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 ?

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

\[ P_{mvO_2} = 40 \text{ mmHg} \quad P_sO_2 = 100 \text{ mm Hg} \]
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

**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
      - 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 O₂
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 $F_1O_2$
The A-a Gradient

Why?

Two Questions

1. Which of these people has a lower than expected $P_aO_2$?
   A. A MS3 in SSOM with a $P_aO_2 = 95$
   B. 70 yo Doc Hering in SSOM with a $P_aO_2 = 80$
   C. 50 yo very athletic lung doc in an airplane with a $P_aO_2 = 50$
   D. A MS3 running at top speed with a $P_aO_2 = 70$

2. Which ABG illustrates abnormal $O_2$ Transfer from Alveolus to Capillary?

<table>
<thead>
<tr>
<th>$PaCO_2$</th>
<th>$PaO_2$</th>
</tr>
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<tbody>
<tr>
<td>A. 40</td>
<td>95</td>
</tr>
<tr>
<td>B. 60</td>
<td>70</td>
</tr>
<tr>
<td>C. 20</td>
<td>95</td>
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Write your answers down…
The A-a Gradient

Why?
To answer the question:
- Is the measured PaO2 what it should be?

What should the measured PaO2 be?
- That Depends….
  - On a lot of things:
    - Age
    - Barometric pressure
    - FIO2
    - \( p_aCO_2 \)
    - RQ
  - i.e., the components of the A-a Gradient equation
A-a Gradient

- Assesses the efficiency of oxygen transfer from the atmosphere to the arterial circulation.
  - In health, O₂ from the atmosphere moves to the alveoli and then efficiently crosses into the pulmonary capillaries.
    - i.e., there is only a small A-a Gradient.
    - Why any gradient?
      - Physiologic Shunt that increases with age
      - Normal = (Age/4) + 4
  - When the A-a Gradient is greater than normal, then – and only then – is there a barrier to O₂ transfer.

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A-a Gradient

- We calculate what the Alveolar O₂ ought to be
- We measure the arterial PO₂
- The Difference is the A-a Gradient.

- So, how do we calculate what the Alveolar O₂ ought to be.....
1. What is the normal $pO_2$ in the atmosphere?

- **Atmospheric Gases**
  - $P_B = 760$ torr at sea level
  - Composition:
    - $O_2 = 21\%$
    - $N_2 = 79\%$
    - Everything else is so trivial as to be measured in PPB
  - Thus, the $P_{atm}O_2 = P_B \times F_{O2} = 760 \times .21 = 160$
Atmospheric gas is humidified as it traverses the pharynx, trachea, and bronchial tree:
- At body temperature at sea level, fully humidified air has a $P_{H_2O} = 47$ torr
- Thus, the $pO_2$ of the air reaching the alveoli is:

$$pO_2 = (P_B - P_{H_2O}) \times F_{I O_2}$$
$$= (760 - 47) \times .21$$
$$= 150$$

2. What is the normal $pO_2$ entering the alveolus?

3. What is the normal $pO_2$ actually in the alveolus available to oxygenate the venous blood?

Finally, alveolar gas has CO$_2$ added and O$_2$ and removed.

Thus, Alveolar O$_2$ is:

$$P_{AlvO_2} = [(P_B - P_{H2O}) \times F_{I O_2}] - (P_{a CO_2}/RQ)$$
$$[(760 - 47) \times .21] - (P_{a CO_2}/RQ)$$
$$150 - (P_{a CO_2}/RQ)$$

“normally” $P_{a CO_2}/RQ = 40/0.8 = 50$

Therefore, $P_{AlvO_2}$ normally* = 150 - 50 = 100

*For people breathing room air at sea level

....$P_B$, $F_{I O_2}$, $P_{a CO_2}$, and RQ can all be manipulated
The A-a Gradient Formula:

- Conceptually:
  - What is the $O_2$ gradient between an ‘ideal’ alveolus and the pulmonary capillaries

- Mathematically:
  - $P_{Alv}O_2 - P_{a}O_2$
  - $\left\{ \left[ (P_B - P_{H2O}) \times F_{O2} \right] - (P_{a}CO_2/RQ) \right\} - P_{a}O_2$

- = 150 if sea level and room air

- $P_{a}CO_2$ from ABG; RQ = 0.8

- $P_{a}O_2$ from ABG
1. Which of these people has a lower than expected PaO₂?

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@ sea level, on room air, normal CO₂ and RQ means Alveolar PO₂ should be @ 100 mm Hg

- A-a = 100-95 = 5... normal
- A-a = 100-80 = 20
  - Age/4 + 4 = 21.5..... Normal
- At 8000 feet, PB is only 565
  - (565-47) * 0.21 – (40/0.8) = 59
  - 59-50 = 9 Normal
- To repeat, normal people don’t desaturate... ABNORMAL

Two Questions

2. Which ABG illustrates abnormal O₂ Transfer from Alveolus to Capillary?

PaCO₂  PaO₂  A-a  Barrier?
A.  150-40/0.8 = 100  95  5  NO
B.  150-60/0.8 =  75  70  5  NO
C.  150-20/0.8 = 125  95 30  YES

Patient A is simply what we expect
Patient B is simply hypoventilating
Patient C is has SIGNIFICANTLY abnormal oxygen transfer despite an overtly normal PaO₂!!!
Clinical Question

- Treatment for pneumocystis pneumonia in a patient whose ABG is 7.48/30/70?

