Chapter 10

Ventilation
Objectives

- Describe the physiologic functions provided by ventilation.
- Describe the pressure gradients responsible for gas flow, diffusion, and lung inflation.
- Identify the forces that oppose gas movement into and out of the lungs.
- Describe how surface tension contributes to lung recoil.
- Describe how lung, chest wall, and total compliance are related.
Objectives (cont.)

- State the factors that affect resistance to breathing.
- Describe how various lung diseases affect the work of breathing.
- State why ventilation is not evenly distributed throughout the lung.
- Describe how the time constants affect alveolar filling and emptying.
Objectives (cont.)

- Identify the factors that affect alveolar ventilation.

- State how to calculate alveolar ventilation, dead space, and the $V_D/V_T$ ratio.
Introduction to Ventilation

- Five main areas will be considered:
  - Mechanics of ventilation
  - Mechanics of exhalation
  - Work of breathing (WOB)
  - Distribution of ventilation
  - Efficiency and effectiveness of ventilation
Mechanics of Ventilation

- Ventilation is cyclic: inspiration and expiration
  - Tidal volume ($V_T$): gas moved per phase
  - Facilitates removal of CO$_2$, replenishes O$_2$

- Lung and thorax compliance and resistance
  - The load the respiratory muscles overcome to produce ventilation
  - Healthy lungs at rest, inspiratory load is minimal, while expiration is passive
Pressure Differences During Breathing

- Gases move due to pressure gradients
  - Produced by thoracic expansion/contraction

- Transrespiratory pressure
  - $P_{rs} = P_{alv} - P_{(bs or ao)}$
  - This gradient causes gas flow in and out of lungs.

- Transpulmonary pressure
  - $P_L = P_{alv} - P_{pl}$
  - $P_L$ keeps alveoli open.
  - Established by opposing lung and thorax recoil
Pressure Differences During Breathing (cont.)

- Transthoracic pressure
  - \( P_w = P_{pl} - P_{bs} \)
  - \( P_w \) pressure across chest wall
  - Represents total pressure required to expand or contract the lungs and chest wall together
Pressure Differences During Breathing (cont.)
Pressure Differences During Breathing (cont.)

- At end of resting exhalation, $P_L$, normally $-5 \text{ cm } H_2O$; $P_{alv}$ is zero, period of no gas flow

- Inspiration begins.
  - Inspiratory muscles expand thorax; $\downarrow P_{Pl}$ widens $P_L$, causing alveoli to expand.
  - Alveolar expansion decreases $P_{alv}$ below zero, resulting in “negative” $P_{rs}$, so gas enters.
  - Alveolar filling slows as $P_{alv}$ approaches $P_{ao}$.
  - End-inspiration $P_{rs}$ is again zero as $P_{alv} = P_{ao}$. 
Pressure Differences During Breathing (cont.)

- **Beginning of expiration**
  - Thoracic recoil causes $P_{pl}$ to begin to rise.
  - $P_L$ declines, so alveoli begin to deflate.
  - Shrinking alveoli increase $P_{alv}$ so “positive” pressure gradient compared to $P_{ao}$ ($P_{rs}$)
  - Gas moves from alveoli to atmosphere.
  - When $P_{alv}$ falls to $P_{atm}$, expiratory flow stops.
Forces Opposing Lung Inflation

- Elastic opposition to ventilation
  - Elastic and collagen fibers provide resistance to lung stretch.
  - Application of pressure causes stretch.
  - Greater pressure causes greater stretch until maximum inflation is reached.
  - Deflation is passive recoil and less force is required to maintain same volume.
  - Hysteresis is the difference between inflation and deflation curves.
Surface Tension Forces

- Hysteresis is partly caused by surface tension.

- If surface tension is removed, the hysteresis becomes very small (see Figure 10-4).

- Lung recoil occurs due to tissue elasticity and surface tension.
Surface Tension Forces (cont.)

- Pulmonary surfactant reduces surface tension,
  - Produced in type II pneumocytes
  - Most effective as surfactant molecules draws closer together as seen on deflation
  - Surfactant stabilizes alveoli by preventing collapse
Lung Compliance

- \( C_L \) defined as change in volume (\( \Delta V \)) per change in pressure (\( \Delta P \)) or

\[
C_L = \frac{\Delta V \text{ L}}{\Delta P \text{ cm H}_2\text{O}} \quad \text{(normal 0.2 L/cm H}_2\text{O)}
\]

- Pulmonary pathology alters \( C_L \).
  - Emphysema increases \( C_L \) (loss elastic tissue).
  - Fibrosis decreases \( C_L \) (↑ elastic tissue).
  - See Figure 10-5, B.
Relationship Between Chest Wall and Lung

- Lungs and chest wall recoil in opposite directions.
  - Compliance in both is ~0.2 L/cm H₂O.
  - They each oppose the other, resulting in a system compliance of ~0.1 L/cm H₂O.
  - FRC established at resting lung level where the tendency of chest wall to expand equals that of the lungs to collapse
    - Occurs at ~40% TLC
Frictional Resistance to Ventilation

- **Tissue viscous resistance (~20% of R)**
  - Frictional forces during displacement of lungs, ribs, diaphragm, and abdominal organs

- **$R_{aw}$ (~80% of R)**
  - Gas flow causes frictional resistance
    \[ R_{aw} = \frac{P_{alv} - P_{ao}}{V \text{ L/sec}} \quad (0.5 - 2.5 \text{ cm H}_2\text{O/L/sec}) \]
  - Airway radius exponential effect ($r^4$) on $R_{aw}$
    - Artificial airway size or bronchospasm
  - $R_{aw}$ is highest at nose (50% of total $R_{aw}$) falls to ~20% of total $R_{aw}$ at the small airways
Mechanics of Exhalation

- Airway size is determined by structural support and transmural pressure ($P_{tm}$).

