Oxygenation and External Respiration

Module D
Chapter 6 Malley

Readings

• Malley – Chapter 6 (Oxygen and External Respiration).

• Key Topics
  • Oxygenation
  • External Respiration
  • Ventilation
  • c / s Ratios
  • Diffusion

Hypoxemia

• Lack of oxygen in the blood
• Decreased PaO2 less than 80 mm Hg (torr)
  • Normal PaO2 is 80 – 100 mm Hg
  • Mild hypoxemia 60 – 79 mm Hg
  • Moderate hypoxemia 40 to 59 mm Hg
  • Severe hypoxemia - < 40 mm Hg
    • Malley says 45 mm Hg. Ignore.

Hypoxemia vs. Hypoxia

Is it possible to have:

• Hypoxemia without Hypoxia?
• Hypoxia without Hypoxemia?

Hypoxia

• Definition
  • Decreased oxygen at the tissue level
  • Lack of oxygen to vital organs causes necrosis or death
  • Local hypoxia
    • MI, CVA
  • Diffuse or generalized hypoxia
    • Severe hypoxemia, low cardiac output

What is the formula for O₂Del (Oxygen Delivery)?

• Oxygen Delivery = CaO₂ * CO * 10

CaO₂      CO
= [(Hb*1.34*Sao₂) + (PaO₂ * 0.003)] * (HR * SV)
Cardiopulmonary System

- When the pulmonary disease reduces the body’s ability to oxygenate the blood adequately and \( \text{Pao}_2 \) falls, the body releases certain hormones that improve cardiac function and increase oxygen delivery.
  - Oxygen Delivery is reduced secondary to reduced oxygen content.
  - Adrenergic stimulation occurs which:
    - ↑ HR (chronotropicity)
    - ↑ Force of Contraction (inotropicity)
  - This may cause heart strain and CHF
  - Weaning a patient from Mechanical Ventilator

Cardiopulmonary System

- If reduced cardiac output is resulting in tissue hypoxia (e.g., MI resulting in reduced cardiac output), pulmonary compensation may be attempted, but are less efficient in improving oxygen delivery.
  - Oxygen Delivery is decreased secondary to reduced stroke volume
  - Increase in Ventilation

Steps in Tissue Oxygenation

- **External Respiration**
  - Oxygen moves from atmosphere to the lungs.
    - Oxygen Loading
- **Oxygen Transport**
  - Transport of oxygen in sufficient volume from the lungs to the cellular destination.
    - Oxygen Delivery
- **Internal Respiration**
  - Diffusion of oxygen to the tissues in response to metabolic needs.
    - Oxygen Unloading

External Respiration

- Three criteria needed to accomplish the movement of oxygen from atmosphere to bloodstream:
  - Ventilation
  - \( \bar{V} \) / \( \bar{Q} \) Matching
  - Diffusion
Ventilation

- Normal PaCO₂ implies normal ventilation
- PaCO₂ above 45 mm Hg – hypoventilation
- PaCO₂ below 35 mm Hg – hyperventilation

- SUSPECT OXYGENATION PROBLEM IN ANY PATIENT WHO IS HYPERVENTILATING!!

Peripheral Chemoreceptors

- Stimulated by low PAO₂ and low PaO₂
  - PaO₂ less than 60 mm Hg
- Stimulated by an ↑ in H ions (↓ pH)
- Stimulation results in
  - Increased Ventilation (hyperventilation)
  - Increased HR and Blood Pressure
- Suppression of the receptors occur when the PaO₂ decreases below 30 mm Hg

Chronic Hypoxemia in COPD

- Progressive deterioration of pulmonary function leads to progressively higher PaCO₂ levels and lower PaO₂ levels.
- Administration of oxygen therapy may lead to worsening of the hypercarbia.
- Hypoxic drive vs. Worsening Mismatch
Ventilation & Perfusion

- Unilateral lung disease where all ventilation goes to the left lung.
- At the same time there is a massive PE which diverts all the blood flow to the right lung.
- What would the \( \frac{V}{Q} \) be?

West’s Zones of the Lung

Perfusion is absent

Perfusion is sporadic

Perfusion is constant

Changes in the Normal Distribution of Ventilation

- Distribution of ventilation depends
  - Compliance
  - Airway Resistance
- Air will follow the path of least resistance
- Air will flow to areas of ↑ Compliance and ↓ Raw
Distribution of Ventilation At Zero Lung Volume

- If the lungs were completely empty then during inspiration:
  - Air would enter the lung apices
  - Alveoli at the top of the lung have higher compliance and lower Raw
    - Less blood flow at the top so it would be easier to inflate

Distribution of Ventilation at FRC

- During inspiration from FRC, the alveoli are so full that further inflation would be more difficult
- Gas flows to the lower lung regions
  - Compliance is now higher at the bottom compared to the apices (easier to inflate)

