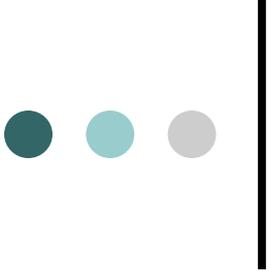


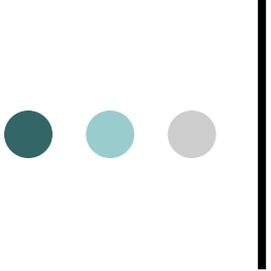
Pulmonary “Tests”

“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

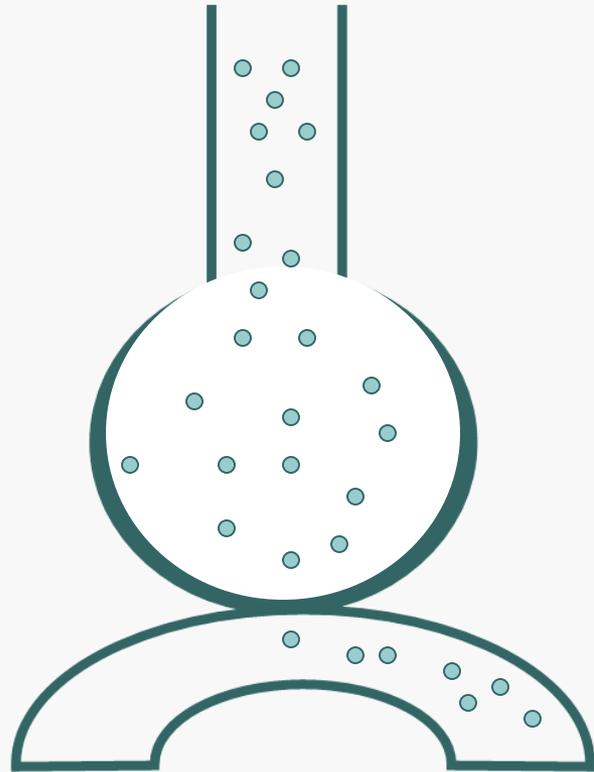
○ Disease-Based

- 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 F_IO₂
- Diffusion-Perfusion Impairment
- Mixed ?

Normal Physiology



$P_{mv}O_2 = 40 \text{ mmHg}$

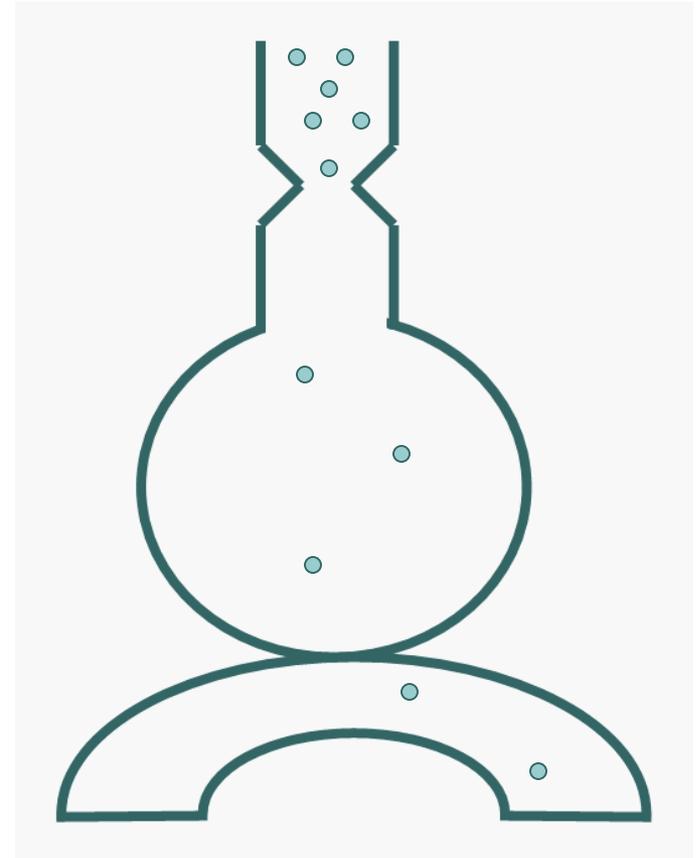
$P_aO_2 = 100 \text{ mm Hg}$

Important Principles:

- Ventilation = Perfusion throughout
 - More of both at the bases
 - Less of both at the apices
- In health, no matter how low the $P_{mv}O_2$ may be, P_aO_2 will be normal

