

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.**

JEMS
JOURNAL OF EMERGENCY MEDICAL SERVICES

FIRE RESCUE

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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**



PHOTO KARI GREER

BIZARRE CRASH PRODUCES UNEXPECTED HAZARD

By A.J. Heightman, Editor-in-Chief, JEMS

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.

The trapped patient's other complaints and visible injuries included a fracture/dislocation of his right femur/hip, an open fracture of his left patella, facial abrasions, neck and back pain, and dyspnea.

I asked state troopers and bystanders to stay with the driver and keep his neck and spine in a neutral position and gave priority attention to the trapped patient. I radioed an incoming mini-pumper to provide me with water to dilute and cool the hot water leaking on the patient and requested 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 methylene 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 pre-



PHOTO 1



PHOTO 2



PHOTO 3

sented with symptoms that included tachycardia, hypotension, lethargy and dyspnea. My pulse was 130, 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. ♦

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THE GREAT IMITATOR

Understanding & treating carbon monoxide poisoning

By Dana Bartlett

A JEMS CONTINUING EDUCATION FEATURE

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Ever since humans discovered that burning wood would keep them warm, people have been exposed to carbon monoxide (CO). Despite widespread public awareness and education, CO poisoning is still all too common; it is the No. 1 cause of death by poison in the United States. In its annual report for 2004, the American Association of Poison Control Centers (AAPCC) recorded more than 75,000 exposures to carbon monoxide and 74 deaths, and it is likely that there are many cases of CO poisoning that are never brought to a physician's attention or are misdiagnosed. CO poisoning causes significant morbidity and death, and consumes first responder and emergency department resources. In short, CO poisoning is a serious national health problem.

OBJECTIVES

- List common sources of CO poisoning.
- Explain how CO poisoning affects oxygen delivery to the tissues.
- Describe treatment options for CO-poisoned patients, including hyperbaric oxygen.

CO sources

Carbon monoxide is aptly called *the silent killer*. Odorless and colorless, CO is produced by incomplete combustion of a carbon-containing fuel, and exposure can be fatal. Oil and other petroleum products,

natural gas, coal and wood can all be sources. Devices such as furnaces, ovens, space heaters and water heaters are all potential CO-generating culprits. Automobile exhaust is the biggest source of CO, followed by fire smoke inhalation.¹

Note: A lesser known source of carbon monoxide is the vapors from *methylene chloride*, a compound commonly found in paint strippers. When the fumes are inhaled, it is converted *in vivo* to carbon monoxide.

CO produced by fuel-burning devices in the home is not usually a problem in well-maintained devices that are properly ventilated. Problems occur when these devices are not properly maintained or serviced, or when ventilation systems become blocked, such as a dryer filter packed with lint or a furnace flue blocked by a bird's nest. Dangerous levels of CO can accumulate quickly with catastrophic results. Many cases of CO poisoning in the home are preventable with maintenance, inspection and proper ventilation.

There are also less-obvious sources of CO exposure. People have been poisoned by CO when riding in the back of a pickup truck, riding in a powerboat, operating forklifts in a factory, using hibachi grills and after being exposed to the fumes from a gas log fireplace.

Lesser known risky activities include glassblowing, working with ceramics, commercial jewelry making and welding. Some outdoor sports involve equipment that can produce toxic levels of CO, such as stoves used in mountaineering tents or faulty SCUBA gear used in diving.

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.²

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).³ However, during overhaul, there may be a tendency to overlook this fact. Fire crews 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.

Pathophysiology

The pathophysiology of CO poisoning is complex. However, despite the many mechanisms by which CO produces poisoning, they each produce the same result: preventing oxygen from reaching tissues. CO produces tissue hypoxia by the following mechanisms:

- **Binding to hemoglobin.** Hemoglobin is a protein-iron complex that binds with oxygen and delivers it to the tissues. CO also binds with hemoglobin (the CO hemoglobin complex is called *carboxyhemoglobin*), but it does so much more avidly; the affinity of CO for hemoglobin is 200–250 times greater than that of oxygen. If CO is present in the blood, there may be adequate oxygen in the inspired air, but it must compete, unsuccessfully, with CO for hemoglobin-binding sites.
- **Transfer of oxygen to the tissues.** Even in cases of CO poisoning, there is some binding of oxygen to hemoglobin. However, in the presence of carboxyhemoglobin, oxygen is much more tightly bound to hemoglobin, and it is not released to tissues. Tissue oxygen pressures must be dangerously low before the oxygen is released.

