

DISEASE OF THE RESPIRATORY SYSTEM

Emphysema

- Is characterized by permanent enlargement of the airspaces distal to the terminal bronchioles accompanied by destruction of their walls, without obvious fibrosis.
- Over inflation.
- Types of emphysema:
 1. Centriacinar - > 95%.
 2. Panacinar.
 3. Paraseptal.
 4. Irregular.

Centriacinar (centrilobular) emphysema

- Occur in heavy smoker in association with chronic bronchitis.
- The central or proximal parts of the acini are affected, while distal alveoli are spared.
- More common and severe in the upper lobes, particularly in the apical segments.
- The walls of the emphysematous space contain black pigment.
- Inflammation around bronchi and bronchioles.

Panacinar (panlobular) emphysema

- Acini are uniformly enlarged from the level of the respiratory bronchiole to the terminal blind alveoli.
- More commonly in the lower lung zones.
- Occurs in α_1 -anti-trypsin deficiency.

Distal acinar (paraseptal) emphysema

- The proximal portion of the acinus is normal but the distal part is dominantly involved.
- Occurs adjacent to areas of fibrosis, scarring or atelectasis.
- More severe in the upper half of the lungs.
- Sometimes forming cyst-like structures with spontaneous pneumothorax.

Emphysema

Incidence

- Emphysema is present in approximately 50% of adults who come to autopsy.
- Pulmonary disease was considered to be responsible for death in 6.5% of these patients.

Irregular Emphysema

- The acinus is irregularly involved, associated with scarring.
- Most common form found in autopsy.
- Asymptomatic.

Pathogenesis

- Is not completely understood.
- Alveolar wall destruction and airspace enlargement invokes excess protease or elastase activity unopposed by appropriate antiprotease regulation (protease-antiprotease hypothesis).
- α_1 -antitrypsin, normally present in serum, tissue fluids and macrophages, is a major inhibitor of proteases secreted by neutrophils during inflammation.
- Encoded by codominantly expressed genes on the proteinase inhibitor (Pi) locus on chromosome 14.
- The protease-antiprotease hypothesis explain the effect of cigarette smoking in the production of centriacinar emphysema.
 - * Smokers have accumulation of neutrophils in their alveoli.
 - * Smoking stimulates release of elastase.
 - * Smoking enhances elastase activity in macrophages, macrophage elastase is not inhibited by α_1 -antitrypsin.

Emphysema

Morphology

- The diagnosis depend largely on the macroscopic appearance of the lung.
- The lungs are pale, voluminous.
- Histologically, thinning and destruction of alveolar walls creating large airspaces.
 - * Loss of elastic tissue.
 - * Reduced radial traction on the small airways.
 - * Alveolar capillaries is diminished.
 - * Fibrosis of respiratory bronchioles.
 - * Accompanying bronchitis and bronchiolitis.

Emphysema

Clinical course

- Cough and wheezing.
- Weight loss.
- Pulmonary function tests reveal reduced FEV1.

Death from emphysema is related to:

1. Pulmonary failure with respiratory acidosis, hypoxia and coma.
2. Right-sided heart failure.

Other types of emphysema

- Compensatory emphysema.
- Senile emphysema.
- Obstructive overinflation.
- Bullous emphysema.
- Mediastinal (interstitial) emphysema.

Emphysema and Chronic Bronchitis

	Predominant Bronchitis	Predominant Emphysema
Appearance	“Blue bloaters”	“Pink Puffers”
Age	40-45	50-75
Dyspnea	Mild, late	Severe, early
Cough	Early, copious sputum	Late, scanty sputum
Infection	Common	Occasional
Respiratory Insufficiency	Repeated	Terminal
Cor pulmonale	Common	Rare, terminal
Airway resistance	Increased	Normal or slightly increased
Elastic recoil	Normal	Low
Chest radiography	Prominent vessels, large heart	Hyperinflation, small heart

Restrictive Lung Diseases

- Reduced total lung capacity, while the expiratory flow rate is normal or reduced.
- Occur in two general conditions:
 1. Chest wall disorder.
 2. Acute or chronic, interstitial and infiltrative diseases, e.g. ARDS, pneumoconiosis.

