Inhalational Injury: pathophysiology, diagnosis, treatment

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At the end of this lecture you should be able to:

- Describe the physiology of the respiration of the patient with inhalation injury.
- Describe how to perform a respiratory assessment on the patient with inhalation injury.
- Appreciate the importance of airway management in the patients with Inhalation Injury.
Causes of inhalation injury:

- Noxious asphyxiant gases released during thermal decomposition include carbon monoxide (CO) and hydrogen cyanide.

- Other byproducts produced by combustion of furniture and cotton (aldehydes) or rubber and plastics (chlorine gas, ammonia, hydrocarbons, various acids, ketones) produce injury.

- Heat generated during combustion can cause significant thermal injury to the upper airway.

- Particulate matter produced during combustion (soot) can mechanically clog and irritate the airways, causing reflex bronchoconstriction.

- Exposure to metal fumes and fluorocarbons, systemic toxins typically released during industrial fires.
Pathophysiology

- Cell injury and pulmonary parenchymal damage by irritants

- Hypoxemia by interruption of oxygen delivery by asphyxiants

- End organ damage by absorption of systemic toxins through the respiratory tract.
<table>
<thead>
<tr>
<th>Type</th>
<th>Inhalant</th>
<th>Source</th>
<th>Injury/Mechanism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Irritant gases</td>
<td>Ammonia</td>
<td>Fertilizer, refrigerant, manufacturing of dyes, plastics, nylon</td>
<td>Upper airway epithelial damage</td>
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<td>Chlorine</td>
<td>Bleaching agent, sewage and water disinfectant, cleansing products</td>
<td>Lower airway epithelial damage</td>
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<td>Sulfur dioxide</td>
<td>Combustion of coal, oil, cooking fuel, smelting</td>
<td>Upper airway epithelial damage</td>
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<td></td>
<td>Nitrogen dioxide</td>
<td>Combustion of diesel, welding, manufacturing of dyes, lacquers, wall paper</td>
<td>Terminal airway epithelial damage</td>
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<tr>
<td>Asphyxiants</td>
<td>Carbon monoxide*</td>
<td>Combustion of weeds, coal, gas, heaters</td>
<td>Competes for oxygen sites on hemoglobin, myoglobin, heme-containing intracellular proteins</td>
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<tr>
<td>Hydrogen cyanide†</td>
<td>Burning of polyurethane, nitrocellulose (silk, nylon, wool)</td>
<td>Tissue asphyxiation by inhibiting intracellular cytochrome oxidase activity, inhibits ATP production, leads to cellular anoxia</td>
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<tr>
<td>Hydrogen sulfide‡</td>
<td>Sewage treatment facility, volcanic gases, coal mines, natural hot springs</td>
<td>Similar to cyanide, tissue asphyxiant by inhibition of cytochrome oxidase, leads to disruption of electron transport chain, results in anaerobic metabolism</td>
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<tr>
<td>Systemic toxins</td>
<td>Hydrocarbons</td>
<td>Inhalant abuse (toluene, benzene, Freon); aerosols; glue; gasoline; nail polish remover; typewriter correction fluid; ingestion of petroleum solvents, kerosene, liquid polishes</td>
<td>CNS narcosis, anesthetic states, diffuse GI symptoms, peripheral neuropathy with weakness, coma, sudden death, chemical pneumonitis, CNS abnormalities, GI irritation, cardiomyopathy, renal toxicity</td>
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<td>Organo Phosphates</td>
<td>Insecticides, nerve gases</td>
<td>Blocks acetylcholinesterase, cholinergic crisis with increased acetylcholine</td>
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<tr>
<td></td>
<td>Metal fumes</td>
<td>Metal oxides of zinc, copper, magnesium, jewelry making</td>
<td>Flulike symptoms, fever, myalgia, weakness</td>
</tr>
</tbody>
</table>
Pathophysiology

- Cell injury and pulmonary parenchymal damage by irritants
- Hypoxemia by interruption of oxygen delivery by asphyxiants
- End organ damage by absorption of systemic toxins through the respiratory tract.
Cell injury and pulmonary parenchymal damage

- Thermal or chemical damage to the epithelial surfaces of the intrathoracic and extrathoracic airways lead to respiratory embarrassment.

- Secondary insult with bacterial pneumonia may occur days after inhalation, causing further cytotoxic damage. Ciliary function is impaired, leading to accumulation of airway debris. The inflammatory cascade initiates neutrophil infiltration.
Hypoxemia:

- a decrease in inspired oxygen concentration at the scene of injury,
- a mechanical inability to exchange gas because of airway obstruction or parenchymal pulmonary disease,
- inhibition of oxygen delivery and tissue use by toxins.
Symptoms

• Inhalation injury does not always cause noticeable symptoms initially.

• All burn victims must be watched carefully for inhalation injuries.

• Inhalation injury should be suspected in anyone trapped in a building fire or who has lost consciousness during a fire or when there is heavy smoke.

• Victims may have a cough, hoarse voice, or sore throat.

• If the injury involves the lower part of the respiratory tract, then they may have chest discomfort or pain or difficulty breathing.
Symptoms (in relation to CO level)

- Between 0 and 10% is not dangerous.
- Heavy smokers commonly have levels approaching 10%.
- Between 10 and 20% a person may experience nausea and a pounding headache.
- Drowsiness and an overall feeling of weakness occur at levels between 20 and 30%.
- Above 30%, confusion and agitation are common.
- Over 40% causes coma and death occurs at levels above 50%.
Signs

• Dyspnoea
• Facial or neck burns
• Singed hair of the eyebrows or in the nose
• Oral mucosal hyperaemia and ulceration
• Wheezing
• Sputum that is black or sooty.
Clinical indications for intubation

absolute:

- Burn of the palate, tongue and pharynx
- Oedema of the posterior pharynx and upper glottis
- Burn of vocal cords

relative:

- Hoarseness
- Facial burn
- Sooty sputum
Diagnosis

- Clinical
- Bronchoscopic
- Blood gas assessment
- Xenon 133 lung scan
- Pulmonary function studies
Fig. 2. A normal pulmonary scan consisting of (A, left) uniform perfusion, (B, right) equal distribution before clearance.
Fig. 2C (left). Clearance of isotope almost complete at 88 seconds, (D, right) complete clearing with no "hot spots."
Targeted arterial blood gas goals

pH 7.25–7.45

PaO2 55–80 mmHg or SaO2 of 88–95%

PaCO2 35–55 mmHg
(permissive hypercapnia can be used if pH 7.25)
Management

• Airway issues and early intubation
• Bronchial hygiene therapy
• Chest physiotherapy
• Early ambulation
• Airway suctioning
• Pharmacological adjuncts
A management protocol

• Titrate humidified oxygen to maintain SaO2s’ > 90%

• Cough, deep breath exercises every 2 h

• Turn patient side to side every 2 h

• Chest physiotherapy every 4 h

• Aerosolize 3 cc’s of 20% N-acetylcysteine every 4 h with a bronchodilator

• Alternate aerosolizing 5000 units of Heparin with 3 cc’s of normal saline every 4 h

• Nasotracheal suctioning as needed

• Early ambulation Sputum cultures for intubated patients
Complications

- Tracheal stenosis
- Obstructive/restrictive airway
• http://emedicine.medscape.com/article/1002413-overview

• http://emedicine.medscape.com/article/771194-overview

• http://www.thedoctorwillseeyounow.com/articles/other/burns_23/