These mechanisms are the “classic” explanations of why CO poisoning affects oxygen delivery. But research suggests that although CO binding to hemoglobin and decreased release of oxygen may be important components of CO poisoning, they do not completely explain the clinical picture. In addition, the damage done by CO is probably not completely explained by those two effects. Other mechanisms are probably more important, and they can be divided into these categories:



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 guanylate cyclase (an enzyme that catalyzes the formation of cyclic guanosine monophosphate, a potent vasodilator) and causes the release of nitric oxide (a potent vasodilator) from platelets. Vasodilation 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 poor perfusion and lack 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

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.



PHOTO MIKE MEADOWS

When responding to a fire, crews must take the necessary PPE precautions to prevent CO exposure.

depression of cardiac function and vasodilation); and 3) stimulates the production of free radicals that damage tissue.

Clinical signs & symptoms

CO has been called the great imitator. The 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 occur when you are driving your car? When was the last time the car's exhaust system was inspected?
- 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 cases 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 be comatose or develop seizures.
- **Cardiac:** The cardiac signs of CO poisoning reflect the decreased myocardial function and vasodilation CO causes (e.g., the patient may be hypotensive) and reflect the decreased oxygen delivery to, and utilization of, oxygen by the myocardium (e.g., the patient may have tachycardia, chest pain, arrhythmias or myocardial ischemia). Most deaths from CO poisoning result from ventricular dysrhythmias.
- **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 this fact: *CO interferes 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.*

Note: 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.

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.

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 oxygen saturation if the patient has been exposed to CO. The readings will be falsely high, and the difference between the pulse oximetry reading and the actual saturation of hemoglobin with oxygen increases as the COHb increases.

What levels of COHb 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.⁴ The current Occupational Safety and Health Administration (OSHA) limit for the average amount of CO that is safe for an eight-hour work 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 neuropsychiatric sequelae are similar to recurrent symptom syndrome, but the 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%.⁵

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PHOTO KARTI GREER

Wildland fires present a significant CO threat to responders who frequently do not have adequate respiratory protection.

The pregnant patient

The pregnant patient presents a special challenge. One exposure to CO affects two patients, and it's 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 declines 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 when it was applied and how long after the exposure ended it was applied. Place the patient on a cardiac monitor and observe for arrhythmias. If 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:

children (compared with adults, their metabolic rate is higher so their need for oxygen is greater); adults with cardiac disease (CO depresses myocardial function, causes vasodilatation and binds with myoglobin [the myocardium depends on myoglobin], and the heart is very active metabolically); pregnant women or women who may be pregnant; patients with an increased oxygen demand (e.g., fever) or decreased oxygen-carrying capacity; and patients with chronic respiratory insufficiency.

Once these steps are complete, it's time for transport. Should the patient go to the closest emergency department, or should they go to a hospital equipped with a hyperbaric oxygen chamber? It's not a simple decision.

Hyperbaric oxygen: To dive or not to dive?

There is no doubt about the mechanism by which hyperbaric 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 atmospheric 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 by 10 times the amount of oxygen dissolved in plasma.

It would appear that HBO would be a very valuable therapeutic tool for treating cases of CO poisoning, but there is a lot of controversy about HBO in the medical world. Given the available evidence—including theoretical, clinical and practical considerations—it's not difficult to understand why. Consider the following:

- Transport time to an HBO chamber is often lengthy; by the time the patient gets to the chamber, their COHb may have declined considerably. Transport to a tertiary care center with HBO capabilities may warrant utilization of air medical transport systems.
- Patients who have traditionally been considered candidates for HBO have been symptomatic patients. The possible benefits of HBO must be weighed against the risk of transporting unstable patients between hospitals. Also, patients who are very symptomatic must be stabilized before they can be moved. This takes time, and, again, by the time they get to the HBO chamber, the patient's COHb level will probably have declined considerably.
- CO poisoning is complex. It almost certainly causes damage by several different mechanisms, and it's not clear which of these HBO might treat. HBO can decrease COHb half-life and increase the amount of oxygen dissolved in the plasma, but as mentioned before, COHb binding to hemoglobin may not be why CO causes damage. CO poisoning may also be a process, and no one knows when during that process HBO might be useful.
- It's unclear which patients are HBO candidates. The criteria used in the past were based on the treating physician's clinical judgment. Physicians traditionally considered patients candidates for HBO treatment if they had a syncopal episode, were comatose, were seizing, had a serious arrhythmia or were pregnant, but no unequivocal proof exists showing HBO is the best oxygen therapy for any particular group of CO-poisoned patients. Should all pregnant patients exposed to CO receive HBO treatment? Many sources state that pregnant women with a COHb >15% should receive HBO therapy. Should all children poisoned with CO

receive HBO therapy? Should it be restricted to patients with a high COHb level regardless of signs and symptoms, or should the patient's clinical status be the deciding factor?

