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Carbon Monoxide Exposure: Evaluation and Management

Carbon monoxide (CO) is responsible for significant morbidity and mortality worldwide and often represents a diagnostic challenge for emergency providers because of its wide range of nonspecific symptoms. Highly oxygen-dependent organs, such as the brain and heart, are particularly vulnerable to CO toxicity. The clinical spectrum is broad and can range from headache and flu-like symptoms to syncope, coma, and death.

The pathophysiology of CO toxicity lies in its strong affinity for hemoglobin (230 to 270 times that of oxygen), which results in a leftward shift of the oxygen-hemoglobin dissociation curve and impairs oxygen delivery to tissues.[1,2] Additionally, carboxyhemoglobin (COHb) causes direct cellular toxicity, nitric oxide formation, and oxidative stress.

Oxygen therapy is the mainstay of treatment, and 100% oxygen should be provided via non-rebreather face mask in any suspected or confirmed CO exposures. Hyperbaric oxygen therapy (HBOT) can further speed CO elimination and may mitigate some of the persistent and delayed neuropsychiatric effects of CO poisoning; however, there is no established standard of care for HBOT at this time.

Definition

Carbon monoxide is a colorless, odorless gas, the inhalation of which can cause a diverse range of nonspecific symptoms, including headache, myalgias, seizure, syncope, or death. It often is referred to as the great mimicker and can easily be mistaken for more benign diagnoses, such as viral infections. Special attention should be paid to the history to identify any potential exposures, such as nonconventional heating sources, inadequate ventilation, or exposure to engine exhaust. It is imperative that the healthcare provider identify subtle exposures in patients with mild symptoms to avoid misdiagnosis and prevent discharge back to dangerous environments.

Epidemiology

Carbon monoxide poisoning is a common presentation in the emergency department (ED). According to the Centers for Disease Control and Prevention (CDC), more than 50,000 ED visits and 430 deaths are related to CO poisoning each year.[3] Although the exact prevalence is unknown, an estimated 50% of patients who present with acute symptomatic CO poisonings will develop neurologic sequelae, which is a substantial contributor to morbidity.[4,5] During the past 25 years, deaths from CO poisoning have decreased significantly (7.2 to 4.6 deaths per million each year); however, overall the incidence of CO poisoning has remained stable.[6] This likely is because of a combination of greater

EXECUTIVE SUMMARY

- The most common symptom of carbon monoxide (CO) poisoning is headache.
- A key clue to suspecting CO poisoning is obtaining a history of exposure to combustion.
- Pulse CO-oximetry (SpCO) can be used as a screening tool in low-risk patients.
- Initiate 100% oxygen therapy via non-rebreather mask upon suspicion of CO poisoning while awaiting test results.
- Direct CO-oximetry of venous or arterial blood samples remains the gold standard for detection of CO exposure.
- For victims exposed to fire in an enclosed space, consider cyanide poisoning in addition to CO poisoning.
- Consultation with a toxicologist or hyperbaric expert is recommended regarding hyperbaric oxygen therapy in patients with moderate or severe CO toxicity.

availability of CO-oximetry testing, earlier implementation of effective treatment, and improved recognition of the high morbidity and mortality related to acute CO exposure. Although there is no difference in incidence of CO poisoning between males and females, men prove to be twice as likely to die from CO poisoning.[6] Age also plays a significant role in mortality, with the global risk of mortality after CO exposure being nearly double in infants and 26-fold higher in the elderly.[7]

Carbon monoxide exposure may be accidental or intentional, and providers should maintain a high suspicion for historical clues that suggest possible exposure. Exposures to high levels of CO may occur due to building fires, charcoal briquettes, propane-powered forklifts, ice resurfacing machines, automobile or boat exhaust, malfunctioning generators, gas camping stoves, and locations with inadequate ventilation.

