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

Ventilation

- **Minute ventilation** \( (V_e) = \) Total volume of air expired in one minute
  - **Respiratory rate** \( \times \) **tidal volume** \( (V_t) \) (total volume = amount of air per breath)
  - Normal minute ventilation = \( 12 \times 500 \text{ ml} = 6000\text{ml} \)
  - **Note:** (hypoventilation can occur with normal or even high respiratory rate)

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
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)

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 PaCO₂ (inversely related to V̇e)
  - PCO₂ > 45 mm Hg indicates alveolar hypoventilation *
  - PCO₂ < 35 mm Hg indicates alveolar hyperventilation

Note: Only one physiologic reason for increased PaCO₂

Treatment of Ventilation Problems

- 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.

Partial pressure of O₂: 100 (104) mmHg

Inspired gas P(I)O₂: 149 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 PaCO₂ to determine if inadequate ventilation contributed to the hypoxemia.

Perfusion

- Definition: The movement of blood through the pulmonary capillaries

Perfusion 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) =
    - Increased capillary recruitment =
    - Decrease in PVR
    - High lung volumes pull pulmonary vessels open. Results in a decrease PVR.

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 FIO$_2$ and increased pressure (CPAP / PEEP) will increase driving pressure of oxygen

Power Your Memory Time!!

- **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

- In decreased ventilation to perfusion ratio
  - V/Q = 0
  - (Intrapulmonary Shunting)
  - Alveolar O$_2$ will fall
  - Alveolar CO$_2$ will rise
Increased V/Q Ratio (Dead Space)

In increased ventilation perfusion ratio
Alveolar O₂ will rise
Alveolar CO₂ 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

A patient is admitted with new onset dyspnea s/p discharge last week from trauma secondary to motor vehicle crash. CTA reveals PE in RLL.

What is true about V/Q ratio:

- Increased
- Decreased
- No effect

You are caring for a patient who is being treated for bilateral community acquired pneumonia. CXR today shows worsening of bilateral infiltrates.

What is true regarding V/Q ratio:

- Increased
- Decreased
- No effect

Assessment Tools and Pearls

Ventilation: Patient End Tidal CO₂ (PetCO₂)

- Capnography: evaluation of the CO₂ level in the respiratory gases.
- Includes both the continuous analysis and the continuous recording of the CO₂.
  - 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 PaO₂ (arterial) without considering alveolar oxygenation content (PAO₂)
  - Increase in FIO₂ will increase PAO₂
  - Increase in PACO₂ will decrease PAO₂

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

PaO₂ and FIO₂ Ratio

• An assessment and trending tool
• PaO₂ / FIO₂ ratio:
  - Normal well above 300
  - Acute lung injury < 300
  - ARDS <= 200

PaO₂ of 60 mmHg with an FIO₂ of 0.5 (50%) represents a PaO₂ /FIO₂ ratio of 60 / 0.5 = 120.
This is a clinically significant intrapulmonary shunt.

Linking Knowledge to Practice with PaO₂ / FIO₂ 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>318</td>
<td>Continued treatment, FIO₂ decreased</td>
</tr>
<tr>
<td>150</td>
<td>40%</td>
<td>375</td>
<td>Clinical improvement, FIO₂ 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

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 pigmentation
- 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
- Plasma: 3% of oxygen is dissolved in plasma
- Represented by the SaO₂
- Represented by the PaO₂ (measurement of O₂ tension in plasma)

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

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

- **pH**
  - Normal 7.35-7.45
  - < 7.35 Acidemia
  - > 7.45 Alkalemia

- **PaCO₂**
  - Normal 35-45 mmHg
  - < 35 mmHg respiratory alkalosis or compensation for metabolic acidosis
  - > 45 mmHg respiratory acidosis or compensation for metabolic alkalosis

- **HCO₃⁻**
  - Normal 22-26 mEq/L
  - < 22 mEq/L metabolic acidosis or compensation for respiratory alkalosis
  - > 26 mEq/L metabolic alkalosis or compensation for respiratory acidosis

- **PaO₂**
  - Normal 80-100 mmHg
  - >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

- **Base Excess (BE)**
  - Normal +2 to -2
  - < -2 (base deficit) metabolic acidosis or metabolic compensation for respiratory alkalosis
  - > +2 metabolic alkalosis or metabolic compensation for respiratory acidosis

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

**Normal**
- Na 140
- Chloride 103
- HCO$_3^-$ 28
- $140 - (103 + 28) = 9$

**Abnormal**
- Na 140
- Chloride 99
- HCO$_3^-$ 18
- $140 - (99 + 18) = 23$

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

Power Your Memory

With Practice ABGs
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

| pH   | 7.30 |
| PaCO₂ | 40 mmHg |
| HCO₃  | 18 mEq/L |
| PaO₂  | 85 mmHg |

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

| pH   | 7.21 |
| PaCO₂ | 60 mmHg |
| HCO₃  | 20 mEq/L |
| PaO₂  | 48 mmHg |
This Blood Gas Represents
1. Respiratory alkalosis
2. Metabolic alkalosis
3. Mixed respiratory and metabolic alkalosis
4. None of above

