CareFusion Critical Care Ventilators

Avea
- Used in both critical & sub-acute settings
- Industry-leading critical care ventilation
- Unique options include minimally invasive & minimally invasive monitoring

Vela
- Used in both critical & sub-acute settings
- Industry-leading critical care ventilation
- Unique options include minimally invasive & minimally invasive monitoring

EnVe
- The only high-frequency oscillatory ventilator currently available
- Used in neonatal & pediatric patients

3100A & 3100B
- Used in Neonatal, Pediatric & Adult critical care settings
- Industry-best neonatal ventilation
- Unique options include volumetric capnography & esophageal pressure monitoring

Questions to be answered
- From what are we protecting the lungs?
- How do we safely manage the "handful" of ventilatory parameters that are currently available to us?
- What clinical evidence supports these methods of protective strategies?
- What does the future hold for lung protective strategies?

Lung-Protective Ventilation

Strategies & Application

What’s So Bad About Mechanical Ventilation?
**What’s So Right with Spontaneous Ventilation?**

- During a spontaneous inspiration, small negative intrapleural, interstitial and alveolar pressures are generated which allow the lung to inflate.

- During a spontaneous exhalation, intrapleural pressures return towards atmospheric, but remain slightly negative, while interstitial and alveolar pressures return towards normal or slightly positive.

**What Is Mechanical Ventilation, Really?**

- Just what is it that is delivered by the ventilator to the patients’ lungs?
  
  - Volume
  
  - Flow
  
  - Pressure

**What’s So Bad With Mechanical Ventilation?**

- Positive-pressure ventilation departs radically from the physiology of breathing spontaneously.

- During a positive-pressure inspiration positive intrathoracic pressures are created:
  
  - These pressures are not homogenously distributed throughout the lung:
    
    - Effectively distributed through compliant lung
    
    - Flow is attenuated in low-compliant areas
  
  - This heterogeneity can result in overdistension of compliant lung with underdistension of less-compliant lung

- The immediate macroscopic physiological side-effects of positive-pressure ventilation are readily recognized and easily understood.

- However, there are significant microscopic physiological interactions which are less obvious, more insidious, and may only produce complications if ventilation is prolonged.
The Other End of the Circuit

Why Are We Ventilating?

ALI & ARDS

- Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS) are common, life-threatening diseases that are the clinical and radiological manifestations of an inflammatory response within the lung secondary to direct and indirect insults.
- 1967 - First described in Lancet by Ashbaugh in 1967
  - Vague criteria for diagnosis, not specific enough to exclude other conditions.
- 1988 – Murray, in American Review of Respiratory Diseases created a ALI scoring system that considered:
  - Level of PEEP
  - PF Ratio
  - Static Lung Compliance
  - Changes in CXR

Which Patients are at Risk for ARDS

- Trauma
- Shock Syndromes
  - Septic
  - Cardiogenic
- Gastric Aspiration
- Burns
- Diffuse Pneumonias
- Near Drowning
- Drug Overdose
- Metabolic Events
  - Pancreatitis
  - Uremia
- Systemic-Mediator-Release Syndromes
- Disseminated Intravascular Coagulopathy
- Cardiopulmonary Bypass
- Anaphylaxis
- Extrapulmonary Infection
- Transfusion Reaction
### How Common is ALI & ARDS?

- Incidence of ALI: 18-79 cases/100,000 persons
- Incidence of ARDS: 13-59 cases/100,000 persons
  - 190,000 cases in the US each year, >500/day
- More than 80% of patients with ARDS will require intubation and mechanical ventilation.

### ALI & ARDS Mortality

- Mortality from ALI & ARDS:
  - Secondary to Sepsis, Pneumonia & Aspiration: 35-40%
  - Secondary to Trauma: 10-15%
- ARDS patients with improvement in Oxygenation and disease severity in first 24 hours after initiation of mechanical ventilation – mortality is 13% - 24%
- ARDS patients with little improvement in first 24 hours in PaO2/FiO2 ratio or with PEEP ≥ 15cmH2O – mortality is 53% - 68%

### Oxygenation Index (OI)

- May be a better predictor of patient outcome than the P/F ratio
- Incorporates oxygenation (P/F Ratio) and Mean Airway Pressure
  
  \[
  OI = \frac{\left(\text{FiO}_2 \times mPaw \times 100\right)}{\text{PaO}_2}
  \]

- Persistently high or rising OI 12 to 24 hours after initiation of mechanical ventilation is identified as a risk factor for increased mortality.
- OI > 30 represents a failure of conventional ventilation and may indicate the need for nonconventional modes.

