Introduction to
MECHANICAL VENTILATION

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Disclosures
• No disclosures

House Rules:
1. Please take your cell phones out, and text IDCCM to 37607 or go to PollEv.com/idccm
2. Open dialogue, feel free to interrupt and ask questions

Objectives
At the end of today's session you will:
• Develop an approach to managing respiratory failure
• Review concepts in initiation and management of patients on mechanical ventilation
• Review evidence behind mechanical ventilation and management of ARDS

RESPIRATORY FAILURE
Many approaches to Respiratory Failure
• Organ/System
• Clinical
• Physiological

TWO MAIN TYPES
• Hypoxemic
• Hypercapneic

Case 1
• 35F pedestrian was found in a back alley following a hit and run. The paramedics arrive to the scene. She is unconscious, and appears to have a lower extremity crush injury. Her initial vitals show:
• GCS 3, HR 130bpm, BP 70/40, O₂Sats 80% on room air

Normal Gas Exchange

Physiologic Causes of Hypoxemia

V/Q mismatch exists as a spectrum between Dead Space and Shunt
Physiologic Causes of Hypoxemia

1. V/Q mismatch
2. Shunt
3. Hypoventilation
4. Diffusion abnormality
5. Low inspired FiO2

How does hypoventilation cause hypoxemia

- Alveolar space is made of both partial pressure of O2 and CO2
- If one goes up, the other goes down
- If PaCO2 ↑, there is “less” space inside the alveolus for O2
- The lower the amount of PAO2, the less is available for gas exchange

Case 1 revisited...

- 35F pedestrian was found in a back alley following a hit and run. Her initial vitals show:
  - GCS 3, HR 130bpm, BP 70/40, O2 Sat 80% on room air
- Why is she hypoxic?
Common causes Respiratory Failure?

- Pneumonia
- ARDS
- Aspiration
- Heart failure/pulmonary edema
- Multi-trauma
- Pulmonary embolism
- Drug/toxic metabolites

Most common physiological cause of hypoxemia?

<table>
<thead>
<tr>
<th>V/Q mismatch</th>
<th>Shunt</th>
<th>Dead Space</th>
<th>Hypo-ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumonia</td>
<td>+++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspiration</td>
<td>+++</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>ARDS</td>
<td>+++</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td>CHF</td>
<td>+++</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PE</td>
<td>++</td>
<td></td>
<td>++</td>
</tr>
<tr>
<td>Drugs/Toxic</td>
<td>+++</td>
<td></td>
<td>+++</td>
</tr>
</tbody>
</table>

Hypercapneic Respiratory Failure

- CO₂ is a common form of respiratory waste by the tissue
- CO₂ – tightly controlled in the body
- PaCO₂ = \( k \times \frac{CO₂ \text{ production}}{CO₂ \text{ removal}} \)

\[ V_A = k \times V_{CO₂} \]

\[ V_A = \text{alveolar ventilation} = V_i \times (1 - V_D/V_T) \]

Causes of hypercapnea

1. Hypoventilation
   - Central CNS
   - Low RR
   - Shallow breathing
   - Drug/toxic metabolites

2. V/Q mismatch
   - COPD
   - Asthma

3. Muscle and chest wall
   - Nerve
   - Muscle
   - Kyphoscoliosis

4. Increased CO₂ production
   - Metabolism
   - Septic/Pot.
   - Hyperthyroid
   - Drug overdose
   - Re-breathing
   - Tachypnea

Case 1 revisited...

- 35F pedestrian was found in a back alley following a hit and run. Her initial vitals show:
  - GCS 3, HR 130bpm, BP 70/40, O₂Sats 80% on room air
- Hypoxemic likely secondary to:
  - Chest trauma, lung contusions, ARDS, shock....
What will you do next?

Oxygen Therapy

• What’s available?

Oxygen therapy

Low-flow systems

• Provide supplemental O₂ without a guaranteed FiO₂
• Flows >2LPM – nasal irritations
  – Add humidity and heat \(\rightarrow\) go up to 5-6LPM
  – Switch to a simple mask
• How much FiO₂ do you get with low flow systems?

