Management of Acute Respiratory Failure

Las Vegas, Nevada
March 15, 2016
No Disclosures

Aaron M Cheng, MD FACS
University of Washington
Division of Cardiothoracic Surgery
Seattle, Washington
Acute Respiratory Failure: Mechanical Ventilator Support

- Partial MV Support
- Full Mechanical Ventilator Support
- Partial MV Support
- Lung Protective Ventilation
- NIV
- Weaning
- Adjunct Rx: Proning, iNO
- ECLS

Severity of Respiratory Failure
Objectives

• Understand the contribution of mechanical ventilation on Ventilator-Induced Lung Injury (VILI)

• Appreciate the different characteristics of breath delivery by commonly used ventilator modes applied for acute respiratory failure

• Recognize important rescue strategies for managing refractory acute respiratory failure
Different Vent Modes: ALL Use Positive Pressure

• Vent MODES: Differences in how positive pressure “ventilator” breath is applied

• Not physiological, therefore...

• ALL VENT MODES CAN CAUSE LUNG INJURY (my opinion)
Ventilator Induced Lung Injury (VILI)

Mechanical Ventilation

OVER-DISTENSION

CYCLIC ALVEOLAR OPENING-CLOSURE

VOLU-trauma

BARO-trauma

ATELECT-trauma

BIO-trauma

LUNG INJURY
Lung Protective Ventilation

The New England Journal of Medicine

VENTILATION WITH LOWER TIDAL VOLUMES AS COMPARED WITH TRADITIONAL TIDAL VOLUMES FOR ACUTE LUNG INJURY AND THE ACUTE RESPIRATORY DISTRESS SYNDROME

The Acute Respiratory Distress Syndrome Network

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>GROUP RECEIVING LOWER TIDAL VOLUMES</th>
<th>GROUP RECEIVING TRADITIONAL TIDAL VOLUMES</th>
<th>P VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Death before discharge home and breathing without assistance (%)</td>
<td>31.0</td>
<td>39.8</td>
<td>0.007</td>
</tr>
<tr>
<td>Breathing without assistance by day 28 (%)</td>
<td>65.7</td>
<td>55.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>No. of ventilator-free days, days 1 to 28</td>
<td>12±11</td>
<td>10±11</td>
<td>0.007</td>
</tr>
<tr>
<td>Barotrauma, days 1 to 28 (%)</td>
<td>10</td>
<td>11</td>
<td>0.43</td>
</tr>
<tr>
<td>No. of days without failure of nonpulmonary organs or systems, days 1 to 28</td>
<td>15±11</td>
<td>12±11</td>
<td>0.006</td>
</tr>
</tbody>
</table>

31% LPV-Mortality vs. 40% Mortality
Why follow lung protective principles in patients on mechanical ventilation

• Needham et al: Prospective cohort study examining 2 year survival in patients with acute lung injury

• 3% decrease in mortality with each adherence to volume-limit lung protective ventilation (≤ 6 ml/kg IBW) and to pressure-limit lung protective ventilation (≤ 30 cm H₂O pPlat)
  • 4.0% ARR in 2-yr mortality with 50% adherence compared to no adherence
  • 7.8% ARR with 100% adherence compared to no adherence

• 18% relative increase in mortality for every 1 ml/kg over IBW tidal volume
Key Points-Part 1

- Mechanical ventilation is NOT physiological → All positive pressure ventilation is potentially deleterious to the susceptible lung
- Minimizing Ventilator Induced Lung Injury is PARAMOUNT in acute respiratory failure/ARDS

  - Principles of Lung Protective Ventilation (LPV) should be followed in all patients who require mechanical ventilator support
    - Tidal Volume ≤ 6 ml/kg IBW
    - Plateau pressures ≤ 30 cm H$_2$O
Objectives

• Understand the contribution of mechanical ventilation on Ventilator-Induced Lung Injury (VILI)

• Appreciate the different characteristics of breath delivery by commonly used ventilator modes applied for acute respiratory failure