### The Treatment of ARDS

- As part of the therapy for the underlying disease (such as shock, trauma, sepsis, pneumonia, aspiration or burns) Mechanical Ventilation is critical for resolving life-threatening hypoxia and hypercapnia
- Studies have shown that mechanical ventilation can further damage lungs due to overinflation, barotrauma and cyclic closing and opening of the alveoli.
- The phenomenon has been named Ventilator-Associated Lung Injury (VALI) or Ventilator-Induced Lung Injury (VILI)
What’s Wrong With Mechanical Ventilation?

- The goal of mechanical ventilation is to provide adequate ventilatory support while minimizing lung damage.
- Limiting lung damage should be accomplished through a reasonable, evidence-based approach to ventilator management which includes volume & pressure limitation and modest PEEP & Plateau pressures
- The need for potentially injurious pressures, volumes, and FiO2’s must be weighed against the benefits of gas exchange support.

Ventilator-Induced Lung Injury

A Early Focus on Peak Pressures

- Early 20th century clinicians noted that excessive lung distension from positive-pressure ventilation would rupture alveolar units and produce extra-alveolar air (pneumothorax, pneumo-mediastinum, and pneumoperitoneum)
- The causative factors for this extra-alveolar air were grouped together under the umbrella term of Barotrauma.
- Research in the 1970’s by Pontoppidan demonstrated that ARDS patients experienced discomfort with the use of small Vt’s.
  - Thus began the practice of using Vt’s in the 10-15 ml/kg range
  - Practice became centered on preventing Barotrauma.
  - As long as Peak Inspiratory Pressures were maintained in the “safe” range then the lungs were protected.

Ventilator-Induced Lung Injury

The Rise of Volutrauma

- Also in the 1970’s, experiments by Webb & Tierney and others demonstrated two important (if not widely applied) concepts:
  - Distending pressures & volumes above normal maximums but below that which was required for alveolar rupture produced lung edema, surfactant abnormalities, tissue inflammation and hemorrhage.
  - Preventing cyclical alveolar collapse & reopening could also significantly reduce the incidence of lung injury.
**Ventilator-Induced Lung Injury**

**Volutrauma**

- More recent studies have shown that alveoli that do not overdistend were unlikely to experience damage
- Normal and excessive alveolar pressures were applied to both volume-limited lungs (chests bound to prevent alveolar expansion) and volume-unlimited lungs (chests unbound with unchecked alveolar expansion)
- Alveolar pressures caused considerably less lung damage in the alveoli with limited expansion than in the alveoli in the unbound chest.

**Ventilator-Induced Lung Injury**

**Inflammation**

- Study investigating the release of "Lung Flooding" factors in Rodents ventilated with three modes:
  - HIP/HV
    - High Pressure (45 cmH2O)
    - High Volume
  - LoP/HV
    - Low Pressure (neg.pres.ventilator)
    - High Volume
  - HIP/LoV
    - High Pressure (45 cmH2O)
    - Low Volume (chest strapped)

**Take-Home Points - Volutrauma**

- Excessive Plateau Pressure
  - Excessive end-inspiratory volume
- May result from a combination of PEEP + Vt
- May severely over-inflate normal alveoli
- Both mechanical and biochemical injury may develop

**QUESTION:**

How can the clinician determine if alveoli are over-distended at end-inspiration?

