Chapter 24

CARBON MONOXIDE

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INTRODUCTION

NONMILITARY SOURCES OF EXPOSURE

MILITARY EXPOSURES

PATHOPHYSIOLOGY

CLINICAL PRESENTATION

DIAGNOSIS

TREATMENT

HEALTH STANDARDS AND GUIDELINES

MEDICAL SURVEILLANCE

SUMMARY

ATTACHMENT: EXAMPLE OF MODELING PERCENT OF CARBOXYHEMOGLOBIN WITH TIME USING MICROSOFT EXCEL

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INTRODUCTION

This chapter is being published as an update to Chapter 11, Carbon Monoxide, in the previous edition of this textbook.[1] Much of the chapter has been revised to reflect carbon monoxide (CO) exposure in the military occupational setting and health regulations for service members. CO is a colorless, odorless, tasteless, and nonirritating gas that forms during the incomplete combustion of carbon-containing materials.[2,3] CO has high inherent toxicity and extensive exposure potential; it is the most significant and widespread toxic gas in the workplace.[4,5]

The Centers for Disease Control and Prevention (CDC) estimates that CO poisoning accounts for 15,000 visits to the emergency department and 500 deaths each year.[6] Over 68,000 CO exposures were reported to poison control centers between 2000 and 2009. Of these exposures, 45% were treated on site and 55% were treated in healthcare facilities.[6] Estimates of annual US incidents of confirmed CO poisoning has ranged from 1.4 to 2.3 cases per 100,000 people.[2,6] Recent natural disasters (eg, hurricanes, floods, and snowstorms) have led to CO poisoning events because people are unfamiliar with the risks of operating emergency generators and cooking indoors without proper ventilation.[6–8]

The CDC found that 12% of the CO exposures reported to poison control centers between 2000 and 2009 and 7% of unintentional CO poisoning deaths occurred in the workplace.[5,6] In 2000, CO poisoning was the top cause of poisonings and one of the ten most frequent occupational exposures in the US Army.[9]

NONMILITARY SOURCES OF EXPOSURE

Exposures in the General Population

Endogenous Sources

CO is endogenously produced in the body through breakdown of heme at a rate of approximately 10 mL/day[2,4] and elevates baseline carboxyhemoglobin (Hbco) levels by 0.3% to 0.7%.[4,10–12] Medical conditions that involve red blood cell breakdown (hemolytic anemia, polycythemia), blood transfusions, and sepsis all increase Hbco levels, though rarely reaching clinically concerning levels.[2,5] Hbco levels increase by 0.4% to 2.6% during pregnancy in nonsmoking mothers.[12]

Outdoor Air Pollution

Approximately 56% of all atmospheric CO emissions come from motor vehicles, while another 22% comes from construction equipment and boat engines. Metal processing, chemical manufacturing, residential wood burning, forest fires, volcanic eruptions, and lightning strikes also contribute to atmospheric CO levels.

Tobacco Smoke

In the 1920s, tobacco smoke was identified as a source of CO that produces an elevation in human Hbco levels.[4] Interestingly, smoke exhaled by a smoker contains only about 5% CO by volume, while smoke produced from burning cigarettes and other tobacco products produces 70% to 90% of the CO generated.[5] Designated smoking areas may have CO levels that exceed 11 ppm, while CO in nonsmoking areas is less than 2 ppm.[5]

Nonsmokers have Hbco levels between 0% and 2%, while smokers have Hbco levels that range from 4% to 20%, based on the number of cigarettes smoked per day. One-pack-a-day smokers can see elevations of 5% to 6% Hbco, two-pack-a-day smokers see a 7% to 9% rise in Hbco levels, and three-pack-a-day smokers can see rises in Hbco levels of 20%.[13,14] On average, Hbco levels rise 2.5% for each pack of cigarettes smoked per day.[15]

Cooking and Heating Appliances

Cooking and heating appliances that burn fuel and are unvented, inadequately vented, or improperly maintained have caused numerous CO poisonings and deaths.[7,13,16] Thousands of fatal and nonfatal human CO poisonings occur in the United States each year because of inadequately vented or malfunctioning water heaters, furnaces, and kerosene heaters. The use of grills for heating or cooking indoors and the misuse of gas stoves and ovens to heat houses contributes to the problem of CO poisonings.[7,13,16,17]

Industrial Exposures

CO exposure can occur in mines after blasting or when fires occur; in petroleum refineries near the catalytic cracking units; and in pulp mills near lime kilns and kraft recovery furnaces. In general industry, CO exposure occurs in boiler rooms and wherever internal combustion engines are used or repaired.[5] CO is used in industrial processes to reduce the oxygen content of iron and other metals, so gas and blast-furnace effluent can contain upwards of 25% to 30% CO.[5] In the chemical industry, CO is the feedstock for acrylate, aldehyde, ethylene, isocyanate, methanol, and phosgene production.[5]

Internal Combustion Engines

When internal combustion engines are run indoors without adequate ventilation, CO exposures occur.[17] Military vehicle crewmembers, law enforcement officers, taxi drivers, ambulance operators, and bus and truck drivers are all at risk of CO exposure. Mechanics in military motor pools, toll takers, garage attendants, and installation security guards are also routinely exposed to CO.[17,18] Workers inside buildings may be exposed to CO when vehicle exhaust enters through improperly placed air intakes.[5] The use of propane-powered forklifts indoors may expose the operator to CO.[5] Pickup truck campers can fill with CO and cause serious CO intoxication, particularly among children.[5] Also, numerous CO poisonings and deaths have occurred when residents slept in their homes with vehicles left running in attached garages.[6,17]

Mines

Blasting operations and fires during mining operations produced some of the worst CO occupational exposures in the late 19th and early 20th century,[5] and recent mine accidents have highlighted the dangers of CO in the workplace.

Structural Fires

CO remains a particular concern for firefighters who must enter enclosed, poorly ventilated spaces and encounter lethal CO concentrations during the "knockdown" (when materials are actively burning) and "overhaul" (searching for "hot spots") phases of firefighting.[5] The most common cause of death in fires is smoke inhalation, and CO is a major contributing factor for individuals who succumb.[18]

Methylene Chloride

Methylene chloride is an unusual industrial hazard that is metabolized into CO. Methylene chloride is widely used in industry for paint stripping and as an aerosol propellant and degreaser.[16]

Methylene chloride is highly volatile, lipid soluble, and readily absorbed, which makes the exposure potential high. An 8-hour exposure of 50 ppm will produce about 3% Hbco.[16,17]

MILITARY EXPOSURES

Exposure to CO has long been recognized as a potential hazard associated with incomplete combustion of carbon fuels, including during combat mining operations in World War I.[1,18] Military personnel face unique and deadly sources of significant CO exposure not found in the private sector. Military crewmembers of armored vehicles (eg, tanks and howitzers) and aircraft (eg, armed helicopters) involved in weapons firing can be exposed to CO.[1,18] Firing of missiles can also result in CO exposure. Exposures to CO may also occur in small arms indoor firing ranges and shoot houses, and exposures have also occurred indoors during the testing of howitzer tubes and during explosive detonation.[1,18]

Historical Exposures

World War I Combat Mining Operations

Defensive mining operations were conducted early in World War I where mines were used to guard important trenches or sectors of the line. After late 1915, mines were also used in offensive operations. This required personnel to dig more tunnels and use larger quantities of high-explosive munitions, resulting in more soldiers being poisoned by CO due to the detonations. Hydrogen, methane, and oxides of nitrogen were also generated, but CO was the most toxic.[1,3]

Mine detonations promoted forward movement of the troops. An exploded mine would create a crater 60 to 90 feet in diameter that could be occupied by infantry troops to advance and establish their position. The CO generated by detonations usually dissipated rapidly, and compressor engines were used to ventilate the area following detonation, but soldiers were frequently overcome as a result of incomplete detonation, subsequent gas collection, and entrapment.[1,3] Burning gas was frequently seen following an incomplete detonation as a blue flame that persisted for hours.[1,3] CO poisoning also occurred when the compressor engine was turned off in order to listen, when fuel or engine lubricants were not available, or when an unexpected breakdown occurred.[1,3] Because CO is odorless, colorless, and non-irritating, an exposed individual often failed to recognize the danger until it was too late, which also increased the incidence of exposures. CO was never used as an offensive chemical warfare agent.[1,3]

World War I Tank Warfare

In the early days of tank warfare, crews spent prolonged time inside their tanks and complained of headache and faintness, and they often lost consciousness. Their symptoms were aggravated when they fired the Hotchkiss and 6-pounder guns.[1,3] Symptoms were caused by the poor design of early tanks, which

permitted heat accumulation and exposure to toxic gases of combustion. CO exposure occurred because of leakage from exhaust lines that ran inside of the tank and back drafts from other tanks. In one 1918 incident, both tank drivers and the tank commander became unconscious. This demonstrated that improved tank ventilation was needed, so fresh outside air was infiltrated around gun ports and other openings to help dissipate the CO accumulation.[1,3]

World War II Tank Warfare

In World War II, CO was a problem for large numbers of soldiers who operated in armored fighting vehicles (AFVs). In response, scientists at the Armored Medical Research Laboratory at Fort Knox, Kentucky, developed a reliable, transportable infrared gas analyzer to measure CO in AFVs. High levels of CO were detected in the M3A4 tank after bursts were fired.[19] After five rounds were fired from the 75-mm gun, CO levels increased rapidly to 0.718% (7,180 ppm) in less than a minute, then decreased back to baseline in 4 minutes. The CO levels generated when the 37-mm was fired were substantially lower than when the 75-mm gun was fired.[1]

CO exposures were measured in a medium tank, the M4A1,[1,20] and in a tank-towing vehicle, the M32B1, on a flat surface and at a 4% grade, for each crew position. Hazardous levels of CO were found in the M4A1 tank and the M32B1 towing vehicle, both of which had engine exhausts directed rearward.[20] Riders in the tank were exposed to levels of CO in excess of 0.2% (2,000 ppm), and the exposures were affected by changes in wind direction, surface grade, monitoring position, and whether the towing cable or towing bar was used. The M4A1 tank and the M32B1 towing vehicle were retrofitted with an exhaust deflector shield to direct exhaust toward the ground.[1,20]

