Pulmonary "Tests"

"What a Loyola MS 3 should know about Oxygenation, CO_2 elimination, and PFT's"

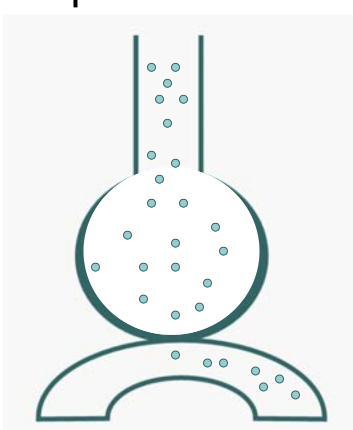
Learning Objectives

- 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



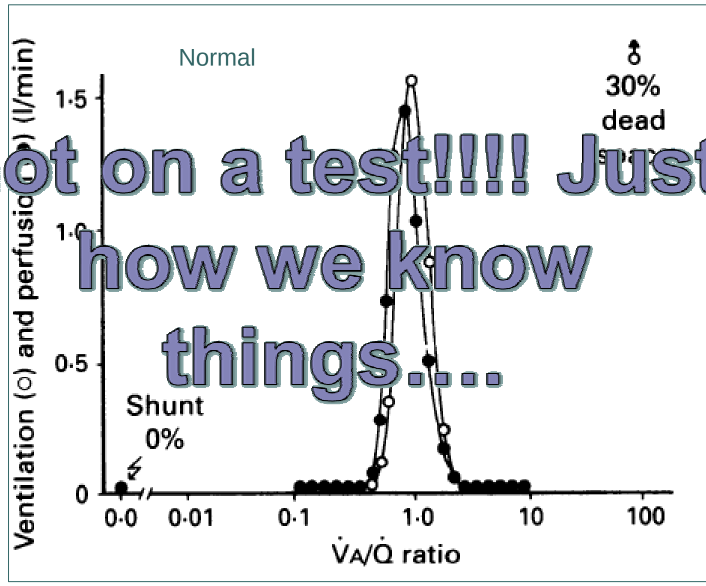
$P_{mv}O_2$ = 40 mmHg **P_aO_2 = 100 mm Hg**

- No obstruction
- No alveolar filling process
- No diffusion barrier

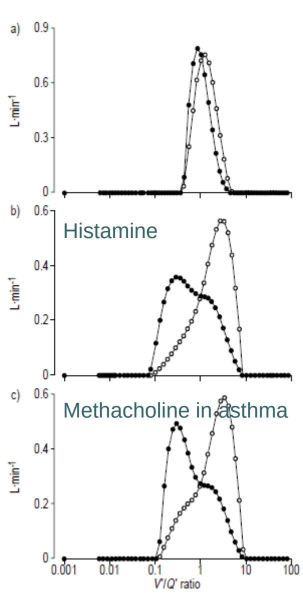
- Ventilation roughly equals Perfusion
  - More of both at the bases
  - Less of both at the apices

- O2 from the bronchus enters the alveolus as rapidly as O2 leaves into the pulmonary capillaries/systemic circulation

MIGET Analysis



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



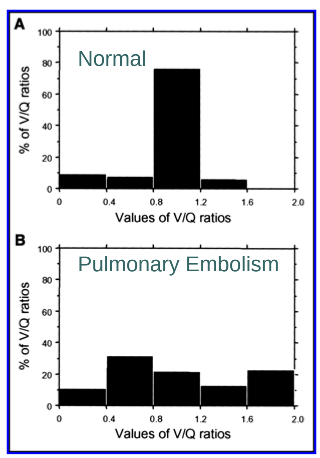
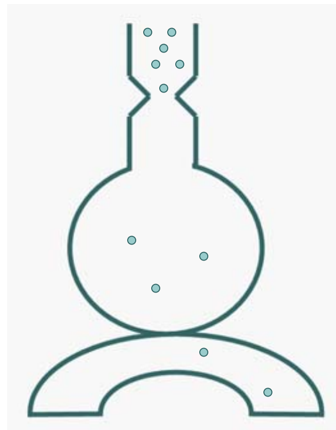


FIGURE 1. Examples of V/Q distribution in patient with normal scan (A) and in patient with confirmed PE (B). Histogram shapes characteristically demonstrate single peak in 0.8–1.2 range in patient with normal scan and bimodal distribution in low (<0.8) and high (>1.2) ranges in PE patient.

