Mechanical ventilation: the basics

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Module “Respiration”
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Mechanical ventilation: the basics

- Physiology and physiopathology
- Modalities
- Indications
- Ventilation strategies
- Adverse events during mechanical ventilation
- Weaning
Mechanical ventilation: the basics

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Physiology and pathophysiology

- Positive pressure ventilation
- Pulmonary barotrauma
- Ventilation induced lung injury
- Diaphragm disuse atrophy
- Heart-lung interactions during positive pressure ventilation
- Dynamic hyperinflation
Positive pressure ventilation

- Improves gas exchange by improving ventilation-perfusion (V/Q) matching (primarily because of decreased physiologic shunting)
- Decreases work of breathing, allowing the ventilatory muscles to recover from their fatigue
- Positive pressure ventilation increases intracranial pressure (ICP) because of impaired cerebral venous outflow.
Pulmonary barotrauma

- Direct consequence of positive pressure ventilation
- Alveolar rupture due to elevated transalveolar pressure (the alveolar pressure minus the pressure in the adjacent interstitial space)
- Prevention: Plateau pressure $< 35$ cmH$_2$O
Air from torn alveolus first enters perivascular interstitium (A), dissecting proximally within the bronchovascular sheath toward the mediastinum (B). As airway pressure rises, decompression occurs into cervical (C), subcutaneous (D), and pericardial (E) tissue spaces. Pleural rupture may result in pneumothorax (F). Small arrows indicate the direction of air movement.

Redrawn from Maunder RJ, Pierson DJ, Hudson LD, Arch Intern Med 1984; 144:1449.
Ventilator associated lung injury (1)

- Ventilator-induced lung injury (VILI) if it can be proven that the mechanical ventilation caused the acute lung injury.
- Ventilator-associated lung injury (VALI) if a causative relationship cannot be proven.
- More frequent in the course of ALI/ARDS
Ventilator associated lung injury (2)

- Due to alveolar overdistension (volotrauma) and cyclic actelectasis (shear forces)
Rapid Disuse Atrophy of Diaphragm Fibers in Mechanically Ventilated Humans


The NEW ENGLAND JOURNAL of MEDICINE

![Image of diaphragm fiber size comparison](image)

![Image of slow and fast myosin heavy chain](image)

![Bar chart of fiber cross-sectional area](image)
Patient triggered ventilation

• Assisted mechanical ventilation
• Avoid ventilator induced diaphragmatic dysfunction
• Providing sufficient level of ventilatory support to reduce patient’s work of breathing
Right ventricle and MV 😞

- Venous return impaired (gradient between IVC and RA ⬡)
- After load of RV ⬆
- Especially when pre-existing RV failure (COPD, pulmonary embolism…)
- Hemodynamic failure or cardiac arrest after initiation of mechanical ventilation
Effect of MV on venous return
Left ventricle and MV 😊

- Preload of LV ↘
- After load of LV ↘
- Favors transudation of alveolar fluid back to the capillaries
- Perfect treatment of cardiogenic pulmonary edema
Dynamic hyperinflation (1)

- Positive airway pressure at the end of expiration due to incomplete exhalation
- Causes:
  - High minute volume due to patient factors or ventilator settings
  - Prolonged inspiratory time
  - Time-constant inequality (COPD)
  - Expiratory flow resistance
  - Expiratory flow limitation and altered respiratory system compliance
Dynamic hyperinflation (2)

• Consequences:
  – exacerbates the hemodynamic effects of positive pressure ventilation
  – increases the risk of pulmonary barotrauma
  – makes it more difficult for the patient to trigger a ventilator-assisted breath

• Management:
  – Changes in ventilator setting
  – Reduce ventilatory demand
  – Reduce expiratory flow resistance
  – Application of external PEEP
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Modalities

- Volume oriented modes
- Pressure oriented modes
- Trigger
- Cycling
- FiO2
- Positive end-expiratory pressure
- Invasive versus non invasive
Confusion 😞

- CMV: Controlled Mechanical Ventilation or Continuous Mandatory Ventilation
- Pressure support or pressure assist?
- PS-PEEP Horus 4 Taema: back-up rate, PSV iVent Versa Med: none
- ACMV mode iVent Versa Med: Pressure limited, ACMV mode Horus 4 Taema: no
- No agreement between manufacturers on mode designation (marketing)
2 main modalities 😊

**Volume oriented:**

→ Variable parameter = inspiratory airway pressure

**Pressure oriented:**

→ Variable parameter = tidal volume
Volume oriented modes

- Inspiratory flow is preset
- Inspiratory time determines the Vt
- The variable parameter is the airway peak and plateau pressure
Pressures in volume oriented ventilation