- The goals of HBO therapy include: 1) preventing deterioration in the patient's clinical status; 2) decreasing recovery time; and 3) preventing permanent damage caused by hypoxia. However, after years of detailed examination of HBO use, there is no agreement as to whether or not HBO works. The results from several large studies differ. This is the most controversial issue about HBO, and despite much research, the question remains.
- A purported benefit of HBO is the prevention (and at times, treatment) of delayed neurological sequelae. Unfortunately, identifying which patients with CO poisoning may develop this problem (and thus which patients are HBO candidates) is not easy. And if HBO is to be used for patients who have developed neurological sequelae, how long after exposure can it be expected to be useful? How many treatments should they receive? Finally, there is no agreed upon testing method for detecting delayed neurological sequelae, so determining the effectiveness of HBO in these cases is difficult.

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 many 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 "serious cases" (patients who are comatose, pregnant patients, patients with a COHb level >30% or patients having a serious arrhythmia or seizing) 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 proved effective. ♦

Dana Bartlett is a registered nurse and a certified specialist in poison information at the Philadelphia Poison Control Center. Contact him at bartlett@email.chop.edu.

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THE GREAT IMITATOR

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 recorded in the United States
2. **The leading cause of CO poisoning is:**
 - a. space heaters
 - b. faulty furnaces
 - c. automobile exhaust
 - d. fire smoke inhalation
3. **During overhaul, the danger from CO poisoning is:**
 - a. higher than during the active fire phase
 - b. still a potential danger that must be considered
 - c. a problem only if the firefighters remain in a closed space
 - d. removed, because no danger from CO poisoning remains
4. **CO causes tissue hypoxia by which of the following mechanisms?**
 - a. CO binds to hemoglobin at a two times greater affinity than oxygen
 - b. CO causes oxygen to bind less tightly to hemoglobin, releasing before the targeted tissues
 - c. CO causes massive vasoconstriction, preventing blood flow to tissues
 - d. CO binds to myoglobin, decreasing oxygen transport in the cells
5. **Diagnosing CO poisoning is:**
 - a. easily completed by assessing signs and symptoms
 - b. difficult, as signs and symptoms of CO imitate 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 fire
6. **Which of the following is expected in mild cases of CO poisoning?**
 - a. seizures
 - b. respiratory alkalosis
 - c. metabolic acidosis
 - d. renal failure
7. **A blood CO level of 4-5% in a person who smokes cigarettes reflects:**
 - a. a normal level of CO in smokers
 - b. acute CO poisoning in a smoker
 - c. a normal CO level even for a non smoker
 - d. a highly elevated level for a smoker
8. **The half life of CO when breathing room air is:**
 - a. 40-60 minutes
 - b. 1-2 hours
 - c. 2-4 hours
 - d. 4-6 hours
9. **The half life of CO when breathing 100% oxygen is:**
 - a. 40-60 minutes
 - b. 1-2 hours
 - c. 2-4 hours
 - d. 4-6 hours
10. **The best indicator of seriousness of CO poisoning is:**
 - a. patient condition
 - b. carboxyhemoglobin level
 - c. CO level in the blood
 - d. pulse oximetry
11. **Which of the following is typical of recurrent symptom syndrome?**
 - a. It occurs after a severe CO poisoning.
 - b. There is an asymptomatic period of 10-15 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. fetus at no risk because CO doesn't cross the placenta
 - b. mother at greater risk for CO poisoning than the fetus
 - c. fetus at greater risk for CO poisoning than the mother
 - d. mother at decreased risk because most of the CO crosses the placenta to the fetus
13. **In hyperbaric oxygen chambers, the patient breathes oxygen at:**
 - a. two to three times the normal atmospheric pressure
 - b. half the normal atmospheric pressure
 - c. a faster than normal respiratory rate
 - d. an increased oxygen percentage
14. **Breathing oxygen in a hyperbaric oxygen chamber decreases the half life of CO to:**
 - a. 20 minutes
 - b. 40 minutes
 - c. 60 minutes
 - d. 90 minutes
15. **Which patient is most likely to benefit from HBO therapy?**
 - a. patient exposed to methylene chloride
 - b. pregnant patient
 - c. patient with a COHb level of 10%
 - d. confused or dizzy patient

CE Test #222

This test is worth 1.5 contact hours.

