



CARBON MONOXIDE TOXICITY

by David Kissin, BS, RRT

Here in the upper Northeast of the United States, as summer ends, we begin to prepare ourselves for winter. This, in health care, includes the inevitable burn, smoke inhalation and carbon monoxide intoxication patients, patients who heat their homes with poorly functioning furnaces and/or woodstoves in poorly ventilated homes essentially "sealed" to maximize heat "efficiency". Due to this inevitability, I am writing a primer of sorts, dispelling some myths and reemphasizing facts to better treat this patient population.

Carbon monoxide is a colorless, odorless fat soluble gas produced from the incomplete combustion of carbon-based products, such as wood and fuel oil. Carbon monoxide (CO) intoxication is the leading cause of accidental poisoning in the United States. CO is one of the by-products of the chemical reactions of anything that produces a flame. The resultant chemicals released from burning are carbon, hydrogen and oxygen. These chemicals in turn combine to form CO, carbon dioxide (CO₂), water and heat. With incomplete combustion and/or with a shortage of oxygen (O₂), CO production increases to dangerous levels. Here it is important to note that hemoglobin has a 240-fold greater affinity for CO than oxygen and will readily displace bound oxygen and impair oxygen delivery. Being a fat soluble compound, CO stores in adipose tissue and can be released into the blood after initial reduction of carboxyhemoglobin (COHb) levels. In addition, the values of COHb obtained from arterial blood gas analysis can be "artificially" lower than actual values. It is because of these "phenomena" that long term sequelae to CO toxicity can manifest up to 6 weeks post exposure.

CO is a natural by-product of hemoglobin metabolism in the human body. However, toxic levels can only occur from exogenous sources. These other sources include cigarette smoke, automobile exhaust and fumes from chemicals such as paint strippers like methylene chloride. COHb has also been reported in pediatric post-heart transplant patients on high doses of nitroprusside. Normal COHb levels in humans are 1 - 3%, usually due to heme catabolism and everyday environmental exposure. Smokers increase this level according to the number of cigarettes they smoke and can tolerate COHb levels as high as 10% asymptotically.

The cherry red skin color associated with CO poisoning most expected by the lay public only occurs at very high COHb levels and only represents 2 - 3% of all cases. People with CO toxicity clinically present with flu-like symptoms, headache, dizziness or nausea. Infants may only exhibit vomiting and treated for gastroenteritis. The elderly, especially those with cardiac disease, may have concomitant

ischemic changes leading to a myocardial infarction. Central nervous system symptoms, obtundation, coma or seizure activity is usually dependent upon prolonged exposure and high COHb levels but can also manifest as a latent symptom. Retinal hemorrhage and lactic acidosis can be resultant of prolonged exposure as well. Persistent metabolic acidosis despite volume resuscitation and with normal cardiac output is a strong indicator of CO interference with O₂ delivery and utilization.

As described previously, diagnosis can be difficult. Clinical presentation is far from definitive and mimics many other disorders. Suspicion of CO exposure is a red flag to pursue more concise diagnosis. Pulse oximetry is not useful in this patient population because of the limitations of the device. Using only red and infrared light wavelengths, a pulse oximeter can only differentiate between saturated and reduced hemoglobin. Dyshemoglobins are detected as saturated due to absorption of similar wavelengths. In other words, a pulse oximeter will display all "saturated" hemoglobin as a percentage. If a patient has an O₂ saturation of 70% and a COHb saturation of 20%, the display will read 90%. Direct measurement of COHb in the blood by cooximetry requiring a blood sample is the definitive measurement tool. (NOTE:COHb levels in venous blood have been shown to underestimate arterial content.) Newer technologies have led to the development of pulse co-oximetry, which detects multiple wavelengths of light and can differentiate between saturated hemoglobin and dyshemoglobins and is a very useful tool in the pre-hospital setting.

