Inhalation Injuries

The Invisible Threat

Sarah Fischer, MSN, RN

Chris Thompson, RN, EMT-P

Ascension

Listening to you, caring for you.®

Disclosure

Presenter has nothing to disclose.

Objectives

- Discuss the significance of inhalation injury
- Explore the pathophysiology of inhalation injury and systemic toxicity
- Describe assessment and ongoing management of the patient with inhalation injury
- Identify treatment adjuncts for inhalation injury and systemic toxicity
- Recognize special considerations for children with inhalation injury

Historical Perspective - 1942

Cocoanut Grove - Boston, Massachusetts
492 fatalities, most occurring in the first 30 minutes
Primary cause of death = smoke inhalation

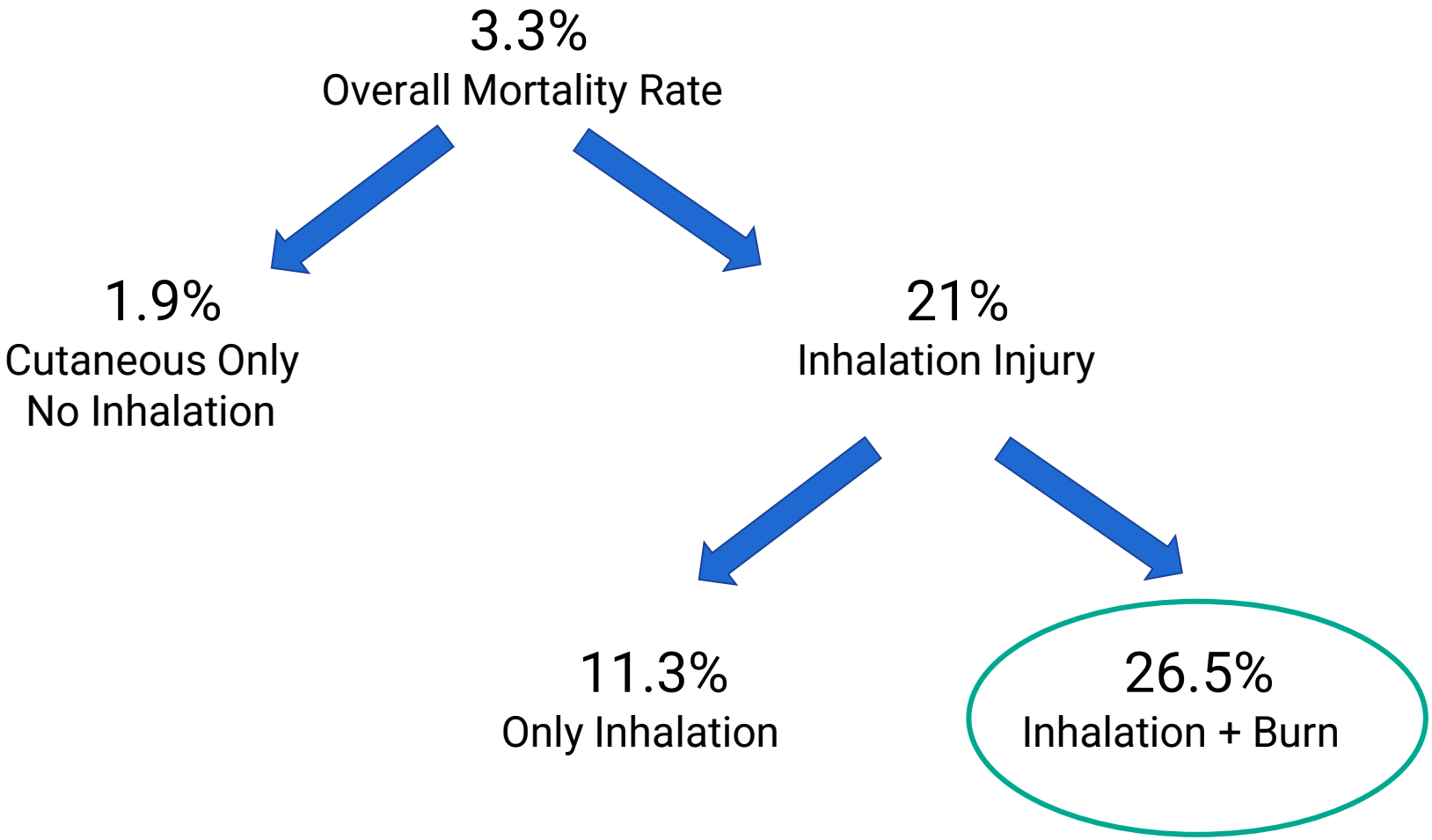
radioboston.legacy.wbur.org/2012/10/31/cocoanut-grove-transcripts