**Atelectrauma**

- Research has also revealed that repeated cyclical collapse/re-expansion of alveoli results in a release of cytokines and the reinforcement and amplification of the local and systemic inflammatory response.
- Interleukin-6
- Interleukin-11
- Interleukin-γ
- Tissue Necrosis Factor-α
**Ventilator-Induced Lung Injury**

**Take-home Points - Atelectrauma**

- Associated with repeated opening and closing of alveoli during ventilatory phasing
- Associated with regional differences in ventilation
- Worsens surfactant dysfunction
- Release of inflammatory mediators into alveolar spaces and into the systemic circulation
- **QUESTION:** How can the clinician determine what PEEP is needed to keep the alveoli open at end-exhalation

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**Atelectrauma vs. Volutrauma**

**Atelectrauma:**
- Repetitive alveolar collapse and reopening of the under-recruited alveoli

**Volutrauma:**
- Over-distension of normally aerated alveoli due to excessive volume delivery

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**The Causes of VILI: Oxygen Toxicity**

- Since the 1950’s we have known that “increased” levels of inspired oxygen can injure lungs through:
  - The formation of cytotoxic oxygen radicals
    - Form disruptive chemical bonds with surrounding lipids, proteins and carbohydrates
    - Cause a chain-reaction that in-turn damages cell membranes, collagen, connective tissue and DNA as well as altering enzymatic reactions within these tissues.
    - Absorption atelectasis
  - What is a safe FiO2?

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**Presumed Mechanism for VILI**

- Upregulation & release of Cytokines, Chemokines
- Subsequent leucocyte attraction and activation
- Pulmonary Inflammation: VILI
- Systemic Spillover: SIRS / MODS
- MECHANOTRANSDUCTION – Conversion of Mechanical Stimulation into Chemical Reaction
- SIRS – Systemic Inflammatory Response Syndrome
- MODS – Multi Organ Dysfunction Syndrome
**Healthy Alveolus vs. Damaged Alveolus**

- Normal Alveolus
  - Alveolar air space
  - Type I cell
  - Surfactant Layer
  - Type II cell
  - Sloughing bronchial epithelium
  - Edema fluid
  - Necrotic or Apoptotic Type I cell
  - Red cell

- Inactivated surfactant

**Pulmonary Inflammatory Cascade**

- Volutrauma, Atelectrauma, Barotrauma, Biotrauma
- Endothelial & Epithelial Damage
- Surfactant Deactivation
- Alveolar Cell Injury and loss
- Protein Leak & Cytokine Release
- Capillary Congestion
- Interstitial/Alveolar Edema & Hemorrhage
- Protein Accumulation in Alveolar Air Space
- Atelectasis
- Hyaline Membrane Formation
- Inflammatory Cell Migration
- Higher FIO2, Volumes, Pressures

**The Sequence of VILI**

**Acute Phase**

- Biochemical damage to Type I Alveolar Cells
  - Disruption of the alveolar/capillary barrier
  - Flooding of alveoli with interstitial fluids, proteins, red blood cells & fibroblasts

- Biochemical damage to Type II Alveolar Cells
  - Decreased surfactant production and deactivation
  - Atelectasis

- Coagulation abnormalities
  - Platelet & fibrin-rich thrombi causing microvascular occlusion

- Ventilation/Perfusion mismatch
  - Decreased compliance
  - Increased dead space

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The Sequence of VILI

Resolution Stage (Day 5-10)

- Dependent upon repair of alveolar epithelium, clearance of pulmonary edema and removal of proteins from alveolar space
- Proliferation of Type II Alveolar Cells across the alveolar basement membrane
  - Eventually differentiate into Type I Cells
- Removal of fluid via active transport through Type II Alveolar Cells
- Removal of non-soluble proteins by endocytosis & transcytosis of Type I Alveolar Cells and phagocytosis by macrophages

Laycock, British Journal of Medical Practitioners, 2010

The Sequence of VILI

Fibrotic Stage (Day 10-14)

- Some patients do not undergo the Resolution Phase but progress to fibrosing alveolitis.
  - Found on autopsy of 55% of all non-survivors
- Alveolar spaces filled with inflammatory cells, blood vessels and excessive deposition of proteins and collagen.
- Interstitial & alveolar fibrosis develops
  - Decreased pulmonary compliance
  - Only partial resolution of pulmonary edema
  - Continued hypoxemia

Laycock, British Journal of Medical Practitioners, 2010

The Villians of VILI

- **Volutrauma** - Overdistension
  - Destruction of Lung Tissue
  - Increased Capillary Permeability
- **Atelectrauma** - Cyclical Collapse & Re-expansion
  - Release of Inflammatory Mediators
  - Surfactant dysfunction
  - Increased regional over-distention and worsening shunt
- **Barotrauma** - Excessive Transpulmonary Pressure
  - Gross Air Leaks
- **Oxygen Toxicity** - Excessive FiO2
  - Cytotoxic radicals
  - Absorption Atelectasis