High-Flow Systems – Venturi Masks

• Provides a fixed FiO₂
• 100% from the wall
• The smaller the O₂ orifice, the higher the flow going through
• Entrainst room air as it passes by

Venturi Masks

<table>
<thead>
<tr>
<th>Color</th>
<th>FiO₂</th>
<th>Wall Flow (LPM)</th>
<th>Total Flow (LPM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue</td>
<td>24%</td>
<td>2-4</td>
<td>51-108</td>
</tr>
<tr>
<td>yellow</td>
<td>38%</td>
<td>6</td>
<td>66</td>
</tr>
<tr>
<td>white</td>
<td>30%</td>
<td>8</td>
<td>72</td>
</tr>
<tr>
<td>green</td>
<td>35%</td>
<td>12</td>
<td>72</td>
</tr>
<tr>
<td>pink</td>
<td>40%</td>
<td>15</td>
<td>60</td>
</tr>
<tr>
<td>orange</td>
<td>50%</td>
<td>15</td>
<td>40</td>
</tr>
</tbody>
</table>
High-Flow Nasal Cannula

- Flows between 10-60 liters
- heated, humidified
- can deliver up to 100%
- maximum flow of 60 LPM via nasal prongs or cannula.
- FiO2 may be guaranteed if patient does not breathe through the mouth

Reservoir Systems

- Mechanisms that gather and store oxygen
- Additional source of O₂, in case patient’s minute ventilation exceed that of the device

Case 1 revisited...

- 35F pedestrian was found in a back alley following a hit and run. Her initial vitals show:
  - GCS 3, HR 130bpm, BP 70/40, O₂ Sats 85% on 100% non-re-breather
- What next?

Indications for Intubation and Mechanical Ventilation

- Airway Protection
  - Altered LOC
  - Absent reflexes
  - Pulm. Toilet
- Respiratory Failure
  - Oxygenation (Type I)
  - Ventilation (Type II)
- Work of Breathing
  - High drive and VO₂ by resp. muscles

Case continues:

She is intubated in the field and given a 2L bolus of NS en route to the hospital. On arrival she is being bagged by EMS on 100%. Her vitals: O₂ Sat 92%, BP 80/60, HR 135bpm, T 35°C. Her breaths seem rapid and shallow

The TTL is barking orders and getting ready for a scan and assessment. As you get dressed the RT looks at you and says "Do you want me to put them on VC or PC?"
What is Mechanical Ventilation?
- Positive pressure ventilation
- Partially or completely replace SPONTANEOUS breathing
- Recognizes the patient is trying to take a breath, then pushes air into the central airways, downstream into the alveoli

What are the TWO BROAD Categories of Mechanical Ventilation?
- Non-invasive Ventilation
  - aka Bi-Level Ventilation
  - Spontaneous (patient triggered) mode
  - Delivered through a mask or helmet
- Invasive Ventilation
  - Delivered through an endotracheal tube
  - Can be spontaneous (patient driven) or controlled (machine driven)

How do you start someone on mechanical ventilation?
- Trigger – What initiates the breath (negative flow or pressure, time)
- Limit or Target – what stops the gas from flowing (flow, or pressure)
- Cycling – when to go from inspiration to expiration (time, flow, volume, pressure)

Phase Variables
**Fraction of Inspired Oxygen (FiO<sub>2</sub>)**
- Concentration of oxygen in inspired gas (Set between 0.21 and 1.0)
- Goals:
  - Use the lowest possible FiO<sub>2</sub> necessary to meet your oxygenation goals (PaO<sub>2</sub>)
  - Reduces the risk of any potential adverse reactions from supplemental oxygen
    - Absorption atelectasis, worsening hypercapnea, airway and parenchymal lung injury

**Tidal Volume (V<sub>T</sub>)**
- Volume of gas that is inhaled and exhaled, measured in mL
- V<sub>T</sub> is made up of:
  - Dead Space Ventilation ~ V<sub>D</sub>
  - Alveolar Ventilation ~ V<sub>A</sub>
  - Almost always 6mL/kg or less
  - Depends on several patient and lung factors (disease state, lung compliance)

**Respiratory Rate (RR or f<sub>tot</sub>)**
- Number of breaths per minute that the ventilator delivers
  - No ideal number is established
  - Most patients start on RR 10-20/minute
  - Patients in respiratory distress may breathe more and the RR will be higher

**PEEP**
- Positive end-expiratory pressure
- Amount of pressure maintained at the end of a breath (cmH<sub>2</sub>O)
  - Applied to maintain open alveoli
  - Generally >5cmH<sub>2</sub>O
  - In normal people - this is represented by your RV

**PEEP - How does it work?**
- Physiological Benefits
  - Improvement in Gas Exchange
  - Improvement in V/Q mismatch
  - Increased Minute Ventilation
- Decreased the W.O.B
  - Reduced auto-peep
  - EPAP balances the auto-peep
  - PSV/IPAP augments your tidal volume