Historical Perspective - 2003

The Station - Warwick, Rhode Island
100 fatalities, 215 injuries
Building completely collapsed within 30 minutes

https://www.cbsnews.com/news/the-station-nightclub-rhode-island-fire-deaths-owners/
https://www.firerescue1.com/firefighting-history/articles/station-nightclub-fire-lessons-code-changes-follow-tragedy-VsJH1dv8rXfbtUlG/

Burn Injury Summary Report (2018-2022)



Clinical Significance

Mortality - immediate anoxic injury versus decompensation over time

Airway obstruction secondary to edema

Increased fluid resuscitation requirements

Impaired gas exchange, tissue ischemia
- Chemical pneumonitis, pulmonary edema, ARDS

Multiple organ failure
- Systemic inflammatory response syndrome

Chronic pulmonary dysfunction
- Laryngeal damage, pulmonary fibrosis

Differential Diagnosis

Determined initially by history and external exam

- Mechanism of injury
- Location - structure, vehicle, outdoors
- Duration of exposure
- Signs and symptoms

Supported by interventional adjuncts after patient is stabilized

- Carboxyhemoglobin
- Bronchoscopy

Pathophysiology

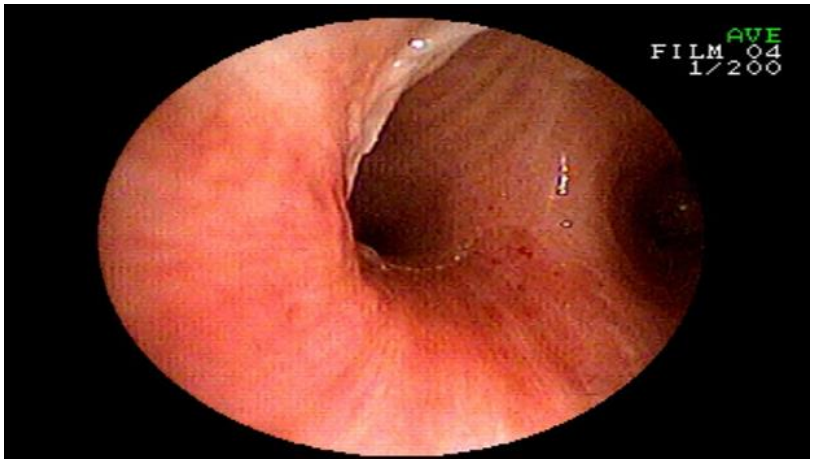
Direct thermal damage
- Irritation and inflammation
- Mucosal sloughing
- Bronchospasm

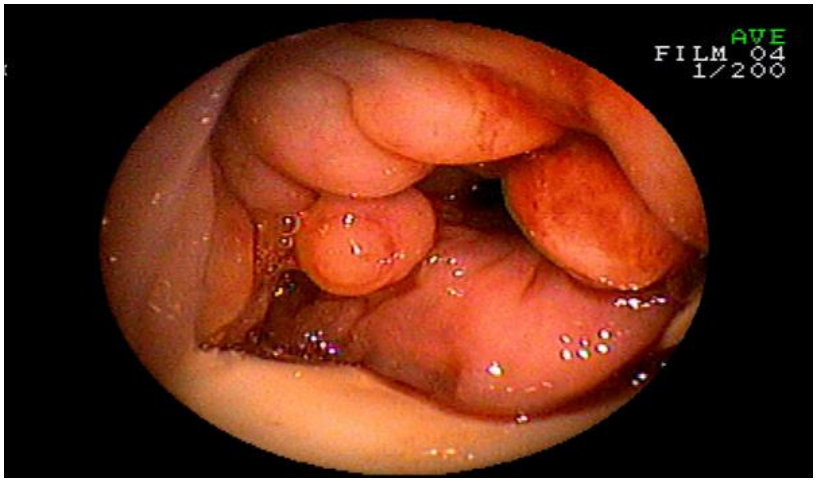
Secondary inflammation
- Local cytokine inflammatory response
- Bronchorrhea, alveolar flooding, cast formation
- Impaired ciliary and surfactant function