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.54</td>
</tr>
<tr>
<td>( \text{PaCO}_2 )</td>
<td>25 mmHg</td>
</tr>
<tr>
<td>( \text{HCO}_3^- )</td>
<td>30 mEq/L</td>
</tr>
<tr>
<td>( \text{PaO}_2 )</td>
<td>95 mmHg</td>
</tr>
</tbody>
</table>

Oxygen Therapy and 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 FIO\(_2\)
- Doesn’t mean a high 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
- FIO\(_2\) depends on rate and depth of ventilation and fit of device
- Doesn’t have to mean low FIO\(_2\)
- 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(s):
  - Nasal Cannula:
    - 1
    - 2
    - 3
    - 4
    - 5
    - 6
  - Oxygen Mask:
    - 5-6
    - 7-8
    - 9-10
  - Mask with Reservoir:
    - 6
    - 7
    - 8
    - 9
    - 10

- FIO\textsubscript{2} (%):
  - 24
  - 28
  - 32
  - 36
  - 40
  - 44
  - 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
  - Paraesthesia 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

Other Therapeutic Gases

- Heliox gas mixture (79% helium and 21% oxygen):
  - May be used in acute lung injury, ARDS, COPD exacerbation, asthma and other conditions
  - Most evidence appears to be in pediatrics
  - Lacks evidence-based recommendations in clinical practice
  - Technical issue: can interfere with ventilator functioning and the delivery of adequate tidal volume
  - Also need to assure adequacy of inspired oxygen

- Nitric oxide:
  - Inhaled gas that is potent pulmonary vasodilator
  - Approved by the United States Food and Drug Administration (FDA) for the treatment of term and near-term neonates with hypoxemic respiratory failure associated with clinical or echocardiographic evidence of pulmonary arterial hypertension.
  - Overdosage with inhaled nitric oxide:
    - Elevations in methemoglobin
    - Acute lung injury.

Mechanical Ventilation

Indications

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

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 H\textsubscript{2}O

Biphasic Positive Airway Pressure:
  - Senses inspiration and delivers higher pressure during inspiration
  - 12 / 6 cmH\textsubscript{2}O is a common setting

Consider as first line strategy
Consider as alternative to failed weaning
Decreased VAE

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.

**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
Adaptive Support Ventilation

- 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 CO$_2$
  - Facilitates oxygenation
  - P High (20–30 cmH$_2$O) and P low (0) (pressure)
  - T high (4-6 seconds) and T low (0.8 seconds) (time)

- **Inverse ratio ventilation**

- 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

- 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.

- Time triggered
- Time cycled
- Pressure limited

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 6 to 8 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 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 H$_2$O 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

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 breath
• 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

Lung volume at end of expiration is greater than functional residual capacity.

Auto PEEP

Factors Shortening Expiratory Time

• High rate
• High VT

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

**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

**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)

**Measured Parameters**

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

**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

**Power Your Memory Again 😊**

- Your ventilator has been persistently alarming due to the exceeding the high pressure alarm. Respiratory therapy is at the bedside and indicates the plateau pressure is high.

- True or False:
  - More frequent suctioning is needed to prevent the high pressures causing the alarms?
**Clinical Practice Guidelines for the Management of Pain, Agitation, and Delirium in Adult Patients in the Intensive Care Unit**


Updated:
Crit Care Med. 2018 Sep;46(9):e825-e873Published: 8/22/2018

---

**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: > 5 ml/kg
- Vital capacity: > 10 ml/kg, ideally 15 ml/kg
- Minute ventilation: < 10 L
- Negative inspiratory pressure: < -25 to -30 cm H₂O
- FIO₂: < 0.50
- PaO₂ / FIO₂ 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.

Failed Ventilator Weaning

- Altered lung mechanics (compliance and resistance issues)
- Respiratory muscle dysfunction
  - Electromyography and ultrasound can evaluate respiratory muscle function
  - Phosphate and magnesium deficiency associated with ventilatory muscle weakness
  - Strength and endurance training
  - Proper nutrition can facilitate ventilator weaning
- Cardiac dysfunction
  - Ultrasound and brain natriuretic peptides can evaluate cardiac dysfunction
  - Possible treatment with vasodilators and inotropes
- Cognitive dysfunction
- Metabolic disorders

Failed Ventilator Weaning

- Focus on determining etiology
- Identify strategies to target the etiology

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

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 PaO\(_2\)
  - Normal PaCO\(_2\)
  - Widened A-a gradient