How to describe the “degree” of hypoxia

- The “P/F” Ratio
  - $P_a O_2/F_i O_2$
  - Normally…
    - $P_a O_2/F_i O_2 = 100/0.2 = 500$
  - Lower P/F Ratios imply worsening degrees of hypoxia
    - P/F < 200 is bad enough hypoxia to count as ARDS
Other Oxygen Issues:

- **Oxygen Content:**
  - i.e., How many mL of O₂ are in each dL of
    - arterial blood?
    - venous blood?

- **Oxygen Delivery:**
  - How many mL of O₂ are delivered per minute to the tissues?

- **Oxygen Extraction:**
  - What percent of the delivered O₂ 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
### Oxygen Content

- Mathematically:
  - \(C_xO_2 = (Hgb)(S_xO_2)(1.34) + (P_xO_2)(0.003)\)
  - \(C_aO_2 = (15)(1)(1.34) + (95)(0.003)\)
    \(\approx 20 \text{ mL } O_2/\text{dL Blood}\)
  - \(C_{mv}O_2 = (15)(0.75)(1.34) + (40)(0.003)\)
    \(\approx 15 \text{ mL } O_2/\text{dL blood}\)
  - \(D_{a-v}O_2 = C_aO_2 - C_{mv}O_2\)
    \(= 20 - 15 = 5 \text{ mL } O_2/\text{dL blood}\)
  - 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:
  - \(D_aO_2 = C.O. \times C_aO_2\)
    \(= 5 \text{ Lpm} \times 20 \text{ mL } O_2/\text{dL} \times 10 \text{ dL/L}\)
    \(= 1000 \text{ mL } O_2/\text{min}\)
Oxygen Extraction

- VO$_2$ = 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:
  - ↓$C_{mv}O_2$
  - ↑$D_{a-v}O_2$
  - ↑ ER
Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% $F_iO_2$
  - ? $F_iO_2$ per liter

- Simple Face Mask
  - 40 –60% $F_iO_2$
Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% FiO2
- Simple Face Mask
  - 40 –60% FiO2
- Non-Rebreather Mask
  - “reservoir” with one-way valve
  - 60-100% FiO2

Oxygen Delivery Devices

- Venturi Mask
  - Includes a valve allowing precise FiO2 delivery (Advantage for COPD patients)
  - 24-40% FiO2
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What about CO₂?

*Conceptually…*

- PaCO₂ is determined by how much CO₂ is produced vs how much is eliminated.
  - and CO₂ elimination depends upon Alveolar Minute Ventilation.
    - and Alveolar Minute Ventilation is Total Minute Ventilation minus Wasted Ventilation
- Hence, the determinants of PaCO₂ are:
  - CO₂ Production
  - Total Minute Ventilation
  - Wasted Ventilation (i.e., “dead space”)
**CO₂: Mathematically…**

- \( \text{PaCO}_2 \propto \frac{\text{VCO}_2}{[\text{MV} \times (1 - \frac{\text{V}_D}{\text{V}_T})]} \)
  - \( \text{VCO}_2 = \text{CO}_2 \text{ Production} \)
    - Normal = 200 ml/min
    - Increases in \( \text{VCO}_2 \) are not a clinically relevant cause of hypercapnea
  - \( \text{MV} = \text{Minute Ventilation} \)
    - Normal = 5 Lpm at rest
    - Up to 100 Lpm at maximum aerobic activity
    - Obviously, hypoventilation leads to hypercapnea
  - Therefore, if there is no increased \( \text{VCO}_2 \) or decreased \( \text{MV} \), hypercapnea must be due to increased \( \text{V}_D/\text{V}_T \)

**Dead Space?**

- \( \text{PaCO}_2 \propto \frac{\text{VCO}_2}{[\text{MV} \times (1 - \frac{\text{V}_D}{\text{V}_T})]} \)
  - \( \frac{\text{V}_D}{\text{V}_T} \) = “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?
$V_D/V_T$

- Normally:
  - $V_T \approx 500 \text{ cc}$
  - $V_D \approx 1 \text{ cc/pound} \approx 150 \text{ cc}$
  - $V_D/V_T \approx 150/500 \approx 30\%$ of an average TV
- $V_D/V_T$ increases when there is no perfusion to ventilated alveoli. Either due to:
  - Abnormally High Alveolar Pressures
    - i.e., Zone 1 of the Lung in which alveolar pressures exceed the pulmonary vascular perfusion pressures
  - Reduced Perfusion to the Alveoli
    - Volume Depletion
    - Pulmonary Hypertension
    - Pulmonary Embolism

**Causes of $V_D/V_T$**

- Increased Alveolar Pressures
  - i.e. PEEP
- Decreased Perfusion due to Volume Depletion or Pulmonary HTN
- Decreased Perfusion due to PE
VD/VT

Why does it matter?

- If increased $V_D/V_T$, one must increase minute ventilation which increases work of breathing.
- Think of Increased $V_D/V_T$, whenever:
  - Increased PaCO₂
  - AND/OR
  - Normal PaCO₂ 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**
  - ↓FEV₁/FVC
    - Obstruction
  - Asthma
  - COPD
  - Bronchiectasis

- **Lung Volumes**
  - ↓TLC
    - Restriction
  - Interstitial Disease
  - Chest Wall Disease
  - Neuromuscular Disease

- **Diffusing Capacity**
  - ↓DLCO
    - Pulmonary HTN
  - Associated with COPD and/or ILD
  - Isolated = Primary Pulmonary HTN

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**Normal**

**Positive Methacholine Challenge**

**Obstruction**

**Restriction**
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