LETHAL EXPOSURE

CARBON MONOXIDE PRESENTS A TOXIC HAZARD FOR FIRST RESPONDERS

An exclusive supplement to JEMS (Journal of Emergency Medical Services), FireRescue Magazine & Wildland Firefighter sponsored by Masimo Corp.
By A. J. Heightman

Vigilance keeps responders from becoming victims
By Greg Jakubowski

On the cover: A Smithtown, N.Y., firefighter responds to a house fire. Wearing SCBA protects fire crews from exposure to carbon monoxide and other toxic byproducts of combustion. PHOTO CRAIG JACKSON

The 21st of June was a hot day—the first day of summer and my birthday. My fire department was dispatched to the westbound lanes of an interstate highway where a tractor-trailer carrying pipes that were loaded too high on the trailer impacted a highway overpass, causing the top row to be sheared off. Immediately after landing on the roadway, the pipes were hit head-on by a painter’s van traveling in the passenger lane. I was the first EMS command officer to arrive on scene. I gave a brief scene assessment to responding units, reporting that we had two victims, one heavily trapped, a painter’s van involved, paint leaking on the highway, and no smoke or fire visible from the highway level. A local police officer photographed the scene from the top of the overpass and later reported seeing a green mist in the air over the van. However, this was not reported to me or other responding units.

I was confronted with a male in his 30s who had been ejected from the passenger side on impact and was lying on the road at the rear of the van. He was conscious and complained of neck, back, and leg pain (see Photo 1).

His passenger, a 22-year-old male, was pinned in the right front seat. The van’s front end was pushed into the passenger compartment, and scalding hot water from the vehicle’s radiator was leaking on the passenger’s genital region.

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I was confronted with a male in his 30s who had been ejected from the passenger side on impact and was lying on the road at the rear of the van. He was conscious and complained of neck, back, and leg pain (see Photo 1).

His passenger, a 22-year-old male, was pinned in the right front seat. The van’s front end was pushed into the passenger compartment, and scalding hot water from the vehicle’s radiator was leaking on the patient and required that a medical helicopter be dispatched.

The engine crew charged a small-diameter line and began flushing the patient’s lower body regions (see Photo 2). With the patient’s C-spine immobilized and high-flow oxygen started, extrication began. During the course of the rescue, the patient’s dyspnea ended and he appeared more coherent than when I first arrived. However, the personnel extricating the patient, myself included, began to show signs of extreme fatigue.

I assumed that the high heat and humidity of the afternoon, the complicated rescue, patient packaging, and our heavy turnout gear were causing our lethargy. But I was wrong. The strange green cloud that the police officer on the overpass observed was actually methyl-chloride vapors being emitted from the paint solvents leaking along with the paint, causing us to unknowingly be exposed to carbon monoxide (see Photo 3).

An alert fire officer realized that the spilled contents were causing our symptoms, particularly because the trapped patient (who was being flushed with high-flow oxygen) was no longer dyspneic, while his rescuers were exhibiting unusual signs of lethargy and dyspnea. He ordered exhaust fans placed around the van to ventilate the area.

After the extrication was completed and the patient was turned over to the MedEvac crew, rescuers began to collapse, and eight of us ended up as priority 1 patients. During triage and treatment, we presented with symptoms that included tachycardia, hypotension, lethargy and dyspnea. My pulse was 150, my respiratory rate was 28 and my BP was 80/60. With high-concentration O2 flowing and bilateral IVs running wide open, I and the other rescuers were transported to area hospitals and treated for carbon monoxide exposure.

We were all released after extensive testing and hours of fluid and oxygen therapy. But the trauma of our exposure didn’t end that day. As a result of our prolonged exposure, several of us sustained lung damage that now results in chronic bronchitis and emphysema.

We learned many lessons at this incident, including the need for a designated safety officer at all scenes, the need for better police-to-EMS communications, to use SCBA any time foreign substances are present, the need for CO monitoring devices—and the invisible and damaging effects of carbon monoxide exposure.

Learn more at www.fire rehab.com
The U.S. Fire Association (USFA) has specific guidelines that recommend the following:

- Decreased oxygen utilization.
- Lethal exposure

Learn more at www.firerehab.com

Transfer of oxygen to the tissues

Lethal exposure

List common sources

Decomposition of carbon monoxide is the main cause of carbon monoxide poisoning. Carbon monoxide poisoning is a serious health problem. In short, CO poisoning is a serious national health problem.

