Objectives

- List the factors that influence mortality rate
- Describe the nature of smoke inhalation and the fire environment
- Recognize the pulmonary and systemic changes that occur following smoke inhalation and burn injury
- List the effects of smoke inhalation injury on the upper and lower airways
- Identify methods to diagnose smoke inhalation injury and CO poisoning
Objectives

- Describe the methods used to determine the type and extent of burn injury
- Recognize the emergent treatment for smoke inhalation injury and CO poisoning
- Describe the airway and ventilatory support strategies for smoke inhalation and burn injury
- Describe the fluid, surgical, and nutritional support used in the treatment of burn injuries
Introduction

- Fire is a major source of injury, death, and economic loss
- Burns rank third most common cause of serious injury and death
  - 80% of deaths in residential fires
  - 5%–10% mortality due to asphyxiation, systemic poisoning, and respiratory tract injury
Introduction

- Prevalence of smoke inhalation among burn victims = 10%–35%
- Pulmonary complications according to the resuscitative phase
  - Early (first 24 hours)
    - Inhalation of toxic or hot gases
    - Fluid loss
    - Heavy sedation
Introduction

- Intermediate (2–7 days) post resuscitative phase
  - Analgesic-related respiratory dysfunction
  - Secretion retention
  - Airway obstruction
  - Atelectasis
  - ARDS
Introduction

- Late (> 7 days)
  - Pneumonia
  - Sepsis
  - Multiple organ dysfunction
  - Pulmonary embolism
  - Chronic pulmonary disease
Etiology

- Fire – residential fires – most common
- Superheated gases
- Scalding liquids
- Chemicals
- Electrical currents
Within 24 hours post-burn

- Carbon monoxide
  - Produced in fire environment, especially if
    - Oxygen levels are low
    - Combustion is incomplete
  - Rapidly absorbs into blood
Carbon monoxide

- It converts HbO₂ into HbCO
  - Normal HbCO: < 3%
  - Minor smoke inhalation: 10%–15%
  - Severe smoke inhalation: > 50%
- Compromise of O₂ transport
- Inability of Hb to transport O₂
- The Hb conversion and inhibition of O₂ release result in *Functional anemia*
Pathophysiology: Early Pulmonary and Systemic Changes

- Carbon monoxide
  - Skeletal and cardiac muscle dysfunction
  - Cerebral vasodilation
  - Rapid loss of consciousness and cerebral edema
  - Lethal CO poisoning typically occurs when COHb > 50%–60%
Pathophysiology: Early Pulmonary and Systemic Changes

- Hydrogen cyanide (HCN)
  - Linked to early and late death in burned patients
  - Easily transported to tissues through circulatory system; blocks tissue use of O2
  - Shift to anaerobic metabolism and elevated lactic acid production
Pathophysiology: Early Pulmonary and Systemic Changes

- Other considerations
  - Reduced oxygen transport
  - Cellular metabolic dysfunction
  - Release of inflammatory mediators
  - Vascular changes

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Compromise of nervous system, cardiovascular system, and skeletal muscle function
(Causes of death)
Other considerations

- **Thermal** injury to the respiratory tract
  - Typically confined to the face, oral and nasal cavities, pharynx, and trachea
  - Blistering, edema, accumulation of thick saliva, and glottic closure if severe
Pathophysiology: Early Pulmonary and Systemic Changes

- Other considerations
  - **Chemical** injury to the respiratory tract
    - Injuries extend into the lungs
    - Tracheobronchitis, bronchospasm, bronchorrhea, mucosal sloughing, airway obstruction
    - Alveolar de-recruitment - atelectasis
    - Pulmonary edema in severe cases
Pathophysiology: Early Pulmonary and Systemic Changes

- Other considerations
  - **Systemic** changes are associated with
    - Decline in O2 transport
    - Metabolic derangement
    - Release of inflammatory mediators
    - Fluid loss
Pathophysiology: Intermediate Pulmonary and Systemic

- Changes (2–7 days post-burn)
  - Signs of respiratory distress often after 24–48 hours
  - PVR returns to normal
  - Hypermetabolic state continues
    - Increased O2 consumption and CO2 production
Pathophysiology

- Airway edema resolves between day 2 and 4
- Increased mucus production
- Atelectasis, pleural effusion, acute lung injury
Pathophysiology: Late Pulmonary and Systemic Changes

- > 7 days post-burn
  - Hypermetabolic state for 1–3 wks
  - Infection is the most common complication in this period
    - Staphylococcus aureus
    - MRSA
    - Pseudomona aeruginosa
  - Pulmonary embolism can develop within 2 weeks of burn injury
Clinical Features

- Brain and heart = first to show dysfunction
- HbCO content is potential indicator of the dose of smoked inhaled
- $\text{SpO}_2$ should not be used since HbO$_2$ and HbCO have similar light absorption
Clinical Features

- Upper respiratory manifestations
  - Stridor – Hoarseness
  - Difficulty speaking – Chest retractions
- Severe form of inhalation injury
  - Cough – Dyspnea
  - Tachypnea – Cyanosis
  - Wheezing – Crackles
  - Rhonchi
Clinical Features: Chest Radiograph

- Frequently no signs in early period
- CT scans may be more useful to determine severity of pulmonary injury
Clinical Features: Arterial Blood Gases

- To trend the patient’s pulmonary insult
- Reduced PaO2 and SaO2
- Reduced PaO2/FiO2 (ALI vs ARDS)
- Respiratory alkalosis in early post-burn period
- Metabolic acidosis and respiratory failure are signs of life threatening injuries
Clinical Features: Hemodynamic Monitoring

- To optimize fluid resuscitation
- Monitor
  - CVP
  - PAP
  - CO
  - Urine output
Treatment

- Goals of respiratory care in burn patient
  - Achieve a patent airway
  - Secretion removal
  - Maintenance of effective ventilation
  - Preservation of lung volume
  - Adequate oxygenation
  - Maintenance of acid–base balance
Treatment: Airway

- Monitor for airway closure
- High Fowler’s position to reduce WOB
- Intubation if airway closure is anticipated
- Extubation if
  - Patient is improving
  - Maintain his/her own secretions
  - Cuff leak
  - Adequate ventilation
Treatment: Carbon Monoxide Poisoning

- Oxygen therapy: cornerstone of therapy (NRM)
- High-flow mask CPAP 5–10 cm H2O if
  - Patient with minimal upper airway thermal injury
  - Increasing dyspnea
  - Increasing hypoxemia
- Intubation if HbCO > 30%
Treatment

- Mechanical Ventilation if
  - Respiratory failure
  - Pneumonia
  - ALI/ARDS
  - Sedation and paralysis are necessary
Treatment: Fluid Balance

- To minimize development of
  - Shock
  - Renal failure
  - Pulmonary edema
Treatment: Prevention of Burn Complications

- Isolation technique
- Room pressurization
- Air filtration
- Wound covering
- Topical silver sulfadiazine
- Prophylactic antibiotics

Front line of infection defense