are most often described in the chronic setting, but in this article, we focus on the mechanisms, pathophysiology, diagnosis, and management of pulmonary atelectasis in the settings of general anaesthesia (GA) and critical care.

Atelectasis related to GA

Atelectasis is common during anaesthesia and is frequently noted in critically ill patients with different underlying aetiologies and pathophysiology. Uneventful anaesthesia can lead to the development of collapse in 10–15% of lung tissue. The following mechanisms have been proposed to contribute to atelectasis during GA.

Compression atelectasis

In a normal healthy adult in the upright position, the functional residual capacity (FRC) is ≈ 3 litres. This decreases by 0.7–0.8 litres in the supine position as abdominal contents push the diaphragm cephalad. This is further accentuated with anaesthesia and paralysis, as abdominal pressure is easily transmitted to the thoracic cavity. The loss of intercostal muscle function also contributes to FRC reduction, particularly in children. Surgical manipulation during thoraco-abdominal procedures may worsen atelectasis caused by GA. Other factors accentuating compression atelectasis include morbid obesity, laparoscopic procedures, and head-down and lateral positioning. During one-lung anaesthesia, the lung on the side of surgery is typically deliberately collapsed to allow surgical access on that side.
Absorption atelectasis

This can occur by two different mechanisms:

(i) Complete airway occlusion can be seen in accidental bronchial intubation, one-lung anaesthesia, and with mucus plugging of small or large airways. Its pathophysiology is similar to that described in obstructive mechanisms.

(ii) Atelectasis can occur in the absence of obstruction. Lung zones with lower ventilation relative to perfusion are susceptible to collapse, this can occur when inspired oxygen concentration is increased leading to a higher flux of oxygen from the alveoli to capillary, and alveoli progressively become smaller.

Atelectasis in the critical care setting

Atelectasis is a common cause of impaired gas exchange and X-ray opacification of lung regions in critically ill patients. It may be encountered in the presence or absence of ALI. The incidence is likely to be high if the patient is immobile, has had a general anaesthetic, or if they have pre-existing lung disease, a smoking history, obesity, or advanced age. Its pathophysiology is multifactorial: obstructive, non-obstructive, or both. Most cases are likely to be multifactorial in origin with prolonged immobility and infection probably being the most common contributors.

Acute lung injury

Extensive evidence exists regarding the maintenance of lung volume in the prevention of lung injury. Atelectasis in patients with ALI differs from atelectasis related to GA. During lung injury, atelectasis is accompanied by inflammatory fluid filling up the alveoli and a phenomenon of cyclical collapse is seen. Also, repetitive lung atelectasis leads to increased neutrophil activation. This leads to an inflammatory response causing localized lung injury alongside systemic and distant organ dysfunction due to systemic release of inflammatory mediators (cytokines, proteases, and reactive oxygen species). It will therefore take time to resolve, unlike the usually fairly rapid resolution after uncomplicated GA and surgery.

Surfactant depletion

Surfactant is a lipoprotein complex secreted by Type II alveolar cells which forms an inner coating for alveoli. It reduces surface tension within a theoretically spherical structure like an alveolus, and so may be considered to follow Laplace’s law (pressure = 2 × tension/radius). This equation indicates that smaller alveoli will contain higher pressure than larger ones, and so would quickly empty into the larger alveoli, and so collapse. Surfactant reduces surface tension in all alveoli, and to a greater extent in smaller ones, thereby maintaining alveolar stability. The lack of surfactant function therefore leads to atelectasis. Also, lung tissue deficient in surfactant is difficult to inflate leading to increased work of breathing and so likelihood of respiratory failure. This situation can be seen in premature neonates who may require artificial surfactant. In adults, surfactant function may be impaired by lung infection or inflammation.

Pathophysiological effects of atelectasis

Decreased compliance

The loss of lung volume as a result of atelectasis causes inspiration–expiration cycles to commence from a lower FRC, so these are occurring on a less efficient section of the pressure–volume curve. As a result, increased transpulmonary pressure is required to achieve a given tidal volume, leading to increased work of breathing.

Impaired oxygenation

Atelectasis can significantly affect systemic oxygenation by the loss of adequate ventilation to perfused lung units. This was first identified during GA and the effect was reversed by passive hyperinflation.

Increased pulmonary vascular resistance

Regional hypoxia in atelectatic lung units leads to hypoxic pulmonary vasoconstriction due to decreased alveolar and mixed venous oxygen tension. If extensive, this phenomenon may lead to right ventricular dysfunction and increased microvascular fluid leakage in susceptible patients.