Modern Era Exposures

During World War II, only 3% of soldiers in the US Army used AFVs. In contrast, nearly all US soldiers used armored vehicles, such as the mine-resistant ambush protected vehicle, in operations in Iraq and Afghanistan. Furthermore, electronic networks and systems on the battlefield have significantly increased the use of internal combustion generators for electricity production. Today's service members are more likely than those in earlier conflicts to be exposed to CO, and at significantly higher levels if control measures fail. Even more effective ventilation systems and cleaner burning propellants have not reduced the risk of CO exposure in today's AFVs.[1]

M1E1 Tank

In 1984, an exposure to CO was reported during the operational test of the M1E1 tank. A firing exercise was run in accordance with a test plan that called for the hatches to be closed, the primary nuclear, biological, and chemical (NBC) system to be off, and backup system (M13A1) to be on. Also, the breech was open, and the engine was off during most of the exercise, although it was started periodically to recharge the batteries. The crew was dressed in mission-oriented protective posture (MOPP) gear. Breathing air was supplied to the protective masks through the M13A1 gas particulate filter unit, although the masks were not worn for most of the exercise. The tank fired 26 main gun and approximately 100 machine gun rounds. At the end of the exercise, the loader slumped forward in his seat. A short time later, the tank commander aroused the loader and assisted him out of the turret. The tank commander was also dizzy and lay down on the tank. The loader and tank commander were taken to the hospital and later admitted.[1,21] During the medical evaluation of the crew, Hbco levels were obtained. The loader had a level of 33% Hbco and the tank commander had a level of 27.8%. It should be noted that these Hbco measurements underestimate the true Hbco levels because of the short biological half-life of Hbco in the blood.

In March 1984, the US Army Human Engineering Laboratory Liaison Office, at Fort Hood, Texas, made the following recommendations to improve the M1E1 tank:[21]

- Reroute the air intake for the M13A1 gas particulate filter unit away from the turret area.
- Advise individuals at all test sites about the CO hazard when the main gun and/or coaxial machine gun is fired with the hatches closed and NBC system off.
- Advise personnel that the M13A1 gas particulate filter provides no CO protection.
- Include a warning in the operators manual about the CO hazard when the weapon is fired and the inability of the M13A1 system to remove the CO, and describe the ventilation steps needed.[21]

Bradley Fighting Vehicle

The Bradley fighting vehicle (BFV) is a tracked, light-armored vehicle. The BFV has an M242 turret-mounted 25-mm chain gun and an M240 7.62-mm coaxial machine gun, and a TOW (tube-launched, optically tracked, wire-guided) anti-tank missile launcher.

In 1980 the US Army Environmental Hygiene Agency conducted toxic gases testing on the BFV.[22] Real-time monitoring was done inside the vehicle during a worst-case firing scenario to determine peak and total CO exposure concentrations.

Two hundred rounds of 25-mm and 75 rounds of 7.62-mm ammunition were fired over 20- and 60-minute periods. Peak exposures in the turret were measured at above 600 ppm; the average exposure concentration was 190 ppm. The total exposure concentration was 11,400 ppm-minutes based on the cumulative exposure being the product of the average airborne concentration and total exposure time. The firing-exposure scenario was repeated, and peak measures in the turret were found to exceed 800 ppm; total exposure measured 24,730 ppm-minutes. In the driver's compartment, CO peaks measured in excess of 400 ppm, and total exposure concentrations were 10,600 ppm-minutes. In the crew compartment, peak exposures were detected above 400 ppm, and total exposure concentrations of approximately 8,200 ppm-minutes were measured.[22]

Total concentration exposures in the BFV exceeded the acceptable limit of 6,000 ppm-minutes established in Military Standard 800.[23] The study concluded that under firing conditions, exposures could result in Hbco levels of about 15% in 15 minutes. These exposure levels were considered a health risk that caused significant signs and symptoms of impairment of combat effectiveness.[22]

In a study of the BFV performed in 1984, real-time CO measurements were obtained using an infrared CO analyzer.[24] The study found that firing conditions in the BFV affected CO concentrations. The type of weapon fired, the position of the hatches (open or closed), the crew position, and the position of the turret with respect to the hull all affected the measured CO levels. Wind speed also affected firing conditions. Closed-hatch firing was not permitted at wind speeds above 10 mph, and open-hatch firing was not permitted at wind speeds above 5 mph. Hull fans were turned off, and gas particulate filter units were turned on during firing of both the 7.62-mm and 25-mm rounds.[24]

The 1984 study concluded that firing BFV weapons generated Hbco levels above 5%, and in three conditions, Hbco levels ranged between 11.0% and 13.4%. The maximum peak concentration, 1,462 ppm, was measured at the driver position, and levels of 1,087 ppm and 1,200 ppm were detected in the crew compartment.[24,25] The M13A1 filter unit had no ability to remove CO and contributed to high levels of CO exposure within the driver's and crew's compartments.[24]

M109 155-mm Self-Propelled Howitzer

The M109 self-propelled howitzer is an armored and air-transportable field artillery weapons system, generally operated by a crew of four. It was designed to provide support to armored and mechanized infantry units. The system has been improved several times, including the addition of NBC protection for the crew and increased projectile range with rocket-assisted projectiles. The M109 carries conventional rounds and two oversized projectiles on board.[26] The main armament is a modified 155-mm M185 cannon assembly (the M284) and an M178 gun mount. The modified muzzle break deflects propellant gases back along the gun tube.

A 1988 Environmental Hygiene Agency health hazard assessment found that many variables affect the measured level of propellant combustion gases generated by the M109 howitzer.[26] Tube-firing elevation, wind speed, wind direction, hatch configuration, ventilator mode, propellant type and quantity, system failure, fire rate, and industrial hygiene sampling practices all influenced the results. The bore evacuator is a pressure-responsive tube evacuation system designed to promote the movement of post-fire combustion gases from the breech toward the muzzle. Compromised bore evacuator function and wind direction are critical variables associated with exposure concentrations after firing. Exposures were highest when the vehicle fired with the hatches closed. The crew compartment had a slight negative pressure, which caused combustion gases to enter the crew compartment from the breech when it was opened. A head wind significantly increased exposure to combustion gases, and reconfiguring the muzzle break did not reduce the exposure.[26]

The M109A6 Paladin weapon system was used from 1993 to 2000. Over its lifespan, the system received a new gun assembly, bore evacuator, and muzzle brake, along with improvements to the breach and recoil system. The Paladin had a range of 20 miles with a rocket-assisted projectile, and fired four rounds a minute. The Paladin's advanced bore evacuator was much more efficient than the standard bore evacuator, which reduced CO concentrations in the crew compartment.[27]

On December 9, 1999, a CO exposure occurred at Fort Carson, Colorado, involving the crew of an M109A6 Paladin that resulted in the gunner and loader experiencing symptoms of CO poisoning. The Paladin crew took part in a routine training exercise in which the main gun was fired with hatches closed to simulate operating in an NBC environment.[27] The intake ventilation system was not used while the rounds were fired, and the exhaust ventilation system was not used afterward.[27] Following the exercise, the gunner and

loader who experienced CO poisoning symptoms were air-evacuated to a hospital with a hyperbaric chamber for treatment.[27] Their Hbco blood levels were 29% and 16%, respectively, upon arrival at the hospital. Their symptoms resolved after 2 hours of hyperbaric oxygen treatment.[27]

On January 13, 2000, another CO poisoning event occurred at Fort Carson during a training exercise with an M109A6 Paladin, when three solders (section chief, gunner, and loader) experienced symptoms.[28] During the exercise, the crew was engaged in direct fire, and the rear hatch was open, the tank commander's hatch was opened briefly after the firing of at least some of the rounds, and the driver's hatch was closed.[28] The intake ventilation system was used during firing, but the exhaust ventilation system was not used at the end of mission.[28] The section chief had the highest Hbco level (20%). The chief and another crew member were treated in a hyperbaric chamber.[28] The other two crewmembers had much lower Hbco levels (8% each).[28]

Following the January 2000 incident, the Fort Carson 223rd Preventive Medicine Medical Detachment conducted four different tests on three different M109A6 Paladins at the National Training Center (NTC) on Fort Irwin, California, from February 11 to 21, 2000.[29] The testing did not detect high concentrations of CO, nor were Hbco levels found in the blood of Fort Carson crews. Hbco levels were well below the American Conference of Governmental Industrial Hygienists (ACGIH) biological exposure index (BEI) Hbco level of 3.5%.[29]

The 223rd did a separate test in February 2000 of nonsmoking crewmembers exposed to CO concentrations ranging from 50 to 83 ppm over 275 minutes that resulted in Hbco levels between 6.4% and 11.4%, assuming an initial Hbco level of 1%. In the same Paladin, 1 week later, average CO exposures over a 155-minute exposure interval were significantly less, ranging from 18 to 22 ppm, and predicted Hbco levels were much less than the ACGIH BEI of 3.5% Hbco.[29] Differences in exposure concentrations between the two tests may possibly be explained by any of the following: differences in the amount of rounds fired and type of powder used; weapons fire dynamics; or wind velocity and direction. It should be noted that the Hbco levels estimated above are likely to be underestimates because CO measurements did not adequately capture the large fluctuations in CO concentrations expected during weapons firing.[29]

The US Army Center for Health Promotion and Preventive Medicine (USACHPPM) was contacted to help review Fort Carson's local board actions and opinions, confirm CO measurements, determine why CO levels were elevated, and recommend corrective actions.[30] USACHPPM provided several explanations as to why the measured Hbco levels did not mirror the signs and symptoms experienced by the crew members.[27] First, the measured Hbco levels would have been lower than the actual Hbco level at the completion of firing, given the biological half-life of Hbco and delay in obtaining blood samples for an hour to several hours after the exposure. Second, blood was drawn from the patients who had been given oxygen therapy. Altitude may have also contributed somewhat to the effects of CO poisoning, but the Paladin crews had been at the altitude for well over 4 weeks, and should have been physiologically acclimatized. So even though altitude increased the crew risk of hypoxia, it was not considered a significant explanation for the observed signs and symptoms. Also, some crewmembers were smokers, which confounded the investigation somewhat. Other noxious gases (eg, nitric oxide) were ruled out as a significant confounder by USACHPPM toxic gas testing; measured nitric oxide concentrations were low.