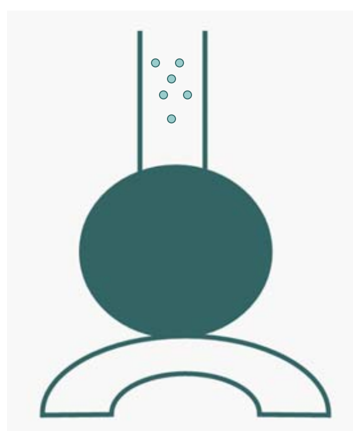
Mechanisms of Hypoxia: VQ Mismatch

- 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

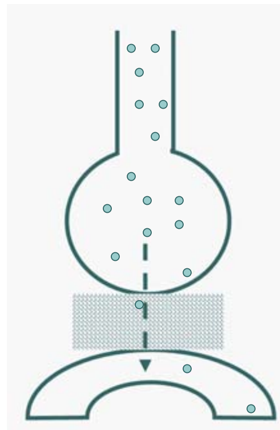
- No O_2 reaches some set of pulmonary capillaries
- Hypoxia is SEVERE
- Hypoxia does NOT improve with supplemental O_2
- Causes:
  - Pulmonary Shunt:
    - NO ventilation to alveoli that are still perfused
      - Blood
      - Pus
      - Water
        - Pulmonary Edema
        - ARDS
      - Atelectasis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



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

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



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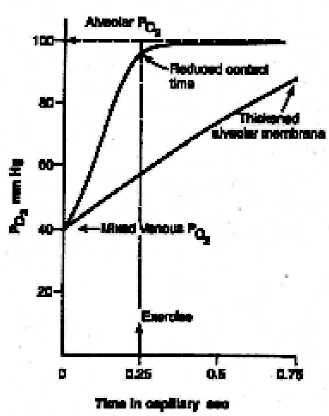
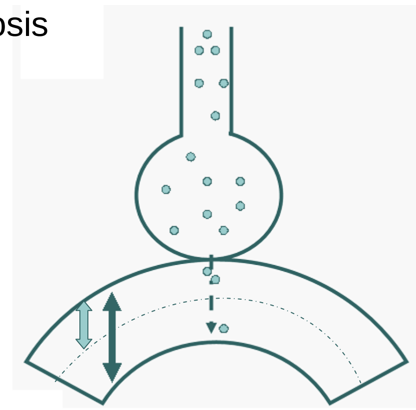


Mechanisms of Hypoxia: Diffusion-Perfusion Impairment

- 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?

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

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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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**

1, 2, and 3 are all NORMAL.

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

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**

If this is obvious, take the next 10 minutes off…..