Overall, more than half of CO poisoning cases in the United States stem from malfunctioning furnaces, usually during cold months.[4] Importantly, catastrophic weather can indirectly precipitate CO exposure, and the diagnosis should be considered in all patients presenting during extreme events like blizzards and hurricanes, often secondary to the use of portable generators.[4,8]

While the majority of cases (72.9%) are believed to be accidental poisoning, there remains a significant portion of CO poisoning that occurs as a result of attempted suicide.[9] Future studies should aim to characterize the burden and harm of intentional CO poisoning, but many cases might never present to the ED or are not suspected because of their subclinical presentation.

Despite the limitations of demographic data for toxicologic exposures like CO, national databases representing the U.S. population characterize the most likely victim of unintentional non-fire-related CO poisoning to be older (aged 45-64 years) white males living in the South or Midwest.[6]

Etiology

Carbon monoxide is formed by the incomplete combustion of carbonaceous fuel. Vehicles are responsible for half of unintentional CO poisoning deaths in the United States, even after the widespread implementation of catalytic converters.[4] Catalytic converters have been mandatory in all U.S. automobiles since 1975 and effectively convert harmful exhaust fumes like CO to steam and other benign gases. While unintentional deaths have remained stable, suicide attempts and deaths using automobile exhaust did decrease with catalytic converter implementation.[10] Even open air vehicles, like pickup trucks, forklifts, ice resurfacing machines, and boats, have been implicated in CO exposures.[4] (*See Table 1.*)

Charcoal and propane burning grills represent another common source of CO exposure, and the ill-advised use of gas stoves as a heating source has been shown to be predictive of CO poisoning in patients presenting with headache and dizziness.[4] For patients presenting with nonspecific symptoms in poor socioeconomic communities, consider asking about alternative heating sources in the home, especially during cold winter months.

Fires represent the most obvious source of severe CO poisoning, and all patients brought in after fire or smoke exposure should be evaluated for CO exposure. In fact, CO poisoning is considered the most important clinical effect of smoke inhalation.[4]

A wide variety of unexpected exposures also have been tied to CO poisoning, including anesthetic absorbents, banked blood, formic acid decomposition, methylene bromide, underground mine explosions, and wood pellet storage.[4] Waterpipe, also known as hookah or shisha, use also has been associated with acute CO poisoning and should be considered in individuals reporting a history of recent use, even in the absence of symptoms.[11,12] In one case report, blood COHb was measured as high as 21% after reported waterpipe use alone.[11,12]

Toxicokinetics

Carbon monoxide is readily absorbed into the bloodstream via the lungs, and with exponential uptake, it may take more than four hours to obtain steady state.[4] Once absorbed, CO interferes with oxygen transport and delivery to body cells. CO has an affinity for hemoglobin that is 230-270 times that of oxygen, resulting in the formation of COHb, which shifts leftward the oxygen-hemoglobin dissociation curve, crippling oxygen delivery to tissues.[1,2] The peak concentration of COHb depends on factors that include baseline patient COHb level, duration of exposure, pulmonary ventilation, and individual pre-exposure health.[1]

The CO molecule also has high affinity for other heme-proteins, including myoglobin and cytochrome c oxidase, further disrupting cellular respiration and causing production of reactive oxygen species.[2,13] Strong binding

Table 1. Sources Implicated in Carbon Monoxide Poisoning

Man-Made	Metabolic Exposure	Natural Sources
• Malfunctioning furnaces • Portable generators • Gas camping stoves • Charcoal briquettes • Building fires • Automobile exhaust • Boat engine exhaust • Propane-powered forklifts • Ice resurfacing machines • Kerosene heaters • Waterpipe (hookah)	• Methylene chloride • Paint stripper	• Forest fires • Volcanic eruptions

Table 2. Elimination Half-Life of Carbon Monoxide[15]

Treatment	Half-Life
• Room air	• 3-4 hours
• 100% normobaric oxygen	• 30-90 minutes
• Hyperbaric oxygen therapy (2.5 atm)	• 15-23 minutes

throughout the body can lead to both organ-specific and global dysfunction, as discussed in more detail in the section on pathophysiology.