Testing was done on March 10, 2000, to re-create the situation. Crewmembers wore supplied-air respirators. Paramedics were present outside the Paladin during the tests.[27] One Paladin was tested in two configurations: (1) with the rear and side hatch open and (2) with the side hatch closed and the rear hatch open. In both instances, the tank commander's and driver's hatches were closed. The NBC scenario with all hatches closed was not re-created, but measured CO concentrations would likely have been much higher than the concentrations found during the tests with open hatches.

When the January 2000 scenario was re-created (rear hatch open, side hatch closed, tank commander's hatch closed, driver's hatch closed), CO concentrations were found to be substantially elevated inside the crew compartment.[27] The CO dosimeters worn by the crew did not record the actual concentrations because they did not read above 1,000 ppm, which was exceeded for about 10 minutes. The average concentrations recorded by the dosimeters for the crewmembers were about 500 ppm for 30 minutes, with 15-minute average concentrations as high as 851 ppm. The 15-minute average concentrations are more representative of the concentrations while firing rounds than the average concentration, which includes lead time and exhaust period at mission end.[27]

The CO concentrations measured during the test in the crew compartment underestimate the actual concentrations. Concentrations may have been as much as double the measured concentrations. Given this uncertainty and the varying CO concentrations measured at different positions in the crew compartment, Hbco levels of about 15% to 30% were predicted over the 30-minute interval for crewmembers.

After the first test, an investigation revealed that the rear bore evacuator seal/O-ring was missing, which reduced the efficiency of the bore evacuator and contributed to the increase in CO inside the crew compartment. Once discovered, the missing bore evacuator seal/O-ring was replaced.[27] This was felt to be the main reason for the elevated CO levels inside the crew compartment, though headwinds and the intake ventilation may have also contributed to the problem.

Testing was repeated once the missing rear bore evacuator seal/O-ring was replaced.[27] This time, the average CO concentrations inside the crew compartment ranged from 4 ppm to 10 ppm, which is well below the level of a health hazard. The highest 15-minute average concentrations ranged from 5 ppm to 12 ppm, which was thought to be more representative of the concentrations seen when rounds were fired. Predicted Hbco levels during the 27 minutes were well below the ACGIH BEI of 3.5%.

USACHPPM made a number of recommendations to avoid repeated CO exposures with Paladins, including the following[27]:

1. Take any Paladins missing the bore evacuator seals/O-rings required by manufacturer out of service, and require each vehicle to carry spare seal/O-ring sets.
2. Ensure preventive maintenance is performed on all Paladins, including a bore evacuator system check.
3. Avoid firing in the presence of headwinds that increase CO exposures in crew compartments.
4. Ensure that the intake ventilation is on during round firing and for 2 minutes after firing; after that the exhaust ventilation should be on for 10 minutes.
5. Request USACHPPM support through Forces Command to study soldier occupational exposures.
6. Have the Paladin program manager examine whether there is sufficient intake ventilation make-up air for the bore evacuator to work efficiently with hatches closed.
7. Consult with Army NBC safety and preventive medicine personnel about whether the ventilation intake should be turned on during firing in an NBC environment (or when training for such an environment), when all hatches are closed.
8. Because CO is readily introduced through the ventilation intake and climate conditioning system intake, consider moving these systems to the rear of the vehicle.
9. Install a CO monitor with an audible and visual alarm in every Paladin that activates at concentrations equivalent to an Hbco level of less than or equal to 10%.

Although 10% Hbco was adopted as an operational limit by the Army,[31] values less than 15% Hbco do not affect performance but may cause a mild headache.[32] Values exceeding 25% Hbco are considered dangerous and require aggressive treatment in addition to removal from exposure.[32]

Military Aviation

Unsafe levels of CO were generated in military aircraft by early piston-driven engines, which caused the deaths of many pilots. Later, CO exposures from engines were eliminated, but high levels of toxic gases were generated when weapons systems were fired aboard aircraft. In 1988, CO levels were tested when an M134 mini-gun mounted in the UH-60A Black Hawk helicopter was fired. In the tests, 2,700 rounds were fired, and the average CO concentration was 79.4 ppm during the 4 minutes of firing. The maximum predicted Hbco level was 4.90%, so firing restrictions were not recommended.

More recently, CO poisonings have been reported in the fixed-wing fighter community in the US Air Force and Navy. F-22 Raptors were grounded in 2011 for several months due to suspected CO poisoning at Elmendorf Air Force Base, Alaska, when planes were started inside hangars.[33] Air Force investigators thought exhaust gases containing CO accumulated inside the hangar and were subsequently taken back up into the engines and entered the On-Board Oxygen Generation System (OBOGS). The Navy has reported similar problems with the F/A-18 Hornet OBOGS,[34] which prompted flight surgeons to look for evidence of hypoxia in pilots in planes equipped with the system.

Other Recent Exposure Incidents

Fort Hood, Texas

In early November 1997, two active duty soldiers, a married couple, woke up at 0200 in their off-base apartment and felt nauseated and dizzy.[35] They felt better when they went outside, but decided to go to the local emergency room for evaluation. The soldiers did not smoke, and their Hbco levels were 31.6% and 28.3%, respectively for the husband and wife. They were diagnosed with CO poisoning, given 100% oxygen with a non-rebreather mask, and sent for

hyperbaric oxygen therapy (HBOT). Their residence did not have a CO detector, and the furnace that was turned on the night before had malfunctioned, causing high levels of CO.

Fort Campbell, Kentucky

In November 1997, the wife of a soldier woke up with a headache, fatigue, and difficulty standing.[35] She went to the emergency room and was tested for CO poisoning. She was a nonsmoker and her Hbco level was 33%. She was treated with 100% oxygen and sent home. She lived off-post in a trailer equipped with a CO detector, which had gone off the day before the incident. The trailer owner told her the CO detector was installed too close to the furnace, so she moved it. The alarm continued to go off but she ignored it the day of the incident. The day after the incident, an inspector checked the furnace and discovered it had a clogged flue.[35]

Olsbrucken, Germany

On November 7, 2000, two children of an Air Force retiree living in Germany awoke at 0330 disoriented and vomiting, with diarrhea and abdominal cramps.[36] Both the father and mother were also affected, with sudden onset of headache, vomiting, and nausea. The German ambulance put the family on oxygen and took them to the Landstuhl Regional Medical Center emergency department. All family members improved after leaving the house. Their history and clinical presentations and improvement after leaving the house were highly suggestive of CO poisoning.

The emergency room staff found that the concentration of Hbco in the family's blood ranged from 19.6% to 26.5%. The family members were all placed on 100% oxygen and observed in the emergency department for several hours, and the emergency room physician reported the CO poisoning to the US Army Public Health Center–Europe.[36] After laboratory results confirmed CO poisoning, industrial hygienists interviewed the family and surveyed their home, finding CO concentrations of 200 ppm at the house's entrance. Based on the reading, the industrial hygienists alerted the German fire department, who sent personnel equipped with a self-contained breathing apparatus to survey the inside of the house. They found the CO concentration in the boiler room was over 500 ppm.[36] It was later determined that ash in the chimney caused the buildup of CO and prevented proper ventilation of the heating system.

Fort Irwin National Training Center, California

On January 12, 2001, two soldiers died at Fort Irwin NTC from CO poisoning and anoxia after purchasing a Coleman Powermate 15,000-BTU unvented propane gas heater to warm up their tent.[37] Testing confirmed that the tent was airtight,[37] that oxygen had been depleted by the soldiers' breathing, and that CO had accumulated from the unvented heater in the tent.[37] Because of these deaths, safety bulletins, alerts, and guidelines have been issued to prevent a recurrence.[38–43]

Poisoning Events, 1998–2008

The Armed Forces Health Surveillance Center recorded 227 CO poisonings over a 10-year period from July 1998 to June 2008, which averaged 23 poisonings per year. These numbers reflect a decrease from 1,000 CO poisoning in the previous 10-year reporting period in the US military.[44] However, the more recent case definition limited cases to those involving hospitalizations or lost duty time, while prior reporting included cases that were not clinically significant. Among the 227 reported cases, 53% required hospitalization, and 9 cases (4%) were fatal.[9] CO poisonings peaked during winter months. Most cases were in the 20 to 29 age group (66%); 56% involved US Army personnel; and two installations, Fort Hood, Texas, and Fort Lewis, Washington, each accounted for 10% of the identified cases.[9]

PATHOPHYSIOLOGY

In the body, CO is produced by heme destruction and excreted by exhalation.[10] CO impairs the oxygen-carrying capacity of hemoglobin in two ways. First, CO competitively binds to hemoglobin with an affinity 200 to 250 times greater than that of oxygen. CO also causes changes in the conformation of the hemoglobin molecule, which shifts the hemoglobin dissociation curve to the left and decreases the amount of oxygen released.[15] In addition, CO interferes with cellular respiration by binding to cytochrome oxidase, which causes neurological and myocardial injury.[44]

Until recently, the primary mechanism of CO toxicity was thought to be the formation of Hbco and subsequent tissue hypoxia. However, the results of recent scientific studies have challenged this principle.[15,45] The clinical presentation of a patient with CO poisoning does not always correlate with blood Hbco level, nor does clinical improvement correlate with the clearance

of Hbco.[2,15,45] Clinical CO toxicity is now thought to result from a combination of hypoxia and inflammatory mechanisms. These mechanisms include the CO's binding to intracellular proteins such as myoglobin and cytochrome a, lipid peroxidation affecting myelin proteins and apoptosis in neurons, increased nitrous oxide production and oxidative stress within the vasculature, and increased amino acid levels.[45,46]