16. **Most deaths from CO poisoning occur from:**
 - a. ventricular dysrhythmias
 - b. respiratory arrest
 - c. congestive heart failure
 - d. massive brain swelling
17. **Which of the following is true of CO poisoning from methylene chloride vapors?**
 - a. The signs and symptoms pass quickly.
 - b. The symptoms from this type of poisoning are different from other CO poisoning.
 - c. Methylene chloride is stored in fat and slowly released.
 - d. Methylene chloride poisoning is less serious than other types of CO poisoning.
18. **Which patient is at the greatest risk for developing delayed neuropsychiatric sequelae?**
 - a. patient exposed to 50 ppm CO over eight hours
 - b. patient who lost consciousness
 - c. patient exposed to methylene chloride
 - d. patient with COHb levels of 5-10%
19. **Cardiac dysrhythmias caused by CO poisoning should be treated with:**
 - a. standard therapy
 - b. double doses of cardiac medications
 - c. half the normal dose of cardiac medications
 - d. no cardiac medications; they are ineffective in the presence of CO
20. **Hyperbaric oxygen therapy works by:**
 - a. recruiting more hemoglobin for oxygen transport
 - b. directly delivering oxygen to hypoxic tissues
 - c. forcing CO from the myoglobin
 - d. increasing the oxygen dissolved in plasma

CE Answer Sheet #222: THE GREAT IMITATOR

To be considered for credit, this test must be postmarked by May 15, 2007.

Name _____

Address _____

City/State/ZIP _____

Phone # _____

E-mail _____

Organization _____

State Certification # _____

State of Licensure _____

Expiration Date _____

National Registry # _____

Skill Level: ☐ EMT ☐ EMT-I ☐ Paramedic

Area of Service: ☐ Rural ☐ Urban

Status: ☐ Volunteer ☐ Paid

Instructions for Obtaining CE Credit

1. Study the CE article in this issue.
2. Answer the test questions on this form that are appropriate to your level of care. (Photocopies of the answer strip are accepted if others wish to take the test; for grading purposes, however, the strip should not be enlarged or reduced.)
3. Mail completed answer strip with \$10 testing fee to: **JEMS COMMUNICATIONS, 525 B ST., STE. 1900, SAN DIEGO, CA 92101-4495, ATTN: JEMS CE**
4. Within six weeks, you will receive your test score and, if you passed, a CE certificate. The passing score is 70%.

Answers

Mark your answers in the appropriate box (1-20) (☐) Paramedics are graded on questions 1-20 (required), 6 errors = fail. EMTs and others are graded on 1-15 (16-20 corrected FYI), 4 errors = fail.

1. ☐ a. ☐ b. ☐ c. ☐ d.
2. ☐ a. ☐ b. ☐ c. ☐ d.
3. ☐ a. ☐ b. ☐ c. ☐ d.
4. ☐ a. ☐ b. ☐ c. ☐ d.
5. ☐ a. ☐ b. ☐ c. ☐ d.
6. ☐ a. ☐ b. ☐ c. ☐ d.
7. ☐ a. ☐ b. ☐ c. ☐ d.
8. ☐ a. ☐ b. ☐ c. ☐ d.
9. ☐ a. ☐ b. ☐ c. ☐ d.
10. ☐ a. ☐ b. ☐ c. ☐ d.
11. ☐ a. ☐ b. ☐ c. ☐ d.
12. ☐ a. ☐ b. ☐ c. ☐ d.
13. ☐ a. ☐ b. ☐ c. ☐ d.
14. ☐ a. ☐ b. ☐ c. ☐ d.
15. ☐ a. ☐ b. ☐ c. ☐ d.
16. ☐ a. ☐ b. ☐ c. ☐ d.
17. ☐ a. ☐ b. ☐ c. ☐ d.
18. ☐ a. ☐ b. ☐ c. ☐ d.
19. ☐ a. ☐ b. ☐ c. ☐ d.
20. ☐ a. ☐ b. ☐ c. ☐ d.



Learn more at www.fire rehab.com

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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.