CO sources

Carbon monoxide is a colorless, odorless gas that is toxic to humans. It is produced by many different sources, including:

- Combustion of carbon-containing fuels, such as coal, wood, and natural gas.
- Automobile exhaust.
- Faulty furnaces and other heating equipment.
- Gas appliances.
- Stoves and other cooking appliances.
- Cigarette smoke.

Pathophysiology

The pathophysiology of CO poisoning is complex. However, despite the many mechanisms by which CO produces poisoning, they each contribute to the overall effect of CO poisoning.

On-scene considerations

Many calls involving carbon monoxide—faulty furnaces, exposure to automobile exhaust, etc.—do not pose a significant risk to EMS or fire personnel, or the risk is easily managed. Dealing with fires is a different matter. The risk of CO exposure during a fire is prolonged and potentially deadly, and it does not end once the fire is under control.

When responding to a fire, fire and EMS crews can quickly find their role of responder change to the role of victim. To prevent that, crews must implement on-scene rehabilitation (i.e., rehab) and remain vigilant regarding potential CO poisoning during overhaul.

Rehab is achieved by periodic, supervised rest periods for firefighters; it is care given to firefighters and other emergency personnel on scene. Fighting fires places personnel at risk for CO poisoning, but there is also the danger of heatstroke, dehydration, and cardiac problems. Incident commanders assess the risk and ensure that responders have access to rest, fluids, food, medical attention, and CO monitoring.

Note: The U.S. Fire Association (USFA) has specific guidelines that specify how rehabilitation services should be set up and provided.

Overhaul is more complicated. It refers to seeking out and extinguishing any remaining fires, eliminating rekindles, stabilizing the incident scene and securing the structure. This phase of fire control can be very time consuming, and personnel may be involved for hours. Overhaul may also appear to be relatively risk-free, and that is one of its dangers. CO levels in smoldering fires and during overhaul operations can be very high, certainly high enough to cause impairment.

A study performed in Phoenix showed that in 20% of the fires examined, the CO level during overhaul exceeded the National Institute of Occupational Safety and Health's short-term exposure limit of 200 parts per million (ppm). However, during overhaul, there may be a tendency to overlook this fact. Firecrews may perceive that because the fire is out, there is no longer a danger, and that it's safe to remove SCBA. This can prove dangerous, exposing personnel not only to CO, but many other byproducts of combustion.

Incident commanders must assess the CO risk to firefighters and ensure that responders have access to rest, fluids, food, medical attention and CO monitoring during operations.

- Depressed cardiac function:
- Action as a vasodilator: CO increases the activity of cyclic guanosine monophosphate, a potent vasodilator)
- Effects on platelets: Vasodilataion decreases the oxygen delivery to tissues by causing pooling of blood in the vascular bed;
- Decreased oxygen utilization: CO also binds to myoglobin, a protein-iron complex that transports oxygen within the cells; and
- Free-radical formation: The release of nitric oxide from platelets initiates the formation of free radicals. Also, the tissue damage caused by peroxidation and loss of oxygen attracts leukocytes to the damaged area. This initiates and sustains an inflammatory response and also causes free radical formation. (This process is essentially a tissue reperfusion injury, similar to what is seen in patients who have suffered a myocardial infarction.)

In summary, CO: 1) prevents oxygen from being delivered (by the formation of COHb and increased binding of oxygen to hemoglobin); 2) causes tissue hypoxia (by the above mechanisms along with decreased tissue oxygen pressure);

Glossary

Overhaul: Extinguishing any remaining fires, eliminating rekindles, stabilizing the incident scene and securing the structure.

Rehabilitation: Periodic supervised rest periods during extended operations, presenting the opportunity to determine if an individual has sustained CO poisoning.

The risk of CO exposure during a fire is prolonged and potentially deadly, and it does not end once the fire is under control.
depression of cardiac function and vasodilation); and 3) stimulates the production of free radicals that damage tissue.