Mechanisms of Hypoxia: VQ Mismatch

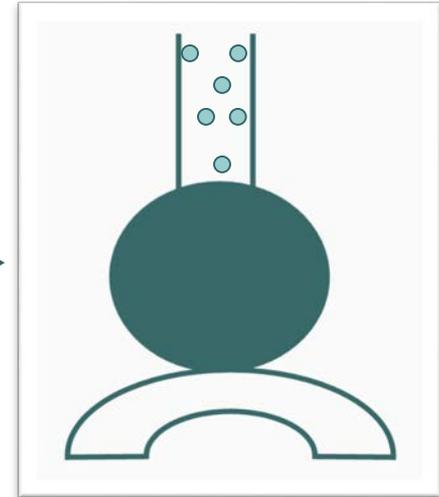
- Decreased V relative to Q
- O_2 exits alveolus more quickly than enters via bronchi
- Hypoxia is MILD
- Hypoxia improves with supplemental O_2
- Causes:
 - Asthma, COPD
 - Pulmonary Emboli
 - ILD



Mechanisms of Hypoxia:

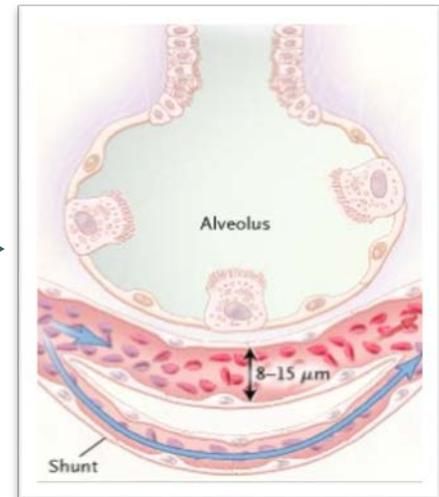
Shunt

- No O_2 reaches some set of pulmonary capillaries
- Hypoxia is SEVERE
- Hypoxia does NOT improve with supplemental O_2
- Causes:
 - Alveolar Disease:
 - NO ventilation to alveoli that are still perfused
 - Blood
 - Pus
 - Water
 - Pulmonary Edema
 - ARDS
 - Atelectasis
 - Anatomic Shunt
 - Pulmonary AVM
 - PFO, ASD, VSD