**Minute Ventilation (V<sub>E</sub>)**
- The average volume of gas entering or leaving the lungs in 1 minute (in L/min)
  - V<sub>E</sub> = V<sub>T</sub> x RR
  - Normal V<sub>E</sub> = 5-10L/min
  - This is closely related to CO<sub>2</sub> clearance by the lungs
    - In normal lungs – increasing the V<sub>T</sub> will decrease PaCO<sub>2</sub>
    - In diseased states, an increase in VE is usually due to increased RR, without effective change in V<sub>T</sub>
Flow Rate (V)
- The highest flow or speed by which a tidal breath is delivered during inspiration
  - Peak flow rates of 60L/min are usually sufficient
  - When flow is insufficient, patients are dyspneic, and the vent waveforms change shape
  - The faster the flow, the faster the gas is delivered, and the shorter the inspiratory time

Inspiratory:Expiratory Time (I:E ratio)
- The ratio or amount of time spent in each phase of breathing
  - Normal is 1:2, or 1s for inspiration
  - If the ratio is increased, meaning more I-time, there may not be enough time for expiration
    - Residual gas is trapped within the alveoli
    - breath stacking, gas trapping, or auto-PEEP
    - CO₂ clearance or ventilation requires enough E-time

Pressure vs. Volume based
Depends on the lung compliance

INVASIVE VENTILATION

Pressure Support (PSV)
- Partial assistance from the ventilator
- Preset level of inspiratory support (Pressure)
- Variable RR, flow, and Vₜ
- Cycles from inspiration to expiration based on a preset level (<25% peak flow)

Pressure Control (PC)
- Set RR and Insp. Pressure
- Vₜ is variable, aim for <6mL/kg
- If the patient takes a breath, they get the set pressure level
- Guarantees a pressure limit
- Ppeak <35cmH2O
Volume/Assist Control (VC/AC)

- Set RR and V̇
- Insp. Pressure is variable, aim for Pplat <30cmH₂O
- If patient triggers a breath, they get the set V̇
- Guarantees V̇̇ because there is a guaranteed V̇

Advantages and Disadvantages

<table>
<thead>
<tr>
<th>Mode</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td>Controlled Ventilation vs. Spontaneous</td>
<td>Reduced work of breathing</td>
<td>Hypotension, hyperventilation</td>
</tr>
<tr>
<td>Volume Control Ventilation</td>
<td>Guarantees a set tidal volume</td>
<td>May cause increases in pressure and barotrauma, hyperventilation</td>
</tr>
<tr>
<td>Pressure Control Ventilation</td>
<td>Limits to pressure reduces risk of barotrauma</td>
<td>Hyperventilation or hypopertilation if lung compliance/resistance changes</td>
</tr>
<tr>
<td>Pressure Support Ventilation</td>
<td>Patient comfort, improved ventilator interaction</td>
<td>Not tolerated by all patients, back up rate only if apneic</td>
</tr>
<tr>
<td>SIMV</td>
<td>Guarantees set number of breaths, less alkalois</td>
<td>Increased work of breathing when trying to initiate spontaneous breaths</td>
</tr>
</tbody>
</table>

Case repeated:

She is intubated at the scene and given a 2L bolus of NS en route to the hospital. On arrival she is being bagged by EMS on 100%. Her vitals: O₂Sat 92%, BP 80/60, HR 135bpm, T 35°C. Her breaths seem rapid and shallow.

The TTL is barking orders and getting ready for the Pan-Scan. As you get dressed the RT looks at you and says “Do you want me to put them on VC or PC, I’m setting the FIQ2 at 1.0 and the PEEP at 5 for now. Is that OK?”

With complete and total confidence you tell the RT...

You tell the RT...

1. Start on Volume Control with PEEP at 10, and set a tidal volume of 6mL/kg.
2. Start on Pressure Control with a PEEP at 10, and aim for Ppeak <40cm H₂O
3. Use SIMV with a back up rate of 15
4. Put them on Pressure Support

Case continues....