Endogenous sources of CO rarely are considered in acute poisoning, since they often are negligible. Carboxyhemoglobin is a byproduct of heme metabolism by heme oxygenase, but baseline levels rarely exceed 2%.[4] However, in patients with hemolytic anemia, baseline COHb levels may increase to 3% to 4% in nonsmokers.[14]

The majority of CO is confined to the blood compartment, but it also can easily diffuse into all compartments, including muscle and brain. At steady state, 15% of total body CO content will be taken up by the body tissues, primarily bound to myoglobin.[1] During treatment with oxygen, CO will be displaced gradually back into blood as COHb blood saturation falls, allowing for further removal and detoxification.

The binding of COHb is dependent on the fractional concentration of oxygen (FiO_2) inhaled. Consequently, the higher the FiO_2, the faster the dissociation of CO from hemoglobin. At room air, the typical half-life of the COHb compound is three to four hours, but this can be decreased to 30 to 90 minutes with 100% normobaric oxygen. The half-life further decreases with HBOT to 15 to 23 minutes at 2.5 atm and 100% oxygen.[15] (*See Table 2.*)

The Coburn-Forster-Kane (CFK) model may be used to predict COHb levels using a given exposure history, but it may overestimate COHb in the setting of acute high CO exposures.[4]

Methylene chloride, such as that found in paint removers, represents another potential CO exposure. It may be absorbed via the skin, ingested, or inhaled and can take eight hours or longer to reach peak serum levels. Methylene chloride is hepatically metabolized to CO and, consequently, has a more prolonged half-life (13 hours).[4]

Pathophysiology

The primary and most obvious effect of CO poisoning is its binding to hemoglobin, which subsequently impairs oxygen delivery to body tissues. Thus, even with adequate partial pressures of oxygen in the blood, hemoglobin is unable to effectively deliver oxygen peripherally, resulting in tissue ischemia. However, CO toxicity cannot solely be attributed to this mechanism and also results from direct cellular toxicity and nitric oxide (NO) formation.

Carbon monoxide is a cellular asphyxiant, meaning it interferes with the ability of cells to use oxygen for respiration.[4] Specifically, CO binds to mitochondrial cytochrome oxidase and cytochrome P450 oxidase, impairing oxidative metabolism and effectively inhibiting ATP production.[13,15] Carbon monoxide also can directly damage cells through direct intracellular protein binding and lipid peroxidation, triggering an inflammatory cascade, leading to apoptosis and cell death.[14]

Myoglobin, another heme protein, also preferentially binds CO with an affinity 60 times greater than its affinity for oxygen. This results in myocardial impairment, reducing oxygen availability in myocytes, with subsequent hypoxia, lactic acidosis, and ischemia. These effects ultimately contribute to dysrhythmias, cardiac dysfunction, and clinical decompensation.[14,15]

Nitric oxide also has been implicated in the cerebral vasodilation and microvascular damage that might account for some of the long-term sequelae seen in CO poisoning. Endothelial dysfunction results from local nitric oxide free radicals, which when displaced from platelets by CO, serve to activate cytochrome oxidase and attract white blood cells to adhere to cerebral vasculature.[4] This, in turn, activates local myelin peroxidase to release proteases, which promotes further oxygen free radical formation and causes neuronal lipid peroxidation. The subsequent neuronal death that follows represents the end point of the damaging inflammatory cascade seen in cerebral vasculature.[4] Oxidative stress simultaneously leads to pathologic activation of glutamate and other excitatory amino acids, which causes neuronal damage and loss due to intracellular calcium release.[4]

