**Mechanical Ventilation & NIPPV**

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**Section I: Indications for the use of mechanical ventilation**

There are a number of physiologic and clinical indications for ventilation, which all of us are very familiar with. Dr. Marino suggests the following “Simple Rules” to decide who needs mechanical ventilation (MV):

**“Simple Rules” for the initiation of MV:**

**Rule 1:** The indication for intubation and mechanical ventilation is thinking of it

Do not delay: like most procedures, elective intubation is much safer than emergent intubation

**Rule 2:** Intubation is not an act of personal weakness.

Airway control in an unstable patient is an act of conviction and is better for the patient

**Rule 3:** Initiating mechanical ventilation is not the “kiss of death.”

Being on the vent does not create vent-dependence; severe illness does

**Characteristics of Intubated Patients**

There have been a number of studies looking at different disease states, to determine the incidence and indications for mechanical ventilation. A prospective 28-day international study in JAMA looked at unselected, heterogeneous, intubated patients, describing their characteristics and outcomes. The study examined 5,183 patients who received MV for > 12h.

**Why do we do it?**

1. Acute Respiratory Failure: 68.8% (Including post-op - 21%, Pneumonia, CHF, etc.)
2. Coma: 16.7%
3. Chronic Pulmonary Disease: 12.8% (COPD - 10%, Asthma, etc.)
4. Neuromuscular Disease: 1.8%

**How do we do it?**

1. Orotracheal tube: 89%
2. Nasotracheal tube: 4%
3. Face mask: 5% (17% of COPD patients). The incidence of this mode has been rising: As many as 35% of MV patients in Europe are not intubated.
4. Tracheostomy: 2%

**Indications for Non-Invasive Positive Pressure Ventilation (NIPPV)**
NIPPV is just another mode of MV. In the Emergency Department (ED), COPD and CHF exacerbations are the most relevant indications. A level of evidence classification for NIPPV has been proposed.\(^3\)

**Level 1 evidence**
- Systematic reviews (with homogeneity) of RCTs and individual RCTs (with narrow CIs)
  - COPD exacerbations
  - Facilitation of weaning/extubation in patients with COPD
  - Cardiogenic pulmonary edema
  - Immunosuppressed patients

**Level 2 evidence**
- Systematic reviews (with homogeneity) of cohort studies - individual cohort studies (including low quality RCTs)
  - Do-not-intubate status
  - End-stage patients as palliative measure
  - Extubation failure (COPD or congestive heart failure) (prevention)
  - Community-acquired pneumonia in COPD
  - Postoperative respiratory failure (prevention and treatment)
  - Prevention of acute respiratory failure in asthma

**Caution advised for**
- Severe community acquired pneumonia
- Extubation failure prevention

**Level 3 evidence**
- Systematic reviews (with homogeneity) of case–control studies, individual case-control study
  - Neuromuscular disease/kyphoscoliosis
  - Upper airway obstruction (partial)
  - Thoracic trauma
  - Treatment of acute respiratory failure in asthma

**Caution advised for**
- Severe acute respiratory syndrome

**Level 4**
- Case series (and poor quality cohort and case-control studies)
  - Very old age, older than age 75 years
  - Cystic fibrosis
  - Obesity hypoventilation

**Caution advised for**
- Idiopathic pulmonary fibrosis

**Common conditions encountered in the ED**
**COPD Exacerbation**

There is a very well established benefit of NIPPV, multiple RCTs support this use. Prevents further deterioration and intubation, improves survival.4

**Cardiogenic Pulmonary Edema**

Reduces intubation rate, improves physiologic variables. 5-8 Impact on mortality is less clear. One meta-analysis failed to detect any difference in mortality, though previously cited trials have demonstrated improvement. 7

**Asthma**

Use of NIPPV has been associated with reduced admissions and increased pulmonary flow rates. 9, 10

**Contraindications to NIPPV**

**Absolute Contraindications**
- Respiratory arrest
- Cannot fit mask (there are a variety available — full face, nose, mouth, helmet)

**Relative Contraindications**
- Clinically unstable
- Agitated and uncooperative – keeps removing mask
- Cannot protect airway
- Excessive secretions
- Recent upper airway or upper gastrointestinal surgery
Section II: How to troubleshoot unstable intubated patients

Mechanical ventilation may contribute to instability of patients, due to the unusual physiology of positive-pressure ventilation, or due to complications of mechanical ventilation. Early recognition and specific treatment of these complications will help reverse the instability. In the next section I will go over each complication, how to recognize and correct each, here I list some general principles of troubleshooting unstable intubated patients in the ED.