- The patient had bilateral chest tubes inserted in ER for pneumothoraces. Subsequently, compartment syndrome is identified and she is taken to the OR for urgent fasciotomy and ongoing resuscitation. She is now being transported back to the ICU for management. She is sedated and paralyzed prior to transport and bagged en route.
- On arrival to ICU, the RT’s are busy initiating MV. You ask “How do you know what to set the vent at?”
Initiating Mechanical Ventilation

- **Goal is to maintain adequate gas exchange**
- Start on a controlled mode of ventilation, either pressure or volume targeted
- In PCV
  - Set PEEP between 5-10
  - Aim for a $V_T$ of 6-8mL/kg
  - Start at $FiO_2$ 1.0 and adjust down later
  - Get a gas and titrate

Improving Oxygenation

- Increase $FiO_2$
- Recruit Lung
  - Increase $P_{inflation}$
  - Increase $I_{time}$
  - Increase PEEP
- Decrease $VO_2$
  - Sedate, NMBA
- Rescue therapy
  - Drugs, Prone position, ECLS

Improving Ventilation

- Increase $V_E$
  - Increase $V_T$ and/or RR
- Reduce $CO_2$ production/asynchrony – sedation/analgesia ± NMBA
  - Treat fever, seizures, etc.
- Prone Positioning to improve V/Q matching
- Rescue therapies
  - HFJV, ECMO

Lower your expectations!

- Target Reasonable Goals, balance iatrogenic harm (VILI)
  - $SpO_2$ >88% or $PaO_2$ >50-55 mmHg
  - $pH$ >7.25
- Depends on what else is going on:
  - Brain Injury
  - Pregnancy....

When starting a patient on the vent you...

A. Pressure-targeted mode of ventilation with ZEEP ($PEEP = 0$)
B. SIMV with a back up rate of 18
C. **Controlled mode of ventilation (either pressure or volume)**
D. Don’t know, I just do what the RT tells me to do
Back to the Case...

• It is ICU day 4 and the patient is still intubated. You notice over the course of the day that she is becoming more hypoxic. She has had her fasciotomy repaired despite being grossly edematous, and she is on fentanyl for pain control.
• Presently, she is on PCV 20/5, FiO2 70%, her VT is ~600ml or 11ml/kg (Current weight is 85kg; IBW=55kg). Her PaO2 is 55, RR is 35, BP 95/60, HR 108bpm.

Which of the following is false

A. She may have ARDS, in which case lowering her VT to ~350ml will improve outcome
B. She may have ARDS, in which case increasing her PEEP will improve outcome
C. She may have ARDS in which case keeping her Pplat < 30cmH2O would improve outcome
D. She may have ARDS and using inhaled NO will improve her outcome

ARDS: The Berlin Definition

Acute Respiratory Distress Syndrome

<table>
<thead>
<tr>
<th>Timing</th>
<th>Within 1 week of a known clinical insult of new/worsening respiratory symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest Imaging *</td>
<td>Bilateral opacities – not fully explained by effusions, lobar/lung collapse, or nodules</td>
</tr>
<tr>
<td>Origin of Edema *</td>
<td>Respiratory failure not fully explained by cardiac failure or fluid overload; Need objective assessment (e.g., echocardiography) to exclude hydrostatic edema if no risk factor present</td>
</tr>
</tbody>
</table>

Mild: PaO2/FiO2 ≤ 300 with PEEP or CMF ≥ 5cmH2O
Moderate: PaO2/FiO2 ≤ 200 with PEEP ≥ 5 cmH2O
Severe: PaO2/FiO2 ≤ 100 with PEEP ≥ 5 cmH2O

* Chest x-ray or CT scan
* If altitude >1000 m, correction should be made: PaO2/FiO2 = x (barometric pressure/760)

Management

• Treat the inciting/underlying cause
• No effective pharmacologic treatments
  – Ketoconazole, lisofylline, antioxidants, exogenous surfactant
  – Inhaled NO improves oxygenation, but increased risk of renal failure, no change in mortality
• Supportive care with mechanical ventilation remains the mainstay of treatment
  – Mechanical ventilation itself, may potentiate existing lung injury
**Primum non nocere...**

- **Principles of mechanical ventilation in ARDS**
  - Restore "normal" physiology
  - Adjust tidal volume, airway pressure, FiO$_2$, minute ventilation to achieve "normal" blood gases

- **Ventilator-associated lung injury (VALI)**
  - Gross barotrauma (e.g., pneumothorax)
  - Uncommon, easy to detect and treat (e.g., chest tube drainage)

- **Ventilator-associated lung injury (VALI)**
  - Barotrauma
  - Volutrauma
  - Atelectrauma
  - Biotrauma

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**Lung Protective Ventilation**

- "Normal" Lung, aka Baby Lung
- Abnormal lung, consolidation, edema, "cellular debris, dependant lung zones

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**ARDSNet Protocol - Goals**

- **Plateau Pressure Goal**
  - Keep $P_{plat} \leq 30$ cmH$_2$O

- **Tidal Volume Goal**
  - 6 vs. 12 mL/kg predicted body weight (PBW)

