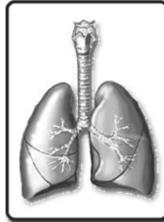


Pulmonary Physiology and Pathophysiology



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2012

1



2

Opening Questions

- Your patient's O₂ saturation is 82%. He does not improve with 100% non rebreather mask. Why not?
- Your patient's O₂ saturation is 95%. Does this assure his tissues are receiving adequate oxygen? Why or why not?
- Your patient's peak inspiratory pressure is high but his plateau pressure is normal. What are some possible causes?

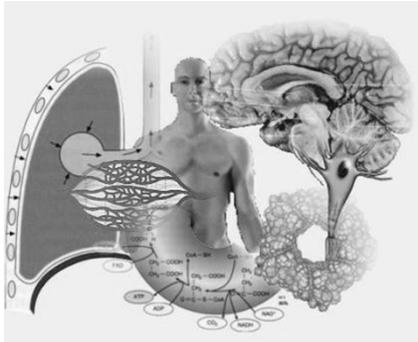
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Opening Questions

- Your patient is on a 50% venti mask and his PaO₂ on blood gas is 90 mmHg. What do you conclude about his oxygenation status?
- Your patient's PCO₂ on blood gas is 55 mmHg. His PaO₂ is 55 mmHg. His pH is 7.27. What treatment do you anticipate?

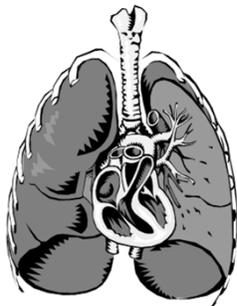
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Pulmonary Physiology



5

Physiology of Pulmonary System



- **Ventilation and Perfusion**
- Diffusion
- Relationship of Oxygen to Hemoglobin
- Oxygen Delivery to the Tissues
- Cellular Respiration

6

Ventilation



7

Ventilation

- **Definition:** The movement of air between the atmosphere and alveoli and the distribution of air within the lungs to maintain appropriate concentrations of oxygen and carbon dioxide in the blood
- Process of ventilation occurs through inspiration and expiration

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Ventilation

- Pressure difference between airway opening and alveoli
 - Contraction of inspiratory muscles
 - Lowers intrathoracic pressure
 - Creates a distending pressure
 - Alveoli expand
 - Alveolar pressure is lowered
 - Inspiration occurs
 - Result: Negative pressure breathing

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Ventilation

- Minute ventilation (V_E) = Total volume of air expired in one minute
 - **Respiratory rate x tidal volume**
 - Normal minute ventilation =
12 x 500 ml = 6000ml
 - Note: (hypoventilation can occur with normal or even high respiratory rate)

10

Alveolar Ventilation (V_A)

- $V_A = V_T -$ anatomical dead space
- Approximately 350 ml per breath

Anatomical dead space:

Walls are too thick for diffusion
Mixed venous blood not present

Approximately 1 cc per ideal pound of body weight

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Respiratory Anatomy

Conducting Airways

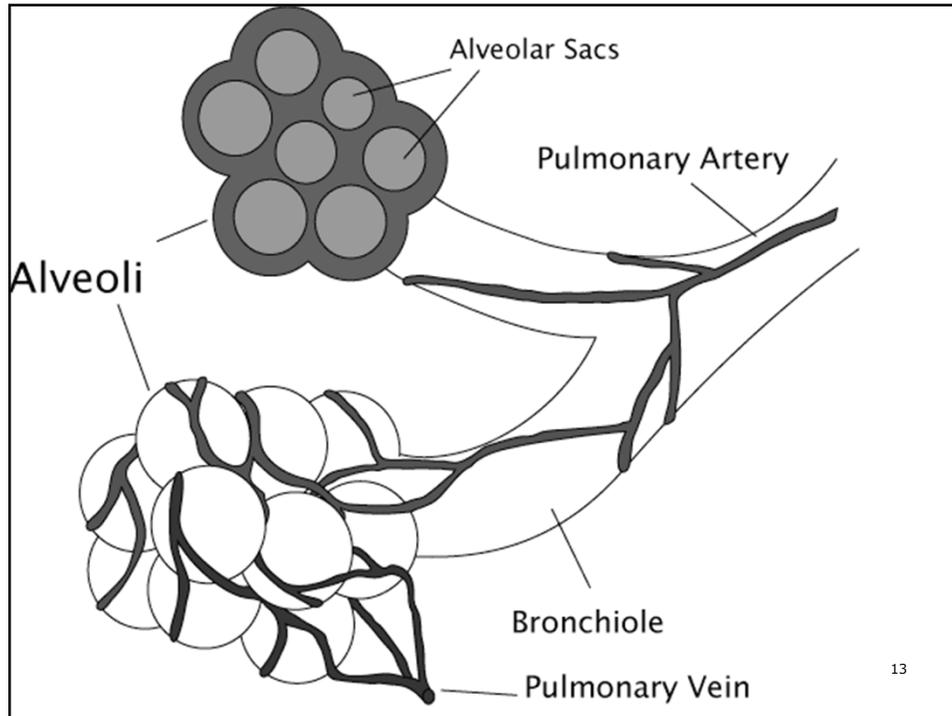
- Nose
- Pharynx
- Larynx
- Trachea
- Right and Left Bronchi
- Non-Respiratory Bronchi

Gas Exchange Airways

- Respiratory Bronchioles (transitional zone)
- Alveolar Ducts
- Alveoli

V_A : Alveolar ventilation

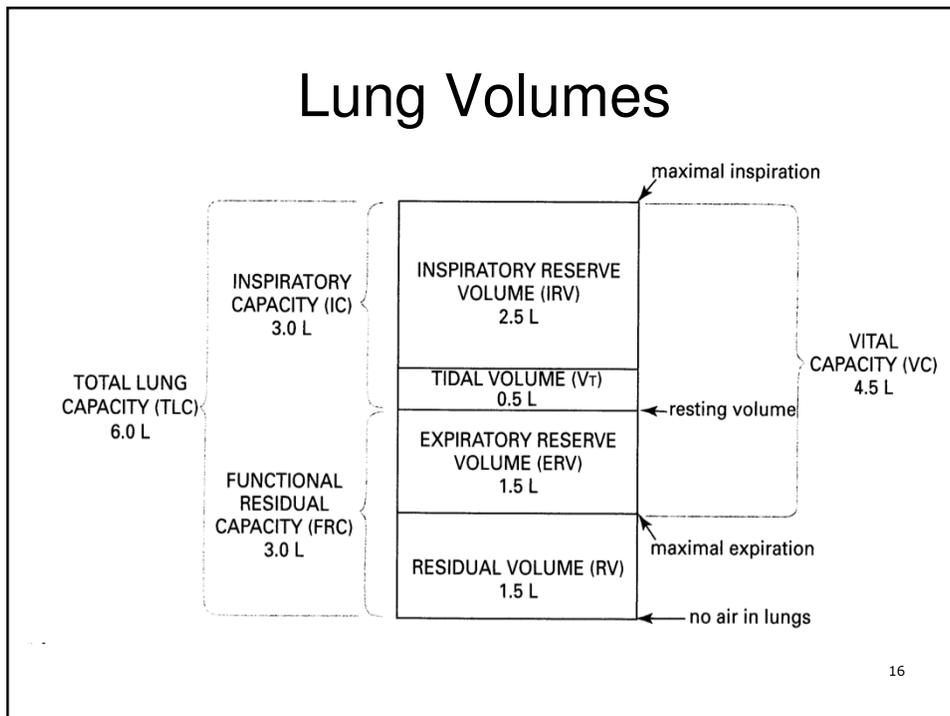
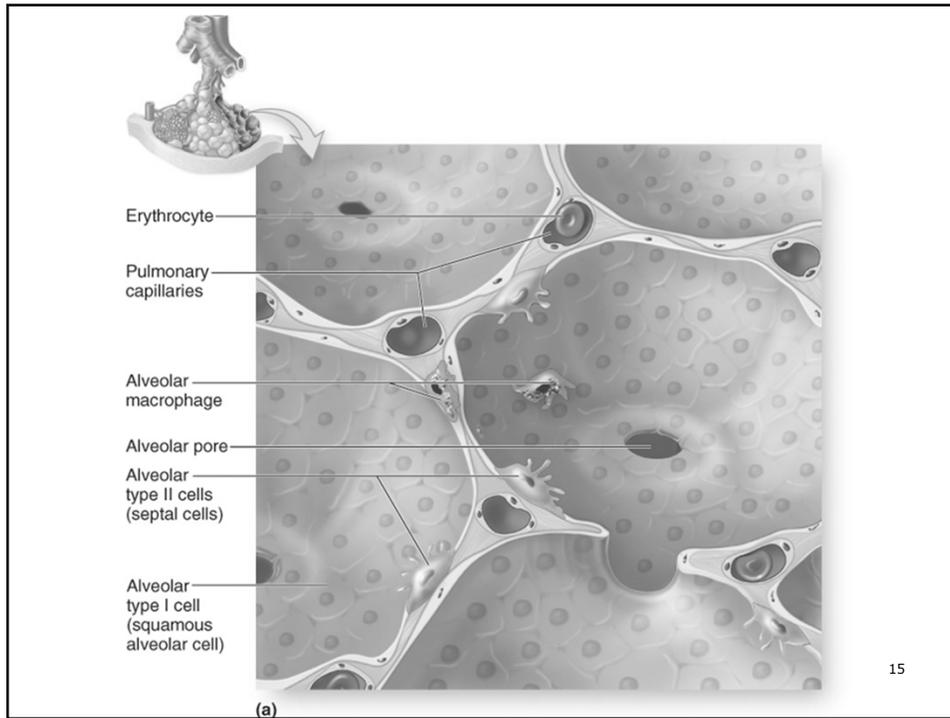
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Alveolar Cells

- **Type I (make up 90% of alveolar surface area)**
 - Squamous epithelium
 - Adapted for gas exchange
 - Prevents fluid from entering alveoli
 - Easily injured
- **Type II**
 - Can generate into Type 1 cells
 - Produces surfactant (**allows alveoli to remain inflated at low distending pressures by decreasing surface tension, decreases work of breathing, detoxifies inhaled gases**)
 - **Lipoprotein (phospholipid)**
 - **Hypoxemia / hypoxia may lead to decreased production or increased destruction**
 - Metabolically active
- **Alveolar Macrophages**
 - Phagocytosis

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Ventilation

- Work of Breathing
Affected by:
 - Compliance (elastic work of breathing)
 - Lungs distend most easily at low volumes
 - Compliance is opposite of elastic recoil

Resistive work of breathing greatest during forced expiration.

- Airway Resistance (flow resistance / resistive work of breathing)
 - Total resistance is comprised of tissue (20%) and airway resistance (80%)
 - Directly proportional to viscosity and length of tube / indirectly proportional to radius
 - Small airway resistance offset by numerous small airways (greatest resistance normally in medium bronchi)

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Assessment of Ventilation

- Efficiency and effectiveness of ventilation is measured by PaCO_2 (inversely related to V_A)
 - $\text{PCO}_2 > 45$ mm Hg indicates alveolar hypoventilation *
 - $\text{PCO}_2 < 35$ mm Hg indicates alveolar hyperventilation

Note: Only one physiologic reason for increased PaCO_2 .

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More on Ventilation

- Normal ventilation on room air results in an alveoli with a partial pressure of oxygen of approximately 100 mmHg.

Partial
pressure
of O₂
100 (104)
mmHg

Inspired gas PI_{O_2}
149 mm Hg.

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Untreated Alveolar Hypoventilation

Untreated alveolar hypoventilation will lead to hypoxemia. The hypoxemia is secondary to uncorrected alveolar hypoventilation.

In acute respiratory failure a blood gas is necessary to assess the PaCO₂ to determine if inadequate ventilation contributed to the hypoxemia.

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Conditions Altering Ventilation

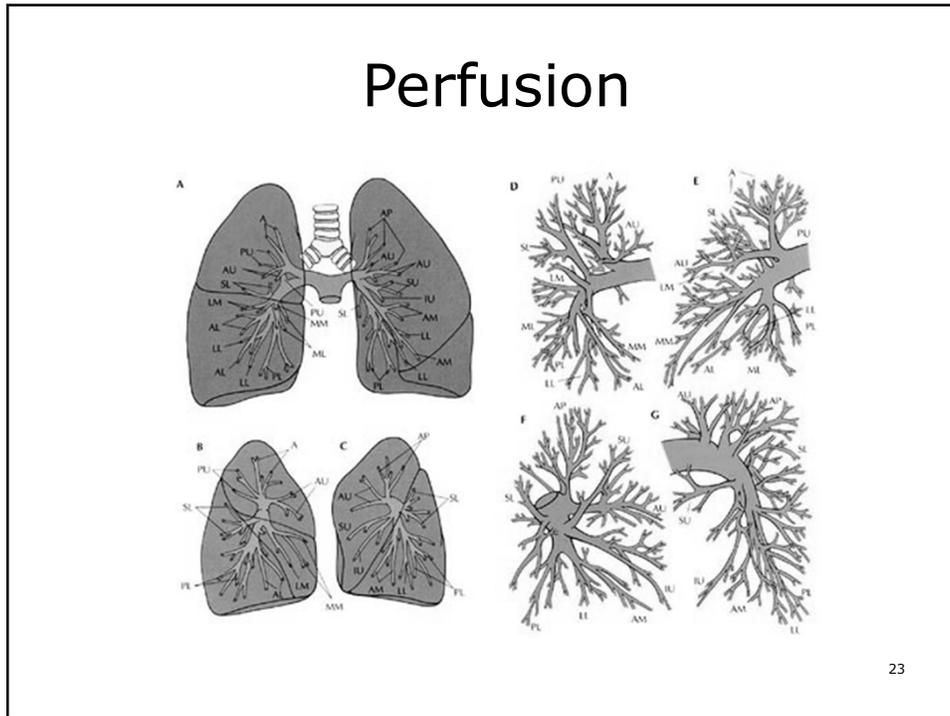
- Non Pulmonary Conditions
- Pulmonary Conditions
 - Decreased Compliance
 - Decreased surfactant production
 - Atelectasis
 - Obesity / musculoskeletal disorders (chest wall compliance)
 - Restrictive disorders (fibrosis, interstitial lung disease)
 - Pulmonary vascular engorgement
 - Air, blood or excess fluid in pleural space
 - Increased Resistance
 - Narrowing of airways (secretions / bronchospasm)
 - Obstructive Disorders
 - Asthma
 - Emphysema
 - Bronchitis
 - Foreign body causes a fixed obstruction
 - Sleep apnea can be obstructive

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Ventilation Review

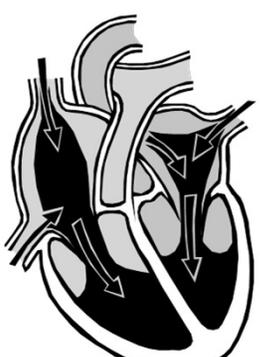
- What physiological factors affect ventilation?
- What non pulmonary conditions can alter ventilation?
- What pulmonary conditions can impact ventilation?
- How is ventilation assessed? (with and without an ABG?)
- How are ventilatory problems corrected?

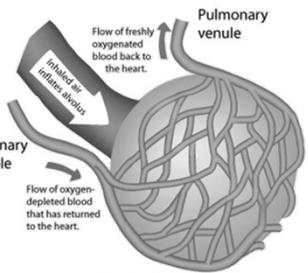
22



Perfusion

- **Definition:** The movement of blood through the pulmonary capillaries





Flow of freshly oxygenated blood back to the heart.

Flow of oxygen-depleted blood that has returned to the heart.

