MECHANICAL VENTILATION – KEEP IT SIMPLE

S. Dial
Rounds 2020
“...an opening must be attempted in the trunk of the trachea, into which a tube of reed or cone should be put; you will then blow into this, so that the lung may rise again...and the heart becomes strong...”

Vesalius 1555
Fun Facts

- Vesalius credited with the first positive pressure ventilation 1555

- Iron Lung
  - invented by Phillip Drinker and Louis Agassiz Shaw
  - Pumps that increase/decrease air pressure
  - Negative pressure ventilation
  - Treatment of coal gas poisoning
  - First used in 1928 at Children’s Hospital, Boston

- Emerson company prototype in late 1950s
  - Positive-pressure ventilation
  - Became famous in mid-1900s during polio outbreak
Ventilation

- Brain
- Nerves
- Rib cage
- Respiratory muscles

- Ventilators can replace the function of these parts of the respiratory system
The functional lung unit

Deoxygenated blood from pulmonary artery

Air

Alveolus

capillary
Gas exchange

- Mixed venous
- Arterial blood
- Alveolus
- Inspired air
- Expired air
- Rate of exchange determined by metabolic rate

\[ CO_2 \]

\[ O_2 \]
# Oxygen Inhaled Therapy

<table>
<thead>
<tr>
<th>Device</th>
<th>Reservoir Capacity</th>
<th>Oxygen Flow (L/min)</th>
<th>Approximate ($\text{FiO}_2$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nasal Cannula</td>
<td>50mL</td>
<td>1</td>
<td>0.21–0.24</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>0.24–0.28</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3</td>
<td>0.28–0.34</td>
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<tr>
<td></td>
<td></td>
<td>4</td>
<td>0.34–0.38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5</td>
<td>0.38–0.42</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6</td>
<td>0.42–0.46</td>
</tr>
<tr>
<td>Face Mask</td>
<td>150–250mL</td>
<td>5–10</td>
<td>0.40–0.60</td>
</tr>
<tr>
<td>Mask–Reservoir bag</td>
<td>750–1250mL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Partial Rebreather</td>
<td></td>
<td>5–7</td>
<td>0.35–0.75</td>
</tr>
<tr>
<td>NonRebreather</td>
<td></td>
<td>5–10</td>
<td>0.40–1.00</td>
</tr>
</tbody>
</table>
High flow nasal cannula

- Humidification improves tolerance of high flows
- Nasal cannula- better tolerated than full face mask – patients can speak and eat
- The high flows delivered results in less dilution by room air in patients with high flows
- Systems – Vapotherm, AIRVO, Ventilator, Optiflo
AIRVO HFNC

AIRVO2
(P10 1AZ/ P10 1UK)

HEATER PLATE

FINGER GUARD

AUTO-FILL WATER CHAMBER (MR290)
(with adapter fitted)

HEATED BREATHING TUBE CONNECTION PORT

MEASUREMENT POINT OF DISPLAYED DEW POINT TEMPERATURE

CHAMBER PORTS

Water chamber

Patient interface

Heated breathing tube

English

OXYGEN INLET PORT

HEATER PLATE

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English
Physiologic mechanisms of action of HFNC

- Washout of nasopharyngeal dead space – the expired air is replaced by a fresh source of air with a lower pCO$_2$ and higher FIO$_2$
- Decreases the resistance of the upper airway on inspiration – prevents potential narrowing related to negative intra-luminal pressure
- Warm humidified air improves conductance and pulmonary compliance
- Humidification of air through the nasopharynx uses energy – therefore saves energy
Physiologic mechanisms

- Provides PEEP which is dependent on the flow
- Drying out of the mucosa can cause damage which interferes with mucociliary clearance, this increases the risk of infections and atelectasis – the humidification of HFNC is thought to decrease these risks

Fig. 3. Pressure profiles from one subject at increasing gas flows with the Optiflow nasal high flow oxygen system.

