PEDIATRIC ARDS: What works, what doesn’t?

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DISCLOSURE STATEMENT

• I have no conflicts of interest to disclose
OUTLINE

- History of ARDS
- Pathology of ARDS
- Physiology of ARDS
- Diagnosing ARDS in pediatric patients
- Management interventions that help
- Management interventions that don’t help
ACUTE RESPIRATORY DISTRESS SYNDROME

• Acute, diffuse, inflammatory lung injury
  – Hypoxemia
  – Radiographic opacities
  – Diffuse alveolar damage
  – Non-cardiogenic
CASE

- 16 yo M with epilepsy, autism, OSA
- Seizure in shower
- Bystander CPR
- OSH Course
  - Aspiration of gastric contents
  - Difficult intubation
  - Bronch: copious gastric contents suctioned
  - O2 sat 60’s-80’s on FiO2 100%
  - ABG: 7.18/53/78/-9, lactate 12
  - hypotensive
INITIAL CXR
INITIAL PICU COURSE

• PaO2 in 50’s on 100% FiO2, PEEP 16, MAP 21
  – P/F: 56
  – OI: 41

• Sedation, neuromuscular blockade, vasoactive infusions
ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

• First described in World War II and Vietnam War
  – “shock lung”
  – “Noncardiogenic pulmonary edema”
  – “wet lung”
  – “white lung”
  – “Da Nang lung”
ACUTE RESPIRATORY DISTRESS IN ADULTS

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Summary The respiratory-distress syndrome in 12 patients was manifested by acute onset of tachypnoea, hypoxaemia, and loss of compliance after a variety of stimuli; the syndrome did not respond to usual and ordinary methods of respiratory therapy. The clinical and pathological features closely resembled those seen in infants with respiratory distress and to conditions in congestive atelectasis and postperfusion lung. The theoretical relationship of this syndrome to alveolar surface active agent is postulated. Positive end-expiratory pressure was most helpful in combating atelectasis and hypoxaemia. Corticosteroids appeared to have value in the treatment of patients with fat-embolism and possibly viral pneumonia.
<table>
<thead>
<tr>
<th>Case</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Illness</th>
<th>Onset of acute respiratory distress (hr. after illness)</th>
<th>Possible contributory factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>29</td>
<td>M</td>
<td>Multiple trauma; lung contusion</td>
<td>8</td>
<td>+ + 7500 ml.</td>
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<tr>
<td>2</td>
<td>19</td>
<td>F</td>
<td>Multiple trauma; lung laceration and contusion</td>
<td>1</td>
<td>+ + + 3000 ml.</td>
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<tr>
<td>3</td>
<td>19</td>
<td>F</td>
<td>Multiple trauma and fractures; fat-embolism</td>
<td>72</td>
<td>+ .. ..</td>
</tr>
<tr>
<td>4</td>
<td>25</td>
<td>M</td>
<td>Shotgun wound to abdomen</td>
<td>96</td>
<td>+ + + 9000 ml.</td>
</tr>
<tr>
<td>5</td>
<td>11</td>
<td>M</td>
<td>Blunt chest injury; lung contusion</td>
<td>1</td>
<td>.. + + ..</td>
</tr>
<tr>
<td>6</td>
<td>43</td>
<td>F</td>
<td>Acute pancreatitis</td>
<td>48</td>
<td>+ + + + + + + + + + + + 5000 ml.</td>
</tr>
<tr>
<td>7</td>
<td>23</td>
<td>F</td>
<td>? viral pneumonia</td>
<td>48</td>
<td>.. .. ..</td>
</tr>
<tr>
<td>8</td>
<td>39</td>
<td>F</td>
<td>Drug ingestion; ? viral pneumonia</td>
<td>24</td>
<td>.. .. + +</td>
</tr>
<tr>
<td>9</td>
<td>19</td>
<td>F</td>
<td>Guillain-Barre; ? viral pneumonitis</td>
<td>96</td>
<td>.. .. + +</td>
</tr>
<tr>
<td>10</td>
<td>18</td>
<td>M</td>
<td>Multiple trauma; crushed chest; severe concussion</td>
<td>1</td>
<td>.. .. ..</td>
</tr>
<tr>
<td>11</td>
<td>48</td>
<td>F</td>
<td>Drug ingestion; ? aspiration; ? viral pneumonia</td>
<td>48</td>
<td>.. .. + + + + + + 10328 ml.</td>
</tr>
<tr>
<td>12</td>
<td>34</td>
<td>M</td>
<td>Gunshot wound left chest</td>
<td>96</td>
<td>.. .. ..</td>
</tr>
</tbody>
</table>
DEADLY LUNG AILMENT HAS BATTLEFIELD ORIGINS