Atelectasis

- Incomplete expansion of the lungs or collapse of previously inflated lung substance.
- Significant atelectasis reduce oxygenation and predispose to infection.

Types of Atelectasis

1. Resorption atelectasis.
2. Compression atelectasis.
3. Microatelectasis (patchy atelectasis).
4. Contraction atelectasis.

Types of Atelectasis

1. Resorption atelectasis.

- Result from complete obstruction of an airway and absorption of entrapped air. Obstruction can be caused by:

a. Mucous plug (postoperatively or exudates within small bronchi seen in bronchial asthma and chronic bronchitis).

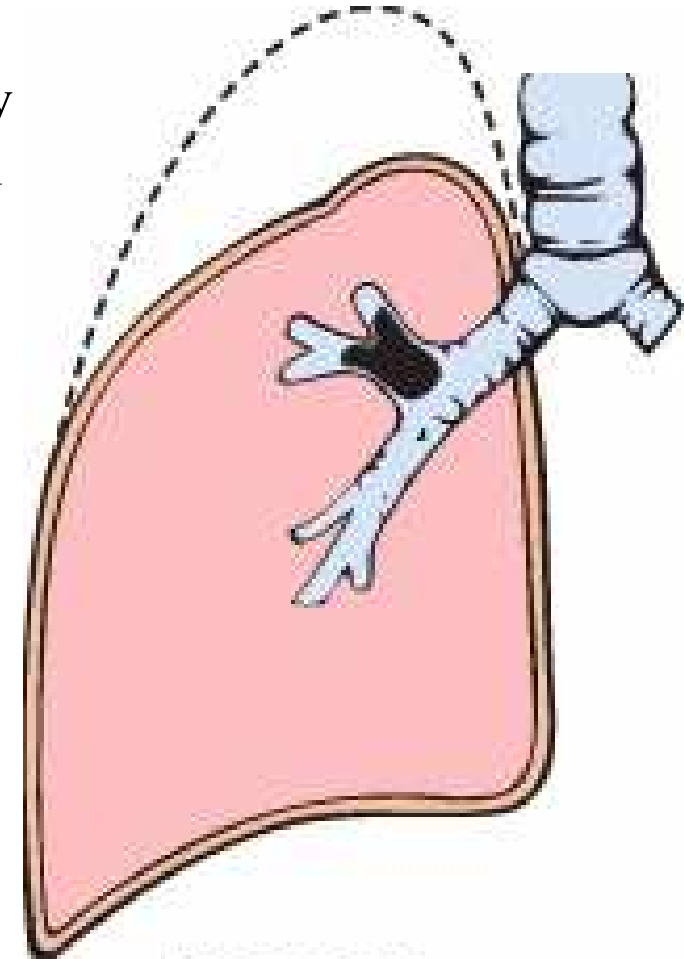
b. Aspiration of foreign body.

c. Neoplasm.

d. enlarged lymph node

- The involvement of lung depend on the level of airway obstruction.

- Lung volume is diminished and the mediastinum may shift toward the atelectatic lung.