• Recognize important rescue strategies for managing refractory acute respiratory failure
Mechanical Ventilator Support

DIFFERENT VENT MODES

- Partial MV Support
- Full Mechanical Vent Support
- Partial MV Support

Volume-AC
Pressure-AC
PRVC

PSV
NAVA
PAV

APRV
HFOV
HFPV

Increasing Ventilator Support

Severity of Respiratory Failure

ECLS
Ventilator Basics

4 Phases during Ventilator Cycle

Breath Initiation

• **Trigger**—Initiation of inspiratory phase: Pressure, Flow, or Time

Flow delivery (Inspiratory) phase

• **Limit**—Sustains inspiratory cycle eg. Limit flow 50 L/min

Breath termination

• **Cycle**—Ends inspiratory cycle eg. Cycle at TV 500 ml

Expiratory phase

• **Baseline**—Usually passive—depends on airway resistance & lung elasticity
Assist-Control (AC):
Most common ICU vent mode

- **Control**: Pressure or Volume
- **Trigger**: (Patient effort & sensitivity) Pressure, Flow, or (Machine) Time
- **Limit**: Pressure, Flow, or Volume
- **Cycle**: (Machine) Flow, Volume, Pressure, Time
Pressure or Volume: Is there a preference?

<table>
<thead>
<tr>
<th>Assist Control--VOLUME</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Constant Tidal Volume (Vt)</td>
<td>Peak alveolar pressure are variable--Over distension risk</td>
</tr>
<tr>
<td></td>
<td>PaCO₂ constant</td>
<td>Cannot respond to changes in ventilator demand--flow limited</td>
</tr>
<tr>
<td></td>
<td>Changes in Peak Inspiratory pressure easy to detect</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Assist Control--PRESSURE</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Peak alveolar pressure is limited</td>
<td>Vt is variable (patient effort → overdistension → Volutrauma)</td>
</tr>
<tr>
<td></td>
<td>Flow responds to patient</td>
<td>PaCO₂ can vary</td>
</tr>
<tr>
<td></td>
<td>Better vent synchrony</td>
<td></td>
</tr>
</tbody>
</table>
Dual Modes: Combines features of pressure & volume target to achieve goals of ventilation

- Known by different names
  - Volume Assured Pressure Support
    - Pressure Augmentation
  - Pressure Regulated Volume Control (PRVC)
    - Auto-flow
    - Variable Pressure Control
Dual Modes: Pressure regulated volume control (PRVC)

- Pressure control waveform but “guaranteed tidal volume”
- Ventilator (Machine) adjusts Pressure target to least value needed to maintain minimum Tidal Volume target
Advanced Ventilator Modes:

APRV
Hi Frequency Oscillation
APRV: Airway Pressure Release Ventilation

- “Inverse-ratio” Bi-Level Mode
- Maintains Lung Volume throughout cycle
  - CO\textsubscript{2} clearance occurs with pressure release & spontaneous breathing

**Settings**
- \( P_{\text{High}} \) & \( P_{\text{Low}} \)
- \( T_{\text{High}} \) & \( T_{\text{Low}} \)
APRV: Spontaneous Breathing
Reported benefits of APRV

- Decreases VILI
  - Reduces peak airway pressures
- Promotes improved aeration at lung bases—where atelectasis commonly occurs
- Allows patient to breathe while providing “OPEN” lung ventilation
  - Less sedation needed
- Improves oxygenation
  - Higher mean airway pressure (mPaw)
The Role of APRV

• Frequently used in awake patients with moderate-severe ARDS
  • Decreased sedation requirement → less vasoactive requirements

• Improved aeration & cardiovascular stability → lost when patient unable to breathe spontaneously

• Can be injurious in patients with high minute ventilation requirements & severe obstructive lung disease
  • Hyperinflation → High alveolar pressure → Barotrauma
High Frequency Oscillation: OSCILLATE & OSCAR