Clinical presentation

This depends on the extent of atelectasis and rapidity with which it develops. Small and slowly developing areas of collapse may be asymptomatic or present as a non-productive cough. Rapidly

Table 1 Causes of lung collapse in anaesthesia and critical care

<table>
<thead>
<tr>
<th>Obstructive</th>
<th>Non-obstructive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Large airway obstruction</td>
<td>Compressive</td>
</tr>
<tr>
<td>Tumour: bronchial, metastatic</td>
<td>Peripheral tumour</td>
</tr>
<tr>
<td>Inflammatory: tuberculosis, sarcoidosis</td>
<td>Interstitial disease: sarcoidosis, lymphoma</td>
</tr>
<tr>
<td>Other: foreign body, malpositioned tracheal tube</td>
<td>Air trapping in adjacent lung: emphysema</td>
</tr>
<tr>
<td>Small airway obstruction</td>
<td>Passive</td>
</tr>
<tr>
<td>Mucus plugs</td>
<td>Bibal collapse under anaesthesia</td>
</tr>
<tr>
<td>Inflammation</td>
<td>Thoracic and abdominal surgery</td>
</tr>
<tr>
<td>Bronchopneumonia, bronchitis, bronchiectasis</td>
<td>Pneumothorax, pleural effusions</td>
</tr>
<tr>
<td>Non-obstructive</td>
<td>Diaphragmatic hernia</td>
</tr>
<tr>
<td>Compressive</td>
<td>Adhesive</td>
</tr>
<tr>
<td>Peripheral tumour</td>
<td>Smoke inhalation</td>
</tr>
<tr>
<td>Interstitial disease: sarcoidosis, lymphoma</td>
<td>Cardiopulmonary bypass</td>
</tr>
</tbody>
</table>

Pulmonary atelectasis

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developing large-scale atelectasis can present with features of hypoxia and respiratory failure. Physical examination will reveal decreased movement in the affected lung area, dullness on percussion, absent breath sounds, and deviation of the trachea to the affected site.

**Investigations**

**Chest X-ray**

The radiological signs of collapse will depend upon the aetiology, degree of collapse, and associated consolidation or pleural pathology. Signs may be direct (related to loss of lung volume and collapsed lobes) or indirect (occurring as result of compensatory changes due to volume loss), leading to shift of the mediastinal structures.

**Direct signs**

These include:

- Increased opacification in the area of atelectasis. Air bronchograms are normally a feature of consolidation but may also be present in lobar collapse.
- Displacement of fissures. This occurs with large degree of collapse.
- Loss of aeration. If the collapsed lung is adjacent to the mediastinum or diaphragm, then loss of definition of these structures indicates loss of aeration (the silhouette sign).
- Vascular signs. In partial collapse, crowding of vessels may be seen.

**Indirect signs**

These include:

- Elevation of a hemi-diaphragm. This sign is of limited value as the normal position of the diaphragm is variable.
- Mediastinal displacement to the side of collapse. Some contents of the mediastinum which are easily seen on plain chest X-rays include the trachea, tracheal tube, central venous catheters in the superior vena cava, and nasogastric tubes in the oesophagus.
- Hilar displacement. The hilum may be elevated in the upper lobe collapse, and depressed in the lower lobe collapse.

**Radiological patterns of collapse**

**Complete collapse**

Collapse of an entire lung leads to complete opacification of a hemithorax (a so-called ‘white out’). This is often confused with a large pleural effusion, but can be distinguished by the presence of mediastinal shift towards a collapsed lung (Fig. 1) compared with movement away from a pleural effusion (Fig. 2). Ultrasound or computerized tomography (CT) allow definitive confirmation of the presence of an effusion.

**Fig 1** Total lung collapse due to obstructive pathology causing a white out of the right hemithorax. Note the trachea is pulled towards the collapsed lung.

**Fig 2** Total lung collapse of the left lung due to pressure from a pleural effusion leading to tracheal shift away from the left lung.

