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## TOXICOLOGY OF SMOKE INHALATION

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In the spring of 2003, two firefighters from a large western city were critically exposed to smoke within a few weeks. One died; the other survived. Both were in peak physical condition, well trained, and very experienced. Both were rescued after becoming unconscious while making an initial attack in a single-family residential fire. Both were found with their masks on and air in their tanks. What caused them to become unconscious? Why were they unable to call for help?

Inhalation of toxic smoke is the primary cause of death from fires. It is a major cause of firefighter death. Smoke inhalation causes acute life-threatening injuries and results in long-term lung and neurological damage. Many toxic products are released during a typical room-and-contents fire. A multitude of variables make it difficult to predict what toxins will be produced. As a result, most information is from animal and autopsy studies. Unfortunately, some toxins, like cyanide, are very difficult to measure. Cyanide's role in smoke inhalation toxicity is just beginning to be understood.

Autopsy and experimental data show that serious injury and death result from exposure to contact irritants, primarily hydrogen chloride, and the central systemic poisons, carbon monoxide (CO) and cyanide.<sup>1</sup> Contact irritants cause cellular damage and death. In response to irritants, cells release fluids, causing massive edema. Additional inflammatory responses cause cells to lose integrity and die.<sup>2</sup>

Systemic poisons are absorbed into the blood through the lungs. They act on specific cells in the body or within specific parts of every cell. Systemic poisons either inhibit critical cell functions or cause cellular death.

Contact irritants include particulate matter such as soot. Particles larger than five microns will lodge in the upper airways, causing mechanical obstruction. They are observed in the nose and the mouth. Particles smaller than one micron are inhaled deep into the lungs, where the carbonaceous soot is toxic to the macrophages. Macrophages are cells that remove foreign particles. Heavy metals coating the surface of soot cause direct lung damage by forming free oxygen radicals which damage cilia and alveolar surfaces.

Fires generate irritating and corrosive gases. Aldehydes and acrolein are released when wood and cellulose burn. These products cause intense tearing, coughing, and choking. Acrolein is highly toxic to lung tissue, causing protein destruction in the deep lung tissue.

Degradation of plastics creates most of the corrosive gases found in fires. Plastics that contain a chlorine molecule such as polyvinyl chloride are deadly. Burning of these plastics forms hydrogen chloride, phosgene, and hydrochloric acid. Hydrogen chloride is the most toxic of these three; its contact with moist mucosa results in the formation of hydrochloric acid. The eyes, oral pharynx, and upper airway are immediately affected; upper airway edema results. If superheated air has also been inhaled, swelling can be severe and cause rapid, significant airway obstruction. Because hydrogen chloride is only moderately water soluble, it will be inhaled into the bronchus and smaller airways. Tissue response includes swelling, cilia paralysis, and massive fluid leak into the smaller airways. Bronchospasm and bronchorrhea will present as wheezing and pulmonary edema. Phosgene is a combination of chlorine, hydrogen, and carbon molecules. It is commonly present in smoke. Phosgene is poorly soluble in water and is inhaled deep into the lungs. When phosgene contacts moisture in the terminal alveoli, hydrochloric acid is formed, and the terminal alveoli are damaged.

Inhalation of these irritants initially causes tearing, coughing, and gagging. If the victim continues to breathe the smoke, damage and swelling to the upper airways will ensue. Deeper airways spasm and swell, and terminal airways are destroyed. Because the cilia are damaged, soot and fluid cannot be removed from the lungs. The alveoli collapse, tissues die and slough away, large amounts of fluid leak into the lungs, and the victim dies from respiratory failure. Serious symptoms may be delayed for up to 48 to 72 hours, although the victim initially may appear to have minor respiratory distress.<sup>3</sup> Systemic cellular toxicants cause sudden unconsciousness and death. Their presence depends on several factors: the length of exposure, availability of oxygen, position of the victim, amount of heat present, stage of the fire, and material being burned. Other systemic toxins

are produced, but CO and cyanide are so deadly that toxicity from other gases is rare. It previously was thought that a low oxygen level was the cause of death in smoke inhalation, but recent studies suggest otherwise. (3) Even in fires with low oxygen levels, CO and cyanide are the greatest threats.

