AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
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Smoke Inhalation: More than Soot in Your Eyes

Victims of smoke inhalation present with a combination of injuries from noxious particulates, irritant chemicals, and cellular asphyxiants. The attendee will learn the initial approach to airway and pulmonary management in these cases. In addition, the systemic effects of toxins such as cyanide and carbon monoxide will be discussed, including indications for specific antidotes.

- Recognize the need for early intervention in the airway management of smoke inhalation victims.
- Discuss the criteria for observation or admission of smoke inhalation victims.
- Review the various toxins present in smoke and their specific antidotes.

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FACULTY

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Smoke Inhalation

The 1999 ACEP Scientific Assembly, Las Vegas

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Course Description

Victims of smoke inhalation present with a combination of injuries from noxious particulates, irritant chemicals and cellular asphyxiants. The attendee will learn the initial approach to airway and pulmonary management in these cases. In addition, the systemic effects of toxins such as cyanide and carbon monoxide will be discussed, including indications for specific antidotes.

Objectives

- Understand that smoke inhalation is a complex toxicological problem.
- Know the most dangerous toxins, clinical effects, treatment guidelines and pitfalls regarding smoke inhalation patients.
- Gain knowledge necessary to make confident decisions about upper airway evaluation and management, antidote therapy, use of HBO, appropriate transfer criteria, and patient disposition.
- Obtain updates of recent literature regarding smoke inhalation poisoning.

Summary

- Toxins in fires are unpredictable. Smoke inhalation is not just CO!
- Fire Chemistry is complex. Fortunately, the pathophysiologic response to smoke is relatively simple.
- Smoke inhalation is a toxicologic disease.
- The final common pathway of all toxins in smoke is tissue hypoxia.
- NOT recognizing upper airway injury is a major pitfall in management.
- Most diagnostic tests for smoke inhalation poisoning are nonspecific. The most reliable are clinical exam, direct visualization of the vocal cords, and markers of tissue hypoxia.
- Elevated carboxyhemoglobin indicates that the patient received a potentially large dose of smoke.
- Empirically treat all seriously ill smoke inhalation patients for cyanide poisoning.
- Check the eyes; Soot can Burn.
- Inhalation PLUS burns is very BAD.
- A period of observation and repeated evaluations is the rule with all smoke inhalation exposures.

History

Most fire related deaths are from inhalation injury

Great Fires in History

Great fire of Babylon – 538 BC
Great fire of Rome – 64 AD
London Burns - 1212
Washington, DC burns - 1814
Great Chicago Fire - 1871
Iroquois Theater burns – 1903 – 602 deaths
Cleveland Clinic Fire – 1929 – 121 dead from x-ray film fumes
Cocoanut Grove, Boston – 1942 – 491 deaths
Beverly Hills Supper Club, Southgate, KY – 1977 – 164 deaths
MGM Las Vegas fire – 1980 – 84 deaths
Hilton Las Vegas – 1982
Imperial Foods, Hamlet, NC 1991
Twin towers bombing in New York City – 1993- Injured 1000

> "The complications encountered were similar to those resulting from inhalation of certain war gases … From the experience of the Cocoanut Grove fire, we know that such pulmonary complications are to be found not solely in warfare, but may be encountered at any moment in civilian life." *Oliver 1943*

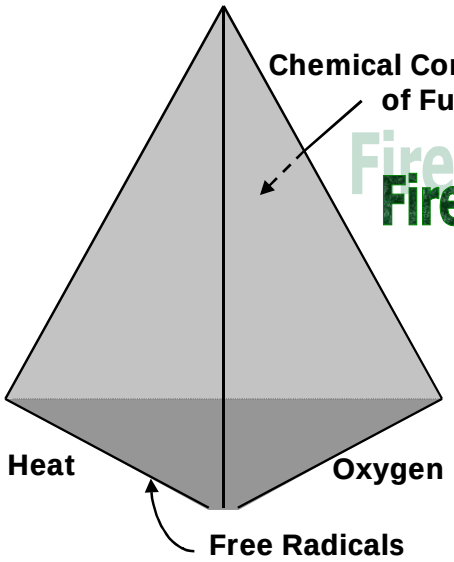
The Complex: Understanding Fire Chemistry

Combustion products are difficult to predict in a fire

COMBUSTION (PYROLYSIS) - Rapid oxidation of a substance (fuel).. It is a chemical reaction that releases energy as heat and light (Flames). During combustion two or more substances chemically unite (usually atmospheric oxygen) to form oxides (carbon dioxide – complete combustion; carbon monoxide – incomplete combustion). When carbon-based fuels do not combine with oxygen combustion produces tiny particles of elemental carbon that manifests as black smoke.