- **Physiological Goals**
  - $\text{PaO}_2$ 55-80 mmHg or $\text{SpO}_2$ 88-95%
  - PEEP/FiO$_2$ titrated according to predefined table
  - pH 7.30-7.45
**ARMA Trial Results**

- Mortality = 31%, vs 40% → ARR 9%

**Consequences of Low VT**

- While low tidal volume ventilation for lung protection may avoid alveolar over-distention (volutrauma), this strategy may lead to derecruitment and atelectrauma
  - Measures to counterbalance derecruitment are becoming increasingly important

**Alveolar Recruitment**

- Dynamic process of reopening collapsed alveoli
  - Pan-inspiratory phenomenon
  - Ventral to dorsal, cephalad to caudal
  - Dependent on transpulmonary pressure

**Rationale for Alveolar Recruitment**

- Recruitment (e.g., increased end-expiratory lung volume) may lead to:
  - Improved oxygenation and decreased shunt
  - Reduced pulmonary edema
  - Protection from VALI (atelectrauma and resultant biotrauma)

**Role of PEEP**

- End-expiratory and end-inspiratory collapse are not independent phenomena
  - PEEP maintains alveoli open that have been recruited by the previous inspiratory pressure, provided PEEP > superimposed pressure

**PEEP – Pro/Con**

- **Pro**
  - Less atelectrauma
  - Increased functional residual capacity
  - Improved oxygenation
  - Decreased shunt
- **Con**
  - Reduction in cardiac output
  - Volutrauma
  - Barotrauma

**Villar J. Minerva Anesthesiol 2006;72:357-362.**
The Problem with ARDS

“Baby Lung”:
- Risk of Overdistention
- High Dead Space
- Pulmonary Vasoconstriction
- Area with V/Q mismatch
- Low Compliance

Consolidated Lung:
- Increased Shunt
- Large area of atelectasis
- Low Compliance

Beyond Lung Protective Ventilation


Proning – why it works

- Recruitment of atelectatic lung
- Reduced abdominal pressure onto diaphragm
- Reduced cardiac weight
- Mobilization of secretions
- Overall: improve V/Q matching, decrease shunt

Back to the Case...

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C. She may have ARDS in which case keeping her $P_{plat} < 30cmH_2O$ would improve outcome
D. She may have ARDS and using inhaled NO will improve her outcome
Caring for patients with COVID-19

- Evidence is rapidly evolving
- Supportive Care is still the standard
- PROTECT YOURSELF:
  - Appropriate PPE for what you are doing

What PPE to wear?

| PPE for all in hospital activities | PPE Droplet Contact | PPE for Protected Code Blue or Aerosol Generating Procedures |

“TWO” Phenotypes

<table>
<thead>
<tr>
<th>L-TYPE</th>
<th>H-TYPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low recruitability</td>
<td>High recruitability</td>
</tr>
<tr>
<td>Low Lung compliance</td>
<td>High lung compliance</td>
</tr>
<tr>
<td>PEEP may not be helpful</td>
<td>PEEP is helpful</td>
</tr>
<tr>
<td>Pulmonary vasoconstriction and Dead Space high</td>
<td>?? iNO</td>
</tr>
<tr>
<td>?? NO</td>
<td>Paralysis/Sedation</td>
</tr>
<tr>
<td>Paralysis/Sedation</td>
<td>PRONING</td>
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</tbody>
</table>

Summary

- There are several ways to approach respiratory failure, clinical, anatomical, and physiological
- The most common physiological cause of hypercapneic and hypoxemic failure is V/Q mismatch
- Mechanical Ventilation can help support a patient in respiratory failure

Summary

- Acutely ill patients often require a controlled mode of ventilation that is *either* volume or pressure targeted
- Lung-protective ventilation improves outcomes in mechanically ventilated patients
- Sedation/Paralysis and Proning are best for those with moderate – severe ARDS

Basics of Mechanical Ventilation

Free online course
April 14 - 17, 2020 from 6:00 pm - 7:30 pm each day

We are happy to announce that we are offering a free online course for “Basics of Mechanical Ventilation” April 14-17, 2020 from 6:00 pm - 7:30 pm each day.

There will be two lectures per day with Questions & Answers.

This course will be presented by:
Interdepartmental Division of Critical Care Medicine (IXCCM)
Critical Care Canada Forum (CCCF)
Centre of Excellence in Mechanical Ventilation (CEMV)
Registration is open to all.

To register kindly RSVP by CLICKING HERE.

https://www.surveymonkey.com/r/H35DSR5
QUESTIONS?
Christie.Lee@sinaihealthsystem.ca