- Type II: Hypoxemic Hypercapnic
  - Low PaO\(_2\)
  - High PaCO\(_2\)
  - Normal A-a gradient

Acute Respiratory Failure: Causes

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

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

Acute Pulmonary Embolus
**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 DVT**

- PROLONGED IMMOBILIZATION
- RECENT TRAUMA
- PLASTER CASTS
- BURNS
- ORTHOPEDIC / SPINE SURGERY
- CENTRAL VENOUS CATHETERS
- PREGNANCY
- ORAL CONTRACEPTIVES
- VARICOSE VEINS
- PHLEBITIS
- DEHYDRATION / HYPOVOLEMIA
- POLYSYTHEMIA VERA
- SICKLE CELL DISEASE
- BEHCET'S DISEASE
- DEFICIENCY IN PROTEIN C, PROTEIN S, OR ANTITHROMBIN III
- FACTOR V LEIDEN MUTATION
- HEART FAILURE
- MYOCARDIAL INFARCTION
- COPD
- HIV / AIDS
- MALIGNANCY
- SHOCK

* Obesity is most common preventable cause of DVT.

**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**

**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 approx 8% of patients presenting with PE (Up to Date 2019).
- Involves both the right and left pulmonary arteries or causes hemodynamic collapse
- Presenting systolic BP of < 90 mmHg for > 15 min
  – Or requiring vasopressor, or
- Mortality rates range from 30% to 60% and most deaths occur within the first 1 to 2 hours (Ouellette et al., 2013; Wood, 2002).
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 ($\downarrow$ CO$_2$)
  - 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 2nd 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

Diagnosis

- Computed tomography pulmonary angiography (CTPA) standard test for the diagnosis of PE
- Portable perfusion scan available in some centers
- Bedside echocardiography
  - Assessment of RV
- Cardiac troponins will be elevated in half of patients with moderate to large PE (Konstantinides, 2008)
- Use of ultrasound to rule out DVT
- VQ scan is used as alternative

ECG in PE

- The ECG has poor sensitivity in PE
- Changes are often transient and non-specific
- Pre-existing comorbid conditions confound the use of the ECG
- ST with non specific ST / T wave changes most common finding (50%)
  - Not specific to PE
- The ECG with the clinical picture can be helpful

Other signs

- S1, Q3, T3
  - 15 to 25% of PE patients
- Short heart axis
- RBBB or incomplete RBBB
  - Can produce ST elevation in V1 or V2
  - Right or left axis deviation
  - Affected by pre-existing disease
- ST (most common) or atrial arrhythmias
- T wave inversion in limb and precordial leads
- Tall P wave in lead II
- P pulmonale
Treatment

• Treatment with anticoagulation in non-massive PE reduces mortality to 2 to 11% (Up to Date 2019)
• 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 / pregnancy
    • Low risk patients can have home treatment or early discharge
    • Management of subsegmental PE is controversial

Catheter Based Techniques

• No superiority proven; institution specific
• Ultrasound assisted thrombolysis
  – High frequency ultrasound to aid thrombolytic agent
• Rheolytic embolectomy
  – Pressurized saline
  – Venous cut down required
  – Adenosine released from platelets
    • Bradycardia, hypoxia, and vasospasm
  – Red blood cell fragmentation: hemoglobinuria
  – Boxed warning

Recurrence is greatest in 1st 2 weeks.

• 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
• There is risk of stroke in patients with PFO

Recurrence is greatest in 1st 2 weeks.

• IVC Filter:
  – Absolute contraindication to anticoagulation or bleeding risk too high
    • Usually even in absence of lower extremity DVT (if PE confirmed)
  – Post survival of massive PE where subsequent PE will prove fatal
  – Presence of venous thromboembolism with adequate anticoagulation
  – retrievable filter preferred
• 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

Catheter Based Techniques

• Rotational embolectomy
• Suction embolectomy
• Thrombus fragmentation with:
  – Angioplasty
  – Aspiration
  – Catheter directed thrombolysis

Treatment

• Individualized and multidisciplinary approach
• 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 – with or without thrombolysis
  – If high bleeding risk / failed systemic therapy / shock
• Surgical pulmonary embolectomy may also be considered in select patients
  – Thrombolysis contraindicated or failed
  – Trapped embolus: PFO, RA, RV
  – High mortality in elderly
  – Distal thrombus not amenable to surgery
Special Considerations Air Embolism

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

- **Treatment**
  - Prevention
  - Aspiration of air
  - Left lateral / Trendelenburg (for venous)
  - RV outflow tract becomes inferior to RV cavity
  - Arterial force moves air bubbles forward even in Trendelenburg
  - Want to avoid exacerbating any cerebral edema
  - 100% FIO₂
  - Increases air reabsorption rate
  - Hyperbaric oxygen for severe cases
  - Support hemodynamics / ventilatory support if needed