- Mean airway pressure: pressure applied to the lung and the chest wall averaged across both phases of ventilation
- Peak airway pressure: total pressure needed to create the tidal volume
- Plateau pressure = Peak airway pressure – resistive component (pressure applied to small airway and alveoli)
- Transpulmonary pressure = plateau pressure – pleural pressure
Equation of insufflated gases in flow assist control ventilation

- Describes interactions between the patient and the ventilator
- Pressure required to deliver a volume of gas in the lungs is determined by elastic and resistive properties of the lung

\[ \text{Paw} = \frac{\text{Vt}}{\text{C}} + \text{VR} + \text{PEP} \]
Peak Airway Pressure

\[ \text{Paw} = \text{Po} + \frac{\text{Vt}}{C} + \text{RV} \]

\[ C = \frac{\text{Vt}}{\Delta P} \quad \text{and} \quad \Delta P = \text{P Plat} - \text{PEEP} \]
Measuring plateau pressure

- apply an inspiratory breath hold (usually 0.5 to 1 second) to the ventilated patient and measure the airway pressure during the breath hold

- In a relaxed patient

- Essential to prevent barotrauma and choose the best tidal volume in ARDS patients.
Measuring auto-peep

Apply an expiratory breath hold (usually 0.5 to 1 second) and then directly measure the airway pressure during the breath hold.
Pressure oriented modes

- Pressure in airway is the preset parameter
- Flow is adjusted at every moment to reach the preset pressure
- The variable parameter is Vt
Equation of motion in pressure support ventilation

\[ \text{Paw} = \text{Passist} + \text{Pmus} = \frac{V_t}{C} + V_x R + \text{PEP} \]

- Pressure applied by the ventilator on the airway + pressure generated by respiratory muscles

- Pmus is determined by respiratory drive and respiratory muscle strength
Tidal volume

• Predicted Body Weight
  – males:
    • $50 + 2.3 \text{ (height in inches} - 60)$
    • $50 + 0.91 \text{ (Height in cm} - 152.4)$
  – females:
    • $45.5 + 2.3 \text{ (Height in inches} - 60)$
    • $45.5 + 0.91 \text{ (Height in cm} - 152.4)$
• Tidal volume: 8-10 ml/kg if normal compliance, 6 ml/kg in ARDS
Flow rate and shape

- Physiological proto-inspiratory peak flow
- Match patient’s demand
- Decelerating flow more physiological
- But no data supporting one shape more than the other
- Determines inspiratory time indirectly
- Plateau
Trigger

Pressure Trigger

Flow Trigger
How to set the trigger?

- As low as possible
- If too low
  - Autotrigerring
  - RR too high
- If too high
  - Patient has difficulty to trigger the cycle
  - Increased work of breathing
Cycling

- Time-cycled breath
- Pressure-cycled breath
- Flow-cycled breath
<table>
<thead>
<tr>
<th>Ventilator</th>
<th>Flow Cycle</th>
<th>Pressure Cycle</th>
<th>Time Cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puritan-Bennett 7200</td>
<td>5 L/min</td>
<td>PEEP + pressure support + 1.5 cm H₂O</td>
<td>3 s</td>
</tr>
<tr>
<td>Puritan Bennett 840</td>
<td>Adjustable (1–80% of peak flow)</td>
<td>PEEP + pressure support + 1.5 cm H₂O</td>
<td>3 s</td>
</tr>
<tr>
<td>Puritan-Bennett 740/760</td>
<td>10 L/min or 25% of peak flow</td>
<td>PEEP + pressure support + 3 cm H₂O</td>
<td>3.5 s</td>
</tr>
<tr>
<td>Servo 900C</td>
<td>25% of peak flow</td>
<td>PEEP + pressure support + 3 cm H₂O</td>
<td>80% of set cycle time</td>
</tr>
<tr>
<td>Servo 300</td>
<td>5% of peak flow</td>
<td>PEEP + pressure support + 20 cm H₂O</td>
<td>80% of set cycle time</td>
</tr>
<tr>
<td>Servoi</td>
<td>Adjustable (1–40% of peak flow)</td>
<td>High-pressure limit</td>
<td>≤ 2.5 s, based on flow-cycle setting*</td>
</tr>
<tr>
<td>Dräger Evita 4</td>
<td>25% of peak flow</td>
<td>High-pressure limit</td>
<td>4 s</td>
</tr>
<tr>
<td>Bear 1000</td>
<td>25% of peak flow</td>
<td>High-pressure limit</td>
<td>5 s</td>
</tr>
<tr>
<td>Hamilton Veolar</td>
<td>25% of peak flow</td>
<td>High-pressure limit</td>
<td>3 s</td>
</tr>
<tr>
<td>Hamilton Galileo</td>
<td>Adjustable (10–40% of peak flow)</td>
<td>High-pressure limit</td>
<td>3 s</td>
</tr>
<tr>
<td>Intrasonics Star</td>
<td>4 L/min</td>
<td>PEEP + pressure support + 3 cm H₂O</td>
<td>3.5 s</td>
</tr>
<tr>
<td>Bird 8400 and TBird</td>
<td>25% of peak flow</td>
<td>High pressure limit</td>
<td>3 s</td>
</tr>
<tr>
<td>Pulmonetic LTV</td>
<td>Adjustable (10–40% of peak flow)</td>
<td>High-pressure limit</td>
<td>Adjustable (1–3 s)</td>
</tr>
<tr>
<td>Viasys Avea</td>
<td>Adjustable (5–45% of peak flow)</td>
<td>High-pressure limit</td>
<td>Adjustable (0.2–5.0 s)</td>
</tr>
<tr>
<td>Newport E500</td>
<td>Variable, based on time constant and pressure above pressure support setting</td>
<td>High-pressure limit</td>
<td>3 s</td>
</tr>
</tbody>
</table>
PEEP