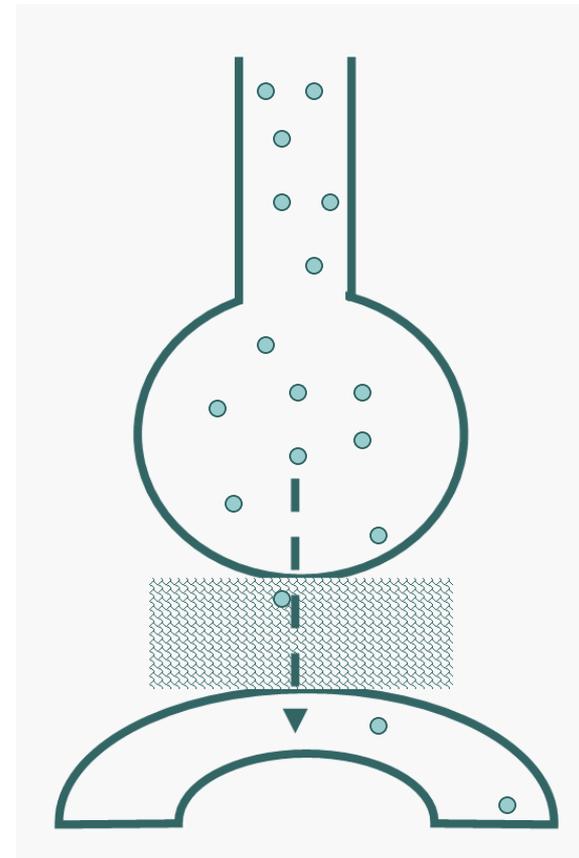


$P_{mv}O_2 = 40$

$P_aO_2 = 40$

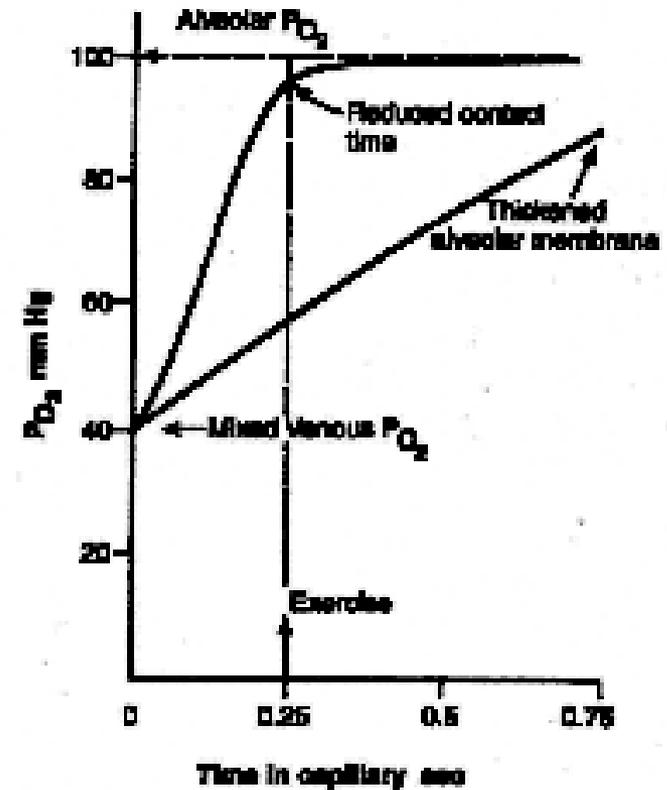


Mechanisms of Hypoxia: Diffusion Impairment



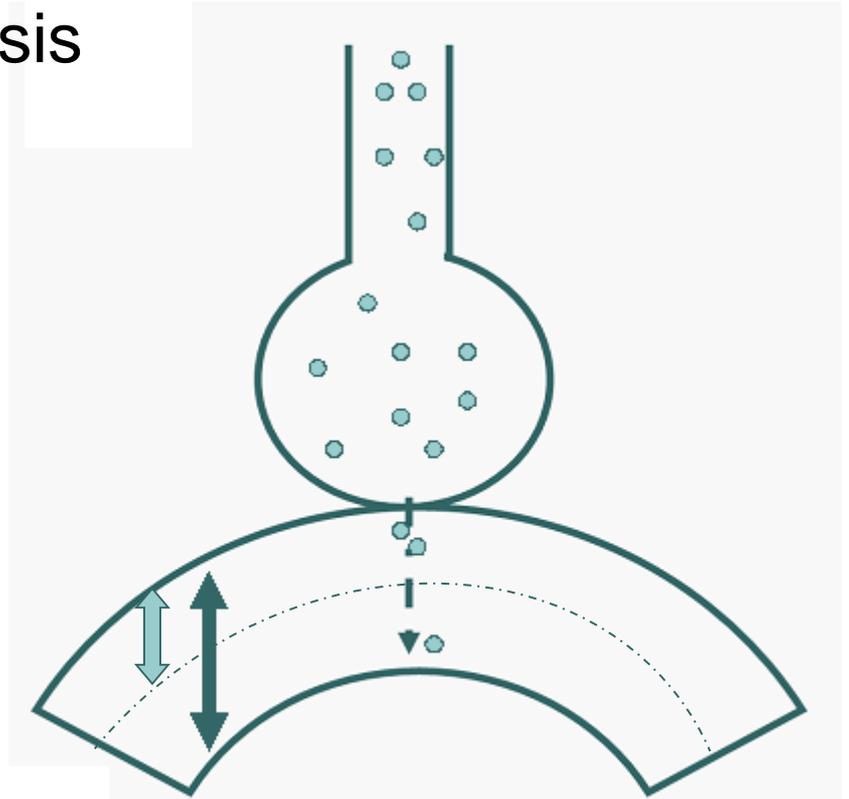
Mechanisms of Hypoxia: Diffusion Impairment

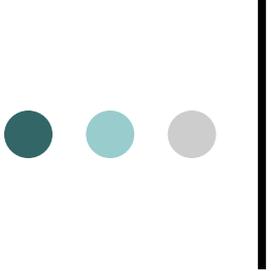
- NOT a common problem
 - Blood is normally fully oxygenated within 25% of its transit through the alveolar capillaries.
 - Therefore, even if slowed by a diffusion barrier, blood usually reaches full saturation
- Hypoxia is MILD
- Hypoxia improves with supplemental O_2