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>
<tbody>
<tr>
<td>Group 1</td>
<td>Pulmonary arterial hypertension</td>
<td>Idiopathic, heritable, drug or toxin induced, associated with conditions such as connective tissue disease, HIV, portal hypertension, chronic hemolytic anemia, congenital heart disease, and others.</td>
</tr>
<tr>
<td>Group 2</td>
<td>Pulmonary hypertension</td>
<td>From left heart disease. This is the most common cause of pulmonary HTN. Can result from systolic dysfunction, diastolic dysfunction, or valvular disease.</td>
</tr>
<tr>
<td>Group 3</td>
<td>Pulmonary hypertension</td>
<td>From lung disease or hypoxia. Can result from COPD, interstitial disease, deep disorders of breathing, and others.</td>
</tr>
<tr>
<td>Group 4</td>
<td>Pulmonary hypertension</td>
<td>From chronic thromboembolic disease.</td>
</tr>
<tr>
<td>Group 5</td>
<td>Pulmonary hypertension</td>
<td>Etiology is unclear multifactorial processes including hematologic, metabolic and systemic diseases.</td>
</tr>
</tbody>
</table>

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
  - 10 to 20% respond; idiopathic, heritable, and drug / toxin induced PAH most likely to respond
  - 3% of responders will have clinical response to calcium / channel blockers – usually short lived
  - 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) – strongest efficacy
  - IV
  - Nebulized inhalation (off label)
  - SE: Jaw pain, diarrhea, flushing, arthralgias
  - Life threatening crisis from infusion interruption less common with simultaneous administration of oral medications

- Treprostinil (Remodulin)
  - Inhalation
  - Oral
  - SQ (can cause severe pain at injection site) or IV continuous infusion
  - Similar SE as above

- Iloprost (Ventavis)
  - Nebulized inhalation: 6 to 9 times per day

- Selexipag (Uptravi)
  - Oral
  - Syncope most common SE

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

Can be used as single agent therapy

- Bosentan (Tracleer)
  - Oral
- Ambrisentan (Letairis)
  - Oral
- Macitentan (Opsumit)
  - Oral

Major side effects are peripheral edema and hepatotoxicity

Double method contraception required

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

Most common SE: HA, GI upset, flushing, muscle and joint pain
### Combination Oral Therapy

- **Ambrisentan** (Letairis) (ERA) and **Tadalafil** (Adcirca) (phosphodiesterase (type 5) enzyme inhibitor) – Preferred

**Other combinations**
- **Macitentan** (Opsumit) (ERA) and **Sildenafil** (Revatio) (phosphodiesterase (type 5) enzyme inhibitor)
- **Bosentan** (Tracleer) (ERA) and **Tadalafil** (Adcirca) (phosphodiesterase (type 5) enzyme inhibitor)
- **Bosentan** (Tracleer) (ERA) and **Riociguat** (Adempas) (Soluble Guanylate Cyclase Stimulator)
- **Selexipag** (Uptravi) prostacyclin analogue plus an ERA or a phosphodiesterase (type 5) enzyme inhibitor

### 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 – relies on RA hypertension
  - 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
  - Reduces RV afterload
  - Brain and myocardium are not exposed to desaturated blood

### 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 hypercapnia 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 (FEV$_1$) are measured after bronchodilator therapy. The ratio of FEV$_1$ / FVC is obtained.
- FEV$_1$/FVC ratio is < 70% indicates obstruction to airflow.

Classification System

### GOLD Classification of COPD

<table>
<thead>
<tr>
<th>Gold Category</th>
<th>Severity</th>
<th>FEV$_1$ Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>GOLD 1</td>
<td>Mild</td>
<td>FEV$_1$ &gt; 80% of predicted</td>
</tr>
<tr>
<td>GOLD 2</td>
<td>Moderate</td>
<td>FEV$_1$ &lt; 80% of predicted</td>
</tr>
<tr>
<td>GOLD 3</td>
<td>Severe</td>
<td>FEV$_1$ &lt; 50% of predicted</td>
</tr>
<tr>
<td>GOLD 4</td>
<td>Very Severe</td>
<td>FEV$_1$ &gt; 30% of predicted</td>
</tr>
</tbody>
</table>

* 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 CO₂ 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 O₂ therapy 15 hours per day
- Dosed to achieve a rest SpO₂ of ≥ 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 and SaO₂ ≤ 88% with or without hypercapnia.
- There are no other required clinical features if the patient meets these criteria.
- 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.

