Principles and Practice: Strategies of Mechanical Ventilation for Brain Injured Patients

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Co-Director, Neurological Intensive Care
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I do not have any financial interests or relationships to disclose
Objectives

• Provide a broad update regarding mechanical ventilation in the operating room.

• Discuss the impact of acute respiratory distress in traumatic brain injury.

• Introduce new terminology to describe the severity of acute respiratory distress syndrome.

• Discuss strategies of ventilator management for patients with severe traumatic brain injury and acute respiratory failure.
Case Presentation

24 year-old male pedestrian struck by vehicle and dragged 100 feet

Obvious head injuries, combative at scene and needs an airway.

How would you intubate this patient?
How would you intubate this patient?

- *Is ketamine safe for patients with severe acute brain injury and suspected intracranial hypertension?*

- *Does succinylcholine increase ICP?*
“Ketamine can be considered 1st line agent and NO evidence to support association between succinylcholine and raised ICP”

“The Ketamine Effect on ICP in Traumatic Brain Injury
F. A. Zeiler · J. Teitelbaum · M. West · L. M. Gillman

“Oxford 2b, GRADE C recommendation that ketamine does not lead to an elevation in ICP in severe TBI, in the setting of an intubated and sedated patient.”
**Table 2: Primary and secondary endpoints and intubation condition for study patients**

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Etomidate (n=234)</th>
<th>Ketamine (n=235)</th>
<th>Difference (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SOFA_max score (mean [SD])</td>
<td>10.3 (3.7)</td>
<td>9.6 (3.9)</td>
<td>0.7 (0.0 to 1.4)</td>
<td>0.056</td>
</tr>
<tr>
<td>Δ-SOFA (median [IQR])†</td>
<td>1.5 (0 to 3)</td>
<td>1.0 (0 to 3)</td>
<td>0.5 (-1 to 1)†</td>
<td>0.20</td>
</tr>
<tr>
<td>28-day mortality (n [%)]</td>
<td>81 (35%, 29 to 41)</td>
<td>72 (31%, 25 to 37)</td>
<td>4 (-4 to 12)</td>
<td>0.36</td>
</tr>
<tr>
<td>Mechanical ventilation-free days at day 28 (median [IQR])</td>
<td>12 (0 to 25)</td>
<td>15 (0 to 26)</td>
<td>-2.4 (-9.9 to 5.7)†</td>
<td>0.36</td>
</tr>
<tr>
<td>Transfusions (n [%], 95% CI)</td>
<td>42 (18%, 13 to 23)</td>
<td>38 (15%, 11 to 21)</td>
<td>2 (-5 to 9)</td>
<td>0.62</td>
</tr>
<tr>
<td>Fluid loading (mL/kg/h; mean [SD])</td>
<td>2 (1)</td>
<td>2 (4)</td>
<td>0 (-0.7 to 0.5)</td>
<td>0.23</td>
</tr>
<tr>
<td>Catecholamine support (n [%], 95% CI)</td>
<td>137 (59%, 53 to 65)</td>
<td>120 (51%, 45 to 57)</td>
<td>7.5 (-1.5 to 16.5)†</td>
<td>0.10</td>
</tr>
<tr>
<td>Catecholamine-free days (until day 28; median [IQR])</td>
<td>37 (14 to 28)</td>
<td>28 (20 to 28)</td>
<td>-0.7 (-2.1 to 0.2)†</td>
<td>0.08</td>
</tr>
<tr>
<td>ICU-free days at day 28 (median [IQR])</td>
<td>4.0 (0 to 22)</td>
<td>6 (0 to 23)</td>
<td>-2 (-13 to 11)†</td>
<td>0.57</td>
</tr>
<tr>
<td>Glasgow outcome score (median [IQR])</td>
<td>3 (1 to 5)</td>
<td>3 (1 to 5)</td>
<td>0 (-1 to 1)†</td>
<td>0.95</td>
</tr>
</tbody>
</table>