- Positive end expiratory pressure
- Direct therapeutic effect in case of atelectasis / cardiogenic pulmonary edema
- Give always minimal PEEP 3 to 5 cm H2O (prevention of injury associated with cyclic actelectasis)
- Effects of PEEP on alveolar recruitment: best PEEP in ARDS ???
- No PEEP if severe acute asthma or pneumothorax
**FiO2**

- Initially FiO2=100%
- Do ABGs with FiO2=100%
- Then titrate downward for SpO2 between 92 and 94% and a PaO2 between 55 and 60 mmHg
- Avoid when possible high FiO2 (>60%) related with oxygen toxicity especially at the early phase of ARDS.
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Indications for invasive positive pressure ventilation

- Decreased mental status
- Hypercapnic respiratory failure
- Hypoxemic respiratory failure
- Intubation to facilitate a procedure
- Ethical issues
Non-invasive ventilation

- Time consuming
- Requires committed medical team
- Discomfort of the mask
- Leaks
- Gastric distension

BUT

- Easy weaning
- Allows feeding and talk between sessions
- No sedation
- Less nosocomial infection
- Reduced mortality
- No tracheal injury, no intubation
When is it appropriate to use NIV?

- ARF / any chronic obstructive or restrictive lung diseases
- ARF / hypercapnia
- ARF with moderate hypoxemia (?)
- Weaning of invasive ventilation in COPD patients
- Cardiogenic pulmonary edema without myocardial ischemia
- Patient refusing intubation
When isn’t it appropriate to initiate NIV?

- Major ventilatory impairment: respiratory arrest and pauses, apnoea,…
- Neurologic impairment: extreme agitation, coma not due to hypercapnia
- Severe cardiovascular impairment: cardiac arrest, severe ventricular arrhythmia, bradycardia, hypotension
- Others (relative): Pulmonary edema with myocardial ischemia, non cooperating patients or severe hypercapnic enkephalopathy, trauma or surgery (face, ENT, esophagus, stomach…)
- Inability to clear secretions
How to initiate NIV?

- Explain the procedure to the patient
- Patient seated
- Limit dead space (humidifier, connections)
- Pressure support + PEEP, FiO2 for SpO2 = 90-92%
- Facial mask (try various shapes and size). Beware of the adaptation of the mask and avoid injury to the face.
- Start with 10 cm H2O of pressure support and PEP<5 cm H2O, then set according to leaks, Vt (>5ml/kg), RR
- Check arterial blood gases after 30 mn
- Do not miss the appropriate timing for intubation in case of failure, especially in hypoxemic patients
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Ventilation strategies

• Impaired neurological status
• Adult respiratory distress syndrome
• Obstructive lung diseases
• Heart failure
ARDS

- P Plat < 30cm H2O
- Protective lung ventilation
- Vt 6-8ml/kg
- Reduce up to 4ml/kg to target P Plat
- Lung recruitment: titration of PEEP and FiO2 (hemodynamic tolerance)
- Oxygen toxicity, auto-peep
- Infections
Severe acute asthma

- Low tidal volume
- Low respiratory rate
- High expiration time
- Permissive hypercapnia
- No PEEP
- Beware of occult auto-peep
- Plateau pressure below 30 cm H2O
- Sedation ± muscle relaxant
- Hydration
- Nebulization
Mechanical ventilation: the basics

• Physiology and physiopathology
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Adverse events

• Respiratory distress in a patient on mechanical ventilation
• Hemodynamic failure
• Patient ventilator asynchrony
• Ventilator acquired pneumonia
• Tracheal stenosis and tracheo-esophageal fistula
Respiratory distress in a patient on mechanical ventilation

- Focused history and physical
- Check gas exchange
- Check respiratory mechanics
- CXR

Is the patient stable?

