Pulmonary “Tests”

“What a Loyola MS 3 should know about Oxygenation, CO₂ elimination, and PFT’s”

Learning Objectives

- **Oxygenation:**
  - Distinguish the various mechanisms of hypoxia
  - Know how to calculate the A-a Gradient
  - Understand oxygen content, delivery, and extraction
  - Recognize the various oxygen delivery devices

- **CO₂ Elimination:**
  - Know the principles determining one’s CO₂
  - Understand the concept of Dead Space Ventilation

- **PFT’s:**
  - Be able to interpret PFT’s recognizing Obstruction, Restriction, and Diffusion Impairments

Approach to Hypoxemia

- **Disease-Based**
  - COPD/Asthma
  - Pulmonary Edema
  - ARDS
  - Pneumonia
  - ILD
  - Hypoventilation
  - Altitude
  - Decreased FIO2
  - Cirrhosis
  - Pulmonary Embolism

- **Mechanism-Based**
  - VQ Mismatch
  - Shunt
  - Diffusion Impairment
  - Hypoventilation
  - Decreased Barometric Pressure
  - Decreased FIO₂
  - Diffusion-Perfusion Impairment
  - Mixed

Mechanisms of Hypoxia: VQ Mismatch

- Decreased V relative to Q
- O₂ exits alveolus more quickly than enters via bronchi
- Hypoxia is MILD
- Hypoxia improves with supplemental O₂
- Causes:
  - Asthma, COPD
  - Pulmonary Emboli
  - ILD

Mechanisms of Hypoxia: Shunt

- No O₂ reaches some set of pulmonary capillaries
- Hypoxia is SEVERE
- Hypoxia does NOT improve with supplemental O₂
- Causes:
  - Pulmonary Shunt:
    - NO ventilation to alveoli that are still perfused
  - PFO
  - ASD
  - VSD
  - Pulmonary Emboli
  - ARDS
  - Pulmonary AVM
  - Cardiac Shunt

Normal Physiology

- No obstruction
- No alveolar filling process
- No diffusion barrier
- Ventilation roughly equals Perfusion
- More of both at the bases
- Less of both at the apices
- O₂ from the bronchus enters the alveolus as rapidly as O₂ leaves into the pulmonary capillaries/systemic circulation

\[ P_{mvO₂} = 40 \text{ mmHg} \quad P_{aO₂} = 100 \text{ mm Hg} \]
Mechanisms of Hypoxia:

Shunt

- No O$_2$ reaches some set of pulmonary capillaries
- Hypoxia is SEVERE
- Hypoxia does NOT improve with supplemental O$_2$

Causes:
- Pulmonary Shunt:
  - NO ventilation to alveoli that are still perfused
  - Blood
  - Pus
  - Water
  - Pulmonary Edema
  - ARDS
  - Atelectasis
  - Pulmonary AVM
- Cardiac Shunt
  - PFO, ASD, VSD

Mechanisms of Hypoxia:

Diffusion Impairment

- NOT a common problem
  - Blood is normally fully oxygenated within 25% of its transit through the alveolar capillaries.
  - Therefore, even if slowed by a diffusion barrier, blood usually reaches full saturation
- Hypoxia is MILD
- Hypoxia improves with supplemental O$_2$

Mechanisms of Hypoxia:

Diffusion-Perfusion Impairment

- Seen occasionally in cirrhosis
- Dilated capillaries pose an impairment to full oxygenation

Mechanisms of Hypoxia

- VQ Mismatch
- Shunt
- Diffusion Impairment
- Diffusion-Perfusion Impairment
- Hypoventilation
- Altitude
- Decreased F$_2$O$_2$

The A-a Gradient
### Two Questions

1. Which of these people has a lower than expected \( P_{\text{O}_2} \)?
   - A. A MS3 in SSOM with a \( P_{\text{O}_2} = 95 \)
   - B. 72 yo Doc Hering in SSOM with a \( P_{\text{O}_2} = 80 \)
   - C. 50 yo Dr. Michelfelder in flight with a \( P_{\text{O}_2} = 50 \)
   - D. A MS3 running at top speed with a \( P_{\text{O}_2} = 70 \)

2. Which ABG illustrates abnormal \( O_2 \) Transfer from Alveolus to Capillary?
   - A. \( \text{PaCO}_2 = 40 \), \( \text{PaO}_2 = 95 \)
   - B. \( \text{PaCO}_2 = 60 \), \( \text{PaO}_2 = 70 \)
   - C. \( \text{PaCO}_2 = 20 \), \( \text{PaO}_2 = 95 \)

Write your answers down...