Clinical signs & symptoms

Clinical signs and symptoms of CO poisoning are changeable, non-specific and often mild. The patient may have a slight headache and complain of a lack of energy. Even if symptoms are severe, CO poisoning can be easily misdiagnosed. CO poisoning may be mistaken for food poisoning, influenza, a cerebral bleed or a migraine headache. If there is no obvious situation involving CO (e.g., a fire), it can be difficult to spot these cases. But remember two points: 1) Examine the patient and take a history, taking into account the situation surrounding the patient and their illness and; 2) CO poisoning causes tissue hypoxia, and the signs and symptoms of CO poisoning will reflect this. If you suspect CO poisoning, but there is no obvious source, ask the patient the following questions:

- **Do you feel sick now?** If not, where, specifically, are you when you feel sick? If the patient reports that they feel sick at home, and their symptoms improve when they are out, that is a sign that CO may be involved.
- **Whom do you live with?** Has anyone else been sick? If the patient is living with other people, but no one else is sick, CO exposure is unlikely.
- **What type of heating system do you have, and was it operating when you were symptomatic?** Patients often suspect they have CO poisoning, but the symptoms occur when the heating system is not on, e.g., during the summer.
- **Have you recently checked/maintained your heating system, water heater, etc., and the devices’ exhaust systems?**
- **Do the symptoms happen at work?** Do you work indoors in a situation in which CO is produced (e.g., inside a garage or warehouse with forklifts operating, etc.)?
- **Have you recently stripped paint or varnish off any furniture?**
- **How long have you suffered the signs and symptoms? What are the signs and symptoms?** A viral illness typically lasts five to seven days. Patients who have CO poisoning from a source that is not obvious may have signs and symptoms for weeks or months. Also, some symptoms of certain viral infections, such as sore throat or fever, are very unlikely to be due to CO poisoning.

- **If there are multiple patients, did everyone become sick at the same time?** A viral/infectious illness usually starts with one person and then spreads to the others. In CO poisoning involving many people, everyone will become sick at approximately the same time. (This is also true of food poisoning that affects large groups of people, but the situations in which CO and food poisoning occur usually differ.)

After the interview, move on to the physical exam. Again, CO poisoning does not produce signs and symptoms that are distinct. However, responders may recognize CO by remembering that CO poisoning causes decreased oxygen delivery to, and decreased oxygen utilization by, organs that are very active metabolically. CO poisoning affects the following systems:

- **Neurologic:** CO poisoning causes central nervous system depression, and the effects of CO poisoning can be arranged on a continuum of impairment. In mild cases, the patient may complain of a headache, dizziness and confusion, or may have difficulty with abstract thinking or have ataxia. In severe cases, the patient may become comatose or develop seizures.
- **Cardiac:** The cardiac signs of CO poisoning reflect the decreased myocardial function and vasodilation caused by the CO. Patients also develop a toxic myocarditis. The signs and symptoms of CO poisoning are similar to specific signs and symptoms of myocardial ischemia.
- **Metabolic:** Respiratory alkalosis is possible in mild cases, and metabolic acidosis is common in severe exposures.
- **Pulmonary:** Pulmonary edema occurs in 10–30% of acute CO exposures. This may be due to a direct effect on the alveolar membrane, left ventricular failure, aspiration or neurogenic pulmonary edema.
- **Renal:** Rhabdomyolysis and renal failure are possible. Remembering all the effects caused by CO can be difficult. It’s much easier to simply remember interfering with the delivery to, and utilization of, oxygen by organs with a high need for oxygen. The signs and symptoms of CO poisoning reflect this fact.

**Notes:** CO poisoning by methylene chloride vapors can be prolonged. The enzymes that metabolize methylene chloride become saturated when the level is too high. Methylene chloride is then stored in fat tissue and slowly released.

**Laboratory confirmation**

The laboratory can provide unequivocal proof that a patient has been exposed to CO. A carboxyhemoglobin level is drawn (both venous and arterial blood can be used; if using venous blood, a lithium heparin tube must be used), and if it is above 1–2%, there is a possibility that the patient was exposed to CO. However, levels must be interpreted with several facts in mind.

CO naturally occurs in the body, and a level of 1–2% is normal. Cigarette smoke contains CO, and smokers can have a “normal” level of 4–5%. Some smokers might have a chronic level of 10%.

Also, the length of time between the exposure and the level is important. The lungs naturally excrete CO; the half-life of CO is four to six hours when the patient is breathing room air, and 40–60 minutes when the patient is breathing 100% oxygen. If transport time is 30 minutes and the patient has been breathing 100% oxygen during that time, it will be difficult to know when the level peaked.