**Lobar collapse**

Characteristic features associated with individual lobar collapse are as follows:

- Right upper lobe (RUL) collapse (Fig. 3a and b) results in elevation of the right hilum and the minor fissure. On the lateral view, elevation of the minor and major fissure may be visible. The minor fissure in RUL collapse is usually convex superiorly but may appear concave because of an underlying mass lesion. This is called the sign of Golden S (Fig. 3c).
- Right middle lobe (RML) collapse (Fig. 4a and b) results in minimal opacity and is often overlooked. The loss of silhouette of the right heart border is almost always a feature on a
posterior–anterior view. The right horizontal and oblique fissures move towards each other leading to a wedge-shaped opacity on the lateral view. A chronically collapsed RML is often associated with bronchiectasis and is known as RML syndrome.

- Right lower lobe (RLL) collapse (Fig. 5A and B) is seen as a triangular opacity adjacent to the right heart border. There is obliteration of the right hemidiaphragm and it may appear elevated. However, the right heart border is clearly seen. On lateral projection, the right hemidiaphragm outline is lost posteriorly and the lower thoracic vertebrae appear more dense.
- Left upper lobe (LUL) collapse (Fig. 6A and B). Owing to the lack of a minor fissure, LUL collapse appears different compared with the RUL. It appears as a veil-like opacity extending from the hilum and fading inferiorly. On the lateral view, the major fissure displaces anteriorly and the lower lobe is hyper-expanded. LUL collapse also causes a hyper-expanded superior segment of the left lower lobe (LLL), which is positioned between the atelectatic upper lobe and the aortic arch in half of cases. This gives the appearance of a crescent of aerated lung, called the Luftsichel sign (Fig. 6A).
- LLL collapse (Fig. 7A and B) is seen as an increased retrocardiac opacity, which silhouettes the left hemidiaphragm. On the lateral view, the left hemidiaphragm outline is lost posteriorly and the lower thoracic vertebrae appear denser than normal.

Fig 3 (A) RUL collapse (PA view). The minor fissure and hilum are displaced upwards. The mediastinal border is obscured. The trachea is deviated to the right. (B) RUL collapse (lateral view). The minor and major fissures (arrows) are pulled upwards causing a triangular opacity with well-defined margins. (C) Golden S sign. This is seen in RUL collapse due to a mass lesion. The superior portion of the ‘S’ is from the displaced minor fissure (arrow), while the inferior portion results from the mass itself (arrowhead).
In clinical practice, particularly in the sicker patient, many of these patterns of collapse can co-exist, and bibasal collapse is commonly seen after major anaesthesia and surgery, or in a critically ill patient with impending respiratory failure.

- Rounded atelectasis. This unusual type of collapse appears as a homogenous mass on a plain film. This is due to segmental or subsegmental atelectasis, and occurs secondary to visceral pleural thickening and entrapment of lung tissue.

**CT scan**

Atelectasis on CT has been defined as pixels with attenuation values of $-100$ to $+100$ Hounsfield units. The Hounsfield unit is a measure of X-ray attenuation used in CT scan interpretation. It characterizes the relative density of a substance, that is, air: $-1000$, fat: $-50$, water: 0, muscle: $+40$, calculus: $+100$ to $+400$, bone: $+1000$. Features of lobar collapse and effusions are more obvious on CT than plain radiographs (Fig. 8) and it is useful for more atypical forms of collapse. CT aids identification and localization of endobronchial lesions and any tumour spread, and differentiation of obstructive lesions from other forms of atelectasis.

**Ultrasound**

Obstructive atelectasis on ultrasound shows as an area of homogenous low echogenicity. An important role for ultrasound is to distinguish basal lung collapse from a loculated pleural effusion (Fig. 9). An affected segment of collapse resembles liver (so-called hepatization of the lung). Echogenic bands may be visible as a result of a fluid bronchogram. No reinflation of the lung is seen during inflation. On using colour-flow Doppler, increased flow signals relative to the liver are seen. Also the ‘lung pulse’ may be used as a dynamic lung ultrasound sign described as absent lung sliding with the perception of heart activity at the pleural line, associated with complete atelectasis (e.g. during endobronchial intubation).

**Other investigations**

These include bronchoscopy, thoracoscopy, and open surgery, but these are often more treatment based than purely for diagnosis.
Prevention of atelectasis is preferable to later treatment to re-open collapsed areas of the lung, but techniques for both are similar and based on the causes of atelectasis. Evidence-based studies on the management of atelectasis are lacking.