A capillary network covers the surface of the alveolus to facilitate oxygen and carbon dioxide exchange.

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Perfusion

- Blood supply to lung
 - **Pulmonary blood flow**
 - **Entire output of right ventricle**
 - **Mixed venous blood**
 - **Gas exchange with alveolar air into pulmonary capillaries**
 - Bronchial blood flow
 - Left ventricle
 - Part of tracheal bronchial tree
 - Systemic arterial blood

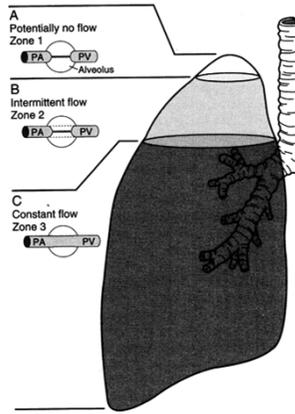
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Perfusion Fun Facts

- Pulmonary capillaries are slightly smaller than average erythrocyte
- Gas exchange actually starts in smaller pulmonary arterial vessels that are not true capillaries (functional pulmonary capillaries)
- 280 billion capillaries supply 300 million alveoli
- Potential surface area for gas exchange is 50-100 m²
- Alveoli are completely enveloped in pulmonary capillaries
- At rest each red blood cell spends only about 0.75 seconds in the pulmonary capillary. Less time during exercise.

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Zones of Perfusion



Zone 1 increased in positive pressure ventilation and PEEP.

- Zone 1: May be no blood flow. (alveolar deadspace – no zone 1 in normal breathing)
- Zone 2: Flow during systole.
- Zone 3: Flow during entire cardiac cycle.

Note: Zones are not static.

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Pulmonary Vascular Resistance

- Comparison with systemic vascular resistance
 - 1/10 of systemic vascular resistance
 - Pulmonary vascular resistance is evenly distributed between the pulmonary arteries, the pulmonary capillaries, and the pulmonary veins.
- Relationship to pulmonary artery pressures and cardiac output
 - Increase in cardiac output = Increase in PAP = Increased capillary recruitment = Decrease in PVR
 - Increased pulmonary artery pressure may lead to pulmonary edema
- Relationship to lung volumes
 - High lung volumes pull pulmonary vessels open. Results in a decrease PVR.

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Pulmonary Vascular Resistance

- During positive pressure mechanical ventilation, both the alveolar and extra-alveolar vessels are compressed during lung inflation and PVR is increased.
- PEEP increases PVR further.

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Hypoxic Pulmonary Vasoconstriction

- Diverts blood away from poorly ventilated alveoli
- Also occurs in response to more global hypoxia
 - Increases pulmonary artery pressure and recruits pulmonary capillaries to improve ventilation and perfusion matching
- Has limitations because of small amount of vascular smooth muscle in the pulmonary arteries
- Hypoxic vasoconstriction greatly increases the workload of the right ventricle
- Increased pulmonary artery pressure may lead to pulmonary edema.

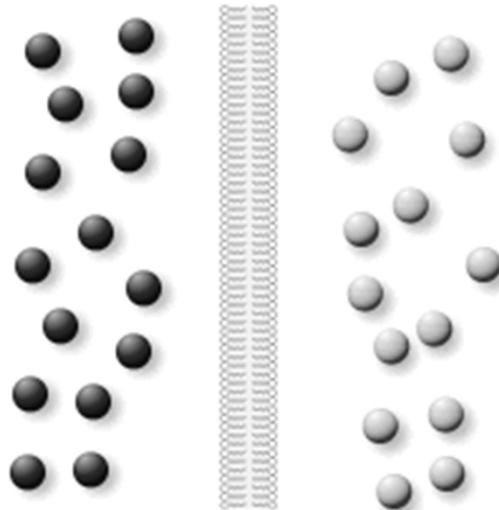
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Perfusion Review

- Do we directly measure pulmonary perfusion?
- What would be a "pulmonary" disorder that results in decreased pulmonary perfusion?
- What would be a non pulmonary reason for decrease in pulmonary perfusion?
- What is the impact of increased pulmonary vascular resistance or increased pulmonary pressures on the right heart?
- Does mechanical ventilation increase or decrease the work of the right heart?

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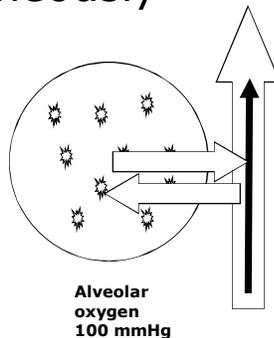
Diffusion



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Prior to Diffusion

- Ventilation and Perfusion Occur Simultaneously



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Diffusion

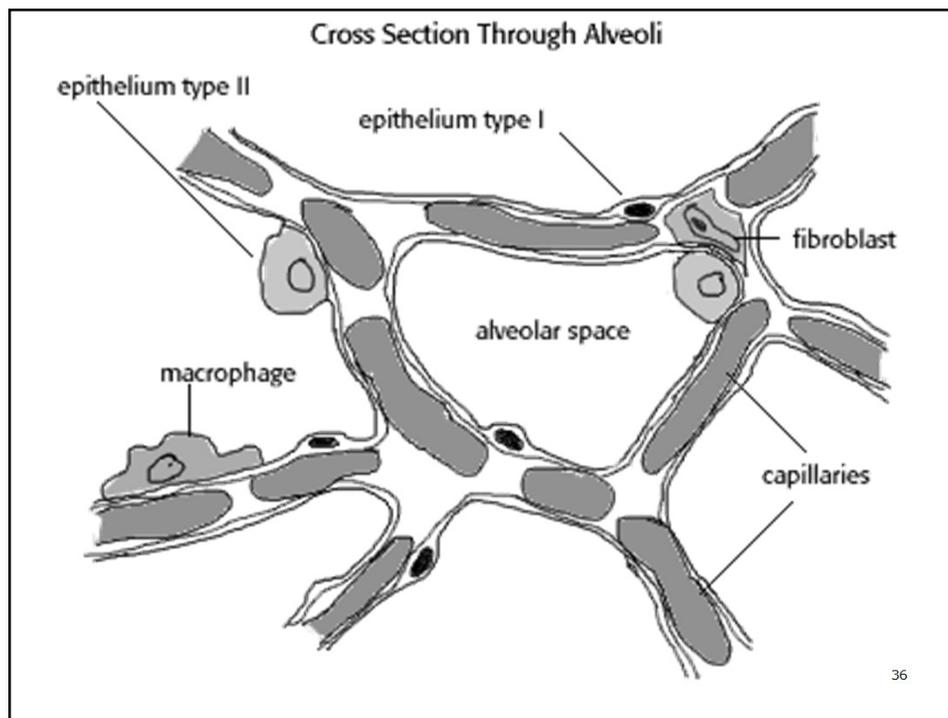
- Movement of gases between the alveoli, plasma, and red blood cells
- Net movement of molecules from an area where the particular gas exerts a high partial pressure to an area where it exerts a lower partial pressure
- Different gases each move according to their own partial pressure gradients
- ***Diffusion of oxygen from alveoli to capillary determines the patient's oxygenation status***

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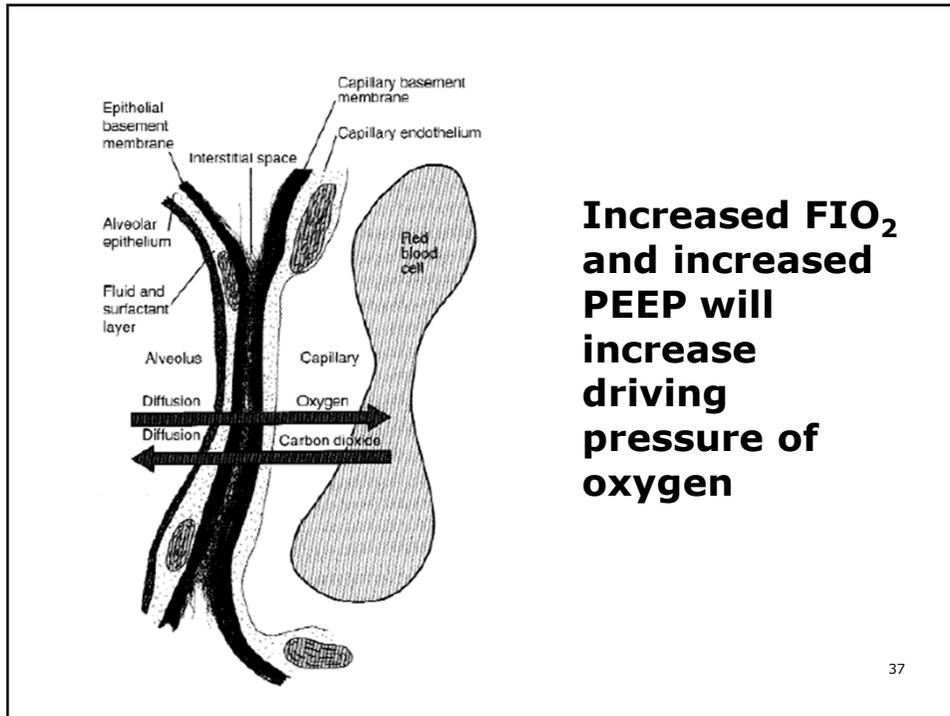
Determinants of Diffusion

- Surface Area: negatively affected by any type of pulmonary resection; tumor, emphysema, pneumothorax
- Driving pressure: negatively affected by low inspired fraction of O₂ (smoke inhalation) or by low barometric pressure (high altitudes)
 - Barometric pressure is the sum of the pressures of all the gases it contains
- Thickness of alveolar capillary membrane (< 1 RBC): negatively affected by pulmonary edema, pneumonia, or fibrosis

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Assessment of Diffusion

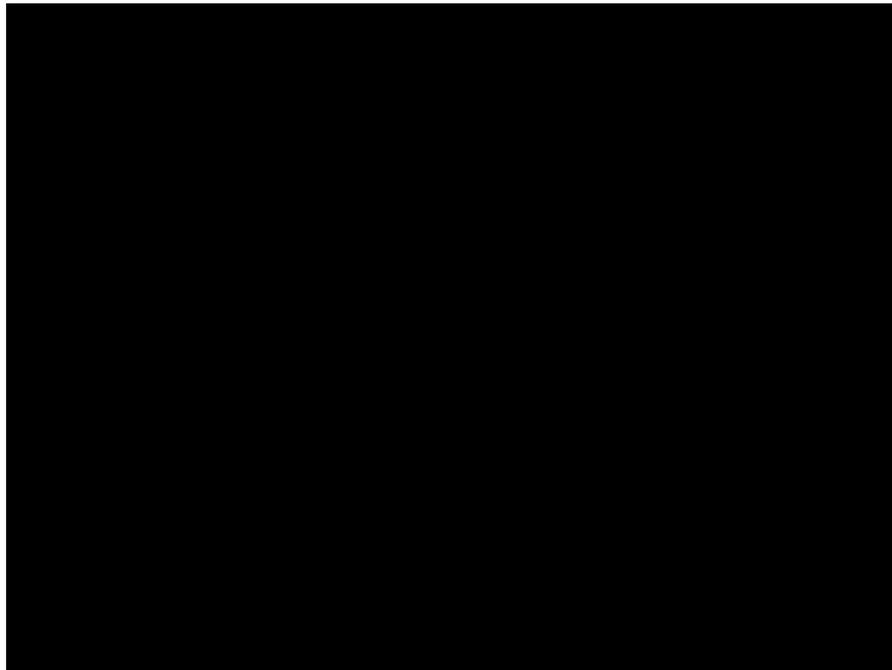
- **PaO_2 and oxygen saturation (SaO_2)**
- *Clinical Application: CO_2 is 20 times more diffusible than O_2 - so a diffusion problem causing hypoxemia does not result in the same problem with CO_2 retention (hypercapnia)*

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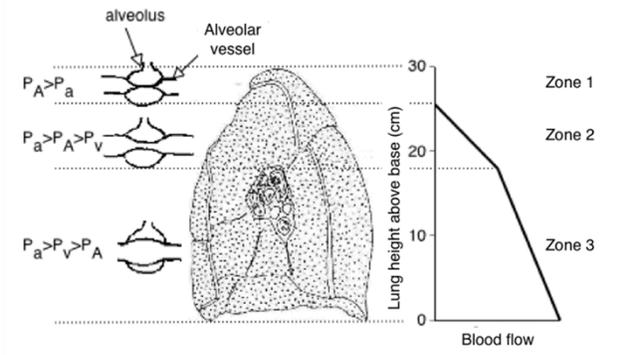
Diffusion Review

- What are the prerequisites for diffusion?
- Which gas is most adversely impacted by barriers to diffusion?
- How do assess if your patient is adequately diffusing?
- How do you correct a simple diffusion problem?

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Ventilation and Perfusion Ratios



Alveoli in upper regions have greater volume and are less compliant. Alveoli in lower parts of lung have a greater change in volume during inspiration and are considered better ventilated.

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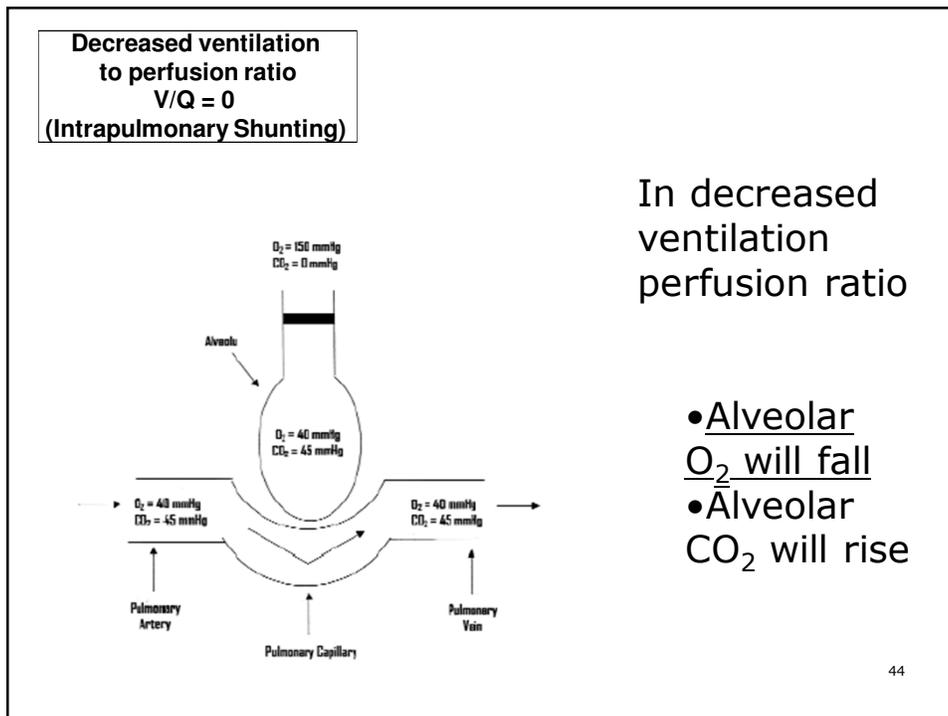
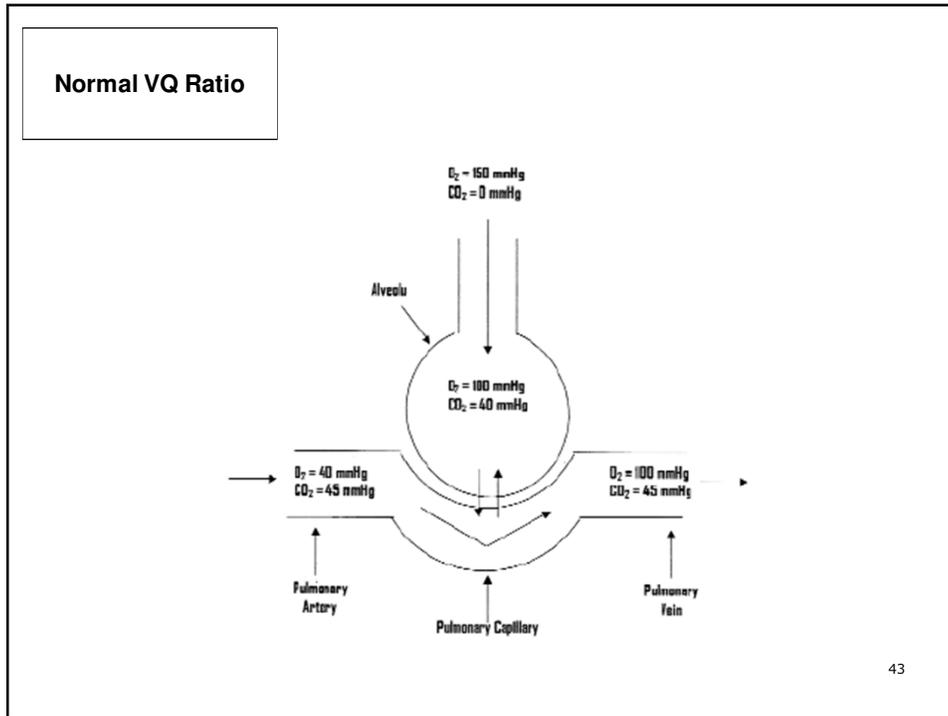
Ventilation / Perfusion Ratio (V/Q)