### **CARBON MONOXIDE**

CO is present in all fires. Thermal degradation of polystyrene plastic releases large quantities of CO. The ability of CO to inhibit the attachment of oxygen to hemoglobin is well known. CO also interrupts the transfer of oxygen from the hemoglobin to the cell. Additionally, CO prevents the capability to store oxygen in muscle cells, principally the cardiac muscle. This triple toxic effect explains why CO is assumed to be the primary cause of death from smoke inhalation. Yet, it takes very high CO concentrations to poison humans (20 percent carboxyhemoglobin for toxic effects and greater than 50 percent to cause death). Twenty minutes of active burning may be required to develop sufficient concentrations of CO to reach lethal levels.<sup>4</sup> But once exposed, 40 percent of those with severe poisonings will have long-term neurological impairment, including cognitive (emotional/behavior) dysfunction, short-term memory effects, and sensory motor (vision) problems. (4)

### **CYANIDE**

The presence of cyanide is difficult to measure. It easily degrades into nitrogen oxides in a combustible atmosphere. Cyanide formation results from burning materials containing a nitrile or cyano group. These groups contain a carbon and a nitrogen molecule bonded together. Plastics, primarily polyurethane foam, melamine, abs (plastic pipe), and nylon, will release cyanide. Cyanide is a highly toxic gas. In a closed room, the cyanide released from the combustion of a simple seat cushion is sufficiently toxic to poison the entire room.<sup>5</sup> Cyanide formation is greatest in fire situations that have low-heat and low-oxygen levels. When inhaled in a low-oxygen environment, cyanide becomes 10 times more toxic.<sup>6</sup> Exposure to hydrogen cyanide results in the loss of consciousness within 30 seconds, apnea in three to five minutes, and cardiac arrest in five to eight minutes.

Cyanide is easily absorbed into the blood and easily released into cells. It affects all cells by preventing the use of oxygen in energy production. The cells become profoundly acidic. Cellular function slows and then fails in an acidic environment. The brain, lungs, and heart are the organs affected. Cyanide inhalation rapidly results in respiratory depression and

arrest. The victim may not have enough time to inhale toxic levels of CO or suffer pulmonary irritation and damage. Without measuring the cyanide level, which is done in U.S. hospitals, the victim may not have evidence of severe smoke inhalation. In addition, inhalation of cyanide and CO together appears to be synergistic, thus explaining why death occurs with lower than lethal levels of CO or cyanide.<sup>7</sup>

CO and cyanide are odorless and undetectable by humans. When exposed, the victim becomes confused, unable to think rationally, and disoriented. Loss of consciousness soon follows. The brain shuts down, the respiratory muscles become fatigued, and breathing slows or stops. The cardiac muscle becomes toxic, the pulse slows, cardiac output falls, myocardial damage occurs, and cardio/pulmonary arrest results. If the patient is rescued and treated before the cessation of cardiac activity, recovery is possible.

### **OXYGEN REQUIREMENTS**

Firefighters exposed to smoke while conducting firefighting operations are at much higher risk than the civilians rescued. The role of the individual's oxygen requirements at the time of exposure appears to be critical. Most civilians are at rest and have minimal oxygen needs; they most frequently are "hugging" the floor, where smoke toxicity is lowest. Civilians are most often rescued when a fire is in the free-burning phase, hence oxygen is present and smoke toxicity is minimal.<sup>8</sup>

During searches and fast attack mode, firefighters are working at maximum or above maximum oxygen requirements. Active searches often occur in rooms that have the highest levels of toxins. Even momentary exposure may introduce enough toxins to begin a cascade of events that result in tragedy.

### **TREATMENT**

Because of the very toxic mechanisms of smoke inhalation, resuscitation must begin as soon as possible to be successful. Initial treatment and administration of antidotes should begin as soon as the victim is rescued. Clinical signs should direct treatment. Aggressive treatment and a very high index of suspicion are paramount.

Treat victims from a closed space fire presenting with seizures, GCS < 8, hypotension, and cardiovascular collapse very aggressively for both CO and cyanide poisoning.<sup>9</sup>

Early signs of toxic exposure include a decrease in cognitive ability, hypotension, confusion, anxiety, a decreased level of consciousness, a rapid pulse, poor tissue perfusion, rapid breathing, and dyspnea. Interpret an arterial blood oxygen saturation (SaO<sub>2</sub>) level with care. A low reading may indicate pulmonary damage, but a normal level means absolutely nothing. Conventional oxygen oximetry does not recognize CO in the blood and will record a normal SaO<sub>2</sub> in the face of severe CO poisoning. A pulse CO-oximeter can differentiate carboxyhemoglobin from oxyhemoglobin and provide valuable information when you suspect CO poisoning. A field carboxyhemoglobin level is important, but severe smoke inhalation can present with subtoxic levels.