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
Medications Adverse Effects

- **Beta 2 – agonists**
  - Resting tachycardia
  - Body tremors in older patients

- **Anticholinergics**
  - Dry mouth

- **Methylxanthines**
  - Therapeutic effect at near toxic doses

- **Corticosteroids**
  - Increase risk for pneumonia
  - Steroid myopathy
  - Chronic respiratory failure

- **Phosphodiesterase -4 inhibitors**
  - GI symptoms and weight loss
  - Roflumilast cannot be used with theophylline and should be used with caution in patients with depression

Other Medications

- **Corticosteroids**: Remains controversial, but they are frequently used in treating exacerbations
- **Antibiotics**: used exacerbations caused by bacterial infections
- **Mucolytics**: Only with very viscous sputum; N-acetylcysteine may have a role in reducing exacerbations in patients not taking inhaled corticosteroids
- **Antitussives**: Cough plays protective role, not routinely used
- **Vasodilator therapy**: Not used in COPD
- **Narcotics**: May be used if benefit outweighs risk

Medications in COPD

<table>
<thead>
<tr>
<th>Medications in the Treatment of COPD</th>
<th>Beta Agonists (Short Acting)</th>
<th>Anticholinergics (Short Acting)</th>
<th>Anticholinergics (Long Acting)</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ Fenoterol</td>
<td>▪ Formoterol</td>
<td>▪ Ipratropium bromide</td>
<td></td>
</tr>
<tr>
<td>▪ Levalbuterol</td>
<td>▪ Arformoterol</td>
<td>▪ Oxitropium bromide</td>
<td></td>
</tr>
<tr>
<td>▪ Inhalebuterol</td>
<td>▪ Indacaterol</td>
<td>▪ Acidinium bromide</td>
<td></td>
</tr>
<tr>
<td>▪ Toprobuterol</td>
<td>▪ Salmeterol</td>
<td>▪ Glycopyrronium bromide</td>
<td></td>
</tr>
<tr>
<td>▪ Terbutaline</td>
<td>▪ Tulobuterol</td>
<td>▪ Tiotropium</td>
<td></td>
</tr>
</tbody>
</table>

- **Methylxanthines**
  - Inhaled Corticosteroids
  - Systemic Corticosteroids
  - Phosphodiesterase -4 Inhibitors

<table>
<thead>
<tr>
<th>Aminophylline</th>
<th>Theophylline (SR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ Aminophylline</td>
<td>▪ Theophylline (SR)</td>
</tr>
<tr>
<td>▪ Beclomethasone</td>
<td>▪ Budesonide</td>
</tr>
<tr>
<td>▪ Fluticasone</td>
<td>▪ Prednisone Methylprednisolone</td>
</tr>
<tr>
<td>▪ Methylprednisolone</td>
<td>▪ Roflumilast</td>
</tr>
</tbody>
</table>

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)*.

Additional Options

- Lung volume reduction surgery
- Bronchoscopic Lung Volume Reduction
- Bullectomy
- Endobronchial valves approved for severe COPD 9/2018
- Lung transplantation
- End of life decision making
  - Palliative Care versus Hospice Care
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

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% *
- The goal is to return the pH to normal – not to return the PaCO₂ to normal

**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

Power Your Memory With A Case Example

- Patient history: COPD (CO₂) retainer
- Initial presentation:
  - Tachypneic with increased work of breathing
  - SpO₂ 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 CO₂ retention

Pending blood gas results, the best initial treatment would include:

1. CPAP
2. BiPAP
3. Increased FIO₂ to achieve saturation 90-92%
4. 1 and 3
5. 2 and 3
ABG
- pH 7.29
- PaCO2 60 mmHg
- HCO3 30 mEq/L
- PaO2 48 mmHg

Blood gas goals
1. Normalization of pH
2. Normalization of PaCO2
3. Both of the above
4. Neither of the above

Status Asthmaticus
Exacerbation of acute asthma characterized by severe airflow obstruction that is not relieved after 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)

CXR likely to normal.

Status Asthmaticus: Presentation

- Tachypnea (rate > 30)
- Use of accessory muscles of inspirations
- Unable to speak full sentences
- Orthopnea
- Tachycardia (HR > 120)
- Pulses paradoxus

Note: 50% of people with severe airflow obstruction will not have any of these signs

Status Asthmaticus: Diagnosis

- Maximal expiratory airflow with peak flow meter or spirometer is best objective assessment of severity of asthma
  - Don’t do if respiratory failure is impending
  - < 200 L/min is severe obstruction in average adult
  - Hypercapnia is rarely present when peak expiratory flow is > 200 L/min
- Severe hypoxemia represents life threatening asthma or other complication (i.e. pneumonia)

Status Asthmaticus: Hypercapnia

- Respiratory drive increases in acute asthma resulting in hyperventilation
  - ▲PaCO2 is late sign
    - Airway narrowing is so severe despite increase central respiratory drive
- Respiratory failure can develop rapidly
- Mechanical ventilation indicated for progressive hypercapnia