- **Ventilation (V)**
 - Alveolar minute ventilation = 4 to 6L
- **Perfusion (Q)**
 - Normal cardiac output = 5 L

Normal ventilation /
perfusion ratio
(V/Q ratio) = 0.8 to 1.2

**Ventilation and perfusion must
be matched at the
alveolar capillary level**

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**Increased V/Q
Ratio
(Dead Space)**

In increased ventilation perfusion ratio

- Alveolar O₂ will rise
- Alveolar CO₂ will fall

Alveoli
Pulmonary Artery
Pulmonary Capillary
Pulmonary Vein

45

Decreased V/Q Ratio: Intrapulmonary Shunting

- Intrapulmonary shunt occurs when there is significant alveolar hypoventilation in relation to normal perfusion (Example: Poorly ventilated alveoli in ARDS)
- V/Q ratio < 0.8

Result

- Poorly oxygenated blood returns to left side of heart resulting in low PaO₂ and SaO₂ (oxygenation₄₆ problem)

Increased V/Q Ratio: Alveolar Dead Space

- Alveolar dead space: When ventilation is greater than perfusion
- Classic example: Pulmonary Embolus
- V/Q ratio > 0.8

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Causes of V/Q Mismatching

- Non uniform ventilation
 - Uneven resistance
 - Collapsed airways (Emphysema)
 - Bronchoconstriction (Asthma)
 - Inflammation (Bronchitis)
 - Uneven compliance
 - Fibrosis
 - Pulmonary vascular congestion
 - Atelectasis
- Non uniform perfusion:**
 - **Pulmonary Emboli**
 - **Compression of pulmonary capillaries (high alveolar pressures)**
 - **Tumors**
 - **Shock (pulmonary vascular hypotension)**

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Assessing Oxygenation

- *Clinical Application: Cannot assess PaO₂ (arterial) without considering alveolar oxygenation content (PAO₂)*
 - Increase in FIO₂ will increase PAO₂
 - Increase in PACO₂ will decrease PAO₂

Note: With normal diffusion the majority of oxygen in the alveoli should diffuse across the alveolar capillary membrane.

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ALVEOLAR O₂

Cannot directly measure the amount of oxygen in the alveoli.
It is a calculated value.

Alveolar Gas Equation:

$$PAO_2 = FIO_2 (PB - 47) - PaCO_2 / .8$$

- PAO₂ = partial pressure of alveolar oxygen
- FIO₂ = fraction of inhaled oxygen
- PB = barometric pressure
- 47: PH₂O = water vapor pressure
- PaCO₂ = partial pressure of arterial carbon dioxide
- .8 = respiratory quotient

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Importance of FIO_2

Normal arterial oxygen content of 80 -100 mm Hg is only normal when the FIO_2 is .21

Expected PaO_2 based on FIO_2
 $(FIO_2 \% \times 6) - PaCO_2$

Example: FIO_2 of 100% or 1.0 with $PaCO_2$ 40 mm Hg
 $(100 \times 6) - 40 = 560$ mm Hg

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PaO_2 and FIO_2 Ratio

- An assessment and trending tool
- PaO_2 / FIO_2 ratio:
 - Normal > 300
 - Acute lung injury < 300
 - ARDS < or = 200

PaO_2 of 60 mmHg with an FIO_2 of 0.5 (50%) represents a PaO_2 / FIO_2 ratio of 120.

This is a clinically significant shunt.

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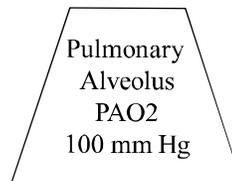
Linking Knowledge to Practice with PaO₂ / FIO₂ Ratios ✦

PaO ₂	FIO ₂	Ratio	Treatment / Notes

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A – a Gradient

- Provides an index regarding diffusion as cause of hypoxemia.
- A large A-a gradient generally indicates that the lung is the site of dysfunction.
- Normal A-a Gradient = 5 to 15 mm Hg



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Critical Thinking Questions Related to Assessment of Hypoxemia

- **What if A – a gradient is normal and PaO₂ is low?????**
- **How do you know when hypoxemia is the result of a simple diffusion problem as opposed to a significant intrapulmonary shunt?**

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Hypoxemia

- Causes
 - Untreated alveolar hypoventilation
 - Diffusion abnormality
 - Ventilation and perfusion mismatching
 - Significant decreased V/Q ratio = intrapulmonary shunting
 - Low inspired oxygen (rare)
- Assessment Clues
 - PaCO₂
 - PaO₂ / SaO₂
 - PaO₂ / FIO₂ ratio
 - A-a gradient

Ventilation versus Diffusion *Assessment and Treatment*

■ **Ventilation problems**

- Assessed by:

- **Corrected with?**

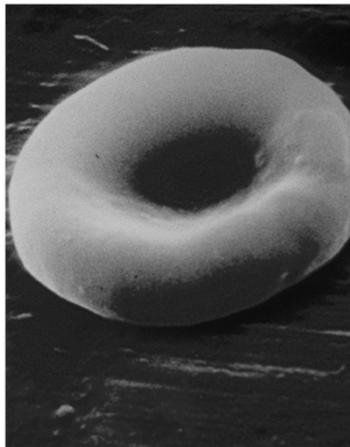
■ **Diffusion problems**

- Assessed by:

- **Corrected with?**

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Relationship Between Oxygen and Hemoglobin



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Oxygen Transportation

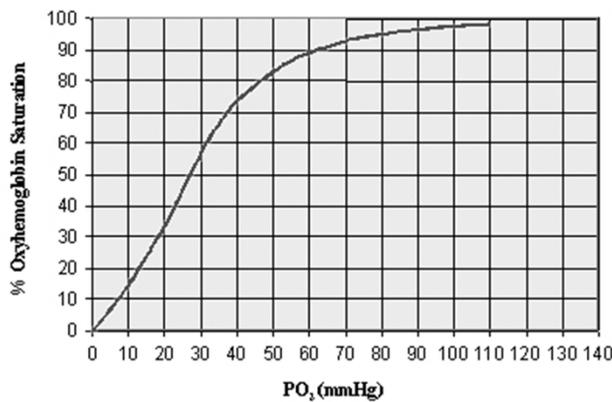
Oxygen is transported both physically dissolved in blood and chemically combined to the hemoglobin in the erythrocytes

- Hemoglobin: 97% of oxygen is combined with hemoglobin
- Plasma: 3% of oxygen is dissolved in plasma
- Represented by the SaO_2
- Represented by the PaO_2 (measurement of O_2 tension in plasma)

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Oxyhemoglobin Dissociation Curve

- Shows the relationship between PaO_2 and SaO_2



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Oxyhemoglobin Dissociation Curve

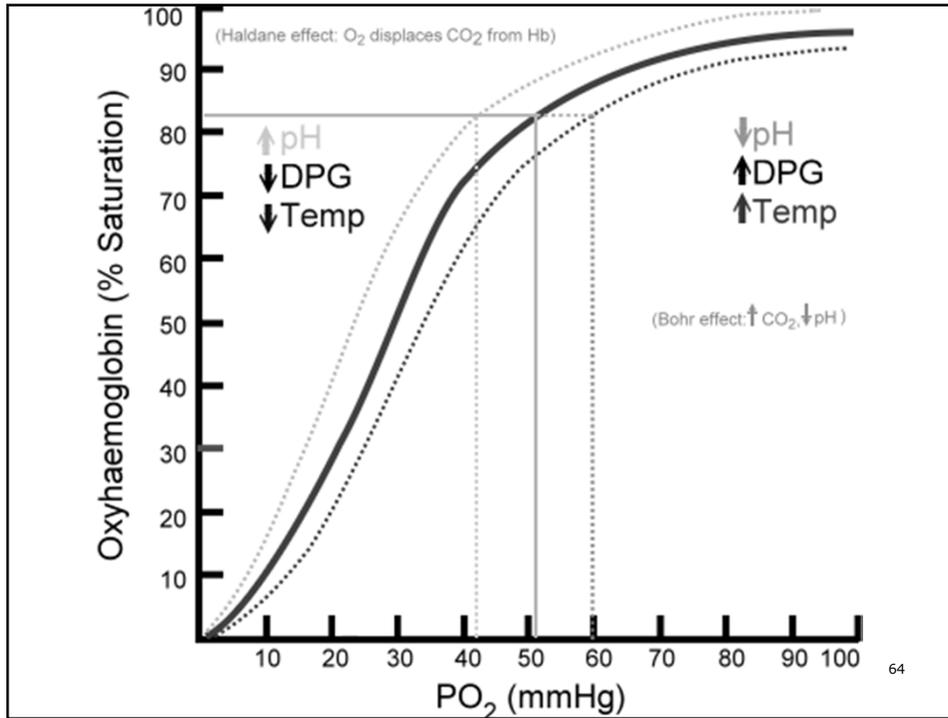
- Horizontal curve shows PaO₂ above 60 results in minimal changes in oxygen saturation
 - Protects body – allowing high saturations with large decreases in PaO₂
- Vertical curve shows PaO₂ below 60 results in significant decreases in oxygen saturation
 - Allows tissues to extract large amounts of O₂ with only small decreases in PaO₂

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Shifts in Oxyhemoglobin Curve

- | | |
|--|---|
| <ul style="list-style-type: none"> ▪ Shift to the Left <ul style="list-style-type: none"> • Easier to pick up at the lung level and more difficult to drop off (unload) at the tissue level • Hemoglobin is more saturated for a given PaO₂ and less oxygen is unloaded for a given PaO₂ | <ul style="list-style-type: none"> ▪ Shift to the Right <ul style="list-style-type: none"> • More difficult to pick up at the lung level but easier to drop off (unload) at the tissue level • Hemoglobin is less saturated for a given PaO₂ and more oxygen is unloaded for a given PaO₂ |
|--|---|

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Shifts in Oxyhemoglobin Curve

■ Causes of Shift to Left

- Hypothermia
- Decreased 2,3 - DPG
- Hypocapnia
- Alkalemia

■ Causes of Shift to Right

- Hyperthermia
- Increased 2,3 - DPG
- Hypercapnia
- Acidemia

65

A Closer Look at 2,3-DPG

- 2,3-Diphosphoglycerate
- Substance in the erythrocyte which affects the affinity of hemoglobin for oxygen (binds to hemoglobin and decreases the affinity of hemoglobin for oxygen)
- Produced by erythrocytes during their normal glycolysis
- Increased
 - Chronic hypoxemia, anemia, hyperthyroidism
- Decreased
 - Massive transfusion of banked blood, hypophosphatemia, hypothyroidism

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Alterations in Oxyhemoglobin Curve

Rise
In
2, 3-DPG
H⁺
Temperature

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Hypoxia and Hypoxemia

■ Hypoxemia

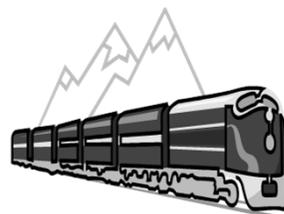
- Insufficient oxygenation of the blood
- Mild: PaO₂ < 80 mm Hg or SaO₂ 95%
- Moderate: PaO₂ < 60 or mmHg or SaO₂ 90%
- Severe: PaO₂ < 40 mmHg or SaO₂ 75%

■ Hypoxia

- Insufficient oxygenation of tissues
- Determined by cardiac index, Hgb, SaO₂, cellular demand, patency of vessels

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Oxygen Delivery to Tissues



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Transport of Gases in the Blood

- **Definition:** movement of oxygen and carbon dioxide through the circulatory system; oxygen being moved from the alveolus to the tissues for utilization and carbon dioxide being moved from the tissues back to the alveolus for exhalation

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Oxygen Delivery To Tissues

- Oxygen delivery measured as DO_2 :
Volume of oxygen delivered to tissues each minute
- $DO_2 = \text{cardiac output} \times \text{arterial oxygen content (hemoglobin} \times \text{arterial oxygen saturation)}$

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Formula for Oxygen Delivery

- DO_2 formula = **CO x Hgb x SaO₂ x 13.4** (constant)
- Normal DO_2 = 900- 1100 ml/min
(1000)
- Normal DO_{2I} = 550 – 650 ml/min

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Improving Oxygen Delivery

- Oxygen delivery can be improved by increasing cardiac output, hemoglobin or SaO₂

Some interventions more effective in clinical practice; interventions can be performed simultaneously

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Oxygen Consumption

- Measured as VO_2
- Volume of oxygen consumed by the tissues each minute
- Determined by comparing oxygen content in arterial blood to the oxygen content in mixed venous blood
 - Normal CaO_2 is 20 ml/dl and normal CVO_2 is 15 ml/dl
- Normal VO_2 : 200 – 300 ml / min (250 ml / min)

74

Causes of Increased VO_2

- | | |
|------------------------|-----------|
| ■ Fever per 1 degree C | ■ 10% |
| ■ Shivering | ■ 50-100% |
| ■ Suctioning | ■ 7-70% |
| ■ Sepsis | ■ 5-10% |
| ■ Non Family Visitor | ■ 22% |
| ■ Position Change | ■ 31% |
| ■ Sling Scale Weight | ■ 36% |
| ■ Bath | ■ 23% |
| ■ CXR | ■ 25% |
| ■ Multi Organ Failure | ■ 20-80% |



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Oxygen Reserve in Venous Blood

- **Measured by mixed venous oxygen saturation (SVO_2)**
- **Normal 60-80% (75%)**

- **Tissues were delivered 1000 ml / min (DO_2)**
- **Tissues uses 250 ml / min (VO_2)**
- **This leaves a 75% reserve in venous blood**
- **Oxygen Extraction Ratio (O_2ER) = 25%**

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Oxygen Consumption and Oxygen Delivery

- Oxygen delivery and oxygen consumption are **independent** until a critical point of oxygen delivery is reached

- Tissues will extract the amount of oxygen needed independent of delivery because delivery exceeds need

77

Relationship of Delivery to Consumption ✦

DO₂	VO₂ (extraction is independent of delivery)	SVO₂ (SVO ₂ will improve when you increase delivery)

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Relation of Delivery to Consumption

- When oxygen delivery reaches a critical level then consumption will depend on delivery
- SVO₂ will not increase with increased delivery while you are in this dependent state
- Anaerobic metabolism occurs here because you have an oxygen deficit

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SVO₂ Monitoring

- Global indicator between oxygen supply and demand
- Influenced by oxygen delivery and oxygen extraction
- Reflects mixing of venous blood from superior vena cava, inferior vena cava and coronary sinus
- Measured using a pulmonary artery fiberoptic catheter

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Significant Changes In SVO₂

- SVO₂ < 60%
 - Decreased delivery
 - Increased consumption
 - SVO₂ > 80%
 - Increased delivery
 - Decreased demand
 - Sepsis (tissues cannot extract)
 - Wedged catheter
 - Clinically significant change is +or - 5 to 10% over 3 to 5 minutes
- SVO₂ < 40% represents limits of compensation and lactic acidosis will occur (oxygen demand is greater than oxygen delivery and reserve can be depleted = oxygen debt)

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ScVO₂

- ScVO₂ reflects oxygen saturation of blood returning to right atrium via the superior vena cava.
 - Can be obtained without a pulmonary artery catheter, using a modified central venous catheter with fiberoptic technology.
 - Normal value is > 70%.
 - ScVO₂ trends higher than SVO₂ but trends with SVO₂.