### The A – a Gradient

1. Mathematically = \( \text{P}_{\text{AlvO}_2} - \text{P}_{\text{aO}_2} \)

   Why is there any gradient?
   - Normal Anatomic and Physiologic Shunting
   - The A-a is normally less than age/4 + 4
   - A higher A-a gradient implies “disease” decreasing the efficiency of oxygen transfer from the atmosphere to the arterial circulation

2. Answers the question:
   - Is your patient’s \( \text{PaO}_2 \) ‘normal’?

### Under “normal” circumstances….

… breathing room air at sea level

A “normal” Alveolar \( O_2 \) is:

\[
\text{AlvO}_2 = \left( (\text{PB} - \text{P}_{\text{H}_2\text{O}}) \times \text{FIO}_2 \right) - \left( \text{PaCO}_2 / \text{RQ} \right)
\]

\[
\text{PaO}_2 \text{ from ABG = 150 - (40/0.8)} = 59 \text{ mmHg}
\]

Therefore, \( \text{P}_{\text{AlvO}_2} \) normally* = 150 - 50 = 100

*But … \( \text{P}_{\text{B}}, \text{FIO}_2, \text{PaCO}_2, \) and \( \text{RQ} \) can all be manipulated

### The Answers:

1. Which of these people has a lower than expected \( P_{\text{O}_2} \)?
   - A. A MS3 in SSOM with a \( P_{\text{O}_2} = 95 \)
   - B. 72 yo Doc Hering in SSOM with a \( P_{\text{O}_2} = 80 \)
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Patient A is simply what we expect
Patient B is simply hypo-ventilating
Patient C is has SIGNIFICANTLY abnormal oxygen transfer despite an overly normal \( \text{PaO}_2 \)!!!
Clinical Question
- Treatment for *pneumocystis jiroveci* pneumonia in a patient whose ABG is 7.48/30/70 on room air?

How to describe the “degree” of hypoxia
- The “P/F” Ratio
  - $P_{a}O_2/FIO_2$
  - Normally...
    - $P_{a}O_2/FIO_2 \times 100/0.2 = 500$
  - Lower P/F Ratios imply worsening degrees of hypoxia
    - $P/F < 200$ is bad enough hypoxia to count as ARDS

Other Oxygen Issues:
- How many mL of $O_2$ are in each dL of:
  - arterial blood?
  - venous blood?
- How much many mL of $O_2$ are delivered per minute to the tissues?
- What percent of the delivered $O_2$ is extracted by the tissues at rest?
- How are these numbers useful clinically?

Oxygen Content
- Conceptually:
  - Oxygen is carried in the blood as both:
    - Hemoglobin-Bound Oxygen
    - Dissolved Oxygen

Mathematically:
- $C_{a}O_2 = (Hgb)(S_{O_2})(1.34) + (P_{a}O_2)(0.003)$
- $C_{v}O_2 = (15)(1)(1.34) + (95)(0.003)$
  - $\approx 20$ mL $O_2$/dL Blood
- $C_{mv}O_2 = (15)(0.75)(1.34) + (40)(0.003)$
  - $\approx 15$ mL $O_2$/dL blood
- $D_{a-v}O_2 = C_{a}O_2 - C_{mv}O_2$
  - $= 20 - 15 = 5$ mL $O_2$/dL blood
  - i.e., the difference in $O_2$ content between arterial and venous blood

Oxygen Delivery
- Conceptually:
  - The amount of oxygen delivered to the tissues is the product of cardiac output and oxygen content.