- Likely problem with ventilator or settings. Change settings to match effort and observe closely. Consider ventilator change if discomfort continues.

Disconnect from ventilator. Manual ventilation with 100 percent oxygen. Distress resolves?

- Consider empiric treatment of life-threatening problems:
  - Tension pneumothorax
  - Auto-PEEP

Continued deterioration?

Assess manual ventilations. Difficult to ventilate?

- Check for air leak. Replace endotracheal tube if necessary

- Attempt to pass suction catheter. Difficult to pass?

Trial of sedation or paralysis. Recheck manual ventilations.

- Reassess need for intubation, consider extubation if gas exchange is adequate and distress does not resolve

No

No

Yes

Yes

Yes

Yes
Effect of high PEEP on RV

- In ARDS increase in mPAP while increasing tidal volume and PEEP
- Management: optimisation of ventilator settings (compromise), fluid challenge to ensure adequate preload, pressors and inotropes
Patient-ventilator asynchrony: a challenge for the intensivist

- Discomfort anxiety
- Increased work of breathing
- Increased requirement of sedation
- Increased length of mechanical ventilation
- Increased incidence of VAP
Patient-ventilator asynchrony

- Mechanical ventilation: 2 pumps
  - Ventilator controlled by the physician
  - Patient’s own respiratory muscle pump

- Mismatch between the patient and the ventilator inspiratory and expiratory time

- Patient « fighting » with the ventilator
Ventilation phases
Trigger asynchrony

• **Insensitive trigger**: triggering that requires excessive patient effort
• **Ineffective triggerring**: muscular effort without ventilator trigger
• **Double triggerring**
• **Auto-triggerring**
Ineffective triggering

![Graph showing ineffective triggering with arrows indicating timing issues.](image)
Double triggering

- Cough
- Sighs
- Inadequate flow delivery
Auto-triggering

- Circuit leak
- Water in the circuit
- Cardiac oscillations
- Nebulizer treatments
- Negative suction applied through chest tube
Flow asynchrony

- Fixed flow pattern (volume oriented)
- Variable flow pattern (pressure oriented)
Volume oriented ventilation
(fixed flow pattern)

• Inspiratory flow demand varies according to the underlying condition
• If patient’s flow demand increases, peak flow should be adjusted accordingly
• Usually, peak flow is too low
• Dished-out appearance of the pressure-wave-form
• Importance of flow-pattern
- Ineffective triggering at 30 l/min
- Increase in flow rate
- Subsequent increase of expiratory time
- Decreased dynamic hyperinflation
- Subsequent decrease in ineffective triggering
Pressure oriented ventilation
(variable flow)

- Peak flow is depending on:
  - Set target pressure
  - Patient effort
  - Respiratory system compliance

- Adjustment: rate of valve opening = rise
time = pressure slope = flow acceleration
Termination asynchrony

• Ventilator should cycle at the end of the neural inspiration time

• Delayed termination:
  – Dynamic hyperinflation
  – Trigger delay
  – Ineffective triggering

• Premature termination
Set inspiratory time < 1 sec
Expiratory asynchrony

- Shortened expiratory time:
  - Auto-PEEP → trigger asynchrony
    - Delay in the relaxation of the expiratory muscle activity prior to the next mechanical inspiration
    - Overlap between expiratory and inspiratory muscle activity
- Prolonged expiratory time
Reduction of patient-ventilator asynchrony by reducing tidal volume during pressure-support ventilation
• International Nosocomial Infection Control Consortium

• 2002 to 2005

• 55 ICUs 46 hospitals

• 8 countries: Argentina, Brazil, Colombia, India, Peru, Mexico, Morocco, Turkey

• Prospective by infection control professionals
**Table 5.** Comparison of Device Use and Rates of Device-Associated Infection in the Intensive Care Units of the International Nosocomial Infection Control Consortium and of the U.S. National Nosocomial Infection Surveillance System*