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Cellular Respiration

- Definition: Utilization of oxygen by the cell
- Estimated by the amount of carbon dioxide produced and amount of oxygen consumed
- Oxygen is used by the mitochondria in the production of cellular energy – prolonged oxygen deficit can result in lethal cell injury

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Oxygen and Ventilator Therapy



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Oxygen Therapy

- Cannula: < 40%
- Simple Mask: 40-60%
- Venturi Mask: Up to 40%
- Nonrebreathing mask: 80-100%
- Bag Valve Mask

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High Flow Oxygen Therapy

- Provides entire inspired gas by high flow of gas
- Provides a predictable FIO₂
- Doesn't mean a high FIO₂
- 100% non rebreather masks, venturi masks and mechanical ventilators are examples of higher flow oxygen delivery systems

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Guidelines for estimating FIO₂ with low flow oxygen devices

■ 100% O ₂ flow rate(L)	■ FIO ₂ (%)
• Nasal Cannula	■ 24
■ 1	■ 28
■ 2	■ 32
■ 3	■ 36
■ 4	■ 40
■ 5	■ 44
■ 6	
• Oxygen Mask	■ 40
■ 5-6	■ 50
■ 6-7	■ 60
■ 7-8	
• Mask with Reservoir	■ 60
■ 6	■ 70
■ 7	■ 80
■ 8	■ 90
■ 9	■ 99
■ 10	

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Oxygen Toxicity

- | | |
|---|--|
| <ul style="list-style-type: none"> ■ Complications of O₂ <ul style="list-style-type: none"> • Absorption atelectasis • Decreased hypoxic drive ■ Signs and symptoms of oxygen toxicity <ul style="list-style-type: none"> • Dyspnea • Decreased lung compliance • Retrosternal pain • Parasthesia in the extremities | <ul style="list-style-type: none"> ■ To reduce risk of oxygen toxicity: <ul style="list-style-type: none"> ■ 100% no more than 24 hours ■ 60% no more than 2-3 days ■ Use 40% for longer term therapy |
|---|--|

89

Mechanical Ventilation

- | | |
|---|---|
| <p>Indications</p> <ul style="list-style-type: none"> ■ Respiratory failure. <ul style="list-style-type: none"> • Hypercapnic • Hypoxemic ■ Excessive work of breathing. <ul style="list-style-type: none"> • Tachypnea • Accessory muscle use • Tachycardia • Diaphoresis ■ Protection of airway | <p>Goals</p> <ul style="list-style-type: none"> ■ Achieve adequate ventilation ■ Achieve adequate oxygenation ■ Provide decreased work of breathing, patient comfort and synchrony with the ventilator ■ Protect the lungs from further injury |
|---|---|

90

Non Invasive Positive Pressure Ventilation

- Continuous Positive Airway Pressure
 - Continuous pressure throughout breathing cycle
 - Most commonly 10 cm H₂O
- Biphasic Positive Airway Pressure
 - Senses inspiration and delivers higher pressure during inspiration
- Consider as first line strategy
- Consider as alternative to failed weaning
- Decreased VAP

91

Non Invasive Positive Pressure Ventilation

- Dedicated ventilator or traditional mechanical ventilator
- Note: Single tubing / dual tubing; ported mask or non ported mask
- Contraindications
 - Decreased level of consciousness
 - Increased gastrointestinal bleeding
 - Hemodynamic instability
 - Progressive decline in respiratory status

92

Low Flow Oxygen Therapy

- Doesn't provide total inspired gas
- Patient breathes varying amounts of room air
- FIO₂ depends on rate and depth of ventilation and fit of device
- Doesn't have to mean low FIO₂
- Nasal cannula is a low flow oxygen delivery system
- Simple face mask is a moderate flow delivery system

93

Intubation and Cuff Pressure

- Cuff pressures should not exceed capillary filling pressures of trachea
 - < 25 cm H₂O or < 20 mmHg
- Adequate seal for positive pressure ventilation and PEEP
- Prevents aspiration of large particles but not liquids
- Low pressure / high volume cuffs used
- Leak in cuff or pilot balloon valve requires replacement
- Routine measurement of cuff pressures

94

Mechanical Ventilation Breaths

- **Volume cycled:** Preset tidal volume
- **Time cycled:** Delivered at constant pressure for preset time
- **Flow cycled:** Pressure support breath. Constant pressure during inspiration.

95

Modes of Ventilation

- **Assist Control Mode (AC)**
 - Volume targeted (volume cycled)
 - Pressure targeted (time cycled)
- **Synchronized Intermittent Mandatory Ventilation (SIMV)**
 - Same breath options as assist control
- **Adaptive Support Ventilation**
- **Airway Pressure Release Ventilation (APRV)**
 - Open lung strategy
- **High Frequency Oscillator Ventilation**
 - Open lung strategy

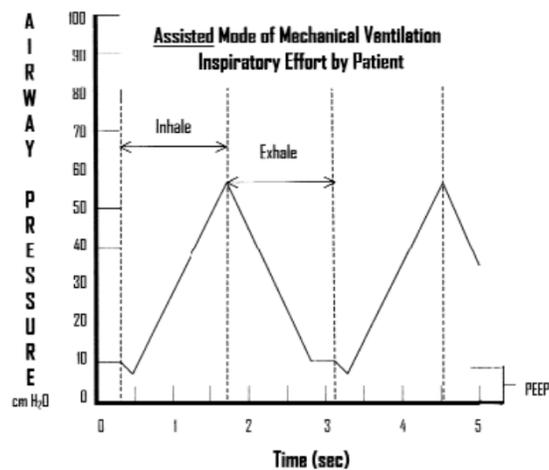
96

Assist Control

- Minimal respiratory rate is set. Set number of breaths delivered at the preset parameters.
- Allows the patient to assist. Maintains control of patient breaths once initiated.
- Differs from (CMV) where no spontaneous breaths are allowed.
- Is effective in decreasing the work of breathing when used with appropriate sedation.

97

Assist Control



98

SIMV

- Delivers a set number of ventilator breaths at preset parameters.
- Also allows the patient to initiate breaths above the preset rate.
- Patient initiated breaths in SIMV are patient dependent and not guaranteed to achieve ventilator set parameters.
- Pressure support is often used during spontaneous breaths.
- The primary disadvantage of SIMV is the increased work of breathing in the patient with respiratory distress.

99

Adaptive Support Ventilation

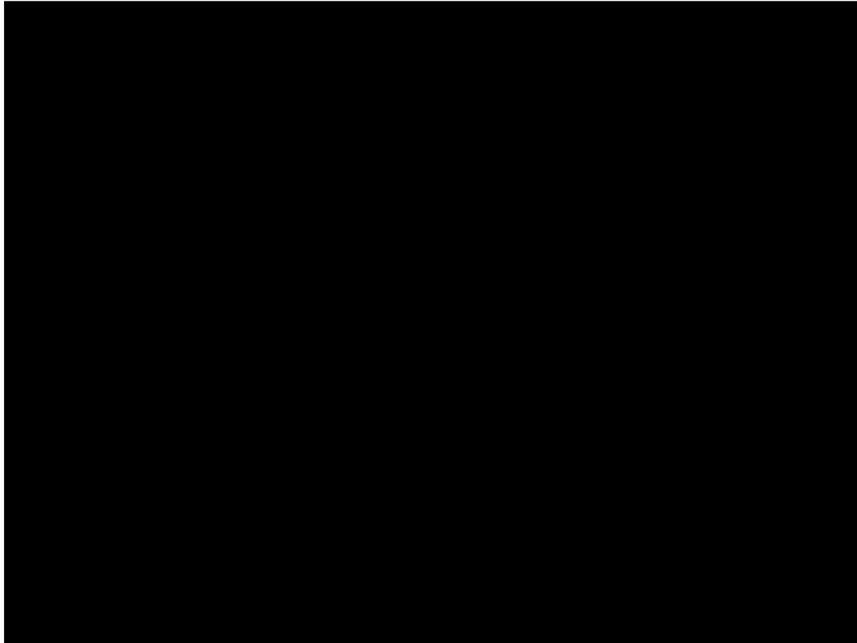
- Dual control
 - Pressure limited
 - Time cycled
- Breath to breath
- Pressure limit of spontaneous and mandatory breaths continuously adjusted
- Other names based on commercial ventilators

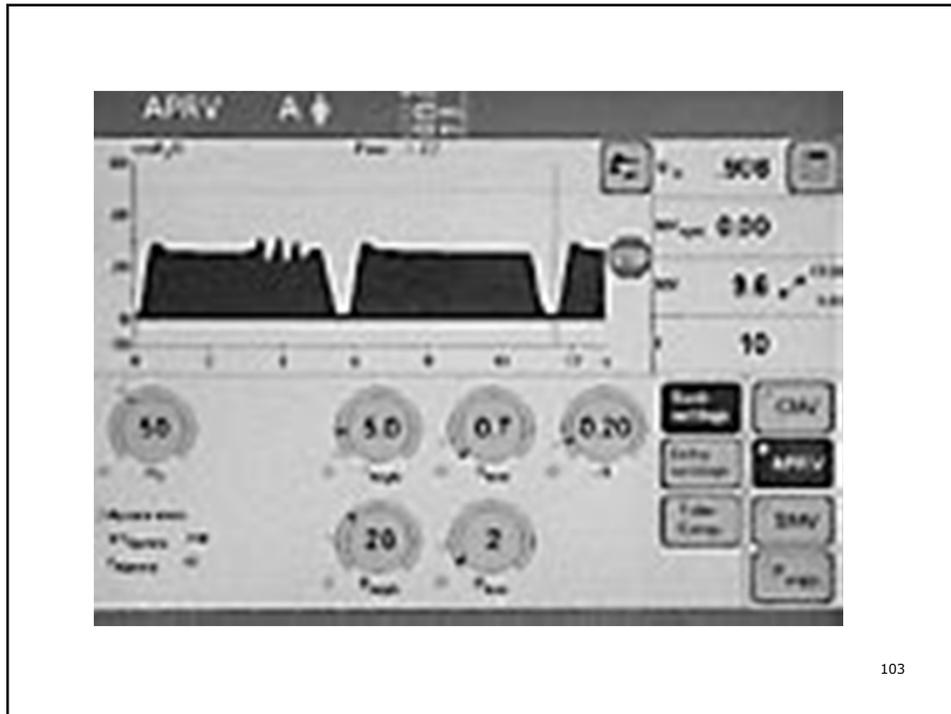
100

Open Lung Strategies: Focus on Mean Airway Pressure

- APRV
 - Similar to CPAP with release
 - Spontaneous breathing allowed throughout cycle
 - Can also be used with no spontaneous effort
 - Release time allows removal of CO₂
 - P High (20 -30 cmH₂O) and P low (0) (pressure)
 - T high (4-6 seconds) and T low (0.8 seconds) (time)
 - Facilitates oxygenation and CO₂ clearance
 - Time triggered
 - Pressure limited
 - Time cycled
- Advantages
- Lower peak and plateau pressures for given volume
 - Decreased sedation / near elimination of neuromuscular blockade

101





Open Lung Strategies: Focus on Mean Airway Pressure

- High frequency oscillation
 - Not jet ventilation
 - Constant mean airway pressure
 - TV 1-3ml/kg
 - Delivers and removes gas: 1/3 time delivery in and 2/3 time delivery out
 - Usually set starting at 5 to 6 HZ (60 oscillations / HZ)
 - Chest wiggle
 - JVD: Tamponade effect

■ Inverse ratio ventilation

104

Initial Ventilator Settings: Acute Respiratory Failure

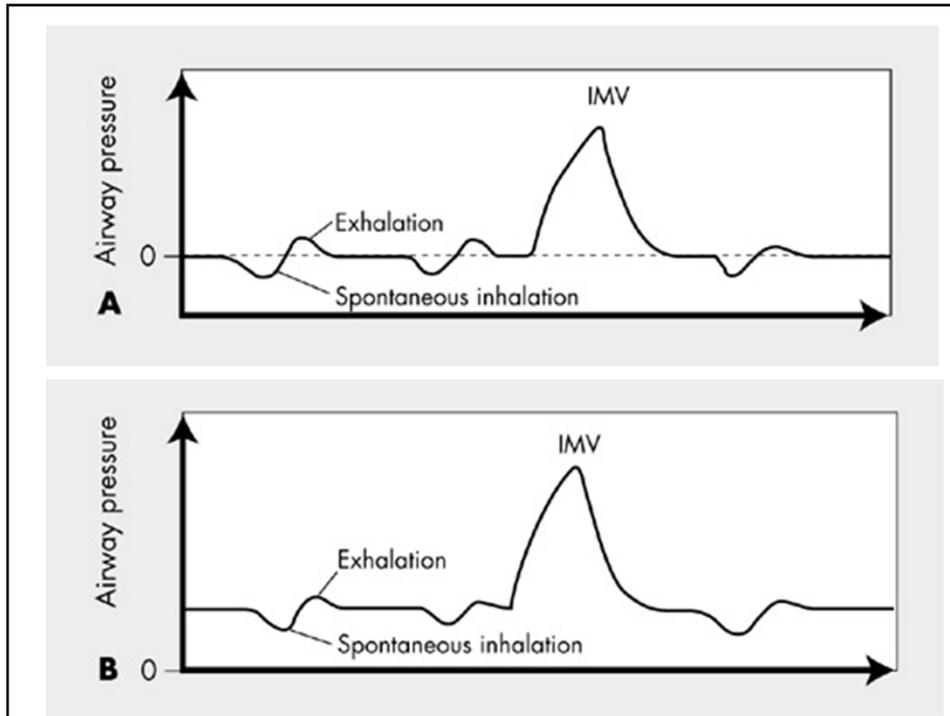
- Most common initial mode of ventilation used in critical care for respiratory failure is AC with volume cycled breathes.
- Tidal volume: (VT): Usually set at 8 – 10 ml/kg of ideal body weight.
- Respiratory Rate: Usually set at 12-16 breaths per minute.
- Fraction of Inspired Oxygen (FIO₂): Started at 1.0 or 100%. Weaning as quickly as possible to .4 or 40% while maintaining an oxygen saturation of 92-94%.
- PEEP: Usually started at 5 cm of H₂O. PEEP is titrated up as needed to achieve adequate oxygenation. > 15 cm H₂O of PEEP is rarely needed.