Mechanisms of Hypoxia: Diffusion-Perfusion Impairment

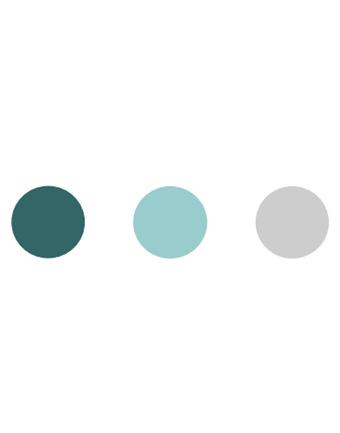
- Seen occasionally in cirrhosis
- Dilated capillaries pose an impairment to full oxygenation





Mechanisms of Hypoxia

- VQ Mismatch
- Shunt
- Diffusion Impairment
- Diffusion-Perfusion Impairment
- Hypoventilation
- Altitude
- Decreased $F_I O_2$



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?

	<u>$PaCO_2$</u>	<u>PaO_2</u>
A.	40	95
B.	60	70
C.	20	95

Write your answers down...

The A – a Gradient

- Mathematically = $P_{\text{Alv}}^{\text{calculated}} \text{O}_2 - P_{\text{a}}^{\text{measured}} \text{O}_2$
 - Why is there any gradient?
 - Normal Anatomic and Physiologic Shunting
 - What is a normal gradient?
 - The A-a is normally less than $\text{age}/4 + 4$
 - What does an elevated A-a gradient imply?
 - A higher A-a gradient implies “disease” decreasing the efficiency of oxygen transfer from the atmosphere to the arterial circulation
- Answers the question “Is your patient’s PaO_2 ‘normal’?”

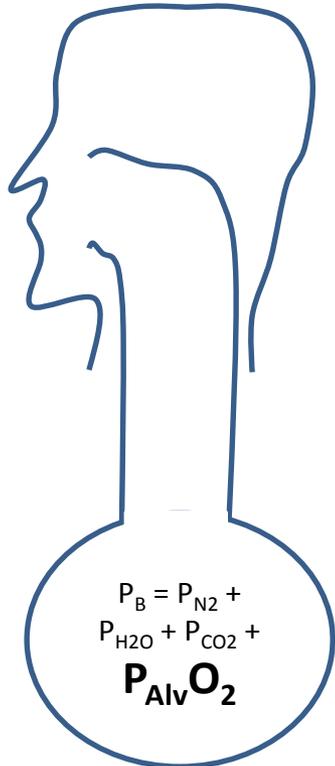
*What PaO_2
would you be
happy with?*

Atmospheric Air
 $P_{atm}O_2 = P_B \times F_{I}O_2$
 $= 760 \times 0.21$
 $P_{atm}O_2 = 160$ mmHg

Air passing through the nasopharynx and tracheobronchial tree is humidified
 $P_{H_2O} = 47$ mmHg

In the alveolus, O_2 is removed as CO_2 is added

Mixed Venous Blood
 $P_{mv}CO_2$ 45 mmHg;
 $P_{mv}O_2$ 40 mmHg



P_aO_2 from ABG

$$A - a \text{ Gradient} = P_{Alv}O_2 - P_aO_2$$

$$= [(P_B - P_{H_2O}) \times F_{I}O_2] - (P_aCO_2 / RQ)$$

= 150 if sea level and room air

P_aCO_2 from ABG; RQ = 0.8

Arterial Blood
 $PaCO_2$ 40 mmHg;
 PaO_2 95 mmHg

Under “normal” circumstances....

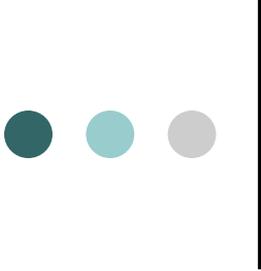
... *breathing room air at sea level*

A “normal” Alveolar O₂ is:

$$P_{Alv}O_2 = \frac{[(P_B - P_{H_2O}) \times F_I O_2] - (P_a CO_2 / RQ)}{150 - (40 / 0.8)}$$

Therefore, P_{Alv}O₂ normally* = 150 - 50 = **100**

**ButP_B, F_IO₂, P_aCO₂, and RQ can all be manipulated*

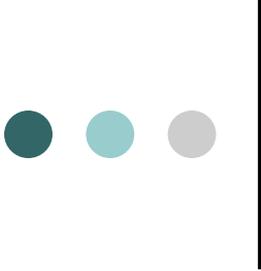


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- C. 50 yo Dr. Michelfelder in flight with a $P_aO_2 = 50$
- D. A MS3 running at top speed with a $P_aO_2 = 70$

