NONINVASIVE CO MEASUREMENT BY FIRST RESPONDERS

A suggested management algorithm

By Neil B. Hampson, MD, & Lindell K. Weaver, MD

A new pulse oximeter is available with the capability to measure blood carbon monoxide (CO) levels, in addition to the conventional variables of heart rate and arterial hemoglobin oxygen saturation.[1] EMS personnel and other first responders will likely begin using the device soon. It's important that they understand the meaning of the CO measurement provided and have a plan for patient triage and management based upon the reading obtained.

CO is a toxic gas produced as a byproduct from burning. Almost all burning produces CO to some degree; the amount varies depending on the material and the efficiency of the combustion. Examples of common sources of CO include malfunctioning furnaces, gasoline-powered engines and fires. Although one can typically see and smell exhaust and smoke, the CO that they contain is colorless, odorless and tasteless. Most people are unaware that they are being exposed to CO until they develop symptoms.

ILLUSTRATION BROOK WAINWRIGHT

CO's toxic mechanisms

Carbon monoxide has a variety of toxic mechanisms of action.[2–5] One mechanism that has been known for more than a century is its effect on hemoglobin. When inhaled, CO binds to hemoglobin in red blood cells passing through the lungs, forming carboxyhemoglobin (COHb). Because CO binds to hemoglobin much more tightly than oxygen and occupies the sites normally used to bind and carry oxygen from the lungs to the tissues, one mechanism of CO toxicity is decreased oxygen content of arterial blood and a resultant reduction in peripheral oxygen delivery.

Poisoning from CO is common in the United States, accounting for an estimated 40,000 emergency department (ED) visits and 3,800 deaths annually.[6,7] Symptoms of CO poisoning range from headache, nausea, vomiting and dizziness to loss of consciousness and even death. Because the milder symptoms of CO poisoning are so nonspecific, patients may be misdiagnosed with such conditions as viral illness, food poisoning or motion sickness, depending on the circumstances of the exposure. It is felt that the 40,000 cases of CO poisoning diagnosed each year in U.S. emergency departments (EDs) underestimate the actual incidence, and that many more cases are either not seen in an ED or are not diagnosed when seen.

Organs with a high metabolic requirement for oxygen, such as the heart and brain, are particularly susceptible to injury from CO. The primary treatment for CO poisoning is oxygen, either normobaric or hyperbaric, depending upon the severity of the poisoning. A large prospective randomized clinical trial recently demonstrated that treatment with hyperbaric oxygen (HBO) is more effective than normobaric oxygen in preventing long-term neurological sequelae in CO-poisoned patients, so it is now generally accepted by experts in the field that at least some patients with CO poisoning should be treated with HBO, if reasonably available.[8,9]

Because CO binds so avidly to hemoglobin, COHb remains in the circulation for hours and is a marker that can be measured to document recent CO exposure.

Normal COHb levels are different for smokers and nonsmokers because smokers regularly inhale CO with cigarette smoke. As can be seen in Table 1 (right), the average COHb level in nonsmokers is less than 1%, while the average level in smokers is about 4%.[14] There is obviously a range of values among individuals in each category, with some having higher levels and some lower than the average. To look at it a different way, 98% of nonsmokers have a COHb level ≤ 2.5% and 98% of smokers have a level ≤ 10.0% (Table 1). Of the 2% of smokers whose levels exceed 10%, COHb has been reported as high as 15–20% immediately after smoking.[10–12] If an individual's COHb measurement is higher than 3% in a non-smoker or 12% in a smoker, it is quite likely that they were exposed to another source of CO.

New noninvasive CO measurement

Until recently, determining an individual's COHb level required drawing a blood sample and measuring it in a laboratory with a CO-oximeter or estimating it by measuring exhaled CO.[13] Laboratory CO-oximeters use multiple wavelengths to distinguish the various forms of hemoglobin (oxy-, deoxy-, carboxy- and met-). Conventional two-wavelength pulse oximeters are incapable of measuring COHb.[15] The new Rad-57 pulse CO-oximeter, developed by Masimo Corp., utilizes eight wavelengths of light and is able to provide a noninvasive measurement of COHb (SpCO) in seconds, in addition to SpO_2 and heart rate. The device's accuracy has been demonstrated up to 40% SpCO, with a range of ± 3% around the measurement.[1]

