Issues in Oxygenation: Understanding the Cardiopulmonary Circuit

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Swimmers never take a breath for granted!

Nurses never take a life for granted!
Pulmonary Physiology

Physiology of Pulmonary System

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

Ventilation

- Minute ventilation ($V_E$) = Total volume of air expired in one minute
  - Respiratory rate x tidal volume
  - Normal minute ventilation = $12 \times 500 \text{ ml} = 6000\text{ml}$
  - Note: (hypoventilation can occur with normal or even high respiratory rate)
Alveolar Ventilation ($V_A$)

- $V_A = V_T - \text{anatomical dead space}$
- 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:** Resistance
  - 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
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
Lung Volumes

Measurements including RV are made by helium dilution or body plethysmography, not spirometry.
Ventilation

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

Resistive work of breathing greatest during forced expiration.

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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
  - CPAP
  - PEEP (positive expiratory pressure)
Assessment of Ventilation

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

**Note:** Only one physiologic reason for increased PaCO$_2$.

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 patient’s 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.
- **Capnography includes both the continuous analysis and the continuous recording of the CO$_2$**
Treatment of Ventilation Problems

VENTILATION PROBLEMS ARE TREATED BY RATE AND $V_T$

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 $O_2$
100 (104) mmHg

Inspired gas $PIO_2$
149 mm Hg.
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.

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
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?

Perfusion
Perfusion

Definition: The movement of blood through the pulmonary capillaries

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
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.

Zones of Perfusion

- 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.
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.

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.
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.

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?
Prior to Diffusion

- Ventilation and Perfusion Occur Simultaneously
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**

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
Assessment of Diffusion

- **PaO\textsubscript{2} and oxygen saturation (SaO\textsubscript{2})**
  - However, a simple diffusion problem rarely results in hypoxemia at rest.

- **Clinical Application**: CO\textsubscript{2} is 20 times more diffusible than O\textsubscript{2} - so a diffusion problem causing hypoxemia does not result in the same problem with CO\textsubscript{2} retention (hypercapnia)
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.

Factors Affecting Accuracy of SpO2 (Pulse Oximetry)

- 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 pigmentation.
- Patients receiving administration of high fat content such as with propofol or TPN can have a falsely high SpO2.
Increased $\text{FIO}_2$ and increased PEEP will increase driving pressure of oxygen.

**Diffusion Review**

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

**Assessment and Treatment**

- **Ventilation problems**
  - Assessed by:
  - **Corrected with?**

- **Diffusion problems**
  - Assessed by:
  - **Corrected with?**

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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.
Ventilation / Perfusion Ratio (V/Q)

- **Ventilation (V)**
  - Alveolar minute ventilation = 4 to 6 L

- **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

Normal VQ Ratio

- $\text{V} = 600 \text{ mmHg}$
- $\text{Q} = 0 \text{ mmHg}$

- $\text{Alveolus}$

- $\text{PAO}_2 = 100 \text{ mmHg}$
- $\text{PACO}_2 = 40 \text{ mmHg}$

- $\text{Fenumary Artery}$
- $\text{Pulmonary Capillary}$
- $\text{Pulmonary Vein}$
**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 (oxygenation problem)
Increased V/Q Ratio (Dead Space)

In increased ventilation perfusion ratio
- Alveolar $O_2$ will rise
- Alveolar $CO_2$ will fall

Increased V/Q Ratio: Alveolar Dead Space

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

Assessing Oxygenation

- Clinical Application: Cannot assess PaO\(_2\) (arterial) without considering alveolar oxygenation content (PAO\(_2\))
  - Increase in FIO\(_2\) will increase PAO\(_2\)
  - Increase in PACO\(_2\) will decrease PAO\(_2\)

Note: With normal diffusion the majority of oxygen in the alveoli should diffuse across the alveolar capillary membrane.
ALVEOLAR O₂

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

**Alveolar Gas Equation:**

\[ PAO₂ = FIO₂ \times (PB - 47) - PaCO₂ / 0.8 \]

- \( PAO₂ \) = 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
- \( 0.8 \) = respiratory quotient

Importance of FIO₂

*Normal arterial oxygen content of 80-100 mm Hg is only normal when the FIO₂ is 0.21*

*Expected \( PaO₂ \) based on \( FIO₂ \)

\[ (FIO₂ \% \times 6) - PaCO₂ \]

**Example:**

FIO₂ of 100% or 1.0 with \( PaCO₂ \) 40 mm Hg

\[ (100 \times 6) - 40 = 560 \text{ mm Hg} \]
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.