PRODUCTS OF COMBUSTION ARE:

- FLAME (Light)
- HEAT
- SMOKE – complex mixture of:
  - Suspended Solid and Liquid Particles - mostly carbon coated with organic acids and aldehydes (intense irritants)
  - Heated air
  - Gases, Fumes, Aerosols, Vapors - major components: CO_2 and CO; Others: CN, HCL, SO_4, NO_4, NH_3, and OTHERS



FIRE CONDITIONS DETERMINE SMOKE COMPOSITION.

1. AVAILABILITY OF OXYGEN
2. COMPOSITION OF MATERIAL (Fuel)
3. TEMPERATURE
   e.g., POLYURETHANE:
   300 - 800°C = ISOCYANATE
   >800°C = Hydrogen Cyanide

Combustion products are difficult to predict in a fire; in fact, the composition of smoke often is quite variable within the same fire environment.

For example: Polyvinyl chloride produces at least 75 potentially toxic agents

Wood produces as many as 200 potentially toxic agents

PRODUCTS OF COMBUSTION FROM SPECIFIC SUBSTANCES

Fuel	Combustion Products
WOOD, COTTON, PAPER	Acetaldehyde, acrolein, formaldehyde, acetic acid, methane, formic acid, Carbon monoxide, Cyanide
NYLON	Ammonia, cyanide
SILK	Sulfur dioxide, hydrogen sulfide, ammonia, cyanide
WOOL	carbon monoxide, hydrogen chloride, phosgene, chlorine, cyanide
PETROLEUM PRODUCTS	Acrolein, acetic acid, formic acid, sulfur oxides, carbon monoxide

POLYSTYRENE	Styrene
ACRYLIC	Acrolein, hydrogen chloride, carbon monoxide
PLASTICS	Cyanide, hydrogen chloride, aldehydes, ammonia, nitrogen oxides, phosgene, chlorine
POLYVINYL CHLORIDE(PVC)	Hydrogen chloride, phosgene, chlorine, CO, carbon dioxide
POLYURETHANE	Cyanide, isocyanates
RUBBER	Hydrogen sulfide, sulfur dioxide
NITROGEN CONTAINING MATERIAL	Cyanide, isocyanates, oxides of nitrogen
NITROCELLULOSE	Oxides of nitrogen, acetic acid, formic acid
FLUORINATED RESINS	Hydrogen fluoride
FIRE RETARDANT MATERIALS	Hydrogen chloride, hydrogen bromide

An assortment of other toxins

Such compounds as metal oxides, hydrocarbons, hydrogen fluoride, and hydrogen bromide, cyclic and straight-chained hydrocarbons, antimony, cadmium, chromium, cobalt, gold, iron, lead, and zinc have been measured in fires. Unusual fires from industries, clandestine drug laboratories, transportation accidents, or natural disasters (e.g., volcanoes) produce additional toxic inhalants.

The Simple: Pathophysiology of Smoke Inhalation

The final common pathway of all of smoke's toxins is hypoxia

Smoke inhalation is a respiratory and systemic disease. Smoke inhalation's pathophysiologic effects impair the body's ability to acquire and use oxygen at every step of respiration. It affects the acquisition of oxygen from the environment to the cellular use of oxygen in energy production.

Classification of Combustion Products by their Toxic Effects

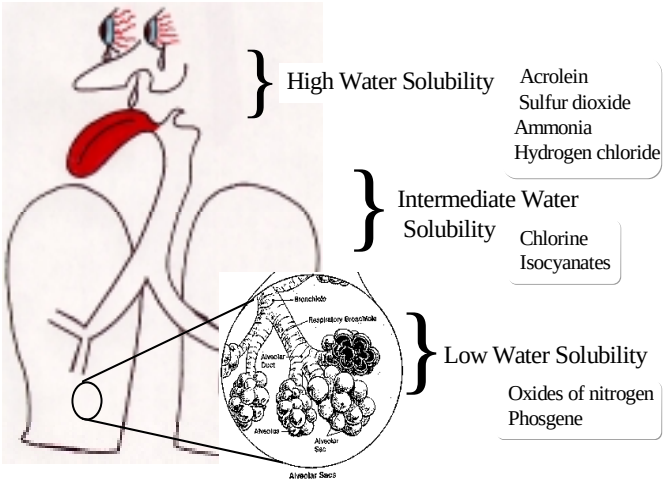
Simple Asphyxiants	**Irritants**
Carbon Dioxide Methane Oxygen-deprived environment **Chemical Asphyxiants** Carbon Monoxide Hydrogen Cyanide Hydrogen Sulfide Oxides of Nitrogen (Methemoglobinemia)	**Highly water soluble** (upper airway injury) Acrolein Sulfur dioxide Ammonia Hydrogen chloride **Intermediately water soluble** (upper & lower respiratory injury) Chlorine Isocyanates **Low water solubility** (pulmonary parenchymal injury) Oxides of nitrogen Phosgene