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

- $A-a = 100-95 = 5 \dots$ Normal
- $A-a = 100-80 = 20$
 - $Age/4 + 4 = 22 \dots$ 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**



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

	<u>PaCO₂</u>	<u>PaO₂</u>
A.	40	95
B.	60	70
C.	20	95

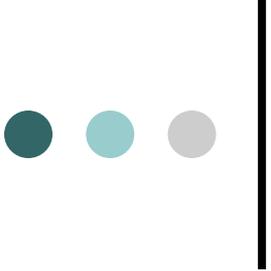
@ sea level, on room air, with a normal RQ,
 $P_{\text{Alv}}\text{O}_2 = 150 - \text{PaCO}_2/0.8$

<u>P_{alv}O₂</u>	<u>A-a</u>	<u>Barrier?</u>
A. 100	5	NO
B. 75	5	NO
C. 125	30	YES

Patient A is simply what we expect

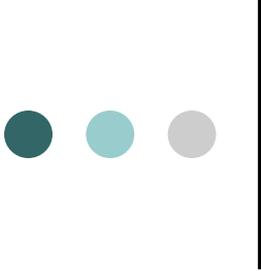
Patient B is simply hypo-ventilating

Patient C is has SIGNIFICANTLY abnormal oxygen transfer despite an overtly normal PaO₂!!!



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

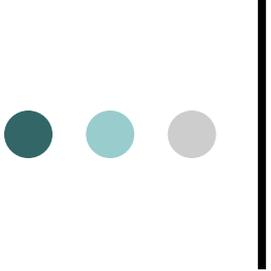
- $P_aO_2/F_I O_2$

- Normally...

- $P_aO_2/F_I O_2 \cong 100/0.2 = 500$

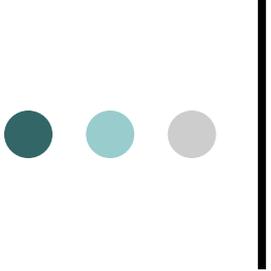
- Lower P/F Ratios imply worsening degrees of hypoxia

- P/F < 300 is bad enough hypoxia to count as ARDS



Other Oxygen Issues:

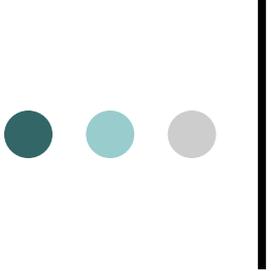
- How many mL of O_2 are in each dL of:
 - arterial blood?
 - venous blood?
- How much many mL of O_2 are delivered per minute to the tissues?
- What percent of the delivered O_2 is extracted by the tissues at rest?
- How are these numbers useful clinically?



Oxygen Content

- Conceptually:

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



Oxygen Content

Mathematically:

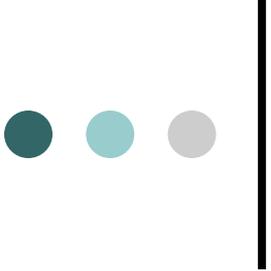
- $C_xO_2 = (\text{Hgb})(S_xO_2)(1.34) + (P_xO_2)(0.003)$

- $C_aO_2 = (15)(1)(1.34) + (95)(0.003)$
 $\cong 20 \text{ mL } O_2/\text{dL Blood}$

- $C_{mv}O_2 = (15)(\underline{0.75})(1.34) + (\underline{40})(0.003)$
 $\cong 15 \text{ mL } O_2/\text{dL blood}$

- $D_{a-v}O_2 = C_aO_2 - C_{mv}O_2$
 $= 20 - 15 = 5 \text{ mL } O_2/\text{dL blood}$

i.e., the difference in O_2 content between arterial and venous blood



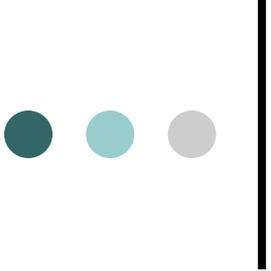
Oxygen Delivery

- Conceptually:

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

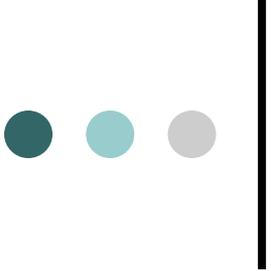
- Mathematically:

- $D_aO_2 = C.O. \times C_aO_2$
= 5 Lpm x 20 mL O₂/dL (x 10 dL/L)
= 1000 mL O₂/min



