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
- Under neurological control
- Occurs through inspiration and expiration
- Pressure difference between airway opening and alveoli
  - **Result:** Negative pressure breathing

### Minute Ventilation

- **Minute ventilation** ($V_E$) = Total volume of air expired in one minute
  - **Respiratory rate** x **tidal volume** ($V_T$) (tidal volume = amount of air per breath)
  - Normal minute ventilation = 12 x 500 ml = 6000ml

- **Note:** (hypoventilation can occur with normal or even high respiratory rate)
Alveolar Ventilation ($V_A$)

- $V_A = V_T -$ anatomical dead space
  - Walls are too thick for diffusion
  - Mixed venous blood not present
    - Approximately 1 ml per ideal pound of body weight (150 ml)

- $V_A =$ Approximately 350 ml per breath
  - This is the ventilation that participates in gas exchange

Respiratory Anatomy

- Conducting Airways: Resistance
  - Nose
  - Pharynx
  - Larynx
  - Trachea
  - Right and Left Bronchi
  - Non-Respiratory Bronchi

- Gas Exchange Airways: Compliance
  - 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)
    • Compliance is opposite of elastic recoil
    • Lungs distend most easily at low volumes

– 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.

Conditions Altering Ventilation

• Non Pulmonary Conditions
  – Drug overdose
  – Spinal cord injury
  – Brain injury

• Pulmonary Conditions
  – Decreased Compliance
  – Increased Resistance
Pulmonary Conditions Altering Ventilation

**Lung or Chest Wall Compliance**
- Restrictive disorders (fibrosis, interstitial lung disease)
- Decreased surfactant production
- Atelectasis
- Pulmonary vascular engorgement
- Air, blood or excess fluid in pleural space
- Obesity / musculoskeletal disorders (chest wall compliance)

**Airway Resistance**
- Obstructive Disorders
  - Asthma
  - Emphysema
  - Bronchitis
  - Foreign body causes a fixed obstruction
  - Sleep apnea can be obstructive
- Narrowing of airways
  - Secretions
  - Bronchospasm

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

• Rate and depth of respirations
• Work of breathing
• 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$_2$.

Treatment of Ventilation Problems

VENTILATION PROBLEMS ARE TREATED BY RATE AND VT

Options: Reverse sedation or underlying cause, ambu bag, BiPAP, or intubation and mechanical ventilation
More on Ventilation

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

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 PaCO$_2$ to determine if inadequate ventilation contributed to the hypoxemia.
Perfusion

- Definition: The movement of blood through the pulmonary capillaries.
Perfusion

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

Perfusion Fun Facts

• 280 billion capillaries supply 300 million alveoli
• 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)
• 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 (PVR)

- \(1/10\) of systemic vascular resistance
- Evenly distributed between the pulmonary arteries, the pulmonary capillaries, and the pulmonary veins
- Increased PVR
  - Hypoxic vasoconstriction
  - Mechanical ventilation
  - PEEP
  - Note: Increased PVR increases work of right ventricle
- Decreased PVR
  - Increase in cardiac output = Increase in pulmonary artery pressure (PAP) = Increase in capillary recruitment = Decrease in PVR
  - High lung volumes pull pulmonary vessels open. Results in a decrease PVR.
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
• **The hypoxic vasoconstriction intended to help with V/Q mismatching increases the workload of right ventricle.**
• Increases in PA pressures to recruit alveoli can also lead to pulmonary edema.

Conditions that Alter Pulmonary Perfusion

• **#1 = pulmonary embolism**
• Any decrease in cardiac output from right ventricle: shock

• Clinical Applications
  – An increase in PVR for any reason can lead to right heart failure
  – Any increase in pulmonary artery pressures can lead to pulmonary edema
Prior to Diffusion

• Ventilation and Perfusion Occur Simultaneously

Alveolar oxygen 100 mmHg
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\(_2\)** and oxygen saturation (**SaO\(_2\)**)  
  – However, a simple diffusion problem rarely results in hypoxemia at rest.

• **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
Treating Diffusion Barriers

Increased $\text{FiO}_2$ and increased pressure (CPAP / PEEP) will increase driving pressure of oxygen.

Ventilation versus Diffusion

**Assessment and Treatment**

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

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

Normal VQ Ratio
Decreased ventilation to perfusion ratio
V/Q = 0
(Intrapulmonary Shunting)

In decreased ventilation perfusion ratio

Alveolar O$_2$ will fall
Alveolar CO$_2$ will rise

Increased V/Q Ratio
(Dead Space)

In increased ventilation perfusion ratio

Alveolar O$_2$ will rise
Alveolar CO$_2$ will fall
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 from high alveolar pressures
  - Tumors

Assessment Tools and Pearls
Ventilation: Patient End Tidal CO$_2$ (PetCO$_2$)

- Capnography: evaluation of the CO$_2$ level in the respiratory gases.
- Includes both the continuous analysis and the continuous recording of the CO$_2$.
  - Continuous waveform capnography is recommended as the most reliable method of confirming and monitoring correct placement of an ET tube.

- Effective tool detecting ventilation abnormalities well before a change in the patient’s oxygenation status.
Assessing Oxygenation

• 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.*

PaO$_2$ and FIO$_2$ Ratio

• An assessment and trending tool
• PaO$_2$/ FIO$_2$ ratio:
  – Normal well above 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

\[
60 \div 0.5 = 120.
\]

This is a clinically significant intrapulmonary shunt.
Linking Knowledge to Practice with \( \text{PaO}_2 / \text{FIO}_2 \) Ratios

<table>
<thead>
<tr>
<th>( \text{PaO}_2 )</th>
<th>( \text{FIO}_2 )</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 (Alveolar) – a (arterial) Gradient (Difference)

- Provides an index regarding diffusion.
- The majority of what is in the “A” should end up in the “a”.
- A large A-a gradient generally indicates that the lung is the site of dysfunction.

- Normal A-a Gradient is small = 5 to 15 mm Hg

Pulmonary Alveolus
\( \text{PAO}_2 \) 100 mmHg

\( \text{PaO}_2 \) (80-100 mmHg)
Hypoxemia

• Causes
  – Diffusion abnormality
  – Untreated alveolar hypoventilation
  – Ventilation and perfusion mismatching

• Assessment Clues
  – PaO₂ / SaO₂: Will be low regardless of etiology
  – Improvement with increased FIO₂: Diffusion problem
  – ↑ PaCO₂ / increased work of breathing: Ventilation failure
  – A-a gradient will be normal in ventilatory failure from neurological abnormality
  – ↓PaO₂ / FIO₂ ratio: Suggests something more severe than simple diffusion abnormality (i.e. intra pulmonary shunting from decreased V/Q ratio

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 being treated with oxygen.

• 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 (carboxyhemoglobin or methemoglobin)

- Other Factors
  - SpO₂ below 70%
  - Low blood flow: hypotension or vasoconstriction
  - IV dyes, fingernail polish, some skin pigmentations
  - Administration of high fat content such as with propofol or TPN can have a falsely high SpO₂

Hypoxia and Hypoxemia

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

- **Hypoxia**
  - Insufficient oxygenation of tissues
    - Determined by oxygen delivery and cellular demand
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
  - 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 $\text{PaO}_2$ and $\text{SaO}_2$
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
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
Alterations in Oxyhemoglobin Curve

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

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
Acid –Base Balance

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 PCO$_2$
    • If pH is up: decrease rate and depth of respiration to retain PCO$_2$
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: PaCO$_2$

• Evaluate acid-base status: pH

• Evaluate source of abnormal pH: respiratory or metabolic

• Evaluate oxygenation: PaO$_2$, SaO$_2$
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).

Anion Gap

- 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.