Linking Knowledge to Practice with PaO$_2$ / FIO$_2$ Ratios

<table>
<thead>
<tr>
<th>PaO2</th>
<th>FIO2</th>
<th>Ratio</th>
<th>Treatment / Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>55</td>
<td>21%</td>
<td>261</td>
<td>Admit; respiratory distress</td>
</tr>
<tr>
<td>60</td>
<td>100%</td>
<td>60</td>
<td>Worsening; NRB Mask</td>
</tr>
<tr>
<td>210</td>
<td>100%</td>
<td>210</td>
<td>Post intubation ABG, antibiotics</td>
</tr>
<tr>
<td>190</td>
<td>60%</td>
<td>316</td>
<td>Continued treatment, FIO$_2$ decreased</td>
</tr>
<tr>
<td>150</td>
<td>40%</td>
<td>375</td>
<td>Clinical improvement, FIO$_2$ decreased</td>
</tr>
</tbody>
</table>
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

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
Hypoxia and Hypoxemia

- **Hypoxemia**
  - Insufficient oxygenation of the blood
  - Mild: PaO2 < 80 mm Hg or SaO2 < 95%
  - Moderate: PaO2 < 60 mm Hg 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

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?**
Relationship Between Oxygen and Hemoglobin

- 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. Represented by the $\text{SaO}_2$.

- Plasma: 3% of oxygen is dissolved in plasma. Represented by the $\text{PaO}_2$ (measurement of $O_2$ tension in plasma).
Oxyhemoglobin Dissociation Curve

- Shows the relationship between PaO$_2$ and SaO$_2$

- Horizontal curve shows PaO$_2$ above 60 results in minimal changes in oxygen saturation
  - Protects body – allowing high saturations with large decreases in PaO$_2$
- Vertical curve shows PaO$_2$ below 60 results in significant decreases in oxygen saturation
  - Allows tissues to extract large amounts of O$_2$ with only small decreases in PaO$_2$
Shifts in Oxyhemoglobin Curve

- **Shift to the Left**
  - 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

- **Shift to the Right**
  - 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
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

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
Alterations in Oxyhemoglobin Curve

\[ \text{Rise} \]
\[ \text{In} \]
\[ 2, 3-	ext{DPG} \]
\[ \text{H}^+ \]
\[ \text{Temperature} \]

Oxygen Delivery to Tissues
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 $\times$ arterial oxygen saturation)
Formula for Oxygen Delivery

- \( \text{DO}_2 \) formula = \( \text{CO} \times \text{Hgb} \times \text{SaO}_2 \times 13.4 \) (constant)

- Normal \( \text{DO}_2 \) = 900-1100 ml/min (1000)

- Normal \( \text{DO}_2 \text{I} \) = 550 – 650 ml/min

Inadequate Delivery: Hypoxia

- Circulatory hypoxia
  - Inadequate cardiac output

- Anemic hypoxia
  - Inadequate hemoglobin

- Respiratory hypoxia
  - Inadequate \( \text{SaO}_2 \)
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

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)
Causes of Increased VO$_2$

- Fever per 1 degree C
- Shivering
- Suctioning
- Sepsis
- Non Family Visitor
- Position Change
- Sling Scale Weight
- Bath
- CXR
- Multi Organ Failure

<table>
<thead>
<tr>
<th>Event</th>
<th>Increase in VO$_2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>10%</td>
<td>10%</td>
</tr>
<tr>
<td>50-100%</td>
<td>50-100%</td>
</tr>
<tr>
<td>7-70%</td>
<td>7-70%</td>
</tr>
<tr>
<td>5-10%</td>
<td>5-10%</td>
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<tr>
<td>22%</td>
<td>22%</td>
</tr>
<tr>
<td>31%</td>
<td>31%</td>
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<tr>
<td>36%</td>
<td>36%</td>
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<tr>
<td>23%</td>
<td>23%</td>
</tr>
<tr>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>20-80%</td>
<td>20-80%</td>
</tr>
</tbody>
</table>

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$_2$ER) = 25%
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.