General Principles: ABCs

1. If intubated, check for tube blockage or displacement
2. Check BP, Pulse, SpO₂, RR, EtCO₂ if available
3. Disconnect patient from vent and bag with 100% oxygen. Bag synchronously with respiration.
4. Look, listen and feel for:
   - Air leaks around endotracheal tube (ETT) or face mask
   - Chest – look for equal expansion, breath sounds, subcutaneous emphysema
5. Obtain Chest X-Ray (CXR), Arterial Blood Gas analysis (ABG)

Using airway pressures to troubleshoot

The peak and plateau pressures can help provide clues to the sudden hemodynamic deterioration of your patient. Please use this flowchart along with the guide for specific complications (Section III) to determine the cause and treat.

<table>
<thead>
<tr>
<th>ACUTE RESPIRATORY DETERIORATION</th>
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<tbody>
<tr>
<td>PEAK INSPIRATORY PRESSURE</td>
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<tr>
<td>(Decreased)</td>
</tr>
<tr>
<td>• Air Leak</td>
</tr>
<tr>
<td>• Hyperventilation</td>
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<tr>
<td>PLATEAU PRESSURE</td>
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<tr>
<td>• Pulmonary Embolus</td>
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<tr>
<td>• Extrathoracic Process</td>
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<tr>
<th>AIRWAY OBSTRUCTION</th>
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<tbody>
<tr>
<td>• Aspiration</td>
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<tr>
<td>• Bronchospasm</td>
</tr>
<tr>
<td>• Secretions</td>
</tr>
<tr>
<td>• Tracheal Tube</td>
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<tr>
<td>• Obstruction</td>
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<table>
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<tr>
<th>DECREASED COMPLIANCE</th>
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<tr>
<td>• Abdominal Distension</td>
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<tr>
<td>• Asynchronous Breathing</td>
</tr>
<tr>
<td>• Alveolar Atelectasis</td>
</tr>
<tr>
<td>• Auto-PEEP</td>
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<tr>
<td>• Pneumothorax</td>
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<tr>
<td>• Pulmonary Edema</td>
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Section III: Evaluate for complications due to mechanical ventilation

Try to identify a specific complication of mechanical ventilation

Remember that agitation may have a unique contribution to instability of MV patients. For example, In Assist-Control ventilation, every time the ventilator is triggered, a full breath is delivered. A patient who’s neural center is triggering rapid shallow breathing will be forced to significantly hyperventilate, and will also be at risk for ventilator induced lung injury. After eliminating all vent complications and other causes of agitation, sedation should always be administered to intubated patients. In certain situations paralysis may also be appropriate.
Complications Related to Airway

An ETT itself causes a number of problems. Some of the complications listed below also apply to NIPPV, but the latter is potentially safer.

Tube migration

With head flexion/extension, there can be 2 cm movement of the ETT in and out of the trachea. Side to side movement of neck, placement in Trendelenburg, all cause movement of ETT in trachea that may lead to esophageal intubation or main bronchial intubation.

The ideal tube length has been estimated using formulae based on age, height, etc. The straight distance between the upper incisors and manubrio-sternal junction in the fully extended position has been proposed as a more accurate estimation.

How to evaluate

Signs of tube migration depend on whether the tube has migrated out (into the esophagus) or in (into a main stem bronchus).

Evaluation for Esophageal Intubation

Hypoxia, air hunger (increased respiratory effort). Vent alarms for low exhaled (return) volume and change in the waveform display.

Breath sounds are known to be unreliable in confirming tube placement. Auscultation of the epigastric area combined with axillae has been shown to be 100% reliable. Water vapor in the tube has been shown to be 100% sensitive but not specific.

EtCO₂ monitoring (either quantitative or qualitative) is the currently recommended standard to confirm ETT placement. This is known to be unreliable in arrest patients where there is insufficient pulmonary blood flow, and if there is a large amount of CO₂ in the stomach (due to carbonated beverages, bag-mask ventilation).

Direct visualization of tube going through cords is ideal, but such visualization may not be possible. Similarly fiberoptic laryngoscopy may not be available; visualization of tracheal rings is quite helpful. A CXR is also a very useful test, the lateral view being superior to the AP view.

In general use of a combination of methods with high clinical suspicion is probably the most appropriate strategy.

Evaluation for Main Bronchus Intubation

First signal for this would be increased peak and plateau airway pressures, because the vent is now trying to deliver a large amount of air into half the volume it was set for.

Bilateral auscultation is recommended to evaluate for main bronchus intubation.

The current ETT depth should be compared with the depth recorded at initial intubation.

