Pulmonary “Tests”

“What a Loyola MS 3 should know about Oxygenation, CO_2 elimination, and PFT’s”

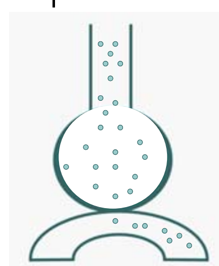
Learning Objectives

- 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 F_iO_2
  - Diffusion-Perfusion Impairment
  - Mixed ?

Normal Physiology



$P_{mv}O_2$ = 40 mmHg P_aO_2 = 100 mm Hg

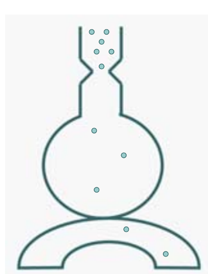
•No obstruction
•No alveolar filling process
•No diffusion barrier

•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

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

$P_{mv}O_2$ = 40 P_aO_2 = 40

Mechanisms of Hypoxia: Shunt

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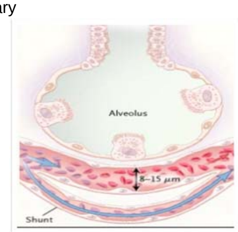
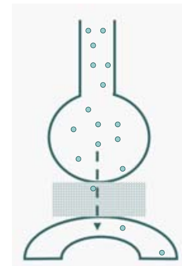
Pulmonary AVM, Cardiac Shunt (PFO, ASD, VSD) → Anatomic Shunts

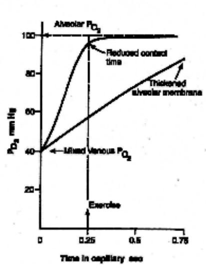


Mechanisms of Hypoxia: Diffusion Impairment



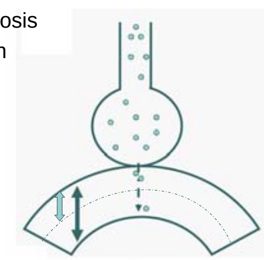
Mechanisms of Hypoxia: Diffusion Impairment

- 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_IO_2

The A-a Gradient

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. 72 yo Doc Hering in SSOM with a P_aO_2 = 80
C. 50 yo Dr. Michelfelder in flight with a P_aO_2 = 50
D. A MS3 running at top speed with a P_aO_2 = 70

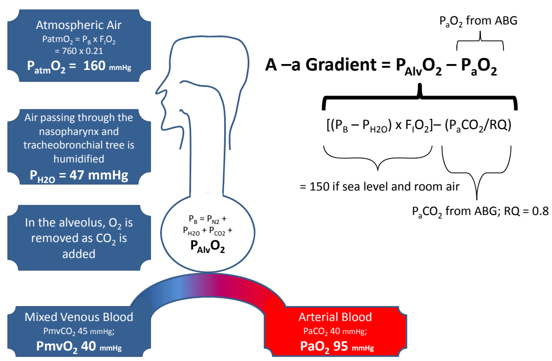
2. Which ABG illustrates abnormal O_2 Transfer from Alveolus to Capillary?

	$PaCO_2$	PaO_2
A.	40	95
B.	60	70
C.	20	95

Write your answers down…

The A – a Gradient

1. Mathematically = $P_{Alv}O_2 - P_aO_2$ (calculated – measured)
   - 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 PaO2 'normal'?



Under "normal" circumstances….

… breathing room air at sea level

A "normal" Alveolar O_2 is:

$$P_{Alv}O_2 = [(P_B - P_{H2O}) \times F_IO_2] - (P_aCO_2/RQ)$$
$$[(760 - 47) \times .21] - (P_aCO_2/RQ)$$
$$150 - (40/0.8)$$

Therefore, $P_{Alv}O_2$ normally* = 150 - 50 = **<u>100</u>**

**But ….P_B, F_IO_2, P_aCO_2, and RQ can all be manipulated*

The Answers:

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@ sea level, on room air, normal CO_2 and RQ means $P_{Alv}O_2$ should be @ 100 mm Hg

- A-a = 100-95 = 5… normal
- A-a = 100-80 = 20
  - Age/4 + 4 = 22….. Normal
- At 8000 feet, P_B 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

@ sea level, on room air, with a normal RQ, $P_{Alv}O_2 = 150 - PaCO_2/0.8$

	$P_{alv}O_2$	A-a	Barrier?
A.	100	5	NO
B.	75	5	NO
C.	125	30	YES

Patient A is simply what we expect

Patient B is simply hypo-ventilating

Patient C is has SIGNIFICANTLY abnormal oxygen transfer despite an overtly normal PaO_2!!!

