

## **Carbon Monoxide**

### **Introduction**

In the United States, carbon monoxide (CO) is responsible for more morbidity and mortality than any other single poison. Exposures to CO are often difficult to diagnose as the symptoms are vague and frequently mimic upper respiratory infections. Health care practitioners must have CO poisoning constantly in their minds so as not to miss this common problem. While CO poisoning is more common during the winter months, CO exposures can occur year round.

In the year 2004, the American Association of Poison Control Centers answered 17,115 calls regarding carbon monoxide exposures. Of these, 62% were mild exposures and managed in the home. The others (6,427) were managed in a healthcare setting with the majority being treated and released. While this data reflects poison control center data, it is estimated that the majority of CO exposures nationwide are not reported to poison centers.

### **Background and mechanism of toxicity**

Carbon monoxide is a colorless, odorless gas that is produced anytime there is incomplete combustion of organic (carbon containing) materials. While the most common sources of CO are gas stoves, furnaces, automobiles and house fires, CO poisoning has also resulted from breathing the exhaust from electrical generators, gasoline-powered high pressure washers and house boats.

Carbon monoxide binds to hemoglobin with an affinity 210-280 times greater than oxygen. It also causes the hemoglobin to hold onto oxygen more tightly than normal, thus preventing the release of oxygen to the tissues. The end result is tissue hypoxia. The areas most affected are those which have high oxygen demands, especially the brain and the heart. Hypoxia does not explain all of the toxicity associated with CO. Carbon monoxide also diffuses into cells and binds to cytochromes in the oxidative phosphorylation system. This inhibits the generation of energy and also leads to the production of oxygen free radicals (O<sup>-</sup>, OH<sup>-</sup>, H<sub>2</sub>O<sub>2</sub>) which lead to further tissue damage. When exposed to CO, white blood cells adhere to the vascular endothelium. When these white blood cells are later re-exposed to oxygen, they release cytokines which cause lipid peroxidation of cell membranes. This lipid peroxidation has been shown in animals to be responsible for the permanent and delayed effects of CO on the central nervous system.

### **Symptoms**

The clinical symptoms of CO poisoning are highly variable and there is very poor correlation between CO blood levels and the patient's symptoms. Mild symptoms are often mistaken for other diseases, such as viral illness, the flu, gastroenteritis, or food poisoning. Symptoms of a mild CO intoxication include headache and nausea. Moderate exposures result in more severe headaches, nausea, vomiting, difficulty

thinking, confusion, lethargy, weakness, or shortness of breath. More severe symptoms include chest pain, EKG changes, syncope, seizures, obtundation or loss of consciousness, cardiac dysrhythmias or cardiac arrest, respiratory distress, or cyanosis. Any condition that increases the body's oxygen demands, such as exertion, trauma, burns, MI, CVA or concurrent drug ingestions, will produce more severe symptoms.

It is also important to recognize secondary injuries associated with CO toxicity. These can include cardiomyopathy, rhabdomyolysis, non-cardiogenic pulmonary edema, multiorgan failure, disseminated intravascular coagulation (DIC), persistent neurologic sequelae, and delayed neurologic sequelae. All of these injuries require further workup and hospitalization. The neurologic sequelae may present from 2 to 28 days from time of exposure and may occur in as many as 15 percent of severely poisoned patients.

### **Evaluation of Carboxyhemoglobin and Ambient CO levels**

Carboxyhemoglobin (COHb) levels should be part of the patient's evaluation, but historically, these levels do not correlate well with patients' symptoms. Levels of COHb drawn at the scene of the exposure are sometimes more clinically useful in the evaluation and, if possible, ambulance services should draw CO levels at the scene in patients who have a significant CO exposure. Another tool in evaluation of a CO exposure is to have the fire department measure ambient levels of CO at the scene. However, if the building has been aired out before the fire department arrives, their measured ambient CO levels may not reflect CO levels at the time of the patient's exposure. It is important to remember that oxygen saturation measured by pulse oximetry in the setting of COHb will be inaccurate, since pulse oximeters can not distinguish between carboxyhemoglobin and oxyhemoglobin.

### **Laboratory**

The following laboratory studies should be performed on patients with CO exposures: (a) carboxyhemoglobin level, (b) serum chemistries to look for acidosis, (c) a complete blood count to look for anemia (patients with anemia are at a higher risk for tissue hypoxia), and (d) an arterial blood gas if the patient is having respiratory problems or there is concern about the patient's oxygenation status. Most patients should have an EKG checked unless they are at very low risk for cardiac ischemia. In the setting of intentional CO poisoning, a urine toxicology screen and blood alcohol level should also be considered. If the patient was involved in a fire, the possibility of cyanide exposure from burning plastics should be considered, and if suspicion is high enough, treat for cyanide intoxication also. While any patient with mental status changes should receive an appropriate neurological evaluation, an MRI or PET scan can be considered to evaluation for CNS sequelae.