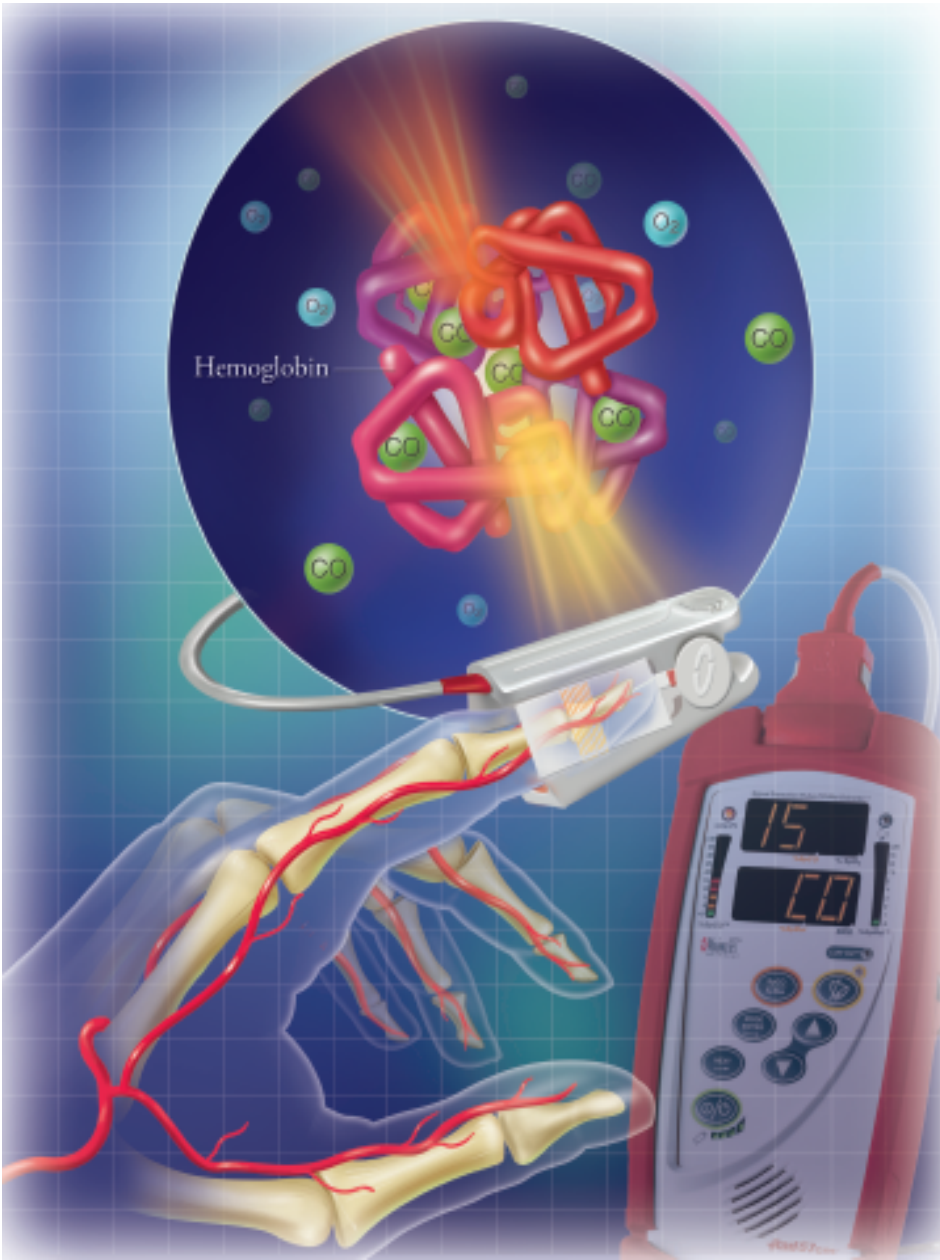
CO's toxic mechanisms

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 non-specific, 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 $\leq 2.5\%$ and 98% of smokers have a level $\leq 10.0\%$ (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 SpO₂ and heart rate. The device's accuracy has been demonstrated up to 40% SpCO, with a range of $\pm 3\%$ 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,

