

Pulmonary edema

Pulmonary edema can result from hemodynamic disturbances or from direct increases in capillary permeability, as a result of microvascular injury.

The **most common** hemodynamic cause of pulmonary edema is **increased hydrostatic pressure**, as occurs in **left-sided congestive heart failure**. Whatever the clinical setting, pulmonary congestion and edema are characterized by heavy, wet lungs. Fluid accumulates initially in the basal regions of the lower lobes because hydrostatic pressure is greater in these sites.

The **second mechanism** leading to pulmonary edema is **injury to the capillaries of the alveolar septa**. The edema results from primary injury to the vascular endothelium or damage to alveolar epithelial cells.

The **early symptoms** of pulmonary edema include dyspnea, orthopnea, and coughing. **Clinical features** include tachycardia, tachypnea, dependent crackles, and neck vein distension. The treatment is designed to reduce extravascular fluid, to improve gas exchange and heart function (*i.e., oxygen, diuretics, vasopressors, positive inotropic agents and antiarrhythmics*).

Bronchiectasis is an **irreversible, abnormal dilatation** of the bronchi or bronchioles caused by destruction of their supporting structures by a chronic necrotizing infection. It is common in children with **cystic fibrosis**. Most common symptom is a chronic, productive cough with a **foul-smelling, purulent sputum**. Recurrent pulmonary infection may lead to **lung abscess**.

Note: Bronchiectasis most often involves the lower lobes of both lungs.

Atelectasis refers either to incomplete expansion of the lungs (*neonatal atelectasis* — which is common in **premature infants** due to a lack of surfactant) or to the collapse of previously inflated lung, producing areas of relatively airless pulmonary parenchyma.

Note: The causes and sequelae of left sided congestive heart failure are discussed on card #162 in this series.