105

Adjuncts to Mechanical Ventilation

- PEEP: Positive end expiratory pressure
- PSV: Pressure support ventilation; positive pressure during inspiration; during spontaneous breaths with SIMV or during non invasive mechanical ventilation

106



More on PEEP

- PEEP is used to improve oxygenation by increasing mean airway pressures and increasing the driving pressure of oxygen across the alveolar capillary membrane.
- Prevents derecruitment, low levels do not recruit
- PEEP and PAOP
- Potential complications:
 - Barotrauma
 - Decreased cardiac output
 - Regional hypoperfusion

**Optimal
PEEP**

108

Other Ventilator Settings

- Peak Flow (gas flow): speed and method of VT delivery, velocity of air flow in liters per minute
-
- Sensitivity: determines patient's effort to initiate an assisted breathe
-
- I:E ratio (inspiratory to expiratory ratio): Typically set at 1:2 (can be altered to facilitate gas exchange and prevent auto peep)

109

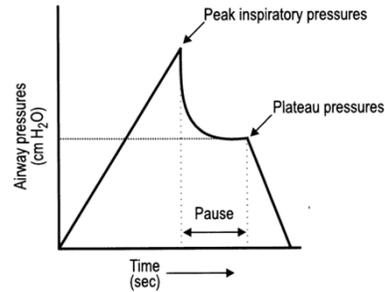
Measured Parameters

- Mean Airway Pressure: Constant airway opening pressure
 - PEEP
 - CPAP
 - Pressure Support
- I:E Ratio
 - Inspiration time and mean airway pressure
 - Development of auto PEEP

110

Measured Parameters

- **Peak Inspiratory Pressure**
 - Accounts for airway resistance and lung compliance
- **Inspiratory Plateau Pressure**
 - Takes resistance out of equation



111

Improving Resistance and Compliance

- ***Airway Resistance***
 - Effective coughing
 - Bronchodilators (albuterol) or steroids for bronchospasm
 - Repositioning and suctioning to mobilize and aspirate secretions
 - Decrease endotracheal tube resistance.
 - > 8 mm
 - Short tubes
- ***Lung / Chest Compliance***
 - Deep breath and hold
 - Incentive spirometry (10 breaths per hour)
 - Prevent abdominal distention / positioning
 - Thoracentesis or chest tube for pleural effusion
 - Diuretics for pulmonary edema
 - Antibiotics for pneumonia
 - CPAP
 - PEP (positive expiratory pressure)

112

Hemodynamic Effects of Mechanical Ventilation

- Decreased venous return
- Pulmonary capillary compression and increased right ventricular afterload
- Decreased right ventricular stroke volume
- Decreased left ventricular afterload

113

Hypotension with Mechanical Ventilation

- **Conversion to positive pressure ventilation.**
 - Assure adequate circulating fluid volume.
- **Tension Pneumothorax**
 - Chest tube required.
- **Development of auto PEEP**
 - Increase expiration time.

114

Complications of Mechanical Ventilation

- **Barotrauma** (caused by excessive pressure)
- **Volutrauma** (caused by excessive volume)
- **Atelectrauma** (caused by low volume resulting in repetitive opening and closing of distal lung units)
- **Biotrauma** (caused by biochemical mediators released in response to mechanical ventilation as opposed to a mechanical complication)

115

Lung Protective Strategies

- Low tidal volume (6 ml / kg) with permissive hypercapnea
- Maintain plateau pressure \leq 30 mm Hg

116

Benefits of Sedation During Mechanical Ventilation

- Reduce anxiety
- Amnesia, particularly during use of neuromuscular blocking agents
- Prevent recall of unpleasant experience
- Decrease level of stress hormones
- Reduce tissue oxygen consumption
- Improve ventilator synchrony

117

Sedation and Analgesia in Mechanical Ventilation

- Pain and anxiety should be assessed together
- Goals:
 - Patient comfort
 - Ventilator synchrony

118

More on Analgesia

- Treating pain is a priority
 - Pain can manifest as anxiety
 - Sedation will not relieve pain
 - Opiates (scheduled or drip) for constant effect
 - Acetaminophen or non steroidal as adjunct

119

More on Sedation

- Target level of sedation: Calm patient who is easily arousable.
 - Note: Use of benzodiazepines increases risk for delirium
 - Use only what is needed (or use other agents)
 - Maintain brief eye contact and follow simple commands
 - No signs of agitation
 - Use of validated assessment tool
 - Richmond Agitation Sedation Scale (RASS)
 - Riker Sedation-Agitation Scale (SAS)

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Daily Interruptions of Sedation

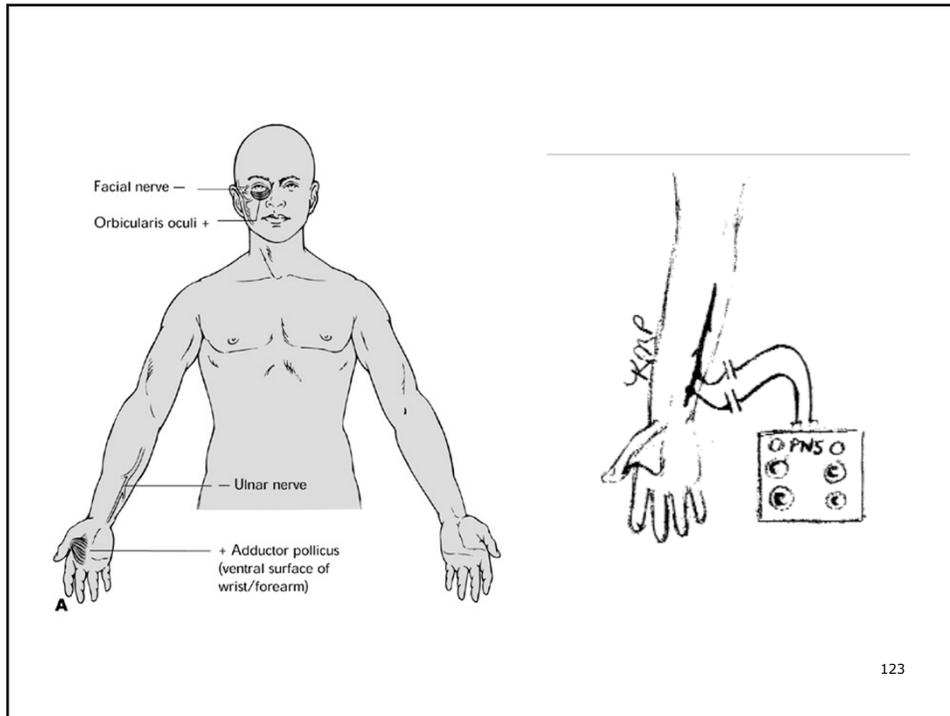
- Allow patients to spend some time awake and interacting
- Time for a thorough neurological assessment
- Opportunity to re-titrate sedatives and analgesics.
- Reduces duration of mechanical ventilation and ICU stay w
- Does not increase unplanned extubations.

121

Neuromuscular Blockade

- | | |
|---|--|
| <ul style="list-style-type: none"> ■ Depolarizing agents <ul style="list-style-type: none"> • Mimic acetylcholine: produces fasciculation followed by paralysis • Example: Succinylcholine | <ul style="list-style-type: none"> ■ Complications of neuromuscular blockade. <ul style="list-style-type: none"> • Deep vein thrombosis, muscle atrophy, and nerve compression syndromes. • Acute quadriplegic myopathy syndrome, or critical illness polyneuropathy is a serious complication |
| <ul style="list-style-type: none"> ■ Non-depolarizing agents <ul style="list-style-type: none"> • Prevents action of acetylcholine • Example: Vecuronium and Atracurium | <ul style="list-style-type: none"> ■ Assessment with neuromuscular blocking agents. <ul style="list-style-type: none"> • Peripheral nerve stimulators • The most commonly used nerve-muscle combinations <ul style="list-style-type: none"> ■ Facial nerve and orbicularis oculi ■ Ulnar nerve and adductor pollicis. • The goal is to have one or two twitches in response to nerve stimulation. |

122



123

Sources of Nosocomial Pneumonia during Mechanical Ventilation		Microbiologic causes of Nosocomial Pneumonia
Exogenous	Endogenous	
Mechanical ventilatory circuit	Gastric pH and volume	Bacteria
Heated humidifier	Buccal mucosa and oropharyngeal flora	Viruses
Suction catheters	Circulating infectious agents—septicemia	Fungi
		Protozoa (<i>Pneumocystis carinii</i>)
		Atypical agents (Legionella)

Evidenced Based Nursing Practice

124

Prevention of VAP

- Hand hygiene
- Oral care, including brushing of teeth, gums, and tongue
- HOB elevated 30 to 40 degrees
- Suction only when necessary (not routine)
- **Routine installation of NS not recommended**
- Cover yankauer catheters when not in use
- Ventilator circuit changes only when soiled, or weekly
- Adequate endotracheal tube cuff pressure
 - Maintain at < 20 mm Hg or < 25 cm H₂O to not exceed capillary filling pressure of trachea.
 - Low pressure high volume cuffs typically used.
 - Inflate to assure no or minimal leak during inspiration.
 - Need for increasing air may be due to tracheal dilation or leak in cuff or pilot balloon valve (tube must be replaced if leak present).
 - Cuff pressures measured routinely every 8-12 hours and with any change in tube position.

125

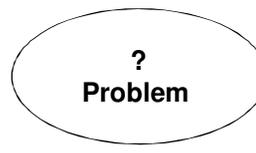
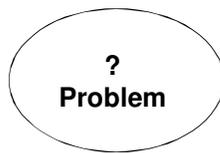
Prevention of VAP

- Subglottic suctioning prior to repositioning or deflating cuff
- Hold tube feedings if residuals > 150 cc
- Discontinue NG tubes as soon as possible
- Extubate as soon as possible
- Avoid nasal intubation
- Stress ulcer prophylaxis with sucralfate rather than H₂ blockers or proton pump inhibitors (potential advantage)
- Avoid overuse of antibiotics

126

Basic Ventilator Changes: Review

- To Change PaCO₂
 - Change _____
 - Change _____
- To change PaO₂
 - Change _____
 - Change _____



127

Ventilator Weaning

- **Spontaneous breathing trial**
 - Short period of time
 - T – Piece with CPAP or CPAP and PSV
 - If patient has not been on ventilator very long
 - Quickly need to extubate if patient tolerates
- IMV
 - Decreasing rate
 - Adding pressure support
 - If patient has been on ventilator for several days and has deconditioning of respiratory muscles

128

Minimum Weaning Parameters

- Spontaneous respiratory rate < 30 breaths per minute
- Spontaneous tidal volume: > 5ml/kg
- Vital capacity: > 10 ml/kg, ideally 15ml/kg
- Minute ventilation: < 10L
- Negative inspiratory pressure: < -25 to -30 cm H₂O
- FIO₂: < 0.50
- PaO₂ / FIO₂ ratio > 200

129

Tracheostomy

- Indications
 - Facilitate removal of secretions
 - Decrease dead space
 - Bypass upper airway obstruction
 - Prevent or limit aspiration with cuffed tube
 - Patient comfort for prolonged mechanical ventilation
- Benefits
 - Decrease laryngeal damage, swallowing dysfunction, and glottic trauma
 - Decrease in airway resistance
 - Improved ability to suction lower airways
 - Decreases risk of sinusitis
 - Improved patient comfort and mobility

130

Bedside Respiratory Monitoring: SpO₂ (Pulse Oximetry)

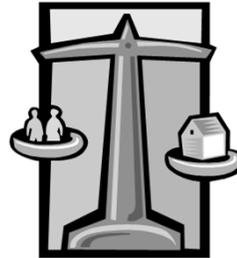
- Used to estimate oxyhemoglobin. The SpO₂ generally correlates with the SaO₂ + or - 2%.
- The goal equal to or greater than 92-94% in most patients.
 - Higher in African Americans
- Requires the presence of a pleth wave detecting an accurate pulse.
 - Patients receiving administration of high fat content such as with propofol or TPN can have a falsely high SpO₂.
- Several factors can interfere with the accuracy
 - Hemoglobin < 5 g/dL or hematocrit <15%.
 - Abnormal hemoglobin, such as carboxyhemoglobin or methemoglobin.
 - SpO₂ below 70%.
 - State of low blood flow, such as with hypotension or vasoconstriction.
 - IV dyes, fingernail polish, and some skin pigmentations

131

Bedside Respiratory Monitoring: Patient End Tidal CO₂ (PetCO₂):

- Expired CO₂ can be measured, directly at the patient and ventilator interface.
- Airway adapter should be placed as close to the patients airway as possible.
- End exhalation represents alveolar gas, and under normal circumstances, parallels PaCO₂.
- The normal gradient between PaCO₂ and PetCO₂ is 1-5 mm Hg.
- Several factors can interfere with the correlation
 - Body temperature, pulmonary disease and cardiac status.
- It can be used to detect changes over time and should be considered in patients who are undergoing deep sedation.

132



Acid –Base Balance

133

Definitions

- Acid: A substance that can give up a H⁺ ion
- Acidemia: A blood pH below 7.35
- Acidosis: The condition that causes acidemia
- Base: A substance that can accept an H⁺ ion
- Alkalemia: A blood with a pH above 7.45
- Alkalosis: The condition that causes the alkalemia

134

Acid – Base Balance

- pH
 - Indirect measurement of hydrogen ion concentration
 - Reflection of balance between carbonic acid and bicarbonate (base)
 - Inversely proportional to hydrogen ion concentration (acids donate H⁺ ions)
 - ▲H⁺ concentration = ▼pH, more acid
 - ▼H⁺ concentration = ▲pH, less acid
 - pH <6.8 or > 7.8 is incompatible with life

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Buffers

- Bicarbonate (the presence of hemoglobin makes this a much more effective buffer)
 - Bicarbonate generated by kidney
 - Aids in elimination of H⁺
- Phosphate
 - Aids in excretion of H⁺ ions by the kidneys
- Proteins

136

Acid - Base Regulation

- Respiratory System
 - Responds within minutes – fast but weak
 - Regulates the excretion or retention of carbonic acid
 - If pH is down: increase rate and depth of respiration to blow off PCO₂
 - If pH is up: decrease rate and depth of respiration to retain PCO₂

137

Acid - Base Regulation

- Renal System
 - Responds within 48 hours – slow but powerful
 - Regulates excretion or retention of bicarbonate and the excretion of hydrogen and non-volatile acids
 - If pH is down: kidney retains bicarbonate
 - If pH is up: kidney excretes bicarbonate

138

ABG Analysis

- Evaluate ventilation: PaCO₂
- Evaluate acid-base status: pH
- Evaluate source of abnormal pH: respiratory or metabolic
- Evaluate oxygenation: PaO₂, SaO₂

139

ABG Analysis: Parameters

- | | |
|--|---|
| <ul style="list-style-type: none">■ pH<ul style="list-style-type: none">• Normal 7.35-7.45• < 7.35 Acidosis• >7.45 Alkalosis | <ul style="list-style-type: none">■ PaCo₂<ul style="list-style-type: none">• Normal 35-45 mm Hg• < 35 alkalosis or respiratory compensation for metabolic acidosis• >45 acidosis or respiratory compensation for metabolic alkalosis |
|--|---|

140

ABG Analysis: Parameters

- HCO_3
 - Normal 22-26 mEq/L
 - < 22 metabolic acidosis or metabolic compensation for respiratory alkalosis
 - > 26 metabolic alkalosis or metabolic compensation for respiratory acidosis
- Base Excess (BE)
 - Normal +2 to -2
 - < -2 (base deficit) metabolic acidosis or metabolic compensation for respiratory alkalosis
 - > +2 metabolic alkalosis or metabolic compensation for respiratory acidosis

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ABG Analysis: Parameters

- Pao_2
 - Normal 80-100 mm Hg
 - >100 hyperoxemia
 - < 80 mild hypoxemia
 - < 60 moderate hypoxemia
 - < 40 severe hypoxemia
- Sao_2
 - Normal 95% or >
 - < 95% mild desaturation of HGB
 - < 90% moderate desaturation of HGB
 - < 75% severe desaturation of HGB

142

Compensation

An acidosis or alkalosis for which there has been compensation causes the pH to return to the normal range while leaning toward the initial disorder. The body never overcompensates. A non leaning pH with two abnormal indicators suggests a mixed disorder (one alkalotic and one acidotic process).