Transpulmonary Pressure

- Transpulmonary Pressure (PL)
  - \( PL = P_{alv} - P_{pL} \)
  - The greater the PL the larger the alveoli and larger the alveolar filling

Changes in the FRC &/or Raw

- Changes in FRC or Raw leads to abnormal distribution of ventilation
  - ↓ Compliance (↓ FRC) or ↑ Raw → gas is shifted upward to the lung apices
**Distribution of Ventilation during Mechanical Ventilation**

- Mechanical ventilation ↑ ventilation to the upper lung zones while it ↓ perfusion to these areas (↓ venous return)
  - ↑ Physiological deadspace
  - ↑ Vd/Vt ratio to 40 – 60%

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**Abnormal Distribution of Perfusion**

- As lung volumes decrease, relatively more perfusion is distributed to nondependent lung regions.
- Result is improved V/Q.
- In the presence of a reduced PAO₂, regional blood flow will be reduced.
  - Effect of worsening hypoxemia with old generation bronchodilators.
  - Similar effect of bronchial smooth muscle in the presence of a pulmonary emboli.

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**Macroscopic V/Q Relationships**

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**Causes of Hypoxemia**

- Reduced PAO₂
  - ↓FiO₂
  - ↓PBaro
  - Hypoventilation
- V/Q Mismatch
  - Deadspace (indirectly)
  - Shunt
  - Diffusion
\( \dot{V}/\dot{Q} \) Relationships

- Normal \( \dot{V}/\dot{Q} \) relationship is
  - 4 L/min of alveolar ventilation = 0.8
  - 5 L/min of Cardiac Output
- \( \dot{V}/\dot{Q} = \dot{V}_A/\dot{Q}_{\text{T}} \)

\( \dot{V}/\dot{Q} \) Relationships

- 5 \( \dot{V}/\dot{Q} \) relationships in the lung
  - Normal \( \dot{V}/\dot{Q} \) relationship
  - High \( \dot{V}/\dot{Q} \) – Increased Physiologic \( V_d \)
  - True Capillary Shunts
  - Low \( \dot{V}/\dot{Q} \) - Relative Shunt
  - Silent Unit
Spectrum of $V/Q$ Relationships

Figure 6-1, p. 149

<table>
<thead>
<tr>
<th>Ventilation (mL)</th>
<th>Perfusion (mL)</th>
<th>V/Q</th>
<th>Unit</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>1</td>
<td>0</td>
<td>Absolute deadspace</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>1</td>
<td>Ideal unit</td>
</tr>
<tr>
<td>3</td>
<td>1</td>
<td>3</td>
<td>Relative deadspace</td>
</tr>
<tr>
<td>1</td>
<td>1</td>
<td>1</td>
<td>Ideal unit</td>
</tr>
<tr>
<td>0.5</td>
<td>1</td>
<td>0.5</td>
<td>Relative shunt</td>
</tr>
<tr>
<td>0.1</td>
<td>1</td>
<td>0.1</td>
<td>Relative shunt</td>
</tr>
<tr>
<td>0</td>
<td>10</td>
<td>0</td>
<td>Absolute shunt</td>
</tr>
<tr>
<td>0</td>
<td>0</td>
<td>0</td>
<td>Silent unit</td>
</tr>
</tbody>
</table>

Deadspace (V_d) is ventilation that does not participate in gas exchange
- Wasted Ventilation
- Energy is consumed to move gas into and out of the lungs, but no gas exchange occurs.
- In order for external respiration to occur, V_t must exceed anatomic V_d
- Rapid, shallow breathing does not facilitate gas exchange.

Deadspace Ventilation
- $V_t > V_d$ → gas exchange is occurring
- $V_t < V_d$ → no gas exchange is occurring

Types of Deadspace
- Anatomic $V_d$
- Alveolar $V_d$
  - True Alveolar $V_d$ 4/0 = infinity
  - Relative Alveolar $V_d$ 4/2 = 2
- Mechanical $V_d$
- Total $V_d$

Alveolar Deadspace
- Too much ventilation in relationship to perfusion can increase alveolar deadspace.
  - Large V_t
  - High PEEP levels (& Auto-PEEP)
- Too little perfusion in relationship to ventilation can likewise increase alveolar deadspace.
  - Pulmonary Emboli
  - Decreased CO/BP (hypovolemia, CHF)

Deadspace
- Under normal conditions, a $V_A$ of 5 L/min creates a P_{aCO_2} of 40 mmHg.
- If you double the alveolar minute ventilation to 10 L/min, you should reduce the P_{aCO_2} to 30 mmHg.
- If you quadruple the alveolar minute ventilation to 20 L/min, you should reduce the P_{aCO_2} to 20 mmHg. (20/20 Rule)
- A patient with the following relationship indicates an increase in Total $V_d$ in the lung (assuming no increased $V_CO_2$)
  - $c_A$ 20 L/min P_{aCO_2} 40 mm Hg
**V / Q Relationships**

- Artificial Airways (Tracheostomy)
  - ↓ Anatomic Vd and therefore Physiologic Vd
  - ↑ Alveolar Ventilation
  - Easier to wean from mechanical ventilation

\[ V_A = (V_t - V_d) \times f \]

**Mechanical Deadspace**

- The volume of any breathing device (ventilator or O₂ mask) in which exhaled gas remains and is inspired on the next breath.
- Extension of anatomic Vd.
- On a ventilator, it is placed between the patient and the vent wye.