The local and systemic inflammation combined with anoxia leads to neurologic and cardiac injury. Recent studies suggest that myocardial injury following moderate to severe CO poisoning increases mortality. In a prospective study of 230 CO poisoning cases, 37% had acute myocardial injury, and an additional 38% died within 7.6 years of follow-up. Among the relatively young, healthy cohort, the observed death rate was 300% higher than the expected death rate.[43] Studies of CO poisoning cases note that a third of patients have persistent headaches and memory problems for 4 weeks following the exposure, and half had neuropsychological symptoms up to 6 weeks later.[45]

CLINICAL PRESENTATION

The signs and symptoms of CO poisoning were described in 1923.[47] Table 24-1 describes acute health effects in healthy adults, based on information in *Patty's Toxicology*[4] and other sources.[5,17,18,48,49] CO targets metabolically active tissue that has a good supply of oxygen-rich blood.[18,50–53]

Signs and Symptoms

Headache, dizziness, and nausea are the most common symptoms. If CO poisoning continues and Hbco levels rise, additional symptoms may include fatigue, dyspnea, chest and abdominal pain, impaired judgment and memory, visual disturbances, drowsiness, and agitation. Common clinical signs include vomiting, ataxia, confusion, syncope, coma, seizures, tachypnea, and cardiac dysrhythmias. The symptoms and degree of impairment with CO exposure are worse with higher concentrations and prolonged exposure. The symptoms of CO exposure are exacerbated by muscular activity that increases oxygen demand, while individuals at rest may experience no symptoms before becoming unconscious.[46]

Providers must have a suspicion about CO poisoning because the symptoms are nonspecific, and CO poisoning is commonly confused with other conditions such as influenza or gastroenteritis.[2,54] The classic sign of "cherry-red" skin or lip color is actually uncommon, and it is a late finding in lethal or near-lethal CO poisoning cases.[2,13,15]

The medical history for CO poisoning cases usually involves an exposure from gas-burning appliances, fireplaces, or gasoline engines being operated in poorly ventilated areas. People who have survived a fire and those who work in enclosed spaces may have elevated Hbco levels. Also, mechanics and others who work with paint strippers and solvent degreasers containing methylene chloride may have delayed onset of symptoms because the methylene chloride is metabolized to CO and can cause a rise in Hbco levels.[16,54]

Severe toxicity occurs through metabolic acidosis due to lactate formation from hypoxia, as well as renal failure due to rhabdomyolysis. Bullae may form from direct toxic effects of CO or from pressure necrosis. CO poisoning affects the basal ganglia in the brain and causes tremor, slowed reaction time, decreased manual dexterity, poor eye-hand coordination, and inability to process complex movements.[50] The ocular signs of CO poisoning occur early and persist for some time; they include retinal vessel congestion and optic disc hyperemia. In 1921 Wilmer noted that amblyopia and complete blindness are common sequelae of CO poisoning.[55]

In combat, soldiers who experienced CO poisoning during World War I had anoxia and first experienced extremity weakness, followed by giddiness, confusion, breathlessness, and palpitations as the CO concentrations increased. At higher CO concentrations, mental confusion caused soldiers to appear drunk. Mental confusion and extremity weakness reduced both the desire and ability to escape, and consequently many individuals went into a coma and died.[3]

In contrast to acute CO poisoning cases at high CO concentrations, individuals with mild cases of CO poisoning develop symptoms more slowly and experience nonspecific clinical signs of headache and nausea, which is often confused with mountain sickness.[3] Mechanics, heavy equipment drivers, armored vehicle crews, and aviators who are chronically exposed to low levels of CO have not developed health problems, but providers must remain vigilant.[54]

Central Nervous System Effects

Individuals with CO poisoning exhibit signs and symptoms of hypoxia, but these symptoms can be caused by any neurological condition. People with acute CO intoxication have been reported to have signs of multiple sclerosis, parkinsonism, bipolar disorder, schizophrenia, and conversion disorder.[50] The neurologic sequelae of CO poisoning, which can occur immediately or be delayed, include headache, myalgia, weakness,

TABLE 24-1

ACUTE HEALTH EFFECTS OF CARBON MONOXIDE EXPOSURE*

Blood Saturation Hbco (%)	Range Notes	Response of Healthy Adults	Response of Patients with Severe Coronary Artery Disease
0.4–0.7, increasing up to 2.6 during pregnancy	Normal range due to endogenous CO production	No known detrimental effect	No known detrimental effect
1–2	Background levels in urban population due to combination of endogenous CO and environmental exposure	No known detrimental effect	No known detrimental effect
2–5	Range found in commuters on urban highways	Possible slight decrements in psychomotor function (eg, reduced video game performance)	Less exertion required to induce chest pain
5–10	Range found in cigarette smokers	• Compensatory increase in CNS and coronary blood flow • Slight decrease in capacity for strenuous exercise • Prolonged levels may affect the performance of tasks requiring a high degree of vigilance (eg, flying an aircraft or monitoring a control panel)	Greater frequency and complexity of ventricular ectopic beats during exercise
10–20 Note: triservice laboratory diagnosis for CO poisoning: >10% in nonsmokers, >15% in smokers†	Range found in cigar smokers	Slight headache, fatigue, lightheadedness	Exertion may precipitate myocardial infarction
20–30		Moderate headache, nausea, fine manual dexterity impaired, visual evoked response abnormal, flushing and tachycardia	No difference
30–40		Severe headache, nausea and vomiting, hypotension and ataxia	No difference
40–50		Syncope	No difference
50–65		Coma and convulsions	No difference
>65–70		Lethal if not treated	No difference

*Exposure to CO at high concentrations (>50,000 ppm or 5% CO) can result in a fatal cardiac arrhythmia and death before the Hbco is significantly elevated.

†Defense Health Agency. *Revised Armed Forces Reportable Medical Events Guidelines and Case Definitions.* Washington, DC: DHA: June 30; 2017. Memorandum. https://health.mil/Policies/2017/07/17/Revised-Armed-Forces-Reportable-Medical-Events-Guidelines-and-Case-Definitions. Accessed September 12, 2017.

CO: carbon monoxide

CNS: central nervous system

Hbco: carboxyhemoglobin

Data sources: (1) Apfelbach G. Carbon monoxide poisoning. In: Kober G, Hayhurst E, eds. *Industrial Health.* Philadelphia, PA: P. Blakiston's Son & Co; 1924. (2) Goldstein M. Carbon monoxide poisoning. *J Emerg Nurs.* 2007;34(6):538-542. (3) American Conference of Governmental Industrial Hygienists. *Documentation of the Threshold Limit Values and Biological Exposure Indices.* Cincinnati, OH: ACGIH; 2001. (4) National Research Council, Committee on Toxicology. *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants*. Vol 4. Washington, DC: National Academy Press; 2007. (5) American Industrial Hygiene Association. *Carbon Monoxide Documentation for Emergency Response Planning Guidelines.* Fairfax, VA: AIHA; 1999.

memory loss, paralysis, cortical blindness, and peripheral neuropathy and convulsions.[56,57] These signs usually resolve within days, but can persist for months to years.[47] Permanent memory loss and personality changes occur in between 0.3% and 10% of CO poisoning cases.[58]

Acute CO poisoning can cause neurobehavioral effects including compromised dark adaptation and impaired visual tracking, which can reduce performance in aircraft handling and target acquisition. Military-specific Hbco limits and equipment-design specifications[31] were developed because visual acuity becomes impaired at Hbco levels between 3% and 5%.[52]

Cardiovascular Effects

The heart is highly sensitive to CO poisoning because it depends almost exclusively on aerobic metabolism. Normally the heart consumes pyruvate and lactate in metabolic oxidation, but this does not occur when Hbco levels rise above 8.7%.[48] Individuals with cardiopulmonary conditions including coronary artery disease, anemia, and lung disease are more likely to have problems with CO-induced tissue hypoxia.[50] At low Hbco levels, people with ischemia have experienced angina. Recent studies suggest that young, otherwise healthy individuals who experience moderate to severe CO poisoning develop myocardial injury and are at increased risk of mortality over time.[44,59]

The crews of armored vehicles are routinely exposed to CO levels that induce ischemic responses in animals and humans.[60,61] It is possible that soldiers with early cardiovascular disease could suffer an adverse myocardial event when the vehicle operates and generates high concentrations of CO. A study conducted by the US Army Biomedical Research and Development Laboratory showed that individuals with 10% to 20% Hbco levels who performed work at 35% of the maximal work rate demonstrated only minimal increases in heart rate after working a period of 3.5 hours; however, individuals with Hbco levels of 40% to 45% had physical work capacity that was dramatically compromised.[18] CO poisoning decreases maximum work capacity (as defined by Vo_2 max) when Hbco levels rise above 5%, and both fatigue and angina develop sooner with CO exposure.[53]

Chronic Effects

Most patients recover completely after being removed from CO exposure, but some patients develop central nervous system (CNS) or cardiovascular sequelae days to weeks after poisoning.[50,57] Prior to 2001, the prognosis for recovery was thought to be related to the degree of asphyxia, and men exposed to CO in mines were thought to develop a permanent weakness of the heart.[4] Vision loss, speech problems, and CNS defects were also reported. However, recent reports show that only long-term mortality due to myocardial injury is correlated with Hbco levels.[45,51]

Magnetic resonance imaging studies taken several days following the event (but not earlier) show low-density lesions in the area of the globus pallidus that are associated with CO-induced encephalopathy, which occurs in 50% of severe CO poisoning cases.[57] Lesions in the basal ganglia gray matter may resolve, but lesions in the white matter that are associated with neuropathy are usually permanent.[50]

CO-induced neuropsychiatric illness may occur up to 6 weeks after the event in between 2% and 30% of CO poisoning cases.[62] Delayed sequelae occur in both young and old, but elderly patients are more at risk. Clinical signs of delayed sequelae may include urinary or fecal incontinence, weakness, gait disturbances, tremor, mutism, speech abnormalities, and mental deterioration. Complete recovery occurs in about 75% of individuals within a year.[62]

