We've long known that up to 45 percent of on-duty firefighter deaths are due to cardiovascular disease. Generally, these deaths are attributed to coronary artery disease. A recent study found that most on-duty firefighter deaths occur during active fire suppression activities (see the table on p. 70). However, deaths were reported during all aspects of firefighter duties.

Some experts have suggested that many of these deaths are due to a lack of physical fitness, obesity and similar risk factors. Indeed, it seems intuitive that cardiovascular...
risks in firefighters should be similar to those of the population as a whole. However, recent research indicates we must consider other risk factors as well. Specifically, firefighters’ exposure to carbon monoxide may significantly increase their risk of coronary heart disease.

**CO DANGERS**

Carbon monoxide (CO) is the most common cause of poisoning in industrialized countries. Typically, following CO exposure, there will be a phase of decreased oxygen in the blood (hypoxemia). This is usually followed by reoxygenation when the victim is removed from the toxic environment and oxygen administered. It also occurs normally when carboxyhemoglobin (COHb), produced as a result of the poisoning, breaks down and is replaced with normal hemoglobin.

These periods of hypoxemia often result in the formation of dangerous chemicals called free radicals, highly reactive chemical compounds that cause cell damage. An increase in free radical compounds results in oxidative stress, which damages body cells and is associated with the development of many diseases, including atherosclerosis, Parkinson’s disease and Alzheimer’s disease. Thus, oxidative stress can cause injury to oxygen-sensitive tissues, such as the brain and the heart, beyond those caused by the initial hypoxemic insult.

CO poisoning can occur following both acute and chronic exposures; both can result in long-term, often permanent, problems. CO is an occupational risk for firefighters. Although a well-fitted self-contained breathing apparatus (SCBA) can protect firefighters from environmental gases, they often don’t wear their SCBA through all phases of fire operations. It’s not uncommon to see COHb levels up to 5 percent (in non-smokers, 0–0.1 percent is normal) during fire overhaul, when firefighters often remove their SCBA.

Deaths from Coronary Heart Disease among Firefighters Based on Duty Type* (1/1/94–12/31/04)

<table>
<thead>
<tr>
<th>DUTY</th>
<th>DEATHS (N=449)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fire Suppression</td>
<td>144 (32.1 percent)</td>
</tr>
<tr>
<td>Alarm Response</td>
<td>60 (13.4 percent)</td>
</tr>
<tr>
<td>Alarm Return</td>
<td>78 (17.4 percent)</td>
</tr>
<tr>
<td>Physical Training</td>
<td>56 (12.5 percent)</td>
</tr>
<tr>
<td>EMS and Non-Fire Emergencies</td>
<td>42 (9.4 percent)</td>
</tr>
<tr>
<td>Fire Station and Other Non-Emergencies Duties</td>
<td>69 (15.4 percent)</td>
</tr>
</tbody>
</table>

**CO & THE HEART**

The heart is highly dependent on a constant supply of oxygen to function normally and is highly susceptible to decreases in available oxygen. As discussed above, myocardial injury results from tissue hypoxia as well as cellular damage due to the release of free radicals.

Victims of moderate to severe CO poisoning are at increased risk of developing cardiovascular complications. These CO complications occur in all age groups regardless of the patient’s underlying health status. In a 2005 study, researchers in Minneapolis studied 230 victims of CO poisoning and found that myocardial injury is common in moderate to severe CO poisoning. In this study, two groups of patients were identified. The first group of patients was younger (average age 43 years) and had few cardiac risk factors. The second group was older (average age 64 years) and had more cardiac risk factors. Based upon this study, the following factors were predictors of myocardial injury:

- Male gender;
- Hypertension; and
- Altered mental status (a Glasgow Coma Score of less than 14)

No other risk factors were found.