**Intubation condition**

| IDS value (median [IQR])                              | 1 (0 to 3)        | 1 (0 to 3)       | 0 (0 to 0)†             | 0.70    |
| Difficult intubation (n [%], 95% CI)‡                | 24 (10%, 6 to 14) | 20 (9%, 5 to 13) | 2 (-4 to 7)†            | 0.52    |
| Change in arterial systolic blood pressure (mm Hg; median [IQR])§ | 5 (-1 to 30)     | 10 (-10 to 33)  | -5 (-13 to 2)‡          | 0.24    |
| Change in arterial diastolic blood pressure (mm Hg; median [IQR])¶ | 1 (-8 to 13)     | 5 (-7 to 18)    | -4 (-8 to 1)‡           | 0.18    |
| Change in SpO₂ (%) (median [IQR])§                    | 1% (0 to 6)       | 2% (0-7)        | -1 (-2 to 1)‡           | 0.98    |
| Cardiac arrest during intubation (n [%])              | 7 (3%)            | 4 (2%)          | 1.3 (-1.5 to 4.0)       | 0.36    |

SOFA\_max—the maximum value of the sequential organ failure assessment (SOFA) score during the first 3 days in intensive care. ICU—intensive care unit. IDS—intubation difficulty score. SpO₂—pulse oxygen saturation. Δ-SOFA—SOFA\_max-SOFA(admission). *Bootstrap CI for median difference. †Difficult intubation is defined as IDS≥5. §Change in arterial systolic blood pressure equals pre-intubation minus post-intubation arterial systolic blood pressure. ¶Change in arterial diastolic blood pressure equals pre-intubation minus post-intubation arterial diastolic blood pressure. |Change in SpO₂ equals post-intubation minus pre-intubation SpO₂.
On arrival to our medical center...

Intubated, *sedated*

**Neuro Exam**
- GCS 6(t)
- Pupils equal and reactive
- Withdraws all extremities

**Vital Signs**
- BP 95/45  HR 115  SaO₂ 87% on FIO₂ 1.0
- Ventilator: Volume control 650 ml  RR 12  PEEP 5
- Peak Inspiratory Pressure 43  Plateau Pressure 35

Underwent the following imaging studies:
Perioperative ultrasonography

Parasternal Long Axis

Apical 4 chamber

 Courtesy of Dr. Achi Oren-Grinberg
Role of perioperative ultrasonography

Lung ultrasound:
Pneumothorax

Lung ultrasound:
Normal

Courtesy of Dr. Achi Oren-Grinberg
What are possible immediate, life threatening injuries in this case (in addition to impending herniation)?

<table>
<thead>
<tr>
<th>Heart and Vessels</th>
<th>Diagnostic Evaluation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac tamponade</td>
<td>Clinical, echocardiography</td>
</tr>
<tr>
<td>Blunt Cardiac injury</td>
<td>Echocardiography</td>
</tr>
<tr>
<td>Aortic Disruption</td>
<td>Chest CT angiogram, echo</td>
</tr>
<tr>
<td><strong>Occult hemorrhage</strong></td>
<td>Hematocrit, angiography</td>
</tr>
<tr>
<td>Lung</td>
<td></td>
</tr>
<tr>
<td>Pneumothorax (Simple, Open, Hemothorax)</td>
<td>Chest XR, Chest CT, Chest ultrasound</td>
</tr>
<tr>
<td>Flail chest</td>
<td>Clinical, Chest XR</td>
</tr>
<tr>
<td><strong>Pulmonary contusion/pneumonitis</strong></td>
<td>Chest CT</td>
</tr>
<tr>
<td>Tracheobronchial Tree disruption</td>
<td>Chest CT</td>
</tr>
<tr>
<td>Esophageal disruption</td>
<td>Chest CT</td>
</tr>
<tr>
<td>Diaphragmatic injury</td>
<td>Chest CT</td>
</tr>
</tbody>
</table>

Adapted from Advanced Trauma Life Support for Doctors®. Student Course Manual, 8th edition
To OR:

Evacuation of left EDH
Right frontal EVD
Ex Fix left femur, Repair popliteal aa
Intraoperative → Postop Course

ICP 14 mmHg, CPP 67 mmHg

Hypotension →: Resolved following volume resuscitation and vasopressor support

Respiratory failure: Increasing peak (40 cm H$_2$O) and plateau airway pressures (32 cm H$_2$O)

STAT CXR = unchanged
Labs = ABG  pH 7.29  PaCO$_2$ 38  PaO$_2$ 77  HCT 32
Bedside echo = Normal
DOES THIS PATIENT HAVE ARDS?

<table>
<thead>
<tr>
<th>Timing</th>
<th>Acute Respiratory Distress Syndrome&lt;br&gt;(The Berlin Definition)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Within 1 week of a known clinical insult or new/worsening respiratory symptoms</td>
<td></td>
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</table>

| Chest Imaging a | Bilateral opacities – not fully explained by effusions, lobar/lung collapse, or nodules |

| Origin of Edema | 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 |

<table>
<thead>
<tr>
<th>Oxygenation b</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>200&lt;(\text{PaO}_2/\text{FiO}_2)≤ 300&lt;br&gt;with&lt;br&gt;PEEP or CPAP ≥ 5 cmH₂O c</td>
<td>100&lt;(\text{PaO}_2/\text{FiO}_2)≤200&lt;br&gt;with&lt;br&gt;PEEP ≥ 5 cmH₂O</td>
<td>(\text{PaO}_2/\text{FiO}_2)≤100&lt;br&gt;with&lt;br&gt;PEEP ≥ 5 cmH₂O</td>
<td></td>
</tr>
</tbody>
</table>

JAMA 2012; 307(23):2526-2533
Practical Solutions

• Can we translate what we know from studies involving critically ill patients to the operating room?

• Is there anything that we can do to improve this patient's outcome?

• Does the presence of severe, acute brain injury matter?
Impact of Acute Lung Injury and Acute Respiratory Distress Syndrome After Traumatic Brain Injury in the United States

Prevalence: 20-25%

Risk Factors:
- Younger patients
- Sepsis, multi-organ system failure

3X increase in odds of in-hospital mortality

Rincon F. Neurosurgery 2012; 71(4): 795-803
Respiratory Failure in TBI

Figure 1. Factors affecting the cross-talk between the brain and the lung.

Pelosi P. Critical Care 2011; 15:168
Ventilator Induced Lung Injury

- **Volutrauma** - lung injury resulting from over-distension of alveoli
  - Related to tidal volume ($V_T$)
  - Often presumed to be related to $P_{plat}$
  - Low $V_T$ is standard of care

- **Atelectrauma** - lung injury resulting from cyclic recruitment / de-recruitment of alveoli during tidal breathing
  - Related to PEEP as demonstrated in numerous animal studies
  - Ideal PEEP setting remains elusive in human trials

- **Barotrauma** - refers to pressure and is a function of transpulmonary pressure (distending pressure).

- **Biotrauma** - Above mechanisms lead to cytokine release, inflammation and further injury to alveoli.
Figure 5. Macroscopic aspect of rat lungs after mechanical ventilation at 45 cm H$_2$O peak airway pressure. *Left:* normal lungs; *middle:* after 5 min of high airway pressure mechanical ventilation. Note the focal zones of atelectasis (in particular at the left lung apex); *right:* after 20 min, the lungs were markedly enlarged and congestive; edema fluid fills the tracheal cannula.
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*
Low tidal volume ventilation: Mortality Benefit

Figure 1. Probability of Survival and of Being Discharged Home and Breathing without Assistance during the First 180 Days after Randomization in Patients with Acute Lung Injury and the Acute Respiratory Distress Syndrome.