Anion Gap

<table>
<thead>
<tr>
<th>Normal</th>
<th>Abnormal</th>
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<tbody>
<tr>
<td>Na</td>
<td>140</td>
</tr>
<tr>
<td>Chloride</td>
<td>103</td>
</tr>
<tr>
<td>$\text{HCO}_3$</td>
<td>28</td>
</tr>
<tr>
<td>140 – (103+28) = 9</td>
<td>140 – (99+18) = 23</td>
</tr>
</tbody>
</table>
Common Causes of Respiratory Acidosis

- Depression of respiratory control centers
- Neuromuscular disorders
- Chest wall restriction
- Lung restriction
- Airway obstruction
- Pulmonary parenchymal disease
- **Anything that causes ventilatory failure**

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
This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis
3. Respiratory alkalosis
4. Metabolic alkalosis
5. Respiratory acidosis with hypoxemia

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.30</td>
</tr>
<tr>
<td>PaCO₂</td>
<td>54 mmHg</td>
</tr>
<tr>
<td>HCO₃</td>
<td>26 mEq/L</td>
</tr>
<tr>
<td>PaO₂</td>
<td>64 mmHg</td>
</tr>
</tbody>
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This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis
3. Respiratory alkalosis
4. Metabolic alkalosis
5. Respiratory acidosis with hypoxemia

| pH  | 7.30 |
| PaCO₂ | 54 mmHg |
| HCO₃  | 26 mEq/L |
| PaO₂   | 64 mmHg |

This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis
3. Respiratory alkalosis
4. Metabolic alkalosis

| pH  | 7.48 |
| PaCO₂ | 30 mmHg |
| HCO₃  | 24 mEq/L |
| PaO₂   | 96 mmHg |
This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis
3. **Respiratory alkalosis**
4. Metabolic alkalosis

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<td>30 mmHg</td>
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<tr>
<td>HCO₃</td>
<td>24 mEq/L</td>
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<tr>
<td>PaO₂</td>
<td>96 mmHg</td>
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</table>

This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis
3. Respiratory alkalosis
4. Metabolic alkalosis

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<tr>
<td>HCO₃</td>
<td>18 mEq/L</td>
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<tr>
<td>PaO₂</td>
<td>85 mmHg</td>
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</table>
This Blood Gas Represents

1. Respiratory acidosis

2. **Metabolic acidosis**

3. Respiratory alkalosis

4. Metabolic alkalosis

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<td>85 mmHg</td>
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This Blood Gas Represents

1. Respiratory acidosis

2. **Metabolic acidosis**

3. Respiratory alkalosis

4. Metabolic alkalosis

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<tr>
<td>HCO₃</td>
<td>33 mEq/L</td>
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<tr>
<td>PaO₂</td>
<td>92 mmHg</td>
</tr>
</tbody>
</table>
This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis
3. Respiratory alkalosis
4. Metabolic alkalosis

[pH 7.50
PaCO₂ 40 mmHg
HCO₃ 33 mEq/L
PaO₂ 92 mmHg]

This Blood Gas Represents

1. Compensated metabolic alkalosis
2. Compensated metabolic acidosis with hypoxemia
3. Compensated respiratory alkalosis
4. Compensated respiratory acidosis with hypoxemia

[pH 7.35
PaCO₂ 54 mmHg
HCO₃ 30 mEq/L
PaO₂ 55 mmHg]
This Blood Gas Represents

1. Compensated metabolic alkalosis
2. Compensated metabolic acidosis with hypoxemia
3. Compensated respiratory alkalosis
4. **Compensated respiratory acidosis with hypoxemia**

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<td>30 mEq/L</td>
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<tr>
<td>$\text{PaO}_2$</td>
<td>55 mmHg</td>
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</tbody>
</table>

This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis with hypoxemia
3. Over compensated metabolic alkalosis
4. Mixed respiratory and metabolic acidosis with hypoxemia

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<thead>
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<th>Parameter</th>
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<td>$\text{PaCO}_2$</td>
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<tr>
<td>$\text{HCO}_3$</td>
<td>20 mEq/L</td>
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<tr>
<td>$\text{PaO}_2$</td>
<td>48 mmHg</td>
</tr>
</tbody>
</table>
This Blood Gas Represents

1. Respiratory acidosis
2. Metabolic acidosis with hypoxemia
3. Over compensated metabolic alkalosis
4. **Mixed respiratory and metabolic acidosis with hypoxemia**

\[
\begin{array}{|c|c|}
\hline
\text{pH} & 7.21 \\
\text{PaCO}_2 & 60 \text{ mmHg} \\
\text{HCO}_3^- & 20 \text{ mEq/L} \\
\text{PaO}_2 & 48 \text{ mmHg} \\
\hline
\end{array}
\]

This Blood Gas Represents

1. Respiratory alkalosis
2. Metabolic alkalosis
3. Mixed respiratory and metabolic alkalosis
4. None of above

\[
\begin{array}{|c|c|}
\hline
\text{pH} & 7.54 \\
\text{PaCO}_2 & 25 \text{ mmHg} \\
\text{HCO}_3^- & 30 \text{ mEq/L} \\
\text{PaO}_2 & 95 \text{ mmHg} \\
\hline
\end{array}
\]
This Blood Gas Represents

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2. Metabolic alkalosis
3. **Mixed respiratory and metabolic alkalosis**
4. None of above

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</table>

Oxygen and Ventilator Therapy
Oxygen Therapy and $\text{FIO}_2$

- Cannula: < 40%
- Simple Mask: 40-60%
- Venturi Mask: Up to 40%
- High Flow Nasal Cannula: Up to close to 100%
- Non-rebreathing mask: 80-100%
- Bag Valve Mask

High Flow Oxygen Therapy

- Provides entire inspired gas by high flow of gas
- Provides a predictable $\text{FIO}_2$
- Doesn’t mean a high $\text{FIO}_2$
- 100% non rebreather masks, high flow nasal cannula, venturi masks and mechanical ventilators are examples of higher flow oxygen delivery systems
**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 FIO\textsubscript{2}
- Nasal cannula is a low flow oxygen delivery system
- Simple face mask is a moderate flow delivery system
Guidelines for estimating FIO\textsubscript{2} with low flow oxygen devices

- 100% O\textsubscript{2} flow rate(L)
  - Nasal Cannula
    - 1
    - 2
    - 3
    - 4
    - 5
    - 6
  - Oxygen Mask
    - 5-6
    - 6-7
    - 7-8
  - Mask with Reservoir
    - 6
    - 7
    - 8
    - 9
    - 10

- FIO\textsubscript{2} (%)
  - 24
  - 28
  - 32
  - 36
  - 40
  - 44
  - 40
  - 50
  - 60
  - 60
  - 70
  - 80
  - 90
  - 99

Oxygen Toxicity

- Complications of O\textsubscript{2}
  - Absorption atelectasis
  - Decreased hypoxic drive

- Signs and symptoms of oxygen toxicity
  - Dyspnea
  - Decreased lung compliance
  - Retrosternal pain
  - Parasthesia in the extremities

- To reduce risk of oxygen toxicity:
  - 100% no more than 24 hours
  - 60% no more than 2-3 days
  - Use 40% for longer term therapy
Mechanical Ventilation

**Indications**
- Respiratory failure.
  - Hypercapnic
  - Hypoxemic
- Excessive work of breathing.
  - Tachypnea
  - Accessory muscle use
  - Tachycardia
  - Diaphoresis
- Protection of airway

**Goals**
- Achieve adequate ventilation
- Achieve adequate oxygenation
- Provide decreased work of breathing, patient comfort and synchrony with the ventilator
- Protect the lungs from further injury

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
  - 12 / 6 cmH₂O is a common setting

- Consider as first line strategy
- Consider as alternative to failed weaning
- Decreased VAE
Non Invasive Positive Pressure Ventilation

- Dedicated non invasive unit or with traditional mechanical ventilator
- Contraindications
  - Decreased level of consciousness
  - Increased gastrointestinal bleeding
  - Hemodynamic instability
  - Progressive decline in respiratory status