Relationship of Delivery to Consumption

<table>
<thead>
<tr>
<th>DO₂</th>
<th>VO₂ (extraction is independent of delivery)</th>
<th>SVO₂ (SVO₂ will improve when you increase delivery)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1000 cc</td>
<td>250 cc (25%)</td>
<td>75%</td>
</tr>
<tr>
<td>750 cc</td>
<td>250 cc (33%)</td>
<td>67%</td>
</tr>
<tr>
<td>500 cc</td>
<td>250 cc (50%)</td>
<td>50%</td>
</tr>
</tbody>
</table>
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.

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.
Significant Changes In SVO$_2$

- SVO$_2$ < 60%
  - Decreased delivery
  - Increased consumption
- SVO$_2$ > 80%
  - Increased delivery
  - Decreased demand
  - Sepsis (tissues cannot extract)
  - Wedged catheter
- Clinically significant change is +or – 5 to 10% over 3 to 5 minutes

- SVO$_2$ < 40% represents limits of compensation and lactic acidosis will occur (oxygen demand is greater than oxygen delivery and reserve can be depleted = oxygen debt)

ScVO$_2$

- ScVO$_2$ 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%.
  - ScVO$_2$ trends higher than SVO$_2$ but trends with SVO$_2$. 
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

Acid –Base Balance
**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

**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
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

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
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

ABG Analysis

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

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Range</th>
<th>Abnormal Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>pH</strong></td>
<td>Normal 7.35-7.45</td>
<td>-7.35 Acidosis, &gt;7.45 Alkalosis</td>
</tr>
<tr>
<td><strong>PaCO2</strong></td>
<td>Normal 35-45 mm Hg</td>
<td>&lt;35 alkalosis or respiratory compensation for metabolic acidosis, &gt;45 acidosis or respiratory compensation for metabolic alkalosis</td>
</tr>
<tr>
<td><strong>HCO₃⁻</strong></td>
<td>Normal 22-26 mEq/L</td>
<td>&lt;22 metabolic acidosis or metabolic compensation for respiratory alkalosis, &gt;26 metabolic alkalosis or metabolic compensation for respiratory acidosis</td>
</tr>
<tr>
<td><strong>Base Excess (BE)</strong></td>
<td>Normal +2 to -2</td>
<td>&lt; -2 (base deficit) metabolic acidosis or metabolic compensation for respiratory alkalosis, &gt; +2 metabolic alkalosis or metabolic compensation for respiratory acidosis</td>
</tr>
</tbody>
</table>
ABG Analysis: Parameters

- **Pao2**
  - Normal 80-100 mm Hg
  - >100 hyperoxemia
  - < 80 mild hypoxemia
  - < 60 moderate hypoxemia
  - < 40 severe hypoxemia

- **Sao2**
  - Normal 95% or >
  - < 95% mild desaturation of HGB
  - < 90% moderate desaturation of HGB
  - < 75% severe desaturation of HGB

Compensation

An acidosis or alkolosis 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).
Anion Gap

- The anion gap is used to help determine the cause of the patient’s metabolic acidosis.
- **Anion Gap = Na+ - [Cl- + HCO3-]**
- 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 HCO3-.
  - Lactic acidosis
  - Ketoacidosis
  - Renal retention of anions

More on Anion Gap

- Most common etiology of normal anion gap acidosis: Diarrhea.
- Second most common: Renal tubular acidosis.
- Both result in a loss of bicarbonate ions.
- To compensate there is an increase in plasma chloride.
- Normal ion gap acidosis is often referred to as hyperchloremic acidosis.
Common Causes of Respiratory Acidosis

