Pulmonary “Tests”

“What a Loyola MS 3 should know about Oxygenation, CO$_2$ 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
- **CO$_2$ Elimination:**
  - Know the principles determining one’s CO$_2$
  - 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

\[ P_{mvO_2} = 40 \text{ mmHg} \quad P_{aO_2} = 100 \text{ mm Hg} \]
MIGET Analysis

Not on a test!!!! Just how we know things....

FIGURE 1. Examples of V/Q distribution in patient with normal scan (A) and in patient with confirmed PE (B). Histogram shapes characteristically demonstrate single peak in 0.5–1.2 range in patient with normal scan and bimodal distribution in low (<0.5) and high (>1.2) ranges in PE patient.
Mechanisms of Hypoxia:

VQ Mismatch

- Decreased \( V \) relative to \( Q \)
- \( O_2 \) exits alveolus more quickly than enters via bronchi
- Hypoxia is MILD
- Hypoxia improves with supplemental \( O_2 \)
- Causes:
  - Asthma, COPD
  - Pulmonary Emboli
  -ILD

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

\[ P_{mvO_2} = 40 \quad P_{O_2} = 40 \]
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
- Hypoventilation
- Altitude
- Decreased $F_iO_2$
- Diffusion Impairment
- Diffusion-Perfusion Impairment
What is a normal $p_aO_2$?

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 Myles Sheehan flying to Vietnam 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>
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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>
</tr>
</tbody>
</table>

Write your answers down…
What is a normal $p_aO_2$?

- Depends….
- On a lot of things:
  - Age
  - Barometric pressure
  - $F_iO_2$
  - $p_aCO_2$
  - RQ

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2. Which ABG illustrates abnormal O$_2$ Transfer from Alveolus to Capillary?
   - A. 40 95
   - B. 60 70
   - C. 20 95

1, 2, and 3 are all NORMAL.

In health, no one desaturates even at peak exercise intensity.

If this is obvious, take the next 10 minutes off….. Otherwise, calculate the A-a gradient
The real question isn’t “What is a normal PaO₂?”

Rather, the real question is:

- “Is the measured PaO₂ lower than it “should” be?
  - The “A-a Gradient” answers this question

The A-a Gradient:

- Assesses the efficiency of oxygen transfer from the atmosphere to the arteries.
- Normally, 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.
  - When the A-a Gradient is greater than normal, then – and only then – is there a barrier to O₂ transfer.

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We *measure* the arterial PO₂

We *calculate* what the Alveolar O₂ ought to be

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 $P_{atm}O_2 = P_B \times F_{I}O_2 = 760 \times 0.21 = 160$
Atmospheric gas is humidified as it traverses the pharynx, trachea, and bronchial tree:

- At body temperature at sea level, fully humidified air has a $P_{H2O} = 47$ torr
- Thus, the $pO_2$ of the air reaching the alveoli is:
  \[
  pO_2 = (P_B - P_{H2O}) \times FIO_2
  \]
  \[
  = (760 - 47) \times .21
  \]
  \[
  = 150
  \]

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, in addition to $O_2$, $H_2O$, and $N_2$, alveolar gas has $CO_2$ added in exchange for $O_2$

Thus, Alveolar $O_2$ is:

\[
P_{AlvO_2} = [(P_B - P_{H2O}) \times FIO_2] - (P_{aCO_2}/RQ)
\]
\[
= [(760 - 47) \times .21] - (P_{aCO_2}/RQ)
\]
\[
= 150 - (P_{aCO_2}/RQ)
\]

"normally" $P_{aCO_2}/RQ = 40/0.8 = 50$

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

*For people breathing room air at sea level

* $P_B$, $FIO_2$, $P_{aCO_2}$, 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:
  - $P_{Alv}O_2 - P_{a}O_2$
  - $\{(P_B - P_{H2O}) \times F_iO_2\} - (P_{a}CO_2/RQ) - P_{a}O_2$

= 150 if sea level and room air

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

$P_{a}O_2$ from ABG
What is a ‘normal’ A-a gradient?

- Why is there a gradient in normal people?
  - Physiologic shunt
  - Increases with age
- How much?
  - Normally, the oxygen gradient between alveolus and artery is:
    \[ (\text{Age}/4) + 4 \]

The Answers:

1. Which of these people has a lower than expected \( \text{P}_{a\text{O}_2} \)?
   - A. A MS3 in SSOM with a \( \text{P}_{a\text{O}_2} \) = 95
   - B. 70 yo Doc Hering in SSOM with a \( \text{P}_{a\text{O}_2} \) = 80
   - C. 50 yo Myles Sheehan flying to Vietnam with a \( \text{P}_{a\text{O}_2} \) = 50
   - D. A MS3 running at top speed with a \( \text{P}_{a\text{O}_2} \) = 70

@ 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
  - \( \text{Age}/4 + 4 = 20 \)… Normal
- At 8000 feet, PB is only 565
  - \( (565-47) \times 0.21 - (40/0.8) = 59 \)
  - 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?