Low Tidal Volume Ventilation Strategy

**Volumes**: 8ml/kg *ideal* body weight, then reduced to 7→6 ml/kg
- Male IBW (kg) = 50 + 2.3 (height (in) – 60)
- Female IBW (kg) = 45.5 + 2.3 (height (in) – 60)

**PEEP**: ≥5 cmH₂O, RR up to 35 (V̇e typically 7-9 L/min)

**FiO₂**: goal SaO₂ 88-92%

A Trial of Intraoperative Low-Tidal-Volume Ventilation in Abdominal Surgery

Futier E. NEJM 2013; 369:428
High versus low positive end-expiratory pressure during general anaesthesia for open abdominal surgery (PROVHILO trial): a multicentre randomised controlled trial

The PROVE Network Investigators* for the Clinical Trial Network of the European Society of Anaesthesiology

**Figure 2:** Kaplan-Meier curve showing the probability of postoperative pulmonary complications by postoperative day 5. PEEP = positive end-expiratory pressure.
What Tidal Volumes Should Be Used in Patients without Acute Lung Injury?

Marcus J. Schultz, M.D., Ph.D.,* Jack J. Haitsma, M.D., Ph.D.,† Arthur S. Slutsky, M.D.,‡ Ognjen Gajic, M.D.§

<table>
<thead>
<tr>
<th>Normal lung* and no ALI risk factors (&quot;hits&quot;)**</th>
<th>Abnormal lung* and/or presence of ALI risk factors (&quot;hits&quot;)**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tidal volume &lt; 10 mL/kg PBW</td>
<td>Tidal volume 6 mL/kg PBW</td>
</tr>
<tr>
<td>Plateau pressure§ &lt; 15 - 20 cm H₂O</td>
<td>Plateau pressure§ &lt; 15 - 20 cm H₂O</td>
</tr>
<tr>
<td>PEEP£ ≥ 5 cm H₂O</td>
<td>PEEP£ ≥ 5 cm H₂O</td>
</tr>
</tbody>
</table>

Schultz et al. Anesthesiology 2007; 106:1226–31
- Relatively low incidence of ARDS (0.2%)
- Preoperative Risk Factors:
  ASA status 3-5
  Emergent Surgery
  Renal Failure
  COPD
- Intraoperative Risk Factors:
  Driving Pressure (PIP-PEEP)
  FIO2
  Fluids and transfusion

Anesthesiology 2013; 118: 19
IMPAIRED OXYGENATION IN SURGICAL PATIENTS DURING GENERAL ANESTHESIA WITH CONTROLLED VENTILATION*

A Concept of Atelectasis

H. H. Bendixen, M.D.,† J. Hedley-Whyte, M.B., B.Chir.,‡ and M. B. Laver, M.D.§

BOSTON
High tidal volume is associated with the development of acute lung injury after severe brain injury: An international observational study*

Luciana Mascia, MD, PhD; Elisabeth Zavala, MD; Karen Bosma, MD; Daniela Pasero, MD; Daniela Decaroli, MD; Peter Andrews, MD; Donatella Isnardi, MD; Alessandra Davi, MD; Maria Jose Areu, MD; Maurizio Berardino, MD; Alessandro Ducati, MD; on behalf of the Brain IT group

Injurious mechanical ventilation affects neuronal activation in ventilated rats

María Elisa Quilez1,2, Gemma Fuster1,2, Jesús Villar1,3, Carlos Flores1,4, Octavi Martí-Sistac1,2,5, Lluís Blanch1,2 and Josefina López-Aguilar1,2*

Management and outcome of mechanically ventilated neurologic patients*

Paolo Pelosi, MD; Niall D. Ferguson, MD, MSc; Fernando Frutos-Vivar, MD; Antonio Anzueto, MD; Christian Putensen, MD; Konstantinos Raymondos, MD; Carlos Apezteguia, MD; Pablo Desmery, MD; Javier Hurtado, MD; Fekri Abrour, MD; José Elizalde, MD; Vinko Tomicic, MD; Nahit Cakar, MD; Marco Gonzalez, MD; Yaseen Arabi, MD; Rui Moreno, MD; Andres Esteban, MD, PhD; for the Ventila Study Group