Simple asphyxiants act by exerting a space-occupying effect, filling an enclosed space at the expense of oxygen.

Chemical asphyxiants exert toxic effects at tissues distant from the lung.

The **Irritant toxins** are chemically reactive compounds that exert a local effect on the respiratory tract. Many irritant chemicals react with the moisture of respiratory mucosa, generating caustics that cause chemical burns and inflammatory reactions.

Water solubility is the most important physical characteristic of irritant gases.



High water solubility gases are immediately acting/ good warning properties.
Low water solubility gases are delayed acting gases/ no warning properties.

Irritant gases cause: Corrosive injury to mucosal surfaces
- Edema/ Mucous secretion
- Laryngospasm/ bronchospasm
- Sloughing of cells

Loss of protective barrier
- Fluid shifts/Bacterial invasion

Smoke Inhalation is a Multilevel Insult to the Respiratory System

Smoke inhalation is a respiratory and systemic disease. It is a complex interaction of physical asphyxiants, irritant gases and systemic asphyxiants with every step of respiration.

UPPER RESPIRATORY TRACT

- Edema and upper airway obstruction as a result of:
  - Corrosive burns from soluble gases reacting with mucosal water
  - Laryngospasm

LOWER RESPIRATORY TRACT

- Corrosive burns from volatile, insoluble gases and chemicals adsorbed onto carbon particles
- Increased airways resistance and obstruction of the smaller airways is the result of:
  - Bronchial edema and bronchospasm
  - Necrosis, ulceration, and sloughing of mucosa
  - Increased mucous production
  - Intense inflammatory response with exudate rich in inflammatory cells and protein
  - Cast formation from exudate, intraluminal debris and mucous
  - Cessation of cilial function impairing clearance of mucous
- Late complications of bacterial invasion leading to bronchopneumonia and bronchitis

ALVEOLI

- Inactivation of surfactant resulting in atelectasis
- Inflammation induced vasoconstriction (ventilation –perfusion mismatch)
- Increased alveolar - capillary barrier permeability
- Destroys alveolar macrophages allowing bacterial proliferation.

SYSTEMIC

- Toxins may inhibit oxygen carrying capacity or oxygen use at the cellular level leading to further hypoxia.

Inhalation Injury Plus Burns is Very BAD

The complex interactions of lung injury, together with the inflammatory effects of skin burns, produce a more serious systemic illness. Burn victims with inhalation injury have a higher morbidity and mortality than those with burns only; the incidence of acute respiratory failure is 61% versus 12%, respectively. In addition, burn edema is accentuated and nonburned tissue has increased vascular permeability when associated with inhalation injuries.

The Clues: The Patient Evaluation

Most Diagnostic Tests for smoke inhalation poisoning are NON-SPECIFIC

Who is at risk for significant smoke inhalation injury? Estimating the Dose can Help.

Basic Principles of Inhalation Toxicology

- **Absorption:** Inhaled toxins have higher, more rapid uptake with immediate health effects as compared to dermal, intramuscular or GI routes of absorption. Peak levels typically occur at time of exposure.
- **Dose** is determined by:

  Concentration

  Percent absorption

  Breathing pattern/minute ventilation rate

  Agent solubility

  Particle size

  Duration of exposure

  - The dose required to produce a given effect varies as a function of ***TIME***
  - The same dose administered over a longer duration will cause less severe effects than if administered over a shorter period of time.
  - Higher concentrations over a shorter duration are more likely to produce adverse health effects than lower concentrations over longer exposure periods.