Villian’s Up Close & Personal:

Volutrauma
**Volutrauma - Stretch Injury**

- Excessive volume at end-inspiration
- Excessive Plateau Pressure
- May result from a combination of PEEP + Vt
- May severely over inflate normal alveoli
- Both mechanical and biochemical injury may develop

**Volutrauma - Stretch injury**

- Mean pulmonary capillary pressure is usually 7-10 mmHg
- Positive-pressure ventilation can produce interstitial pressures that exceed capillary pressures.
  - Tend to compress the capillary and impede its flow, especially in the ventilated alveoli.
- Increased pulmonary artery pressures and high inflation pressures can cause Capillary Stress Failure
  - Leak of proteinacious material into the alveoli promoting atelectasis.

**Volutrauma - Diffuse Lung Injury**

- Small Vt

**Volutrauma – Histologic Evidence**

- Saline-lavaged Rabbit Model - 4 hours
Villian’s Up Close & Personal:  

**Atelectrauma**

- Associated with repeated opening and closing of alveoli during ventilatory phasing
- Associated with regional differences in ventilation
- Worsens surfactant dysfunction
- Release of inflammatory mediators into alveolar spaces and into the systemic circulation

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**Cyclic Shearing**

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**What Can We Do?**  

*Identifying the Problem*
Using Graphics to Prevent VILI: Recognizing VILI on the PV Loop

- **Overdistension**
  - Edematous fluid accumulation
  - Surfactant degradation
  - Increased oxygen exposure
  - Mechanical disruption

- **Derecruitment/Atelectasis**
  - Repeated closure and re-expansion
  - Stimulation of inflammatory response
  - Inhibition of Surfactant Production
  - Local hypoxemia
  - Compensatory overexpansion

Using Graphics to Prevent VILI: The Pressure-Volume Curve

- **Graphic representation of Volume vs Pressure**

- **Upper Inflection Point**
  Represents the pressure and volume at which if exceeded regional overdistension may occur.

- **Lower Inflection Point**
  Represents the minimal pressure for maintenance of adequate alveolar recruitment

Using Graphics to Prevent VILI: The P-V Curve in Acute Respiratory Failure

- **Reduced lung compliance:**
  - Curve is "more horizontal"
  - Decreased range of volume excursion

- Flattening of the curve at low and high volumes:
  - Increased risk of Volutrauma at upper section of curve
  - Increased risk of Atelectrauma at lower section of curve

Yum, Yum, Pig Lungs

- Increased risk of ARDS

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What Can We Do?
Managing the Ventilator

What We’ve Learned from ARDSNet –
www.ardsnet.org

- Network of 42 hospitals (organized into 12 clinical sites) conducting ongoing research into the best treatments for ARDS.
- Studies Completed or Underway:
  - ARMA Lower Vt
  - KARMA Lower Vt with Ketoconazole
  - LaSRS Late Steroids
  - LARMA Lysophylline (cytokine inhibitor)
  - ALVEOLI High PEEP/Low FiO2 vs Low PEEP/High FiO2
  - FACTT Wet vs. Dry Fluid Balance
  - Eden-OMEGA Enteral
  - SAILS Statins

Current Concepts in Lung Protective Strategy

- Small Vt
  - 4-6ml/Kg IBW
  - Minimizes Stretching Force
  - Permissive Hypercapnea

- Low Plateau Pressure
  - Below Upper Pflex
  - Try to keep <30 cmH20
  - Reduces regional overdistension

- PEEP
  - High vs. Low PEEP strategy still uncertain
  - What do YOU call HIGH PEEP?

