ADVANCED MODES OF VENTILATION:
CONCERNS FOR THE OR

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WHAT I AM NOT GOING TO Cover
“Adaptive” Advanced Modes that focus on synchrony
- Proportional assist
- Adaptive Support
- Neurally Adjusted Ventilatory Assist

Why?
- Evidence of benefit is lacking
- Generally interchangeable with standard intraop modes

DISCLOSURES
NONE

LEARNING OBJECTIVES
1. Review the pathophysiology of the diseased or injured lung
2. Understand recent strategies in mechanical ventilation, particularly focusing on “low-stretch” and “open-lung” techniques.
3. Discuss strategies for OR management of patients on “advanced” vent modes
4. Apply these concepts to routine OR vent management

PATHOPHYSIOLOGY OF THE SICK LUNG
1. ARDS
   Ashbaugh and Petty: 1967 case series of 12 ICU patients
   - Tachypnea and hypoxemia
   - Opacification on CXR
   - Poor lung compliance
   - Diversity of primary insult

PATHOPHYSIOLOGY OF THE SICK LUNG
ARDS: The Berlin Definition (c. 2011)
   ARDS is an acute diffuse, inflammatory lung injury, leading to increased pulmonary vascular permeability, increased lung weight, and loss of aerated lung tissue...[With] hypoxemia and bilateral radiographic opacities, associated with increased venous admixture, increased physiological dead space and decreased lung compliance.

**PATHOPHYSIOLOGY OF THE SICK LUNG**

**ARDS: The Berlin Definition (c. 2011)**

<table>
<thead>
<tr>
<th>Timing</th>
<th>Acute Respiratory Distress Syndrome</th>
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<tbody>
<tr>
<td>Within 1 week of a known clinical insult or new or worsening respiratory symptoms</td>
<td></td>
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<tr>
<td>Recent illness or injury</td>
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<table>
<thead>
<tr>
<th>Chest imaging*</th>
<th>Bilateral opacities – not fully explained by effusions, lobar lung collapse, or consolidation</th>
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<tbody>
<tr>
<td>Origin of edema</td>
<td>Respiratory failure not fully explained by cardiac failure or fluid overload</td>
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<tr>
<td>- Hypoxic respiratory failure</td>
<td></td>
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<tr>
<td>- Non-hypoxic respiratory failure</td>
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<table>
<thead>
<tr>
<th>Oxygenation</th>
<th>Acute</th>
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<tbody>
<tr>
<td>200 mm Hg &lt; Pao2/FiO2 &lt; 300 mm Hg with PEEP or CPAP</td>
<td>Acute respiratory distress syndrome</td>
</tr>
<tr>
<td>Moderate</td>
<td>100 mm Hg &lt; Pao2/FiO2 &lt; 200 mm Hg with PEEP or CPAP</td>
</tr>
<tr>
<td>Severe</td>
<td>Pao2/FiO2 &lt; 100 mm Hg with PEEP or CPAP</td>
</tr>
</tbody>
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* Pao2/FiO2 = arterial oxygen tension/partial pressure of inspired oxygen

**VENTILATING THE NON-COMPLIANT LUNG**

What happens if we use conventional modes?

**MECHANISMS OF INJURY**

- Barotrauma: high peak pressure
- Volutrauma: over distention
- Atelectotrauma: cyclic opening/closing of alveoli
- Biotrauma: inflammatory mediators

Scott, Benjamin K., MD

Advanced Modes of Ventilation: Concerns for the OR
THE EMERGENCE OF "LUNG PROTECTIVE" OR "LOW STRETCH" MECHANICAL 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

ARDSNET

6 vs 12 ml/kg (IBW)

PEEP and FiO2 protocolized

Mean Peep approx 8-9 cm H2O

Stopped after 861 patients enrolled

22% reduction in mortality in the low-stretch group

Conclusion: low tidal volumes are protective

HOW DO WE OPERATIONALIZE LOW STRETCH?

