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Inhalational Burns

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3rd Annual EMS Medical Directors' Conference

W. Graham Carlos, MD

August 26, 2016

SCHOOL OF MEDICINE

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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

Clinical Investigations

Respiration

Respiration
DOI: 10.1159/000443798

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Smoking-Related Home Oxygen Burn Injuries: Continued Cause for Alarm

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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

Image courtesy of www.fotosearch.com

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

Audience Participation Poll

Image courtesy of www.fotosearch.com

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.[10]

Smoke Inhalation in the 3rd World

www.unfoundation.org

"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."

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 injury[4]

- Strongest predictors of mortality in burn patients[4]:
  - 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

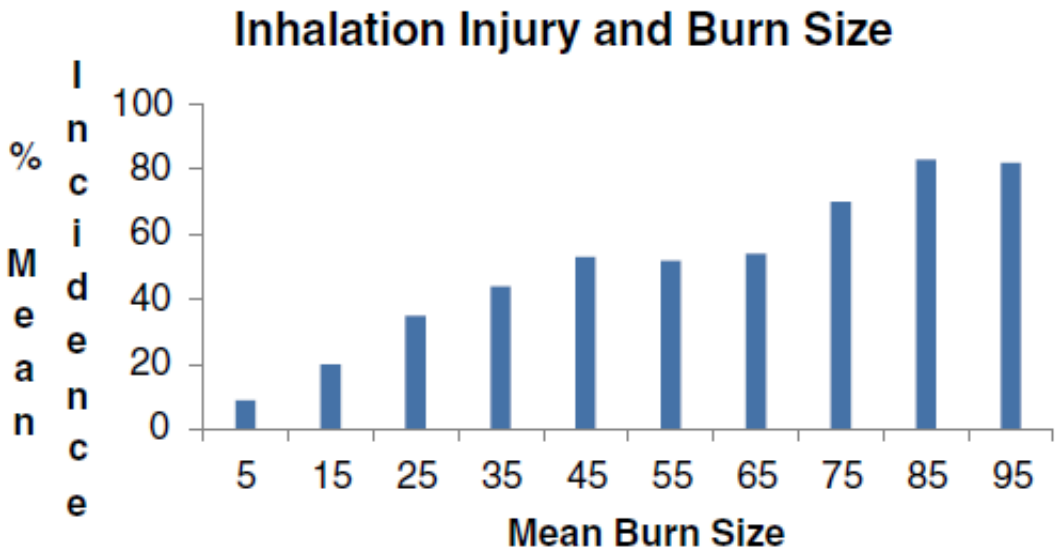


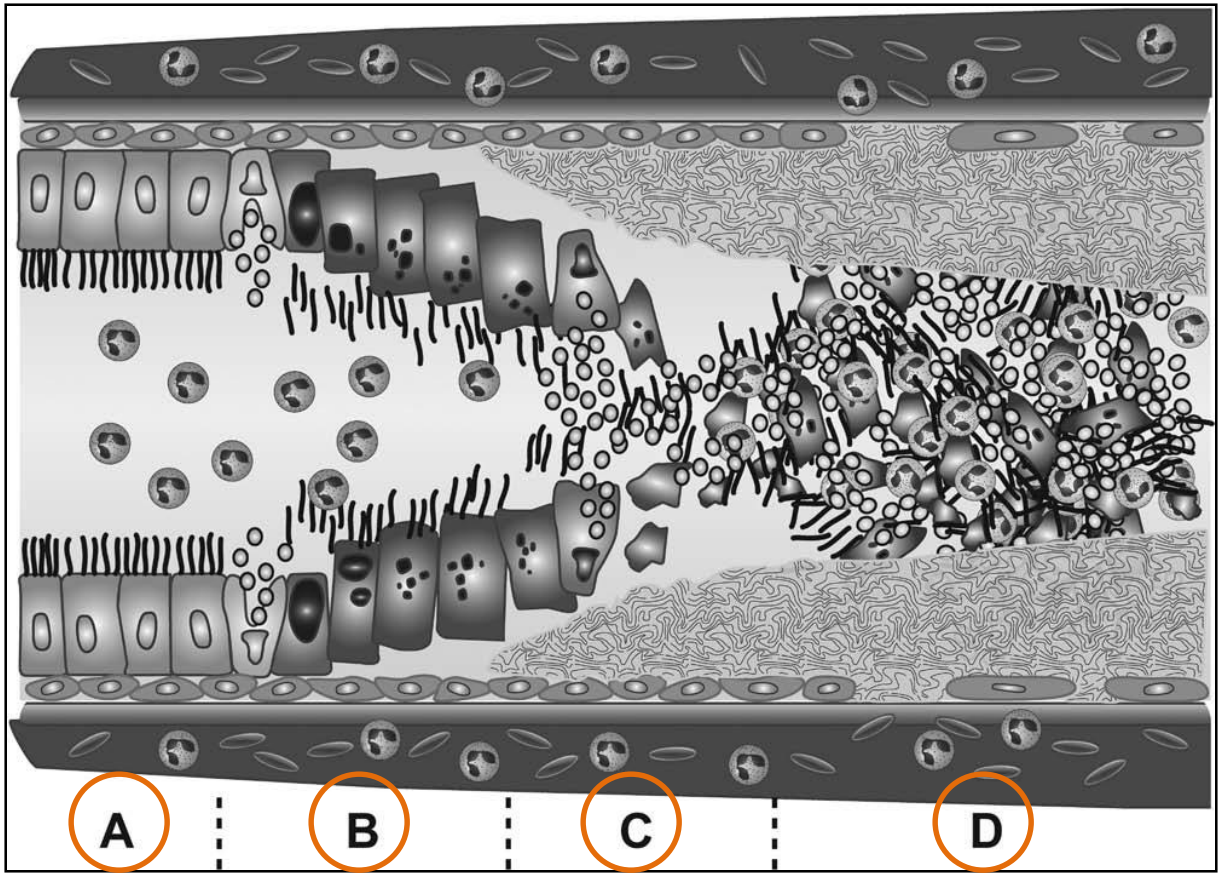
Figure 1 Relationship between burn size and incidence of inhalation injury illustrates the rise in occurrence of inhalation injury with increasing burn size [5].