Fiberoptic laryngoscopy and CXR are definitive tests for detection.

How to treat

Deflate cuff, reposition tube
In case of main bronchus intubation, gradual withdrawal of the tube while listening for breath sounds is a useful solution. A CXR should always be done after repositioning the tube.

**Trauma**

Insertion of the ETT or the presence of an ETT in the upper airway can cause local trauma to a number of structures in the area. NIPPV is not immune to such trauma, as pressure points on the face can cause necrosis and bleeding.\(^1\)

It is recommended that the ETT cuff pressure should not exceed 25 mm Hg (35 cm H\(_2\)O). Higher pressure can lead to esophageal fistulae and erosion into the innominate artery. Early tracheostomy has been shown to mitigate many of these complications and is recommended for many intubated patients in the ICU.

**How to evaluate**

Watch for obvious bleeding, or blood on suctioning around tube

**How to treat**

Direct pressure/surgical intervention as required

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**Tube Blockage**

Blockage of the airway (either the endotracheal tube, or the natural airway) is an important complication seen in MV patients. Routine suctioning is a standard practice in all MV patients, especially those who have an impaired gag reflex.

**How to evaluate**

Ventilator alarms: peak inflation pressure very high, low airflow. Plateau pressure is often not increased, which helps differentiate this from conditions like pneumothorax

Disconnect from ventilator and bag: difficult or unable

Try to suction ETT – may encounter obstruction when passing catheter

CXR may be necessary

**How to treat**

Suctioning through inline suction and around ETT

Saline instillation not helpful (biofilm is hydrophobic, saline just pushes bacteria down into lungs)\(^1\)

Instillation of NAC directly into the trachea may be useful (or aerosolized NAC, avoid in asthmatics)\(^1\)

Replace ETT if necessary

Fiberoptic or rigid bronchoscopy may be required in recalcitrant cases
Air Leak

An air leak implies that the inflation air from the vent is escaping into the atmosphere. This can lead to hypoventilation, increased work of breathing and hypoxia, defeating the purpose of mechanical intubation in the first place.

How to evaluate

Audible sound on each inhalation/exhalation
Can feel air flowing around the tube/mask
Significant difference in inhaled and exhaled volumes on vent
Peak pressure decreased on vent
Check all vent tubing for leaks and loose connections

How to treat

For ETT, check if the cuff is still up or has ruptured (indicator balloon is present near the inflation valve)
Ensure ETT still in trachea (see tube migration section above)
When ETT position has been confirmed, add more volume to the cuff. Make sure cuff pressures do not exceed 35 cm H₂O.
Replace ETT if necessary
For NIPPV, adjust fit of face mask, consider alternative interface such as nasal, full face, helmet, etc.

Aspiration

Cuff inflation for tracheal seal does not protect against aspiration. Aspiration may contribute significantly to development of ventilator associated pneumonia.¹⁶ There has been a lot of activity lately regarding oral decontamination for MV patients in the ICU.

Complications Related to Ventilation

Hemodynamic Effects

Positive airway pressures are transmitted to other intrathoracic structures such as the great vessels, heart, pulmonary capillaries, etc. MV therefore has a number of effects on hemodynamics that can be exacerbated in unstable patients with minimal cardiac reserve. Additionally many parameters used as goals of resuscitation may be affected by MV, for example PEEP can cause spurious increases in cardiac filling pressures (such as CVP).

PEEP directly affects cardiac compliance, decreases right ventricular filling and increases right ventricular volume overload by increasing pulmonary vascular resistance. The overall effect of MV is decreased cardiac output.

How to evaluate

Decreased cardiac output manifested by hypotension, tachycardia, decreased SvO₂, increasing lactate.

How to treat

Fluids, vasopressors as required (CVP may be falsely elevated)
Temporary reduction in PEEP
Check for intrinsic PEEP and adjust vent as required
Use PEEP only if necessary

**Alveolar Rupture**

Alveolar rupture is the final complication of Ventilator Induced Lung Injury (VILI), discussed below. This can present as pneumothorax, pneumomediastinum or pneumoperitoneum. The incidence rises with higher inflation pressures and volumes or with use of PEEP. The extent of damage to the lungs from underlying disease also effects the development of VILI.

**How to evaluate**

Clinical exam is unreliable in diagnosis of alveolar rupture; there may be no findings until the air accumulation is causing direct pressure effects. Subcutaneous emphysema is pathognomonic of alveolar rupture.

CXR is diagnostic. In pneumothoraces, basilar and subpulmonic collections of air on CXR are characteristic in the supine position, typically air does not accumulate in the apices. CT scanning may be required to make the diagnosis.