- Support comes from cartilage, and “traction” from surrounding tissues.
  - Small airways lack cartilage, so they are more subject to collapse.
Mechanics of Exhalation (cont.)

- During quiet breathing the $P_{tm}$ is negative, maintaining airway’s patency

- Forced exhalation can cause collapse
  - EPP: pressure inside airway = pressure outside, downstream airway compression occurs
    - May result in airway collapse
Mechanics of Exhalation (cont.)

Work of Breathing (WOB)

- Respiratory muscles perform work.
  - Resting inhalation requires work.
  - Resting exhalation is passive.
  - Forced exhalation requires work.

- Pulmonary disease can dramatically increase WOB.
  - Restrictive disease work is greater due to elastic tissue recoil.
  - Obstructive disease work is greater due to increased $R_{aw}$. 
Pathology’s Affect on WOB

(A) Normal

(B) Restrictive disease: slope of the volume-pressure curve is less, showing increased elastic work

(C) Obstructive disease: Frictional resistance increases dramatically, noted as bulging inspired and expired curves.
Metabolic Impact of Increased WOB

- Respiratory muscles consume $O_2$ to perform work.

- $O_2$ cost of breathing (OCB) is an indirect measurement of WOB.
  - Normal OCB <5% of oxygen consumption
  - In disease, OCB may increase dramatically.
    - Limits exercise tolerance
    - Impacts ability to wean from mechanical ventilation
Distribution of Ventilation

- In upright lung ventilation and perfusion (V/Q) are matched best at bases (dependent area).
  - Apical alveoli are larger but harder to ventilate compared to those at bases.
  - Gravity pulls more blood to bases.
  - In local disease, place good lung down for better V/Q matching.
Factors Affecting Distribution of Ventilation

- Regional factors that interact with gravity
  - Thoracic expansion
    - The shape of lungs and muscle action causes greater expansion at bases, so more gas flow
  - Transpulmonary pressure gradients: Directly related to $P_{pl}$, closest alveoli most affected
    - Apical $P_{pl} = 10 \text{ cm H}_2\text{O}$, while at bases $= 2.5$
  - Local CL and Raw influence local ventilation.
    - Time constants: time required for local filling
      - Time constant $= R \times C$. 

Efficiency of Ventilation

- **Efficiency**
  - Lungs should have a low OCB and produce little CO$_2$.

- **Healthy lungs waste some gas due to**
  - Anatomic deadspace of conducting airways
  - Alveoli that are ventilated but have little or no perfusion (alveolar deadspace)

\[ V_E = V_A - V_D \quad \text{or} \quad V_T = V_A - V_D \]
Minute and Alveolar Ventilation

- **Minute ventilation ($\dot{V}_E$)**: normal 5–10 L/min
  - Total volume moved in and out per minute
    \[ \dot{V}_E = RR \times V_T \]
    \[ 6 \text{ L/min} = 12 \text{ breaths/min} \times 0.5 \text{ L} \]
  - $\dot{V}_E$ driven by CO$_2$ production and subject size

- **Alveolar ventilation**
  - Amount of fresh gas reaching alveoli per minute
  - Determined by $V_T$, deadspace, and RR
    \[ \dot{V}_A = (V_T - V_D) \times RR \]
  - $\dot{V}_A$ is always less than $\dot{V}_E$ due to deadspace.
Deadspace Ventilation

- Physiologic deadspace ($V_{Dphs}$) = anatomic ($V_{Danat}$) + alveolar deadspace ($V_{Dalv}$)
  - $V_{Danat}$: volume in conducting airways
    - 1 ml/lb of IBW (2.2 ml/kg)
  - $V_{Dalv}$: alveoli receive gas but no perfusion or have ventilation that exceeds perfusion
  - Measured clinically using the Bohr equation
Bohr Equation: $V_D / V_T$ Ratio

- $V_{Dphys}$ is expressed as a ratio ($V_D / V_T$).

- Provides an index of wasted ventilation

  $$V_D / V_T = \frac{(PaCO_2 - PeCO_2)}{PaCO_2}$$

- If there were no deadspace, then $PaCO_2$ would equal $PeCO_2$. 
Bohr Equation: $V_D/V_T$ Ratio (cont.)

- Normal $V_D/V_T$ ratio is 0.2–0.4.

- $V_D/V_T$ increases with disease
  - Tough to wean from mechanical ventilation if $>0.6$
Effectiveness of Ventilation

- Ventilation is effective when CO$_2$ is removed so as to maintain normal pH

- Resting CO$_2$ production ($\dot{V}_{CO_2}$) = 200 ml/min

- CO$_2$ level determined by $V_A$ and $\dot{V}_{CO_2}$

$$PCO_2 = \frac{\dot{V}_{CO_2}}{V_A}$$
Effectiveness of Ventilation (cont.)

- Hyperventilation occurs when the PaCO₂ < 35 mm Hg
- Hypoventilation occurs when the PaCO₂ > 45 mm Hg