Managing the Ventilator:
The Modes
Ventilator Management:

**Ventilatory Modes**

- **Volume-Limited**
  - Stable Vt in the presence of varying compliance
  - Flow is “Back-End” loaded

- **Pressure-Limited**
  - Higher Mean Paw
  - Flow is “Front-End” loaded
  - More rapidly fills alveoli

- Dual or hybrid breath types may offer both desired effects

- Choice depends largely on goal

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**Volume-Limited Ventilation**

- Gives clinician control of minute ventilation and CO2 clearance
- Airway and alveolar pressures become variable
- May be less comfortable for actively breathing patients

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**Pressure-Limited Ventilation**

- Pressure limited and time cycled.
- Flow is variable and is based on distending pressure
- May overinflation with improving compliance

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**Choosing Volume or Pressure Ventilation**

- The choice of pressure vs. volume targeted breaths depends on which feature is required for the clinical goal.
- Specifically, if CO2 clearance is of primary concern and patient comfort and lung stretch are less of an issue, **volume targeted** ventilation would be preferable.
- On the other hand, if over-distension risk if high and/or patient synchrony is more of an issue than CO2 clearance, **pressure targeted** ventilation is probably the correct choice.

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*MacIntyre, New Approaches to Mechanical Ventilatory Support, 2003*
Hybrid Modes

- Combine the features of volume and pressure
- Available on most current ventilators
- PRVC, VC+, Autoflow are the predominate types

Managing the Ventilator:

A Handful of Settings

Ventilator Management:

A Handful of Settings

- FiO2 Accurately measured
- Respiratory Rate Accurately measured
- Tidal Volume Accurately measured
- PEEP Measured but not accurate
- Plateau Pressure Measured but not accurate

The Ventilatory Handful

The Three We Do Well
The Pinkie: Tidal Volume (Vt)

- Vt Goal: 4-6 ml/kg IBW (Ideal Body Weight)
  - Males = 50 + 2.3 [height (inches) - 60]  
  - Females = 45.5 + 2.3 [height (inches) - 60]

- Initial Vt of 8 ml/kg IBW then reduce Vt by 1 ml/kg at intervals ≤ 2 hours until Vt = 6ml/kg IBW.

- Recent research has indicated that variable Vt's ("noisy ventilation") may improve respiratory function and reduce inflammatory response.

The Ring Finger: Respiratory Rate (RR)

- RR Goal: Approximate the patients baseline Spontaneous Ve (along with Vt)
  - RR not to exceed 35 bpm
  - Adjust Vt and RR to achieve pH and plateau pressure goals

The Bird: FIO2

- Oxygenation Goal 1: PaO2 55-80 mmHg
- Oxygenation Goal 1a: SpO2 88-95%

The Ventilatory Handful

The Two We Guess
The Pointer: Plateau Pressure

- Plateau Pressure Goal: Keep < 30 cmH2O
- Check Pplat (minimum 0.5 second inspiratory pause) at least q 4h and after each change in PEEP or Vt.
  - If Pplat > 30 cmH2O:
    - Vt by 1 ml/kg to minimum of 4 ml/kg.
  - If Pplat < 25 cmH2O and Vt < 6 ml/kg:
    - Vt by 1 ml/kg until Pplat > 25 cmH2O or Vt = 6 ml/kg.
  - If Pplat < 30 but breath stacking or dysynchrony occurs:
    - Vt by 1 ml/kg to a Vt of 7-8 ml/kg if Pplat remains < 30 cmH2O

The Thumb: PEEP

- PEEP Goals:
  - Recruit available alveoli while not overdistending “good” alveoli.
  - Avoid overdistention (monitor plateau pressure).
- Many methods to determine optimal PEEP
  - Static Compliance
  - PFlex Maneuver
  - Stress Index
  - Esophageal Pressure Monitoring

Use of PEEP < 10 cmH2O leads to an increase in mortality

Ventilator Management: PEEP Tables

- Consider the use of Incremental FiO2/PEEP Tables to achieve goal.