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.
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

---

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.**

• Is effective in decreasing the work of breathing when used with appropriate sedation.
Assist Control

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.
Adaptive Support Ventilation

• Capable of increasing or decreasing support as needed
  – No spontaneous breathing: Uses AC with time cycled (pressure controlled) breaths
  – Spontaneous breathing below target: Uses SIMV mode with time cycled (pressure controlled) breaths
  – Spontaneous breathing above target: the ventilator changes to the pressure support mode using flow cycled breathes

• Plateau pressure is set and Vt varies breath to breath
• Mandatory breaths are adjusted to assure adequate minute ventilation
• Pressure limits can be adjusted if needed to assure adequate ventilation
• I:E ratio is also adjusted to prevent auto peep
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
  - Facilitates oxygenation
  - P High (20-30 cmH₂O) and P low (O) (pressure)
  - T high (4-6 seconds) and T low (0.8 seconds) (time)

- Time triggered
- Time cycled
- Pressure limited

**Advantages**
- Lower peak and plateau pressures for given volume
- Decreased sedation / near elimination of neuromuscular blockade

Airway Pressure Release Ventilation
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
  - Hemodynamic effects: Can increase JVP and PAOP and decrease CO.

- **Inverse ratio ventilation**

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** ($V_T$): 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** ($FIO_2$): 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 H$_2$O. PEEP is titrated up as needed to achieve adequate oxygenation. > 15 cm H2O of PEEP is rarely needed.
Adjuncts to Mechanical Ventilation

• PEEP: Positive end expiratory pressure

• PSV: Pressure support ventilation; positive pressure during inspiration; during spontaneous breaths such as with SIMV
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
- Potential complications:
  - Barotrauma
  - Decreased cardiac output
  - Regional hypoperfusion

Optimal PEEP

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)
  - Longer inspiration time increases mean airway pressure
  - Too short of expiratory times can lead to auto PEEP
Auto PEEP

Factors Shortening Expiratory Time
- High rate
- High $V_T$

Strategies to Lengthen Expiratory Time
- Decrease rate
- Decrease VT
- Increase peak gas flow
Measured Parameters

• Mean Airway Pressure: Constant airway opening pressure
  – PEEP
  – CPAP
  – Pressure Support

Measured Parameters

• Peak Inspiratory Pressure
  – Accounts for airway resistance and lung compliance

• Inspiratory Plateau Pressure
  – Takes resistance out of equation
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

Hypotension with Mechanical Ventilation

- **Conversion to positive pressure ventilation / PEEP**
  - Assure adequate circulating fluid volume
- **Response to sedation**
  - Titrate sedation
- **Development of auto PEEP**
  - Increase expiration time
- **Tension Pneumothorax**
  - Chest tube required
Complications of Mechanical Ventilation

- **Barotrauma** (caused by excessive pressure)
- **Volutrauma** (caused by excessive volume)
- **Ateletrauma** (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)

Lung Protective Strategies

- Low tidal volume (6 ml / kg) with permissive hypercapnea
- Maintain plateau pressure ≤ 30 mm Hg
Clinical Practice Guidelines for the Management of Pain, Agitation, and Delirium in Adult Patients in the Intensive Care Unit


Analgesia and Mechanical Ventilation

- **Opiates (scheduled or drip) for constant effect**
  - Opiates typically do not have hemodynamic effects in patients who are not hypovolemic.
  - Fentanyl duration of action 30 to 60 minutes
  - Morphine duration of action 4 hours
- **Acetaminophen or non-steroidals as adjunct**
Level of Sedation

- Target level of sedation: Calm patient who is easily arousable.
  - 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)
When sedation goal not met - Consider pain or delirium as contributing factor.

Benzodiazepines

- Dose dependent effect.
- Anxiolytic, amnestic, and anticonvulsant properties.
- May enhance analgesic effect of opiates
- Little hemodynamic effect with adequate volume status.

- Increased risk for delirium
  - Avoid or use only what is needed
  - Continuous infusions = longer ventilation times and longer ICU LOS.
    - Bolus dosing preferred.
Benzodiazepines

- Onset 30 seconds to 5 minutes; Duration typically < 2 hours
- Parental form lipid soluble
- Prolonged or continuous infusion = increased peripheral distribution in areas where no metabolism occurs; after the drug is stopped the tissues can release it back into circulation
- Active metabolite can accumulate
- High risk features for prolonged effect: Obesity and elderly with hepatic and renal impairment

Midazolam

- Slower onset of action and less lipid soluble compared to midazolam
- Metabolism less influenced by age and liver function
- No active metabolite

Lorazepam

Propofol

- Rapid onset and offset.
- Decreases awareness and respiratory drive.
- Anxiolytic, amnestic agents, and anticonvulsant effect.
- Preferred option for sedation over benzodiazepines
- Indicated when mechanical ventilation is anticipated to be < 48 hours and / or if frequent neurologic exams are required.
Propofol

- Use lowest possible infusion rate for shortest period of time to achieve sedation goal.
- Initiated slowly at a rate of 5 mcg/kg/min. Increase by increments of 5 to 10 mcg/kg/min. Minimum of 5 minutes between dose adjustment.
- Typical maintenance dose of 5 to 50 mcg/kg/min.
- Half life with short term use: 3 to 12 hours

Propofol Complications

- Hypotension (common)
  - Venous vasodilatation and mild cardiac depressive effect.
  - Bolus doing and hypovolemia increase risk.

- Elevated triglycerides, pancreatitis, and infection
  - High lipid content (1.1 kcal/ml of fat)

- Propofol-related infusion syndrome.
  - Potentially life threatening complication characterized by the onset of metabolic acidosis, dysrhythmias, hyperkalemia, rhabdomyolysis (or elevated CPK levels), and cardiac failure
Dexmedetomidine

- Selective α2-receptor agonist
  - Sedative
  - Non opioid analgesic
  - Sympatholytic properties
  - No anticonvulsant properties
- Patients more easily arousable
- Minimal respiratory depression – can be used in non intubated patient

Dexmedetomidine

- Only approved for short-term sedation of ICU patients (< 24 hrs) at a maximal dose of 0.7 μg/kg/hr
- Several studies have demonstrated safety at higher doses and for longer duration of infusion
- Analgesic mechanism: α-2 receptors are located in the dorsal region of the spinal cord and in supraspinal sites – however, analgesic effects extend beyond spine; ?
  - Mechanism of action
  - Evidence of lower delirium compared to benzodiazepines
Dexmedetomidine

- Preferred over benzodiazepines to improve clinical outcomes in mechanically ventilated adult ICU patients
- Maintenance dose: 0.2 to 0.7 mcg/kg.hour
- Half life: 1.8 to 3.1 hours
- Side effects:
  - Hypotension and bradycardia
  - HTN or hypotension with loading dose
  - Loss of oropharyngeal muscle tone – potential obstruction in non intubated patients

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
- Does **not** increase unplanned extubations.
Neuromuscular Blockade

• Adequate analgesia and sedation are prerequisites before deciding if neuromuscular blockade is indicated.

• Sedation to achieve complete amnesia is required (Society of Critical Care Medicine and American Society of Health-System Pharmacists, 2002).

Neuromuscular Blockade

• **Depolarizing agents**
  – Mimic acetylcholine: produces fasciculation followed by paralysis
  – Example: Succinylcholine

• **Non-depolarizing agents**
  – Prevents action of acetylcholine
  – Example: vecuronium, atracurium, pancuronium and cisatracurium

- Pancuronium is a commonly used agent.
  - Vagolytic medication and cannot be used in patients who cannot tolerate an increase in heart rate.
  - Cannot be used in patients with hepatic or renal dysfunction.