Oxygen Extraction

- $\dot{V}O_2$ = 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:
 - $\downarrow C_{mv}O_2$
 - $\uparrow D_{a-v}O_2$
 - $\uparrow ER$

Oxygen Delivery Devices

- Nasal Cannula
 - 24-44% $F_{I}O_2$
 $F_{I}O_2$ increases ~ 3%
for each additional
liter per minute



Oxygen Delivery Devices

- Nasal Cannula
 - 24-44% $F_{I}O_2$
- Simple Face Mask
 - 40-60% $F_{I}O_2$

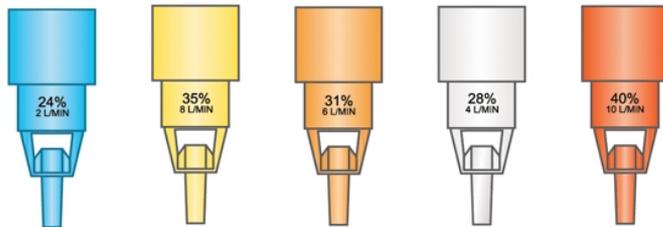
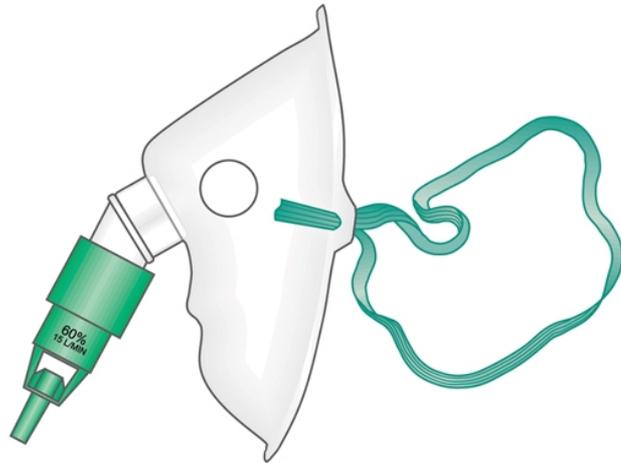


Oxygen Delivery Devices

- Nasal Cannula
 - 24-44% $F_{I}O_2$
- Simple Face Mask
 - 40-60% $F_{I}O_2$
- Non-Rebreather Mask
 - “reservoir” with one-way valve
 - 60-100% $F_{I}O_2$



Oxygen Delivery Devices



Interchangeable Venturi Valves

- Venturi Mask
 - Includes a valve allowing “precise” $F_I O_2$ delivery
 - ? Advantage for COPD patients
 - 24-60% $F_I O_2$

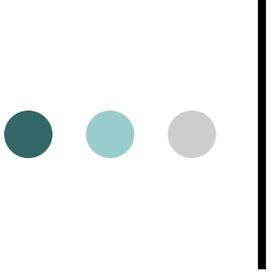
Oxygen Delivery Devices



○ Optiflow[®]

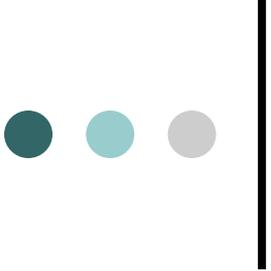
“Nasal High Flow Oxygen”

- Heated and Humidified
- “Flushes” out dead space
- Provides a tiny amount of CPAP
- Up to 100% $F_{I}O_2$



Oxygen Delivery Devices

- Nasal Cannula
 - 24-44% F_1O_2
- Simple Face Mask
 - 40 –60% F_1O_2
- Non-Rebreather Mask
 - 60-100% F_1O_2
- Venturi Mask
 - 24-60% F_1O_2
- Nasal HF O2
 - Up to 100% F_1O_2



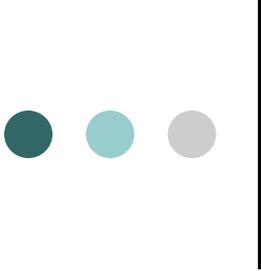
What about $P_a\text{CO}_2$?

Conceptually:

- $P_a\text{CO}_2$ 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 $P_a\text{CO}_2$ are:

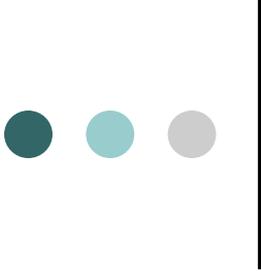
- CO_2 Production
- Total Minute Ventilation
- Wasted Ventilation (i.e., “dead space” or V_D/V_T)



$P_a\text{CO}_2$

Mathematically...