Autopsy Findings

Anatomic autopsy findings are helpful in determining cause of death. The skin color of a CO-poisoned person differs from the skin color of other deceased individuals.[48] The face may be bright red and there may be rose-red spots on the face, neck, breast, and limbs. The color of the skin between the red areas is likely to be discolored and may be cyanotic. Blood ranges in color from bright red to black. Ecchymoses, effusions, or hemorrhages may occur even with no change in blood coagulation. The respiratory tract is generally unchanged, although mucus or digestive contents have been found in the upper respiratory tract. The brain swells and the intraventricular fluids become blood tinged. Hemorrhagic lesions ranging in size from microscopic to "apple sized" have been seen.[47] Other common pathologic findings of CO poisoning include bronchopneumonia, blood vessel deterioration, necrosis in the lenticular nucleus, thrombosis, and encephalitis.[4]

DIAGNOSIS

The differential diagnosis for CO poisoning includes influenza, gastroenteritis, food poisoning, cerebrovascular events, myocardial infarction, asphyxia, delayed parkinsonism, ethanol intoxication, sedative-hypnotic overdose, hypothermia, and myxedema coma.[13,16,54] Headache and dizziness are common symptoms of

many of the conditions in the differential diagnosis, which increases the likelihood of misdiagnosis, especially with winter visits to the emergency department.[46] Mental status changes and ataxia associated with ethanol intoxication or medication overdose may confuse the provider's neurological assessment. Other gas exposures may cause the altered mental status often seen with CO poisoning. Physical asphyxiants (eg, natural gas, carbon dioxide, acetylene, helium) may lead to temporary unconsciousness. Irritant gases (eg, formaldehyde, hydrogen chloride, nitrogen dioxide) are more likely to be associated with coughing and bronchospasm due to pulmonary and mucous membrane irritation. Cellular asphyxiants that bind cytochrome oxidase (eg, hydrogen cyanide, hydrogen sulfide) cause more abrupt loss of consciousness than does CO.[54] The clinician should consider coexisting cyanide toxicity if the patient suffered from smoke inhalation.[2]

Because the signs and symptoms of CO poisoning are variable depending on the severity and duration of exposure, headaches, dizziness, nausea and vomiting, blurred vision, impaired cognition, and seizures can all occur to some degree and severity.[63] Physicians must be alert to the possibility of CO poisoning when a patient presents with this constellation of signs and symptoms. The diagnosis can only be made when the Hbco test confirms toxicity due to CO poisoning.

When ordering confirmatory laboratory tests, other causes of mental status changes should be considered (eg, hypoglycemia, hypoxemia, metabolic changes, systemic infection, drug/alcohol intoxication, toxins, adverse drug reactions). The laboratory tests should include complete blood count, metabolic profile, urinalysis, urine drug screen, and serum ethanol. Creatine kinase may be useful for detecting rhabdomyolysis. In suspected CO poisoning cases, the patient's blood should be sampled and analyzed to determine Hbco levels.[4,13,16] The diagnosis of CO poisoning is confirmed when the Hbco level is greater than 3.5% in nonsmokers, or greater than 10% in smokers.[2,15] (However, as previously stated, Hbco level does not predict long-term outcomes well, nor is it a good measure for monitoring clinical progress.)

Hampson et al[15] proposed three criteria for diagnosing CO poisoning: (1) history of exposure to CO, (2) elevation in blood Hbco, and (3) signs or symptoms of CO toxicity. Clinicians should consider initiating treatment with oxygen therapy if they suspect CO poisoning until the Hbco test results are made available.

It is important to note that pulse oximetry is a poor test to use when CO poisoning is suspected because the standard pulse oximeter uses two wavelengths (660 and 990 nm) and cannot discern between oxyhemoglobin and Hbco.[15]

The DoD no longer requires providers to report CO poisoning since it has been removed from the list of reportable medical events.[64] However, many local jurisdictions still consider CO poisoning a reportable event, and healthcare providers are still encouraged to report any patient diagnosed with CO poisoning to their local preventive medicine service or public health detachments. Case definitions for a CO poisoning event vary by state, so clinicians are encouraged to report any suspected case.

TREATMENT

Any patient suspected of CO poisoning must first be removed from the contaminated environment to prevent further poisoning. The basic "CABD" (circulation, airway, breathing, disability) approach should be followed in CO poisoning cases. Once the patient is safely removed from the exposure, the following interventions should be considered:

- Place an advanced airway if mental status or upper airway is compromised.
- Mechanically ventilate if the gas exchange is poor during respiration.
- Obtain intravenous access to infuse resuscitative fluids and vasoactive drugs if necessary.

The patient should get 100% supplemental oxygen as soon as possible. If the patient is not intubated, a tight-fitting facemask will be required to deliver the oxygen. Oxygen should be continued for 6 to 12 hours to permit shifting the oxygen hemoglobin saturation curve to the right and displacing the CO.[46]

Electrocardiogram and cardiac monitoring should be used to evaluate for cardiac ischemia. Patients with Hbco levels greater than 25% often get ST-segment depression, which is one of the criteria for instituting HBOT.[48] The goal of treatment is to increase the partial pressure of oxygen in the lungs to displace CO from the Hbco. While delivering 100% oxygen at 1 atmosphere absolute (ata) is effective, the displacement can be accelerated by using hyperbaric oxygen. The half-life of Hbco is 4 to 6 hours at room air, 1 hour at 100% oxygen, and 20 minutes with hyperbaric oxygen at 3 ata.[46]

Hyperbaric oxygen may help reduce cerebral and myocardial hypoxia and cerebral edema, and also may enhance CO elimination sooner than other treatment options.[56] HBOT supports metabolic oxygen requirements

EXHIBIT 24-1

CONSULTATION AND ASSISTANCE WITH LOCATING HYPERBARIC OXYGEN TREATMENT FACILITIES

Unit Name	Availability	Phone Contact
*Navy Experimental Diving Unit, Panama City, Fl	Normal hours (0730–1600) After 1600	850-230-3100 850-234-4351
*Navy Dive School, Panama City, FL	Normal hours (0730–1600)	850-234-4651
*Army Hyperbaric Medicine Service, Eisenhower Army Community Hospital, Augusta, GA	Normal hours (0730–1600) After 1600	706-787-3110 706-787-9284
US Divers Alert Network (offers emergency phone consult)	Available 24 hours a day	919-684-9111

*Hyperbaric chamber on site

by rapidly providing dissolved plasma oxygen and enhancing Hbco dissociation and CO clearance.[48,50] HBOT is thought to benefit patients with cerebral edema by reducing secondary intracranial pressure by 50% within 1 minute of its administration.[48]

Despite this relatively simple concept of decreasing the elimination time for Hbco, as well as its physiologic benefits, HBOT remains controversial in practice. Controlled trials of HBOT in CO poisonings have not consistently found benefit. Consequently, HBOT remains controversial both in terms of indications for use and in treatment protocols. According to expert opinion, HBOT should be attempted for individuals with no other risk factors if Hbco levels are over 25%.[2] In patients with Hbco levels over 15% and under 25%, HBOT is generally indicated when the patient has altered mental status or loss of consciousness, abnormal neurologic findings, seizures, hypotension, cardiac ischemia, or pregnancy. Other indications may include persistent metabolic acidosis, concurrent burns, or pregnancy with any history of CO exposure.[11,15] No superior hyperbaric treatment protocol has been identified. An initial treatment at 2.5 to 3.0 ata is recommended; more treatment sessions can be added if symptoms persist.[46] Hyperbaric oxygen facilities range in size from large walk-in chambers to much smaller chambers that can only accommodate one person.

The fetus is more susceptible to CO poisoning than the mother. The fetal oxyhemoglobin dissociation curve is to the left of the adult dissociation curve. CO reacts to form fetal Hbco, which accentuates the left shift. While the Hbco levels in the fetus lag behind those in the mother, the final fetal Hbco level may be 10% to 15% higher than the maternal level.[14] Most importantly, the half-life of fetal Hbco is 15 hours, so it takes substantially (5 times) longer to regenerate oxyhemoglobin in the fetus than in the mother.[50] Exhibit 24-1 contains information on locating HBOT centers in the United States.

HEALTH STANDARDS AND GUIDELINES

Developing consistent exposure standards has been problematic because it is difficult to determine the exposure levels at which CO causes health effects; a large number of variables affect exposure levels, many of which fluxuate widely. Therefore, the guidelines below should not be substituted for sound clinical judgment in determining what constitutes a safe or dangerous exposure.

When deciding which standard or guideline to apply, the following factors must be considered:

- whether a healthy or susceptible population is exposed;
- whether exposures are to the general public or to a smaller subset of the population;
- whether or not operations are military-specific;
- whether exposures are occupational or residential; and
- whether exposures are chronic and relatively low-level or acute and high-level.

Tools are provided below to help investigators estimate Hbco and CO concentrations at the time and place of exposure.

On occasion, signs and symptoms may not correspond with what is expected based upon the measured

Hbco in blood or CO concentrations measured or predicted at the incident site. The presence of confounders may provide a partial explanation for the observed signs and symptoms that may not be satisfactorily explained by Hbco levels.

Military Occupational Exposures

The Army has established CO standards for military-unique workplaces, operations,[31] equipment, and systems.[65] These include combat and operations, as well as testing and maintenance of military weapons, aircraft, ships, submarines, missiles, early warning systems, military space systems, ordnance, and tactical vehicles. Also included are peacekeeping missions; field maneuvers; combat training; naval operations; flight and missile operations; military research, development, test, and evaluation activities; and national defense contingency conditions.