By LAWRENCE K. ALTMAN, M.D.
Published: April 9, 1985

LOUISVILLE, Ky., April 8— It is an often fatal lung condition that strikes suddenly and unexpectedly, often among healthy people, producing calamitous breathing problems. Doctors first recognized it when treating battlefield wounded in recent wars.

Now doctors diagnose the problem perhaps as many as 100,000 times a year among American civilians who suffer it as a complication while recovering from accidents, serious infections or operations. Two of the most recent cases were artificial heart patients.

The condition stiffens the lungs, fills them with water and causes shortness of breath from respiratory failure. It is called adult respiratory distress syndrome, or ARDS, and it was invariably fatal when doctors first became aware of it under different names in World War II and in the Vietnam War.
ETIOLOGY

- Direct lung injury
  - Pneumonia
  - Aspiration
  - Drowning

- Secondary to a non-pulmonary insult
  - Sepsis
  - Burns
  - Non-pulmonary trauma
PATHOLOGIC PHASES

• Acute Exudative Phase

• Subacute Proliferative Phase

• Fibrosis
Time course of acute respiratory distress syndrome (ARDS)

Schematic representation of the time course of the acute respiratory distress syndrome (ARDS). During the early (or exudative) phase, the lesion is characterized by high permeability pulmonary edema followed by the formation of hyaline membranes. After seven to ten days, a proliferative phase may develop, with marked interstitial inflammation, fibrosis, and disordered healing.

ACUTE EXUDATIVE PHASE

• First week
  – Capillary-alveolar barrier injury
    • Damage to type I pneumocytes
  – Development of protein-rich noncardiogenic pulmonary edema
  – Neutrophil activation and alveolar infiltration
  – Hyaline membrane formation
  – Pulmonary HTN
  – Surfactant dysfunction
    • Damage to type II pneumocytes

• Clinically:
  – pulmonary edema, atelectasis, IPS, hypoxia, SIRS
SUBACUTE PROLIFERATIVE PHASE

• 7-10 days into course
  – Fibroblast proliferation
  – Ongoing inflammation
  – Widening of alveolar septae due to cellular proliferation and organization of hyaline membrane
  – Worsening pulmonary HTN

• Clinically:
  – Ventilation impaired due to increasing dead space, improved SIRS
FIBROSIS

Normal lung

High power photomicrograph shows alveoli containing capillaries within a narrow interstitium. The alveoli are lined with thin, elongated type I pneumocytes (arrow) and smaller numbers of cuboidal type II pneumocytes (dashed arrow).

Late diffuse alveolar damage

High power photomicrograph shows changes typical of the proliferative or late stage of diffuse alveolar damage. Although hyaline membranes are still identifiable, the histologic picture is now dominated by thickening and reorganization of interstitial structures due mainly to marked proliferation of mesenchymal spindle cells, including both fibroblasts and myofibroblasts.

Courtesy of Jeffrey L Myers, MD.
PHYSIOLOGICAL EFFECTS
An imbalance of forces across the pulmonary capillary walls can lead to interstitial and then alveolar pulmonary edema.
• Disruption of alveolar-endothelial barrier
  – Protein-rich fluid fills alveoli
  – Diminishes effectiveness of surfactant to reduce surface tension
  – Alveolar collapse
  – Further edema
  – Reduced lung compliance
  – Reduced FRC
NORMAL LUNG COMPLIANCE