Crit Care Med 2007; 35(8): 1815

Critical Care 2011; 15:R124

Crit Care Med 2011; 39: 1482
HOW DO WE BALANCE THE GOALS BETWEEN “LUNG PROTECTIVE” VENTILATION AND “NEURO PROTECTIVE” VENTILATION?
Hypercapnia and ICP

- concept of “permissive hypercapnia”
- relationship between PaCO$_2$ and CBF
- estimating intracranial compliance
Cerebral blood flow as a function of CO2

Am J Physiol Heart Circ Physiol
PaCO$_2$ and Compliance

$$\text{Compliance} = \frac{\Delta V}{\Delta P}$$

ICP (mm/Hg) vs. Intracranial volume (mL)
Strategy: PaCO$_2$ and ICP

**ARDS and ICP <20mmHg**
- monitor clinical status and ICP
- may be safe to allow PaCO$_2$ to increase to 45-55 mmHg

**ARDS and ICP >20mmHg or requiring active Rx**
- avoid hypercapnia, at expense of increasing tidal volume and minute ventilation
- goal eucapnia, PaCO$_2$ 35-40 mmHg

Lowe G. Curr Opin Crit Care 2006; 12:3-7
PEEP and ICP

function of:

respiratory system compliance
Intracranial compliance
venous return
volume status
alveolar hyperinflation
Positive end-expired pressure (PEEP) and effect on ICP: Impact of respiratory system compliance

- Increased $V_D$
- Elevated intra-thoracic pressure $\rightarrow$ Increased RAP
- And decreased MAP
Strategy: PEEP and ICP

- Consider setting PEEP < ICP

- PEEP to 15 cmH\(_2\)O likely safe

- Measure lung compliance for best PEEP & recruitment
  Mascia L. Intensive Care Med 2005; 31:373
  Caricato A. J Trauma 2005; 58: 571

- Watch hemodynamics when making vent changes
Oxygenation and TBI

- oxygenation and low tidal volume ventilation
- is there a role for prophylactic hyperoxia?
- does systemic oxygenation influence brain tissue oxygenation?
ARDSnet: 6 vs. 12 Vₜ Trial

PaO₂ / FiO₂ Ratio

Study Day

0 1 2 3 4 7 14 21

P/F

120 140 160 180 200

6 ml/kg
12 ml/kg

* *

ARDSnet. NEJM 2000; 342:1301

Courtesy of Ray Ritz, RRT
Acute Lung Injury Is an Independent Risk Factor for Brain Hypoxia After Severe Traumatic Brain Injury

**BACKGROUND:** Pulmonary complications are frequently observed after severe traumatic

**FIGURE.** Histograms of mean (standard deviation) brain tissue oxygen tension ($PbO_2$) values according to the $PaO_2/FiO_2$ ratio range. **$P < .01$** for comparisons with samples obtained at $PaO_2/FiO_2$ ratio $>300$. 

Neurosurgery 2010; 67(2):338-344
Mechanical Ventilation Guided by Esophageal Pressure in Acute Lung Injury


![Graph showing PaO2/FiO2 levels over time for different treatments.](image)
Compliance - Respiratory System ($C_{RS}$), Lung ($C_L$) & Chest Wall ($C_{CW}$)

- Compliance ($C$) = $\Delta V / \Delta P$

- $C_{RS} = C_{Lung} + C_{ChestWall}$

- $C_L = V_T / (P_{L-insp} - P_{L-exp})$

- $C_{CW} = V_T / (P_{es-insp} - P_{es-exp})$
# Diseases Affecting Compliance

<table>
<thead>
<tr>
<th>Affecting $C_L$</th>
<th>Affecting $C_{CW}$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute Respiratory Distress Syndrome (ARDS)</td>
<td>Morbid Obesity</td>
</tr>
<tr>
<td>Pulmonary Fibrosis</td>
<td>Abdominal Hypertension</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>Pleural Effusions</td>
</tr>
<tr>
<td>Aspiration</td>
<td>Dystonia / Hypertonia</td>
</tr>
<tr>
<td>COPD</td>
<td>Burns / Radiation</td>
</tr>
</tbody>
</table>
Strategy: Oxygenation and TBI