CO poisoning's persistent and delayed effects are varied and generally referred to as delayed neurologic sequelae (DNS). Interestingly, these effects may represent persistent symptoms following acute exposure or a delayed deterioration after a latency period of two to 40 days.[4,14] While true prevalence is difficult to ascertain,

prospective studies have demonstrated that 34% of patients report symptoms like headache or memory deficits at four weeks even with normobaric oxygen therapy, and 46% of patients had neuropsychiatric sequelae at six weeks.[13,16] These delayed psychiatric and neurologic effects likely involve lesions of the cerebral white matter with poor vascular blood supply.[4] Autopsy data have demonstrated necrosis of the white matter, globus pallidus, cerebellum, and hippocampus.[4] Magnetic resonance imaging studies confirm damage and atrophy to the basal ganglia and hippocampus years after CO poisoning.[13]

Similar to the previously discussed mechanisms, the delayed neurocognitive effects of CO have been hypothesized to be due to several factors, including CO-mediated brain tissue hypoxia, oxygen-based free radicals, and membrane peroxidation, rather than simply being an effect of direct hypoxia-induced damage.[5,17] Etiologies that include inflammation and autoimmunity, ischemia-hypoxic injury, and cellular apoptosis also have been proposed without definitive evidence to support any single theory.[18,19]

Clinical Features

The diagnosis of CO poisoning may be clinically challenging given its varied presentation that can be readily confused with other illnesses, such as a benign viral syndrome. Mild CO exposures can result in headache, dizziness, nausea, fatigue, or myalgias. More severe exposures can result in chest pain, confusion, loss of consciousness, arrhythmias, cardiac arrest, or even death. (*See Table 3.*) Carbon monoxide concentration and duration of exposure are the most important determinants of toxicity.[20] Unfortunately, patients who are sleeping or intoxicated may never experience any symptoms before succumbing to CO poisoning.

Headache is the most common presenting symptom, usually described as dull, frontal, and continuous.[21] It can easily be misdiagnosed as a tension headache without a thorough history or adequate suspicion for occult CO exposure. As exposure increases, patients may present with more pronounced or severe symptoms, such as altered mental status, cardiac ischemia, and neurologic deficits. Both the heart and brain are highly oxygen-dependent, and even healthy patients may present with dizziness and ataxia at COHb levels as low as 15% to 20%.[4]

Table 3. Clinical Signs and Symptoms of Carbon Monoxide Poisoning

Mild-Moderate	Moderate-Severe
• Headache • Fatigue • Nausea • Vomiting • Dizziness • Blurred vision • Confusion • Chest pain • Shortness of breath • Weakness	• Cardiac ischemia • Non-cardiogenic pulmonary edema • Syncope • Dysrhythmias • Syncope • Seizure • Hypotension • Altered mental status • Coma • Cardiac arrest

Early cardiovascular effects include tachycardia as a direct response to hypoxia but may progress to angina and cardiac ischemia. Troponin-I can be used to evaluate for myocardial damage following CO exposure, which may occur even in the absence of pre-existing coronary artery disease or electrocardiogram (ECG) changes.[22] CO poisoning also can act as an exercise stress test, unveiling underlying cardiovascular disease.[14] The cardiovascular effects of CO lower the threshold for malignant dysrhythmias, the ultimate cause of acute mortality in most CO deaths.[4] Myocardial stunning may be seen in acute CO poisoning, manifesting as global hypokinesia or Takotsubo cardiomyopathy.[23] Increased serum concentrations of B-type natriuretic peptide, lactate, and troponin can last several days.[23]

Additional features of CO poisoning include rhabdomyolysis, acute renal failure, noncardiogenic pulmonary edema, and cutaneous bullae.[14] The often-described "cherry-red" skin color is seldom seen in medical practice and usually is seen only in autopsies and severe cases involving prolonged exposure.[4] The distinctive color is postulated to occur as a result of a combination of CO-induced vasodilation, concomitant tissue ischemia, and failure to extract oxygen from arterial blood, but it cannot be used reliably to detect CO exposure.[4]