“Optimal PEEP for open lung strategy ventilation in ARDS.” Derangedphysiology.com
COMPLIANCE IN ARDS
V/Q MISMATCH

\[ P_{A\text{CO}_2} \text{ (mm Hg)} \]

\[ P_{A\text{O}_2} \text{ (mm Hg)} \]

\[ V/Q = 0 \]
R-L Shunt

\[ V/Q = 1 \]
Normal

\[ V/Q = \infty \]
Dead Space

pathwaymedicine.org/ventilation-perfusion-ratio
DIAGNOSIS OF PEDIATRIC ARDS
ISSUES WITH ADULT DEFINITIONS

• Reliance on PaO2
• Reliance on mechanical ventilation
• PaO2/FiO2 ratio does not address vent management
Pediatric Acute Respiratory Distress Syndrome: Consensus Recommendations From the Pediatric Acute Lung Injury Consensus Conference

The Pediatric Acute Lung Injury Consensus Conference Group

<table>
<thead>
<tr>
<th>Age</th>
<th>Exclude patients with peri-natal related lung disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Timing</td>
<td>Within 7 days of known clinical insult</td>
</tr>
<tr>
<td>Origin of Edema</td>
<td>Respiratory failure not fully explained by cardiac failure or fluid overload</td>
</tr>
<tr>
<td>Chest Imaging</td>
<td>Chest imaging findings of new infiltrate(s) consistent with acute pulmonary parenchymal disease</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Oxygenation</th>
<th>Non Invasive mechanical ventilation</th>
<th>Invasive mechanical ventilation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PARDS (No severity stratification)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mild</td>
</tr>
<tr>
<td></td>
<td>Full face-mask bi-level ventilation</td>
<td>4 ≤ OI &lt; 8</td>
</tr>
<tr>
<td></td>
<td>or CPAP ≥5 cm H₂O</td>
<td>8 ≤ OI &lt; 16</td>
</tr>
<tr>
<td></td>
<td>PF ratio ≤ 300</td>
<td>OI ≥ 16</td>
</tr>
<tr>
<td></td>
<td>SF ratio ≤ 264</td>
<td></td>
</tr>
</tbody>
</table>

| OI ≥ 16                                      |                                 |

<table>
<thead>
<tr>
<th>Special Populations</th>
<th></th>
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</thead>
<tbody>
<tr>
<td>Cyanotic Heart Disease</td>
<td>Standard Criteria above for age, timing, origin of edema and chest imaging with an acute deterioration in oxygenation not explained by underlying cardiac disease.</td>
</tr>
<tr>
<td>Chronic Lung Disease</td>
<td>Standard Criteria above for age, timing, and origin of edema with chest imaging consistent with new infiltrate and acute deterioration in oxygenation from baseline which meet oxygenation criteria above.</td>
</tr>
<tr>
<td>Left Ventricular dysfunction</td>
<td>Standard Criteria for age, timing and origin of edema with chest imaging changes consistent with new infiltrate and acute deterioration in oxygenation which meet criteria above not explained by left ventricular dysfunction.</td>
</tr>
</tbody>
</table>

**Figure 2.**
Pediatric acute respiratory distress syndrome definition. OI = oxygenation index, OSI = oxygen saturation index. Use Pao₂-based metric when available. If Pao₂ not available, wean Fio₂ to maintain Spo₂ ≤ 97% to calculate OSI or oxygen saturation/Fio₂ ratio. For nonintubated patients treated with supplemental oxygen or nasal modes of noninvasive ventilation, see Figure 3 for at-risk criteria. Acute respiratory distress syndrome severity groups stratified by OI or OSI should not be applied to children with chronic lung disease who normally receive invasive mechanical ventilation or children with cyanotic congenital heart disease. OI = (Fio₂ × mean airway pressure × 100)/Pao₂. OSI = (Fio₂ × mean airway pressure × 100)/Spo₂.
OXYGENATION INDEX

• OI = (MAP X %FiO2)/PaO2
  – > 16 severe ARDS
  – 25-40 Consider transfer for ECMO
  – > 40 Consider ECMO

• Oxygenation Saturation Index
  – OSI = (MAP x %FiO2)/SpO2
    • Wean FiO2 for sat <97%
INTERVENTIONS THAT WORK
INTERVENTIONS THAT HELP

• Protective/open lung strategy

• Improving oxygen delivery, decreasing oxygen consumption

• Optimize fluid balance
• Always use cuffed ETTs!
  – For all pediatric patients intubated for any reason
VENTILATOR MANAGEMENT