Status Asthmaticus: Treatment

- Avoid benzodiazepines
- Effective bronchodilatation
- Reassurance
- Encouragement to take slow deep breaths
  - Minimize dead space ventilation and breath stacking
- Eliminate or treat cause
- No routine use of antibiotics
- IV hydration
- Oxygen to keep SpO2 > 92% (higher in pregnancy and cardiac disease) (lower if high risk for hypercapnia)

- Inhaled beta 2 agonists
  - Metered dose inhaler
  - Treat hypokalemia
- Inhaled anticholinergic (ipratropium)
- Glucocorticosteroids are the most important treatment for status asthmaticus refractory to intensive bronchodilator therapy
- Magnesium sulfate 2 g IV if nonresponsive to initial treatment
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 Pneumonia

- Older age (≥ 65 years)
- Previous viral respiratory infection
- Comorbid conditions
  - COPD
  - Heart failure
  - Stroke
  - Diabetes
  - Malnutrition
  - Immunocompromised state (corticosteroids)
- Gastro esophageal reflux disease (GERD)
- Chronic alcohol over use / cigarette smoking / opioid use
- Decreased level of consciousness / anesthesia / intubation
- Environmental toxins
- Crowded or low-income living conditions

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

Community Acquired Pneumonia

- Leading cause of morbidity and mortality worldwide
- 2nd most common cause of hospitalization
- Most common infectious cause of death
- Responsible pathogen only detected in ½ of cases

Community Acquired Pneumonia:
Causative Agents: Typical Bacteria

- Streptococcus pneumoniae (S. pneumoniae) (most common bacterial agent in community acquired pneumonia)
  - Also called pneumococcus
  - Incidence declining due to vaccination
  - Can be resistant to one or more antibiotics
- Haemophilus influenzae common among smokers
- Moraxella catarrhalis (particularly common in patients with chronic bronchitis).
- Staphylococcus aureus
- Group A streptococci
- Aerobic gram-negative bacteria (Klebsiella spp or Escherichia coli)
- Microaerophilic bacteria and anaerobes (associated with aspiration)
Community Acquired Pneumonia: Causative Agents: Atypical
- Legionella spp
- Mycoplasma pneumoniae
- Chlamydia pneumoniae
- Chlamydia psittaci
- Coxiella burnetti

Community Acquired Pneumonia: Causative Agents: Viruses
- Influenza A and B viruses
- Rhinoviruses
- Parainfluenza viruses
- Adenoviruses
- Respiratory syncytial viruses
- Human metapneumovirus
- Coronavirus
- Human bocaviruses
- Respiratory virus detected in 1/3 of community acquired pneumonia

Nosocomial Pneumonia
- Includes hospital acquired pneumonia
  - Pneumonia acquired ≥ 48 hours after hospital admission
- Includes ventilator associated pneumonia
  - Pneumonia acquired ≥ 48 hours after endotracheal intubation

Hospital Acquired Pneumonia
- Causative agents
  - Aerobic gram negative rods
    - Klebsiella sp.
    - Pseudomonas sp.
    - Enterobacter sp.
    - Escherichia coli.
    - Proteus sp
    - Serratia sp.
    - Enterococci.
  - Staphylococcus aureus (including methicillin-resistant Staphylococcus aureus [MRSA])
  - Group B streptococci
- Nosocomial pneumonia is typically caused by bacterial agents that are more resistant to antibiotic therapy.
- Sources
  - Contamination of pharynx and perhaps stomach with bacteria
  - Repeated small aspirations of oral pharyngeal secretions.
  - Retrograde contamination from GI tract.

Pneumonia Presentation
- Cough – with or without sputum
- Dyspnea
- Pleuritic chest pain
- Chills
- Fatigue
- Malaise
- Anorexia
- 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)
- Hypoxemia / central cyanosis
- Altered mental status
- S & S of sepsis

S&S can be subtle in older adult and in patients who are immunocompromised.

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
- Tactile fremitus
- Tracheal deviation
- Lymphadenopathy
- Pleural friction rub
Testing in Pneumonia

- Sputum gram stain
- Sputum culture
- Urinary antigen testing for *S. pneumoniae*
- Testing for *Legionella* spp
- Testing for respiratory viruses
- Bronchoscopy may be done
- Blood cultures (bacteremia not present in most)
- Leukocytosis / Shift to left of WBCs.
  - 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 (pus in pleural space)
  - 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**
- **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)
Criteria for Admission

**Hospital Admission**
- O₂ sat < 92% on room air
- PSI score > III risk class
  - Pneumonia severity index (PSI) score as a guide for inpatient care and mortality risk.
- CURB-65 score ≥ 3 or ≥ 2 if > 65 years of age

**ICU Admission**
- Respiratory failure requiring intubation
- Sepsis requiring vasopressor
- Other features suggesting severe pneumonia