Mathematically:
- $D_{a}O_2 = C.O. \times C_{a}O_2$
  - $= 5$ Lpm $\times 20$ mL $O_2$/dL (x 10 dL/L)
  - $= 1000$ mL $O_2$/min
Oxygen Extraction

- \( \text{VO}_2 = \text{Oxygen Consumption} \)
  - Normal = 250 cc/min at rest
- Extraction Ratio
  - % of delivered oxygen actually consumed
    - At rest:
      - 250 cc/min consumed
      - 1000 cc/min delivered
      - ER = 25%
      - Can increase to 75%

Oxygen Content, Delivery, Extraction: Summary

- Evidence of Inadequate Delivery relative to Consumption:
  - \( \dot{\text{CmvO}}_2 \)
  - \( \dot{\text{DAvO}}_2 \)
  - \( \dot{\text{ER}} \)

Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% \( \text{F}_O_2 \)
  - \( \text{F}_O_2 \) increases ~ 3% for each additional liter per minute
- Simple Face Mask
  - 40-60% \( \text{F}_O_2 \)
- Non-Rebreather Mask
  - "reservoir" with one-way valve
  - 60-100% \( \text{F}_O_2 \)
- Venturi Mask
  - Includes a valve allowing "precise" \( \text{F}_O_2 \) delivery
    - ? Advantage for COPD patients
    - 24-60% \( \text{F}_O_2 \)
Oxygen Delivery Devices

- Optiflow®
  - "Nasal High Flow Oxygen"
  - Heated and Humidified
  - "Flushes" out dead space
  - Provides a tiny amount of CPAP
  - Up to 100% FIO₂

Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% FIO₂
- Simple Face Mask
  - 40–60% FIO₂
- Non-Rebreather Mask
  - 60-100% FIO₂
- Venturi Mask
  - 24-60% FIO₂
- Nasal HF O₂
  - Up to 100% FIO₂

What about PₐCO₂?

Conceptually:
- PₐCO₂ depends upon how much CO₂ is produced vs how much is eliminated.
- CO₂ elimination depends upon Alveolar Ventilation.
  - i.e., Total Ventilation minus Wasted Ventilation

Hence, the determinants of PₐCO₂ are:
- CO₂ Production
- Total Minute Ventilation
- Wasted Ventilation (i.e., "dead space" or VD/VT)

Mathematically...

\[ PₐCO₂ \propto \frac{VCO₂}{MV \times (1 - VD/VT)} \]

- VCO₂ = CO₂ Production
  - Normal = 200 ml/min
  - Increases in VCO₂ are not a clinically relevant cause of hypercapnea
- MV = Minute Ventilation
  - Normal = 5 lpm at rest
  - Up to 100 lpm at maximum aerobic activity
  - Obviously, hypoventilation leads to hypercapnea
- Therefore, if there is no increased VCO₂ or decreased MV, hypercapnea must be due to increased VD/VT

Dead Space?

\[ PₐCO₂ \propto \frac{VCO₂}{MV \times (1 - VD/VT)} \]

- VD/VT = "Dead Space" Ventilation
  - i.e., the percent of each tidal volume which does NOT participate in gas exchange
  - Includes 'anatomic' dead space
  - i.e., the air in the trachea and bronchi down to the conducting airways
  - And includes physiologic dead space
  - i.e., air in alveoli that nonetheless is not participating in gas exchange
- Three Questions:
  - How much dead space is normal?
  - What are causes of increased dead space?
  - What is the consequence of increased dead space?

Normal:
- VT ≥ 500 cc
- VO ≥ 1 cc/pound ≥ 150cc
- VD/VT ≥ 150/500 ≥ 30% of an average TV
VD/VT

Causes of increased VD/VT:
- Decreased Perfusion of Ventilated Lung:
  - Pulmonary Emboli
  - Pulmonary Hypertension
  - Volume Depletion
- Increased Alveolar Pressures:
  - PEEP (mechanical ventilation)
  - auto-PEEP (emphysema)

Why does it matter?
- If increased VD/VT, one must increase minute ventilation which increases work of breathing.
- Think of increased VD/VT, whenever:
  - Increased P_aCO_2
  - And/OR
  - Normal P_aCO_2 with increased MV

PFT’s – practically speaking....
- Calculate expected values:
  - Age
  - Height
  - Sex
  - Race
- Measure patient values
- Compare
  - “normal” is defined by measured values that are between 80% and 120% of the predicted values

PFT’s: 3 Main Components
- Spirometry
  - FEV_1/FVC
  - Asthma
  - COPD
  - Bronchiectasis
- Lung Volumes
  - TLC
  - Interstitial Disease
  - Chest Wall Disease
  - Neuromuscular Disease
- Diffusing Capacity
  - DLCO
  - Pulmonary HTN
  - Associated with COPD and/or ILD
  - Isolated = Primary Pulmonary HTN