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

Common Causes of Respiratory Alkalosis

- Central nervous system disorders
- Drugs
- Hormones
- Bacteremia
- High altitude
- Over mechanical ventilation
- Acute asthma
- Pulmonary embolism
Common Causes of Metabolic Acidosis

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

Common Causes of Metabolic Alkalosis

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

ABG Analysis Practice

- pH: 7.30
- PaCO2: 54
- HCO3: 26
- PaO2: 64
ABG Analysis Practice

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

ABG Analysis Practice

- pH: 7.30
- PaCO2: 40
- HCO3: 18
- PaO2: 85
ABG Analysis Practice

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

ABG Analysis Practice

- pH: 7.35
- PaCO2: 54
- HCO3: 30
- PaO2: 55
ABG Analysis Practice

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

ABG Analysis Practice

- pH  7.54
- PaCO2  25
- HCO3  30
- PaO2  95
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.
Acute Respiratory Failure

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

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

**Ventilatory Failure**

**Oxygenation Failure**

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**
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) / 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.

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
# COPD: Clinical Manifestations

<table>
<thead>
<tr>
<th>Chronic Bronchitis</th>
<th>Emphysema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic cough and sputum production daily for minimum of 3 months/year for at least 2 consecutive years</td>
<td>Increased responsiveness to hypoxemia</td>
</tr>
<tr>
<td>Can have chronic hypoxemia / right sided heart failure</td>
<td>Dyspnea with adequate oxygenation</td>
</tr>
<tr>
<td>Exacerbations related to infection</td>
<td>Initial dyspnea on exertion</td>
</tr>
<tr>
<td></td>
<td>Dyspnea at rest</td>
</tr>
</tbody>
</table>

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

![Diagram of normal bronchi and bronchitis compared to emphysema](image-url)
COPD: Clinical Manifestations

- Blended symptoms
- Large lung volumes / diminished breath sounds
- Ventilation / perfusion mismatching
- High \( \text{PaCO}_2 \) / low \( \text{PaO}_2 \)
- Increase erythropoietin for increased RBCs
- Right sided heart failure

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.
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?

Case Example

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

- Treatment options?
- Goals for ABG values?
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

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
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)
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)

Status Asthmaticus: Treatment

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

- Myasthenia Gravis
  - Neuromuscular junction disorder

- Guillain-Barre
  - Acute polynuropathy

- Muscular Dystrophy

The hallmark of restrictive disorders is a decrease in lung volumes, particularly TLC and VC.

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
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

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).
Pulmonary Embolism: Pathophysiology

- Clot lodges in pulmonary vessels
- Ventilation continues but perfusion decreases
  - Increase in alveolar dead space
  - Alveolar CO2 decreases
- Over perfusion 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.

Pulmonary Embolus: Clinical Presentation

- Large to massive when 50% of pulmonary artery bed is occluded
  - Impending doom
  - Hypoxemia
  - Syncope
  - Signs and symptoms of right heart strain or right-ventricular failure
  - Signs of right-ventricular strain on ECG.
  - Sudden shock
  - Pulseless electrical activity
- Medium-sized emboli
  - Dyspnea
  - Substernal chest discomfort/pleuritic chest pain
  - Many non-specific signs
  - Tachypnea
  - Tachycardia
  - Rales
  - Accentuated 2nd heart sound
Pulmonary Infarction

- Pulmonary infarction is infrequent
- More common
  - Large embolus
  - Pre-existing lung disease
- Results in alveoli filling with RBCs and inflammatory cells
- Complicated by infection
  - Abscess

Signs and Symptoms
- Pleuritic chest pain
- Dyspnea
- Hemoptysis
- Cough
- Pleural friction rub
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.
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 despite 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
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

Pulmonary Hypertension

- PPH (primary pulmonary hypertension)
- IPAH (idiopathic pulmonary arterial hypertension)
- Rare disease

* Most common reason for secondary pulmonary HTN is LV failure.
Associated Conditions

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

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.
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

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
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

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)
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

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
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

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
CXR In Pulmonary Edema

- Prominent vascular markings: upper lung fields
- Kerley B lines
- Peribronchial thickening
- Patchy alveolar filling in a perihilar distribution – progressing to diffuse infiltrates

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.
Pneumonia

- Acute infection of the lung parenchyma, including alveolar spaces and interstitial space

- Causes:
  - Bacteria (Community acquired versus Hospital acquired)
  - Virus
  - Fungi
  - Parasites
  - Mycoplasma

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
**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

**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 influenza* 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.
  - Psuedomonas 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.