**Calculation of Deadspace**

- Physiologic Vd = \( \frac{P_{aCO_2} - P_{ETCO_2}}{Paco_2} \times V_t \)
- \( V_d/V_t \) ratio = \( \frac{P_{aCO_2} - P_{ETCO_2}}{Paco_2} \)
- A widening of the \( P_{aCO_2} - P_{ETCO_2} \) gradient indicates increasing deadspace.

**How can you decrease physiologic deadspace?**

- On ventilator
  - Decrease Vt
  - Check level of PEEP
  - Eliminate Auto-PEEP
  - Increase Cardiac Output
    - Inotropic Agents
    - Give Blood
  - Treat Pulmonary Emboli
  - Trach a patient

**Pulmonary Embolism and Hypoxemia**

- Normal Alveolar Gas Exchange
  - \( PAO_2: 105 \text{ mm Hg} \)
  - \( PACO_2: 40 \text{ mm Hg} \)
- With pulmonary Embolism
  - \( PAO_2: 145 \text{ mm Hg} \)
  - \( PACO_2: 0 \text{ mm Hg} \)
- When these two alveoli mix?
  - \( PAO_2: 125 \text{ mm Hg} \)
  - \( PACO_2: 20 \text{ mm Hg} \)
- So why is their hypoxemia?

**Cause of Hypoxemia in Pulmonary Embolism**

- NOT the actual pulmonary embolism!
- When a Pulmonary Embolism occurs, blood flow has to be diverted elsewhere, causing over-perfusion of some alveoli and a ventilation-perfusion mismatch.
- Also, there are some cellular mediators released at the site of embolism (secondary to local infarction) and these mediators cause bronchial constriction, reduced ventilation, and a ventilation-perfusion mismatch.
Shunt

• Shunting: Blood that passes through to the left side of the heart without participating in gas exchange.
  • Perfusion with no ventilation (True)
  • Perfusion in excess of ventilation (Relative)
• Net effect: Blood enters and leaves the lung with identical blood gases

Types of Shunts - Anatomic

• Anatomic Shunting
  • Blood flow never “sees” alveolus
  • Normally 2-5% of cardiac output
    • Pleural, Thebesian, and Bronchial Veins
  • Congenital Cardiac Anomalies (R to L)
    • ASD
    • VSD
    • PDA

Types of Shunts – True Capillary Shunt

• No alveolar ventilation, Perfusion present:
  • LOSS OF FUNCTIONAL ALVEOLI
  • \( \dot{V}/\dot{Q} = 0 \) (zero)
• Virtually non-existent in normal humans.
• Can exist when alveoli fill with fluid
  • Pulmonary Edema
  • ARDS
  • Pneumonia
  • Atelectasis

Types of Shunts – Relative Capillary Shunt

• Volume of perfusion exceeds the volume of ventilation
  • \( \dot{Q}_v > \dot{V}_A \)
  • Some ventilation does exist
  • \( \dot{V}/\dot{Q} > 0 \), but not by much.
• Lung bases in normal lung.
• \( \dot{V}/\dot{Q} \) mismatch exists with some, limited gas exchange.
  • Increased pulmonary secretions
  • Bronchospasm
• Net effect – limited oxygen supply

Capillary Shunting

• True or Absolute shunt
  • \( \dot{V}/\dot{Q} \) ratio = 0 L/min ventilation = 0
    5 L/min perfusion

• Relative shunt (V/Q mismatch)
  • \( \dot{V}/\dot{Q} \) ratio = 2 L/min ventilation = 0.4
    5 L/min perfusion
  • \( \dot{V}/\dot{Q} \) ratio = 4 L/min ventilation = 0.4
    10 L/min perfusion

Physiologic Shunting

• The combined effect of anatomic, true, and relative capillary shunting.
• Expressed as \( \dot{Q}_{ep}/\dot{Q}_T \)
• Calculated by using the classic shunt equation.
• Normal \( \dot{Q}_{ep}/\dot{Q}_T \) is approximately 3%.
• Shunt fractions in excess of 15% can be clinically significant.
• Carbon Dioxide excretion is less of a problem than oxygenation.
Calculating Shunts #1