Although most studies address immediate deaths from CO poisoning, other research is starting to demonstrate that long-term mortality may be related to both acute and chronic CO exposure. In a Swedish study, researchers measured COHb levels in men who never smoked and correlated these figures with subsequent mortality. The COHb levels in the never-smokers ranged from 0.13–5.47 percent. Never-smokers with COHb levels in the top quartile (25 percent) had a significantly higher incidence of cardiac events and deaths compared to those in the lowest quartile. The researchers concluded the incidence of cardiovascular disease and death in nonsmokers is related to COHb levels and suggested that measurement of COHb levels be a part of risk screening for cardiovascular disease.

In a prospective study of 230 victims of moderate to severe CO poisoning, 85 patients (37 percent) had an associated myocardial injury. These patients were followed for an average of 7.6 years after their initial poisoning. Interestingly, of the 85 who had myocardial injury, 32 patients (38 percent) eventually died. In contrast, only 22 (15 percent) of the 145 patients who did not sustain myocardial injury eventually died. According to the researchers, “While the precise mechanism for the increase in mortality is not clear, cardiovascular death was much more common (44 percent vs. 18 percent) among patients who initially sustained myocardial injury.”

**CO & THE BRAIN**

CO poisoning affects more than just the heart. Like the heart, the brain is highly dependent upon a constant supply of oxygen. CO poisoning can interrupt oxygen delivery to the brain, causing brain hypoxia,
which is later followed by oxidative stress. The detrimental effects of hypoxia and oxidative stress can be either temporary or permanent. Both acute and chronic neurological problems have been documented following CO poisoning regardless of whether the CO exposure is acute or chronic. It is believed that the mechanism of brain injuries is related to the production of free radicals—primarily nitric oxide (NO). NO is normally found in the body, causes vasodilation and can injure or kill cells through oxidative stress. NO levels increase with CO exposure.6

Numerous neurological findings have been reported following CO exposure, primarily affective (mood) and cognitive (thought) in nature. In a study of 127 CO-poisoned patients, researchers found that depression and anxiety were common; in fact, these conditions were present in 45 percent of the patients at 6 weeks and in 44 percent of the patients at 6 months. Depression and anxiety were higher initially in patients whose CO exposure was due to a suicide attempt. However, at 12 months post-exposure there was no difference in anxiety and depression levels between those exposed to CO accidentally or as a suicide attempt.7

A phenomenon called delayed neurologic syndrome (DNS) has been identified as a complication of acute and chronic CO poisoning.8 In DNS, recovery from the initial CO poisoning is seemingly apparent, only to have the victim develop behavioral and neurological deterioration anywhere from 2–40 days later. The true prevalence of DNS...
is uncertain, with estimates ranging from 1–47 percent of all CO poisoning victims. Patients who have more symptoms initially appear more apt to develop DNS. In addition, DNS is more common when there is a loss of consciousness in the acute poisoning.

The signs and symptoms of DNS are listed in the sidebar on p. 71. Other neurologic complications, such as Parkinsonism (findings that mimic Parkinson’s disease), have also been reported with DNS.

WHAT DOES THIS MEAN FOR FIREFIGHTERS?

Most line-of-duty firefighter deaths result from cardiovascular disease. Certainly, cardiovascular risk factors such as smoking, obesity, lack of exercise and dietary indiscretion are similar in firefighters to that of the population as a whole—and firefighters must work to avoid these factors.

However, these studies indicate that additional factors might be increasing firefighters’ risks for heart disease above the normal population. Thus far, studies of duty-related firefighter deaths have not identified employment as a particular risk factor for the development of cardiovascular disease and ultimately death. However, these studies have not looked at the effects of CO poisoning (both acute and chronic) as a confounding variable in firefighter duty-related deaths.

In addition to the tissue damage to the heart brought on by hypoxia, new knowledge about CO poisoning suggests that chronic exposure to CO-induced free radicals may, in fact, be a major occupational risk factor for cardiovascular disease and early death. Furthermore, exposure to cyanide and other toxic gases may compound the effects of CO in firefighters.

The bottom line: Firefighters must minimize, as much as possible, their exposure to CO. In addition, more research is needed to determine whether a true link exists between occupational CO exposure and the development of cardiovascular and neurologic disorders among firefighters.

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REFERENCES