Multi-center randomized controlled trials comparing Adult patients with ARDS to treatment with Conventional mechanical ventilation versus HFOV
OSCILLATE trial terminated early due to increased in-hospital mortality in the HFOV arm (47% versus 35%)

OSCAR trial demonstrated no difference in 30-day mortality between groups: HFOV was not superior

CONCLUSIONS
In adults with moderate-to-severe ARDS, early application of HFOV, as compared with a ventilation strategy of low tidal volume and high positive end-expiratory pressure, does not reduce, and may increase, in-hospital mortality. (Funded by the Canadian Institutes of Health Research; Current Controlled Trials numbers ISRCTN42090778 and

(IMPORTANCE OF RANDOMIZED CLINICAL TRIALS)
Key Points-Part 2

• No conclusive evidence that “advanced ventilator modes” are superior to Assist Control in ARDS mortality
• New vent technologies try to adapt the ventilator to the patient
• The **BEST VENT MODE** is the one you and your team is most comfortable using
Objectives

• Understand the contribution of mechanical ventilation on Ventilator-Induced Lung Injury (VILI)

• Appreciate the different characteristics of breath delivery by commonly used ventilator modes applied for acute respiratory failure

• Recognize important adjuncts for managing acute hypoxemic respiratory failure—Severe ARDS
Hypoxemia

• Sufficient oxygen delivery depends on effective interaction of several important components:
**Acute Respiratory Distress Syndrome (ARDS)**

- ARDS: Pathological states of impaired GAS EXCHANGE by lungs

<table>
<thead>
<tr>
<th>Timing</th>
<th>Within 1 week of a known clinical insult or new or worsening respiratory symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest imaging&lt;sup&gt;a&lt;/sup&gt;</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 (eg, echocardiography) to exclude hydrostatic edema if no risk factor present</td>
</tr>
<tr>
<td>Oxygenation&lt;sup&gt;b&lt;/sup&gt;</td>
<td>Mild: (200 \text{ mm Hg} &lt; \frac{\text{PaO}_2}{\text{FiO}_2} \leq 300 \text{ mm Hg}) with PEEP or CPAP (\geq 5 \text{ cm H}_2\text{O})</td>
</tr>
</tbody>
</table>
Hypoxemic Respiratory Failure: the Value of PEEP (Positive END EXPIRATORY Pressure)

THE BENEFITS

PEEP improves gas exchange

- Recruits ALVEOLI thereby decreases SHUNT
- Less ATELECTRAUMA (reduce lung injury from repetitive alveoli open-closure cycle)

- APPLYING PEEP can be favorable in congestive systolic heart failure:
  - REDUCES PRELOAD to over distended heart
  - Decreases SYSTEMIC AFTERLOAD
Hypoxemic Respiratory Failure: the Importance of PEEP

THE DRAWBACKS

• Can be difficult to determine OPTIMAL PEEP
  • Balancing recruitment of “recruitable” alveoli from overdistension of already open alveoli
    • OVERDISTENSION  ➔ VILI & Collapse of pulmonary capillaries (V/Q mismatch)

• Hemodynamic effects of PEEP are hard to predict
  • Increased intrathoracic pressure can reduce preload
  • Right ventricular afterload can increase due to decreased pulmonary capillary area
  • Decreased atelectasis can improve oxygenation & decrease hypoxemic pulmonary vasoconstriction
  • PEEP reduces transmural LV pressure gradient ➔ Decrease afterload may augment cardiac output
Determining “Optimal” PEEP

• Many techniques to try to find best PEEP have been undertaken
  • Simple Rule: Roughly twice ~$\text{FiO}_2$ --> example: $\text{FiO}_2$ 70% PEEP of 14; $\text{FiO}_2$ 60% PEEP 12
  • Esophageal Pressure Measurement
  • Bedside Ultrasound Lung Recruitment & PEEP assessment
  • CT imaging
  • PEEP Ladders
Patients with increased intrathoracic pressure from external factors can (e.g. chest wall edema, morbid obesity, abdominal distension) can require “higher” vent pressures to keep lung open.