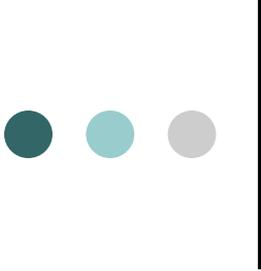
- $P_a\text{CO}_2 \propto \text{VCO}_2 / [\text{MV} \times (1 - \text{V}_D/\text{V}_T)]$
 - $\text{VCO}_2 = \text{CO}_2$ Production
 - Normal = 200 ml/min
 - Increases in VCO_2 are not a clinically relevant cause of hypercapnea
 - MV = Minute Ventilation
 - Normal = 5 lpm at rest
 - Up to 100 lpm at maximum aerobic activity
 - Obviously, hypoventilation leads to hypercapnea
 - Therefore, if there is no increased VCO_2 or decreased MV, hypercapnea must be due to increased V_D/V_T



Dead Space?

$$P_a\text{CO}_2 \propto V\text{CO}_2 / [\text{MV} \times (1 - V_D/V_T)]$$

- 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:

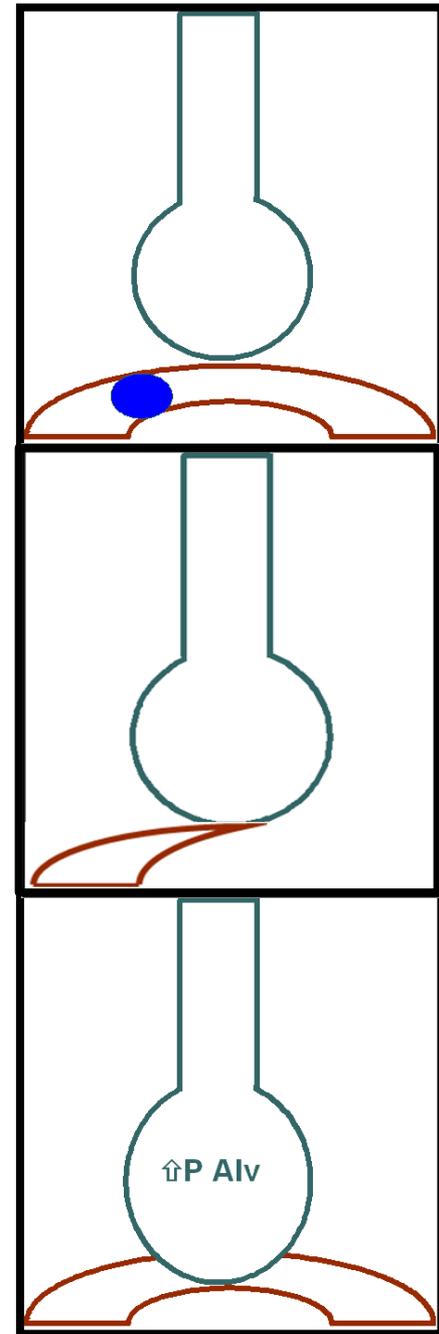
● $V_T \cong 500 \text{ cc}$

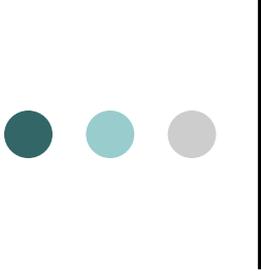
● $V_D \cong 1 \text{ cc/pound} \cong 150 \text{ cc}$

● $V_D/V_T \cong 150/500 \cong 30\% \text{ of an average TV}$

● ● ● | V_D/V_T

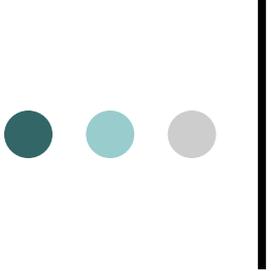
- Causes of increased V_D/V_T :
 - Decreased Perfusion of Ventilated Lung:
 - Pulmonary Emboli
 - Pulmonary Hypertension
 - Volume Depletion
 - Increased Alveolar Pressures:
 - PEEP (mechanical ventilation)
 - auto-PEEP (emphysema)




$$V_D/V_T$$

Why does it matter?