Work performed in some DoD workplaces and operations is similar to work performed in private sector businesses. These operations are not considered militarily-unique operations and can include work performed in weapon, vessel, aircraft, or vehicle maintenance; construction; supply; engineering; public works; medical services; and administrative office work.[65]

Military-Unique Standards

Where occupational exposures occur in military-unique settings, the CO concentration has to be reduced to the lowest level feasible,[31] and less than the OSHA permissible exposure limit (PEL) where practicable.[65] The Army, Navy, and NASA apply a CO exposure standard for military-unique workplaces and operations.[31] The ACGIH threshold limit value (TLV) may also be applied in military-unique situations.[65]

The Department of Defense design standard for human engineering (MIL-STD-1472G) requires that "Personnel shall not be exposed to concentrations of CO that will result in COHb [Hbco] levels in their blood greater than 5.0 percent for all system design objectives and aviation system performance limits and 10 percent for all other system performance limits."[31] The DoD handbook for human engineering design (MIL-HDBK-759C), paragraph 5.13.7.4.5, specifies use of the following empirical formula to predict Hbco blood content as a result of exposure to CO, and is based upon the Coburn, Foster, and Kane equation (CFKE)[66,67]:

$$Hbco_t = \%(Hbco_0\,(e^{(-t/A)}) + 218(1-e^{(-t/A)}) \bullet (1/B + \text{ppm CO}/1{,}403)$$

In this formula, $Hbco_t$ is the predicted Hbco in the exposed individual; $Hbco_0$ is the amount of Hbco usually found in nonsmoking adults; t is the exposure duration in minutes; and ppm CO is the CO concentration in ppm in the contaminated environment. The value of e, a numerical constant, is equal to 2.71828, and variables A and B are constants that depend on the physical activity level of the individual during the exposure, obtained from Table 24-2.

TABLE 24-2

WORK-EFFORT CONSTANTS FOR PREDICTING CARBOXYHEMOGLOBIN BLOOD CONTENT

Work-Effort Scale	Work-Effort Description	Alveolar Ventilation Rate (L/min)	A Value*	B Value*
1	Sedentary	6	425	806
2	Light work (eg, cooking, truck driving)	12	241	1,421
3†	Moderate work (eg, light walking, cycling)	18	175	1,958
4†	Heavy work (eg, loading, shoveling)	24	134	2,553
5	Very heavy work (eg, jogging, hill climbing)	30	109	3,144

*A and B values for each work-effort level is described in paragraph 5.13.7.4.5 of US Department of Defense. *Handbook for Human Engineering Design Guidelines*. Washington, DC: DoD; 1995: 302–303. MIL-HDBK-759C: 302-–03.

†A work-effort level of 4 should be chosen for periods of weapons fire, and a work-effort level of 3 should be chosen for periods of pause, when no weapons are fired.

Data sources: (1) Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health. Coburn equation calculator. https://www.cdc.gov/niosh/topics/co-comp/default.html. Accessed August 28, 2017. (2) US Department of Defense. *Design Criteria Standard (Human Engineering).* Washington, DC: DoD; 2012. MIL-STD-1472G. (3) Smith SR, Steinberg S, Gaydos JC. Errors in derivations of the Coburn-Forster-Kane equation for predicting carboxyhemoglobin. *Am Ind Hyg Assoc J.* 1996;57(7):621–625.

This equation allows the calculation of a predicted $\%Hbco_t$ from a CO exposure (in ppm CO) over a time interval and with respect to a particular work-effort level, and is very useful in situations where CO concentrations are episodic and fluctuate significantly over time, such as during the firing of a main tank gun or machine gun. During firing, the levels often rise steeply; during pauses in firing, levels fall. Because of the dynamic nature of such exposures, the use of this equation is a more accurate means of assessing exposure risk than by measuring and averaging CO exposures over an 8-hour time-weighted average (TWA) and comparing the result to an 8-hour PEL for CO. The equation accounts for the minute respiratory volume of contaminated atmosphere actually inhaled by an exposed individual based on the level of physical activity (either estimated or specified). The equation also accounts for the elimination of CO by the body. The equation is applicable to short-duration high-level exposures as well as low-level exposures of long duration.[66,67]

A value of 1% $Hbco_0$ must be chosen as the initial value before exposure begins.[67] The $\%Hbco_t$ predicted for one time interval becomes the $\%Hbco_0$ for the next time interval, and the process is repeated to predict %Hbco with time.[67] This model can be programmed into a National Institute of Occupational Safety and Health (NIOSH) computer model[68] and Microsoft Excel (see the attachment at the end of the chapter for an example).

Some studies show that nonsmoking, healthy personnel do not experience any significant effects of CO exposures with Hbco levels less than 5%.[17,66] However, other studies suggest that visual acuity may degrade at or below Hbco levels of 15%.[69] Because of the uncertainties evident in the research and the critical nature of crew tasks involving visual perception (night flight operations), the DoD limited CO exposures in aviation systems to keep Hbco levels below 5%. The system design objectives for CO exposures in all other systems was set to keep Hbco levels below 10% (paragraph 5.7.9.4.2 of MIL-STD-1472G).[31]

Other available computer models predict %Hbco. Tikuisis developed a computer program for the Canadian Department of National Defense in 1996 that allows the user to predict %Hbco over time based upon work-effort level (1–5), exposure concentration, and exposure duration.[32] Compared to the model described in MIL-HDBK-759C, the Tikuisis model is more flexible in allowing the user to select more variables that could affect the %Hbco level in a particular individual. For instance, the Tikuisis model allows entry of barometric pressure, and demographic variables (eg, height and weight, nonsmoker, light smoker, and heavy smoker).[32]

The Navy has set a separate standard for submarine environments. At the Navy's request, the National Research Council's Committee on Toxicology (NRC COT) developed and recommended a continuous exposure guideline level (CEGL) for CO. A CEGL is recommended for specific situations in which exposure to a chemical may occur continuously for up to 90 days.[18] It is defined as a ceiling limit designed to prevent adverse health effects, either immediate or delayed, and to avoid degradation in crew performance that might endanger the objectives of a particular mission. The 90-day CEGL for CO is 20 ppm and should not result in blood Hbco above 3.3%.[18] The CEGL is intended to be applied to a young, healthy military population; it is not intended to be applied to other occupational groups or the general public.[18]

Nonmilitary-Unique Standards

Where exposures are occupational in nature and where the workplace or operation is not considered military-unique, the Army applies either the ACGIH TLV or the OSHA PEL, whichever is more stringent.[70] Currently, the 8-hour ACGIH TLV-TWA of 25 ppm is more stringent than the OSHA 8-hour PEL-TWA, which is 50 ppm.[71,72] However, in maritime operations, workers must be removed from exposure if the CO concentration exceeds 100 ppm for any duration, even if the 8-hour TWA of 50 ppm has not been exceeded.

Permissible exposure limits. The OSHA 8-hour PEL-TWA for CO of 50 ppm produces an Hbco level of 8% to 10% in most workers. Generally workers free of cardiovascular conditions do not exhibit signs or symptoms of health impairment when exposed at this level in nonstressful conditions.[73] The 8-hour PEL-TWA for CO in maritime operations is set at 50 ppm; workers must be removed from exposure if the CO concentration exceeds 100 ppm.[73] This exposure level corresponds to a predicted Hbco level of 1.4% based on the predictive equation in MIL-HDBK-759C, assuming moderate work effort (level 3), an exposure duration of 5 minutes, an initial Hbco level of 1%, and no other CO exposures. A 200-ppm peak CO level was set for employees engaged in roll-on, roll-off operations during cargo loading and unloading. This concentration results in an Hbco level of 1.8% using the equation in MIL-HDBK-759C, with the same assumptions as noted above.

Threshold limit values. The ACGIH set the 8-hour TLV-TWA for CO at 25 ppm.[14,72] This was done to maintain blood Hbco levels below 3.5% in order to minimize adverse neurobehavioral and cardiovascular effects and maintain work capacity.[14,72] The ACGIH adopted this CO level to maintain a margin of safety

for susceptible individuals, including pregnant workers and their fetuses and those with cardiopulmonary conditions.[14,72] The ACGIH recommends that CO exposures be kept below 5 times the TLV-TWA, or 125 ppm, at all times, and kept at 75 ppm for no more than 30 minutes a day.[14,72]

Nonetheless, it must be understood that some exposures less than the 25-ppm TLV-TWA may result in Hbco levels that exceed 3.5%. Variation in exposures during the day may produce higher Hbco levels. For example, a 60-minute exposure to 200 ppm would result in an 8-hour TWA of 25 ppm, but the Hbco level could be as high as 10%. It is likely that the Hbco level would be less than 1% to 2% at the end of the shift in the absence of CO exposure for the remainder of the 8-hour shift.

CO is eliminated through the lungs.[14] The NIOSH established a BEI for CO of 3.5% Hbco and a CO level of 20 ppm in end-exhaled breath at the end of the work shift.[14] This exhaled breath concentration corresponds to a Hbco level of 3.5%.[14] The BEI is not applicable to tobacco smokers or people who drive on congested roadways,[14] nor is it applicable in emergency situations, during the first 3 hours of the shift, later than 15 minutes after the end of the work shift, or when there are large fluctuations in exposure concentrations.[14] It should be noted that when analyzing end-exhaled breath, false positives can occur in workers with lactose intolerance, those with intestinal malabsorption,[5] and anyone who has been drinking alcohol.[54]

Army Housing and Tents

Family Housing

To avoid CO poisoning in housing, occupants should follow the guidelines developed by the Consumer Product Safety Commission and the CDC, which describe the sources of CO in the home and what steps are needed to prevent CO poisoning. These steps include regular inspection and routine maintenances of fuel-burning appliances and use of CO detectors or alarms.[74]

CO alarms are highly recommended by the CDC. The US Army Corps of Engineers is required to install CO alarms in new and renovated family housing units with fuel-burning appliances, fireplaces, or an attached garage.[75] Generally, one CO alarm should be located on each level of the housing unit in or near the bedroom. The alarms should be hardwired and wall-mounted 50 inches off the floor. They should also be audible and have a continuous digital display, peak level memory, test button, and test reset button and be approved by Underwriters Laboratory (UL) as meeting UL standard 2034. Housing unit occupants can check the UL website to ensure their CO alarm is a UL-certified product.

CO detectors and alarms are important as a secondary defense; their use does not preclude the responsibility for proper use, regular inspection, or preventive maintenance of fuel-burning appliances. The intent of using such devices is to warn occupants of CO concentrations well before they have reached levels that may result in death or the incapacity to take action or exit. It is conceivable that there may be low-level CO exposures within the living area that do not activate the alarm but may be harmful to persons with cardiovascular disease.

