



## Firemedically

with Mike McEvoy

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### Response to CO Alarms: Time For Another Detector?

Carbon monoxide incidents account for an increasing number of fire department responses. Based on the most contemporary medical and scientific knowledge of CO and how it affects people, it seems high time for some modernization of fire service response to CO alarms and incidents. CO is not only a colorless, odorless and invisible gas, but a toxin with symptoms so elusive that up to half of all CO poisoned patients are routinely misdiagnosed by EMS and emergency department physicians. Sadly, and sometimes catastrophically, missing this diagnosis will often result in the return of a patient to a poisoned environment. With new technologies, the fire service is poised to become either part of the problem or the solution to screening CO poisonings in the community. Lives are clearly on the line.

An analysis of National Fire Incident Reporting System data coupled with NFPA fire experience surveys<sup>(1)</sup> put the number of municipal fire department responses to CO incidents at 61,100 in 2005 when calls where nothing was found or fire was present were excluded. The last time when false alarms, CO detector malfunctions, and CO incidents where nothing was found were included was 2003, due to the increasingly large size of the NIFRS database. These events more than doubled the number of CO responses reported.

It's no secret that CO incidents are on the rise and the NFPA analysis seems to put the increase at about 9 percent per year, which would result in roughly 77,600 incidents in 2008 if we exclude CO incidents where nothing was found or fire was present, and just under 100,000 incidents if we include CO false alarms and detector malfunctions. Statistics also reveal that while nearly one-third of CO incidents in the United States occur during December and January, 69 percent happen during October and March, the prime home heating months. Speaking of homes, 92 percent of CO incidents occur in residential occupancies, while three quarters of incidents occur between the hours of 9:00 a.m. and 10:59 p.m.

While most dispatch protocols assign EMS to CO alarms only when illness is present, everything we know about the gas tells us not to rely on signs and symptoms to identify poisoned patients. If physicians miss up to 50 percent of CO poisonings, the lay public is undoubtedly even less capable of distinguishing CO poisoning from the flu, gastrointestinal maladies, headache or general malaise, all of which are CO poisoning symptoms<sup>(2)</sup>. While symptoms of CO poisoning can be stratified according to the level of CO bound to hemoglobin in blood (carboxyhemoglobin or COHb), attempts to correlate COHb with symptoms or symptoms with COHb levels have consistently failed<sup>(3,4)</sup>. The only reliable indicator of significant CO poisoning is measured blood COHb levels, which until recently required laboratory testing of a blood sample.

#### 'Readily disperse'

As a gas, CO has a vapor density of 0.97, very close to ambient air (1.00). This means that CO will readily disperse throughout the atmosphere, traveling throughout an enclosed structure or immediately dispersing into the outdoors when doors, windows or other sources of ventilation are provided. It has Lower and Upper Explosive Limits of 12.5 percent (LEL) and 74 percent (UEL), providing a degree of flammability significant enough to warrant careful control

of ignition sources. The Immediate Danger to Life and Health (IDLH) of 1200 PPM is relatively high compared to other toxic gases, but as a virtually invisible poison CO is unlikely to provide any warning to unsuspecting responders entering a toxic environment without breathing apparatus. CO also displaces oxygen from ambient air, and at IDLH levels and above will often result in oxygen levels incompatible with life. Several major research studies have demonstrated serious and long-term health consequences from short term and low level CO exposures.

The most significant of these are delayed neurologic syndrome (DNS) <sup>(5)</sup>, a highly variable but life altering set of brain and cognitive symptoms, and substantially increased risk of major cardiovascular events such as stroke, heart attack (often leading to earlier death) following even a single moderate to severe CO exposure<sup>(6)</sup>. DNS occurs in 11 to 33 percent of CO poisoned patients, often with devastating consequences resulting from memory loss, intelligence decline, seizures, concentration difficulties and speech problems<sup>(7,8)</sup>. Equally alarming are studies demonstrating a 3.7 times greater risk of having a cardiovascular event and 2.2 times greater risk of death during a 19-year study of Swedish never-smokers who lived or worked with smokers, exposing them to slight, but measurable levels of atmospheric CO<sup>(9)</sup>.

CO binds extremely tightly to hemoglobin in the human bloodstream, taking many of the seats normally occupied by oxyhemoglobin. It creates additional hypoxia by increasing the affinity of oxygen molecules already bound to hemoglobin units. This unwillingness of existing oxyhemoglobin to unload into the tissues theoretically results in the (rarely seen) cherry red skin color appearance of a CO poisoned patient. That is, normally deoxygenated venous blood remains fairly pink from the stubborn oxygen molecules that refuse to step off into the cells. CO also binds with myoglobin (muscle tissue), significantly inhibiting skeletal and cardiac muscle function. While these weaknesses were long thought consequential to hypoxic injury at the cellular level, recent studies in mice suggest the process is far more sinister<sup>(10)</sup>. CO also induces nitric oxide formation, leading to increased production of oxygen free radicals now implicated in progressive and delayed damage to blood vessel walls, the heart and brain.