- Cisatracurium and atracurium.
  - Recommended agents for hepatic or renal dysfunction.
Neuromuscular Blockade

Assessment

• Objective measures of brain function (i.e., BIS) are recommended
• Peripheral nerve stimulators
  – The most commonly used nerve-muscle combination ulnar nerve and adductor pollicis.
  – Goal: One or two twitches in response to nerve stimulation.

Complications

• Prolonged recovery from the agents
• Myopathy
  – With corticosteroids
  – Use longer than 1 to 2 days.
• Acute quadriplegic myopathy syndrome
  – Acute paresis
  – Myonecrosis (increased CPK enzymes)
  – Abnormal EMG
Suctioning

- Shallow technique (pre determined depth) preferred
- Negative pressure only during withdrawal
- Each pass = 1 event
- Maximum of 15 seconds per event
- 100% FIO2 30 to 60 seconds pre and 60 seconds post
- Smaller suction catheters over larger
- Only done as needed to remove secretions.
- No instillation of normal saline
- Closed systems of benefit with high levels FIO2 or PEEP. Fully withdrawal catheter.

Prevention of VAE

- 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
- Subglottic suctioning prior to repositioning or deflating cuff
- 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.
  - Adequate seal for positive pressure ventilation and PEEP
  - Prevents aspiration of large particles but not liquids
  - 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.
Additional Prevention of VAE

- Avoid nasal intubation
- Extubate as soon as possible
- Discontinue NG tubes as soon as possible
- Avoid overuse of antibiotics

Readiness to Wean

- Reversal of the underlying cause of respiratory failure
- Adequate oxygenation
- Hemodynamic stability as defined by no active myocardial ischemia and no clinically significant hypotension
- Patient ability to initiate an inspiratory effort
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 H$_2$O
- FIO$_2$: < 0.50
- PaO$_2$ / FIO$_2$ ratio > 200

Ventilator Weaning:

Spontaneous Breathing Trial

- Done once per 24 hours if patient meets criteria; continue daily even if patient fails initial SBT
- Short period of time: 30 to 120 minutes
- CPAP, pressure support or T – Piece
- Tolerance: Work of breathing, adequacy of gas exchange, hemodynamic stability, and subjective comfort.
Ventilator Weaning

- Proper nutrition can facilitate ventilator weaning
- Phosphate and magnesium deficiency associated with ventilatory muscle weakness

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
Additional Pulmonary Procedures

**Bronchoscopy**
- Diagnostic purposes
  - Airways can be inspected
  - Biopsies can be obtained
- Therapeutic purposes
  - Removal of mucous plugs
  - Dilatation of airway
  - Drainage of abscess
  - Therapeutic lavage

**Thoracentesis**
- Used for large pleural effusions, diagnostic purposes, or empyemas
- Most common complication is pneumothorax
- Patients may experience pain
Sleep Apnea

**Obstructive**
- Repetitive interruption of ventilation during sleep caused by collapse of pharyngeal airway.
- >10 second pause in respiration associated with **ongoing ventilatory effort**

**Central**
- Repetitive cessation of ventilation during sleep resulting from loss of ventilatory drive
- >10 second pause with **no** associated ventilatory effort
- >5 events per hour considered abnormal

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
- 2nd most common cause of sudden death
- 3rd most common cause of death in hospitalized patient
  - 80% of unexpected hospital deaths
- Often recurrent
Risk Factors for PE in Hospitalized Patient

- Admitted to the medical intensive care unit
- Admitted with pulmonary disease,
- Post myocardial infarction
- Post cardiopulmonary bypass surgery

(Ouellette, Harrington, & Kamangar, 2013)

Pulmonary Embolism

**Acute**
- Located centrally within the vessel lumen or causes vessel occlusion
- Results in distention of vessel wall

**Chronic**
- Adjoins to vessel wall & reduces vessel diameter by > 50%
- Recannulization through thrombus
Pulmonary Embolism

Central
- Main pulmonary artery, the left and right main pulmonary arteries, the anterior trunk, the right and left interlobar arteries, the left upper lobe trunk, the right middle lobe artery, and the right and left lower lobe arteries
- Can cause massive PE

Peripheral
- Segmental and subsegmental arteries of the three lobes of the right lung, the two lobes of the left lung, and the lingula (a projection of the upper lobe of left lung)
- Pain by initiating inflammation close to the parietal pleura.

Massive PE
- Present in less than 5% of patients presenting with PE (Kucher, Rossi, De Rosa, & Goldhaber, 2006).
- Involves both the right and left pulmonary arteries or causes hemodynamic collapse
- Presenting systolic BP of < 90 mmHg
- Mortality rates range from 30% to 60% and most deaths occur within the first 1 to 2 hours (Ouellette et al., 2013; Wood, 2002).
Pathophysiology

• Deep vein thrombosis (DVT) occurs at valves of vein due to physiological abnormality
• Clot can embolize or grow to occlude the vein
• Embolized clot returns to right heart and into pulmonary vasculature
• Lower lobes frequently affected due to increased perfusion
• Additional *humoral response*
Pathophysiology

• Increased PVR
  – Proximal clots
  – Substances (thromboxane A and serotonin) released in humoral response also cause vasoconstriction
• PA pressures double to compensate
• Increased work load of RV
  – Right heart failure
  – Leftward shift of septum
  – Right coronary branches can be compressed

Pathophysiology

• Increased V/Q ratio (alveolar dead space)
• Decreased V/Q ratio to other areas due to redistribution of blood flow
• Hypoxemia due to V/Q mismatching
• Increased minute ventilation to compensate for increased dead space – respiratory alkalosis – however, hypercapnea in massive
• Alveolar shrinkage (↓ CO₂)
  – damage Type 2 alveolar cells – loss of surfactant – atelectasis – non cardiac pulmonary edema
• Pulmonary infarction rare due to dual blood supply
Clinical Presentation

• Pleuritic chest pain, shortness of breath, and hypoxemia is not present in the majority of patients
• May have no respiratory complaint
• Atypical presentation: flank pain, abdominal pain, delirium, syncope, and seizures

• Potential diagnosis in any patient with respiratory symptoms in whom there is not another clear etiology
• Suspect when there is respiratory alkalosis

Physical Exam Findings

• The most common physical sign, present in almost everyone with PE, is tachypnea (defined as respiratory rate > 16 per minute)
• Other:
  – Dyspnea, rales, cough, hemoptysis
  – Accentuated 2\textsuperscript{nd} heart sound, presence of right sided S3 or S4, new systolic murmur of tricuspid regurgitation
  – Tachycardia, low grade fever, diaphoresis
  – Signs of thrombophlebitis, lower extremity peripheral edema
  – Hypoxemia, cyanosis
More on Assessment

<table>
<thead>
<tr>
<th>Massive PE</th>
<th>Multiple Emboli</th>
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<tbody>
<tr>
<td>• Shock presentation</td>
<td>• More signs of pulmonary hypertension and cor pulmonale</td>
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Diagnosis

- Cardiac troponins will be elevated in half of patients with moderate to large PE (Konstantinides, 2008)

- **Use of ultrasound to rule out DVT**

- **Computed tomography angiography (CTA) has become the standard test for the diagnosis of PE**

- VQ scan is used as alternative
ECG in PE

• Changes in only 20% of pts

• Non specific
  – ST
  – Atrial fibrillation

• Small T wave inversion in limb and chest leads

• S1,Q3,T3

• Signs of right heart strain
  – RV hypertrophy
  – Right axis deviation
  – Large R waves in V1 and V2
  – Deep S waves in leads V5 and V6
  – Right atrial enlargement (tall P waves in lead II or dominant first ½ of P wave in V1)
  – Incomplete right bundle branch block (RBBB)
  – Delayed intrinsicoid deflection in leads V1 and V2
Treatment

• Treatment with anticoagulation in non-massive PE reduces mortality to less than 5%