More importantly, there is a poor correlation between a COHb level and the clinical presentation of the patient. This is particularly true of the neurologic effects of CO exposure. This is also true of patients with coronary artery disease; they may develop angina and/or arrhythmias at CO levels that are tolerated by patients with healthy hearts. This is thought to be because the COHb level in the blood does not accurately predict the CO level in the tissues. An elevated COHb level is certainly a cause for concern, but the clinical presentation of the patient is a better indicator of how sick he or she is. Also, the COHb level is a poor predictor of the outcome of any particular exposure.

It is important to note that pulse oximetry does not accurately reflect CO poisoning. The pulse oximeter will provide false readings. The pulse oximeter readings will be falsely high, and the difference between the pulse oximeter reading and the actual saturation of hemoglobin with oxygen increases as the COHb increases.

What levels of CO are dangerous? Some authors have tried to correlate the COHb level to specific signs and symptoms, but there is ample evidence that suggests this can’t be done; patients with high levels will not necessarily be sicker than patients with lower levels. The current Occupational Safety and Health Administration (OSHA) limit is for the average amount of CO that is safe for an occupant for an 8-hour day is 50 ppm, although the CO limits vary slightly depending on the source. Symptoms will usually become apparent when the CO level reaches 100 ppm.

**Recurrent symptom syndrome**

Unfortunately, the effects of CO poisoning can resolve and then return. The recurrent symptom syndrome occurs after a “moderate” case of CO poisoning. There is an asymptomatic period of one to 40 days, then the neurologic effects, such as headache, nausea, mood swings, confusion and memory problems occur. This syndrome affects approximately 12–21% of all patients with a moderate case of CO poisoning. The majority of these patients recover, although this process can take 40 days.

**Delayed neurotoxic sequelae**

Delayed neurotoxic sequelae are similar to recurrent symptom syndrome, but signs and symptoms are more severe, and their onset can be (approximately) two to 240 days after exposure. Again, there is a lucid, asymptomatic period before the effects of CO poisoning recur, and then the patient may have significant neurological and psychiatric deficits. The recovery period may be up to a year, and there are a significant number (figures of 14–43% have been cited) of patients who suffer permanent damage. It appears that patients who were exposed to CO and lost consciousness are at the greatest risk for this disorder. The exact incidence of this problem is not clear; the range reported in the literature is 3–40%.

**Patients at high risk for negative outcome**

**Children;**

**Adults with cardiac disease;**

**Pregnant women or women who may be pregnant;**

**Patients with increased oxygen demand or decreased oxygen-carrying capacity; and**

**Patients with chronic respiratory insufficiency.**

**The pregnant patient**

The pregnant patient presents a special challenge. One exposure to CO affects two patients, and it is not easy to assess the fetus’s condition. The fetus is exposed to the CO through the placenta, and fetal hemoglobin has an even higher affinity for CO than maternal hemoglobin; at any given percent of CO, the fetus will have a higher COHb level than the mother. CO poisoning also interferes with the release of oxygen to the fetal tissue, and absorption and elimination of CO are much slower in the fetus than in the mother. CO levels in the mother that would not be considered particularly high may be dangerous to the fetus. A high COHb level and significant signs/symptoms in a pregnant patient are very serious; in these cases there is a significant risk for fetal central nervous system damage and stillbirth. However, even in minor exposures (e.g., no loss of consciousness) there can be poor fetal outcomes. Oxygen therapy is safe for the fetus.

**Treating the CO-exposed patient**

The signs and symptoms of CO poisoning are subtle and changeable, and at times it takes a skilled observer to notice them. On-scene personnel must also be aware of situations in which CO poisoning is likely; some are obvious, some are not. An accurate assessment and a complete history are needed to determine the amount of risk and to identify high-risk patients. Also, accurate charting of the timing of assessments and therapies proves critical. A CO level drops when the exposure is stopped, and the level declines more rapidly when oxygen is applied, so precise charting of the patient’s signs and symptoms and their response to treatment will suggest the severity of the patient’s poisoning. Caring for a patient with CO poisoning is relatively simple. Assess the airway, breathing and circulation (ABCs), and consider endotracheal intubation if the patient is comatose. Apply oxygen via a non-rebreather mask, and carefully document what was applied and how long after the exposure ended it was applied. Place the patient on a cardiac monitor and observe for arrhythmias. If any of these occur, treat the patient per your local protocol. Check the vital signs, and if the patient is hypotensive, treat them per your local protocol. If the patient is alert and oriented, begin the history and assessment:

Determine if the patient is in the high-risk category. These include:
CE program is coordinated by the Center for Emergency Medicine and the University of Pittsburgh.