Risk Factors from History

Closed space exposure
Loss of consciousness
Entrapment

Risk Factors from Physical Examination

Respiratory distress
CNS depression
Carbonaceous sputum
Edema of posterior pharynx
Face or neck burns
Hoarseness/ Stridor
Singed nasal hairs

Diagnostic studies

- ***Smoke inhalation injury causes hypoxia, therefore diagnostic studies should focus on assessing oxygenation and ventilation.***

Arterial blood gas analysis:

- Assesses both arterial oxygenation and alveolar ventilation
- Serial measurements identify hypoxemia or ventilatory failure
- Detects acidemia: clue to tissue hypoxia

Oxygen saturation measurement - accuracy of depends on the method used

- Calculated oxygen saturation from an ABG may be unreliable
- Measured oxygen saturation determined by *cooximeter* accurately reflects the percent saturation of oxygenated hemoglobin
- Cutaneous pulse oximetry is unreliable. COhgb and Methgb overestimate oxygen saturation

Carboxyhemoglobin level: *An elevated level in a fire victim indicates substantial exposure (large dose) to combustion products and a greater possibility of developing smoke inhalation toxicity*

- Elevation considered an index of cyanide poisoning: significant correlation between measured levels of both toxins
- Carboxyhemoglobin level alone is a poor predictor of the severity of toxicity
  - Low levels do not exclude the possibility of inhalation injury
  - Admission COhgb measurements are inaccurate predictors of peak levels

Methemoglobin levels have been elevated in fire victims.

- Accurately measured by cooximeter
- Cyanomethemoglobin levels are not measured as methemoglobin

Blood cyanide analysis: little clinical use because it takes hours to obtain results.

- Therapy should not await laboratory confirmation of elevated blood cyanide
- Accurate measurement depends on acquiring the sample soon after exposure

Lactic acidosis is a nonspecific indicators of tissue hypoxia

- Results from oxygen deprivation, CO poisoning, cyanide poisoning, and/or tissue hypoperfusion
- High lactate levels correlated with elevated concentrations of cyanide and CO

Chest radiograph is an insensitive indicator of pulmonary injury

- Commonly normal in the early course of smoke inhalation
- Within 24 hours of exposure: perivascular haziness, peribronchial cuffing, bronchial wall thickening, and subglottic edema
- More than 24 hours after inhalation injury: Widespread airways disease such as ARDS, aspiration, volume overload, infection, or pulmonary edema
- ***Serial chest radiographs following a baseline study are extremely helpful in detecting pulmonary disease following smoke inhalation***

Nuclear imaging and pulmonary function testing:

- Xenon ventilation studies can detect small airway and alveolar injury before radiographic changes occur
- Abnormal flow volume curves can indicate early upper airway obstruction
- Abnormal spirometry, especially forced expiratory volume (FEV1) detects early obstructive pulmonary defects of smoke inhalation, which may precede abnormalities of arterial blood gases or radiography

The Actions: Overview of Patient Management

Pathophysiologic changes of smoke inhalation injury lead to hypoxia; therefore, the most important initial treatment goal is to ensure adequate tissue oxygenation

Managing a Patient with Smoke Inhalation

Primary Survey

- Airway/Breathing/Circulation
- Improve Oxygenation
    - Supplemental Oxygen
    - Bronchodialators
    - Frequent suctioning to remove secretions and debris
    - Mechanical ventilation/ Positive end-expiratory pressure
- Treat hypotension and dysrhythmias
- Treat thermal skin burns (fluid resuscitation)
- Decontaminate skin and eyes to remove soot

Secondary Survey

- Assess Upper Airway for subtle evidence of airway injury
    - Direct Visualization/ Fiberoptic bronchoscopy
    - Prophylactic intubation
- Look for trauma or other underlying illnesses
- Search for clues of tissue hypoxia
    - Arterial blood gases with measured O_2 saturation
    - Carboxyhemoglobin/ Methemoglobin
    - Electrolytes (metabolic acidosis)
    - EKG/ Chest X-ray
    - Arterial lactate level
- Treat Specific Poisonings
    - Cyanide antidote Kit - Empirically administer sodium thiosulfate.
    - Hyperbaric Oxygen

Clinical presentations of smoke inhalation vary

The natural course of inhalation injury is immediate or very early compromise from cyanide or carbon monoxide poisoning or upper airway obstruction, then progression over several hours to days to acute respiratory failure due to atelectasis, airway obstruction, or pulmonary edema. Toxin-induced effects may contribute to adult respiratory distress syndrome (ARDS), sepsis, or pneumonia, leading to prolonged illness or delayed death.