Perioperative management

Prevention of atelectasis begins in the preoperative period by identifying high-risk patients and introducing intensive respiratory therapy of physiotherapy, bronchodilators, cessation of smoking, and antibiotics when indicated, at least 5–7 days before operation for elective surgery. In the setting of more urgent surgery, outcomes may be improved if the operation can be delayed for preoperative respiratory therapy. Common risk factors include patients with pre-existing lung problems (chronic obstructive pulmonary disease, asthma, bronchiectasis), smoking, obesity, advanced age, and sleep apnoea. Baseline X-rays, blood gases, and lung function tests are useful for patients with moderate to severe respiratory and cardiovascular disease who are undergoing more major procedures. Lung function test are not recommended as part of routine preoperative testing for adults (>16 yr) of any age classified as ASA I with no co-morbidities undergoing surgery of any severity.

On induction

Using 100% oxygen at induction is common practice to improve margins of safety in relation to hypoxaemia, but there is good evidence that the use of 100% oxygen is associated with atelectasis. A compromise is to reduce $F_{O_2}$ to 80% to reduce atelectasis, or perform a recruitment manoeuvre after induction. During induction of anaesthesia, application of continuous positive airway pressure (CPAP) can prevent the formation of atelectasis and can increase the margin of safety for oxygenation before intubation. For example, application of CPAP 10 cm H$_2$O in morbidly obese patients is effective for the prevention of atelectasis during induction.

During anaesthesia

Optimal modes of ventilation during anaesthesia are unclear, but it is likely that positive pressure ventilation with PEEP, rather than spontaneous ventilation is preferable in longer procedures in at-risk patients. Whether it is practical or beneficial to allow some
spontaneous breaths in longer cases rather that have a patient fully paralysed is unknown. Overall, ventilation strategies should follow those advocated in critical care (see below) with limited tidal volumes and peak inspiratory pressures.

Recruitment of atelectasis should be attempted if it is suspected clinically or in high-risk patients. Research has shown that recruitment of lung units follows a Gaussian distribution; hence, different units recruit at different pressures, range 10–45 cm H₂O. In addition to this critical opening pressure, lung recruitment requires time to allow the delivered gas volume to redistribute.

Suggested recruitment manoeuvres include:

- Vital capacity manoeuvre using an inflation pressure of 40 cm H₂O sustained for 10–15 s.
- Increasing PEEP to 15 cm H₂O and then increasing tidal volumes to achieve peak inspiratory pressure of 40 cm H₂O for 10 breaths before then returning to standard ventilator settings.

Higher levels of PEEP are generally not beneficial as the shunt does not improve due to redistribution of blood flow in the lung, and the high intra-thoracic pressure leads to decreased venous return and haemodynamic compromise.

**Postoperative period**

Atelectasis is one of the most common pulmonary complications in the postoperative period, although it is often clinically insignificant. The altered compliance of lung tissue, impaired regional ventilation, and retained airway secretions contribute to the development of atelectasis. Postoperative pain interferes with spontaneous deep breathing and coughing resulting in decreases in FRC, leading to atelectasis. In addition to general measures, a variety of lung expansion exercises may reduce postoperative pulmonary complications in selected patients, including chest physical therapy, deep breathing exercises, incentive spirometry, intermittent positive pressure breathing, and CPAP. Good postoperative pain control may help to minimize postoperative pulmonary complications by enabling earlier ambulation and improving the patient’s ability to take deep breaths.

**Management in critical care**

Treatment of atelectasis in critically ill patients differs from anaesthesia in that there is commonly a presence of background ALI or infection. The ‘open-lung’ approach to ventilating patients with severe
ALI consists of recruitment manoeuvres with high sustained airway pressures to open atelectatic alveoli, followed by application of PEEP to maintain alveolar patency.

**Ventilation strategies**

There are numerous strategies to consider when attempting to minimize atelectasis during artificial ventilation in critical care patients.

CPAP is useful for the management of spontaneously breathing patients with non-obstructive atelectasis, who are unable to breathe deeply. The aim is to open up collapsed alveoli to reduce shunt and improve ventilation–perfusion homogeneity, hence reversing hypoxaemia.