Hyperbaric oxygen: To dive or not to dive?
There is no doubt about the mechanism by which hyperoxic oxygen (HBO) works. The patient or patients are placed in a closed chamber and breathe an atmosphere of 100% oxygen at pressures that are two to three times the normal atmospheric pressure (which is 14.7 lbs per square inch). Breathing 100% oxygen at these elevated pressures (e.g., 2.5 atmospheres absolute) decreases the half-life of COHb to 20 minutes. HBO also increases the amount of oxygen available to the tissues. Hemoglobin quickly becomes saturated, and the increase in percentage of inspired oxygen and the elevation in atmospheric pressure cannot change that.

HBO works by increasing the rate of COHb dissociation in the blood. HBO leads to an increase in the partial pressure of oxygen in the blood and an increase in the oxygen content of the blood.

The effects of HBO on COHb are well documented. HBO decreases the concentration of COHb in the blood by several different mechanisms, and it's not clear which patients are HBO candidates.

Consider the following:

HBO is not new; it was first used in 1960. Although HBO has been used thousands of times, and the side effects are usually mild and reversible, there are important unanswered questions about HBO.

More research is needed, but at this time, it appears that because HBO might help, and it has virtually no side effects, most emergency physicians will seek the consultation of the local HBO facility physician, and "screening" and "sizing" procedures are underway. Pregnant patients, patients with a COHb level > 30% of patients with a serious history of anemia or obstruction will probably receive HBO therapy.

Conclusion
Although we don't know with certainty how CO poisoning works, it's clearly preventable. It's also clear that fast, effective treatment by first responders can do much to prevent damage. Emergency care of a patient with CO poisoning is straightforward, and it has proven effective.

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4. Within 4 weeks of mailing your test, you will receive your test score and, if you passed, a CE certificate. The passing score is 70%.

Mark your answers in the appropriate box (1-20).

1. Which of the following is true of CO poisoning?
   a. Approximately 10-15 people die annually, but many more are injured.
   b. CO is the leading cause of death by poison in the United States.
   c. CO poisoning is almost always found in industrial settings.
   d. Annually, 10,000 CO exposures are reported in the United States.

2. The leading cause of CO poisoning is:
   a. space heaters
   b. faulty brakes
   c. automobile without d. the smoke inhalation

3. Define what the danger from CO poisoning is:
   a. higher than during the active phase
   b. a potential danger that must be considered
   c. a problem only if the firefights remain in a closed space
   d. removed, because the CO danger from CO poisoningremains

4. CO causes tissue hypoxia by which of the following mechanisms?
   a. CO causes tissue oxygen to be less than 70 mmHg
   b. CO causes tissue oxygen to be less than 90 mmHg
   c. CO causes tissue oxygen to be less than 100 mmHg
   d. CO causes tissue oxygen to be less than 120 mmHg

5. Diagnosing CO poisoning is:
   a. easily completed by assessing signs and symptoms
   b. difficult, as signs and symptoms mimic many other conditions
   c. possible only with long-term observation and blood monitoring
   d. possible only when the patient has a history of exposure to the

6. Which of the following is expected in mild cases of CO poisoning?
   a. nosebleeds
   b. epistaxis
   c. headache
   d. nausea

7. A blood CO level of 4-6% in a person who smokes cigarettes reflects:
   a. acute CO poisoning in a smoker
   b. normal CO level in a current smoker
   c. highly oxidized level for a non-smoker
   d. level oxidized level for a smoker

8. The half life of CO when breathing room air is:
   a. 4-5 hours
   b. 1-2 hours
   c. 2-4 hours
   d. 4-6 hours

9. The half life of CO when breathing 100% oxygen is:
   a. 4-5 hours
   b. 1-2 hours
   c. 2-4 hours
   d. 4-6 hours

10. The best indicator of seriousness of CO poisoning is:
    a. pulse abnormality
    b. respiratory acidosis
    c. metabolic acidosis
    d. pulse oximetry