Army policy prohibits military family housing residents from using portable gas or liquid fuel space heaters in family quarters or in tents.[76] Exceptions for interim emergency heating can only be granted by the installation commander, and only when CO alarms are present and working properly.[76]

Troop Tents

The deaths in Fort Irwin described above demonstrate the dangers of gas heaters. Commanders should only allow the use of vented tent heaters that meet military field heating requirements and are proven safe and effective, such as those listed in the Natick Soldier Center's Commanders' Smart Book Equipment Catalogue.[43] Only trained and licensed personnel are allowed to set up and operate heaters.[43] Individually owned heaters and commercial propane or natural gas heaters must not be used under any circumstances.[43] Unvented kerosene heater use should be restricted to areas where people do not sleep, such as guard houses, ranges, or training areas (or when approved by the commander as interim emergency heating with CO alarms in place).[5]

Acquisition of commercial nonstandard heaters is justifiable only in mission-critical circumstances; if nonstandard heaters are needed, the first general officer in the unit's chain of command must approve their purchase and use, based on the recommendations of safety, health, and fire protection personnel.[43] A complete risk assessment should be done if the commander decides that operational necessity requires use of an unvented kerosene heater in tents or other enclosed shelters. The risk of cold stress must be balanced against the risks of using the heater.[43] Precautions must be followed when using these heaters, and personnel must be trained on the health and safety issues related to heater use, including identifying heaters as a potential source of CO and teaching people about the symptoms of CO poisoning.[43]

Environmental Exposure Standards for the General Population

The Environmental Protection Agency (EPA) set the US national ambient air quality standards for CO at 9 ppm for 8 hours and 35 ppm for 1 hour.[74,77] These outdoor limits are intended to protect the general population and most sensitive subpopulations, including those with heart disease, by maintaining Hbco in nonsmokers below 2.0%.[4] Studies have noted that Hbco levels between 2.4% and 2.9% have aggravated angina and other cardiac conditons[72,77] and decreased exercise capacity.[77] Continuous exposure at 9 ppm would result in an Hbco level of about 1.5% when using the equation in MIL-HDBK-759C.

The EPA uses air monitoring stations to measure CO levels around the country, and the measurements are then compared to these standards. The EPA maintains a list of US areas not meeting these standards; these places are required to develop and carry out plans to reduce CO emissions. In 1995, 80% of CO emissions were generated by transportation (highway and off-road vehicles) and construction sources.[5]

Emergency and Accidental Exposures

NIOSH set the IDLH level for CO at 1,200 ppm based on acute toxicity data in humans.[72] OSHA requires employers to consider whether the range of exposures anticipated includes the NIOSH IDLH when selecting respiratory protection.[78] Per NIOSH, the IDLH value was set to the airborne concentration from which a worker could escape without injury or health effects in the event of the failure of respiratory protection equipment. The IDLH is considered a maximum concentration above which only SCBA should be permitted. In determining IDLH values, NIOSH considered the ability of a worker to escape without loss of life or irreversible health effects along with certain transient effects, such as severe eye or respiratory irritation, disorientation, and lack of coordination, which could prevent escape. IDLH values incorporate a margin of safety based on effects that might occur as a consequence of a 30-minute exposure.[72]

A 1,200-ppm exposure to CO would result in a Hbco level of about 30% when using the formula in MIL-HDBK-759C, using the standard set of assumptions with a work-effort level of 3. At a work-effort level of 4 (heavy work), a 1,200-ppm CO exposure for 30 minutes would result in a Hbco level of 38%.

The NRC COT developed emergency exposure guidance levels (EEGLs) for a healthy military population,[18] as well as submarine escape action levels (SEALs) for the Navy to protect crewmembers from toxic gases in disabled submarines.[79] Additionally, the American Industrial Hygiene Association established emergency response planning guidelines (ERPGs) to protect the general public and workers from exposures.[80]

Committee on Toxicology Emergency Exposure Guidance Levels

The NRC COT EEGLs were established for a military population of healthy soldiers and are not intended for other occupational groups or the general public.[18] The EEGLs and the predicted %Hbco levels associated with these EEGLs are provided in Table 24-3. The EEGLs were developed using air concentrations that do not exceed Hbco levels of 10%.[18]

EEGLs represent the ceiling limit for a single exposure (rare in a lifetime) of 60 minutes or less.[18] They consider the statistical likelihood that a nonincapacitating, reversible health effect may occur in the

TABLE 24-3

NATIONAL RESEARCH COUNCIL EMERGENCY EXPOSURE GUIDANCE LEVELS

Exposure Duration	EEGL*	Maximum %Hbco, Sedentary, 6 L/min, Work-Effort Level 1	Maximum %Hbco, Moderate Work, 18 L/min, Work-Effort Level 3	Maximum %Hbco, Heavy Work, 24 L/min, Work-Effort Level 4
10 min	1,500 ppm	6.4%	14%	18%
30 min	750 ppm	8.9%	19%	24%
60 min	400 ppm	9.1%	19%	23%
24 h	50 ppm	7.8%	7.9%	7.8%

*National Research Council, Committee on Toxicology. *Emergency and Continuous Exposure Guidance Levels for Selected Airborne Contaminants*. Vol 4. Washington, DC: National Academy Press; 2007.
EEGL: emergency exposure guidance level
Hbco: carboxyhemoglobin

exposed population.[18] EEGLS are designed to prevent substantial performance impairment during emergencies.[18] EEGLs must not be used for routine, predictable, and controllable operations such as in firing the main gun in a tank or howitzer.

Navy Submarine Escape Action Levels

SEALS were established to protect crew members in disabled submarines from the effects of exposure to high concentrations of toxic gases[79]; CO is one of eight gases of concern. A collision or explosion that causes onboard fires can expose crewmembers to high concentrations of toxic combustion products. Exposures to any of the eight gases can damage the respiratory system and CNS, which could result in death, either directly or by impeding crew members' ability to escape after a serious incident. The Navy developed two SEALS for each of the eight gases and requested that the COT independently review the available toxicological and epidemiologic data and evaluate the scientific validity of the two SEAL levels. The NRC reviewed the data and recommended that the Navy adopt a SEAL 1 (the maximum concentration of CO in a disabled submarine to which healthy submariners can be exposed for up to 10 days without irreversible health effects) of 80 ppm. The COT recommended the Navy adopt a SEAL 2 (the maximum concentration of CO in a disabled submarine to which submariners can be exposed for up to 24 hours without experiencing irreversible health effects) of 96 ppm.[79]

Emergency Response Planning Guidelines

The American Industrial Hygiene Association developed ERPGs to assist emergency responders in planning for chemical releases into the community with a goal of protecting the general public.[80] The predicted %Hbco levels presented in Table 24-4 were calculated using the equation in MIL-HDBK-759C using the same assumptions as stated previously.

Confounders

Many confounders alter the effects of CO poisoning, which may explain why the expected signs and symptoms do not always correlate to measured Hbco levels. There is a greater risk of CO toxicity in a population with decreased oxygen-carrying capacity or reduced oxygen availability.[14] Respiratory disease can impair oxygen exchange, and increased oxygen-hemoglobin affinity in the fetus may also increase the risk of CO poisoning.[14] Reduced atmospheric pressure at high altitudes, as well as increased work, can put workers at increased risk when exposed to CO.[14] In addition to CO, chemicals such as hydrogen cyanide and nitric oxide are products of combustion that may cause chemical asphyxiation, albeit by different mechanisms. Other chemicals such as methylene chloride are metabolized to CO.

Altitude

Oxygen deficiency generally causes no physiologic effects in healthy adults when the partial pressure of oxygen (Po_2) is greater than 132 mm Hg.[72] However, people living at elevations of 5,000 ft or above, where the Po_2 of the atmosphere may be less than 120 mm Hg, are more sensitive to CO and other asphyxiants.[72]

The effects of CO and of hypoxia from altitude are additive and similar, though the decreased Po_2 at high altitudes and increased Hbco produce different physiological responses.[74,81] The Po_2 in the atmosphere decreases as a function of increased altitude, though the $\%O_2$ remains the same.[74,76] For example, at sea level the Po_2 is about 159 mm Hg (ie, 0.21 × 760 mm Hg), whereas at an altitude of 7,000 ft above sea level, the Po_2 is about 121 mm Hg (ie, 0.21 × 580 mm Hg).[72] An ambient level of 121 mm Hg corresponds to an alveolar Po_2 level of 60 mm Hg due to dead space, carbon dioxide, and water vapor. Hemoglobin will be 90% saturated and normal levels of oxygen transport will occur in healthy adults provided the alveolar Po_2 stays above 60 Po_2.[72] An altitude of 6,000 ft (about 128 mm Hg Po_2) is the approximate physiologic equivalent to a CO exposure of 25 ppm CO at equilibrium (ie, at equilibrium, the Hbco level does not rise or decrease upon subsequent exposure to CO).[82]

The effects of altitude are more likely to be a contributing factor for someone who is not acclimatized. The body acclimatizes over about 4 weeks[83] through five mechanisms: (1) increased pulmonary ventilation, (2) increased red blood cells, (3) marked increase in diffusing capacity of the lungs, (4) increased blood vessels in the tissue, and (5) increased cell ability to use oxygen at low Po_2.[83] At an elevation of 17,000 ft, unacclimated and acclimated work capacities are 50% and 68% respectively.[83] Some workers in the Peruvian Andes live at an altitude of 17,500 ft and work in a mine at 19,000 ft.[83] The work capacity of these workers is 87%.[83]

CO exposure limits may be multiplied by an adjustment factor (AF) to account for the approximate effects of altitude on persons unacclimated to high altitudes. For instance, at an altitude of 6,000 ft and where the ACGIH TLV-TWA is applicable, an 80% AF might be applied to the 8-hour TWA exposure limit to get 20 ppm (ie, 0.8 AF × 25 ppm) until the person is acclimatized.[1(p49)]