143

Anion Gap

- The anion gap is used to help determine the cause of the patient's metabolic acidosis.
- **Anion Gap = $\text{Na}^+ - [\text{Cl}^- + \text{HCO}_3^-]$**
- A normal anion gap is 12 + or - 4 mEq/L.
- An increased anion gap typically indicates an increased concentration of anions other than Cl^- and HCO_3^- .
 - Lactic acidosis
 - Ketoacidosis
 - Renal retention of anions

144

Common Causes of Respiratory Acidosis

- Depression of respiratory control centers
- Neuromuscular disorders
- Chest wall restriction
- Lung restriction
- Airway obstruction
- Pulmonary parenchymal disease

145

Common Causes of Respiratory Alkalosis

- Central nervous system disorders
- Drugs
- Hormones
- Bacteremia
- High altitude
- Over mechanical ventilation
- Acute asthma
- Pulmonary embolism

146

Common Causes of Metabolic Acidosis

- Ingested toxic substances
- Loss of bicarbonate ions
- Lactic acidosis
- Ketoacidosis
- Renal failure

147

Common Causes of Metabolic Alkalosis

- Loss of hydrogen ions
 - Vomiting
 - Diuretics
 - Steroids
- Excess bicarbonate

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Practice ABGs

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ABG Analysis Practice

- pH 7.30
- PaCO₂ 54
- HCO₃ 26
- PaO₂ 64

150

ABG Analysis Practice

- pH 7.48
- PaCO₂ 30
- HCO₃ 24
- PaO₂ 96

151

ABG Analysis Practice

- pH 7.30
- PaCO₂ 40
- HCO₃ 18
- PaO₂ 85

152

ABG Analysis Practice

- pH 7.50
- PaCO₂ 40
- HCO₃ 33
- PaO₂ 92

153

ABG Analysis Practice

- pH 7.35
- PaCO₂ 54
- HCO₃ 30
- PaO₂ 55

154

ABG Analysis Practice

- pH 7.21
- PaCO₂ 60
- HCO₃ 20
- PaO₂ 48

155

ABG Analysis Practice

- pH 7.54
- PaCO₂ 25
- HCO₃ 30
- PaO₂ 95

156



157

Acute Respiratory Failure

Failure of the respiratory system to provide for the exchange of oxygen and carbon dioxide between the environment and tissues in quantities sufficient to sustain life

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Acute Respiratory Failure

- Type I: Hypoxemic Normocapnic
 - Low PaO₂
 - Normal PaCO₂
 - Widened A-a gradient

Oxygenation Failure

- Type II: Hypoxemic Hypercapnic
 - Low PaO₂
 - High PaCO₂
 - Normal A-a gradient

Ventilatory Failure

159

Acute Respiratory Failure: Causes

- Type I (oxygenation failure)
- Pathophysiology: Decreased V/Q ratio (shunting), diffusion defect
 - Pneumonia
 - Pulmonary edema
 - Pleural effusion
 - ARDS

Alveolar / capillary exchange impacted

- Type II (acute ventilatory failure)
- Pathophysiology: Hypoventilation
 - CNS depressant drugs
 - Spinal cord injury
 - Chest trauma
 - Acute exacerbation of COPD

**Oxygen Therapy and
PCO₂ goals
in COPD**

160

COPD

- Disorders of emphysema, chronic bronchitis, and small airway disease.
- Obstructive disease causes resistance to airflow.
- Decreased expiratory airflow is central to COPD.
 - Residual volume, functional residual capacity, and total lung capacity can increase.
 - Increased resistance during forced expiration from dynamic compression.
 - FEV (expiratory airflow) 1 / FVC < .80
- Chronic inflammation of all structures of the lungs.
 - Excessive mucous secretion and ciliary dysfunction.
 - Leads to repeated damage and repair of the airways.
- Vascular changes can lead to pulmonary hypertension and subsequent acute cor pulmonale can develop.

161

Emphysema and Chronic Bronchitis

- Emphysema
 - Destruction of alveolar walls and elastic tissue support of small airways
 - Enlargement of air spaces distal to terminal bronchioles
 - **Air trapping**
 - Airway resistance increased; also loss of pulmonary capillaries
 - Decreased surface area for gas exchange
 - V/Q mismatching
- Chronic bronchitis
 - Mucous glands hypertrophy
 - Decreased cilia
 - Increased bronchial wall thickness
 - **Chronic inflammation and excessive secretions block airways**
 - Increased resistance – ventilation impairment

162

COPD: Clinical Manifestations

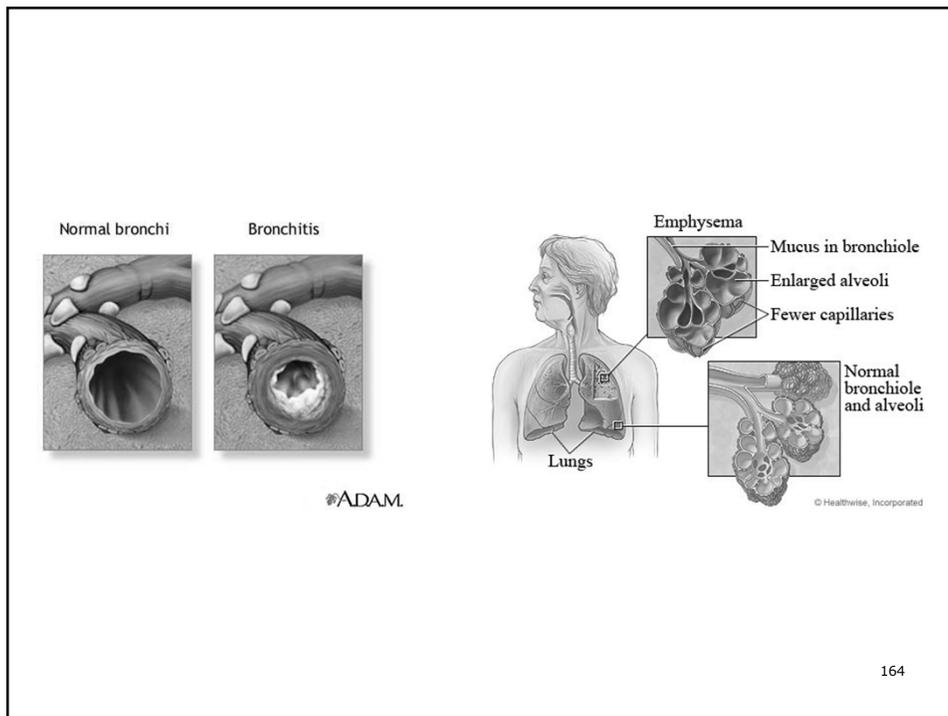
Chronic Bronchitis

- Chronic cough and sputum production daily for minimum of 3 months/year for at least 2 consecutive years
- Can have chronic hypoxemia / right sided heart failure
- Exacerbations related to infection

Emphysema

- Increased responsiveness to hypoxemia
- Dyspnea with adequate oxygenation
- Initial dyspnea on exertion
- Dyspnea at rest

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164

COPD: Clinical Manifestations

- Blended symptoms
- Large lung volumes / diminished breath sounds
- Ventilation / perfusion mismatching
- High PaCO₂ / low PaO₂
- Increase erythropoietin for increased RBCs
- Right sided heart failure

165

COPD: Treatment

- Smoking cessation
- Bronchodilators
 - Anticholinergics are the first-line medication in maintenance therapy.
 - ipratropium (Atrovent).
 - Beta-agonists can be added
 - Short acting
 - racemic albuterol (Ventolin, Proventil, Accuneb).
 - levalbuterol (Xopenex).
 - metaproterenol (Alupent).
 - pirbuterol (Exirel, Maxair).
 - Long acting
 - salmeterol (Serevent).
 - formoterol (Foradil, Oxeze).
 - Theophylline is a long acting weak bronchodilator.

166

COPD: Treatment

- Antibiotics - acute exacerbations can be caused by bacterial infections.
- Corticosteroids: Remains controversial, but they are frequently used in treating exacerbations. Steroids are used as part of chronic treatment in some patients. Corticosteroids can also be combined with other medications.
 - budesonide (Pulmicort)
 - fluticasone and salmeterol (Advair)
- Expectorants/mucolytics.

COPD: Treatment

- Oxygen (Can improve survival in patients who are hypoxemic)
 - Criteria
 - Room air: PaO₂ < 55 mm Hg with saturation < 85%. PaO₂ 56-59 and saturation 86-89%, with a qualifying secondary diagnosis.
 - Goal of oxygen therapy is to obtain PaO₂ of 65-80 mm Hg while awake and at rest.
 - Typically delivered at 1-4 L/min, with an increase of 1 L during sleep and exercise.
 - Should be given continuously at least 19 hours of each day.
- Pneumonia and influenza vaccines

Case Example

- Patient history: COPD (CO₂) retainer
- Initial presentation: Tachypneic with SaO₂ of 78%

- Cause of exacerbation ?
- Initial interventions?

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Case Example

- ABG
 - 7.29
 - PaCO₂ 60
 - HCO₃ 30
 - PaO₂ 48

- Treatment options?
- Goals for ABG values?

170

Status Asthmaticus

Exacerbation of acute asthma characterized by severe airflow obstruction that is not relieved **after 24 hours of maximal doses of traditional therapy**

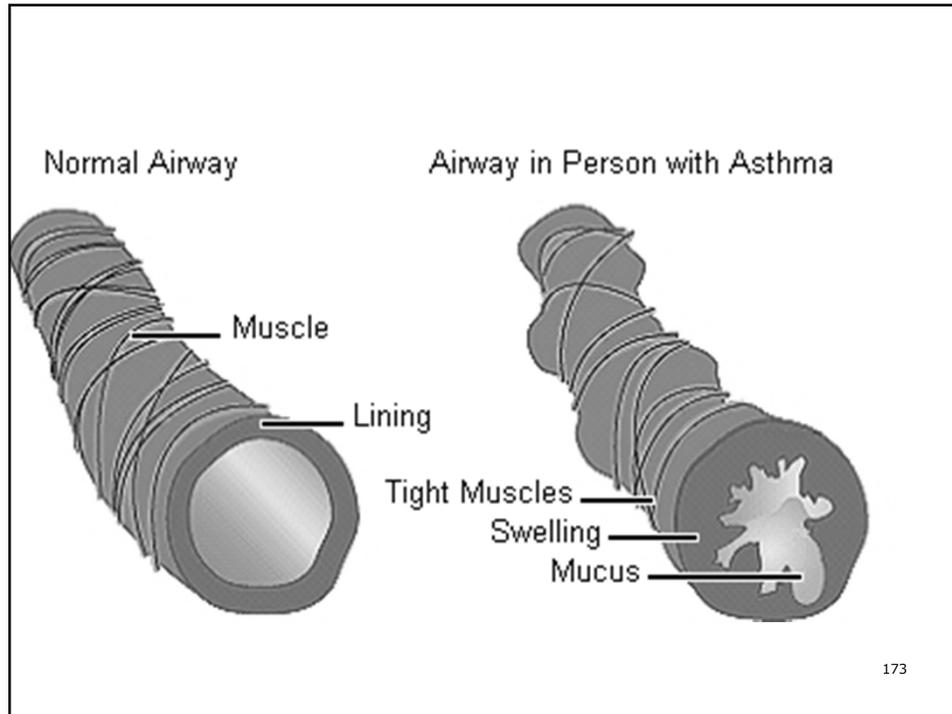
Characterized by expiratory wheezing

171

Status Asthmaticus: Etiology

- Extrinsic (specific allergy can be related to attack)
 - Pollen
 - Dust
 - Pets
 - Smoke
 - Food
 - Drugs
- Intrinsic (attack is seemingly unrelated to an allergen)
 - Infection
 - Stress
 - Exercise
 - Aspiration

172



Status Asthmaticus: Pathophysiology

- Trigger (extrinsic or intrinsic)
- Intrinsic trigger causes imbalance of sympathetic and parasympathetic nervous systems
- Extrinsic: IgE released ► histamine and slow-reacting substance of anaphylaxis (SRS-A)
- Histamine ► swelling and inflammation of smooth muscle of large bronchi (and mucous membrane swelling)
- Swelling of smooth muscle of small bronchi and release of prostaglandins (enhance histamine)

174

Status Asthmaticus: Pathophysiology

- Histamine causes excessive secretion of mucous ► narrows the airway lumen
- Tachypnea increases insensible water loss ► thicker secretions
- Mucous in small airways
- Increased work of breathing (**impaired ventilation**) (**Note: ▲PaCO₂ is late sign**)

175

Status Asthmaticus: Treatment

- Eliminate or treat cause
- Steroids
- Need to ventilate when PaCO₂ becomes elevated
- Additional similar treatment as pneumonia

176

Pulmonary Embolism

- Obstruction of blood flow to one or more arteries of the lung by a thrombus (other emboli – fat, air, amniotic fluid) lodged in a pulmonary vessel
- Lower lobes frequently affected due to increased perfusion

177

Risk Factors for PE

- Stasis of blood
 - Prolonged immobilization after surgical procedures
 - Plaster casts
 - Venous obstruction
 - Heart failure / Shock / Hypovolemia
 - Varicose veins
 - Obesity
- Hypercoagulability
 - Polycythemia vera
 - Sickle cell disease
 - Malignancy
 - Pregnancy
 - Recent trauma
 - Oral contraceptives
- Injury to the vascular endothelium
 - Central venous and arterial catheters
 - Phlebitis

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Pulmonary Embolism: Pathophysiology

- > 90% of thrombus develop in deep veins of iliofemoral system
 - Can also originate in the right side of the heart, pelvic veins, and axillary or subclavian veins.
 - Another source is around indwelling catheters.
- Thrombus formation leads to platelet adhesiveness and release of serotonin (vasoconstrictor).
- Dislodgement of thrombus
 - Intravascular pressure changes (standing, massaging legs, fluid challenge, valsalva maneuver).
 - Natural clot dissolution (7-10 days after development).

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Pulmonary Embolism: Pathophysiology

- Clot lodges in pulmonary vessels
- Ventilation continues but perfusion decreases
 - Increase in alveolar dead space
 - Alveolar CO₂ decreases
- Overperfusion of uninvolved lung results in a decreased V/Q ratio
 - Hypoxemia can occur due to ventilation / perfusion mismatching.
- Decreased blood flow damages type II pneumocytes, which results in a decrease in surfactant production. (atelectasis)
 - Pulmonary edema can develop as secondary complication
- Increased PVR can lead to pulmonary hypertension and potential acute cor pulmonale.
- Cardiogenic shock can occur as the result of right-ventricular failure.