Otherwise, calculate the A-a gradient

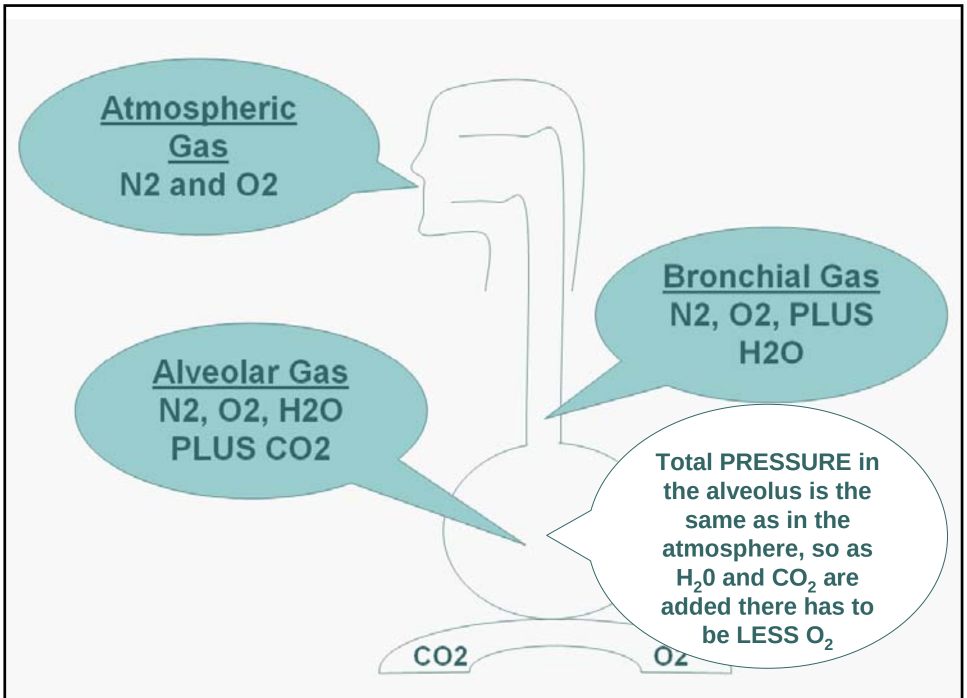
A-a Gradient

- The real question isn't "What is a normal PaO_2?"
- Rather, the real question is:
  - "Is the measured PaO_2 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, O_2 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 O_2 transfer.

A-a Gradient

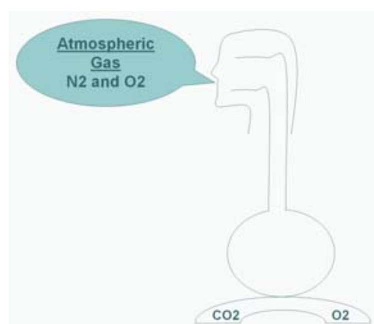
- We ***measure*** the arterial PO_2
- We ***calculate*** what the Alveolar O_2 ought to be
- The **Difference** is the A-a Gradient.

- So, how do we ***calculate*** what the Alveolar O_2 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 $P_{atm}O_2 = P_B \times F_IO_2$ = 760 x .21 = **160**



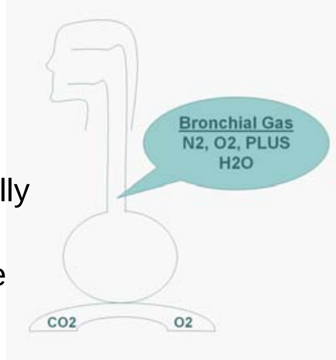
2. What is the normal pO_2 entering the alveolus?

- 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_{H20} = 47 torr
  - Thus, the pO_2 of the air reaching the alveoli is:

$pO_2 = (P_B - P_{H2O}) \times F_IO_2$

$= (760 - 47) \times .21$

$=$ **150**



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:
  - $P_{Alv}O_2=[(P_B - P_{H2O}) \times F_IO_2] - (P_aCO_2/RQ)$

    $[(760 - 47) \times .21] \quad - (P_aCO_2/RQ)$

    $150 \quad - (P_aCO_2/RQ)$
  - "normally" $P_aCO_2/RQ = 40/0.8 = 50$
- Therefore, $P_{Alv}O_2$ normally* = 150 - 50 = **100**

  For people breathing room air at sea level
  - *….P_B, F_IO_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:
  - $P_{Alv}O_2 – P_aO_2$
  - $\{[(P_B – P_{H2O}) \times F_IO_2]– (P_aCO_2/RQ)\} – P_aO_2$

The A-a Gradient Formula:

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= 150 if sea level and room air

P_aCO_2 from ABG; RQ = 0.8

P_aO_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:
    - (Age/4) + 4

The Answers:

1. Which of these people has a lower than expected P_aO_2 ?

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@ sea level, on room air, normal CO_2 and RQ means Alveolar PO_2 should be @ 100 mm Hg

- A-a = 100-95 = 5… normal
- A-a = 100-80 = 20
  - Age/4 + 4 = 20….. 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

@ sea level, on room air, with a normal RQ,
Alveolar PO_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 what hypoventilating

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

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

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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:

$= [150 - PaCO_2/RQ] - PaO_2$

$= [150 – 30/0.8] – 70$

$= [150 – 37.5] – 70$

$= 112.5 – 70$

$= 42.5$

How to describe the "degree" of hypoxia

- The "P/F" Ratio
  - 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)
    ≅ 20 mL O_2/dL Blood
  - $C_{mv}O_2$ = (15)($\underline{0.75}$)(1.34) + ($\underline{40}$)(0.003)
    ≅ 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

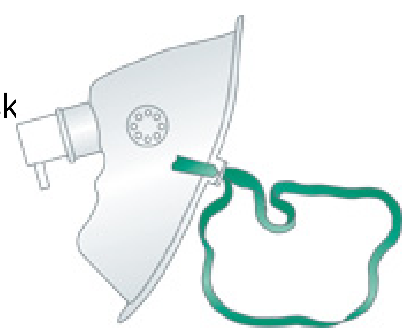
- Evidence of Inadequate Delivery relative to Consumption:
  - ⇩$C_{mv}O_2$
  - ⇧ $D_{a\text{-}v}O_2$
  - ⇧ ER

