## **GENERAL PATHOLOGY**

# LEC 1 DR.ASSEEL M. GHAZI

# **RESPIROTORY SYSTEM**

# INTRODECTION

The major function of the lung is to replenish oxygen and excrete carbon dioxide from blood.

Developmentally, the respiratory system is an outgrowth from the ventral wall of the foregut.

The midline trachea develops two lateral outpocketings, the lung buds. The right lung bud eventually divides into three main bronchi, and the left into two main bronchi, thus giving rise to three lobes on the right and two on the left.

The main bronchi branch giving rise to progressively smaller airways, termed **bronchioles**, which are distinguished from bronchi by the lack of cartilage and submucosal glands within their walls.

Additional branching of bronchioles leads to *terminal bronchioles*; the part of the lung distal to the terminal bronchiole is called an *acinus*.

Pulmonary acini are composed of *respiratory bronchioles* (emanating from the terminal bronchiole) that proceed into *alveolar ducts,* which immediately branch into *alveolar sacs,* the blind ends of the respiratory passages, whose walls are formed entirely of *alveoli,* the ultimate site of gas exchange.

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The microscopic structure of the alveolar walls (or alveolar septa) consists of the following components, proceeding from blood to air



• The capillary endothelium and basement membrane.

shown.

• The pulmonary interstitium is composed of fine elastic fibers, small bundles of collagen, a few fibroblast-like cells, smooth muscle cells, mast cells, and rare mononuclear cells.

It is most prominent in thicker portions of the alveolar septum.

• Alveolar epithelium contains a continuous layer of two principal cell types: flattened, platelike type I pneumocytes covering 95% of the alveolar surface and rounded type II pneumocytes.

The latter synthesize pulmonary surfactant and are the main cell type involved in repair of alveolar epithelium after damage to type I pneumocytes.

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The alveolar walls are not solid but are perforated by numerous **pores of Kohn**, which permit passage of air, bacteria, and exudates between adjacent alveoli.

• A few alveolar macrophages usually lie free within the alveolar space. In the adult, these macrophages often contain phagocytosed carbon particles.

There are multiple primary lung diseases that can broadly be divided into those primarily affecting (1) the airways, 2) the interstitium, and (3) the pulmonary vascular system.

# **ATELECTASIS (COLLAPSE)**

Atelectasis, also known as collapse, is loss of lung volume caused by *inadequate expansion of air spaces*.

It results in shunting of inadequately oxygenated blood from pulmonary arteries into veins, thus giving rise to a ventilation perfusion imbalance and hypoxia.

On the basis of the underlying mechanism or the distribution of alveolar collapse, atelectasis is classified into three forms :

• **Resorption atelectasis.** Resorption atelectasis occurs when an obstruction prevents air from reaching distal airways.

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The air already present gradually becomes absorbed, and alveolar collapse follows.

Depending on the level of airway obstruction, an entire lung, a complete lobe, or one or more segments may be involved.

The most common cause of resorption collapse is obstruction of a bronchus by a mucous or mucopurulent plug.

This frequently occurs postoperatively but also may complicate bronchial asthma, bronchiectasis, chronic bronchitis, tumor, or foreign body aspiration, particularly in children.

• **Compression atelectasis.** Compression atelectasis (sometimes called *passive* or *relaxation atelectasis*) is usually associated with accumulation of fluid, blood, or air within the pleural cavity, which mechanically collapses the adjacent lung.

This is a frequent occurrence with pleural effusion, caused most commonly by congestive heart failure (CHF).

Leakage of air into the pleural cavity (pneumothorax) also leads to compression atelectasis.

# • Contraction atelectasis.

Contraction (or *cicatrization*) atelectasis occurs when either local or generalized fibrotic changes in the lung.

Atelectasis (except when caused by contraction) is potentially reversible and should be treated promptly to prevent hypoxemia.