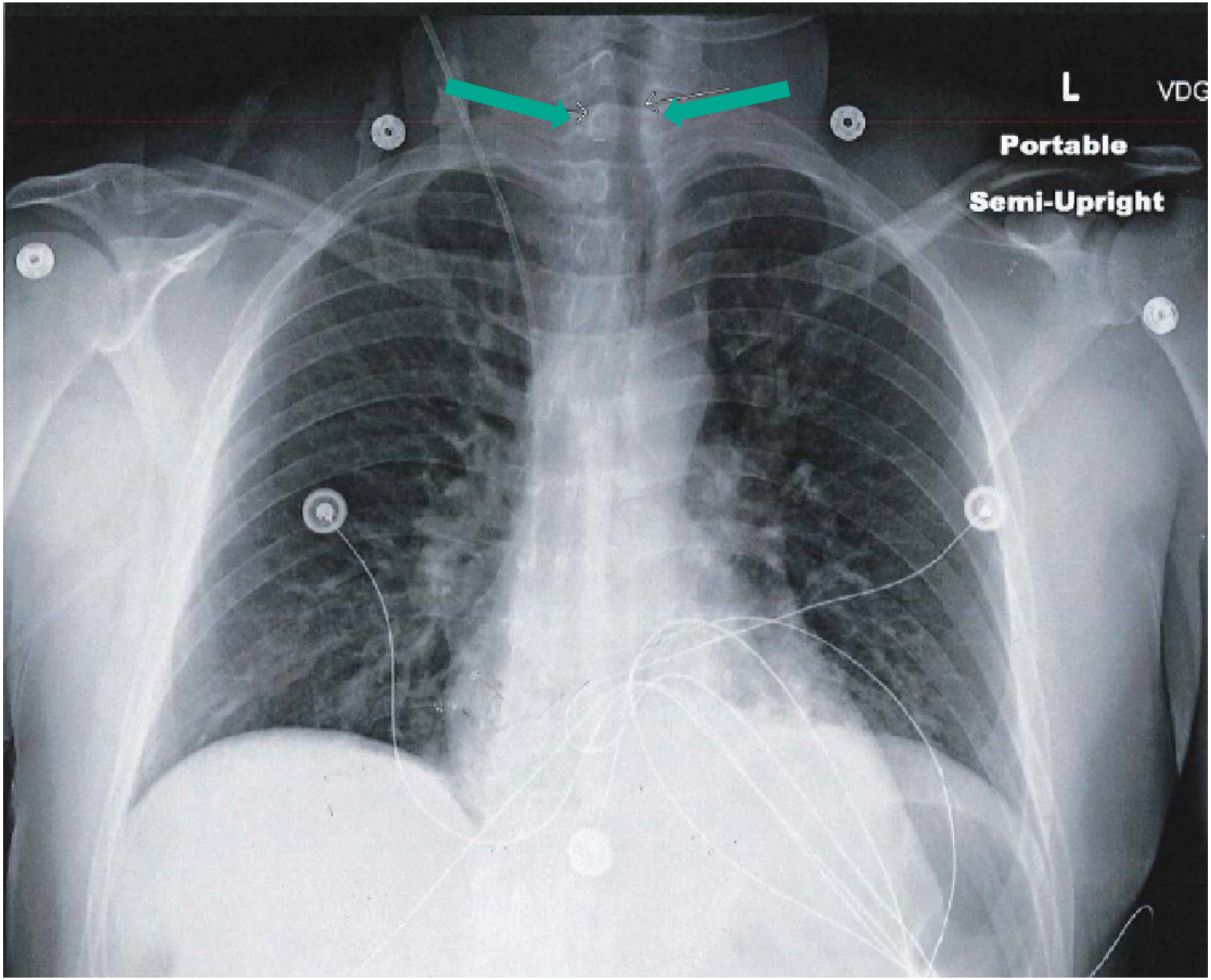
Systemic toxicity
- Tissue ischemia
- Anoxic brain injury
- End organ dysfunction

Supraglottic Injury

- Consequence of direct thermal injury to the upper airway
- Glottal reflex protects the lower airway from heat exposure
- Airway obstruction is the biggest threat