### **Special Populations**

Certain populations are more susceptible to the deleterious effects of carbon monoxide exposures. Younger children can be more difficult to evaluate since they can not describe their symptoms. Also, children have a higher respiratory rate than adults and will breathe in more CO than an adult in the same environment. Older people with pre-

existing medical conditions, such as coronary artery disease, cerebral artery disease and COPD, are at higher risk for toxicity due to already compromised physiology.

A special population that is important to recognize are pregnant patients. The fetus can be greatly affected by CO exposure because fetal hemoglobin has an even higher affinity for CO than adult hemoglobin. Pregnant females should be treated very aggressively for CO exposure and early referral for hyperbaric oxygen should be considered.

Another population at increased risk of CO poisoning are people who are either recent immigrants or are non-English speaking. Immigrants who are used to using cooking fires in their previous, well ventilated, home may not intuitively understand that an indoor fire can be quite dangerous. Also, it may not be intuitive that a home stove should not be used to heat the house. Use of an oven or stove to heat the home is a significant risk factor for CO poisoning. Education materials addressing the dangers of CO and sources of CO may not be accessible to these populations due to language barriers or literacy issues.

### **Treatment**

The patient should immediately be removed from the source of carbon monoxide. After removal from the source, high concentrations of oxygen should be given to the patient since oxygen is the antidote to CO toxicity. The half life of COHb, while breathing room air, is approximately 320 minutes. With administration of 100% oxygen, the half-life is decreased to about 60 minutes. The addition of hyperbaric oxygen (100% oxygen at 2.8 times the normal atmospheric pressure) lowers the half-life to 23 minutes. Patients with mild to moderate symptoms can be treated with oxygen and observation for 3 to 6 hours, and if symptoms are resolved, they can be discharged to home. If there are severe symptoms, or if there is no improvement with normobaric oxygen, hyperbaric oxygen should be considered.

Hyperbaric oxygen (HBO) in the treatment of CO toxicity continues to be a very controversial issue. There are no good randomized studies that definitively prove whether or not HBO is beneficial in treating CO poisoning. Many small non-randomized studies have reported a benefit. Weaver et al. found that hyperbaric-oxygen treatment significantly reduces the incidence of carbon monoxide-induced delayed neurologic sequelae. They also found that for every six patients treated, one case of delayed neurological sequelae could be avoided.

Generally accepted indications for HBO include any signs of serious toxicity, such as altered mental status, history of loss of consciousness, focal neurological deficits, seizures, syncope, ischemic chest pain, EKG changes, new dysrhythmias, hypotension, or shock. COHb levels are generally not as helpful in the decision to use HBO as clinical signs, but most hyperbaric chambers have some guidelines.

At Hennepin County Medical Center, the indications for HBO are:

- 1) COHb level > 25% in the setting of patients with cardiovascular or cerebrovascular disease, age >60 years or <2 years, Hb <10 or exposure >5 hours
- 2) COHb level >40%
- 3) Pregnancy with COHb >20%, exposure time >5 hours, or signs or fetal distress.

At the Iowa and Wisconsin PCC's indications for HBO are:

1. COHb >20%
2. Coma, seizures, syncope, respiratory distress
3. Myocardial ischemia
4. Persistent CNS changes even in the presence of 100% O<sub>2</sub>
5. Pregnancy
6. Metabolic acidosis due to CO exposure\*

\* Iowa Statewide Poison Center indication

Consultation with a physician trained in hyperbaric medicine may be of benefit if the treating physician has questions about the appropriateness of HBO in a specific patient.

## References

Tintinelli et al. Chapter 198 Carbon Monoxide Poisoning. Keith W. Van Meter. Emergency Medicine: A Comprehensive Study Guide. McGraw-Hill pp 1302-5, 2000.

Ernst and Zibrak. Carbon Monoxide Poisoning. New England Journal of Medicine. 339(22) pp 1603-8, Nov 1998.

Hardy and Thom. Pathophysiology and Treatment of Carbon Monoxide Poisoning. Clinical Toxicology. 32(6) pp 613-29, 1994.

Juurlink, Stanbrook, and McGuigan. Hyperbaric Oxygen for Carbon Monoxide Poisoning. Cochrane Database of Systematic Reviews. Issue 2, 2001.

Piantadosi, Claude A. Carbon Monoxide Poisoning. New England Journal of Medicine. 347(14) pp 1054-55, Oct. 2002.

Shochat and Lucchesi. Toxicity, Carbon Monoxide. eMedicine Journal. 2(5) May 2001. <http://www.emedicine.com/EMERG/topic817.htm>

Weaver et al. Hyperbaric Oxygen for Acute Carbon Monoxide Poisoning. New England Journal of Medicine. 347(14) pp 1057-1067, Oct. 2002.