It has been repeatedly demonstrated that the COHb level correlates poorly with the clinical condition of the CO-poisoned patient. As such, most experts have traditionally recommended using the COHb level to confirm the diagnosis in a patient with symptoms suspected to be due to CO exposure, using the actual level to guide management only when elevated to the range of 25% or greater.[9]

TABLE 1: COHB LEVELS IN PERSONS 3–74 YEARS OF AGE[14]

Smoking Status	Percent COHb (mean ± SD)	Percent COHb (98th percentile)
Nonsmokers	0.83 ± 0.67	≤ 2.50
Current smokers	4.30 ± 2.55	≤ 10.00
All smoking statuses combined	1.94 ± 2.24	≤ 9.00

Because a clinician has traditionally ordered blood measurement of COHb only when the condition was suspected, it is likely that there has been a tendency to measure COHb only in the more symptomatic patient or in those whose exposure history was known. Because EMS providers and paramedics commonly use a pulse oximeter to measure SpO_2 at the scene, one can predict that many instances of elevated SpCO will be discovered among patients without a classic history or recognized exposure to CO.

Managing an elevated SpCO level

When first-responders encounter elevated SpCO levels, they will need guidance and/or a protocol for triage and management. We have suggested such an algorithm in Figure 1 (below).

Because smoking history may be unreliable or unobtainable, we do not recommend attempting to determine whether an individual is a smoker or nonsmoker in the field and have not included smoking status in decision making.

For SpCO levels up to 3%, no further evaluation is necessary because they are likely normal (Table 1). If the patient has other indications for treatment or transport, those should obviously be taken into consideration.

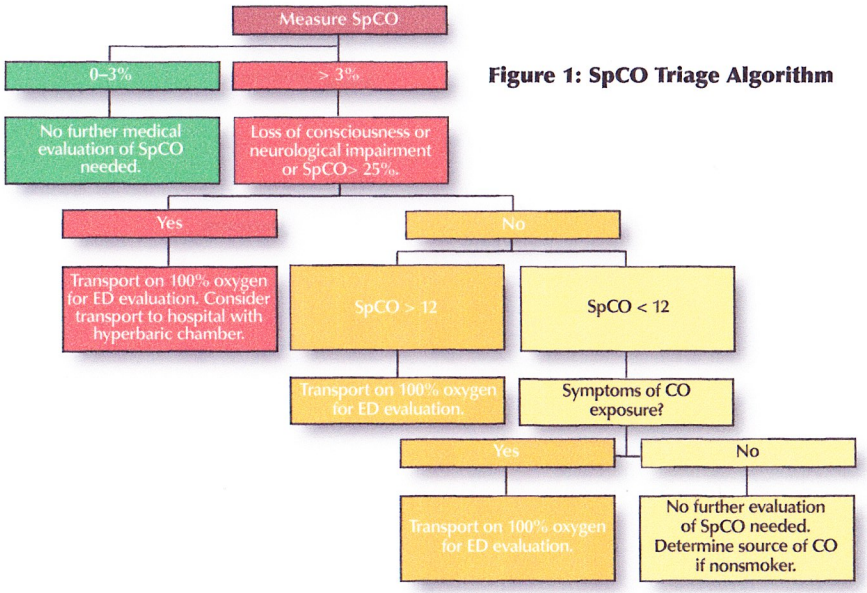
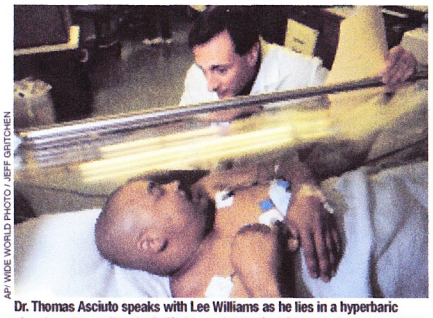


Figure 1: SpCO Triage Algorithm



AP/ WIDE WORLD PHOTO / JEFF GRITCHEN

Dr. Thomas Asciuto speaks with Lee Williams as he lies in a hyperbaric chamber at Long Beach (Calif.) Memorial Medical Center in December 2005. Williams, his wife and two children were overcome with carbon monoxide from a defective heater in their home.

