Complications of mechanical ventilation

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Objective

- Describing Categories of complications associated with mechanical ventilation process in general
- Describing complications occurring during mechanical ventilation in particular
- Describing specific pulmonary complications (VILI, VAP)
- How can we minimize complications?
Categories of Complications during MV process

I. Attributable to intubations
II. Associated with ET-Tracheostomy Tubes
III. Attributable to operation of the ventilator
IV. Occurring during assisted ventilation
I. Attributable to Intubations

- Prolonged intubation attempt
- Intubation of Right Main Stem Bronchus
- Premature or accidental Extubation
II. Associated with ETT or Tracheostomy tube

- Tube Malfunction (e.g. impacted secretions, Air Leak etc.)
- Nasal Necrosis
- Erosions and Bleeding
- Sinusitis
- Stenosis and Dilatation
- Tracheomalacia
III. Attributable to Operation Of the Ventilator

- Machine Failure
- Alarm Failure
- Alarm Found Off
- Inadequate humidification
### IV. Complications Occurring during Mechanical Ventilation

#### Spectrum of complications

<table>
<thead>
<tr>
<th>Pulmonary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Barotraumas / Volutrauma / ALI / VILI</td>
</tr>
<tr>
<td>Air Trapping or Intrinsic PEEP (PEEPi)</td>
</tr>
<tr>
<td>Air-leak syndrome</td>
</tr>
<tr>
<td>Infections {Ventilator-associated pneumonia (VAP)}</td>
</tr>
<tr>
<td>Patient-ventilator dysynchrony</td>
</tr>
<tr>
<td>Oxygen</td>
</tr>
<tr>
<td>Hypoxia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Systemic Cardiovascular &amp; Hemodynamic:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adverse pulmonary homodynamic effects</td>
</tr>
<tr>
<td>Hypotension</td>
</tr>
<tr>
<td>Cardiac arrest</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Airway</th>
</tr>
</thead>
<tbody>
<tr>
<td>Early</td>
</tr>
<tr>
<td>Late</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Other Systemic</th>
</tr>
</thead>
<tbody>
<tr>
<td>CNS</td>
</tr>
<tr>
<td>Muscles</td>
</tr>
<tr>
<td>Kidney: Increase ADH</td>
</tr>
<tr>
<td>GI: stress ulcer</td>
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</tbody>
</table>
Discussion of Specific Pulmonary complications

- Ventilator induced lung Injury (VILI)
- Intrinsic PEEP
- Ventilator associated pneumonia (VAP)
- \(O_2\) toxicity / Hypoxia
- Ventilator –patient dys-synchrony
- Airway complication (early and late)
- Systemic complication
Ventilator-Induced Lung Injury (VILI)

- **Definition**: lung damage caused by application of positive or negative pressure to the lung by mechanical ventilation
- **Etiology & Type**: Volutrauma, Barotrauma, atelectotrauma, Biotrauma, oxygen toxicity
- **Encompasses mostly**: Stretch Injury (Volutrauma, Barotrauma)
- **Also includes**: any ongoing ventilator related lung damaging pathology e.g. air trapping, atelectasis, inflammatory reaction etc.
Manifestation of VILI

**Volutrauma:**
- High volume leads to non-homogenous alveolar over-distension.

**Barotrauma:**
- High pressure induced lung damage and air-leak (PIE)

**Histological appearance:**
- High-permeability pulmonary edema
- Inflammatory reaction
- Hemorrhage
Ventilation-induced Lung Injury (VILI) or Ventilator associated lung injuries (VALI)

**Barotrauma: (high pressure)**
- Repetitive alveolar collapse and reopening of the under-recruited alveoli:
- Epithelial cell damage
- Leakage of air

**Volutrauma (high volume):**
- Over-distension alveoli due to excessive volume delivery:
  - Direct physical damage to alveolar capillary architecture
  - Cytokines releases, lung edema,
  - Surfactant damage,
  - Decrease lung compliance

*Dreyfuss: J Appl Physiol 1992*
Barotraumas

Presence of air in the extra-pulmonary space due to high transpulmonary pressure generated between the internal (alveolar) pressure and the external (intra-thoracic) pressure

Effects of High pressure mechanical ventilation on lungs

• Necessary method used to mechanically support people who have ALI

• If ventilation is used improperly, or is done for too long, it can lead to VILI

Fig. Normal Rat Lungs and Rat Lungs after Receiving High-Pressure Mechanical Ventilation at a Peak Airway Pressure of 45 cm of Water.
Volutrauma

Damage caused by the “delivered VT”, due to the mechanical stretch of the alveolar surface of the reduced number of open alveoli that receive the entire amount of VT leading to diffuse alveolar damage, pulmonary edema, increased fluid filtration, increased epithelial & microvascular permeability.