Because cyanide does not allow oxygen to release from the hemoglobin, it will be fully saturated with oxygen, even on the venous side. The SaO<sub>2</sub> will read artificially high, and venous blood will appear very red. A correlation between the CO level and cyanide level has not been proven.

There is no direct measure for the cyanide level. Serum lactate can easily be measured and has a sensitivity of 90 percent for cyanide toxicity if above 10mmol/l.<sup>10</sup> This test is done in the emergency department. Serum lactate can also be done in the field using a point-of-care device such as an iSTAT®. Without a serum lactate level, assume that cyanide is present in every fire and in every victim.

Because even low levels of CO and cyanide exposure have delayed and long-term effects, all victims removed from an enclosed space fire should be on 100-percent oxygen while an examination is conducted.

Consider early tracheal intubation if there is evidence of upper airway edema or respiratory insufficiency. Insert the largest possible endotracheal tube. Hospital treatment will require a bronchoscopy, and a large tube facilitates this procedure. The use of Rapid Sequence Intubation (RSI) has to be balanced with the fact that the airway may rapidly obstruct and bag/valve ventilation may be impossible. A rescue cricothyroidotomy should be available before RSI is attempted. Simple sedation may be all that is required for intubation. Treat wheezes with inhaled beta agonists. Copious tracheal suction may be required. Specialized pulmonary intensive care centers are required for successful long-term care.

According to Dr. William Hurley, director of the Washington Poison Center, treat any patient from an enclosed space fire with an altered level of consciousness with hydroxocobalamin (the current treatment for cyanide poisoning). This drug should be available at the scene to be most effective. If the airway is at risk or if hydroxocobalamin is not available, immediately transport the patient to the nearest emergency department.

If the airway is controlled, institute antidotal treatment for CO and cyanide. Then, transport the patient to the tertiary facility. The use of hyperbaric oxygen may be indicated. Its use is controversial, so contact local medical control for direction. Because the presentation of corrosive lung injury may be delayed several hours, observe and evaluate all patients who were in an enclosed space fire delayed injury.

Toxic exposure to products of combustion can occur during overhaul. Many departments use CO levels to determine the safety of the fire scene. Because cyanide is produced at much lower temperatures than CO, cyanide may be present when the CO level is safe. Soot that is disturbed becomes airborne and inhaled.

Breathing masks recently have been marketed that claim to protect the wearer from particulates, cyanide, and CO. Currently, these masks are not certified by the National Institute for Occupational Safety and Health (NIOSH) or the National Fire Protection Association. Until your department has the capability to properly protect you from these toxins or measure cyanide levels, wear your SCBA during overhaul and possibly during the investigation.

Conducting fire operations outside a structure reduces the danger of toxic smoke inhalation but does not eliminate the threat. NIOSH Firefighter Fatality Report F2003-36 investigated the death of a firefighter from CO inhalation during a wildland fire, and the disability of individuals related to chronic pulmonary conditions caused by exposure to the New York World Trade Center fires on 9/11 testify to the dangers of low-level smoke exposure while outside a structure.

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What happened to the firefighters discussed at the beginning of this article? The firefighter that survived had just relinquished the nozzle to his lieutenant. Because of extreme heat and smoke, they were unable to access the source of the fire (it was a basement fire; they

were at the top of the stairs). A truck company conducting ventilation stumbled into the firefighter. He was not moving and was unresponsive. His PASS alarm had not activated. They rapidly rescued him from the house. He was in respiratory arrest and possibly cardiac arrest. Paramedics already on the scene rapidly intubated and resuscitated the patient. He had no burns, and his airway was not injured. He was transported to the hospital in a coma. CO levels were not available at the scene. At the hospital, his CO level was normal. He awakened 12 hours later, without injury, and has returned to the fire service.

The officer that died had been initiating a “fast attack” because of a trapped civilian. The officer and two firefighters entered the house and were met with “moderate smoke conditions in the kitchen and donned their masks.” They went up a set of stairs, looking for a bedroom fire. At the top of the stairs, they were in a very cluttered hallway and encountered heavy smoke and heat. They entered a small bedroom to look for the victim and the fire. The officer began a left-handed search; the two firefighters began a right-handed search with the nozzle. After pulling the ceiling and still not finding any fire, the firefighters thought they heard the officer order them out of the room. They exited, thinking the officer was behind them.