PEEP is a core component of artificial ventilation in ALI patients, but the level of PEEP required will depend on the clinical scenario. It is the airway pressure above PEEP that is responsible for alveolar recruitment. PEEP will then prevent recollapse. Patients at risk of ALI should have open-lung techniques instituted to optimize oxygenation. PEEP has a protective role in ALI by attenuating surfactant depletion, and reducing shearing stresses, parenchymal injury, and cytokine release. An open-lung strategy using low tidal volume (6–8 ml kg\(^{-1}\)), limiting distending pressure (plateau pressure <35 cm H\(_2\)O), and setting PEEP above the lower inflection point on the pressure–volume curve is suggested to decrease mortality, length of intensive care unit (ICU) stay, and days on ventilator.\(^9\)

**Typical recruitment measures**

Typical recruitment measures used in intensive care include:\(^10\)

- Three consecutive volume-limited breaths per minute with a plateau pressure of 45 cm H\(_2\)O (also called sigh).
- PEEP increased by 5 cm H\(_2\)O every 30 s with a 2 ml kg\(^{-1}\) decrease in tidal volume. When PEEP reaches 25 cm H\(_2\)O, CPAP at 30 cm H\(_2\)O is used for 30 s.
- CPAP: 35–40 cm H\(_2\)O for 30 s.

In general recruitment, measures are well tolerated, although systemic hypotension can occur due to reduced cardiac output when a sustained inflation pressure is used in critically ill patients, this can be reduced by adequate fluid filling pre-recruitment. Optimal recruitment strategies are unknown.

Inspired oxygen concentration has a strong influence on atelectasis. This has been described above for absorption atelectasis. In addition, high concentrations of oxygen used during resuscitation may increase the production of reactive oxygen species and contribute to reperfusion injury.
Muscle tone during artificial ventilation can also affect gas exchange. Allowing spontaneous breathing up to 10–20% of total ventilation improves gas exchange by redistribution of ventilation and end-expiratory gas to dependent areas, thus promoting alveolar recruitment. This can be achieved with airway pressure release ventilation or biphasic positive airway pressure.11

High-frequency oscillation ventilation (HFOV) may be considered and facilitates lung inflation and recruitment by maintaining mean airway pressure at a constant elevated level while using a piston to cycle the ventilation rate at several hundred times per minute. This results in tidal volumes that are smaller than the anatomical dead space of lungs with the potential to reduce volutrauma-related lung injury. HFOV minimizes cyclical alveolar distension and collapse. The mean airway pressure is set at 2–3 cm H2O above the mean airway pressure on conventional ventilation (usually similar or higher than PEEP but below plateau pressure) and increased in increments of 1–2 cm H2O until improvement in oxygenation occurs. Clinical studies of HFOV in severe ALI to date have not shown improvements in outcomes.12

Others: A variety of further strategies which may be considered in the management of atelectasis are shown in Table 2. These strategies are more likely to be used in ICUs and are not commonly required or used in theatre. There is no strong evidence to support the application of these techniques and so their use should be preceded by an individual risk–benefit analysis for each patient.

**Conclusion**

Atelectasis is commonly encountered in the perioperative and critical care settings and may lead to hypoxaemia and respiratory failure. Timely diagnosis and management is crucial for a good outcome. Prevention is better than cure when dealing with patients at high risk of developing atelectasis.

**Acknowledgement**

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**Declaration of interest**

We thank Medical illustration at St James Hospital Leeds for producing the line diagrams.

**References**


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**Table 2. Other strategies which may be used in the management of artificial ventilation in patients with atelectasis. FRC, functional residual capacity**

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Potential benefit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prone position</td>
<td>Promotes ventilation–perfusion matching, ↑ FRC, ↓ shunt</td>
</tr>
<tr>
<td>Kinetic bed (40° rotation on each side with pause of 10 min on each side and 5 min in the supine position)</td>
<td>Prevents collection of secretions, ↓ ventilator-associated pneumonia, redistribution of pressure to allow lung expansion</td>
</tr>
<tr>
<td>Chest physiotherapy</td>
<td>Aids airway clearance and improves ventilation</td>
</tr>
<tr>
<td>Bronchoscopy</td>
<td>Aids airway clearance in patients with poor cough, e.g. neuromuscular disease</td>
</tr>
<tr>
<td>Mucolytics or DNase</td>
<td>Clears tenacious secretions, good results in pediatrics</td>
</tr>
<tr>
<td>Surfactant</td>
<td>↓ surface tension to allow alveoli stability and prevent collapse</td>
</tr>
<tr>
<td>Fluorocarbons</td>
<td>Facilitates lung recruitment by separating adherent lung surfaces by virtue of their low equilibrium surface tension and positive spreading coefficient</td>
</tr>
</tbody>
</table>

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**Fig 8.** Computerized tomogram of RML collapse. There is a wedge-shaped opacity (arrow) in the midzone.

**Fig 9.** Lung ultrasound. There is a large pleural effusion causing underlying lung collapse.


Please see multiple choice questions 29–32.