Low PEEP/High FiO2 Protocol

<table>
<thead>
<tr>
<th>FiO2</th>
<th>0.3</th>
<th>0.4</th>
<th>0.4</th>
<th>0.5</th>
<th>0.6</th>
<th>0.7</th>
<th>0.7</th>
<th>0.8</th>
<th>0.8</th>
<th>0.9</th>
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High PEEP/Low FiO2 Protocol

<table>
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<tr>
<th>FiO2</th>
<th>0.3</th>
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<td>22</td>
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<td>22</td>
<td>24</td>
</tr>
</tbody>
</table>

- Couple of considerations:
  - Assumes a pleural pressure of ZERO!
  - Does not address “PEEP Hunger”
  - The need for higher PEEP’s with lower FiO2’s

Ventilator Management: PEEP Controversies

- Two recent studies have shown no reduction in patient mortality with the application of an aggressive PEEP strategy (with Vt of 6cc/kg)
  - ExPress Trial (Mercat, Eur Soc Int Care-Barcelona 2006)
    - 850 Patients – Exper: PEEP @ Plateau / Control: PEEP @ 7
    - Mortality NS
  - LOVS Trial (Mead, Eur Soc Int Care-Barcelona 2006, JAMA 2008)
    - 983 Patients – Exper: PEEP 16 / Control: PEEP @ 10
    - Mortality 35 / 40
Why Airway Pressures Aren’t Accurate: Transpulmonary Pressure ($P_{TP}$)

- The airway pressure displayed by the ventilator ($P_{aw}$) actually reflects the pressure within the entire respiratory system ($A$).
- $P_{aw}$ is actually the combination of both chest wall & pleural pressures ($B$) and lung pressure ($C$).
- To determine the actual pressure within the lung itself it is necessary to account for pressures created by the chest wall, abdomen and pleura ($P_{cw}$).
- Once $P_{cw}$ has been subtracted from the $P_{aw}$ you have the actual pressure within the lung ($C$) — Transpulmonary Pressure ($P_{TP}$).

$$P_{TP} = P_{aw} - P_{cw}$$

The Problem with PEEP & Plateau Pressures: The Benefit of Transpulmonary-Guided Ventilation

- Although Plateau and PEEP pressures are vitally important in ventilator management, the values displayed by ventilators do not actually represent the pressures in the lung but are instead the combination of pressures of both the Lung and the Pleura.
- Pleural Pressures are affected by chest wall and abdominal pressures.
- Only when these pleural pressures are accounted for can the true lung pressure be determined.
- True Lung Pressures are better known as Transpulmonary Pressure ($P_{TP}$).
  - $P_{TP \_PLAT}$ is the true plateau pressure in the lung.
  - $P_{TP \_PEEP}$ is the true end-expiratory pressure in the lung.

Transpulmonary-Guided Ventilation: How Common are Increased Intra-Abdominal Pressures?

- Must measure and then subtract from the PAW the pressures that are outside of the lung pushing inwards.
  - Pleural Pressure
  - Chest Wall Pressure
  - Abdominal Pressure

How can we Measure True Lung Pressures ($P_{TP}$)?

- Esophageal Pressure Monitoring
  - Esophageal Pressures have been shown to correlate to pleural pressures.


Abdominal Pressure Total Prevalence MICU SICU

<table>
<thead>
<tr>
<th>Pressure</th>
<th>MICU</th>
<th>SICU</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;12 mmHg</td>
<td>58.8%</td>
<td>54.4%</td>
</tr>
<tr>
<td>&gt;15 mmHg</td>
<td>28.9%</td>
<td>29.8%</td>
</tr>
<tr>
<td>&gt;20 mmHg</td>
<td>8.2%</td>
<td>10.5%</td>
</tr>
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</table>

13 ICU’s, 6 countries, 97 patients S/P Decompressive Laparotomy
The Basics of Transpulmonary-Guided Ventilation

- Recent studies have shown that utilizing Transpulmonary Pressure (PTP) in the management of mechanical ventilators can:
  - Enhance Lung Protective Ventilation
  - Determine the true effectiveness of PEEP
  - Determine actual Plateau Pressures in the lung
  - Determine true Lung Compliances & Resistances and WOB
  - Determine the pressure surrounding the myocardium and calculate the pressure impeding venous return.

The Basics of
Transpulmonary-Guided Ventilation

A Brief Case Study: Guiding PEEP & Plateau with Transpulmonary Pressures

HPX:
Morbidly Obese 24 y/o Female with Pancreatitis

Settings:
PRVC/AC, RR-24, VT-540, PEEP-7, PCO2-43, TL-7

ABG:
PH-7.36, PCO2-50, Pao2-57, SaO2-93%

Pump up the PEEP

Some Heart Border & Diaphragm now visible

Which Plateau Pressure is Correct?