A truck company at the top of the stairs heard the PASS alarm and found the officer unconscious under a mattress in the small bedroom. He was removed with difficulty and was in cardiac arrest. The medics at the scene initiated resuscitation efforts. He never regained consciousness and died a week later from hypoxic brain injury secondary to smoke inhalation. He had a lung injury consistent with corrosives from smoke inhalation, and his CO level was 24 percent on arrival at the hospital.<sup>11</sup>

Because both firefighters were found with their masks on and air in their tanks but were suffering from serious smoke inhalation, the possibility of equipment malfunction was considered. However, their breathing apparatus were determined to be in normal working condition. The firefighter who survived became unconscious for unknown reasons. He survived because of his limited time in the structure and aggressive treatment by the medics at the scene. I suspect that he had a momentary exposure to cyanide, enough to render him unconscious.

It is obvious that the firefighter who died had significant exposure to hydrogen chloride, CO, and probably cyanide. It is likely that his mask did not have a full seal while he was

down and under the mattress. Because this happened in 2006, hydroxocobalamin was not available in the United States. No mention of cyanide was found in his autopsy record.

It is never "just a room-and-contents" fire. Toxins are often the greatest in this type of fire, frequently worse in the rooms above the fire. You must be careful and vigilant. You must wear your breathing apparatus not only for the interior attack but also during overhaul. A high index of suspicion and aggressive treatment is necessary for any case of suspected smoke inhalation, not only for the acute danger but also for the chronic disability that may follow.

### Endnotes

1. Alarie, Y. "Toxicity of Fire Smoke," *Critical Reviews in Toxicology*, 2002; 32(4):259-289.
2. Guy JS, MD Peck, "Smoke Inhalation Injury: Pulmonary Implications," *MedGenMed*, 1999; 1(3). [Formerly published in *Medscape Pulmonary Medicine Journal*, 1999; 3(2), <http://www.medscape.com/viewarticle/408744/>.]
3. Cone DC, D MacMillan, V Parwani, C Van Gelder, "Threats to life in residential structure fires," *Prehosp Emerg Care*, Jul-Sept, 2008;12(3):297-301.
4. Gilmer B., J Kilkenny, C Tomaszewski, & JA Watts, "Hyperbaric oxygen does not prevent neurologic sequelae after carbon monoxide poisoning," *Acad Emerg Med Jrn*; 2002;9 (1), 1-8.
5. Borron, S. Emergency physician's role in appropriate treatment of cyanide poisoning in smoke inhalation. Oral presentation, Seattle, Wash., 10/09/2008.
6. Tuovinen H , P Blomqvist, "Modeling of Hydrogen Cyanide Formation in Room Fires." SP Swedish National Testing and Research Institute, SP Report 2003:10 Box 857, SE-501 15, BORAS, Sweden, 2003, ISBN 91-7848-941-5.
7. Griggs TR. "The role of exertion as a determinant of carboxyhemoglobin accumulation in firefighters," *Journal of Occupational Medicine*; 1997 Nov;19(11):759-61 from <http://www.ncbi.nlm.nih.gov/pubmed/915571/>.
8. O'Brien, D. J Augustine, D. Walsh. "Cyanide Exposure, Smoke Inhalation, and Pre-Hospital Treatment: Recognizing the Signs and Symptoms and Available Treatment

Options," "Smoke, Cyanide and Carbon Monoxide, The Toxic Twins of Smoke Inhalation," Cyanide Poisoning Treatment Coalition, Indianapolis, Ind, 2009, [www.FireSmoke.org/](http://www.FireSmoke.org/).

9. Baskin S, T Brew. "Cyanide poisoning: Medical aspects of chemical and biological warfare," Chapter 10. U.S. Army Medical Research Institute. 2000, 271-286, [jepmadmin.org/VolumeVI/Issue1/Abs.spring.04/](http://jepmadmin.org/VolumeVI/Issue1/Abs.spring.04/).

10. Baud FJ, P Barriot P, V Toffis, et al, "Elevated blood cyanide concentrations in victims of smoke inhalation," *N Engl J Med*. Dec 19, 1991; 325(25):1761-1766. On [Medline].

11. National Institute for Occupational Safety and Health. (2007). Career Lieutenant Dies in Residential Structure Fire. (NIOSH, 200619), <http://www.cdc.gov/niosh/fire/reports/face200619.html/>.

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