INVERSE RATIO VENTILATION

THE CONCEPT OF THE "OPEN LUNG"
**Recruitment Maneuvers**

- Theoretical benefit to periodic recruitment in both ARDS and routine intraoperative setting
- Avoidance of tidal shear stress
- Small studies have shown benefit but data are equivocal
- How much, how long? (Ramp, cyclic, brief sustained)
- Which patients? (Responders vs non-responders)
- Risk of hypotension and hemodynamic collapse

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**Benefits of APRV are Mostly Linked to Maintenance of Spontaneous Respiration**

- Recruitment, especially adjacent to diaphragm
- Decreased intrathoracic pressure (Lower Ppl but local increase in Ptp)
- Improved PaO2
- Better V/Q matching (when 10-30% of VE is spont)
- Improved CI (periodic reduction in transthoracic pressure)
- Improved organ perfusion (renal, gut)

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**Low Stretch and Open Lung: APRV**

- Time-cycled switching between high and low pressure
- Delta Pressure = mechanically delivered V̇
- Setting T_{high} and T_{low} enables rate adjustment
- Unrestricted spontaneous breathing (PSV)
- True APRV, T_{low} ≤ 1.5 seconds

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**Goals When Setting APRV**

- Tidal volume 6ml/kg, Plateau ≤ 30 cm H₂O
- Duration of high pressure should allow spontaneous breathing
- Duration of low pressure should allow complete expiration and minimize intrinsic PEEP
- Target light to moderate sedation (Ramsay 2-3, RASS 0 to -2)
Asynchrony is most likely when pressure is released during spontaneous inspiration or reapplied during spontaneous expiration.

Timing of high and low pressures may need to be adjusted.

Titrate sedation.

“Synchronized” APRV is being developed.

Increase low pressure (may effect Ve).

Decrease T_{low}.

Ensure adequate spontaneous respiratory rate.

Inhaled nitric oxide or flolan may decrease shunt.

Mild to moderate acidosis may be protective, but worsening acidosis can be problematic.

Try to minimize intrinsic PEEP by lengthening T_{low}.

Raise high pressure setting to increase V_{T}.

Bronchodilator therapy, corticosteroids (if COPD).

Prone Positioning:

- Theoretical benefit

- Several meta-analyses showed benefit, but RCTs had not, potential risks

Prone Positioning:

- 466 patients with early severe ARDS
- 12-24h stabilization period
- Randomized to supine versus prone for at least 16h per day
- All ICUs had at least five years proning experience
- Mortality reduction of 16%
Two major trials published in 2013:
- OSCAR showed no benefit vs conventional low-stretch
- OSCILLATE showed increased mortality


ECMO: CESAR TRIAL LIMITATIONS
- Single center trial
- No safety analysis (6% mortality prior to transport)
- Randomized early and degree of ARDS was severe
- 17 patients (19%) were transferred and improved prior to ECMO
- No standardized protocol (many controls did not receive lung protective ventilation)

ECMO: CESAR TRIAL
Efficacy and economic assessment of conventional ventilatory support versus extracorporeal membrane oxygenation for severe adult respiratory failure (CESAR): a multicentre randomised controlled trial

180 Pts with potentially reversible ARDS (Murray score ≥ 3)
- Excluded PIP >30 or FIO >80% for >7d
- Randomized to conventional ventilation or transfer to a single center with intent to start ECMO
- 6m survival 63% in ECMO group vs. 47% in controls (p=0.03)


Non-conventional salvage modes:
- Appear to depend on experience of the center
- Which patients will benefit?
- What is the optimal timing and duration of therapy?

Practical considerations for the OR:
- Does the procedure need to be done in the OR?
- Does the patient need the ICU ventilator?
- Can the patient tolerate repositioning (trial in the ICU)
- Can the patient tolerate even a transient disconnect (consider clamping the tube)
- Is paralysis necessary? (If so, APRV = pressure control)
**Relative Contraindications:**

- Difficult anatomy
- Significant coagulopathy
- Proximity to site of recent surgery or trauma
- Potential instability (hemodynamics, ICP)
- Severe gas exchange problems: e.g. \( Fio2 > 0.6 \) and PEEP > 10 cm H2O

Intensive Care Society UK, Standards 2014

**DOES THIS APPLY TO MY PATIENTS?**

**What we do know:**

- 90% of patients undergoing general anesthesia with mechanical ventilation will have atelectasis
- Blum et al. found that 46% of patients had hypoxemia during anesthesia, and 4% had P/F ratios under 100 mmHg
- Multiple recent investigations have found that the majority of patients are ventilated with tidal volumes 9-10 ml/kg and without application of PEEP


**DOES THIS APPLY TO MY PATIENTS?**

- Lung injury from mechanical ventilation is well-documented
- ARDS likely represents a multiple hit model
- We should focus on minimizing the blows we deliver

**QUESTIONS?**