- Hyperoxia does not improve outcome

  Reinert M. Acta Neurochir 2003; 145:341
  Magnono S. J Neurosurg 2003;98:952

- Unclear if ARDSnet PEEP/FIO2 tables apply to brain injury

- Target “reasonable” oxygen saturation of 91-94%

- Cerebral microdialysis or brain tissue oxygenation potentially useful when making ventilator changes
Sedation
Sedation for tolerance of ventilator settings may impair neurologic exam

- Low tidal volume ventilator strategy is uncomfortable and typically requires sedation for tolerance
- Commonly believed that higher levels of sedation are required, which may impede frequent neurologic exams
- Several trials have reported similar sedation practice between the low tidal volume and “conventional” groups

Crit Care Med 2005; 33:766-771
Intensive Care Med 2014;40(7):950-7
Management Strategies

Adapted from Intensive Care Med 2012; 38:1582

Intensity of intervention

P/F ratio

300 200 100 50

Mild Moderate Severe

Low tidal volume (6cc/kg PBW)

“Physiologic” PEEP

Consider paralysis

Rescue

ECMO

??Restrictive fluid management strategy ??

ARDSnet PEEP
Prolonged heparin-free extracorporeal membrane oxygenation in multiple injured acute respiratory distress syndrome patients with traumatic brain injury

Ralf M. Muellenbach, MD, Markus Kredel, MD, Ekkehard Kunze, MD, Peter Kranke, MD, Julian Kuestermann, MD, Alexander Brack, MD, Armin Gorski, MD, Christian Wunder, MD, Norbert Roewer, MD, and Thomas Wurmb, MD, Würzburg, Germany
The Effect of APRV Ventilation on ICP and Cerebral Hemodynamics

Paul E. Marik · Alisha Young · Steve Sibole · Alex Levitov
Lung protective ventilation (ARDSNet) versus airway pressure release ventilation: Ventilatory management in a combined model of acute lung and brain injury