### 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.**
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
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)*
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)

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
Aspiration

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

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).
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.

Risk Factors in ARDS

- Sepsis (most common)
- Transfusion
- Aspiration
- Trauma
- Massive transfusion
- Pancreatitis
Acute Respiratory Distress Syndrome: Etiology

- Direct lung injury
  - Chest trauma
  - Near drowning
  - Smoke inhalation
  - Pneumonia
  - Pulmonary embolism
  - Or: Change in pulmonary vascular pressure

- Indirect lung injury
  - Sepsis
  - Shock
  - Multi system trauma
  - Burns
  - CABG
  - Head injury

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

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

ARDS: Treatment

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

High Mortality Persists so Prevention Remains Key
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

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
Case Example

- 65 year old female; 85 kg
- Post witnessed cardiac arrest
- Initial $\text{PaO}_2 / \text{FIO}_2$ ratio 102
- Initial diagnosis?

Case Example

- Ventilator settings:
  - AC
  - Rate 12
  - TV 700 ml
  - FIO$_2$ 80%
  - PEEP 5 cm
- 2$^{nd}$ ABG
  - pH – 7.33
  - PaCO$_2$ – 40 mmHg
  - HCO$_3$ – 14
  - PaO$_2$ – 92

- Ventilator adjustment?
- Other treatment considerations?
# Pulling it All Together

<table>
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<th>Ventilation</th>
<th>Perfusion</th>
<th>Diffusion</th>
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<td>Rate and depth of respirations</td>
<td>Global: Decreased C.O.</td>
<td>Oxygenation status</td>
</tr>
<tr>
<td></td>
<td>Work of breathing</td>
<td>VQ Scan</td>
<td>SpO₂ / SaO₂</td>
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<tr>
<td></td>
<td>PaCO₂</td>
<td>Spiral CT</td>
<td>PaO₂</td>
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<td>S&amp;S associated with P.E.</td>
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<td>Respiratory alkalosis</td>
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<tr>
<td>Treatment</td>
<td>Increase rate or Vt</td>
<td>Support cardiac output</td>
<td>Increase FIO2</td>
</tr>
<tr>
<td></td>
<td>Reverse sedation, Ambu bag, intubate and ventilate, adjust ventilator settings</td>
<td>Anticoagulation</td>
<td>Add PEEP if necessary to increase driving pressure</td>
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<tr>
<td></td>
<td></td>
<td>Fibrinolytic</td>
<td>Treat underlying cause</td>
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<td></td>
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<td>Embolectomy</td>
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</tbody>
</table>

## Associated Pathological Conditions

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<th>Ventilation</th>
<th>Perfusion</th>
<th>Diffusion</th>
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<tr>
<td>Neurological conditions (brain or spinal cord injury), Any condition that affects airway resistance or lung compliance COPD: Increased airway resistance</td>
<td>Pulmonary Embolus</td>
<td>Any disorder that affects the alveolar capillary membrane</td>
<td></td>
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<tr>
<td>Asthma: Increased airway resistance</td>
<td></td>
<td>Pneumonia: Exudate</td>
<td></td>
</tr>
<tr>
<td>Pneumonia: Increased airway resistance from secretions</td>
<td></td>
<td>Pulmonary edema: Fluid</td>
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<tr>
<td>Pulmonary edema: Decreased lung compliance</td>
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<tr>
<td>Pleural effusion</td>
<td></td>
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</tr>
</tbody>
</table>

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