<table>
<thead>
<tr>
<th>P_{alv}O₂</th>
<th>A-a</th>
<th>Barrier?</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. 100</td>
<td>5</td>
<td>NO</td>
</tr>
<tr>
<td>B. 75</td>
<td>5</td>
<td>NO</td>
</tr>
<tr>
<td>C. 125</td>
<td>30</td>
<td>YES</td>
</tr>
</tbody>
</table>

PaCO₂, PaO₂

A. 40, 95
B. 60, 70
C. 20, 95

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

Clinical Question

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

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

*Steroids are recommended if the pO2 is less than 70 or the A-a Gradient is > 35.

Here the A-a gradient:
- \[ \text{A-a gradient} = [150 - \frac{\text{PaCO}_2}{\text{RQ}}] - \text{PaO}_2 \]
- \[ = [150 - 30/0.8] - 70 \]
- \[ = [150 - 37.5] - 70 \]
- \[ = 112.5 - 70 \]
- \[ = 42.5 \]

How to describe the “degree” of hypoxia

- The “P/F” Ratio
  - \( \frac{\text{P}_a\text{O}_2}{\text{F}_1\text{O}_2} \)
  - Normally…
    - \( \frac{\text{P}_a\text{O}_2}{\text{F}_1\text{O}_2} \approx 100/0.2 = 500 \)
  - 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
**Oxygen Content**

- **Mathematically:**
  - \( C_{xO_2} = (Hgb)(S_xO_2)(1.34) + (P_xO_2)(0.003) \)
  - \( C_{aO_2} = (15)(1)(1.34) + (95)(0.003) \) 
    \( \approx 20 \text{ mL O}_2/\text{dL Blood} \)
  - \( C_{mvO_2} = (15)(0.75)(1.34) + (40)(0.003) \) 
    \( \approx 15 \text{ mL O}_2/\text{dL blood} \)
  - \( D_{a-vO_2} = C_{aO_2} - C_{mvO_2} \)
    \( = 20 - 15 = 5 \text{ mL O}_2/\text{dL blood} \)
    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

- VO₂ = 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:
  - ⬇️ CₘᵥO₂
  - ⬆️ Dₐ₋ᵥO₂
  - ⬆️ 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
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
- Venturi Mask
  - Includes a valve allowing precise FiO2 delivery (advantage for COPD patients)
  - 24-40% FiO2

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…**

- \( \text{PaCO}_2 \propto \frac{\text{VCO}_2}{[\text{MV} \times (1 - \frac{V_D}{V_T})]} \)
  - \( \text{VCO}_2 = \text{CO}_2 \text{ Production} \)
    - 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 \( \frac{V_D}{V_T} \)

**Dead Space?**

\( \text{PaCO}_2 \propto \frac{\text{VCO}_2}{[\text{MV} \times (1 - \frac{V_D}{V_T})]} \)

- \( \frac{V_D}{V_T} = \text{“Dead Space” Ventilation} \)
  - 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?
\[ \frac{V_D}{V_T} \]

**Normally:**
- \( V_T \approx 500 \text{ cc} \)
- \( V_D \approx 1 \text{ cc/pound} \approx 150 \text{ cc} \)
- \( \frac{V_D}{V_T} \approx 150/500 \approx 30\% \) of an average TV

**\( \frac{V_D}{V_T} \) 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 Embolism
  - Pulmonary Hypertension

**Causes of \( \uparrow \frac{V_D}{V_T} \)**
- 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 PaCO$_2$
  - AND/OR
  - Normal PaCO$_2$ with increased MV

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**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

<table>
<thead>
<tr>
<th>Spirometry</th>
<th>Lung Volumes</th>
<th>Diffusing Capacity</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓FEV₁/FVC</td>
<td>↓TLC</td>
<td>↓DLCO</td>
</tr>
<tr>
<td>Obstruction</td>
<td>Restriction</td>
<td>Pulmonary HTN</td>
</tr>
</tbody>
</table>

- Asthma
- COPD
- Bronchiectasis
- Interstitial Disease
- Chest Wall Disease
- Neuromuscular Disease

Associated with COPD and/or ILD
Isolated = Primary Pulmonary HTN

Normal vs Positive Methacholine Challenge

Obstruction vs Restriction
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