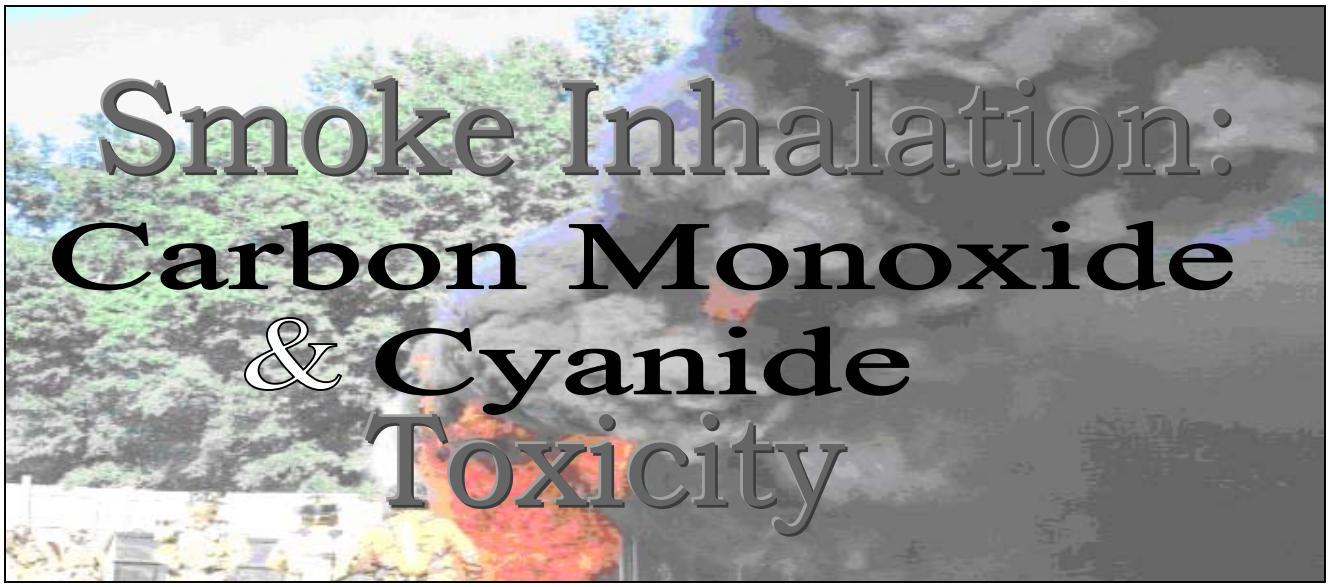


OVERVIEW

Exposure to smoke can lead to a variety of severe complications in the fire victim as well as the fire fighter. In fact, the leading cause of death in fires is caused by smoke inhalation, not the burn from a fire. The smoke also makes escape much more difficult.

Death or disability at the scene from smoke is caused by smoke toxins, especially carbon monoxide, cyanide and lack of oxygen. It is also important to note that the lung damage caused by smoke toxins can lead to airway obstruction several hours later. Ultimate damage to the lung itself is the leading cause of disability and death in the hospitalized burn patient. The discussion of the topic of smoke inhalation will be divided into two sections, “The Silent Killers,” and Direct Lung Damage from Smoke Toxins.

Exposure to Incomplete Products of combustion is the leading cause of death in fires

The “Silent Killer,” Carbon Monoxide, and Cyanide, commonly found in any smoke are rapidly absorbed from the lung and into the blood and then quickly turn off the body’s ability to obtain and utilize oxygen. The victims become oxygen starved. Symptoms can be subtle usually beginning disorientation, with greater exposures resulting in a coma followed by cardiac arrest, and death.

CARBON MONOXIDE TOXICITY

Carbon Monoxide (CO) is a colorless, odorless gas produced by the combustion of any carbonaceous material which means any smoke or fire. Improperly vented gas, water heaters, auto exhaust stoves all release CO in addition to any flammable object.

Carbon monoxide, CO, is rapidly transported across the lung and into the blood where it preferentially binds with the hemoglobin molecule in place of oxygen. Because CO leads to hemoglobin 250 times more avidly then oxygen, even small amounts of CO exposure to CO over long periods can lead to significant toxicity.

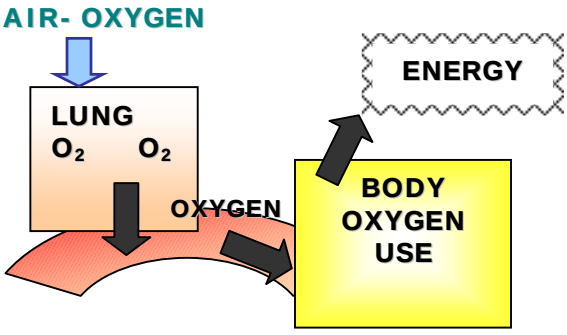
The result is a major impairment in oxygen delivery to the body, since 98% of oxygen is carried to the tissues on hemoglobin. Peak CO toxicity will be at the scene when the victim is first removed from the smoke exposure. The magnitude of the carbon monoxide toxicity roughly corresponds with the peak percent of the circulating hemoglobin bound by carbon monoxide, (carbonboxyhemoglobin (COHgb) level).