Infants are at a particularly increased risk for CO toxicity because CO binds more tightly to fetal than adult hemoglobin.[14] As a result, even low exposures can be life-threatening for an infant. Young children remain at elevated risk for toxicity, given their higher metabolic rate and oxygen update. Similar to the adult patient, symptoms in the pediatric population are nonspecific and include headache, nausea, vomiting, and lethargy, which might be easily misdiagnosed as a viral illness or gastroenteritis.[4]

Pregnant patients represent a unique high-risk population for CO poisoning, since severe fetal effects can result even when the mother appears asymptomatic. Carbon monoxide passes readily through the placenta to the fetus, and diffusion capacity increases with gestational age and fetal weight.[24,25] The accumulation of fetal COHb is insidious, rising later and more slowly over time. The CO blood levels in the fetus are thought to be 10% to 15% higher than in maternal blood, due to the increased affinity of fetal hemoglobin.[24,25]

The subsequent fetal CO toxicity results from two mechanisms: direct toxicity due to fetal COHb and decreased oxygen delivery via placenta secondary to maternal hypoxia.[24] As previously discussed, COHb competitively binds to hemoglobin, displacing oxygen, and inhibits cytochrome P450 and cytochrome oxidase, crippling cellular respiration. In the setting of decreased maternal oxygen availability, these effects can be catastrophic, especially since the fetus is otherwise

unable to increase cardiac output to compensate for the decreased oxygen saturation.[24] In animal studies and case reports, the effects on the fetus can include fetal distress, premature birth, anatomic malformations, brain injury, and death following maternal exposures.[4,14]

Chronic subacute CO exposures likely are further underreported and represent an even greater diagnostic challenge for medical providers. Symptoms may include chronic fatigue, headache, memory issues, cognitive slowing, emotional distress, sleep disturbances, neuropathy, paresthesias, vertigo, polycythemia, and cardiomegaly.[4,13,14] Further demographic data or prognostication is limited by confounding factors and lack of information regarding the nature of exposure. Unfortunately, the nonspecific nature of CO poisoning symptoms — especially subacutely — make diagnosis difficult.

Both treatment and study of chronic exposure require recognition of symptoms and eventual source identification. For these reasons, successful diagnosis of chronic exposure relies on taking a thorough history. Temporality of symptom onset and a keen ear for relevant environmental changes can be helpful, as can a low threshold for suspicion for CO testing in the proper context.

Persistent or delayed neuropsychological symptoms after CO poisoning are common, and the syndrome is referred to as DNS. The syndrome can manifest with neurologic or psychiatric features, which persist or appear after an apparent full recovery from acute CO poisoning.[14] Symptoms may include ataxia, dementia, psychosis, parkinsonism, cognitive disability, and behavioral changes.[4,20,26] Correlated neuroimaging has demonstrated hippocampal atrophy as well as changes in subcortical structures and pallidum in affected patients; however, it should be noted that these abnormalities are not specific to CO poisoning.[20]

Diagnostic Studies

Patients with suspected acute or occult CO poisoning should have the COHb level measured by spectrophotometry via CO-oximeter. This device enables differentiation of various forms of hemoglobin, such as oxyhemoglobin, deoxyhemoglobin, methemoglobin, and COHb, by their absorptive properties.[27] This is in notable contrast to conventional pulse oximetry, which uses only two wavelengths (660 and 930 nm).[27] Standard pulse oximetry cannot reliably distinguish oxyhemoglobin from COHb given the proximity of their wavelengths, so it will unavoidably overestimate blood oxygenation.[16,27]

Clinicians should maintain high index of suspicion for making the diagnosis of occult CO poisoning, since COHb levels may be normal, especially when patients have already received 100% oxygen or there has been significant time since exposure.[16]