- PAW Plateau
  - 41 cmH2O
- PEEP PLAT
  - 21 cmH2O
Further Pumpage

- PEEP increased to 25 cmH2O
- Ptp PEEP now +2.4 cmH2O
  - Lungs are remaining open at end-exhalation

Hey, I Know, Let’s Try APRV!

- P_Low of 0
  - PTP PEEP of -15 cmH2O
  - IMMEDIATE Derecruitment!

Uh-Oh, Now What?

- Returned to PC/AC
  - PEEP of 25 cmH2O
- Ptp PEEP now +2.4 cmH2O
  - No derecruitment!
  - PAW Peak of 46 cmH2O
  - PTP Peak of 27 cmH2O
  - Physicians were hesitant to maintain PEEP of 25

Outcome

- Six Days after adjustment of PEEP using PES monitoring
- PEEP 16 cmH2O with FiO2 of .40
- Heart border and diaphragms visible
Other Ventilator Management Techniques

Ventilator Management: Permissive Hypercapnea

- pH Goal: 7.30-7.45
  - First described in 1990 by Hickling’s study of 50 ARDS patients in which the actual mortality was 16% when it was predicted to be 40%
  - Volume-targeted SIMV with Vt’s as low as 5ml/kg
  - PIP’s < 40 cmH2O
  - FiO2 ≤ 60
  - PEEP of 9 cmH2O (≥ 6)
  - PaCO2 averaged 60 mmHg

Ventilator Management: Inspiratory/Expiratory Timing

- Normally 1:2 to 1:4
- Prolonging inspiratory time may improve shunt in severe ventilatory failure as an alternative to PEEP
  - Usually reserved for patients with plateau pressures ≥35 cm H2O
- Understanding ventilator graphics very important
- Usually effective only with pressure control breaths

Cellular Effects: In the absence of hypoxemia, intracellular acidemia appears to be well tolerated.

Cardiovascular Effects: Increased HR, BP & stroke volume.

CNS: Variable effects, some agitation may occur.
Alternative Modes of Ventilation & Ventilator Management

Alternative Modes of Ventilation: Airway Pressure Release Ventilation (APRV)

- Time-cycled, pressure targeted ventilation that employs alternating high and low CPAP settings while allowing spontaneous ventilation.
- Transition from high to low setting (release) creates volume exchange.

Benefits of APRV

- Lower Paw for a same Vt as compared with volume-targeted modes (AC, SIMV)
- Longer inflation phase recruits the more slowly-filling alveoli
- Limited adverse effects on cardio-circulatory function
- Spontaneous breathing throughout entire ventilatory cycle may make support more tolerable.
- Decreased sedation use and near elimination of neuromuscular blockade.

APRV - Initial Settings

- Pressure High (P_{HIGH}): Current Plateau Pressure or Mean Airway Pressure
- Time high (T_{HIGH}): 4-6 seconds
- Pressure low (P_{LOW}): 0-5 cm H₂O
- Time Low (T_{LOW}): 0.3 to 1.0 second
  - Key Variable – should be short enough to prevent derecruitment but long enough to achieve an adequate Vt.
Alternate Modes of Ventilation: High Frequency Ventilation

- Defined by FDA as a ventilator that delivers more than 150 breaths/min.
- Delivers a small tidal volume, usually less than or equal to anatomical dead space volume.
- While HFV’s are frequently described by their delivery method, they are usually classified by their exhalation mechanism (active or passive).
  - Oscillator – Active Exhalation
  - Jet – Passive Exhalation
  - Percussive – Passive Exhalation

Saline-Lavaged Rabbit’s with HFOV

Multicenter Oscillator ARDS Trial (MOAT2)

- Prospective, Randomized Controlled Trial of the Cardinal 3100B High Frequency Oscillatory Ventilator for adults with ARDS
- Ten Institutions, North American Study