• Maximize PEEP
  – often require 10-15 cm H2O
  – Alveolar recruitment
  – Increases FRC
  – Decreases shear forces

• Minimize VILI
  – Small tidal volume (3-6 ml/kg) and low rates
  – Permissive hypercarbia
    • goal arterial pH >7.20
MAXIMIZING PEEP

• In volume controlled mode
  – Increase in PEEP → increase in PIP less than increase in PEEP until overdistension occurs

• In pressure controlled mode
  – Increase in PEEP → increased tidal volume until overdistension
OXYGEN DELIVERY/CONSUMPTION

• Improve oxygen delivery ($DO_2$)
  – Correct anemia
  – Correct low cardiac output

• Minimize oxygen consumption ($VO_2$)
  – Treating fever
  – Minimize pain
  – Adequate sedation
Comparison of Two Fluid-Management Strategies in Acute Lung Injury

The National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network*

ABSTRACT
INTERVENTIONS THAT SEEM LIKE THEY SHOULD HELP – BUT DON’T
INTERVENTIONS THAT DON’T HELP (IN STUDIES)

• Interventions that can’t be routinely recommended:
  – Mode of ventilation
  – HFOV
  – iNO
  – Prone positioning

• Interventions that really don’t work:
  – Corticosteroids
  – Exogenous surfactant
  – Prostaglandin therapy
MODE OF VENTILATION
Pressure-controlled versus volume-controlled ventilation for acute respiratory failure due to acute lung injury (ALI) or acute respiratory distress syndrome (ARDS)

Binila Chacko¹, John V Peter¹, Prathap Tharyan², George John¹, Lakshmanan Jeyaseelan³

¹Medical Intensive Care Unit, Christian Medical College & Hospital, Vellore, India. ²Cochrane South Asia, Prof. BV Moses Center for Evidence-Informed Health Care and Health Policy, Christian Medical College, Vellore, India. ³Department of Biostatistics, Christian Medical College, Vellore, India

Cochrane Database of Systematic Reviews 2015, Issue 1. Art. No.: CD008807.
Pressure-Controlled vs Volume-Controlled Ventilation in Acute Respiratory Failure
A Physiology-Based Narrative and Systematic Review

Nuttapol Rittayamai, MD; Christina M. Katsios, MD; François Beloncle, MD; Jan O. Friedrich, MD, PhD; Jordi Mancebo, MD; and Laurent Brochard, MD

CHEST 2015; 148(2):340-355
Original Investigation

Comparison of High-Frequency Oscillatory Ventilation and Conventional Mechanical Ventilation in Pediatric Respiratory Failure

Punkaj Gupta, MBBS; Jerril W. Green, MD; Xinyu Tang, PhD; Christine M. Gall, DrPHc; Jeffrey M. Gossett, MS; Tom B. Rice, MD; Robert M. Kacmarek, PhD, RRT; Randall C. Wetzel, MBBS

Published online January 20, 2014.
NITRIC OXIDE (iNO)

• Pulmonary HTN common

• Studies show temporary improvement in SpO2
  – Not sustained
  – No effect on outcome
ECMO

- Consider when lung protective strategies result in inadequate gas exchange
- Cause is reversible or patient suitable for lung transplant

CASE #2

- 4 week old ex-33 week twin
- Both twins home for 10 days
- Both developed cough, decreased PO intake, “funny breathing”
- RSV positive
- Presented to OSH and placed on NCPAP
- Arrival to UVMMC required intubation for apnea
PICU COURSE

- HD 7 worsened
- Hypercarbia to pCO2 of 70’s
- Desaturation despite FiO2 100%
- OI = 26
- No response to iNO trial
- No difference in VC vs PC
CXR HD 7
CASE #2

- Transferred for ECMO
- VA ECMO x 27 days
- Total intubation = 60 days

SUMMARY

• Diagnosis of Pediatric ARDS can be made by OI or OSI
• Vent strategy should focus on:
  – maximizing recruitment with PEEP
  – Minimizing VILI with low tidal volume, permissive hypercarbia
• Some ancillary treatment can be considered on case-by-case basis
• Anticipate need for ECMO
REFERENCES


THANK YOU