



Summary

- This lecture covers pulmonary capillary dynamics, focusing on the balance of Starling forces that control fluid exchange. A slight net filtration pressure normally exists, causing a small, continuous flow of fluid into the interstitial spaces, which is removed by lymphatics to keep the alveoli dry. Pulmonary edema occurs when this balance is disrupted, primarily due to increased capillary hydrostatic pressure (e.g., from left heart failure) or capillary membrane damage. The text also describes the respiratory membrane's structure, which is highly efficient for gas exchange due to its large surface area and thinness; and explains the process of arterialization of blood and the different diffusion rates of O₂ and CO₂.

Highlighted Content

1. Dynamics of Fluid Exchange (Starling Forces):

https://youtu.be/_w_73H4MHqU?si=TcnheyDhMRB5NBmv



Forces Favoring Filtration (Outward):

- Pulmonary capillary hydrostatic pressure: 7 mmHg
- Pulmonary interstitial fluid colloid osmotic pressure: 14 mmHg
- Negative interstitial fluid pressure: -8 mmHg
- Total Outward Force: +29 mmHg

Forces Favoring Absorption (Inward):

- Plasma colloid osmotic pressure: 28 mmHg
- Total Inward Force: -28 mmHg

· Net Filtration Pressure: +1 mmHg. This causes a slight, continual fluid flow into the interstitial spaces, which is removed by the pulmonary lymphatic system.

2. Mechanism of Keeping Alveoli Dry:

- The low net filtration pressure.
- Rich lymphatic drainage.
- Negative pressure in the interstitial spaces.

3. Pulmonary Edema:

- Definition: Accumulation of excess tissue fluid in the lungs; starting in the interstitial spaces and then filling the alveoli.
- Main Causes:
 - Increased pulmonary capillary hydrostatic pressure (e.g., left ventricular failure, mitral stenosis).
 - Damage to pulmonary capillary membranes (e.g., pneumonia, irritant gases).
 - Obliteration of lymph drainage (e.g., thoracic tumors).
 - Hypoproteinemia (decreases plasma colloid osmotic pressure).
 - Respiratory Distress Syndrome (RDS).

4. Pulmonary Edema Safety Factors:

- Low capillary hydrostatic pressure and high plasma colloid osmotic pressure.
- Normal negative interstitial fluid pressure.
- Rich pulmonary lymphatic drainage.

· Acute Safety Factor: Pulmonary capillary pressure must rise from 7 mmHg to over 28 mmHg to cause edema (a safety factor of 21 mmHg). A rapid rise can cause death in 20-30 minutes.

· Chronic Condition Safety Factor: Lymph vessels can expand, increasing fluid removal capability up to 10-fold, allowing tolerance of higher pressures (40-45 mmHg) without lethal edema.

5. Respiratory (Pulmonary) Membrane:

- A blood-air barrier between alveoli and capillaries.
- Surface Area: $\sim 70 \text{ m}^2$.
- Thickness: 0.2-0.6 microns.
- Favors gas exchange due to its large surface area, thinness, and high permeability to O_2 and CO_2 .

6. Gas Exchange and Arterialization:

- O_2 Diffusion: From alveolar air ($\text{PO}_2 \sim 100 \text{ mmHg}$) to venous blood ($\text{PO}_2 \sim 40 \text{ mmHg}$).
- CO_2 Diffusion: From venous blood ($\text{PCO}_2 \sim 46 \text{ mmHg}$) to alveolar air ($\text{PCO}_2 \sim 40 \text{ mmHg}$).
- After equilibration, gas pressures in pulmonary venous blood nearly equal those in alveolar air.

7. Equilibration of Gases:

- The diffusion rate of CO_2 is about 20 times faster than O_2 due to its higher solubility coefficient.
- In diseases causing alveolo-capillary block (e.g., pulmonary fibrosis), O_2 diffusion is impaired much earlier than CO_2 diffusion, leading to severe hypoxia without significant CO_2 retention.

8. Respiratory Distress Syndrome (RDS):

- Caused by deficient surfactant, leading to increased surface tension, alveolar collapse (atelectasis), and edema.
- Common in premature infants, hypothyroid infants, and infants of diabetic mothers.