3rd Annual EMS Medical Directors’ Conference
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@INDTrauma   #EMSMDCConf2016
Inhalational Burns

Dr. W. Graham Carlos
Inhalational Burns

Indiana State Dept. of Health
3rd Annual EMS Medical Directors’ Conference
W. Graham Carlos, MD
August 26, 2016
Disclosures

None.
Goals and Objectives

1. Understand the epidemiology of smoke inhalation injury
2. Apply tenets of thermal injury diagnosis and pathophysiology
3. Understand inhalation injury complications
4. Analyze the basics of pharmacologic management
Smoking-Related Home Oxygen Burn Injuries: Continued Cause for Alarm

William G. Carlos, Mary S. Baker, Katie A. McPherson, Gabriel T. Bosslet, Rajiv Sood, Alexia M. Torke

Divisions of Pulmonary/Critical Care/Allergy and Occupational Medicine and Plastic Surgery, Department of Medicine, Indiana University School of Medicine, Charles Warren Fairbanks Center for Medical Ethics, Indiana University Health, and Richard M. Fairbanks Burn Center, Eskenazi Health, Indianapolis, Ind., USA
Findings

- Single-center retrospective study at Eskenazi Hospital studied burn injury patients related to home oxygen use$^2$
  - 4 years
  - 55 patients admitted to BURN unit
  - Hospital mortality rate was 14.5%
  - Hospital LOS ~8days
  - Concomitant substance abuse in 27% of patients
Audience Participation Poll

Q:
What percentage of home oxygen users continue to smoke while on oxygen?

Image courtesy of www.fotosearch.com
Audience Participation Poll

Q:
What percentage of home oxygen users continue to smoke while on oxygen?

A:
Ranging from 14-51% (Linford, et al.)
Burn Epidemiology

• Estimated **265,000 deaths/year worldwide** due to burns and inhalation
  – American Burn Association reported **3,275** fire/smoke inhalation-related deaths in 2016 (ABA)

• From 2005 – 2014, **73%** of U.S. inpatient burn admissions were related to residential fires.

• 49% of the 790 injured survivors from the WTC attacks developed inhalation injury.
Smoke Inhalation in the 3rd World

“Today, nearly half the world’s population - close to 3 billion people – will eat meals cooked over fires that use charcoal, wood, or even animal waste for fuel. A year from now, 1.9 million of those people will be dead. Their death certificates will cite pneumonia, lung cancer or tuberculosis, but the underlying cause is exposure to cooking smoke.”

www.unfoundation.org
Prognosis and Outcomes

• Systematic review of 13 cohort studies revealed a mortality rate of 13.9% in burn patients but 27.6% specifically with thermal inhalation injury⁴

• Strongest predictors of mortality in burn patients⁴:
  • Increased % Total Body Surface Area affected
  • Presence of smoke inhalation injury
  • Advanced age
  • Other less closely associated predictors include burn depth, comorbid conditions, etc.
Higher %TBSA = Inhalation Injury

Figure 1 Relationship between burn size and incidence of inhalation injury illustrates the rise in occurrence of inhalation injury with increasing burn size [5].

DJ Dries and FW Endorf, 2013
Pathophysiology of Inhalation Injury

- **Upper Airway Injury**
  - Airway temps in a fire reach 1000°F but is dissipated in the airway
  - Massive swelling of oropharyngeal structures within hours

- **Tracheobronchial Tree Injury**
  - Bronchospasm
  - Formation of pseudomembranous airway casts

- **Lung Parenchyma Damage**
  - Alveolar destruction and atelectasis due to transvascular fluid flux and loss of surfactant
  - Activated neutrophils cause direct cell damage via proteases/free radicals

- **Systemic Effects**
  - Cyanide and Carbon monoxide toxicity
  - Diffuse inflammatory response
Airway Changes in Thermal Injury

# More Than the Heat: Chemical Breakdown of Smoke

<table>
<thead>
<tr>
<th>Compound</th>
<th>Source</th>
<th>Clinical Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arolein, propenal</td>
<td>Textiles, wall coverings, upholstery, cellulose-based materials</td>
<td>Marked upper respiratory tract necrosis. Rapid death with concentrations &gt;50ppm</td>
</tr>
<tr>
<td>Aldehydes</td>
<td>Textiles, wall coverings upholstery, household goods</td>
<td>Corrosive, denatures proteins</td>
</tr>
<tr>
<td>Carbon monoxide</td>
<td>Anything combustible</td>
<td>Organ failure, death with concentrations of &gt;80-90%</td>
</tr>
<tr>
<td>Hydrogen chloride</td>
<td>Polyester, PVC materials</td>
<td>Tracheobronchitis</td>
</tr>
<tr>
<td>Hydrogen cyanide</td>
<td>Polymeric materials (-amide, -amine, -urethanes)</td>
<td>Tissue hypoxia. Death with concentrations &gt; 1ug/mL</td>
</tr>
<tr>
<td>Hydrogen sulfide</td>
<td>Rubber, silk, wool</td>
<td>Local irritant</td>
</tr>
</tbody>
</table>