If SpCO is greater than 3%, we recommend evaluation for signs or symptoms of severe CO poisoning that might prompt consideration of treatment with hyperbaric oxygen. The Undersea and Hyperbaric Medical Society recommends hyperbaric oxygen therapy for CO-poisoned individuals with transient or prolonged unconsciousness, neurological signs, cardiovascular dysfunction or severe metabolic acidosis, irrespective of the degree of elevation of their COHb levels.[16] It is noted, however, that a majority of hyperbaric physicians do use HBO to treat patients with less severe symptoms when COHb levels are elevated to the range of 25–30%.[17]

If the SpCO level is 12–25% and severe symptoms are not present, the patient should receive 100% oxygen and be transported to a hospital for further ED evaluation and treatment.

If the SpCO level is 3–12%, the elevation could be due either to smoking or another source. If a patient with SpCO in this range has such symptoms as headache, nausea or vomiting (less severe than those listed above as warranting HBO), they should receive 100% oxygen and be transported for further ED evaluation. If the SpCO level is 3–12% and the individual is asymptomatic, further medical evaluation of the SpCO level is not necessary in most cases. However, if a source other than cigarette smoking is likely, it is imperative to remove the individual from the environment and determine the CO source.

Although it is expected that many unsuspected cases of CO exposure will be identified through use of this device, we recommended that EMS personnel consider reconfirming the SpCO reading if it appears abnormally low or high for the clinical situation.

The new pulse CO-oximeter represents a major advance in field screening for CO exposure and poisoning. With it, the number of individuals diagnosed with CO poisoning each year is likely to increase dramatically. Because many of these will initially be discovered to have an elevated SpCO level by first-responders, it is very important that triage and management protocols be available as the device is put into use.

We have proposed an algorithm for use in the field; however, providers are advised to follow local EMS guidelines and consult their local EMS medical director before using this device or making triage and treatment decisions based on readings from it. ◆

Neil B. Hampson, MD, is head of the Section of Pulmonary and Critical Care Medicine and medical director of the Center for Hyperbaric Medicine at Virginia Mason Medical Center in Seattle. Dr. Hampson serves as the Northwest Regional Coordinator for Divers Alert Network and is immediate past-president and a current board of directors member of the Undersea and Hyperbaric Medical Society. He has written extensively on the topic of carbon monoxide poisoning.

Lindell K. Weaver, MD, FACP, FCCP, FCCM, is medical director of the Department of Hyperbaric Medicine and medical co-director of the Shock, Trauma and Respiratory ICU at LDS Hospital in Salt Lake City. He serves as a professor in the Department of Internal Medicine at the University of Utah School of Medicine and is president of the Undersea and Hyperbaric Medical Society.

References

1. Masimo Corp.: "Rad-57 Pulse CO-oximeter." www.masimo.com/rad-57/index.htm. Accessed Sept. 26, 2005.
2. Piantadosi CA: "Carbon monoxide intoxication." In: Vincent JL, ed.: *Update in Intensive Care and Emergency Medicine.* 10:460–471, 1990.
3. Zhang J, Piantadosi CA: "Mitochondrial oxidative stress after carbon monoxide hypoxia in the rat brain." *The Journal of Clinical Investigation.* 90:1193–1199, 1991.
4. Thom SR: "Leukocytes in carbon monoxide mediated brain oxidative injury." *Toxicology and Applied Pharmacology.* 123:234–247, 1993.
5. Thom SR, Bhopale VM, Fisher D, et al: "Delayed neuropathology after carbon monoxide poisoning is immune-mediated." *PNAS USA.* 101(37):13,660–665, 2004.
6. Hampson NB: "Emergency department visits for carbon monoxide poisoning." *Journal of Emergency Medicine.* 16(5):695–698, 1998.
7. Mott JA, Wolfe MI, Alverson CJ, et al: "National vehicle emissions policies and practices and declining US carbon monoxide-related mortality." *JAMA.* 288:988–995, 2002.
8. Weaver LK, Hopkins RO, Chan KJ, et al: "Hyperbaric oxygen for acute carbon monoxide poisoning." *New England Journal of Medicine.* 347:1057–1067, 2002.
9. Hampson NB, Mathieu D, Piantadosi CA, et al: "Carbon monoxide poisoning: Interpretation of randomized clinical trials and unresolved treatment issues." *Undersea Hyperbaric Medicine.* 28:157–164, 2001.
10. Ashton H, Telford R: "Smoking and carboxyhaemoglobin" (letter). *Lancet.* 2(7833):857–858, 1973.
11. Danaher BG, Lichtenstein E, Sullivan JM: "Comparative effects of rapid and normal smoking on heart rate and carboxyhemoglobin." *Journal of Consulting and Clinical Psychology.* 44(4):556–563, 1976.
12. Smith CJ, Guy TD, Stiles MF, et al: "A repeatable method for determination of carboxyhemoglobin levels in smokers." *Human & Experimental Toxicology.* 17(1):29–34, 1998.
13. Cunnington AJ, Hormbrey P: "Breath analysis to detect recent exposure to carbon monoxide." *Postgraduate Medical Journal.* 78(918):233–237, 2002.
14. Radford EP, Drizd TA: "Blood carbon monoxide levels in persons 3–74 Years of Age: United States, 1976–80." *US Dept of Health and Human Services PHS.* 82–1250; March 17, 1982.
15. Hampson NB: "Pulse oximetry in severe carbon monoxide poisoning." *Chest.* 114(4):1036–1041, 1998.
16. Thom SR, Weaver LK: "Carbon monoxide poisoning." In: *Hyperbaric Oxygen 2003, Indications and Results, The Hyperbaric Oxygen Therapy Committee Report.* Undersea and Hyperbaric Medical Society. 11–18, 2003.
17. Hampson NB, Dunford RG, Kramer CC, et al: "Selection criteria utilized for hyperbaric oxygen treatment of carbon monoxide poisoning." *Journal of Emergency Medicine.* 13:227–231, 1995.