L
VDG
Portable
Semi-Upright

Supraglottic Injury

Signs and symptoms

- Burns of face and/or neck
- Singed nasal and/or facial hair
- Hoarseness
- Difficulty swallowing
- Dyspnea
- Stridor
- Signs of hypoxia
  - Agitation in the presence of significant inhalation injury may be related to hypoxia versus psychosocial behaviors or illicit substances

Supraglottic Injury

Treatment in the immediate post-burn period

- Maintain open airway
  - Early intubation to prevent obstruction
- Provide supplemental oxygen
- Close monitoring for deterioration
- Appropriate fluid resuscitation
  - Inadequate resuscitation exaggerates local cytokine inflammatory response
  - Over-resuscitation exaggerates edema

Maximum edema should occur within 24 - 48 hours of injury

Subglottic Injury

- Consequence of smoke, chemicals, or pressurized steam
- Glottal reflex is not triggered = inhalants deeper into lungs
- Smaller particles are deposited distally
- Difficult to mitigate complications as effects are diffuse

Subglottic Injury

Signs and symptoms

- Burns of face and/or neck
- Singed nasal and/or facial hair
- Hoarseness, sore throat, cough, difficulty swallowing
- Carbonaceous sputum
- Shortness of breath, dyspnea
- Wheezing, stridor
- Signs of hypoxia
  - Disorientation, restlessness, confusion, agitation

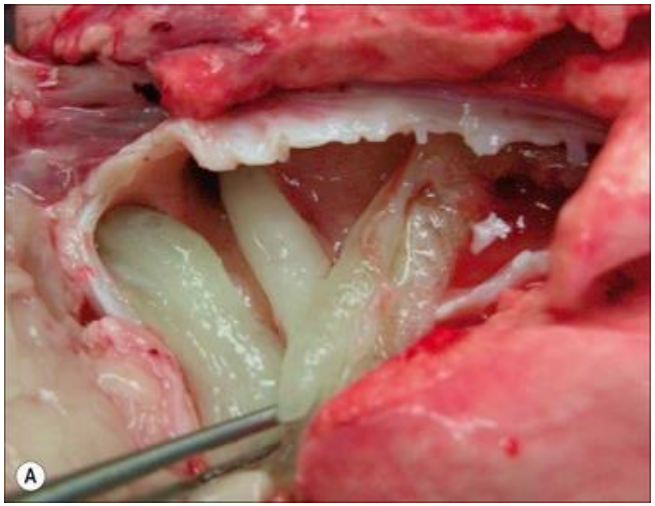
Subglottic Injury

- Sloughing of epithelial lining
- Impaired ciliary action
- Mucus hypersecretion
- Surfactant inactivation
- Inflammation
- Pulmonary edema
- Fibrin cast formation

Subglottic Injury

Treatment begins in the immediate post-burn period but often lasts longer than supraglottic injury

- Protect airway and support pulmonary function
- Appropriate fluid resuscitation
- Chest physiotherapy
- Therapeutic bronchoscopy
- Monitor for signs of pulmonary edema/ARDS
- Targeted inhalant medications
    - Bronchodilators (albuterol) - decrease airflow resistance and improve compliance of smooth muscle
    - Mucolytic agents (acetylcysteine) - breakdown mucus while mitigating inflammatory response
    - Anticoagulants (heparin) - protect against the formation of fibrin casts



plasticsurgerykey.com/the-pathophysiology-of-inhalation-injury/

Systemic Toxicity

Occurs with or without cutaneous injury
Destruction at the cellular level
Interferes with oxygen utilization