**TABLE 4. Cerebral Microdialysis Biomarkers Following Lung and Brain Injury**

<table>
<thead>
<tr>
<th>Variables</th>
<th>30 min, Mean (SD)</th>
<th>1.5 h, Mean (SD)</th>
<th>2.5 h, Mean (SD)</th>
<th>3.5 h, Mean (SD)</th>
<th>4.5 h, Mean (SD)</th>
<th>5.5 h, Mean (SD)</th>
<th>6.5 h, Mean (SD)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Glucose</strong>**</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APRV</td>
<td>1.8 (1.2)</td>
<td>0.84 (0.65)</td>
<td>0.23 (0.071)</td>
<td>TSTD</td>
<td>TSTD</td>
<td>TSTD</td>
<td>TSTD</td>
<td>—</td>
</tr>
<tr>
<td>ARDSNet</td>
<td>1.4 (1.3)</td>
<td>1.8 (1.5)</td>
<td>2.4 (1.1)</td>
<td>3.9 (0.20)</td>
<td>4.3 (1.1)</td>
<td>2.4 (0.10)</td>
<td>2.2 (0.66)</td>
<td></td>
</tr>
<tr>
<td><strong>Lactate</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>APRV</td>
<td>4.5 (2.0)</td>
<td>5.6 (2.9)</td>
<td>6.9 (1.9)</td>
<td>6.5 (1.4)</td>
<td>6.2 (0.60)</td>
<td>6.5 (1.1)</td>
<td>6.6 (1.2)</td>
<td>0.013†</td>
</tr>
<tr>
<td>ARDSNet</td>
<td>5.1 (1.8)</td>
<td>4.7 (2.9)</td>
<td>8.0 (6.0)</td>
<td>7.9 (6.2)</td>
<td>3.8 (2.9)</td>
<td>4.6 (2.9)</td>
<td>7.8 (5.3)</td>
<td></td>
</tr>
<tr>
<td><strong>Glycerol</strong>**</td>
<td></td>
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<td></td>
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<tr>
<td>APRV</td>
<td>80 (41)</td>
<td>63 (26)</td>
<td>102 (118)</td>
<td>156 (254)</td>
<td>185 (292)</td>
<td>355 (454)</td>
<td>262 (403)</td>
<td>—</td>
</tr>
<tr>
<td>ARDSNet</td>
<td>154 (139)</td>
<td>123 (96)</td>
<td>143 (86)</td>
<td>175 (84)</td>
<td>113 (140)</td>
<td>111 (134)</td>
<td>209 (197)</td>
<td></td>
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<tr>
<td><strong>Pyruvate</strong></td>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>APRV</td>
<td>136 (67)</td>
<td>130 (70)</td>
<td>102 (110)</td>
<td>147 (148)</td>
<td>126 (153)</td>
<td>68 (38)</td>
<td>96 (56)</td>
<td>0.051†</td>
</tr>
<tr>
<td>ARDSNet</td>
<td>164 (45)</td>
<td>107 (35)</td>
<td>300 (283)</td>
<td>95 (52)</td>
<td>106 (73)</td>
<td>142 (99)</td>
<td>191 (186)</td>
<td></td>
</tr>
<tr>
<td><strong>Lactate/Pyruvate</strong></td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>APRV</td>
<td>35 (10)</td>
<td>58 (44)</td>
<td>174 (132)</td>
<td>106 (82)</td>
<td>145 (115)</td>
<td>124 (69)</td>
<td>93 (52)</td>
<td>0.16†</td>
</tr>
<tr>
<td>ARDSNet</td>
<td>30 (9.0)</td>
<td>39 (17)</td>
<td>41 (21)</td>
<td>105 (130)</td>
<td>66 (81)</td>
<td>68 (83)</td>
<td>83 (105)</td>
<td></td>
</tr>
</tbody>
</table>

*Interaction between group (ARDSNet vs. APRV) and time was tested using a mixed-effects repeated-measures model. Normality and heteroscedasticity were checked using multivariable log-normal; p < 0.05.
**Insufficient data to compute likelihood function.
†When appropriate, a stabilizing transformation was applied to the data (e.g., logarithm).
Continuous data were analyzed using either Student’s *t* test or Wilcoxon rank-sum, depending on the normality of distribution.
TSTD, too small to determine.
Does Prone Positioning Increase Intracranial Pressure? A Retrospective Analysis of Patients with Acute Brain Injury and Acute Respiratory Failure

Christian Roth · Andreas Ferbert · Wolfgang Deinsberger · Jens Kleffmann · Stefanie Kästner · Jana Godau · Marc Schüler · Michael Tryba · Markus Gehling

Fig. 1 Mean intracranial pressure (ICP) of 119 different episodes of prone positioning shows a significant increase compared to supine positioning.

Neurocrit Care (2014) 21:186–191
Back to the 24 year old with severe TBI, Left femur fx and ARDS

We attributed his ARDS to aspiration pneumonitis, pulmonary contusions

Monitored ICP with EVD

Decreased tidal volume to 6 ml/Kg PBW to keep $P_{pl} < 30$

RR increased to 20, near normal $PaCO_2$

Increased PEEP to 12 cmH$_2$O based on best compliance

Reduced FIO2 from 1→.4 for $O_2$ sat >92%
Practical Solutions?

Mild Hypercarbia probably safe for ICP <20 mmHg

PEEP < ICP, measure lung compliance

Monitoring brain tissue oxygenation for patients with low P/F ratio

Limit sedation for tolerance of ventilator (in the ICU)
Conclusions

ARDS is common in severe TBI and increases mortality

Both intracranial and respiratory system compliance are important considerations when adjusting the ventilator in brain-injured patients

Multi-modal neurologic monitoring may improve safety during lung recruitment
Thank you