11. Which of the following is typical of recurrent symptom syndrome?
    a. 2 or fewer seizures in a 24 hour period
    b. an asymptomatic period of 5-15 hours
    c. Most patients never return to an asymptomatic state.
    d. The recovery period can be up to 40 days

12. Exposure to CO during pregnancy places the:
    a. mother at decreased risk because most of the CO is transferred to the
    b. fetal brain swelling
    c. possible only with long-term observation and blood monitoring
    d. an increased oxygen percentage

13. In hyperbaric oxygen chambers, the patient breathes oxygen at:
    a. less than two times the normal atmospheric pressure
    b. the normal atmospheric pressure
    c. a lesser than normal respiratory rate
    d. an increased oxygen partial pressure

14. Breathing oxygen in a hyperbaric oxygen chamber decreases the half life of CO to:
    a. 2 weeks
    b. 45 minutes
    c. 45 minutes
    d. 30 minutes

15. Which patient is most likely to benefit from HBO therapy?
    a. patient exposed to methane inhalation
    b. patient exposed to methylene chloride
    c. patient with COHb level of 10%
    d. d.f. patient with COHb level of 20%
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. 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 is odorless and tasteless. Most people are unaware that they are being exposed to CO until they develop symptoms.

CO’s toxic mechanisms
Carbon monoxide has a variety of toxic mechanisms of action. 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, the average COHb level in nonsmokers is less than 3%, while the average level in smokers is about 4%.10 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.11–12 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 SpO2 and heart rate. The device’s accuracy has been demonstrated up to 40% SpCO, with a range of 0–3% 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.8

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 SpO2 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.

A suggested management algorithm
By Neil B. Hanspou, MD, & Lindell K. Weaver, MD

Figure 1: SpCO Triage Algorithm

<table>
<thead>
<tr>
<th>SpCO</th>
<th>Transport on 100% oxygen for ED evaluation.</th>
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<tbody>
<tr>
<td>≤ 3%</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>&gt; 3%</td>
<td>No</td>
<td>Yes</td>
</tr>
</tbody>
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TABLE 1: COHb LEVELS IN PERSONS 3–74 YEARS OF AGE

<table>
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<th>Percent COHb (90th percentile)</th>
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<tr>
<td>Nonsmokers</td>
<td>0.83 ± 0.67</td>
<td>≤ 2.5%</td>
</tr>
<tr>
<td>Current smokers</td>
<td>4.30 ± 3.55</td>
<td>≤ 10.0%</td>
</tr>
<tr>
<td>All-smoking status N</td>
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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.14 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%.27

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% 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 recommend 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 management protocols be available as the device is put into use. 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% 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 recommend 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.26

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References

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ometimes, 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. As 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. As with any response, firefighters must begin to size-up the incident. By Greg Jakubowski

Learn more at www.firerehab.com
true if a sudden cold spell occurs, and windows that are normally left open are suddenly closed. Another potential problem occurs when heating and air-conditioning systems wind up with outside air intake shut, so all of the air in the area is simply recirculated.

Buildings located near very busy roadways may experience higher than normal CO levels, particularly during periods of heavy traffic. Weather conditions can also play a role, such as when an atmospheric inversion prevents pollutants from escaping the immediate atmosphere, forcing them to build up near their generation point.

Size-up must continue once the fire department arrives. Life safety takes precedence. Protect firefighters first and foremost, and utilize protective equipment based on the conditions found. If you discover elevated CO levels in the home, utilize full turnout gear and breathing apparatus, even if police and EMS units appear to be operating in the building without it. Evacuate all occupants and responders who aren’t wearing turnout gear and breathing apparatus.

Once you remove everyone from the building, attempt to track down the source of the CO buildup. Some departments leave this responsibility to another organization, but the only way to confirm there’s CO in a building is with a CO meter. The fire department may carry a CO meter, or they may rely on a different agency (e.g., police, health department, hazmat team, utility company, etc.) to respond with a meter to assist with the investigation. Important: Calibrate any meters (for LEI, CO or other gases) utilized in an emergency response as per the meter manufacturer’s recommendations. Uncalibrated meters may provide inaccurate information. Departments can’t purchase a meter, place it on an apparatus and forget about it until a response requires them to use it.