• Full anticoagulation is the priority in any patient with suspected or confirmed PE.
  – Intravenous unfractionated heparin is the drug of choice in massive PE, in patients with renal failure, and when there is concern about subcutaneous absorption.
    • An initial bolus of 80 U/kg followed by an infusion of 18 U/kg/hour
  – IV anticoagulation given before dabigatran and edoxaban; overlapped with warfarin
  – IV anticoagulation does not need to be given before rivaroxaban or apixaban
  – LMWH is preferred in cancer associated thrombus
  – Low risk patients can have home treatment or early discharge

Fibrinolytic therapy
  – Hemodynamic compromise as evidenced by systolic BP < 90 mmHg and no high risk for bleeding
  – Deterioration on anticoagulation and low bleeding risk

• Catheter assisted thrombosis removal if high bleeding risk / failed systemic therapy / shock
  – Surgical pulmonary embolectomy may also be considered in select patients
Treatment

• Includes PE, DVT, and VTE (venous thromboembolic event)
• 3 month long term anticoagulation if no cancer and if provoked
  – Dabigatran, rivaroxaban, apixaban, edoxaban preferred over warfarin
• If unprovoked – minimum of 3 months and then evaluation for risk benefit ratio
  – High bleeding risk – 3 months
  – Low to moderate bleeding risk – extended anticoagulation
• Active cancer
  – LMWH preferred agent
  – Extended anticoagulation even in high bleeding risk
• LMWH if recurrent VTE on oral anticoagulation

Treatment

• IVC Filter:
  – Absolute contraindication to anticoagulation
  – Post survival of massive PE where subsequent PE will prove fatal
  – Presence of venous thromboembolism with adequate anticoagulation
  – May be retrievable in certain conditions

• Chronic thromboembolic pulmonary hypertension requires long term anticoagulation
  – Pulmonary thromboendarterectomy

• Compression stockings are not recommended for prevention of post thrombotic syndrome
  – Can 30 to 40 mmHg for symptoms
Special Considerations Air Embolism

- Large volume of air into venous system

- **Risk Factors**
  - Dialysis
  - Pulmonary artery catheters
  - Surgical procedures
  - CABG

- **Symptoms**
  - Dyspnea, chest pain, agitation, confusion, cough

- **Treatment**
  - Prevention
  - Aspiration of air
  - Left lateral / Trendelenburg
  - 100% oxygen
  - Hyperbaric oxygen
  - Support hemodynamics and oxygenation

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### Pulmonary Hypertension

PA Systolic Pressure > 40 mmHg; Mean > 25 mmHg

<table>
<thead>
<tr>
<th>WHO Group</th>
<th>Name</th>
<th>Description</th>
</tr>
</thead>
</table>
| Group 1   | Pulmonary arterial hypertension   | Idiopathic, heritable, drug or toxin induced, associated with conditions such as connective disease disease, HIV, portal hypertension, chronic hemolytic anemia, congenital heart disease, and others  
* Increased pulmonary arteriole resistance as result of abnormal structure or function of pulmonary arterioles |
| Group 2   | Pulmonary hypertension            | From left heart disease. This is the most common cause of pulmonary HTN. Can result from systolic dysfunction, diastolic dysfunction, or valvular disease. |
| Group 3   | Pulmonary hypertension            | From lung disease or hypoxia. Can result from COPD, interstitial disease, sleep disordered breathing, and others.                             |
| Group 4   | Pulmonary hypertension            | From chronic thromboembolic disease.                                                                                                         |
| Group 5   | Pulmonary hypertension            | Etiology is unclear multifactorial processes including hematologic, metabolic and systemic diseases.                                           |
Clinical Pearls for Pulmonary HTN

Assessment

• PA systolic increases with age and obesity
• Most common reason for pulmonary HTN is left heart disease
  – Left atrial size will be large if LV failure is etiology
  – Elevated left heart filling pressures result in a passive increase in PA pressure
  – Termed isolated post capillary pulmonary HTN
  – PVR and transpulmonary gradient normal
• Patients with left heart disease can also have combined pre and post capillary pulmonary HTN
  – PVR and transpulmonary gradient elevated

Pulmonary Arterial Hypertension (PAH)

• PAH
  – Rare disease
• 15-20% have familial component
• Females are affected more than males
  – Women of child bearing age more often affected
• Perhaps caused by insult to endothelium in patient with susceptibility to pulmonary vascular injury
  – Vascular scarring
  – Endothelial dysfunction
  – Intimal and medial smooth muscle proliferation
Associated Conditions

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

Treatment Overview

- PAH has no cure
- Untreated leads to right sided heart failure and death
- New drugs have improved survival rates
  – Prostacyclin analogues
  – Endothelin receptor antagonists
Presentation

• Average time from symptom onset to diagnosis is 2 years
• Most common symptoms in one study:
  – Dyspnea (60%)
  – Weakness (19%)
  – Recurrent syncope (13%)
  – Same symptoms as aortic stenosis

• 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
  – Diuretics
  – Digoxin
  – Oxygen with hypoxemia
  – Anticoagulation
    • Mixed data and recommendations

• Calcium channel blockers
  – Nifedipine / diltiazem
  – Only used in patients who are responders to acute vasodilator testing
  – Only in patients without overt right sided heart failure
  – High doses are used
  – Can have rebound pulmonary hypertension when withdrawn
Approved Pulmonary Vasodilators for PAH: Prostacyclin analogues

- Epoprostenol (Flolan)
  - IV - Parental
- Treprostinil (Remodulin)
  - IV / SQ – Parental
- Iloprost (Ventavis)
  - Nebulized inhalation
- Selexipag (Uptravi)
  - Oral

Approved Pulmonary Vasodilators for PAH: Endothelin receptor antagonist (ERA)

- Bosentan (Tracleer)
  - Oral
- Ambrisentan (Letairis)
  - Oral
- Macitentan (Opsumit)
  - Oral
Approved Pulmonary Vasodilators for PAH: 
Drugs Interfering with the Nitric Oxide Pathway

• Sildenafil (Revatio)  
  – Phosphodiesterase (type 5) enzyme inhibitor  
  – Oral
• Tadalafil (Adcirca)  
  – Phosphodiesterase (type 5) enzyme inhibitor  
  – Oral
• Riociguat (Adempas)  
  – Soluble Guanylate Cyclase Stimulator)  
  – Oral

Other Treatment Options

• Cardiopulmonary rehab for mild symptom limited aerobic activity
• Pulmonary endarterectomy for WHO group 4
• Single or double lung transplant (cardiac transplant may or may not be needed)
• Atrial septostomy  
  – Creates right to left shunt  
  – Decrease in oxygenation is compensated for by increase in cardiac output  
  – Palliative or bridge to transplant
• Transcatheter Potts shunt  
  – Retrograde needle perforation of the descending aorta at the site where it connects to the left pulmonary artery  
  with deployment of a covered stent between two vessels  
  – Brain and myocardium are not exposed to desaturated blood
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
Acute Respiratory Failure: Causes

- **Type I (oxygenation failure)**
  - Pathophysiology: Decreased V/Q ratio (shunting), diffusion defect
    - Pneumonia
    - Pulmonary edema
    - ARDS
  - Alveolar / capillary exchange impacted

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

COPD

- **Enhanced chronic inflammatory response**
  - Caused by noxious stimuli
- Enlarged mucous secreting glands / increased goblet cells: Increased mucous production / Ciliary dysfunction
- Inflammation and increased mucous production = increased airway resistance
- Persistent airflow limitation usually progressive in nature
  - Small airway disease (obstructive bronchiolitis)
  - Destruction of the alveoli and other lung structures (termed emphysema)
  - Processes persist even after tobacco cessation
- Airways cannot remain open during expiration: Trapped air and hyperinflation
- Tissue destruction also leads to impaired gas exchange
  - Decreased surface area for gas exchange
Pathophysiology

• Neutrophils play major role
  – Cigarette smoking increases neutrophils
• Neutrophils and macrophages release enzymes that digest elastin
• Neutrophil elastase is intended to destroy bacteria
  – But, due to excess destroys elastin found in connective tissue
• Some patients have Alpha-1 antitrypsin deficiency which is responsible for which protects tissues from neutrophil elastase

Manifestations of Disease

• **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.