- **Classic** Shunt Equation
  \[ \frac{Q_{\text{sp}}}{Q_{\text{T}}} = \frac{C_{\text{CO}_2} - C_{\text{aO}_2}}{C_{\text{CO}_2} - C_{\text{vO}_2}} \times 100 \]

- Where:
  - \( C_{\text{CO}_2} = (1.34 \times Hb \times 1.0) + (PA_{\text{O}_2} \times .003) \)
  - Assumes 100% saturation in the ideal alveolus
  - Requires a Pulmonary Arterial Catheter (BTFDC)

Calculating Shunts #2

- **Clinical** Shunt Equations
  \[ \frac{Q_{\text{sp}}}{Q_{\text{T}}} = \frac{A-aDO_2 \times .003}{A-aDO_2 \times .003 + (CA_{\text{O}_2} - C_{\text{vO}_2})} \]

- Requires a Pulmonary Arterial Catheter (BTFDC)
- Only accurate at lower FIO2

Other Indices of Increased Shunting

- \( A-aDO_2 \) [P(A-a)O2]
- \( PA_{\text{O}_2}/PA_{\text{O}_2} \)
- \( PA_{\text{O}_2}/FIO_2 \) (P/F ratio)

Shunts

- All shunts cause **hypoxemia!!**
- True shunts cause refractory hypoxemia
  - \( PaO_2 \) does not respond to increasing FIO2
  - Evaluation of congenital heart defects.
- Treatment is to re-expand the collapsed alveoli
  - PEEP
  - APRV
  - Recruitment maneuvers
  - Proning

Silent Units

- Areas in the lung with no ventilation and no perfusion.
- No \( V/Q \) (Contrast with True Shunt Unit)
- \( V/Q \) ratio = 0 L/min ventilation
  - 0 L/min perfusion
- Silent units have no direct effect on external respiration but they do represent a loss of surface area for gas exchange.
Clinical Causes of $\dot{V}/\dot{Q}$ Imbalance

- **Deadspace**
  - Pulmonary Emboli
  - Excessive $V_t$
  - Excessive PEEP
  - Auto-PEEP
  - ↓ Cardiac Output

- **Shunts**
  - Atelectasis
  - Pneumonia
  - Pulmonary Edema
  - Mucous Plugging

Effective Diffusion requires:
- Time
- Surface Area

Time Available for Diffusion

- Pulmonary Capillary Transit Time.
  - Normal transit time is 0.75 seconds.
  - During exercise, this time shortens (0.34 sec).

Normally oxygen diffuses in 0.25 seconds.
- First 1/3 of capillary transit time

Actual Time or Speed of Diffusion

The speed of gas diffusion is determined by:
- Molecular Size – Molecular weight
  - Oxygen is lighter than CO2 and therefore diffuses better in a gaseous phase.
- Solubility Coefficient – In a liquid environment, however, the ability to dissolve is important
  - CO2 diffuses 20 times faster than oxygen across the AC membrane.
  - Solubility Coefficients: CO2 0.510 mL and O2 is 0.023 mL
- Driving Pressure
  - $O_2$: 62-63 mm Hg
  - $CO_2$: 6-8 mm Hg

Fick’s Law of Diffusion

$$\text{Diffusion} = A D (P_1 - P_2)$$

- **A** = Surface Area
- **D** = Diffusion Constant
- **P1-P2** = Pressure Gradient
- **T** = Thickness of membrane
Diffusion Barriers

- Thickening of AC membrane
- Decreased Driving Pressure (low PAO₂ e.g. high altitude)
- Decreased Surface Area

Thickening of the AC Membrane

- Normal thickness is 1 μ (1/1000 mm).
- Increased in pulmonary fibrosis or pulmonary edema.
- Doubling the thickness would double the equilibration time.
- Not a significant problem unless cardiac output increases.
- Decrease capillary transit time.

Decreased Driving Pressure

- High Altitude
  - Low PBRÖ, Low PIO₂, Low PAO₂
  - Exemplified by patient experiencing dyspnea with mild exertion.

Incomplete Equilibration

- Increased thickening of AC membrane or a decreased driving pressure will result in a diffusion defect when combined with a decreased capillary transit time.
  - This is seen when cardiac output increases.
    - Exercise and early Septic Shock & Hypoxemia!

Surface Area

- Surface area of the lung is 70 – 85 m²
  - Tennis Court
- Diseases where surface area is reduced include:
  - Lobectomy
  - Pneumonectomy
  - Tumors
  - Emphysema with bleb/bullae

Clinical Complaint

- First Symptoms of Pulmonary Fibrosis, Emphysema or exacerbation of CHF
  - SOB and hypoxemia on exertion
    - DOE
    - $SpO_2$ with exercise
Detecting Abnormalities in Diffusion

- Pulmonary Function Lab
- $D_{LCO}$