Treatment for CO toxicity is relatively straightforward. Limiting further exposure to the gas is imperative as is quick reoxygenation of the blood. The reported "therapeutic window" for optimal treatment is 6 - 12 hours. The half-life of CO in the blood of otherwise healthy patients with normal cardiopulmonary function is 300 minutes breathing room air. The half-life can be reduced by 50% every 20 - 30 minutes breathing oxygen enriched gas with O₂ concentrations of 90 - 100%, bringing the half-life to 90 - 120 minutes. If response to high O₂ concentration is unsuccessful at lowering COHb levels or if initial COHb levels are high, greater than 25%, hyperbaric oxygen therapy is recommended at 3 atmospheres. This achieves an arterial PO₂ of greater than 1800 mmHg displacing the CO from the hemoglobin, myoglobin, cytochromes and other hemoproteins and will further reduce the half-life of CO to 15 - 20 minutes. Hyperbaric oxygen therapy has also been shown to reduce neurologic symptomology by improving metab-

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olism and decreasing neutrophil adherence. However, the optimal number of treatments has yet to be determined and obstacles to concomitant care, such as burns, etc., while in the hyperbaric chamber preclude this mode of therapy to the most severe cases. Most practitioners recommend 6 hours of 100% O₂ therapy via face mask as the optimal therapy. When the dysfunction of the patient's pulmonary status is so severe that maximum ventilatory support is ineffective, extracorporeal membrane oxygenation (ECMO) has been used successfully. Other treatment methods are being explored. Laser treatment facilitating the dissociation of CO with the hemoglobin molecule is being studied. A device that delivers high concentration of O₂ with carbon dioxide has been developed that will allow for hyperventilation without hypocapnea, thusly maximizing CO dissociation.

CO poisoning should be suspected in any patient that presents with flu-like symptoms and has any possible exposure to combustibles or CO. If CO toxicity is part of the differential diagnosis, cooximetry by direct blood analysis or pulse cooximetry are easy and useful diagnostic tools. Treatment should include high concentration O₂ therapy for 6 hours or until COHb levels are less than 7%, cardiopulmonary support if needed and, in some more severe cases, hyperbaric oxygen therapy. New diagnostic and treatment options are being studied. Prevention of exposure is key as is expedited diagnosis and treatment. Increased use of CO detectors, better diagnostic tools and treatment options for this commonly misdiagnosed and leading cause of accidental poisoning is needed. Optimal patient care is dependent upon any possible associated problems, including cardiopulmonary compromise, burns, etc. A multifactorial approach is then needed to treat the impaired oxygen delivery as well as the other conditions.

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Failure to fulfill subordinates' expectations

If you feel tortured by a boss's expectations, you're going to be upset by some of the items on subordinates' agendas. We've talked about the desire of employees to be mentored, but that's only the beginning. Most subordinates are hypersensitive to lack of feedback. Even if you don't give them the information they need they expect you to tell them where to get it.

With companies' emphasis on references from subordinates, you need to be sure those references will be enthusiastic. For example, most departing employees won't give exit interviews, but our experience says that when they do, they dis the boss for disappointing them. The most common complaint is, "My boss was neither a leader nor a role model." Worse is "She did not know what should be done and therefore couldn't explain it to me."

Inappropriate show of independence

Nothing will put you on the boss's list of people to throw into the next layoff pool as quickly as being too independent. How does that square with the ideas of empowerment and entrepreneurship both highly touted right now? Here's what makes independence inappropriate. An employee who sets clear limits on his/her obligations to the company will be seen as too independent. This kind of independence spells lack of team spirit to some, which may not be discussed openly but will definitely be resented. Avoiding political hot seats is something like playing musical chairs. You have to be nimble and vigilant.

Marilyn Moats Kennedy is founder and managing partner of Career Strategies, a 34-year old management consulting firm in Illinois. Kennedy holds a MSJ from Northwestern University and is a regular columnist in Focus. MMKCareer@aol.com