• Airway resistance = abnormal ventilation = alveolar hypoventilation = hypercapnia / alveolar hypoxia = eventual arterial hypoxemia

• Pulmonary hypertension develops:
  – Hypoxic vasoconstriction
  – Endothelial damage leading to intimal and smooth muscle hyperplasia
  – Acute cor pulmonale can develop
Comparison

**Emphysema**
- Abnormal permanent enlargement of the airspaces distal to the terminal bronchioles accompanied by destruction of the alveolar wall with no obvious evidence of fibrosis
  - Air sacs are replaced by bullae
- Emphysema is a component within COPD

**Chronic Bronchitis**
- Independent disease entity
- Defined as a chronic cough and sputum production on a daily basis for a minimum of three months a year, and not less than two consecutive years, with other causes of the cough excluded
- Can precede or follow development of airflow limitation / can also accelerate airflow limitation / some pts no airflow limitations
Clinical Presentation

- Reduced inspiratory capacity, particularly during exercise due to hyperinflation

- Hypoxemia and potential hypercapnea due to V/Q mismatching
  - Increased hypoxemia during sleep

- Increased RBC production in response to chronic hypoxemia
  - May also have central cyanosis

- Potential for right sided heart failure

Diagnosis

- Suspect COPD in patients > 40 years
  - Dyspnea that is persistent, progressive, and worse with exercise.
    - Chronic cough (often 1st sign) including intermittent and nonproductive coughs.
  - Chronic sputum production.
  - Exposure to tobacco smoke, smoke from home cooking or heating, and exposure to environmental chemicals.
  - Family history of COPD.

* Physical exam findings usually not present until advanced disease.
Diagnosis

• Formal diagnosis is made with spirometry.
• Forced vital capacity (FVC) and forced expiratory volume in the first second (FEV₁) are measured after bronchodilator therapy. The ratio of FEV₁ / FVC is obtained.
• FEV₁/FVC ratio is < 70% indicates obstruction to airflow.

Classification System

<table>
<thead>
<tr>
<th>GOLD Classification of COPD</th>
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<tbody>
<tr>
<td>Gold Category</td>
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<tr>
<td>GOLD 1</td>
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<td>GOLD 2</td>
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<td>GOLD 3</td>
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<td>GOLD 4</td>
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</tbody>
</table>

Source: GOLD, 2014

* Only applies when FEV₁/FVC ratio is < 70%.
Nursing Implications

• QOL related to dyspnea
• Etiology of exacerbations
• Anorexia, weight loss, and fatigue
• Skeletal muscle dysfunction and opportunity for improvement

COPD: Treatment

• Smoking cessation
• Vaccines
• Treatment of sleep apnea
• Pulmonary Rehab
  – Improves perceived breathlessness and increases exercise capacity,
  – Enhances effect of long term bronchodilators
  – Reduces anxiety and depression and improves health-related quality of life
  – Reduces hospitalization and length of stay, enhances recovery after exacerbation, and improves survival
COPD: Treatment

• Medications to reduce symptoms, exacerbations and to improve exercise tolerance; no evidence for slowing of disease progression
• Oxygen
  – Improves survival in severe resting hypoxemia
  – Reversal of hypoxemia more important than CO\(_2\) retention
  – Reduces pulmonary vasoconstriction and improves V/Q mismatching

More on Oxygen Therapy

• Worn 24 hours per day in patients with resting hypoxemia
• Survival benefit requires \(O_2\) therapy 15 hours per day
• Dosed to achieve a rest \(SpO_2\) of \(\geq 90\%\).
  – 20 to 30 minutes between adjustments
• Sleep level of oxygen
  – Increasing the flow by 1 L during sleep, or
  – Using a sleep study to determine the optimal level of oxygen.
• Exercise oxygen levels should be titrated to maintain a \(SpO2 > 90\%\).
Oxygen Criteria

Criteria for Long Term Oxygen Administration

| Room air / resting PaO₂ ≤ 55 mmHg with SaO₂ ≤ 88% with or without hypercapnia. | Room air / resting PaO₂ between 55 and 59 mmHg or SaO₂ of 89% with evidence of pulmonary hypertension, right sided heart failure, or polycythemia (hematocrit > 55%). | Room air / resting PaO₂ of ≥ 60 mmHg or SaO₂ of ≥ 90%, with special clinical circumstances such as sleep time desaturations not corrected by continuous positive airway pressure (CPAP), exercise desaturations, or severe dyspnea that improves with oxygen therapy. |

There are no other required clinical features if the patient meets these criteria.

Source: American Thoracic Society, 2014

Bronchodilator Therapy

- Treat the reversible component of the airway obstruction
- Long acting and inhaled preferred
- Anticholinergics and beta 2-agonists are most commonly used agents
- Single agents or agents in combination
  - Combination short acting beta 2-agonist and anticholinergic in one inhaler
  - Combination long acting beta 2-agonist and anticholinergic in one inhaler
  - Combination long acting beta 2-agonist and corticosteroids in one inhaler
- Toxicity is dose related
Inhaled Therapy

- Dry powder, metered dose breath activated devices, supplemental spacer devices, nebulizers - often used in exacerbation.
- Incorrect technique:
  - Increased ED visits,
  - Increased hospital admissions
  - Increased use of corticosteroids and antibiotics
- Factors affecting correct use:
  - Advanced age
  - Lower levels of education
  - Most significantly, lack of instruction by a health care provider
  *(Melani et al., 2011).*

Acute Exacerbation

- Viral infection of the upper respiratory tract or the tracheobronchial tree
- Other
  - Bacterial: 3 most common are *Hemophilus influenza*, *Streptococcus pneumoniae*, and *Moraxella catarrhalis*. *Pseudomonas aeruginosa* in more advanced COPD.
  - Environmental
  - Non adherence
  - Unknown
- Associated with worsening lung function and increased mortality
- Required hospitalization = poor prognosis
- Several week recovery period
### Clinical Presentation in Acute Exacerbation

- Dyspnea, cough, or sputum production that is a change from baseline.
- Use of accessory muscles when breathing
- Paradoxical chest wall movement
- New or worsened central cyanosis
- Altered mental status
- Evidence of right sided heart failure

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### Specific Treatment Issues in Acute Exacerbation

#### Oxygen / Ventilation

- Important to know if patient is a chronic CO₂ retainer
- If so – drive to ventilate is based on hypoxic drive
- Keep saturation between 88-92% * treatment of hypoxemia is priority
- It is the oxygen saturation level that is important – not the FIO₂
- Ventilatory support required if respiratory acidosis or increased work of breathing – prefer BiPAP

#### Blood Gas Goals

- Important to know if patient is a chronic CO₂ retainer
- If so – the key to assessing decompensation is when the pH become abnormal (no longer is this a compensated respiratory acidosis)
- The goal is to return the pH to normal – not to return the PaCO₂ to normal
Case Example

• Patient history: COPD (CO$_2$) retainer
• Initial presentation:
  – Tachypneic with increased work of breathing
  – SpO$_2$ of 78%

The previous patient most likely has what type of acute respiratory failure:

1. Oxygenation failure
2. Ventilatory failure
3. This is not acute respiratory failure due to history of CO$_2$ retention
Pending blood gas results, the best initial treatment would include:

1. CPAP
2. BiPAP
3. \( \uparrow \text{FIO}_2 \) to achieve saturation 90-92%
4. CPAP and increased FIO\(_2\)
5. BiPAP and increased FIO\(_2\)