- Carbon monoxide
- Hydrogen cyanide
- Chemical warfare

Carbon Monoxide

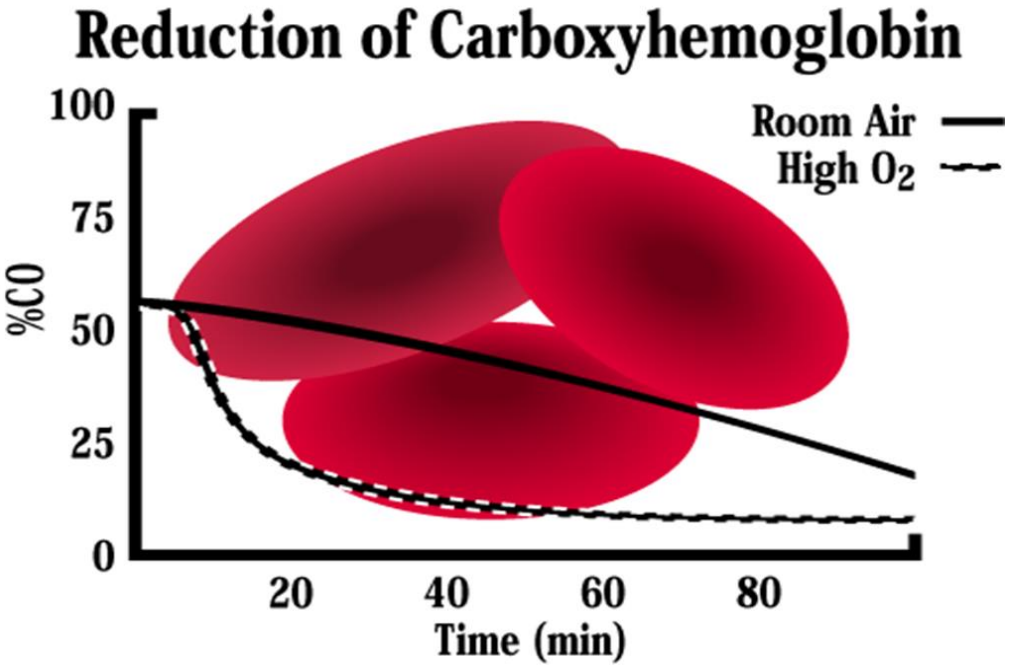
- Carbon binds to receptors on hemoglobin
- Affinity 220 times greater than oxygen
- Hemoglobin continues to circulate causing ischemia
- SpO2 is unreliable
- Carboxyhemoglobin
  - Goal <5%
  - Smokers range from 3 - 15%

Carbon Monoxide

- Lower levels
  - Headache, fatigue, flu-like symptoms, weakness, dizziness, nausea, blurred vision, intoxicated appearance
- Higher levels
  - Vomiting, confusion, palpitations, seizures
- Levels > 60 often associated with cardiopulmonary arrest and death

Carbon Monoxide

- 100% oxygen
- Flood receptors
- Half-life of CO
  - RA = 4 hours
  - 100% = < 1 hour



Hydrogen Cyanide

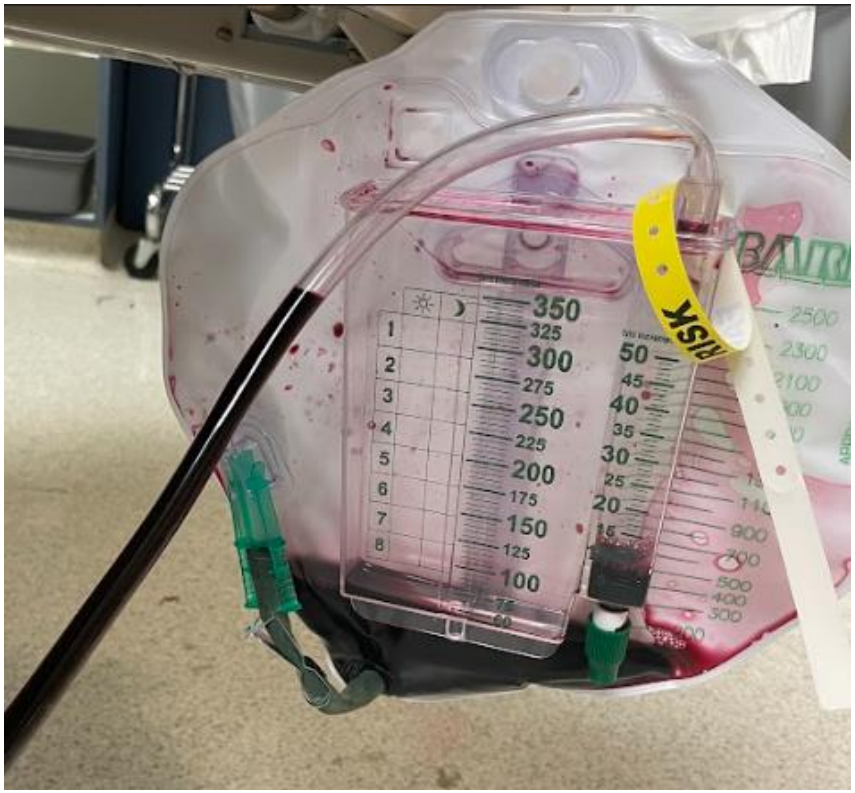
- Less common than carbon monoxide
- Blocks oxygen utilization in mitochondria
  - Limits ATP production
  - Increases ischemia-causing free radicals
- “Cherry-red” skin
- Metabolic acidosis
- Lactate > 10
- High venous pO2 as cells are not using available oxygen