Pneumonia Treatment

**Factors guiding choice of empiric antibiotic therapy**
- Severity of pneumonia
- Setting of treatment
- Comorbid conditions
- Age
- Recent hospitalization or antibiotic use
- Known colonization for suspicion for MRSA / Pseudomonas

**Antibiotic Duration**
- De-escalation if a pathogen becomes known
- Minimum 5-day treatment
  - Mild infection usually 5 to 7 days
  - Severe infection or comorbid 7 to 10 days
- Extended course for immunocompromised or certain pathogen
- Treat until afebrile and clinically stable x 48 hours
- Patient’s do not need to stay overnight when switched from IV to oral

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).
Lung Abscess / Abscess Pneumonia

- Subacute infection
- Area of necrosis in lung parenchyma
- Involves right lung more often, seen in dependent area of lung
- Often complication of aspiration of oropharyngeal secretions
  - Insidious onset 1 to 2 weeks after aspiration

Pleural Conditions

Pleural Effusion

- Increased pulmonary interstitial fluid along with increased capillary vascular permeability and/or decreased lymphatic drainage
- Transudates versus exudates
  - Transudative: manage the underlying medical disorder
    - Heart failure
    - Hypoalbuminemia
  - Exudative
    - Pneumonia
    - TB
    - Malignancy
    - If infected it is labeled as complicated parapneumonic effusion
    - Complicated effusions can lead to adhesions and loculated fluid collections

Pleural Conditions

Treatment Options in Pleural Effusion

- Therapeutic thoracentesis
- Tube thoracostomy
- Pleurodesis (pleural sclerosis)
  - Irritant is instilled into pleural space
    - Talc: Fever, chest pain, and nausea
    - Inflammation bridges fibrosis between visceral and parietal pleura and obliterates the pleural space
    - Pleural space has to be fully drained and lung fully re-expanded for success
    - Chest tubes clamped for 2 hours after installation
- Indwelling tunneled pleural catheters
  - Can be intermittently drained at home

Pleural Conditions

Empyema

- Pus in the pleural space
- Also called pyothorax or pureulent pleuritis
- Typically complication of pneumonia
- Also can be seen as complication of trauma, lung surgery, or after chest tube or thoracentesis

Pulmonary Fibrosis

- Form of interstitial lung disease which results in restrictive lung disease
- Irreversible lung scarring
- Causes:
  - Asbestos, grain dust, bird and animal droppings, radiation treatments, chemotherapy, amiodarone, some antibiotics (sulfasalazine), systemic lupus erythematosus, rheumatoid arthritis, sarcoidosis, and scleroderma
- Termined idiopathic when no known cause
- Natural course variable and unpredictable
- S&S
  - SOB, dry cough, fatigue, unintentional weight loss, muscle and joint aching
Pulmonary Fibrosis

- Two antifibrotic medications for idiopathic
  - Nintedanib
  - Pirfenidone
- Oxygen
- Pulmonary Rehab
- Lung transplant
  - Leading cause for lung transplant in US

Postoperative Nursing Care: Lung Resection

- Lung cancer is most common indication
- **Worst complication acute hypoxemia: ALI / ARDS** – leading cause of death
- Other complications
  - MI
  - PE
  - Arrhythmia
  - Pneumonia
  - Sepsis
- ECG and pulse oximetry monitoring
- Chest tube drainage q 1 hour until next AM
- Pain control

Restrictive Lung Disorders

- Myasthenia Gravis
  - Neuromuscular junction disorder
- Guillain-Barre
  - Acute polyneuropathy
- Muscular Dystrophy

ARDS

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
- 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
- Hyposic 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

**ARDS: Treatment**
- Optimal ventilation / oxygenation
- Avoid over hydration
- Corticosteroids in early (within 14 day onset) in moderate to severe disease
  - Methylprednisone 1mg/kg/day for onset up to 7 day
  - > 6 days onset 2mg/kg/day
  - Followed by slow taper up to 13 days
- Pulmonary vasodilators

**Non Cardiac Pulmonary Edema: ARDS**

**ARDS: Treatment**
- Optimal ventilation / oxygenation
- Avoid over hydration
- Corticosteroids in early (within 14 day onset) in moderate to severe disease
  - Methylprednisone 1mg/kg/day for onset up to 7 day
  - > 6 days onset 2mg/kg/day
  - Followed by slow taper up to 13 days
- Pulmonary vasodilators

**High mortality persists so prevention remains key**
Ventilator Strategies in ARDS

• Low tidal volume (4 to 8 ml / kg of predicted body weight) with permissive hypercapnea

• Maintain plateau pressure < 30 mm Hg

• Prone position > 12 hours per day in severe ARDS

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).