Case Example

- ABG
  - 7.29
  - \( \text{PaCO}_2 \) 60 mmHg
  - \( \text{HCO}_3 \) 30 mEq/L
  - \( \text{PaO}_2 \) 48 mmHg
Blood gas goals include:

1. Normalization of pH
2. Normalization of $\text{PaCO}_2$
3. Both of the above
4. Neither of the above

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.
Bacterial Pneumonia: Presentation

- Hyperthermia (fever, typically >38°C) or hypothermia (< 35°C)
- Tachypnea (>18 respirations/min)
- Use of accessory respiratory muscles
- Tachycardia (>100 bpm) or bradycardia (< 60 bpm)
- Central cyanosis
- Altered mental status

Potential Physical Exam Findings

- Adventitious breath sounds, such as rales/crackles, rhonchi, or wheezes
- Decreased intensity of breath sounds
- Egophony
- Whispering pectoriloquy
- Dullness to percussion
- Tracheal deviation
- Lymphadenopathy
- Pleural friction rub
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
Sputum in Pneumonia

- *Streptococcus pneumoniae*: Rust-colored sputum
- *Pseudomonas*, *Haemophilus*, and pneumococcal species: May produce green sputum
- *Klebsiella* species pneumonia: Red currant-jelly sputum
- Anaerobic infections: Often produce foul-smelling or bad-tasting sputum
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

- **Decision to admit**
  - Pneumonia severity index (PSI) score as a guide for inpatient care and mortality risk.
- **Prevent nosocomial infections**
- **Timely Antibiotics**
  - Cover pneumococcus
- 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
- Patients with severe periodontal disease, putrid sputum, or a history of alcoholism may be at greater risk of anaerobic infection.
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 mmHg or no evidence of increased left-atrial pressure
- No evidence of COPD
- No other explanation for above
Non Cardiac Pulmonary Edema: ARDS

ARDS: Treatment

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

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 arterial hypertension

Mechanical Ventilator Management Strategies for ARDS

- Lower tidal volume ventilation
  - Permissive hypercapnia
- Maintain plateau pressure < 30 mmHg
- Uninterrupted PEEP
- Avoidance of auto PEEP
- Open Lung / Recruitment Modes of Ventilation
  - Airway pressure release ventilation
  - High frequency ventilation (Oscillatory)
Additional Options in ARDS

Independent lung ventilation
- If degree of alveolar collapse is not uniform

- ECMO (extracorporeal membrane oxygenation)
- Venous – venous: facilitates gas exchange but does not provide for hemodynamic support because the blood is returned to the right side of the heart before it enters pulmonary circulation.
- Used as an alternative strategy in adults with ARDS to rest the lungs and avoid insult of mechanical ventilation.
- Hemorrhage and infection are the two most common complications of ECMO.

Partial Liquid Ventilation

- Lung is partially filled with perfluorocarbons
  - To level of functional residual capacity (40% of lung capacity)
  - Properties similar to surfactant
- Patient ventilated conventionally with Vt
- Improvement in compliance:
  - Recruitment of alveoli
  - Possible direct effect on surface tension
- Other possible benefits
  - Protective effect from infection
  - Wash out of inflammatory debris
Prone Position in Severe ARDS

- Multicenter prospective randomized trial.
- Randomized to 16 hours of the prone position or to usual care of the standard supine position.
- The 28-day mortality rate was 16.0% in the prone group and 32.8% in the supine group (statistically significant). Also associated with a reduction in 90-day mortality.
- There were no significant adverse effects with the proning intervention. Patients in the supine group had a higher rate of cardiac arrest (Guérin et al., 2013).

Case Example

- 65 year old female; 85 kg
- Post witnessed cardiac arrest
- Initial $\text{PaO}_2 / \text{FIO}_2$ ratio 102
Based on the PaO$_2$/FIO$_2$ ratio, the initial diagnosis in the previous patient is:

1. ARDS
2. Acute lung injury

Case Example

- Ventilator settings:
  - Assist control
  - Rate 12
  - Vt 700 ml
  - FIO$_2$.8
  - PEEP 5 cmH$_2$O

- 2$^{nd}$ ABG
  - pH: 7.33
  - PaCO$_2$: 40 mmHg
  - HCO$_3$-: 14 mEq/L
  - PaO$_2$: 92 mmHg
Most probable anticipated ventilator changes include:

1. Increased rate
2. Increased Vt
3. Increased FIO₂
4. Increased PEEP

Ventilator settings:
- Assist control
- Rate 12
- Vt 700 ml
- FIO₂ 0.8
- PEEP 5 cmH₂O

2nd ABG
- pH: 7.33
- PaCO₂: 40 mmHg
- HCO₃⁻: 14 mEq/L
- PaO₂: 92 mmHg

If this patient develops ARDS, what potential open lung ventilation mode might be considered:

1. Controlled mandatory ventilation
2. Synchronized intermittent mandatory ventilation
3. Airway pressure release ventilation
4. Spontaneous breathing trial
Blunt Trauma

• Etiology
  – Motor vehicle crash: Responsible for 70 to 80% of blunt trauma

• Chest wall injury (ribs)

• Direct lung injury (pulmonary contusion)

• Direct injury to the heart and great vessels

Diagnosis

• CXR is initial diagnostic study
  – Tension pneumothorax does not require a CXR for treatment
  – May not always be necessary in stable patients

• CT scan is more sensitive (chest, abdomen, and cervical)

• TEE to assess rupture of thoracic aorta

• Transthoracic Echo for tamponade

• Fiberoptic or rigid bronchooscopy for tracheobronchial injuries
  – Endotracheal tube can be loaded on scope
Treatment: Immediate Surgery

• Loss of chest wall integrity
• Blunt diaphragmatic injuries
• Massive air leak after chest tube insertion
• Massive hemothorax (1500 ml of blood on CT insertion)
• Continued high blood loss after CT insertion (250 ml/hour for 3 consecutive hours)
• Confirmed tracheal, major bronchial, or esophageal injury
• GI contents in chest tube

Treatment: Immediate Surgery

• Cardiac tamponade
• Great vessel injury
• Embolism (pulmonary artery or heart)
Rib Fractures

- Seen in 50% of patients with blunt trauma
- Ribs 4-10 common
  - 8-12 associated with abdominal trauma
  - 1-2 are well protected: Must rule out other significant injury
- Tenderness
- Crepitus over sight

- Increases pneumonia and mortality in elderly
- Pain control is hallmark of treatment
  - Early mobilization
  - Pulmonary toilet
Flail Chest

- 3 or more consecutive rib fractures in 2 or more places
  - Free floating, unstable segment of chest wall
- Can also be caused by costochondral separation

- S&S
  - Pain
  - Dyspnea (increased work of breathing)
  - Paradoxical motion of flail segment
  - Associated injuries are common due to force required to produce flail injury
Treatment of Flail Chest

• Same as for fractured ribs
• Caution with fluids – respiratory failure can occur especially when there is co-existing pulmonary contusion
• **Surgery is not superior to supportive care**
• However, surgical intervention for stabilization if thoracotomy is indicated for another reason

Sternal Fracture

• Upper and middle 1/3 most commonly affected
• Inspiratory pain
• 55-70% of patients will have associated injuries
• Blunt cardiac injury present in < 20% of patients
  – Begin workup with ECG
Traumatic Asphyxia

• Crushing (pinned) etiology

• Presentation
  – Cyanosis of head and neck
  – Subconjunctival hemorrhage
  – Periorbital echymosis
  – Petechiae of head and neck

• Treatment focus
  – Elevate HOB 30 degrees
  – Assure adequate airway
  – Follow up neuro evaluation
  – No specific surgical intervention

Pulmonary Contusion

• Pulmonary infiltrates with hemorrhage in the lung tissue

• S&S: Depend on the extent of injury

• Can cause hemothoraces

• Treatment
  – Pain control
  – Pulmonary toilet
  – Oxygen
  – Thoracotomy to get surgical control of bleeding vessels
  – Pneumonectomy may be necessary
Blunt Cardiac Injury

- Impact can range from transient arrhythmias to myocardial rupture

- Valvular Injury
  - Aortic valve most affected
  - Aortic regurgitation / CHF

- Rupture
  - Septum
  - Valves
  - Free wall

- Free wall rupture:
  - Right side most often affected
  - Treat as tamponade
  - Beck’s triad:
    - Hypotension, JVD, Muffled heart sounds
Blunt Injury of Thoracic Aorta and Thoracic Arteries

• Many die at the scene
• S&S
  – Those of aortic dissection
  – May also have signs of cardiac tamponade
  – May also have signs of hemothorax
Blunt Trauma Summary

• > 80% of patients require no invasive therapy or only a tube thoracostomy
• High mortality associated with cardiac tamponade and great vessel rupture.

