Atelectasis: Types and pathogenesis in adults

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INTRODUCTION

Atelectasis describes the loss of lung volume due to the collapse of lung tissue. It can be classified according to the pathophysiologic mechanism (eg, compressive atelectasis), the amount of lung involved (eg, lobar, segmental, or subsegmental atelectasis), or the location (ie, specific lobe or segment location).

The classification of atelectasis according to the pathophysiologic mechanism is reviewed here, as are the mechanisms of each type of atelectasis. Radiologic manifestations of atelectasis are described separately. (See "Radiologic patterns of lobar atelectasis".)

TYPES OF ATELECTASIS

Atelectasis can be divided pathophysiologically into obstructive and nonobstructive atelectasis.

Obstructive atelectasis — Obstructive (ie, resorptive) atelectasis is a consequence of blockage of an airway [1]. Air retained distal to the occlusion is resorbed from nonventilated alveoli, causing the affected regions to become totally gasless and then collapse. The rate at which this occurs depends upon several factors, particularly the amount of collateral ventilation and the composition of inspired gas.

Obstruction of a segmental bronchus is less likely to result in segmental atelectasis than obstruction of a lobar bronchus is to produce lobar atelectasis. This difference is the consequence of collateral ventilation between segments within a lobe. Collateral ventilation occurs via three distinct channels (the pores of Kohn, canals of Lambert, and fenestrations of Boren) and all of these channels must be obliterated for obstructive atelectasis to occur. Such collateral ventilation does not occur between lobes, unless the interlobar fissures are incomplete (which is the case in ≥50 percent of normal persons) [2]. Of note, collateral ventilation is age-dependent:

- Children have poorly developed collateral pathways and, therefore, obstructive atelectasis can occur readily. This is why atelectasis is common in children who have aspirated a foreign body. (See "Airway foreign bodies in children", section on 'Imaging'.)
- In contrast, elderly adults with emphysema have extensive collateral ventilation through fenestrae of Boren, which are larger than the pores of Kohn. This explains why an obstructing lesion, such as a tumor, may have a long latent period in adults before it causes atelectasis [3].

Patients who are breathing gas with a high fraction of inspired oxygen (FiO_2) develop atelectasis more rapidly than patients who are breathing gas with a lower FiO_2. Under normal conditions, the ambient air contains 79 percent inert nitrogen, which is in equilibrium with the nitrogen that is dissolved in pulmonary arteriolar and capillary blood. After complete occlusion of a bronchus, intraalveolar oxygen distal to the obstruction is rapidly reabsorbed along a steep pressure gradient into deoxygenated mixed venous blood. The pressure gradient forcing nitrogen into the mixed venous blood is minimal. Thus, nitrogen disappears slowly and acts like an alveolar splint or scaffolding, delaying the formation of atelectasis for several hours. In contrast, in patients who inhale gas with a high FiO_2, the alveolar nitrogen is washed out. With less nitrogen in the alveoli at the moment of bronchial obstruction, atelectasis develops more rapidly, sometimes within minutes.

Nonobstructive atelectasis — Nonobstructive causes of atelectasis include loss of contact between the parietal and visceral pleurae, parenchymal compression, surfactant dysfunction, replacement of lung tissue by scarring or infiltrative disease, and strong vertical acceleration forces.

Relaxation — Relaxation (ie, passive) atelectasis ensues when contact between the parietal and visceral pleurae is eliminated [2]. While this is usually due to a pleural effusion or pneumothorax, a large emphysematous bulla can have a similar effect. In this case, the residual physiologic elastic recoil of normal lung parenchyma allows the normal lung to pull away from the bulla with subsequent loss of volume.

Normal lung usually preserves its shape, even when its volume is decreased. This quality is called form elasticity and is the reason why relaxation atelectasis is more likely to be generalized than localized. Less frequently, the lobes of the lung may behave independently. As examples, the middle and lower lobes may shrink more than the upper lobe in the presence of a pleural effusion, while the upper lobe may be affected more by a pneumothorax [4]. The loss of contact between the visceral and parietal pleurae may be local in this setting, which may cause local rather than generalized atelectasis.