Additional Options in ARDS

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

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

With A Case Example

• 65 year old female; 85 kg
• Post witnessed cardiac arrest
• Initial PaO₂ / FiO₂ 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:
  - AC
  - Rate 12
  - TV 700 ml
  - FIO2 80%
  - PEEP 5 cm
- 2nd ABG
  - pH 7.33
  - PaCO2 40 mmHg
  - HCO3 14 mEq/L
  - PaO2 92 mmHg

Most probable anticipated ventilator changes include:

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

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

1. Controlled mandatory ventilation
2. SIMV
3. Airway pressure release ventilation
4. SBT

### Transfusion Related Acute Lung Injury (TRALI)

- Pulmonary syndrome after transfusion of almost any blood product plasma containing products
  - Fresh frozen plasma
  - Whole blood
  - Packed red blood cells
  - Platelet transfusions
  - Cryoprecipitate
- TRALI often occurs through transfusions from donors with anti-HLA or anti-granulocyte antibodies (89% of cases one or both of these antibody types)
- Seen in patients with underlying inflammation prior to transfusion
TRALI: New Proposed Definitions

• TRALI Type I (without an ARDS risk factor)

• TRALI Type II (with an ARDS risk factor or with mild existing ARDS)

• Clinical diagnosis and does not require detection of antibodies

TRALI Recognition

• Fever

• Dyspnea

• Hypoxemia / increased $O_2$ requirements

• Pink frothy secretions in ventilated patients

• Cyanosis

• Hypotension

Symptoms begin in 1 to 2 hours and fully manifest by hour 6

Can be fatal if not immediately recognized and treated

TRALI

• CXR: bilateral infiltrates: no cardiac compromise or fluid overload
  – Non cardiogenic
  – No diuretics indicated

• Pathophysiology is microvascular injury to pulmonary vasculature

• Wide range of mortality based on the patient’s pre TRALI condition

TRALI Treatment

• Stop transfusion

• Start oxygen and supportive care with CPAP / BiPAP

• However, 70% to 80% of patients will require intubation and mechanical ventilation
  – Most 3 to 10 days

• Notify blood center
  – Return remaining product for antibody testing

Transfusion-associated circulatory overload (TACO)

• Produces a cardiogenic pulmonary edema
  – Increased hydrostatic pressure

• Seen in patients with cardiac and renal disease or fluid overload state

PULMONARY TRAUMA
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 bronchoscopy 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

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)

  – Paradoxic 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

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 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.
Air Leaks Syndromes

- Alveoli can over distend and rupture high transpulmonary pressures
- Air leaks into interstitium
  - Remain local or spread
  - Rupture visceral pleura or pulmonary hila (point of attachment between the lung root and lung; consists of major bronchi and pulmonary arteries and veins)
- Trapped gas causes restriction
- Inflammatory response at site of rupture
- Atelectasis

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: Hemothorax

- Types / Etiology
  - Primary (no underlying lung disease)
    - Blebs / bullae
    - Smoking
  - Secondary (underlying lung disease)
    - Air enters through damaged alveoli
    - COPD
    - Blunt trauma (lung laceration by rib fracture)
    - Iatrogenic – from medical procedure
    - Positive pressure ventilation (rupture of weak alveoli, bleb or bullous)

- 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 watersal
    - No suction in spontaneous pneumo due to late presentation and risk of re-expansion pulmonary edema

Closed (Simple) Pneumothorax

- Spontaneous primary pneumothorax may take 12 weeks to resolve.

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

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

Tension Pneumothorax

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

- 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
**Hemothorax**

- **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

**Tension Pneumonia**

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

**Chest Tubes**

- **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 an 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.

- 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>Ventilation</th>
<th>Perfusion</th>
<th>Diffusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assessment</td>
<td></td>
<td>Oxygenation status</td>
</tr>
<tr>
<td>V/Q Scan</td>
<td>SpO₂ or SaO₂</td>
<td>Pao₂</td>
</tr>
<tr>
<td>Spiral CT</td>
<td>VO₂ Scan</td>
<td>Global: Decreased C.O.</td>
</tr>
<tr>
<td>S&amp;S associated with P.E.</td>
<td>Respiratory alkalosis</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase rate or Vt</td>
</tr>
<tr>
<td>Reverse sedation</td>
</tr>
<tr>
<td>Ambu bag</td>
</tr>
<tr>
<td>BiPAP</td>
</tr>
<tr>
<td>Intubate and ventilate</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
</tbody>
</table>

Pulling it All Together

- Neurological conditions (brain or spinal cord injury),
- Any condition that affects airway resistance or lung compliance
- COPD and Asthma: Increased airway resistance from obstruction / secretions
- Pneumonia: Increased airway resistance from secretions
- Pulmonary edema: Decreased lung compliance
- Pleural effusion: Decreased lung compliance

- Pulmonary Embolus
- Shock

- Any disorder that affects the alveolar capillary membrane
- Pneumonia: Exudate
- Pulmonary edema: Fluid

The Joy of Learning

Thank You

www.cardionursing.com