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Pulmonary Embolus: Clinical Presentation

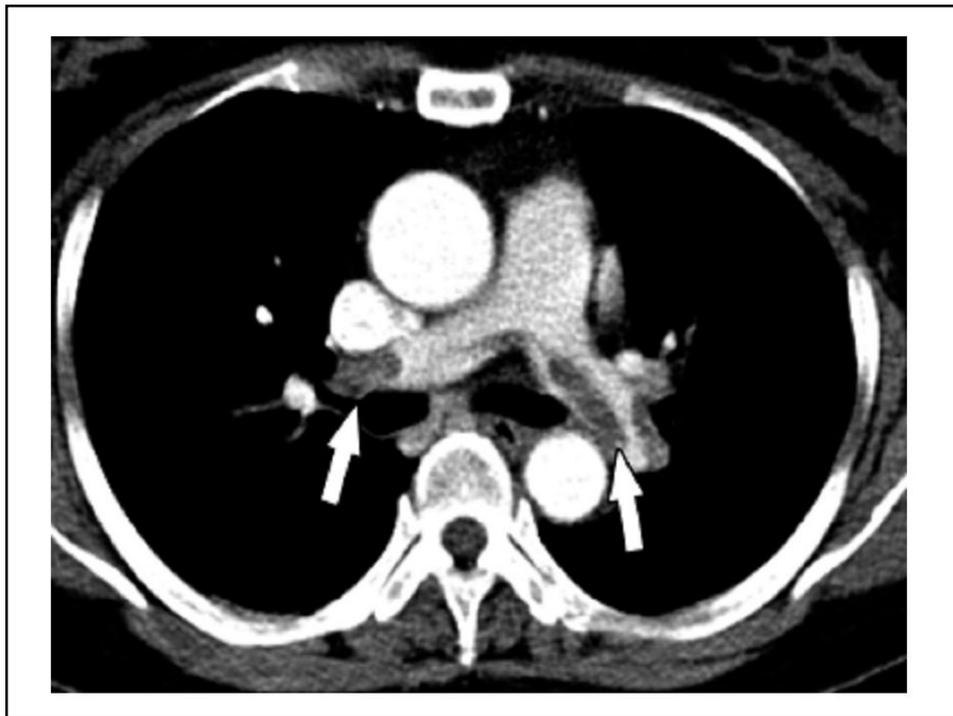
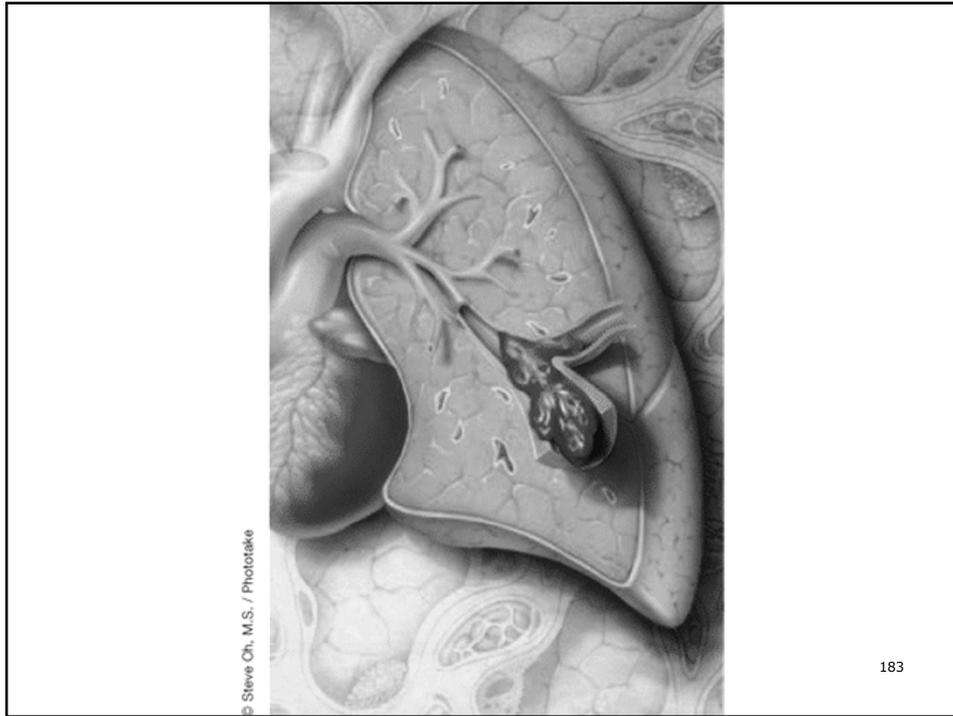
- | | |
|---|---|
| <ul style="list-style-type: none"> ■ Large to massive when 50% of pulmonary artery bed is occluded <ul style="list-style-type: none"> • Impending doom • Hypoxemia • Syncope • Sign and symptoms of right heart strain or right-ventricular failure • Signs of right-ventricular strain on ECG. • Sudden shock • Pulseless electrical activity | <ul style="list-style-type: none"> ■ Medium-sized emboli <ul style="list-style-type: none"> • Dyspnea • Substernal chest discomfort/pleuritic chest pain • Many non-specific signs • Tachypnea • Tachycardia • Rales • Accentuated 2nd heart sound |
|---|---|

181

Pulmonary Infarction

- | | |
|---|--|
| <ul style="list-style-type: none"> ■ Pulmonary infarction is infrequent ■ More common <ul style="list-style-type: none"> • Large embolus • Pre-existing lung disease ■ Results in alveoli filling with RBCs and inflammatory cells ■ Complicated by infection <ul style="list-style-type: none"> • Abscess | <ul style="list-style-type: none"> ■ Signs and Symptoms <ul style="list-style-type: none"> • Pleuritic chest pain • Dyspnea • Hemoptysis • Cough • Pleural friction rub |
|---|--|

182





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Pulmonary Embolus: Treatment

- Prevent thrombus formation
 - Compression stockings that provide a 30-40 mm Hg or higher gradient
 - Low molecular weight heparin
- Heparin is the treatment of choice for reducing mortality in PE
 - Initiated prior to a confirmed diagnosis
 - Slows or prevents clot progression and decreases risk of further emboli
- Fibrinolytic therapy
 - Indicated in patients with hypotension (even if resolved), hypoxemia, or evidence of right-ventricular strain
 - Troponin levels can also be used to guide decision-making in patients with sub-massive PE
- Pulmonary embolectomy is a surgical option when fibrinolytic therapy is contraindicated.

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Pulmonary Embolus: Treatment

- Oxygen is indicated, even in the absence of hypoxemia
- Pulmonary vasodilators to help reduce pulmonary vascular resistance
- Treat right-ventricular failure with fluids and inotropes
- **Obstructive Shock**
- Warfarin
 - 3 to 6 months if there is identifiable reversible risk factor
 - Minimum of 6 six months if there is no identifiable risk factor
 - Long term with recurrent PE or in patients with ongoing risk factors
- Surgical interruption of inferior vena cava with a filter
 - Patients with contraindication to anticoagulants.
 - Recurrent thromboembolism despite⁶⁸ anticoagulant.

Special Considerations Fat Emboli

- **Risk Factors:**
 - Skeletal Trauma: femur and pelvis)
 - Major orthopedic surgery
 - 24 to 72 hours post insult
- **Signs and Symptoms:**
 - Vague chest pain
 - Shortness of breath
 - Sudden restlessness – drowsiness
 - Fever
 - Petechiae (transient – axillary or subconjunctival)
- Release of free fatty acids causes endothelial injury and toxic vasculitis
- Hemorrhage into lungs (**decrease H&H and platelets**)
- CXR pattern similar to ARDS
- Steroids

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Special Considerations Air Emboli

- Large volume of air into venous system
- **Risk Factors**
 - Dialysis
 - Pulmonary artery catheters
 - Surgical procedures
 - CABG
- **Symptoms**
 - Dyspnea, chest pain, agitation, confusion, cough
- **Treatment**
 - Prevent
 - 100% oxygen
 - Left lateral / trendelenburg
 - Positive pressure ventilation
 - Aspiration of air

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Pulmonary Edema

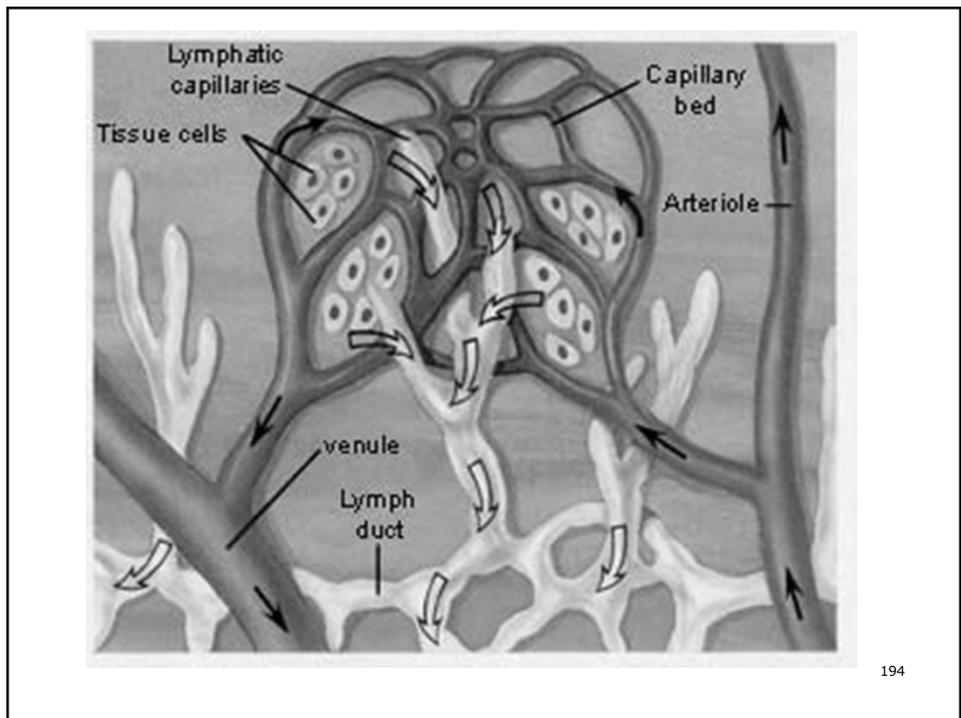
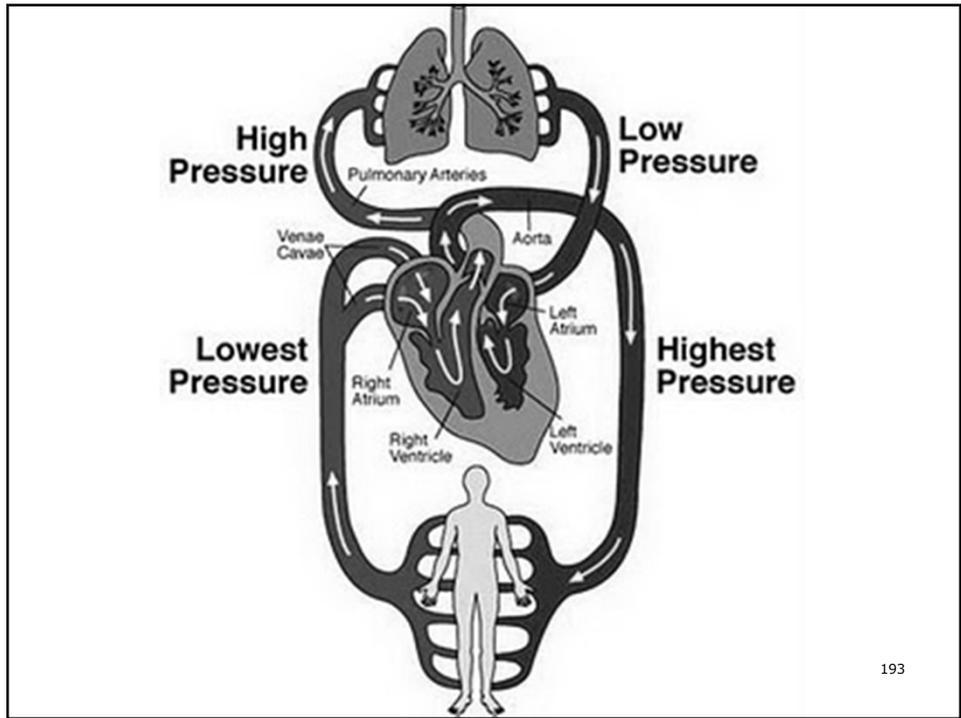
- **Extra vascular accumulation of fluid in the lungs (cardiac or non cardiac)**
 - Results in impaired diffusion of oxygen due to increase in interstitial space
 - Results in decreased V/Q ratio due to poorly ventilated fluid filled alveoli
 - Fluid in alveoli also impacts compliance of lungs and therefore ventilation
- Capillary endothelium more permeable to water and solute than alveolar endothelium
- Edema accumulates in the interstitium before the alveoli

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Pulmonary Edema

- Fluid in pulmonary interstitium is removed by lymphatic drainage of the lung
- Volume of lymph flow from the lung can increase ten fold in pathological conditions
- Only when this large safety factor is taxed does pulmonary edema occur

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Pulmonary Edema: Risk Factors and Treatment

- Loss of integrity of alveolar capillary membrane
 - Infection
 - Inhaled toxins
 - Oxygen toxicity
- Increase in pulmonary capillary hydrostatic pressure
 - Left sided heart failure
 - Excessive fluid administration
 - Occlusion of pulmonary vein

Other: Blockage of lymphatic system

- Cardiac pulmonary edema is treated as acute decompensated heart failure.
- Non cardiac pulmonary edema is treated like ARDS.

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Pneumonia

- Acute infection of the lung parenchyma, including alveolar spaces and interstitial space
- Causes:
 - Bacteria (Community acquired versus Hospital acquired)
 - Virus
 - Fungi
 - Parasites
 - Mycoplasma

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Risk Factors for Bacterial Pneumonia

- Previous viral respiratory infection
- Gastro esophageal reflux disease (GERD)
- Chronic alcohol abuse
- Cigarette smoking
- Decreased level of consciousness
- Anesthesia
- Intubation
- Lung disease
- Diabetes mellitus
- Use of corticosteroids
- Elderly

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Pneumonia: Pathophysiology

- Causative agent is inhaled or enters pharynx via direct contact
- Alveoli become inflamed
- Alveolar spaces fill with exudate and consolidate
- Diffusion of O₂ obstructed
 - Hypoxemia.
- Goblet cells are stimulated to increase mucous
 - Increased airway resistance and work of breathing

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Pneumonia: Causative Agents

- Common agents in community-acquired pneumonia (younger and healthier population)
 - *Streptococcus pneumoniae* (most common agent in community acquired pneumonia).
 - *Mycoplasma pneumoniae*.
 - *Chlamydia pneumoniae*
 - Viral.
- *Haemophilus influenzae* common among smokers
- *Klebsiella pneumoniae* in patients with chronic alcoholism
- Agents in the older population commonly include gram negative bacilli
 - *Moraxella catarrhalis* (particularly common in patients with chronic bronchitis).
 - *Staphylococcus aureus* (in the setting of post viral influenza).
- Methicillin-resistant *Staphylococcus aureus* (MRSA) also as a cause of community-acquired pneumonia

Hospital Acquired Pneumonia

Causative agents

- Aerobic gram negative rods
 - *Klebsiella* sp.
 - *Pseudomonas* sp.
 - *Enterobacter* sp.
 - *Escherichia coli*.
 - *Proteus* sp
 - *Serratia* sp.
 - Enterococci.
- *Staphylococcus aureus* (including methicillin-resistant *Staphylococcus aureus* [MRSA])
- Group B streptococci

- Nosocomial pneumonia is typically caused by bacterial agents that are more resistant to antibiotic therapy.