Rehberg S, et al. (2009)
Assessing Severity of Airway

• First, rely on history/setting and physical exam findings
  – Singed facial hair, carbonaceous deposits on skin/mouth most likely to predict need for intubation

• Major challenges previously in stratifying severity

Table 1. Abbreviated Injury Score (AIS) bronchoscopic gradation of inhalation injury

<table>
<thead>
<tr>
<th>Grade</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 0 (no injury)</td>
<td>Absence of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction</td>
</tr>
<tr>
<td>Grade 1 (mild injury)</td>
<td>Minor or patchy areas of erythema, carbonaceous deposits in proximal or distal bronchi (any or combination)</td>
</tr>
<tr>
<td>Grade 2 (moderate injury)</td>
<td>Moderate degree of erythema, carbonaceous deposits, bronchorrhea, with or without compromise of the bronchi (any or combination)</td>
</tr>
<tr>
<td>Grade 3 (severe injury)</td>
<td>Severe inflammation with friability, copious carbonaceous deposits, bronchorrhea, bronchial obstruction (any or combination)</td>
</tr>
<tr>
<td>Grade 4 (massive injury)</td>
<td>Evidence of mucosal sloughing, necrosis, endoluminal obliteration (any or combination)</td>
</tr>
</tbody>
</table>

Look Worse = Do Worse

Endorf, et al. (2007)

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (Grades 0 and 1)</th>
<th>Group 2 (Grades 2, 3, 4)</th>
<th>( P ) Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>mL/kg/%TBSA</td>
<td>6.6 (±0.7)</td>
<td>6.7 (±0.4)</td>
<td>.88</td>
</tr>
<tr>
<td>Ventilator days</td>
<td>8.6 (±1.4)</td>
<td>12.8 (±2.2)</td>
<td>.11</td>
</tr>
<tr>
<td>Survival</td>
<td>21 (84%)</td>
<td>20 (57%)</td>
<td>.03</td>
</tr>
<tr>
<td>Initial compliance</td>
<td>49.9 (±4.4)</td>
<td>49.7 (±3.1)</td>
<td>.98</td>
</tr>
<tr>
<td>Initial P:F Ratio</td>
<td>371.5 (±32)</td>
<td>329.7 (±29)</td>
<td>.33</td>
</tr>
</tbody>
</table>
Bronchoscopy in Smoke Inhalation

Post Inhalation, Day 0

Post Inhalation, Day 4
Cast formation

Airway cast

Inhaled Therapies

• **Bronchodilators**
  – Ovine model of tiotropium demonstrated improved P/F ratio and decreased peak pressures 24h after injury\(^{12}\)
  – Beta-agonists also helpful and may decrease inflammatory response
  – Epinephrine shown to decrease TNF levels, potentiate IL-10\(^{12}\)

• **Mucolytic Agents**
  – N-acetylcysteine can thin secretions and may have a role in ameliorating effects of free radicals\(^{5,12}\)
  – Often dosed with heparin, pre-dosed with a beta-agonist

• **Anti-inflammatory and Anticoagulation Agents**
  – Heparin protocol q2 (alternate w/ NAC/Albuterol)
Nebulized Heparin Review

- Human studies demonstrated overall decrease in lung injury scores/mortality with nebulized heparin\(^7\)
- No change in duration of ventilation with heparin protocols, but one study was associated with decreased re-intubation\(^7\)
- No systemic bleeding risks noted\(^7\)

### TABLE 3. Summary of the Pathophysiological and Clinical Effects of Nebulized Heparin Regimens in Human Clinical Studies of Smoke Inhalation-Associated Acute Lung Injury

<table>
<thead>
<tr>
<th>Reference</th>
<th>Lung Injury Score</th>
<th>Pneumonia Prevalence</th>
<th>Mechanical Ventilation Duration</th>
<th>Unplanned Reintubation</th>
<th>Hospital Length of Stay</th>
<th>Bleeding Risk</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Desai et al (2)</td>
<td>↓</td>
<td>↓</td>
<td>No change</td>
<td>↓</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Rivero et al (48)</td>
<td>↓</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Holt et al (50)*</td>
<td>No change</td>
<td></td>
<td></td>
<td>No change</td>
<td>No change</td>
<td></td>
<td></td>
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<tr>
<td>Miller et al (27)</td>
<td>↓</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Yip et al (49)</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>No change</td>
</tr>
</tbody>
</table>

*No randomization or allocation into treatment groups. Patients treated at attending physician discretion with a dosing regimen half the strength of the studies by Rivero et al (48) and Miller et al (27).*
References


Questions?

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