**How to treat**

Attempt to reduce airway pressures

Tube thoracostomy is the definitive treatment for pneumothorax. Generally a 2 cm water seal is sufficient, suction is not necessary and may make things worse by contributing to development of a bronchopleural fistula.

**Increased Work of Breathing**

Ideally, the period of mechanical inflation much match the period of neural inspiratory time, and the period of mechanical inactivity (passive deflation) must match neural expiratory time. If not, we may actually increase work of breathing by placing the patient on vent.

PEEP, whether intrinsic or extrinsic, can also increase work of breathing. To trigger the vent, a patient must create a negative pressure in the lungs that exceeds the PEEP. Therefore if there is PEEP of 5 cm H$_2$O and a breath is triggered at -2 cm H$_2$O, the patient must generate a pressure of -7 cm H$_2$O to trigger the vent. Additionally PEEP places the lungs on a flatter section of the volume-pressure curve, so higher pressures are needed to deliver the same tidal volume.

**How to evaluate**

Failure to achieve the goals of intubation – persistence of hypoxia, increased respiratory rate, patient agitation and obvious discomfort

Rising lactate may indicate respiratory muscle overuse

**How to treat**

Proper selection of vent settings is key

Recommended settings: The ARDSnet has a recommended protocol *(see reference for URL).*

1. Calculate Predicted Body Weight (PBW)
   - Males: 50 + [2.3 × (height in inches - 60)]
   - Females: 45.5 + [2.3 × (height in inches - 60)]
2. Select any ventilator mode (Assist-Control most common)
3. Set tidal volume (TV) to 8 ml/kg PBW
4. Set initial respiratory rate (RR) to match baseline ventilation, but not > 35 breaths/min
5. Set FiO₂ to 100%, PEEP to 5 cm H₂O
6. Every 1-2h:
   - Reduce TV by 1 ml/kg, until 6 ml/kg PBW
   - Reduce FiO₂ for goal SpO₂ 88-95% or PaO₂ 55-80 mm Hg
   - Adjust RR, TV to achieve pH 7.30 – 7.45, and plateau pressure ≤ 30 cm H₂O
   - Check for intrinsic PEEP, treat if necessary (see below)

**Intrinsic PEEP**

Incomplete deflation of alveoli during exhalation leads to a pressure gradient between the alveoli and the atmosphere at end-expiration. This creates persistent airflow at the end of expiration, a phenomenon called *intrinsic PEEP, auto PEEP or dynamic hyperinflation*.

Intrinsic PEEP is more common when there is a relative decrease in exhalation time compared to inspiratory time, such as with rapid breathing, high inflation volumes or airway obstruction (in Asthma, COPD).

Hemodynamic effects mirror those of “extrinsic” PEEP, with overall decreased cardiac output that may become life threatening. Intrinsic PEEP also predisposes to alveolar rupture and increased work of breathing.

**How to evaluate**

- End-expiratory occlusion is most popular method
- Presence of airflow at end of expiration is sensitive but not specific.²¹
- Most accurate method to quantify is intraesophageal or intrapleural pressure monitoring.

**How to treat**

- Optimize time allowed for exhalation: decrease respiratory rate, tidal volume. This runs the risk of hypoventilation therefore ABG monitoring is key.
- Modification of inspiratory flow time or I:E ratio can also help decrease PEEP

**Ventilator Induced Lung Injury**

Often does not manifest in the ED, however choice of optimal setting will minimize development of the same later. Lung compliance varies in different areas of the lung, so application of a single pressure at the trachea leads to different pressures and volumes delivered to different regions of the lung.

**Mechanisms of Injury**

- **Volutrauma:** Overexpansion of certain regions of the lung
- **Barotrauma:** Injury to alveoli and airways caused by increased pressure
- **Atelectrauma:** Repeated opening and closing of alveoli that causes damage to their epithelial lining
- **Biotrauma:** Release of inflammatory mediators, and inactivation of surfactant triggered by large alveolar surface area oscillations. These mediators may spread beyond the lung and cause multi-organ system failure.
How to evaluate

May have a variety of presentations, from alveolar rupture to multi organ dysfunction

How to treat

Lung protective ventilation strategies as suggested by the ARDSnet (described earlier) are efficacious. Further management is mostly done in the ICU

Complications that Can Develop in Ventilated Patients

- Pneumonia, decubiti, stress ulcers, etc.
- NIPPV has lower risk of infections (including UTI, line sepsis), shorter LOS, less mortality.
  - Additionally NIPPV decreases need for sedation, which is an independent risk factor for weaning failure.

References