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

	$PaCO_2$	PaO_2
A.	40	95
B.	60	70
C.	20	95

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_aO_2/F_IO_2
  - Normally…
    - $P_aO_2/F_IO_2 \cong 100/0.2 = 500$
  - 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 = $(15)(1)(1.34) + (95)(0.003)$
  $\cong 20$ mL O_2/dL Blood
- $C_{mv}O_2$ = $(15)(\underline{0.75})(1.34) + (\underline{40})(0.003)$
  $\cong 15$ mL O_2/dL blood
- $D_{a\text{-}v}O_2$ = $C_aO_2 - C_{mv}O_2$
  = 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_aO_2 = C.O. x C_aO_2
    = 5 Lpm x 20 mL O_2/dL (x 10 dL/L)
    = 1000 mL O_2/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:
  - ⇩ $C_{mv}O_2$
  - ⇧ $D_{a\text{-}v}O_2$
  - ⇧ ER

Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% F_IO_2

  F_IO_2 increases ~ 3% for each additional liter per minute

Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% F_IO_2
- Simple Face Mask
  - 40-60% F_IO_2

Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% F_IO_2
- Simple Face Mask
  - 40-60% F_IO_2
- Non-Rebreather Mask
  - "reservoir" with one-way valve
  - 60-100% F_IO_2

Oxygen Delivery Devices

- Venturi Mask
  - Includes a valve allowing "precise" F_IO_2 delivery
    - ? Advantage for COPD patients
  - 24-60% F_IO_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% F_IO_2

Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% F_IO_2
- Simple Face Mask
  - 40 –60% F_IO_2
- Non-Rebreather Mask
  - 60-100% F_IO_2
- Venturi Mask
  - 24-60% F_IO_2
- Nasal HF O2
  - Up to 100% F_IO_2

What about P_aCO_2?

Conceptually:

- P_aCO_2 depends upon how much CO_2 is produced vs how much is eliminated.
- CO_2 elimination depends upon Alveolar Ventilation.
  - i.e., Total Ventilation minus Wasted Ventilation

Hence, the determinants of P_aCO_2 are:

- CO_2 Production
- Total Minute Ventilation
- Wasted Ventilation (i.e., "dead space" or V_D/V_T)

P_aCO_2

Mathematically…

- $P_aCO_2 \propto VCO_2 / [MV \times (1 - V_D/V_T)]$
  - VCO_2 = CO_2 Production
    - Normal = 200 ml/min
    - Increases in VCO_2 are <u>not</u> 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 V_D/V_T

Dead Space?

$P_aCO_2 \propto VCO_2 / [MV \times (1 - V_D/V_T)]$

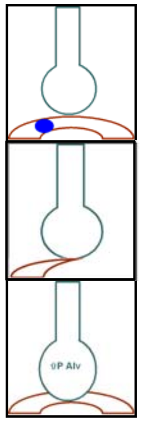
- V_D/V_T = "Dead Space" Ventilation
  - i.e., the percent of each tidal volume which does NOT participate in gas exchange
    - Includes 'anatomic' dead space
      - i.e., the air in the trachea and bronchi down to the conducting airways
    - AND includes physiologic dead space
      - i.e., air in 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 ≅ 500 cc
  - V_D ≅ 1 cc/pound ≅ 150cc
  - V_D/V_T ≅ 150/500 ≅ 30% of an average TV

V_D/V_T

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



V_D/V_T 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 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	Lung Volumes	Diffusing Capacity
↓	↓	↓
↓FEV_1/FVC Obstruction	**↓TLC Restriction**	**↓DLCO Pulmonary HTN**
↓	↓	↓
Asthma COPD Bronchiectasis	Interstitial Disease Chest Wall Disease Neuromuscular Disease	Associated with COPD and/or ILD Isolated = Primary Pulmonary HTN