Hydrogen Cyanide

- Lab results are not timely - treat based on history, lactic acidosis
- Lower levels
  - Faintness, flushing, anxiety, perspiration, vertigo, headache, drowsiness, tachypnea, tachycardia
- Higher levels
  - Hypotension, tremors, arrhythmia, convulsions, stupor, paralysis, coma, respiratory depressions, cardiopulmonary arrest

Hydrogen Cyanide

- 100% oxygen
- Intravenous hydroxocobalamin - Cyanokit
  - Converts cyanide to a nontoxic form that can be excreted in the urine



Intubation

- Rapid sequence intubation
- Place the largest recommended size per age/weight
- Cuffed tube for all ages
- Secure the tube
  - Adhesives will not stick to burned skin
  - Very important, especially for transport
- Cuff leak test prior to extubation

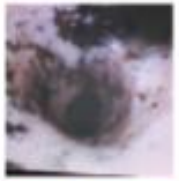
Bronchoscopy

- Supports clinical diagnosis

- Allows for visual assessment of the airway
- Allows for therapeutic removal of debris
- Allows for direct sampling, when indicated

- Not sensitive to clinical outcomes
- Helpful but not essential

Inhalation Injury Bronchoscopic Scoring

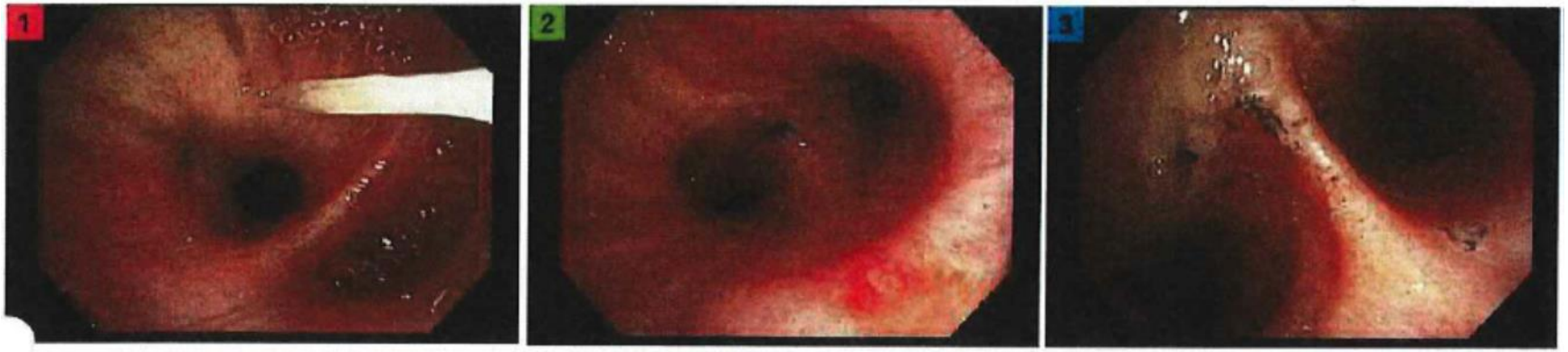
Classification	Grade	Bronchoscopic criteria	Example
None	0	Absence of carbonaceous deposits, erythema, oedema, bronchorrhoea and obstruction	
Mild	1	Minor/patchy areas of erythema and/or carbonaceous deposits in the proximal or distal bronchi	
Severe	2	Moderate degree of erythema, carbonaceous deposits and/or bronchorrhoea, with or without compromise of the bronchi	
	3	Severe inflammation with friability, copious carbonaceous deposits, bronchorrhoea and/or bronchial obstruction	
	4	Evidence of mucosal sloughing, necrosis and/or endoluminal obliteration	

https://www.researchgate.net/deref/https%3A%2F%2Fdoi.org%2F10.1016%2Fj.burns.2021.11.018?_tp=eyJjb250ZXh0Ijp7ImZpcnN0UGFnZSI6InB1YmxpY2F0aW9uIiwicGFnZSI6InB1YmxpY2F0aW9uIn19