Sources

- Contamination of pharynx and perhaps stomach with bacteria
- Repeated small aspirations of oral pharyngeal secretions.
- Retrograde contamination from GI tract.

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Pneumonia: Clinical Presentation

- Flu-like symptoms.
- Pleuritic chest pain.
- Confusion in elderly.
- Tachycardia, tachypnea, fever.
- Crackles and wheezes.
- Productive cough.
- Clinical signs of dehydration.

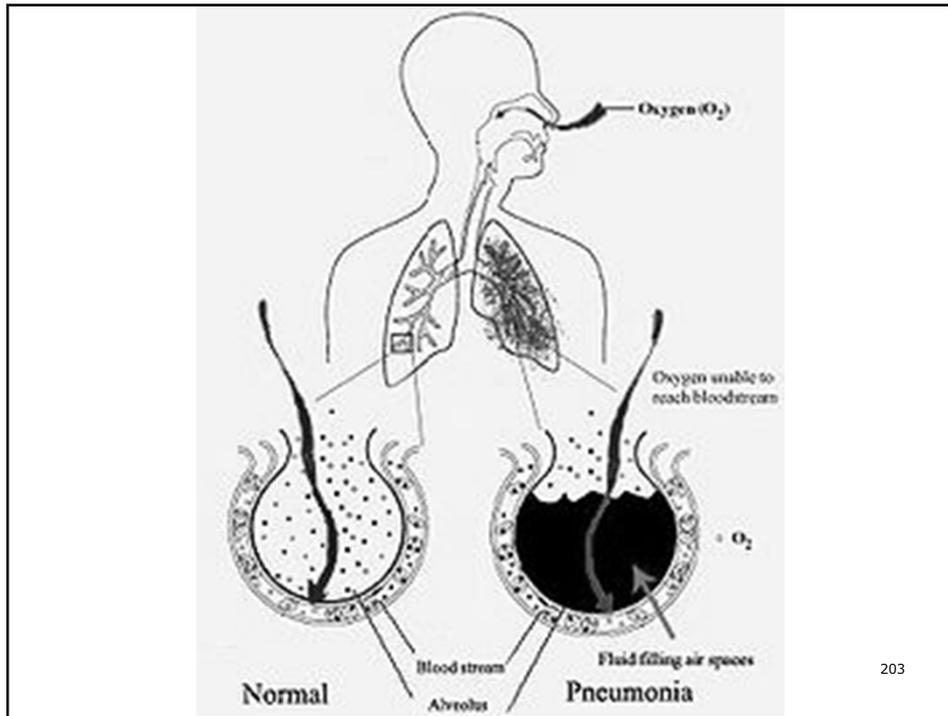
The clinical presentation in the elderly may be more subtle including confusion, dehydration, and fever.

201

Diagnosis of Pneumonia

- Sputum gram stain
- Sputum culture
- Blood cultures (bacteremia not present in most)
- Leukocytosis / Shift to left of WBCs.
 - Leukocytosis and a left shift is expected in bacterial pneumonia.
 - Failure of the white blood cell count to rise in the presence of a bacterial infection is associated with an increased mortality
- Blood gases/oxygen saturation
- Chest x-ray – produces variable results but infiltrates are frequently seen
 - A chest CT may also be used to aid in the

202



Complications of Pneumonia

- Abscesses may form and rupture into pleural space leading to pneumothorax and/or empyema
 - Video assisted thoracoscopy with debridement is a treatment option for empyema in the early organizing phase
 - Full thoracotomy with decortication may be necessary in later organizing phases
- Pleural Effusion
- Acute respiratory failure
- ARDS
- Sepsis

Mortality rates for nosocomial or hospital-acquired pneumonia are higher than those for community acquired pneumonia (particularly in the elderly) 205

Pneumonia: Treatment

- **Prevent nosocomial infections**
- Timely Antibiotics
- Hydration (Electrolyte Monitoring)
- Deep breathing / incentive spirometry
- Bronchodilators, expectorants, mucolytics
- Avoid: sedatives and antitussives
- Early activity and mobility (DVT Prophylaxis)

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Aspiration

- Vomiting or regurgitation
- Large particles – airway obstruction
- pH of liquid determines injury
 - pH < 2.5 or large volume
 - Chemical burns destroy type II cells
 - May induce bronchospasm
 - Increase alveolar capillary membrane permeability
 - Decrease compliance
 - Decrease V/Q ratio

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Aspiration

- Non acidic aspiration
 - More transient
- Food stuff / small particles
 - Inflammatory reaction
 - Hemorrhagic pneumonia within 6 hours
- Contaminated material with bacteria can be fatal

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Aspiration: Possible Prevention Strategies

- Avoiding sedation.
- Resting prior to meal time.
- Eating slowly.
- Flexing the head slightly to the “chin down” position.
- Determining food viscosity best tolerated (thickening liquids will improve swallowing in some patients).

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Acute Respiratory Distress Syndrome

A syndrome of acute respiratory failure characterized by non-cardiac pulmonary edema and manifested by refractory hypoxemia. ARDS does not include mild or early acute lung injury, but rather involves severe and diffused lung injury.

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Risk Factors in ARDS

- Sepsis (most common)
- Transfusion
- Aspiration
- Trauma
- Massive transfusion
- Pancreatitis

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Acute Respiratory Distress Syndrome: Etiology

- | | |
|--|--|
| <ul style="list-style-type: none"> ■ Direct lung injury <ul style="list-style-type: none"> • Chest trauma • Near drowning • Smoke inhalation • Pneumonia • Pulmonary embolism | <ul style="list-style-type: none"> ■ Indirect lung injury <ul style="list-style-type: none"> • Sepsis • Shock • Multi system trauma • Burns • CABG • Head injury |
|--|--|

Time from injury of alveolar capillary membrane to onset of symptoms is 12-48 hours.

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ARDS: Pathophysiology

- Stimulation of **inflammatory** and immune systems
- Release of toxic substances, causing **micro vascular injury**
- Pulmonary capillary membranes are damaged
 - **Increase in capillary permeability.**
- Cells and fluids leak into interstitium and alveolar spaces
 - **Pulmonary Edema**
- Impaired production and **dysfunction of surfactant**
 - **Alveolar collapse** and massive atelectasis.
- **Intrapulmonary shunting**
- Hypoxic vasoconstriction
- **Decreased the compliance of lung**
 - High-peak inspiratory pressures to ventilate the lungs.
- Potential development of **pulmonary fibrosis** in chronic phase.
 - Endothelium, epithelium, interstitial space expand.
 - Protein exudate inside the alveoli produces a hyaline membrane.

Acute Respiratory Distress Syndrome: Diagnosis

- Predisposing condition
- PaO₂/FIO₂ ratio < 200
- Chest x-ray: Diffuse bilateral infiltrates (Chest CT may also be used)
- Decreased static compliance of lungs
- PAOP < 18 mm Hg or no evidence of increased left-atrial pressure
- No evidence of COPD
- No other explanation for above

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ARDS: Treatment

- Optimal ventilation / oxygenation
- Avoid over hydration
- No routine use of steroids
- Pulmonary vasodilators

**High Mortality
Persists so
Prevention
Remains Key**

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Drugs Used to Decrease Right Sided Afterload / Treat Pulmonary Hypertension

- Oxygen
- Pulmonary vasodilators
 - NTG
 - Sodium nitroprusside
 - Inhaled nitric oxide
 - See medications used in the treatment of pulmonary artery hypertension

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Mechanical Ventilator Management Strategies for ARDS

- Lower tidal volume ventilation
 - Permissive hypercapnia
- Maintain plateau pressure < 30 mmHg

- Uninterrupted PEEP
- Avoidance of auto PEEP

- Airway pressure release ventilation
- High frequency ventilation (Oscillatory)
- Independent lung ventilation
- ECMO

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Case Example

- 65 year old female; 85 kg
- Post witnessed cardiac arrest
- Initial PaO₂ / FIO₂ ratio 102

- Initial diagnosis?

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Case Example

- Ventilator settings:
 - AC
 - Rate 12
 - TV 700 ml
 - FIO2 80%
 - PEEP 5 cm
- 2nd ABG
 - pH - 7.33
 - PaCO2 - 40 mmHg
 - HCO3 - 14
 - PaO2 - 92
- Ventilator adjustment?
- Other treatment considerations?

219

Pulmonary Hypertension

- PPH (primary pulmonary hypertension)
- IPAH (idiopathic pulmonary arterial hypertension)
- Rare disease
- Perhaps caused by insult to endothelium in patient with susceptibility to pulmonary vascular injury
 - Vascular scarring
 - Endothelial dysfunction
 - Intimal and medial smooth muscle proliferation

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Pathophysiology

- Early: Pulmonary artery pressures continue to increase due to increasing work of right ventricle
- Then: Thrombotic pulmonary arteriopathy
 - In situ thrombosis of small muscular pulmonary arteries
- Later: Plexogenic pulmonary arteriopathy
 - Remodeling of pulmonary vasculature with intimal fibrosis

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Associated Conditions

- Portal hypertension
- Connective tissue diseases
 - Associated
- Anorexigens
- Alpha adrenergic stimulants (i.e. cocaine / amphetamines)
- HIV

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- IPAH has no cure
- Untreated leads to right sided heart failure and death
- New drugs have improved survival rates
 - Prostacyclin analogues
 - Endothelin receptor antagonists
- APAH occurs more frequently than IPAH
- 15-20% have familial component
- Females are affected more than males
 - Women of child bearing age more often affected.

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Presentation

- Average time from symptom onset to diagnosis is 2 years
- Most common symptoms in one study:
 - Dyspnea (60%)
 - Weakness (19%)
 - Recurrent syncope (13%)
- Possible Physical finding:
 - Increased pulmonic component of 2nd heart sound
 - Palpable 2nd heart sound
 - Murmurs of pulmonic and tricuspid regurgitation
 - Right ventricular heave
 - JVD
 - Large V waves
 - Other signs of right heart failure
 - Normal lungs

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Treatment

- **General**
 - Anticoagulation (Warfarin)
 - Digoxin
 - Diuretics
 - Oxygen with hypoxemia
 - May be of no benefit in left to right shunt
- **Conventional Pulmonary Vasodilators**
 - Calcium channel blockers
 - Nifedipine / diltiazem
 - Only used in patients who are responders to acute vasodilator testing (25%)
 - High doses are used
 - Can have rebound pulmonary hypertension when withdrawn
 - Only in patients without overt right sided heart failure

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Approved Pulmonary Vasodilators for PPH

- **Epoprostenol (Flolan)**
 - IV - Parental
 - Prostacyclin analogue (prostanoid)
- **Treprostinil (Remodulin)**
 - IV / SQ - Parental
 - Prostacyclin analogue (prostanoid)
- **Iloprost (Ventavis)**
 - Nebulized inhalation
 - Prostacyclin analogue (prostanoid)

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Approved Pulmonary Vasodilators for PPH

- Bosentan (Tracleer)
 - Oral
 - Endothelin antagonist (ERA)
- Ambrisentan (Letairis)
 - Oral
 - Endothelin antagonist (ERA)
- Sildenafil (Revatio)
 - Phosphodiesterase (type 5) enzyme inhibitor
 - Oral

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Other Treatment Options

- Single or double lung transplant (cardiac transplant may or may not be needed)
- Atrial septostomy (palliative)
 - Creates right to left shunt
- Cardiopulmonary rehab for mild symptom limited aerobic activity

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Closed (Simple) Pneumothorax

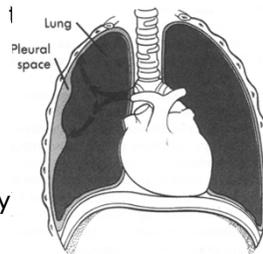
- Air enters the intra pleural space through the lung causing partial or total collapse of the lung
 - Between visceral and parietal pleura
- **Possible etiology**
 - Primary (no underlying lung disease)
 - Blebs / bullae
 - Smoking
 - Secondary (underlying lung disease)
 - Air enters damaged aveoli
 - COPD
- Blunt trauma (lung laceration by rib fracture)
- Positive pressure ventilation (rupture of weak alveoli, bleb or bullous)
- Iatrogenic – from medical procedure

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Closed (Simple) Pneumothorax

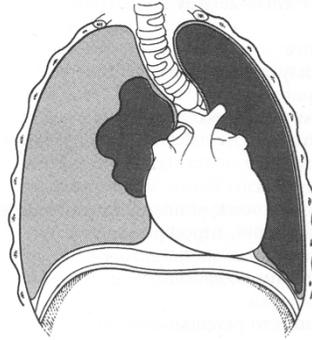
- **Pathophysiology**
 - Disruption of normal negative intrapleural pressure
 - Lung collapse
 - Decreased vital capacity
 - Decreased surface area for gas exchange
 - Acute respiratory failure (particularly secondary)
- **Signs and Symptoms**
 - Chest pain, dyspnea, cough, tachycardia
 - Asymmetrical chest excursion, diminished or absent breath sounds on affected side, dramatic increases in peak inspiratory pressures on a mechanical ventilator
- **Treatment**
 - Oxygen
 - Analgesics
 - Observation (asymptomatic, small primary)
 - Aspiration (symptomatic small primary)
 - Chest Tube Criteria
 - Secondary

Spontaneous primary pneumothorax may take 12 weeks to resolve.



Tension Pneumothorax

- Accumulation of air into the pleural space without a means of escape causes complete lung collapse and potential mediastinal shift
- **Etiology**
 - Blunt trauma
 - Positive pressure mechanical ventilation
 - Clamped or clotted water seal drainage system
 - Airtight dressing on open pneumothorax



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Tension Pneumothorax

- Pathophysiology
 - Air rushes in-cannot escape pleural space
 - Creates positive pressure in pleural space
 - Ipsilateral lung collapse
 - Mediastinal shift \rightleftarrows contralateral lung compression \rightleftarrows potential tearing of thoracic aorta
 - Can also compress heart \rightleftarrows decrease RV filling \rightleftarrows shock

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Tension Pneumothorax

Signs and Symptoms

- Similar to closed pneumothorax
- If mediastinal shift:
 - Tracheal shift away from affected side
 - JVD
 - Hypotension

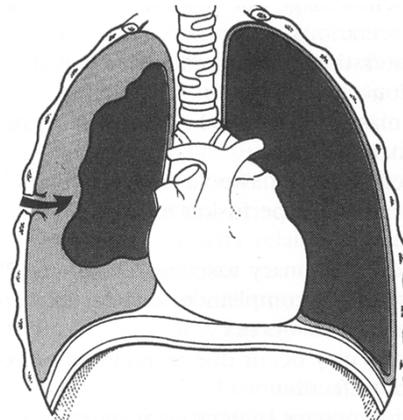
Treatment

- Oxygen (100%)
- Emergency decompression
- Chest Tube
- Other as with closed pneumo

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Open Pneumothorax

- Air enters the pleural space through the chest wall
- **Etiology**
 - Penetrating Trauma



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Open Pneumothorax

- **Pathophysiology and Signs and Symptoms**
 - Equilibrium between intra thoracic and atmospheric pressures
 - Patient condition depends on size of opening compared to trachea
 - The affected lung collapses during inspiration
 - May cause a tension pneumothorax
 - Subcutaneous emphysema usually present
- **Treatment**
 - Similar to closed pneumothorax
 - Closure of open wound with petroleum jelly gauze
 - End expiration
 - Modification for tension pneumothorax
 - Chest tube and water seal drainage²³⁵



Reflection

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