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rate of device use†</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mechanical ventilators</td>
<td>0.43 (0.23–0.62)</td>
<td>0.38 (0.19–0.64)</td>
</tr>
<tr>
<td>CV/Cs</td>
<td>0.57 (0.36–0.74)</td>
<td>0.54 (0.22–0.97)</td>
</tr>
<tr>
<td>Urinary catheters</td>
<td>0.78 (0.65–0.90)</td>
<td>0.73 (0.48–0.94)</td>
</tr>
<tr>
<td><strong>Rate per 1000 device days†</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventilator-associated pneumonia</td>
<td><strong>5.4 (1.2–7.2)</strong></td>
<td><strong>24.1 (10.0–52.7)</strong></td>
</tr>
<tr>
<td>CVC-associated bloodstream infection</td>
<td>4.6 (1.7–7.6)</td>
<td>12.5 (7.8–16.9)</td>
</tr>
<tr>
<td>Catheter-associated UTI</td>
<td>3.9 (1.3–7.5)</td>
<td>8.9 (1.7–12.8)</td>
</tr>
<tr>
<td><strong>Proportion of device associated infections with resistance, %‡</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MRSA</td>
<td>59</td>
<td>84</td>
</tr>
<tr>
<td>Ceftriaxone-resistant <em>Enterobacteriaceae</em></td>
<td>19</td>
<td>55</td>
</tr>
<tr>
<td>Ciprofloxacin-resistant <em>Pseudomonas aeruginosa</em></td>
<td>29</td>
<td>59</td>
</tr>
<tr>
<td>Vancomycin-resistant enterococci</td>
<td>29</td>
<td>5</td>
</tr>
</tbody>
</table>

* Data are from an NNIS report (1). CVC = central venous catheter; ICU = intensive care unit; INICC = International Nosocomial Infection Control Consortium; MRSA = methicillin-resistant *Staphylococcus aureus*; NNIS = National Nosocomial Infection Surveillance System; UTI = urinary tract infection. † Overall (pooled) and 10th to 90th percentile range for U.S. NNIS teaching hospitals; overall (pooled) and range of individual countries for the INICC hospitals. ‡ Overall (pooled) data from NNIS, 1992–2004 (300 hospitals), and from INICC, 2002–2005.
VAP in developing countries

<table>
<thead>
<tr>
<th>Variable</th>
<th>Country</th>
<th>Overall</th>
</tr>
</thead>
<tbody>
<tr>
<td>VAP associated pneumonia, n</td>
<td>A 234</td>
<td>B 136</td>
</tr>
<tr>
<td>Rate per 100 patients (range)†</td>
<td>3.2 (0.0-9.0)</td>
<td>13.1 (0.0-16.5)</td>
</tr>
<tr>
<td>Rate per 1000 ventilator days (range)†</td>
<td>30.1 (0.0-51.4)</td>
<td>21.2 (0.0-22.1)</td>
</tr>
<tr>
<td>Proportion of cases, %§</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Enterobacteriaceae</td>
<td>44</td>
<td>15</td>
</tr>
<tr>
<td>Pseudomonas aeruginosa</td>
<td>8</td>
<td>36</td>
</tr>
<tr>
<td>Acinetobacter spp.</td>
<td>15</td>
<td>28</td>
</tr>
<tr>
<td>Staphylococcus aureus</td>
<td>32</td>
<td>14</td>
</tr>
<tr>
<td>Enterococci</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Coagulase-negative staphylococci</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Candida spp.</td>
<td>0</td>
<td>5</td>
</tr>
<tr>
<td>Susceptibility of resistant microorganisms, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MRSA</td>
<td>83</td>
<td>93</td>
</tr>
<tr>
<td>Ceftriaxone-resistant Enterobacteriaceae</td>
<td>52</td>
<td>94</td>
</tr>
<tr>
<td>Fluoroquinolone-resistant P. aeruginosa</td>
<td>50</td>
<td>67</td>
</tr>
<tr>
<td>Vancomycin-resistant enterococci</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

*MRSA = methicillin-resistant Staphylococcus aureus
†Ranges for individual countries are for the individual hospitals; overall ranges are for the individual countries.
‡Range not given because only 1 participating hospital was from country F.
§Partial listing of major pathogens; does not total 100%.
Ventilator-Associated Pneumonia: Diagnosis, Treatment, and Prevention

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FIG. 3. Potential strategies to prevent VAP.
Tracheal complications (1)

• Prevention !!!
• High volume low pressure cuffs for endotracheal and tracheostomy tubes
Tracheal complications (2)

• Avoid over inflation of cuffs (monitoring cuff pressure if manometer available)
Tracheal complications (3)

- Remove ETT / NG tubes ASAP !!!
- Deflate cuff of tracheostomy tubes when airway protection / mechanical ventilation no more necessary
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Thank you!