Oxygen Delivery Devices

- Nasal Cannula
  - 24-44% F_IO_2
  - ? F_IO_2 per liter

Oxygen Delivery Devices

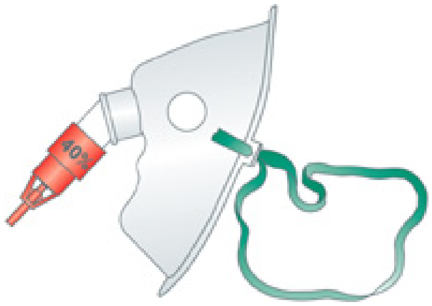
- Nasal Cannula
  - 24-44% FiO2
- Simple Face Mask
  - 40 –60% FiO2



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

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 CO_2? *Conceptually…*

- $PaCO_2$ is determined by how much CO_2 is produced vs how much is eliminated.
  - and CO_2 elimination depends upon Alveolar Minute Ventilation.
    - and Alveolar Minute Ventilation is Total Minute Ventilation minus Wasted Ventilation
- Hence, the determinants of $PaCO_2$ are:
  - CO_2 Production
  - Total Minute Ventilation
  - Wasted Ventilation (i.e., “dead space”)

CO_2: *Mathematically…*

- $PaCO_2 \propto VCO_2 / [MV \times (1 – V_D/V_T)]$
  - $VCO_2 = CO_2$ Production
    - Normal = 200 ml/min
    - Increases in VCO_2 are 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?

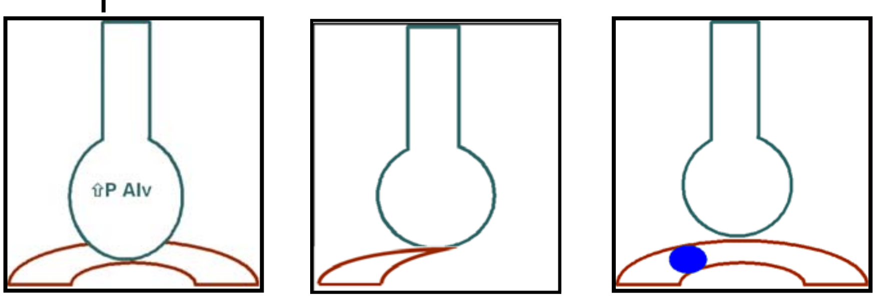
$PaCO_2 \propto VCO_2 / [MV \times (1 – V_D/V_T)]$

- 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 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 ⇧V_D/V_T



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

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 $PaCO_2$

    AND/OR
  - Normal $PaCO_2$ with increased MV

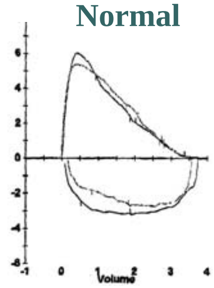
PFT's – practically speaking….

- 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

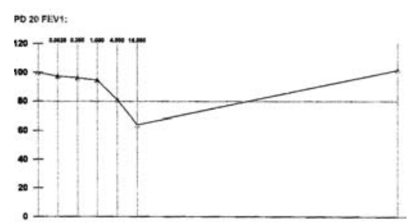
PFT’s: 3 Main Components

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

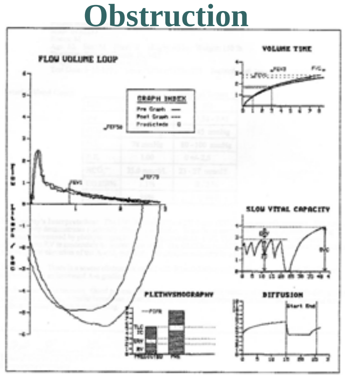
Normal



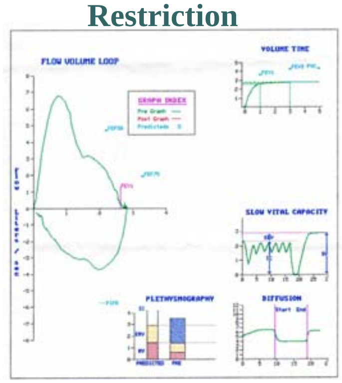
Positive Methacholine Challenge



Obstruction



Restriction



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