TABLE 1: COHB LEVELS IN PERSONS 3–74 YEARS OF AGE¹⁴

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

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

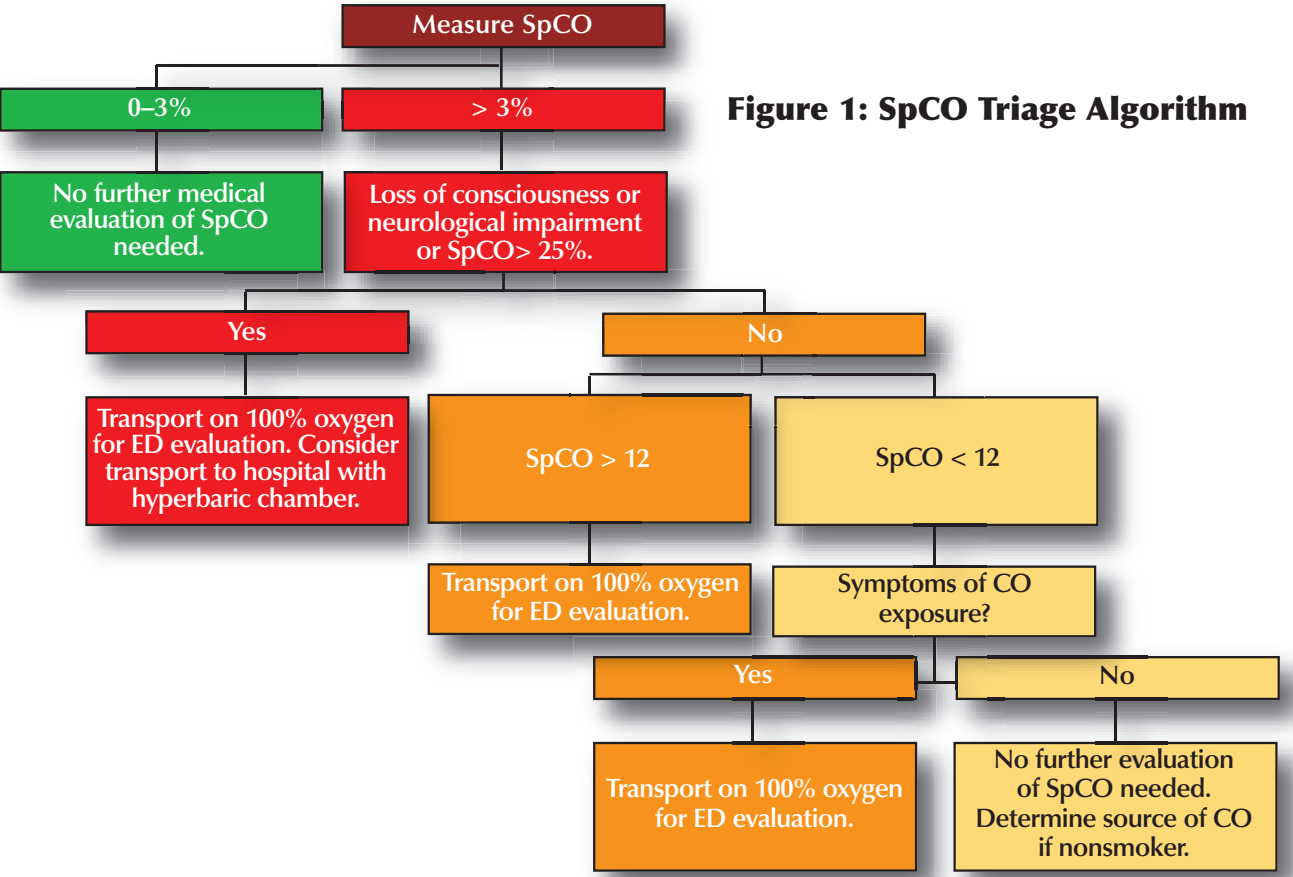
Because a clinician has traditionally ordered blood measurement of COHb only when the condition was suspected, it is likely that there has been a tendency to measure COHb only in the more symptomatic patient or in those whose exposure history was known. Because EMS providers and paramedics commonly use 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.





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.

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FIREFIGHTER RESPONSE TO CO INCIDENTS

Vigilance keeps responders from becoming victims

By Greg Jakubowski



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.

AP/WIDE WORLD PHOTO / MISHA JAPARDZE

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

A SEALED-UP HOME, BELOW-GRADE AREA, GARAGE OR SIMILAR LOCATION PRESENTS CONDITIONS WHERE CARBON MONOXIDE LEVELS COULD BE HIGH.