Patients may present with:

- Cardiac arrest at scene with/or without response to ACLS
- Immediate airway compromise/wheezing
  - Only upper airway obstruction
  - Severe respiratory compromise from a combination of upper respiratory, lower respiratory, and systemic toxicity
- Minimal early symptoms that progress to respiratory failure w/in hours or days
- Transient irritation with rapid resolution and no sequelae
- Smoke inhalation injury and extensive thermal burns

A major pitfall in managing a patient with smoke inhalation is failure to appreciate the potential for rapid deterioration

Upper Airway Obstruction

- Critical airway compromise may be present on arrival at the hospital or may develop suddenly or insidiously
- Degree of injury may be underestimated because of nonspecific history and physical findings
- ***Direct evaluation*** of the upper airway is essential in assessing patients at high risk for inhalation injury
- **Fiberoptic endoscopy** is the preferred method, but when not possible empiric, **prophylactic intubation** is justified to avoid precipitous intubation that may occur with abrupt decompensation.

Relative indications for early intubation:

- Stridor
- Central nervous system (CNS) depression
- Visible burns/ edema of the oropharynx/ full-thickness burns to lips
- Full-thickness circumferential burns to the neck
- Burns requiring massive fluid resuscitation and at high risk of inhalation injury

Lower Respiratory Tract Injury

Smoke inhalation causes airflow obstruction by producing mucosal edema, intraluminal debris, inspissated secretions, and bronchospasm

- Airflow obstruction induced by irritant toxins in smoke should be partially reversible by **inhaled beta2-adrenergic agonists**
- Corticosteroids may be considered in selected patients with ***isolated inhalation injury*** and refractory bronchospasm.
- **Corticosteroids** are effective in the management of refractory acute asthma but have been shown to *increase* mortality and infection rate in the presence of burns and inhalation injury. Benefits of steroids for smoke inhalation injury have not been demonstrated in either clinical or animal studies.
- The treatment of progressive respiratory failure includes **endotracheal intubation, frequent suctioning to remove airway secretions and debris, mechanical ventilation, and positive end expiratory pressure**

Toxins may injure the skin or mucous membranes in addition to the respiratory mucosa.

- Evaluate for corneal burns caused by thermal or irritant chemical injury
- **Irrigate eyes** when signs of ocular irritation are present
- Rinse skin to prevent dermal burns from toxin-laden soot adherent to the skin (especially if contacting burned skin)
- Rapid removal of soot from skin or eyes may prevent continued injury because a chemical's **duration of contact** with tissue determines extent of injury

Treating Systemic Poisonings

HOW IS CARBON MONOXIDE POISONING TREATED IN SMOKE INHALATION?

Incomplete combustion of organic materials generates carbon monoxide. It is the most common, serious acute health hazard to victims of smoke inhalation.

Mechanisms of Carbon monoxide poisoning:

- Binds to hemoglobin ("relative anemia")
- Hinders release of O_2 from Hgb to tissues (Left shift of oxyhgb dissociation curve)
- Binds to hemoproteins (myoglobin, cytochrome oxidase)
- Lipid peroxidation
- Neural messenger that activates guanyl cyclase
- Modulates excitatory neurotransmission in CNS at Glutamate (NMDA) receptors

Treatment for CO poisoning is *supplemental oxygen* therapy administered by:

- High flow, tight-fitting mask
- Endotracheal tube
- Hyperbaric oxygen therapy

Hyperbaric oxygen used to treat carbon monoxide toxicity has been shown to decrease burn edema of the skin and airways, preserve marginally viable burn tissue, promote wound closure, enhance host defenses, reduce extravascular lung water in pulmonary injury, and possibly treat cyanide poisoning.

Consider hyperbaric oxygen therapy if it is immediately available AND:

- History of loss of consciousness
- Coma
- Focal neurological findings
- Pregnancy
- Elevated carboxyhemoglobin level (consider if greater than 25%)?
- Any seriously ill patient with isolated smoke inhalation injury should be considered for transport to a facility with hyperbaric oxygen capabilities

Based on current literature, patients with LOW risk for significant sequelae are those with LOW carboxyhemoglobin (COHb) levels AND NO HISTORY OF:

- Syncope
- Coma
- Focal neurologic findings
- Cardiovascular instability
- Pregnancy

- **Treatment Recommendations for LOW Risk Patients**: 100% oxygen therapy (normobaric)

Controversy: lengthy transport of critically ill patients to a facility with hyperbaric oxygen capabilities

Recommendations: Hyperbaric oxygen should be administered to patients with significant associated trauma, burns covering more than 40% of the body, or hemodynamic instability **ONLY after life-threatening conditions have been treated** and the patient's condition has stabilized.