Effects of ventilation with large tidal volume on the lungs

**Fig.** H&E staining of mouse lungs with non-ventilation (Control) and ventilated with high tidal volume (HTV).
Effects of High pressure mechanical ventilation on lungs

• Necessary method used to mechanically support people who have ALI

• If ventilation is used improperly, or is done for too long, it can lead to VILI

Fig. Normal Rat Lungs and Rat Lungs after Receiving High-Pressure Mechanical Ventilation at a Peak Airway Pressure of 45 cm of Water.
Atelectrauma

Repeated Recruitment / De-recruitment (Opening / Closing) that is responsible for mechanical stress on the alveolar surface of both healthy and atelectatic alveoli due to repeated shear forces.

Manifestations Of Barotrauma / Volutrauma / High PEEP

- Pulmonary Interstitial Emphysema
- Pneumothorax
- Pnuemomediastinum
- Pnuemopericardium
- Subcutaneous Emphysema
- Increased Physiologic Dead Space
Volutrauma: Chest X-ray
Example of ventilator induced lung injuries

Pneumo-mediastinum
Multiple organ failure associated with mechanical ventilation
Graphics can illustrate problems and help adjusting the ventilator to prevent VILI.
Manifestation and predication of intrinsic (Auto) PEEP
Complications of intrinsic PEEP

- Impaired gas exchange
- Pulmonary overdistention (Volutrauma/Barotrauma)
- Elevated mean intra-thoracic pressure
- Air-leak syndrome
Other Possible Complications Associated With PEEP or CPAP

- Effect on Pulmonary Mechanics
- Decreased Cardiac Output
- Decreased Cerebral Perfusion Pressure
- Altered Renal Blood Flow &/or Function
- Decreased Splanchnic blood Flow
- Alveolar Overdistension
Strategies to Minimize VILI

- Lung protective ventilatory strategies
- Goals of MV in pts at risk of VILI: ventilate & oxygenate within “safe window”: avoiding
  - High FiO₂
  - Overdistension “more gentle V₀” “lung rest”.
  - De-recruitment (alveolar collapse) ‘open lung’
  - Alveolar collapse & overdistension,
  - Accept permissive hypercapnia,
Infection complications of mechanical ventilation

- Nosocomial Pnuemonia VAP
- Sinusitis
- Pharyngitis
- Tracheitis
**VAP**: Ventilator associated pneumonia

- Diagnostic criteria: fever, leukocytosis,
- New (progressive) infiltrates, purulent sputum
- New infiltrate (> 48 H after MV) with at least one of:
  - Histopathologic evidence of pneumonia
  - Positive blood or pleural fluid culture (matching TA)
  - New fever and leukocytosis
  - Purulent tracheal aspirates
Risk/Predisposing factors for VAP

- Organ-system failure $\geq 3$
- Extreme age
- Supine head position
- Immuno-suppression
- Renal failure
- Hepatic insufficiency
- Major CNS disease
Patient-ventilator asynchrony

- Fighting the ventilator, acute respiratory distress
- May indicate a potentially life-threatening problem
- Physical signs: anxiety, agitation, tachypnea, use of accessing muscles, thoracic cage-abdominal asynchrony, tachycardia, hypotension, arrhythmia
Patient-ventilator asynchrony

- Check Ventilator for malfunction:
- Remove patient from ventilator, manual ventilation + 100% O2
- Rapid assessment of vital signs (trends), physical exam (cardiopulmonary aspects)
- Assess airway patency, suction airway
Patient-ventilator asynchrony

- Airway obstruction: copious/thick secretions plugging ETT → aggressive suction, CPT, mucolytic agents
- Bronchospasm
- Pneumonia
- Pulmonary edema.
- Dynamic hyperinflation, auto-PEEP: air-trapping
Patient Ventilator Dys-Synchrony

![Graphs showing pressure over time with annotations indicating increased trigger work.]

