Understanding the Pulmonary System: Ventilation, Oxygenation, & Perfusion.
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Presentation Summary
- This session will review the importance of the pulmonary system as it relates to the principles of oxygenation versus ventilation. Various pathological disease processes such as pneumonia, atelectasis, pulmonary edema, and obstructive disorders seriously affect the ability of the lungs to function properly furthering impairing patient outcomes.

Learning Objectives:
1. Discuss the pulmonary anatomy reviewing the principles of oxygenation versus ventilation.
2. Describe the various ventilation/perfusion mismatches that result from various disease states.
3. Discuss interventions to improve both ventilation and perfusion

Function of the Pulmonary System

Gas Exchange: Semi-permeable membrane allows gas exchange according to PRESSURE GRADIENTS
Acinus: terminal respiratory unit

Alveolar Cells
- Type I: Squamous epithelium
  - GAS EXCHANGE
- Type II:
  - Manufacture surfactant which prevents alveolar collapse
  - Surfactant: Decreases work of breathing
    - Allows alveoli to stay open at low distending pressures
    - DECREASED SURFACANT MAKES LUNG EXPANSION DIFFICULT

Alveolar Ventilation
- Minute Ventilation: Amount of air exchanged in 1 minute
  - Composed of both alveolar & dead space ventilation
    - Tidal Volume x Respiratory Rate = Min Ventilation
      - 500 ml x 12 = 6000 ml (6 L)
  - 6 L is the normal resting minute ventilation in adults

Ventilation
- Alveolar ventilation isn’t directly measured
- INVERSELY RELATED TO ARTERIAL CO₂ (PaCO₂)
  - Normal CO₂ = adequate alveolar ventilation
  - Decreased CO₂ = increased alveolar ventilation
    - HYPERVENTILATION
  - Increased CO₂ = decreased alveolar ventilation
    - HYPOVENTILATION
**Pulmonary Elastic Resistance**
- Lungs collapse due to elastic recoil. Chest wall expansion prevents collapse

**Compliance:**
determined by change in lung volume by change in pressure
- If compliance is high, the lung is more easily distended
- If compliance is low, lung is more difficult to distend (stiff)

**Oxyhemoglobin Dissociation Curve:**
- Describes the relationship between O\(_2\) saturation & the PaO\(_2\)
- Depicts the ability of hemoglobin to bind O\(_2\) at normal PaO\(_2\) levels & release O\(_2\) at low PaCO\(_2\) levels

**Respiratory Failure**
- Respiratory system unable to perform one or both of its main functions:
  - Oxygenation
  - Removal of carbon dioxide
- Adequacy of gas exchange determined by:
  - Balance between pulmonary ventilation & capillary blood flow
- Characterized by ABG abnormalities
  - Hypoxemia: PaO\(_2\) < 55 mmHg
  - Hypercarbia: pCO\(_2\) > 50 mmHg

**Etiologies**

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<th>Pulmonary</th>
<th>Neurological</th>
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<td>Flail Chest</td>
<td>Pneumonia</td>
<td>Guillain-Barre</td>
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<td>Pulmonary Contusion</td>
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<td>Spinal Cord Injury</td>
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<td>Pleural Effusion</td>
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**Dead Space Ventilation**
- 2 Types:
  - **Anatomic Dead Space:** Gas in the large conducting airways (pharynx, trachea, bronchi, etc)
  - **Physiologic Dead Space:** alveolar gas that does not fully participate in gas exchange with capillary blood
    - Ventilation without perfusion

**Dead Space Ventilation**
- Increases when:
  - Alveolar-capillary interface is destroyed (i.e.: COPD)
  - Blood flow is reduced (i.e.: cardiogenic shock)
  - Alveoli are distended (i.e.: positive pressure ventilation)
- Normal V\(_D\) accounts for 20-30% of V\(_T\)
- Hypercapnea occurs when V\(_D\) > 50% V\(_T\)

**Intrapulmonary Shunt**
- Capillary blood flow that occurs in excess to ventilation
- The extra blood volume doesn’t participate in gas exchange
- Perfusion without ventilation
- Increases with:
  - Small airway occlusion (asthma)
  - Fluid filled alveoli (pulmonary edema, pneumonia)
  - Alveolar collapse (atelectasis)
  - Excess capillary blood flow (areas of lung without perfusion in a patient with PE)
**Diagnostic Tests**
- ABG, CXR
- Chest CT/CTA
- CBC, BMP, sputum cultures
- Cardiac enzymes & EKG if AMI suspected
- Find & treat the underlying cause!!!

**Acute Respiratory Failure Treatment:**
- High flow oxygen
  - Non-invasive positive pressure ventilation (CHF exacerbation)
- Mechanical ventilation (pneumonia, ARDS, severe chest trauma)
- Antibiotics (pneumonia)
- DVT prophylaxis, stress ulcer prophylaxis
- Supportive care: nutrition, hydration, oral care, turning
- Diuretics (pulmonary edema)

**Ventilation Abnormalities**
- Obstructive – inability to exhale, air trapping, prolonged exhalation time, barrel chest
  - Asthma
  - Chronic Bronchitis
  - Emphysema
- Restrictive – inability to inhale, Stiff, non-compliant lungs
  - Structural abnormality
  - Atelectasis
  - Pneumonia
  - Pneumothorax
  - Pulmonary edema
  - Pulmonary fibrosis
  - ARDS

**Pathophysiology of Ventilation/Perfusion abnormalities**

**Strategies**
- *Improve oxygenation: PEEP*
  - Improves PaO2 without increasing FiO2, decreases surface tension, decreases intrapulmonary shunt
  - Uses: severe hypoxemia, ARDS, drowning, sepsis, acute respiratory failure
  - Adverse effects: hemodynamic changes, baro-trauma, Contraindicated with untreated hypovolemia
**Oxygenation**
- $\text{FiO}_2$
  - Increases FRC
  - Increasing $\text{FiO}_2$ or PEEP will increase oxygenation

**Improve removal of CO2: improve ventilation**
- Frequency (rate)
- Tidal volume
- Flow rate (compliance, alveoli recoil)

**Ventilation**: Removes CO$_2$ from Blood
- Minute ventilation = TV x RR
  - Adjusting TV or RR will INVERSELY affect CO$_2$
- Increasing either TV or RR will decrease CO$_2$ & improve ventilation
- Decreasing either TV or RR will increase CO$_2$ & decrease minute ventilation