If a patient has a major burn injury in addition to an inhalation injury, consultation with the burn center **BEFORE** transporting to a hyperbaric oxygen facility is necessary. It is recommended that these patients receive hyperbaric oxygen, but not at the expense of expedient, experienced resuscitation and burn care. Many burn units in the United States do not use hyperbaric oxygen.

HOW SHOULD CYANIDE POISONING BE TREATED IN THE PRESENCE OF SMOKE INHALATION?

Cyanide poisoning should be suspected in seriously ill patients with smoke inhalation

High cyanide levels measured in air samples from fires and blood of fire survivors and fatalities

Mechanisms of Toxicity

- Blocks the use of oxygen at cellular level (cytochrome c inhibition)
- Synergistic effects in combination with Carbon monoxide

The Challenge

- The amount of cyanide exposure (dose) and its contribution to the overall toxicity of a patient with smoke inhalation is not predictable
- Hypoxia and other toxins in smoke create similar signs and symptoms to cyanide poisoning. Cyanide toxicity causes agitation, coma, seizures, cardiovascular compromise, and metabolic acidosis.
- No rapid laboratory test exists to confirm cyanide toxicity

Treatment Recommendations

Cyanide poisoning should be suspected in seriously ill patients with smoke inhalation. Consider specific treatment for cyanide toxicity only after life-support measures, including 100% oxygen therapy, are instituted.

Treatment options include:

- Supportive care alone
- Administration of all or part of the Cyanide Antidote Kit
- Hyperbaric oxygen therapy
- Hydroxycobalamin (Someday)

The Cyanide Antidote Kit (amyl nitrite, sodium nitrite, and sodium thiosulfate) is the only antidote for cyanide poisoning available in the United States. Nitrite-induced methemoglobin and sodium thiosulfate work synergistically to detoxify cyanide. Sodium thiosulfate donates sulfur to the enzyme, rhodanese, which converts cyanide to thiocyanate. Sodium thiosulfate has few adverse side effects and can be safely administered to all seriously ill smoke inhalation patients. ***When coma, seizures, cardiac dysrhythmias, hypotension, or metabolic acidosis are present, sodium thiosulfate alone should be given empirically.***

Warning: ***Impairing oxygen-carrying capacity and oxygen delivery to tissues with nitrite-induced methemoglobinemia is a valid concern in the presence of tissue hypoxia from carboxyhemoglobinemia and other factors.*** Remember, pathophysiologic changes of smoke inhalation injury lead to hypoxia; therefore, the most important initial treatment goal is to ensure adequate tissue oxygenation.

The Dispostion

A period of observation and repeated evaluations is the rule with all smoke inhalation exposures.

Patients with smoke inhalation have a spectrum of presentations and clinical courses. Risk factors, clinical findings and diagnostic studies should determine disposition.

Patients with mild symptoms or resolving respiratory symptoms:

- Determine the risk of smoke injury
- If high risk: consider direct visualization of cords or prolonged observation
- If low risk: give detailed discharge instructions for symptoms that indicate worsening condition

Transfer issues:

- Consider transfer of patients only after stabilization of inhalation, burn, other trauma and other medical conditions.
- Burn Centers versus Hyperbaric Oxygen Facility
- When hyperbaric oxygen is indicated, it is recommended that transfer is not at the expense of expedient, experienced resuscitation and burn care. Especially if hyperbaric oxygen is not available at the burn center.

Consultants:

- Medical Toxicology
- Hyperbaric Medicine
- Pulmonary/Critical Care
- ENT
- Burn Surgeon

The Future: Cutting Edge

- Technetium-99m diethylene penta-acetic acid radioaerosol lung scintigraphy for diagnostic testing
- Antioxidants/Free radical scavengers (dimethyl sulfoxide)
- Aerosolized heparin/ N-acetylcysteine
- Ibuprofen
- Perfluorocarbon-assisted gas exchange
- Bronchoscopic lavage with perfluorocarbon
- High frequency percussion ventilation
- Exogenous Surfactants
- Hydroxycobalamine for cyanide poisoning

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