If the department chooses to search for the CO source, do not ventilate the building. CO is just slightly lighter than air, with a vapor density of 0.968 (air=1.0). CO will likely mix with air in the building, so it is important to check air at the breathing level as well as at the ceiling. Monitor the area for any potential CO sources, with the sources running, if possible. If it becomes difficult to pinpoint the source, or CO levels become transient, try running the hot water to activate the water heater, and activate the home’s heater, assuming both are fossil-fueled. If you still have difficulty pinpointing the source, seek additional outside expert assistance.

Signs & indications

You can’t see or smell carbon monoxide, but at high levels it can kill in minutes. The Immediately Dangerous to Life and Health (IDLH) level of carbon monoxide is 1,200 parts per million (ppm). Symptoms at moderate levels include severe headaches, dizziness, mental confusion, nausea and syncope (see table below). Symptoms normally lessen once victims are moved to fresh air and/or placed on oxygen therapy. Continuous exposure to moderate to high levels can be fatal. The very young, the very old and those who are already ill are likely to be more susceptible to CO exposure.

Symptoms of low-level CO exposure may include shortness of breath, mild nausea and mild headaches. Long-term exposure to low CO levels may have longer term effects on health. CO incidents often occur during cold weather, which is also flu season, so the symptoms may be confused with someone having the flu. However, CO poisoning victims will not normally present with a fever. Symptoms of exposure may also be confused with food poisoning or other illnesses.

Overhaul risks

Overhaul at a fire scene is a time when many dangers present themselves to firefighters, although it is also a time when firefighters may be less aware of the potential for threats. Among the perils during overhaul is the potential for inhalation hazards. More than 12 hours after a devastating fire, respiratory protection filters are good at a generation site and that contributed to building collapse, carbon monoxide readings taken outside at the front door exceeded 100 ppm while crews wetted down the remains.

Overhaul: The Phoenix study

In 1998, the City of Phoenix Personnel Department Safety Section joined with the Phoenix Fire Department, the University of Arizona Prevention Center and Arizona State University to conduct a scientific study of firefighter exposures to a variety of contaminants during fire overhaul, including carbon monoxide. This study was scientifically based, with trained personnel conducting it, and involved monitoring the air during the overhaul phase of 25 structure fires. The study found, among other things, that carbon monoxide levels exceeded the National Institute for Occupational Safety and Health’s (NIOSH) ceiling value of 200 ppm at five of the 25 incidents.

Concentrations of air contaminants during fire overhaul exceed occupational exposure limits. Without the use of respiratory protection, firefighters are exposed to irritants, chemical asphyxiates and car- cinogens. Therefore, respiratory protection is recommended during fire overhaul. SCBA should be used in atmospheres with CO concentrations above 150 ppm, and air purifying respirators (APRs) may be used when CO concentrations are below 150 ppm. Finally, CO concentra- tions should not be used to predict the presence of other contaminants found in the overhaul environment.

Carbon monoxide is a true hazard during the overhaul phase of structure fires. The OSHA Permissible Exposure Limit (PEL) for carbon monoxide is 50 ppm over an eight-hour period. In the Phoenix study, the average sample concentration was 52.6 ppm for 65 samples taken, with a maximum reading of 260 ppm. Although the calculated time-weighted average (TWA) for exposures were actually less than the OSHA PEL, it is clear that potentially haz- ardous CO levels are present during overhaul. As firefighters overex- ert themselves in the firefighting, they will breathe harder and thus have the potential to inhale larger quantities of carbon monoxide.

For this reason, it is critical to control hazards so they are not generated by the fire itself. If a sudden cold spell occurs, and windows that are normally left open are suddenly closed. Another potential problem occurs when heating and air-conditioning systems wind up with outside air intake shut, so all of the air in the area is simply recirculated. Buildings located near very busy roadways may experience higher than normal CO levels, particularly during periods of heavy traffic. Weather conditions can also play a role, such as when an atmospheric inversion prevents pollutants from escaping the immediate atmosphere, forcing them to build up near their generation point.

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Fatigue or CO Poisoning?

You need to know. A firefighter with carbon monoxide (CO) poisoning needs more than rest - he needs immediate treatment. Undetected, untreated episodes of CO poisoning can lead to permanent neurological damage.

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