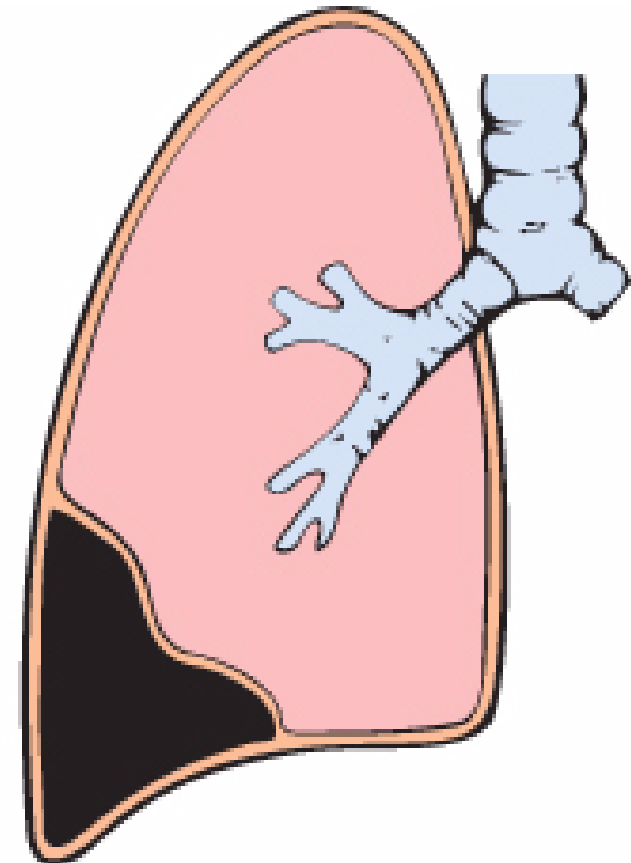
Resorption

Types of atelectasis

2. Compression atelectasis

Results whenever the pleural cavity is partially or completely filled by fluid, blood, tumor or air, e.g.

- patient with cardiac failure
- patient with neoplastic effusion
- patient with abnormal elevation of diaphragm in peritonitis or subdiaphragmatic abscess.



Compression

Types of atelectasis

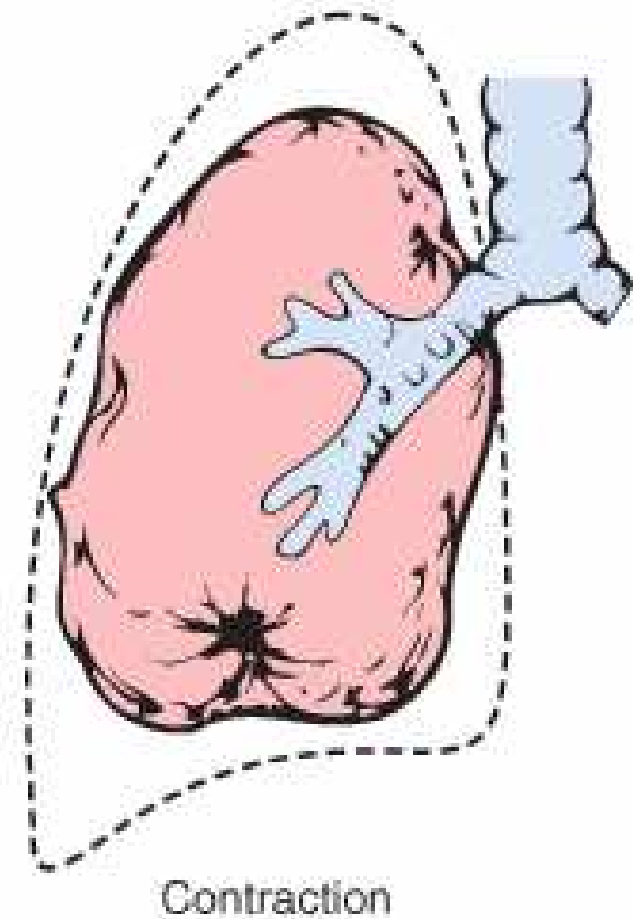
3. Microatelectasis (patchy atelectasis).

- Develops when there is loss of pulmonary surfactant.
- Occur in neonatal or adult respiratory distress syndrome, interstitial inflammation and after surgery.

Types of atelectasis

4. Contraction atelectasis.

- Local or generalized fibrotic changes in pleura or lung preventing full expansion of the lung.



Atelectasis

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- It is reversible disorder except for contraction atelectasis.

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