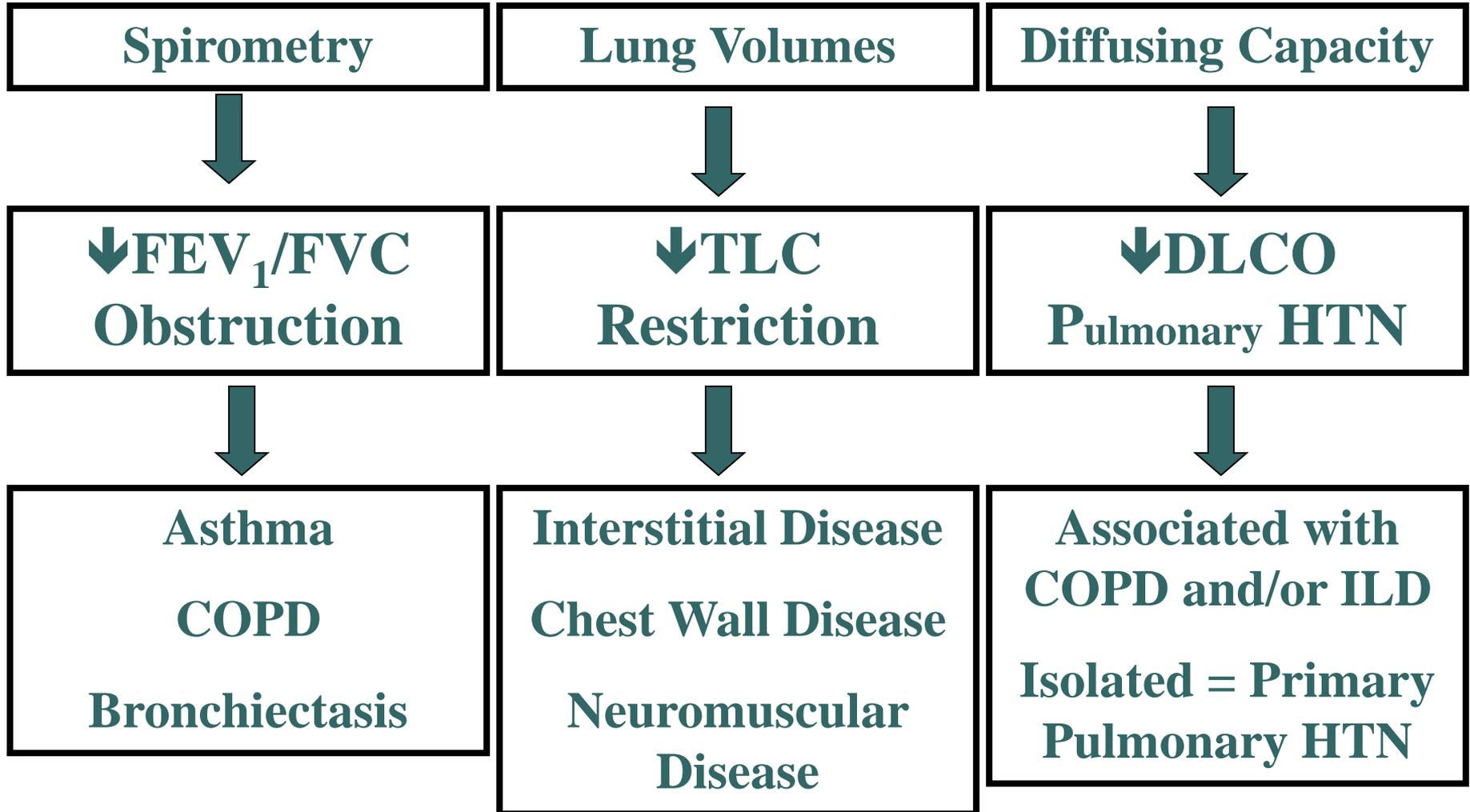
- If increased V_D/V_T , one must increase minute ventilation which increases work of breathing.
- Think of increased V_D/V_T , whenever:
 - Increased $P_a\text{CO}_2$
AND/OR
 - Normal $P_a\text{CO}_2$ with increased MV

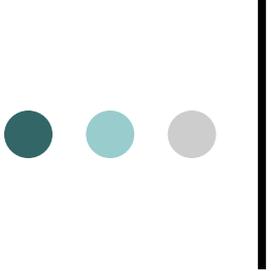


PFT's – practically speaking....

- 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





Learning Objectives

- Oxygenation:
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 - Know how to calculate the A-a Gradient
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 - Recognize the various oxygen delivery devices
- CO₂ Elimination:
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 - Understand the concept of Dead Space Ventilation
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