Compressive — Compressive atelectasis occurs when a space occupying lesion of the thorax (eg, loculated pleural effusion, elevated hemidiaphragm, or solid mass of the chest wall, pleura, or parenchyma) presses on the lung and causes the lung volume to diminish to less than the usual resting volume (ie, the functional residual capacity) [1]. Compressive atelectasis has a lot in common with relaxation atelectasis (ie, both eliminate contact between the pleurae), except compressive atelectasis is more likely to be focal or localized [2].

Adhesive — Adhesive atelectasis is a consequence of alveolar instability due, in part, to surfactant deficiency or dysfunction [1]. In the normal lung, surfactant reduces the surface tension of alveoli and decreases the tendency of alveoli to collapse. Decreased production or inactivation of surfactant leads to alveolar instability and collapse. Adhesive atelectasis is a major problem in respiratory distress syndrome of premature infants, acute respiratory distress syndrome (ARDS) in adults, acute radiation pneumonitis, and posttraumatic lung contusion [2].

Cicatrization — Cicatrization (ie, cicatricial atelectasis) results from diminution of lung volume due to severe parenchymal scarring [1]. Common underlying etiologies include granulomatous disease (eg, sarcoidosis), necrotizing pneumonia, and radiation pneumonitis [2].

Replacement — Replacement atelectasis occurs when the alveoli of an entire lobe are filled by tumor (eg, mucinous adenocarcinoma, previously known as bronchioloalveolar cell carcinoma), with ensuing loss of volume.

Acceleration atelectasis — This type of atelectasis has been described in pilots subjected to very high, vertical accelerative forces between 5G and 9G: at 5G, up to 50 percent of pulmonary airways are distorted and closed due to gravitational forces. The atelectasis is exacerbated by breathing a high fractional concentration of oxygen. Decreases in vital capacity are a reflection of this type of atelectasis in pilots. Acceleration atelectasis can cause symptoms like chest pain, coughing, and dyspnea [5].

Rounded — Rounded atelectasis (also called folded lung, Blesovsky's syndrome, or atelectatic pseudotumor) is a distinct form of atelectasis associated with pleural disease, particularly following asbestos exposure. It is discussed separately. (See "Radiologic patterns of lobar atelectasis", section on 'Rounded atelectasis'.)

Plate-like — Plate-like atelectasis, also known as subsegmental, discoid, or linear atelectasis, is a very common type of fleeting atelectasis. It occurs in regions of the lung that are poorly ventilated, close to linear scars, or adjacent to subtle accessory pulmonary fissures.

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5^{th} to 6^{th} grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10^{th} to 12^{th} grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Atelectasis (The Basics)")

SUMMARY AND RECOMMENDATIONS

- Atelectasis describes the loss of lung volume due to the collapse of lung tissue. It can be classified as obstructive or nonobstructive. (See 'Introduction' above.)
- Obstructive (ie, resorptive) atelectasis results from the blockage of an airway. It is more common after obstruction of a lobar bronchus than of a segmental bronchus and it occurs more rapidly among patients who are breathing gas with a high fraction of inspired oxygen (FiO_2). (See 'Obstructive atelectasis' above.)

- Nonobstructive causes of atelectasis include loss of contact between the parietal and visceral pleurae (ie, passive atelectasis), parenchymal compression (ie, compressive atelectasis), surfactant deficiency or dysfunction (ie, adhesive atelectasis), replacement of lung tissue by scarring (ie, cicatricial atelectasis) or infiltrative disease (ie, replacement atelectasis), and strong acceleration forces in pilots and astronauts. (See 'Nonobstructive atelectasis' above.)
- Rounded atelectasis is a distinct form of atelectasis associated with pleural disease, particularly following asbestos exposure. It is discussed separately. (See "Radiologic patterns of lobar atelectasis", section on 'Rounded atelectasis'.)

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