TABLE 24-4

EMERGENCY RESPONSE PLANNING GUIDELINES*

Maximum Exposure Concentration Over 60 Min	Notes	Maximum %Hbco Sedentary, 6 L/min Work-Effort Level 1	Maximum %Hbco, Light Work, 12 L/min, Work-Effort Level 2	Maximum %Hbco Moderate Work, 18 L/min, Work-Effort Level 3	Maximum %Hbco, Heavy Work, 24 L/min, Work-Effort Level 4
200 ppm (ERPG-1)	Maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to 1 h without experiencing symptoms of exposure. Designed to keep Hbco levels < 5%–6%.	5%	7.7%	9.8%	12%
350 ppm (ERPG-2)	Maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to 1 h without experiencing irreversible or other serious adverse health effects that could impair the ability to take protective action. Designed to keep Hbco levels < 10%–12%.	8.1%	13%	17%	20%
500 ppm (ERPG-3)	Maximum airborne concentration below which it is believed nearly all individuals could be exposed for up to 1 h without experiencing life-threatening health effects. Designed to keep Hbco levels < 15%.	11%	18%	23%	29%

*The values in the table are intended for planning purposes only and not intended for use in routine operations or to distinguish between safe and unsafe exposure levels. The work-effort level used in developing the ERPGs is between sedentary (level 1) and light work (level 2).
ERPG: emergency response planning guideline
Hbco: carboxyhemoglobin

Studies of laboratory animals and humans show that Hbco levels are elevated at altitude.[74] Also, higher Hbco levels have been observed in individuals breathing CO (9 ppm) at rest at altitude compared to those Hbco levels observed at sea level.[74] Exercise in a CO atmosphere (50–150 ppm) at altitude produced lower Hbco levels than those found under similar conditions at sea level, which may be due to either suppressed Hbco formation or a shift in the CO storage.[74]

Hypoxic hypoxia caused by high altitude (25,000 ft) seems to be better tolerated by smokers than nonsmokers, who tend to experience more severe symptoms and have less work capacity.[74] This may be due to the fact that smokers have chronic hypoxemia and develop a partial tolerance to hypoxic hypoxia.[74]

Heat Stress/High Temperature

Heat stress and high temperature when combined with CO exposure produced a decrement in the exercise performance at concentrations of 50 ppm CO.[14,74]

Tobacco Smoking

Tobacco smokers have an elevated Hbco level, ranging from 4% to 20%, and subsequent exposures to other sources of CO will further raise their Hbco. The Hbco level rises with the amount of smoking, with a mean of 5% to 6% Hbco for one pack of cigarettes per day; a mean of 7% to 9% Hbco for two to three packs of cigarettes per day; and up to 20% Hbco for cigars.[14]

An empirical formula in MIL-HDBK-759C (paragraph 5.13.7.4.5)[66] based upon the CFKE predicts Hbco blood content as a result of exposure to CO and can be used to roughly estimate the impact of tobacco smoking by entering an initial Hbco expected for a smoker.

Nitric Oxide

Additive toxicity can be anticipated upon simultaneous exposure to nitric oxide and CO.[14,17] Nitric oxide exposure results in the formation of nitrosyl hemoglobin, a compound that is incapable of oxygen transport.[14,17] Following inhalation of very high nitric oxide concentrations (eg, 80 ppm), circulating methemoglobin concentrations of up to 15% can be reached.[14,17]

Hydrogen Cyanide

There seems to be a slight additive interaction between CO and cyanide, reducing the lethal concentration for 50% of test animals by about 10% when rats have been exposed simultaneously to high concentrations of CO and cyanide.[14,17] Cyanide easily diffuses into all parts of the body and inhibits the metabolic enzyme cytochrome oxidase, which is involved in the transfer of electrons to molecular oxygen.[84] As a result, cyanide quickly halts practically all cellular aerobic respiration.[85]

Methylene Chloride

Methylene chloride is a solvent and paint stripper that is metabolized in the body to CO.[14,17] Exposure at the ACGIH TLV-TWA of 50 ppm produces a 3% Hbco level.[14,17]

Additive Effects Formula for Chemical Mixtures

When individuals are exposed to chemical mixtures involving CO and other substances such as nitric oxide and methylene chloride, the following formula should be applied to determine the exposure concentration and time limit:

$$C_1/T_1 + C_2/T_2 + \ldots C_n/T_n$$

The combined exposure is determined by adding the concentrations divided by time interval of the exposure for each chemical encountered, where C_1 indicates the observed atmospheric exposure concentration and T_1 is the corresponding exposure time limit.[72]

Health Status

Individuals with cardiopulmonary conditions including chronic obstructive pulmonary disease, coronary artery disease, and congestive heart failure have reduced blood oxygen content and are at greater risk from CO exposure because the additional reduction in blood oxygen-carrying capacity resulting from Hbco formation increases the relative hypoxemia.[74] Individuals with medical conditions affecting the blood including anemia and polycythemia are also at increased risk from CO poisoning.[74]

Ototoxicity

In 2003, USACHPPM published a fact sheet listing occupational ototoxins that contribute to hearing loss.[86] CO is on the list; it contributes to noise-induced hearing loss when CO exposure is combined with hazardous noise exposure.[85,87–92] The mechanism of ototoxicity for CO poisoning is thought to be free radical formation, which potentiates the effects of hazardous noise exposure on the hair cell.[90]

For workers enrolled in a hearing program due to excessive noise exposure, clinicians must be aware of the possible potentiating effects of CO exposure. They may need to initiate actions to reduce exposure to both noise and CO.[86] USACHPPM recommends that workers' exposures be kept below 50% of the occupational exposure limit for ototoxic substances, regardless of the actual noise level. Thus, audiograms should be performed when exposures are at one-half the 8-hour TLV-TWA of 25 ppm, or when the CO exposure exceeds 12 ppm and hazardous noise is present in the workplace.

Exposures should be documented in the comments section of DD 2215, Reference Audiogram, and DD 2216, Hearing Conservation Report, noting the ototoxins present in the workplace and the exposure levels of each. For CO exposures, the following should be included: CO exposure concentrations over the course of the day; predicted Hbco levels over the course of the day; noise exposure levels over the course of the day with respect to CO concentrations throughout the day; and other activities conducted outside of work that may have combined CO and noise exposures (eg, volunteer firefighting, playing in a rock band). Also, if the worker is a smoker, the number of packs of cigarettes or number cigars smoked per day should be noted.[86]

MEDICAL SURVEILLANCE

Workers routinely exposed to CO must receive hazardous communications training on the potential hazards of CO, as well as the role of engineering controls and use of personal protective equipment in controlling the risk of exposure. Pregnant workers and others considering pregnancy should be encouraged to quit smoking, advised about CO toxicity risks to the fetus, and trained on the ways to minimize the exposure potential. The employer must provide and properly maintain personal protective equipment. The criteria for enrolling workers into periodic medical surveillance for CO is if exposures exceed the action level for 30 days a year. An industrial hygienist should provide the occupational health clinic with documentation of CO exposures for placement in the medical records of all affected employees.

Workers must also undergo a preplacement physical examination, including a complete history, to identify medical conditions that put them at increased risk from CO toxicity, including smoking, chronic obstructive pulmonary disease, coronary artery disease, cardiovascular disease, CNS disorders, and anemia. In addition, workers must receive periodic and termination examinations. All three exams should emphasize the cardiovascular system, the pulmonary system, and the CNS. A complete blood count baseline should be obtained in the preplacement exam, and subsequent counts should be obtained when clinically indicated. A complete blood count should also be obtained immediately following acute exposure, and the worker should be examined for evidence of CO toxicity. After exposure, a venous blood sample should be obtained and examined for Hbco level.[93]

An occupational medicine physician can perform and document evaluations of the patient's mental, baseline neurological, and visual or ophthalmological status. A useful tool is the CO neuropsychological screening battery, which tests short-term memory, concentration, visual spatial ability, agnosia, and aphasia.[94]

SUMMARY

Exposure to CO can cause acute clinical illness. Military exposures to CO may occur outdoors; inside homes, vehicles, and workplaces; and while using military vehicles and weapons systems. Soldiers exposed while operating vehicles or weapons systems may experience a cyclic exposure, in which high peak exposures are followed by periods of minimal exposure, followed by successive peaks and troughs.

Cellular enzyme systems are adversely affected by CO poisoning due to hypoxia produced by formation of Hbco. Individuals with coronary artery disease appear to be at higher risk of ischemia following CO poisoning, but there have been reports of ophthalmologic and neurologic problems as well. Timely diagnosis and early oxygen therapy will help reduce Hbco levels and facilitate tissue oxygenation, which can reduce both the morbidity and mortality in CO poisoning cases.

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ATTACHMENT: EXAMPLE OF MODELING PERCENT OF CARBOXYHEMOGLOBIN WITH TIME USING MICROSOFT EXCEL

Example with Values Shown

Interval	Operation Description	CO Air Concentration (ppm)	Exposure Duration (min)	$\%Hbco_0$ (beginning of interval)	A	B	$\%Hbco_t$ (end of interval)
1	Weapons fire	500	5	1	134	2,553	3.81
2	Pause	10	5	3.81	175	1,958	3.75
3	Weapons fire	1,000	5	3.75	134	2,553	9.31
4	Pause	10	5	9.31	175	1,958	9.09

Same Example with Formulas Shown

	A	B	C	D	E	F	G	H
1	Interval	Operation Description	CO Air Concentration (ppm)	Exposure Duration (min)	$\%Hbco_0$ (beginning of interval)	A	B	$\%Hbco_t$ (end of interval)
2	1	Weapons fire	500	5	1	134	2,553	=E2*EXP(-D2/F2)+218*(1-EXP (-D2/F2))*(1/G2+C2/1403)
3	=A2+1	Pause	10	5	=H2	175	1,958	=E3*EXP(-D3/F3)+218*(1-EXP (-D3/F3))*(1/G3+C3/1403)
4	=A3+1	Weapons fire	1,000	5	=H3	134	2,553	=E4*EXP(-D4/F4)+218*(1-EXP (-D4/F4))*(1/G4+C4/1403)
5	=A4+1	Pause	10	5	=H4	175	1,958	=E5*EXP(-D5/F5)+218*(1-EXP (-D5/F5))*(1/G5+C5/1403)

CO: carbon monoxide
Hbco: carboxyhemoglobin
$\%Hbco_0$: initial percent carboxyhemoglobin
$\%Hbco_t$: final percent carboxyhemoglobin
ppm: parts per million