Acute exposures to high levels of CO produce a constellation of symptoms reflective of how CO behaves in the body. These typically include headache, nausea and vomiting, dizziness, altered mental status, weakness progressing to collapse, hypotension, and ultimately death resulting from ventricular fibrillation. Most humans would collapse and die within one to two minutes in a 12,800 PPM CO environment, within 10-15 minutes at 6,400 PPM, 30 minutes at 3,200 PPM, and at least lose consciousness after one hour at 1,000 PPM<sup>(11)</sup>. The affinity of CO for hemoglobin also results in significant damage and many deaths from chronic, low level exposure to CO. Environmental exposure to CO from pollutants and combustion typically averages 10 PPM and can reach as high as 30 PPM in urban areas.

A well known Salt Lake City attorney was found dead in his home from a week-long exposure to 130 PPM of CO emitted by a faulty boiler. He had complained to coworkers of nausea and flu like symptoms for several days<sup>(12)</sup>. While the half life of CO in the bloodstream is approximately four hours on room air, it shortens to about 45 minutes on 100 percent oxygen<sup>(8)</sup>. Meanwhile, the CO inhaled from smoking a cigarette can be measured in the bloodstream for up to 48 hours (a fact used in smoking cessation clinics in the U.K.).

A constellation of physical symptoms associated with increasing levels of COHb (carboxyhemoglobin, or carbon monoxide in the blood) is often used to assist health care providers and consumers with recognizing CO poisoning. But it may actually encourage misdiagnosis rather than aid in identifying CO poisoning for two very important reasons. First, the experience of signs and symptoms vary widely across the population and are so inconsistent that no medical model has ever been even remotely successful at predicting CO levels based on symptoms. Second, a quick glance through the list will reveal that the entire constellation of CO poisoning symptoms is remarkably similar to symptoms of the flu, viral illness, general malaise and a host of other vague or poorly defined diagnoses.

<b>SpCO%</b>	<b>Clinical Manifestations</b>
< 5%	None
5-10%	Mild headache, tire easily
11-20%	Moderate headache, exert ional shortness of breath (SOB)

21-30%	Throbbing headache, mild nausea, dizziness, fatigue, slightly impaired judgment
31-40%	Severe headache, vomiting, vertigo, altered judgment
41-50%	Confusion, syncope, tachycardia
51-60%	Seizures, unconsciousness

null

The inability to screen for CO based on symptoms and close resemblance to viral illnesses is a frequent reason why up to half of CO poisonings are missed. It has also led many progressive public health and medical authorities to call for routine screening of every patient for CO poisoning, especially now that a quick, non-invasive tool exists to accomplish this. The RAD-57 is a non-invasive pulse CO-Oximeter that utilizes additional wavelengths of light beyond the two infrared wavelengths used by conventional pulse oximeters to distinguish not only oxyhemoglobin, but other forms of hemoglobin including carboxyhemoglobin<sup>(13)</sup>. The technology has been available to the fire service and medical community in a portable, handheld unit since January of 2006. While the merits of screening every patient for CO might be questioned, it is now an expectation that a prudent health care provider would screen any patient with symptoms of, or suspected or known exposure to CO<sup>(14,15)</sup>. That implies that every response to a CO detector activation is a response to suspected CO exposure. In addition to atmospheric monitoring, people who were inside the premises must be screened for CO.

When it comes to residential CO detectors manufactured after 1998, they'll activate for a variety of conditions including CO of 30 PPM for 30 days, 70 PPM for one to four hours, 150 PPM for 10 to 50 minutes, 400 PPM for four to 15 minutes, CO<sub>2</sub> levels above 5,000 PPM, and specified levels of methane, butane, heptane, ethyl acetate and isopropyl alcohol<sup>(11, 16)</sup>. Few, if any detectors tell firefighters why they activated. Properties of CO allow homeowners to ventilate a residence within seconds or minutes, sometimes merely by exiting the premises while awaiting fire department response. There is no consistent, widely practiced, evidence based procedure for atmospheric monitoring at the scene of a CO incident or alarm activation. Inspecting and operating appliances while measuring for ambient CO is a widely used practice with extremely variable results. Behavior of CO in the human body, however, is extremely predictable. Although CO symptoms vary widely from person to person, an individual removed from a CO poisoned environment will carry evidence of CO in their bloodstream for hours or days following the exposure. All the fire service needs to do is measure it.

Atmospheric monitoring at CO alarms and incidents is an essential safety practice to protect firefighters and the public from toxic environments. CO is the leading cause of poisoning deaths in industrialized nations, the United States included. Consistently missing unsuspected cases of CO poisoning is no longer acceptable given the ability for any health care provider to non-invasively screen people with suspected or known exposure to CO for poisoning. Every CO call with civilians on scene is an EMS call. If people knew when they were CO poisoned, there would be no need for CO detectors. The detector is telling us to be suspicious. Screen the building with atmospheric monitoring. Screen the people on scene with non-invasive CO-oximetry. Sleep better at night knowing you never assured a homeowner it was safe for their family to return to a CO poisoned residence. CO can be ventilated from a structure within minutes; it takes hours to disappear from human blood. The time has come to add a human CO detector (CO-oximeter) to the tools carried by the fire service when responding to CO alarms and incidents.

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