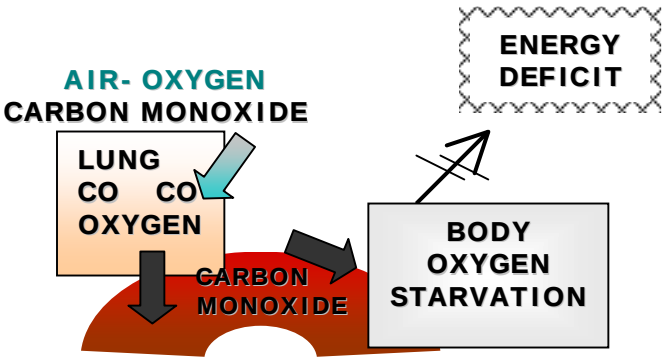
SYMPTOMS

- Mild Toxicity *(reversible)*
  - disorientation, confusion
- Moderate Toxicity *(reversible)*
  - hallucination, fatigue
  - nausea
  - blurry vision
- Severe toxicity *(often reversible)*
  - coma
  - shock like state
  - cardio pulmonary arrest
  - death

✶ Neurological changes with severe toxicity can be permanently white

CO TOXICITY





CARBON MONOXIDE TOXICITY		
CO Hgb	✳ SYMPTOMS RELATIVE TO CARBOXY HEMOGLOBIN LEVEL	
0-5	**Normal value**	**SKIN COLOR**
15-20	**Headache-confusion**	
20-40	**Disorientation, fatigue, nausea, visual changes**	
40-60	**Hallucination, combativeness, coma, shock state** *Note: skin color and mucus membrane color remains pink to red COHgb is red in color*	
60 or above	**Cardiopulmonary arrest**	

✳**CO Hgb – carboxyhemoglobin**

DIAGNOSIS

- History of smoke exposure or possible CO exposure
- Symptoms compatible CO toxicity
- Elevated carboxyhemoglobin levels (COHgh > 5%)

The Carbon Monoxide is rapidly removed from Hemoglobin using 100% O_2 with half of the CO removed every 20 minute O_2 exposure.

PREVENTING CO EXPOSURE

Use of a CO detector in the home as a warning against exposure

TREATMENT

Treatment of carbon monoxide toxicity consists of the early displacement of carbon monoxide from hemoglobin by administration of 100% oxygen.

Endotracheal intubation and use of 100% oxygen with mechanical ventilatory assistance is indicated for those patients with severely impaired neurologic function and a risk of CO toxicity.

Hyperbaric oxygen (2 to 3 atm) produces an even more rapid displacement and is most useful in cases of prolonged exposure.

Hyperbaric oxygen is best used in cases in which the patient has neurologic compromise, but not requiring life support or close monitoring.

Treatment of CO Toxicity

Awake	Obtunded
High flow by mask oxygen ($Fi0_2$ 100%) until CoHgb is between 5-10%	Intubate Use 100% oxygen via positive pressure ventilation Hyperbaria used if patient not responding to 100% oxygen alone (specific indications remain undefined)

CYANIDE TOXICITY

Cyanide gas is released as a by product of combustions from a number of synthetic materials. Cyanide gas is rapidly absorbed from the lung into the blood, where once carried to the tissues, blocks the production of energy. The result is a rapid energy crises and organ failure. The oxygen cannot be used. Cyanide in the gas form as in smoke leads to toxicity much faster than if given in any other form such as ingestion.

SOURCES: BURNING

- **Wool, Plastic**
- **Polyurethane**
  - car seat
  - upholstery
  - mattresses

Common Source of Cyanide Toxicity is combustion of mattresses

DIAGNOSIS

- High index of suspicion
- Symptoms comparatable with cyanide toxicity
  - high heart rate
  - shock state
  - pink color
  - oxygen debt
- Blood value cyanide over 0.1 mg/liter/blood
- Toxicity value is over 0.1 mg/liter/blood
- Fatal value is over 1.0 mg/liter/blood

TREATMENT

There are two components. The <u>first</u> is to restore blood volume and adequate circulation as the liver can detoxify the cyanide, thru the enzyme rhodenase so restoration of adequate circulation is key.

The <u>second</u> approach is the pharmacological alteration of cyanide. This approach is not used routinely in burn patients but only if there is a concern for cyanide toxicity. The unexplained persistent metabolic acidosis is an active acidosis in this case.

Editors

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An unexplained metabolic acidosis

Pharmacological Treatment	
Sodium Nitrate then	**10ml, 3% solution IV over 3-5 minutes in adults**
Sodium Thiosulfate	**12.5g IV over 10 minutes and product is Sodium Thiocyanate**
(Any <u>NITRATE</u> can be used as temporary treatment)	