FIREFIGHTER RESPONSE TO CO INCIDENTS

Vigilance keeps responders from becoming victims

By Greg Jakubowski

AP / WIDE WORLD PHOTO / MISHA JAPARIDZE

Firefighters attempt to extinguish a fire at a publishing house in central Moscow in February 2006. The blaze sent two people to the hospital with suspected carbon monoxide poisoning.

Sometimes, the call for a carbon monoxide (CO) incident is obvious—a report of a CO alarm activation with residents feeling ill or an attempted suicide with an auto running in a garage. Other times, the call is less obvious—a person feeling sick or even someone arriving home to find a family member unconscious. At the obvious incidents, not many clues are necessary to confirm that CO poisoning is the cause of the situation.

At the not-so-obvious incidents, responders may not realize the problem's root cause and simply treat the patient's symptoms. They may have no idea that while they are treating the patient, they are being exposed to the same conditions that caused the patient's illness. In these situations, responders need to maintain a high level of awareness and work diligently to determine the cause of the illness or unconsciousness. They must keep in mind that CO poisoning may be the potential cause for the victim's condition. Based on previous incidents that were eventually traced to CO poisoning, some ambulance units now carry a portable CO monitor in their first-in bag, permitting the crew to continuously monitor their environment for this potential hazard.

How to respond

As with any response, firefighters must begin to size-up the incident upon dispatch to the call. Activated CO detectors are not necessarily emergencies—unless victims are still in the building and/or victims are experiencing symptoms of exposure. Carbon monoxide doesn't normally trap victims inside a building, unless they've fallen unconscious. In that case, emergency response is appropriate, and proper personal protective equipment (PPE) is needed to access and remove the victim from the hazard. Exposed victims who are outside of the hazard zone would generally warrant an emergency response from EMS, although not necessarily from the fire department, unless fire units are providing first response medical care.

Firefighters also may need to respond at emergency speed if CO is building up rapidly in the building; at high levels, CO can be explosive. The lower explosive limit of CO is relatively high at 12.5%, but the explosive range is wide, up to an upper explosive limit of 75%. Once CO is identified as a hazard in the building, it is important to apply positive-pressure ventilation. Opening windows will normally do the trick by allowing the building's interior to air out while minimizing collateral damage.

What to look for

CO is generated from equipment that burns fuel, such as natural gas, propane, gasoline, oil, wood, kerosene or charcoal. Sources include heaters, water heaters, ranges, grills and clothes dryers. Other potential sources of exposure include propane-fueled hot tubs, kilns, fireplaces and various other household appliances. CO can quickly build up from the use of gasoline-powered equipment, such as blowers, cut-off saws, lawn equipment and similar apparatus utilized in poorly ventilated areas. Propane-fueled construction heaters and lift trucks can also cause problems.

CO buildup is much less likely in well-ventilated areas. However, a sealed-up home, below-grade area, garage or similar location presents conditions where CO levels could be high. This may be particularly