Bronchoscopy

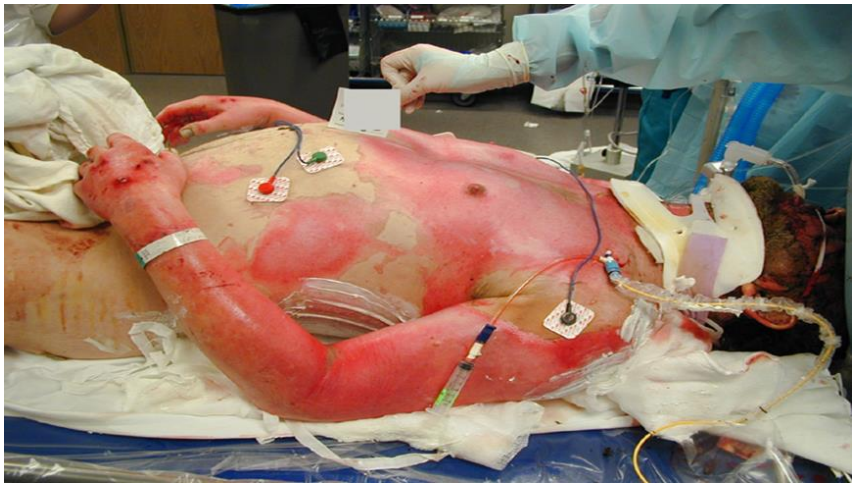
Before Treatment

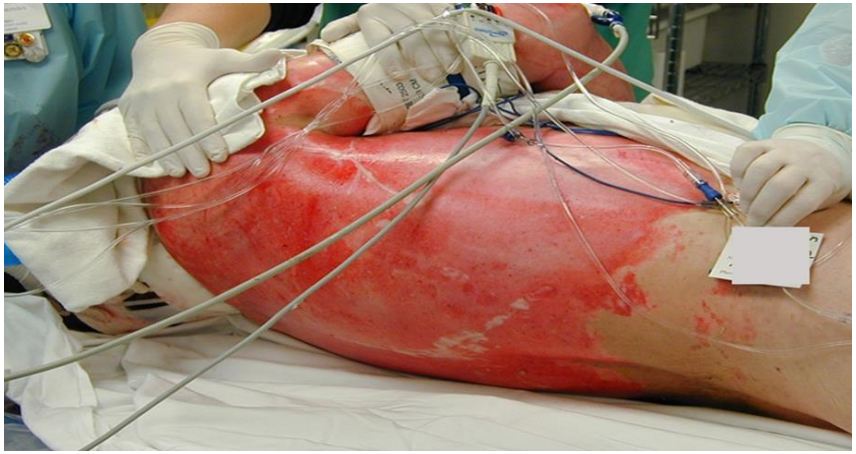
After Treatment



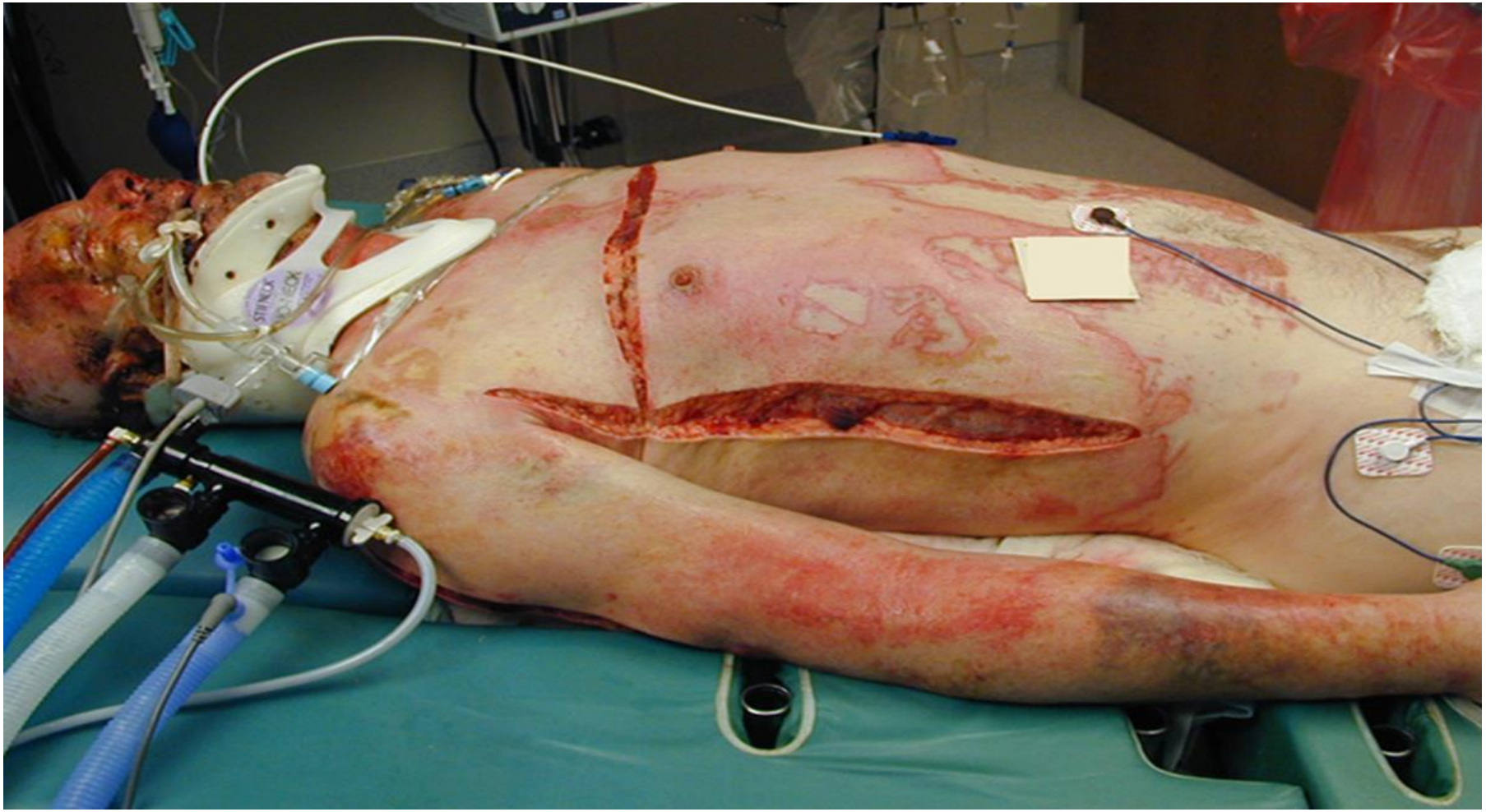
Compartment Syndrome

- Circumferential, full thickness torso burns
- Compromises chest wall excursion and ventilation
- Increased peak pressures as airway compliance decreases





Escharotomy



Hyperbaric Oxygen

- May be used to treat carbon monoxide poisoning, severe metabolic acidosis, and end organ ischemia associated with inhalation injury
- No evidence to suggest superiority over 100% oxygen therapy
- Consider risks versus benefits

Pediatric Consideration

Pediatric Considerations

- Airway obstructs easily
- Avoid hyperextension
- Smaller oxygen reservoirs
- Limited compensatory mechanisms
- Weaker accessory muscles

- Intubation
  - Be proactive, loss of airway will be detrimental
  - Use the appropriate size tube for age
  - Cuffed endotracheal tube
  - Securement is critical

Final Thoughts

- Do not underestimate the significance of inhalation injuries
- Mortality risk increases in the presence of inhalation injury
- Loss of airway from supraglottic edema is catastrophic
- When in doubt - intubate
- 100% oxygen is the mainstay of treatment
- Consult with your local/regional Burn Center

References

American Burn Association. (2023). *Advanced Burn Life Support*.

American Burn Association. (2023). *Annual Burn Injury Summary Report.*

Dyamenahalli K., et. al. (2019). Inhalation injury: Unmet clinical needs and future research. *JBCR. 40*(5), 570-584. doi: 10.1093/jbcr/irz055

Herndon, D. (2018). *Total burn care* (5th ed.). Edinburgh: Elsevier.

Lan, X., et. al. (2020). Nebulized heparin for inhalation injury in burn patients: A systematic review and meta-analysis. *Burns & Trauma. 8*, 1-10. doi: 10.1093/burnst/tkaa015

Sheridan, R. (2016). Fire-related inhalation injury. *The New England Journal of Medicine. 375*(5), 464-469. doi: 10.1056/NEJMra1601128

Walker, P., et. al. (2015). Diagnosis and management of inhalation injury: An updated review. *Critical Care. 19*(351). doi: 10.1186/s13054-015-1077-4