Esophageal pressure ($P_{es}$) acts a surrogate for pleural pressure ($P_{PL}$).

Use $P_{es}$ to guide PEEP and to limit Tidal Volume.

- Increase PEEP to keep Transpleural pressure ($P_{L}$) positive at end expiration to keep alveoli open $\Rightarrow$ PEEP minus $P_{es}$ = 0 to 10cm $H_2O$
Lung imaging-guided PEEP

Lung Ultrasound

Chest CT

Bouhemad B et al. AJRCCM 2011; 183: 341-47

Gattinoni L et al. NEJM 2006; 354: 1775-86
PEEP Ladders

Adjust along PEEP Ladder to achieve goal of PaO₂ 55-80 or O₂ sat 88-95%
Key Points regarding PEEP

- Optimizing PEEP is an important mainstay of improving oxygenation in ARDS (Goal 88-95% O₂ saturation)
- Set PEEP to prevent repetitive lung opening and closing
- Increase PEEP to achieve $P_{\text{plat}} \sim 28-30$ cm H₂O
- Use higher PEEP if the lung is recruitable
  - Severe ARDS (P/F < 200)
- User lower PEEP if the lung is not recruitable
  - Decrease risk of VILI
RESCUE THERAPIES—OTHER CONSIDERATIONS For SEVERE ARDS

• NEUROMUSCULAR BLOCKADE (NMB)
• PRONE POSITIONING
  • Inhaled nitric oxide/prostacyclin
• ECLS (VV-ECMO)
Neuromuscular Blockade

- Short administration (48hr) of NMB (cisatricurium) early in the course of severe ARDS (P/F < 150) leads to improved outcomes
  - Less time on ventilator
  - Improved 90-day adjusted survival
    - 31.6% vs. 40.7% (p=0.08)*
    - 28-day mortality 23.7% vs. 33.3% (p=0.05)
  - No increase in muscle weakness
PRONING for ARDS

• Improves oxygenation in severe ARDS (P/F < 100)
• Ventilation (PaCO2) may not necessarily improve
• Proning should be performed early in severe ARDS
• Studies indicate benefit of proning occurs with prolonged with proning (≥ 16hrs/day)
ECLS and ARDS: VV-ECMO
1979: Prospective trial of ECMO in ARDS

“In the meanwhile, study of the effect of bypass route on the injured lung and research to improve biomaterials are essential to improve ECMO.”

Improvement in Technology

Motorola DynaTac—
First cell phone call
April 3, 1973

Contemporary smart phones—Today
Improvement in ECMO Technology

1970’s

Today
Contemporary Era

Number of ECMO cases per million adult discharges in the United States from 1999 to 2011.
Basic Concept of ECMO
(now also referred to as ECLS)

• Take deoxygenated blood from the patient...
• ... pump the blood...
• ... through an oxygenator, and...
• ... return the oxygenated blood to the patient
Circuit components

- ECMO Device “The Pump”
- Cannulas
- Tubing
- Oxygenator
- Heat exchanger
- Hemoconcentrator
VV-ECLS Configuration: Dual Cannulas

Inflow (Drainage) Cannula: Inferior Vena Cava

Outflow (Return) Cannula: Right Atrium

• Blood is removed from the venous system
  • Oxygenated
  • Carbon Dioxide removed

• Blood is then returned back to the body centrally to right atrium.
VV-ECLS Configuration: Double lumen Single Cannula

- Cannula placed to right IJ with ECHO guidance
- Inflow from SVC & IVC
- Outflow returned to right atrium
- Cannula positioned so return flow is toward the tricuspid valve
ECMO for Severe Respiratory Failure: VV-ECLS

• Institute in situations after failure (or anticipated failure) of other conventional treatment modalities for pulmonary failure

• Provides TEMPORARY support—Improves oxygen delivery & CO₂ removal

• Secondary GOAL of VV-ECMO is to protect patient lungs to allow recovery
Indications for VV-ECLS: Respiratory Failure Conditions