- Increased Trigger Work
Oxygen Toxicity

- Generally agreed: $\text{FiO}_2 > 0.6$ is toxic.
- In addition to VILI; $\text{O}_2$ toxicity $\rightarrow$ systemic VC $\downarrow$, C.O, absorption atelectasis, hypoventilation.
- One of the first goals of ventilator management is to reduce $\text{FiO}_2 < 0.6$ to minimize further lung injury.
**Oxygen Toxicity**

• Onset varies from patient to patient depending on patient characteristics

• Contributing elements are:
  - Fraction (more specifically Partial Pressure) of Inspired Oxygen
  - Length of Exposure
  - Underlying Lung Disease
Oxygen Toxicity

- Important Patient Characteristics:
  - Age
  - Nutritional Status
  - Previous Exposure to Oxidants
  - Antioxidants on board
Hypoxia

Frequent complications in MV patients.

Low $\text{PaO}_2$, desaturation, needs for higher $\text{FiO}_2$.

Mechanisms:

- Hypoventilation
- diffusion abnormalities
- shunting V/Q mismatching
- low alveolar $[\text{O}_2]$. 
Possible causes of worsening oxygenation during MV

- Ventilator-related problems
  - Endotracheal or tracheostomy tubes
  - Ventilator circuit
  - Function of ventilator itself
  - Inappropriate ventilator settings
Possible causes of worsening oxygenation during MV

- Progression of underlying disease process
  - Acute respiratory distress syndrome
  - Cardiogenic pulmonary edema
  - Pneumonia; sepsis
  - Acute exacerbation of asthma or COPD
Possible causes of worsening oxygenation during MV

- Onset of new medical problem
  - Pneumothorax
  - Atelectasis (lobar collapse; diffuse microatelectasis)
  - Aspiration of gastric or oropharyngeal contents
  - Nosocomial pneumonia; sepsis
Possible causes of worsening oxygenation during MV

- Onset of new medical problem
- Pulmonary thromboembolism
- Fluid overload
- Bronchospasm
- Retained secretions
- Shock; any process causing a fall in cardiac output in the presence of a right-to-left intrapulmonary shunt
Airway Complications of MV

I. Complications of endotracheal intubation
   1. Early
   2. Late

II. Complications of tracheostomy
   1. Early
   2. Late
Complications Related to ETT

- Prolonged intubation attempt
- Esophageal intubation
- Right main stem intubation
- Self extubation
- Malfunction of the endotracheal tube
  - Partial dislodgement
  - Inappropriate endotracheal tube length
  - Cuff laceration
  - Endotracheal tube occlusion
  - Cuff leak
Early complications related to ETT

- Acute laryngotraacheal injury
  - Tooth avulsion
  - Laryngospasm
  - Edema of the vocal cords, glottis
  - Pharyngeal injury
  - Tracheal ulcers
  - Nose bleeds
  - Pain in the nose, mouth, chest

- Stridor and upper airways obstruction

- Early aspiration of gastric contents
Example ET Tubes Malfunction due to secretions
Late Complications Related to ETT

- Hoarseness
- Sore throat
- Paranasal sinusitis
- Cough, sputum, and hemoptysis
- Late laryngotracheal injury
  - Ring-shaped tracheitis
  - Granuloma
  - Tracheal stenosis
  - Tracheomalacia
- Late aspiration of gastric contents
Other Systemic complications

1. **CNS:**
   a. Hyperventilation $\rightarrow$ respiratory alkalosis $\rightarrow$ cerebral VC (cerebral ischemia): Bohr effect (hypoxia)
   b. Increased ITP $\rightarrow$ ↓ cerebral venous drainage $\rightarrow$ ↑CBV $\rightarrow$ ↑ICP
   c. Increased RR or short expiratory time $\rightarrow$ ↓ cerebral venous drainage
2. **Muscles:** disuse respiratory muscle hypotrophy (difficult weaning).

3. **Kidney:** MV + PEEP $\rightarrow$ increased ADH $\rightarrow$ fluid retention

4. **Psychiatric and emotional:** complications
Final word

- The science of mechanical ventilation is to optimize gas exchange
- The art is to achieve that without damaging the lung
Thank you