Conflict of interest

* No conflict of interest to declare
Objectives

- Understand different mechanisms of injury
- Anticipate types of injury based on clinical presentation
- Understand criteria for airway intervention
- Treat potential toxic exposures
Mechanisms of Injury

- Heat (Thermal)
- Hypoxia
- Direct toxins
- Systemic toxins
Thermal Injury

- Smoke is hot, but dry
- Low specific heat capacity
  - Rare to have subglottic burns from smoke alone

- Can burn if moist heat, or with general burn injury (ambient heat)
Hypoxia

- Fire consumes oxygen
- FiO2 of inhaled air decreases
  - Causes neural damage
  - Potentiates other toxins
  - Increases minute ventilation
    - More smoke inhaled
Direct Toxins

- Substances toxic to respiratory epithelium
  - SO2, NO2, N2O, Chlorine, multiple organic compounds
  - HCl, HBr, HF, other inorganic compounds
- Effects usually appear 12-48h after exposure
  - Neutrophilic invasion
  - Mucociliary sloughing
  - Increased epithelial permeability
Airway evaluation

- Should be carefully performed in any potential inhalational injury victim
- Indications for early intubation
  - Hoarse voice
  - Stridor
  - Burns above clavicles
  - Soot in nares or mouth, carbonaceous sputum
  - Singeing of facial hair
  - Significant (>30% BSA) burns
  - Altered mental status
Bronchoscopy

- Controversial
- Demonstrates
  - depth of burn
  - carbonaceous sputum
- May help with unclear airway status
- No clear correlation with long-term prognosis
ARDS

- Common after inhalational injury
- Due to toxin inhalation
  - Edema, inflammation, bronchospasm, mucociliary sloughing
  - Microatelectasis, loss of surfactant, capillary leak
  - Can form bronchial casts
- Secretions build up, can obstruct ETT
- Peaks at ~72h post injury
ARDS

- PEEP
- Permissive hypercapnia
- Frequently complicated by pneumonia (30-70%)
Systemic Toxins

- Carbon Monoxide and Cyanide
Carbon Monoxide Poisoning

- Has 240 times the affinity for Hb as O2
- Also binds to porphyrins elsewhere
  - Mitochondrial cytochromes, NADPH reductase

- Half-life:
  - Room air: 300 minutes
  - 100% FiO2: 90 minutes
  - 3 atmospheres 100% FiO2: 30 minutes
Carbon Monoxide Toxicity - Diagnosis

- Cannot use SpO2
- Only co-oximetry, or, where not available, high index of suspicion
Carbon Monoxide Poisoning - Treatment

- 100% O2 for all potential victims
- Hyperbaric therapy considered if:
  - Unconscious, altered MS, neurologic deficit
  - ECG changes, CO-Hb >40% (20% if pregnant)
Carbon Monoxide - Treatment

- Hyperbaric therapy after acute injury
  - Controversial
  - 2 RCT’s - opposite conclusions about neurocognitive sequelae
  - Both flawed studies
    - Poor randomization in one
    - Poor follow-up in the other
Carbon Monoxide - Treatment

- Isocapnic hyperpnea
  - Hyperventilating with a small amount of CO2 in the circuit to maintain PaCO2 at 40
  - Doubles rate of CO-Hb elimination
Hydrogen Cyanide (HCN)

- Liberated from any burned substance that has carbon and nitrogen
  - Plastics, natural fibres, insulation, upholstery
STAND BACK
I'M GOING TO TRY SCIENCE
Cyanide Toxicity

- Binds Fe3+ on Cytochrome Oxidase a3
- Stops oxidative phosphorylation
- Cells switch to anaerobic metabolism
  - Vomiting, abdominal pain
  - Confusion, headache, seizures
  - Hyper- and hypotension, tachycardia, CV collapse, AV block, Ventricular arrhythmias
  - Tachypnea
  - Flushing of skin, renal failure, hepatic necrosis
Cyanide Toxicity - Diagnosis

- Presumed present if significant burn or smoke inhalation
- Any base deficit, lactic acidosis, or anion gap in a burn/smoke inhalation victim
- VBG with abnormally high PO2
Cyanide toxicity - Treatment

• Lilly Kit
  ➔ Induction of methemoglobinemia
    – Cyanide binds Fe3+ >> Fe2+
    – Inducing methemoglobinemia creates a competitive site for the cytochrome binding site for CN- a cyanide “sink”
    – CN-Hb is relatively nontoxic
Cyanide Toxicity - MetHb

- Sodium nitrite
  - Given IV - dosed to achieve 10-30% MetHb
  - Causes left-shift of Oxygen-Hemoglobin dissociation curve

- Contraindicated in concomitant CO poisoning
Oxygen saturation of hemoglobin, percent

Partial pressure of oxygen, mm Hg

Left-shifted

Right-shifted
Cyanide Toxicity - Sulfur Donation

- Sulfur donor to allow natural elimination of cyanide by rhodanese
Cyanide Toxicity - Sulfur Donation

- Given via sodium thiosulfate
- Often in combination with inducing methemoglobinemia
  - Together increase lethal dose of cyanide 10-15x
Cyanide Toxicity - Treatment

Hydroxocobalamin

Cyanocobalamin
Cyanide Toxicity - Hydroxocobalamin

● Standard of care
● Rapid, safe, nontoxic
  – Side effects: Red skin, urine, mucous membranes
  – May interfere with co-oximetry
● May be used with thiosulfate
Conclusion

- Inhalational injury requires rapid diagnostics and interventions
- Must have high index of suspicion for toxic exposures
- Airway evaluation is critical
- Beware your sat probe!
References

- http://pedscmm.org/FILE-CABINET/Pulmonary/Smoke_inhalation.html