**Reversible respiratory failure**

- Acute respiratory distress syndrome (ARDS)
- Pneumonia
- Trauma
- Status Asthmaticus
- Primary graft failure following lung transplantation
- Bridge to lung transplantation
Physiologic indications for VV-ECLS

• “Refractory” hypoxic/hypoxemic respiratory failure:
  - P-to-F ratio < 80-100
  - AaDO$_2$ > 600 mmHg
  - Transpulmonary Shunt fraction > 30%
  - Hi Positive pressure Settings

• CO$_2$ Retention
  - PaCO$_2$ > 80 mmHg
Contraindications for VV-ECLS

• Major contraindications
  • Need for circulatory support (i.e. need for VA-ECLS)
  • Futility
  • Most other contraindications are relative:
  • Prolonged mechanical ventilation at high settings
  • Major pharmacological immunosuppression
  • Age
  • Weight (BMI > 40)
  • Pre-existing conditions which affect quality of life
  • Risk of systemic bleeding from anticoagulation
Management of VV-ECLS

• Flow & ventilator rest settings
• Gas exchange
• Anticoagulation
• Monitoring
• Other critical care patient issues
Concepts: Oxygenation on VV-ECLS

• Arterial blood oxygenation (patient) ≠ ECMO return cannula oxygenation

• Patient arterial blood oxygenation affected by:
  • ECMO BLOOD FLOW
  • Re-Circulation
  • Cardiac output
  • Pulmonary shunting (Ability of native lungs to participate in effective gas exchange)
Concepts: Oxygenation of ECMO Blood Depends on ECMO BLOOD FLOW

• Oxygen transfer from ECMO oxygenator to patient’s blood depends primarily on ECMO blood flow
  • Transfer (removal) of Carbon Dioxide depends on Sweep gas flow

• ECMO blood flow determined by adequacy of venous drainage
Determinants of ECMO flow

- Right heart filling pressure
  - ECMO pump preload
- Venous Cannula Size ➔ Ideal cannula is the one with shortest length & largest internal diameter to minimize resistance
- Pump speed
Recirculation

• Portion of oxygenated blood returning to ECLS circuit via the inflow cannula immediately after being infused to patient from the ECLS circuit

• **Confirm recirculation when:**
  - $\uparrow$ pre-oxygenator venous $O_2$ sat
  - **but**
  - $\downarrow$ patient arterial $O_2$ sat
Factors affecting Recirculation

• Pump speed & flow

• Cannula(s) position & configuration

Equation—*hard to measure SvO$_2$*:

\[
\text{Recirculation (\%)} = \frac{(\text{SpreO}_2 - \text{SvO}_2)}{(\text{SpostO}_2 - \text{SvO}_2)} \times 100
\]
Recirculation: Pump Flow

- Pump flow increases recirculation linearly
- Increase in pump flow will eventually decrease “effective ECMO oxygen flow”
Recirculation: Cannula type & position
VV-ECLS: Interface with Cardiac Output & Native Lung Function

5.0 LPM

4.0 LPM

Recirculation

1.0 LPM

6.0 LPM

4.0 LPM

Recirculation

5.0 LPM

10.0 LPM

10.0 LPM
ECLS Potential Benefits

- Decreased risk of Ventilator Associated Events
- Better oral nutrition
- Decreased need for analgesia/sedation
- Comfort
- Facilitate physical rehab/mobility
- Better patient communication
Management of ARDS: Putting it all together

**PATIENT WITH MODERATE/SEVERE ARDS**

- P:F<200 or desats?
  - Initiate LPV protocol
  - Assess/treat ventilator dysynchrony
- P:F<150 or desats?
  - Initiate high PEEP LADDER
  - Initiate 48 hours of NMB
  - Consider PRONE positioning
- P:F<100 or desats?

**Consider other rescue therapies:**
- Inhaled Prostacyclin or Nitric Oxide
- Esophageal manometry with additional PEEP titration
- Extracorporeal Life Support
Management of Acute Respiratory Failure

Aaron M Cheng, MD
March 15, 2016