Mouth opening only decreases PEEP effect on expiration
Not as bad as you think, nebulizer – clean particles, not patient
What they did in China
Other ways to give high $O_2$

- You can add a filter to the end of this
- Filter
CPAP
What does CPAP do

■ Increase FRC

■ Bigger lung = bigger surface area for gas exchange
  - *Recruit partially collapsed alveoli*

■ Improve compliance
CPAP effect on compliance

The initial part of the curve shows the substantial pressure needed to inflate alveoli from the collapsed state. Once initially inflated, the lungs then become very compliant until the chest wall approaches its limit of expansion, thus reducing compliance markedly.
Ventilation

- Brain
- Nerves
- Rib cage
- Respiratory muscles

Ventilators can replace the function of these parts of the respiratory system
Positive pressure
Compliance and Resistance

\[ R = \frac{\Delta P}{\Delta F} \]

\[ dP = R \times \text{Flow} \]

\[ C_s = \frac{\Delta V}{\Delta P} \]

\[ dP = \frac{dV}{C_{st}} \]
Resistive and Elastive Forces

**Dynamic Characteristics:**
\[ dP = \frac{dV}{Cdyn} \]

**Resistance:**
\[ dP_{\text{resist.}} = R \times \text{Flow} \]

**Static Compliance:**
\[ dP_{\text{dist.}} = \frac{dV}{Cst} \]

\[ dP = dP_{\text{resist.}} + dP_{\text{dist.}} \]

\[ dP = R \times \text{Flow} + \frac{dV}{Cst} \]
Plateau pressure measurements
Plateau pressure

distending pressure → Heavy chest wall, ascites
Positive pressure and PEEP

PEEP = 10

0 PEEP
What do you order

- RR - Respiratory rate = 12 – 24
- Vt or Tidal volume = 4 – 8 ml /kg
- PEEP – 5 – 24? Average on COVID = 10
- This is a recipe that has been used to which I have some reservations – treatment arm outcome was no different than controls

Table 2. Allowable PEEP Ranges at Specified Levels of \( \text{FiO}_2 \)

<table>
<thead>
<tr>
<th>Fraction of Inspired Oxygen (( \text{FiO}_2 ))</th>
<th>0.3</th>
<th>0.4</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
<th>0.8</th>
<th>0.9</th>
<th>1.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control PEEP ranges, cm H(_2)O</td>
<td>5</td>
<td>5-8</td>
<td>8-10</td>
<td>10</td>
<td>10-14</td>
<td>14</td>
<td>14-18</td>
<td>18-24</td>
</tr>
<tr>
<td>Lung open ventilation PEEP ranges, cm H(_2)O</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Before protocol change</td>
<td>5-10</td>
<td>10-14</td>
<td>14-20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20-24</td>
</tr>
<tr>
<td>After protocol change</td>
<td>5-10</td>
<td>10-18</td>
<td>18-20</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>20-22</td>
<td>22</td>
</tr>
</tbody>
</table>
VILI

- Volutrauma or baro – likely combination of both
  - Physiology – think of upper and lower inflexion points
Some terminology

- How does the ventilator when the patient is inspiring = trigger
- Tidal volume ?= you tell it how much to give or you provide a pressure and the Vt can vary (Pressure support of pressure control)
- How fast is the tidal volume delivered = flow
- How does machine know when to stop delivering the breath = cycle
Invasive Ventilatory Support

Volume-controlled ventilation
Invasive Ventilatory Support

- Pressure-controlled ventilation
  - Airway pressure and inspiratory time are controlled
  - Dependent variable are flow and volume
  - Decreases volu-trauma
  - Better alveolar recruitment
  - Can result in hypoventilation in the presence of auto-PEEP
Invasive Ventilatory Support

- Pressure Support Ventilation

![Diagram](Image)
Hypercarnobia

- Clinically elevations in the pCO2 are more likely to be a consequence of decreases in the alveolar ventilation than increased production.
- Alveolar ventilation ($V_A$) = Total minute ventilation – dead space ventilation.
- Dead space = non perfused alveoli – larger airways.
- $V_E = \text{frequency of respiration (breaths/minute)} \times \text{tidal volume (Vt)}$.
- Decreases in alveolar ventilation can occur either from a decrease in the total minute ventilation or an increase in the dead space ventilation or both.
Alveolar ventilation

- $V_A = f (V_t - V_d)$
- Effect of breathing pattern
  - $12 \times (600-150) = 5400\text{ml/min}$
  - $24 \times (300 - 150) = 4200\text{ml/min}$
- Effect of machine added dead space
  - $12 \times (600 - (150anat + 100apparatus)) = 4200\text{ml/min}$
Relation between minute ventilation and pCO$_2$

At higher pCO$_2$ values, smaller changes in alveolar ventilation result in large increases in pCO$_2$
Prone positioning
Proning – PROSEVA trial - ARDS

- You turn the patient on their stomach and leave them for 12-16 hours of the day
- There are videos on how to do it – NEJM