DJ Dries and FW Endorf, 2013

Pathophysiology of Inhalation Injury

- **Upper Airway Injury**
  - Airway temps in a fire reach 1000ºF but is dissipated in the airway[5]
  - Massive swelling of oropharyngeal structures within hours
- **Tracheobronchial Tree Injury**
  - Bronchospasm
  - Formation of pseudomembranous airway casts[8]
- **Lung Parenchyma Damage**
  - Alveolar destruction and atelectasis due to transvascular fluid flux and loss of surfactant[10]
  - Activated neutrophils cause direct cell damage via proteases/free radicals[10]
- **Systemic Effects**
  - Cyanide and Carbon monoxide toxicity
  - Diffuse inflammatory response

Airway Changes in Thermal Injury



Miller AC, et al. *Crit Care Med* 2014

More Than the Heat: Chemical Breakdown of Smoke

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

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

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

Mosier MJ, et al. (2012)

Look Worse = Do Worse

Table 2 Comparison for bronchoscopic grade of inhalation injury

	Group 1 (Grades 0 and 1) 25 Patients	Group 2 (Grades 2, 3, 4) 35 Patients	*P* Value
mL/kg/%TBSA	6.6 (±0.7)	6.7 (±0.4)	.88
Ventilator days	8.6 (±1.4)	12.8 (±2.2)	.11
Survival	21 (84%)	20 (57%)	.03
Initial compliance	49.9 (±4.4)	49.7 (±3.1)	.98
Initial P:F Ratio	371.5 (±32)	329.7 (±29)	.33

Endorf, et al. (2007)

Bronchoscopy in Smoke Inhalation

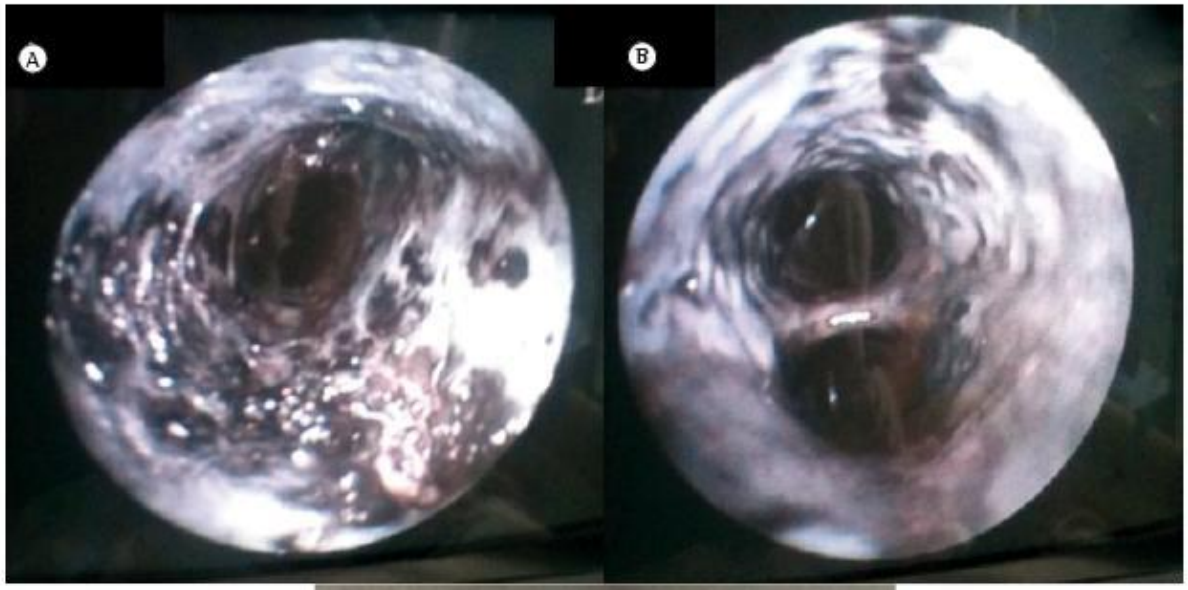
Post Inhalation, Day 0

Walker, et al. (2015)

Post Inhalation, Day 4

Walker, et al. (2015)

Cast formation



Airway cast

Walker, et al. (2015)

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

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

Reference	Lung Injury Score	Pneumonia Prevalence	Mechanical Ventilation Duration	Unplanned Reintubation	Hospital Length of Stay	Bleeding Risk	Mortality
Desai et al (2)	↓	↓	No change	↓			
Rivero et al (48)	↓						↓
Holt et al (50)[a]		No change			No change		No change
Miller et al (27)	↓						↓
Yip et al (49)						No change	

[a]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).

- 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]

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Questions?

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