Future:
– Increased use of thoroscopy for diagnosis and treatment.
– Ultrasound to diagnose tamponade and hemothorax.
– Spiral CT for major vascular lesions.
– Endovascular repair for great vessels.

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
  – If associated with hemorrhage: Hemopneumothorax

• Types / Etiology
  – Primary (no underlying lung disease)
    • Blebs / bullae
    • Smoking
  – Secondary (underlying lung disease)
    • Air enters through damaged aveoli
    • COPD
  – Blunt trauma (lung laceration by rib fracture)
  – Iatrogenic – from medical procedure
  – Positive pressure ventilation (rupture of weak alveoli, bleb or bullous)
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 to absent breath sounds on affected side, dramatic increases in peak inspiratory pressures on a mechanical ventilator

• **Treatment**
  - Oxygen
  - Pulmonary toilet
  - Observation (asymptomatic, small primary)
  - Aspiration (symptomatic small primary)

  - **Chest Tube Criteria**
    - Pneumothorax secondary to trauma
      - Suction used until air leak resolved, then change to waterseal
    - No suction in spontaneous pneumo due to late presentation and risk of re-expansion pulmonary edema

  - Spontaneous primary pneumothorax may take 12 weeks to resolve.
Open Pneumothorax

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

• **Pathophysiology**
  – Equilibrium between intrathoracic and atmospheric pressures
  – Patient condition depends on size of opening compared to trachea
  – The affected lung collapses during inspiration
  – May cause a tension pneumothorax

• **Signs and Symptoms**
  – Significant to complete loss of breath sounds
  – Subcutaneous emphysema usually present

Open Pneumothorax

• **Treatment**
  – Closure of open wound with 3 way petroleum jelly gauze
    • End expiration
  – Chest tube and water seal drainage
  – Possible surgical debridement and wound closure (patches)
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
  – Clamped or clotted water seal drainage system
  – Airtight dressing on open pneumothorax
  – **Positive pressure mechanical ventilation** – rule out if hypotension

Tension Pneumothorax

• **Pathophysiology**
  – Air rushes in-cannot escape pleural space
  – Creates positive pressure in pleural space
  – Ipsalateral lung collapse
  – Mediastinal shift ➔ contralateral lung compression ➔ potential tearing of thoracic aorta
  – Can also compress heart ➔ decrease RV filling ➔ shock
Tension Pneumothorax

**Signs and Symptoms**

- Decreased / absent lung sounds and hyper-resonance on percussion.
- If mediastinal shift:
  - Tracheal shift away from affected side
  - JVD
  - Hypotension

**Treatment**

- Oxygen (100%)
- Chest tube
- Emergency decompression
  - Large bore needle (14 to 16 gauge)
  - 2nd ICS, MCL

---

Tension Pneumonia

![Image of a chest X-ray and diagram of a ribcage with labels for parietal pleura and visceral pleura, indicating air and a syringe entering the chest cavity.](image-url)
Hemothorax

- Blood enters the pleural space
  - Bleeding from chest wall (lacerations of intercostal or internal mammary vessels)
  - Hemorrhage from lung parenchyma or major thoracic vessels

- S&S: Decreased breath sounds, **dullness to percussion**

- Treatment:
  - Tube thoracostomy
  - May need multiple CTs
  - * Monitor drainage to determine need for surgery
  - Clot may need surgically evacuated
  - Pain control
  - Pulmonary toilet
Chest Tubes

May also be in mediastinal space after open heart surgery.

Chest Tubes: Drainage

- **Assessment of chest tube patency is a key nursing function**
  - Dumping of blood with a position change may indicate an acute onset of bleeding (if dark in color and minimal additional drainage then not acute)
- **Chest tubes should always be assessed for evidence of hemorrhage when there is a low blood pressure**
  - Decreased breath sounds, increased inspiratory pressures on the ventilator, or widening of the mediastinum on CXR: suspicion for undrained blood in the pleural space or in the mediastinum
Chest Tube: Water Seal

- Water seal allows air to exit from the pleural space on exhalation and prevent air from entering the pleural cavity or mediastinum on inhalation

- To maintain an adequate water seal it is important to monitor the level of water in the water seal chamber and to keep the chest drainage unit upright at all times.

- Assess the water seal chamber for slight fluctuation (tidaling)
  - Tidaling (rising during spontaneous inspiration and falling during expiration) is normal
  - Lack of fluctuation with respiration may indicate kinking or other problems interfering with drainage.
    - May also be a good sign indicating lung re-expansion.

Chest Tube: Water Seal

- Assess for air leak by checking water seal chamber for bubbles during inspiration.
  - May bubble gently with insertion, during expiration and with a cough.
  - Continuous bubbling represents an air leak.
  - Check for system leaks by clamping before each connection (system may need to be replaced).
  - Check for leak where tube enters chest.
  - Check chest x-ray to assure last hole of chest tube is inside chest.
Chest Tube: Other Nursing Considerations

- Assess the insertion site for subcutaneous emphysema.
- Keep unit below the level of the patient’s chest
- Do not clamp chest tube for transport (can cause tension pneumothorax with pleural chest tubes or tamponade with mediastinal chest tubes)
  - Use portable suction or transport on gravity drainage with tubing from suction chamber open to air

Chest Tubes

- Rare but serious complication is the development of unilateral pulmonary edema in response to rapid re-expansion or rapid evacuation of pleural fluid.
  - Capillary permeability can increase as result of rapid treatment, resulting in pulmonary edema.
  - Strategies for prevention:
    - Avoiding suction with the initial drainage of a large pleural effusion or the expansion of a large pneumothorax
    - Clamping after the 2 liters of initial drainage
## Pulling it All Together

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Ventilation</th>
<th>Perfusion</th>
<th>Diffusion</th>
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</thead>
<tbody>
<tr>
<td>• Rate and depth of respirations&lt;br&gt;• Work of breathing&lt;br&gt;• PaCO₂</td>
<td>• VQ Scan&lt;br&gt;• Spiral CT&lt;br&gt;• S&amp;S associated with P.E. * Respiratory alkalosis&lt;br&gt;Global: Decreased C.O.</td>
<td>• Oxygenation status&lt;br&gt;• SpO₂ or SaO₂&lt;br&gt;• PaO₂</td>
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<table>
<thead>
<tr>
<th>Treatment</th>
<th>Ventilation</th>
<th>Perfusion</th>
<th>Diffusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Increase rate or Vt&lt;br&gt;• Reverse sedation&lt;br&gt;• Ambu bag&lt;br&gt;• BiPAP&lt;br&gt;• Intubate and ventilate</td>
<td>• Anticoagulation&lt;br&gt;• Fibrinolytic&lt;br&gt;• Embolectomy&lt;br&gt;• Support right ventricular function</td>
<td>• Increase FIO₂&lt;br&gt;• CPAP&lt;br&gt;• Add PEEP if necessary to increase driving pressure&lt;br&gt;• Treat underlying cause</td>
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## Associated Pathological Conditions

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