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 intakes 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 LEL, CO or other gases) utilized in an emergency response as per the meter manufacturer's recommendations. Uncalibrated meters could 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's source, do not ventilate the building. CO is just slightly lighter than air, with a vapor



The best way for firefighters to protect themselves from carbon monoxide exposure during overhaul operations is to wear SCBA.

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 all 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 least aware of the potential for threats. Among the perils during overhaul is the potential for inhalation hazards. More than 12 hours after a devastating multiple-alarm fire at a good-size one-story office building that resulted in the roof of the building collapsing, 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 overexposed to irritants, chemical asphyxiates and carcinogens. 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 concentrations should not be used to predict the presence of other contaminants found in the overhaul environment.¹

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 hazardous CO levels are present during overhaul. As firefighters overexert themselves in the firefight, they will breathe harder and thus have the potential to inhale larger quantities of carbon monoxide.

The best way to reduce this hazard is first by engineering controls—move as much clean air as possible into the working space and monitor the air for hazardous CO levels. The second best way to reduce the hazard is with respiratory protection. Although air-purifying respirators that protect against CO are available on the market, the best way for firefighters to protect themselves against exposure during overhaul, when engineering controls are not working, is to wear self-contained breathing apparatus.

CO testing in rehab

At a fire scene, CO may not just be generated by the fire itself.

RESOURCES FOR CO INCIDENT RESPONSE

The following Web sites provide standard operating guidelines for response to CO incidents:

- www.madison-maine.org/media/PDF/SOG_CO.PDF
- www.firerescue.navajo.org/Documents/CO%20Response.doc
- www.cstone.net/firedept/book3/3-iv-15.htm

Numerous vehicles are likely to be operating on scene, and their exhaust is likely to result in elevated CO levels in the vicinity. Firefighters have generally begun to recognize the importance of good rehab to their physical well being. However, rehab must be done properly. It is important to locate the rehab/rest area away from vehicle exhaust to avoid continuously exposing firefighters to carbon monoxide. Although general good practice would put the rehab area upwind of the fire scene and most of the operating vehicles, it would still be beneficial to verify that the air in the rehab area is clean via a quick check with a CO meter. The use of a CO detector, such as the Rad-57, in the rehab area will also aid in detecting emergency personnel exposed to dangerously high CO levels.

Final note

CO presents risks to firefighters. Responders need to remember to consider CO poisoning when responding to medical incidents that involve patients with suspicious symptoms and those located in enclosed areas that have the potential for CO buildup. Firefighters also need to remember that carbon monoxide, as well as other inhalation hazards, are likely to be present during the overhaul phase of a fire. Efforts should be made via engineering controls—principally ventilation—to minimize the hazard. If the hazard cannot be minimized, firefighters should wear breathing apparatus. Finally, those establishing rehab areas on the fireground should do so upwind of the fire building and the majority of the operating vehicles to minimize the potential for ongoing CO exposure while firefighters are resting. Keeping these concepts in mind will help responders maintain their health and live to protect their communities another day. ♦

Greg Jakubowski is a fire protection engineer and a certified safety professional with 27 years of fire service experience. He is a Pennsylvania State Fire Instructor and serves as a captain with the Linghocken Fire Company in Bucks County, Pa. Greg is also a principal in Fire Planning Associates, Washington Crossing, Pa., a company dedicated to helping fire departments and businesses preplan for emergency situations.

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Recommended Reading

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CARBOXYHEMOGLOBIN LEVELS & CLINICAL MANIFESTATIONS		
COHb LEVEL (%)	MANIFESTATIONS	TREATMENT & TRANSPORT DECISION
5	Mild headache	100% O ₂
10	Mild headache, shortness of breath with vigorous exertion	100% O ₂
10–20	Shortness of breath with mild exertion, moderate headache	100% O ₂
20–30	Worsening headache, nausea, dizziness, fatigue	Hyperbaric Oxygen (HBO)
30–40	Severe headache, vomiting, vertigo, altered judgment	HBO
40–50	Confusion, syncope, tachycardia	HBO
50–60	Seizures, shock, apnea, coma	HBO
60–70	Seizures, coma, cardiac arrhythmias, death	HBO
> 70	Death within minutes	HBO

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.

Now there's a way you can detect carbon monoxide poisoning in the field—The Rad-57™ from Masimo®. Just clip the sensor on the firefighter's finger and press a button. The Rad-57 detects the percentage of carbon monoxide in the bloodstream by measuring absorbed light.

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Rad-57

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