<table>
<thead>
<tr>
<th></th>
<th>HFOV</th>
<th>CMV</th>
</tr>
</thead>
<tbody>
<tr>
<td>APACHE 2</td>
<td>22 (6)</td>
<td>22 (9)</td>
</tr>
<tr>
<td>Sepsis</td>
<td>47%</td>
<td>47%</td>
</tr>
<tr>
<td>Pneum infection</td>
<td>19%</td>
<td>19%</td>
</tr>
<tr>
<td>Trauma</td>
<td>25%</td>
<td>25%</td>
</tr>
<tr>
<td>Other</td>
<td>25%</td>
<td>25%</td>
</tr>
</tbody>
</table>

Primary Outcome: Status at 30d

<table>
<thead>
<tr>
<th></th>
<th>HFOV</th>
<th>CMV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Died</td>
<td>37%*</td>
<td>53%</td>
</tr>
<tr>
<td>Alive + RS</td>
<td>43%**</td>
<td>22%</td>
</tr>
<tr>
<td>Alive - no RS</td>
<td>21%</td>
<td>26%</td>
</tr>
</tbody>
</table>

*P=0.098 (chi square)
**HFOV 43% on vent vs CMV 22% on vent
Overall outcome: P=0.08 (chi square)
### Alternate Techniques: Recruitment Maneuvers

**ARDS** are characterized by a loss of gas-exchange area due to collapsed and consolidated alveoli.

- Periodic application of sustained pressure intended to re-recruit collapsed alveoli.
- No standard has been established.
  - Several techniques used: 40/40 method, sigh breaths, etc...
- No strong evidence to suggest benefit.
  - LOV study showed 22% of patients undergoing a 40/40 RM had significant complications.

### Alternate Techniques: Prone Positioning

- In the supine position:
  - Abdominal contents push upward on the diaphragm and collapse lower lobes of the lung.
  - Over 40% of left lung and 15% of right lung are located under the heart and may be compressed.

- May restore FRC in the lung injured patient.
- May improve distribution of ventilation in dependent lung regions.
- Current studies suggest improvement in oxygenation, possibly affecting mortality in more severe ARDS patients.

### Alternate Techniques: Surfactant Replacement Therapy

- May be administered by aerosol or instillation.
- No evidence that surfactant improves oxygenation, ventilator days or mortality have been found with aerosol delivery.
- Small study showed a trend toward improved survival with direct instillation compared to control.

### Alternate Techniques: Tracheal Gas Sufflation

- Provides gas flow near the carina to wash-out CO2 from the large airways.
- Continuous gas flow will increase airway pressures & AutoPEEP.
- Some systems can synchronize gas flow with expiratory cycle.
- Has been shown to substantially reduce PaCO2.
Alternate Techniques: Future Considerations

- Inhaled nitric oxide
  - Short-lived improvement in oxygenation but no change in mortality
- Steroids
  - No improvement in mortality
  - Concern with their role in the development of neuromuscular disorders in critically-ill patients
- ECMO
  - Improvement in mortality
  - Limited access

Alternate Techniques: Future Considerations

- Intravenous Beta 2 Agonists
  - Early experiments have shown:
    - Increased fluid clearance from alveolar space
    - Decreased inflammation
    - Bronchodilation
- Activated protein C
  - Antithrombotic
  - Antiinflammatory
  - Granulocyte-Macrophage Colony-Stimulating Factor (GM-CSF)
  - Limits damage & enhances repair of lung cells

Summary

Lung-Protective Ventilation

- Positive-pressure ventilation is a proven, effective modality but is the cause of immense physiological derangements:
  - Redistribution of alveolar ventilation
  - Altered capillary perfusion
  - Functional changes in surfactant
  - Transcapillary fluid shifts
  - Impaired lymphatic drainage
  - Impeded venous return
- These derangements all contribute to wet, harder-to-ventilate lungs which necessitate even more aggressive positive-pressure ventilation.
Lung-Protective Ventilation

- To minimize these derangements clinical evidence supports:
  - Small $V_t$ (4-6 ml/kg IBW)
  - Optimal PEEP therapy ($p_{IPEP}$)
  - Plateau Pressure maintained below 30cmH2O

- APRV & HFOV appear beneficial but more studies are needed to assess efficacy.

- Prone Positioning and Tracheal Gas Sufflation appear promising but more studies are needed.

- Transpulmonary-Guided Ventilation through Esophageal Pressure monitoring has been shown to improve management of PEEP and Plateau pressures

Questions?