A COHb level greater than 5% in nonsmokers or 10% in smokers confirms exposure to CO. However, blood levels do not correlate with severity of initial symptoms nor outcome.[4,28] Rarely, the COHb level will exceed 10% in heavy smokers with underlying lung pathology, but the COHb generally will rise 2.5% for each pack of cigarettes smoked per day.[29] It should be noted that arterial blood gas is not necessary because of the high correlation of the COHb levels between arterial and venous samples.[14]

In recent decades, noninvasive pulse CO-oximeters have been developed for quick, indirect measurement of blood SpCO levels. While convenient, this technology is hamstrung by high false-negative rates.[21] Even under laboratory conditions, noninvasive CO-oximeter specificity for CO poisoning only reaches 54%; however, its sensitivity has been observed as high as 94% with fair concordance with direct CO-oximetry of blood samples.[21,30,31] Accordingly, noninvasive pulse CO-oximetry (SpCO) can be viewed as a cheap, rapid screening modality for low-risk patients with nonspecific symptoms, but it should never be used independently for diagnosis of CO poisoning. Conventional direct CO-oximetry of blood samples remains the gold standard for detection of CO exposure and always should be sought in the setting of high suspicion for CO exposure.

Some studies have raised concern that spectrophotometric methods like CO-oximetry for the diagnosis of CO poisoning can be greatly affected by degradation of samples due to protein catabolism during blood draws, transport, and storage.[32] For this reason, total blood CO measured chromatographically has been proposed as an alternative diagnostic modality.[32] However, the factors that contribute to dissociation of CO from hemoglobin, such as sample reopening, thaw and re-thaw cycles, and storage parameters, are unlikely to cause clinically significant differences in samples properly drawn and sent immediately for analysis.[32]

Other recommended diagnostic testing includes 12-lead ECG, troponin, and creatinine kinase to evaluate for acute myocardial injury and rhabdomyolysis. The ECG may demonstrate supraventricular or ventricular arrhythmias, disturbances of repolarization, or QT prolongation in addition to ischemia.[15] Chest radiography can show evidence of noncardiogenic pulmonary edema with ground glass opacities, perihilar haze, or peribronchial cuffing.[14,27] Head computed tomography also should be obtained in severe CO poisoning to assess for cerebral edema, infarction, and focal lesions. One well-reported finding in CO poisoning is low-density lesions in the globus pallidus bilaterally.[33] These lesions may be delayed for several days after presentation, but, when seen, should alert the medical provider to possible CO exposure.[14] (*See Table 4.*)

Since both cyanide and CO exposures are common in smoke inhalation injury, cyanide toxicity also must be suspected in such scenarios, especially in patients presenting with severe metabolic acidosis. Direct testing for cyanide rarely is available, and, thus, empiric treatment must be considered in the appropriate clinical scenarios. Blood samples should be obtained for CO-oximetry prior to administration of hydroxocobalamin therapy for cyanide poisoning because of its reddening effect on the skin and urine. The red color, which typically resolves in two to three days, interferes with several laboratory tests, including COHb, methemoglobin, and oxyhemoglobin.[24] Other cyanide antidotes, such as nitrites and 4-dimethylaminophenol, should not be administered, as methemoglobin will be formed as a byproduct,

Table 4. Differential Diagnosis

Diagnosis	Differentiating Clinical Features	Differentiating Laboratory Features	Treatment
Carbon monoxide poisoning	Headache, nausea, vomiting, lethargy, history of exposure	COHb level > 5% in nonsmokers and > 10% in smokers	100% oxygen therapy, hyperbaric oxygen
Influenza	Myalgias, fever, rhinorrhea	Positive PCR for influenza virus	Oseltamivir (early initiation), supportive care
Viral upper respiratory infection	Rhinorrhea, eye discharge, sinus/lung congestion	Respiratory viral pathogen panel (rarely indicated)	Supportive care
Tension headache	Similar headache as carbon monoxide poisoning in the absence of other symptoms	Not applicable (negative COHb)	NSAIDs, acetaminophen, caffeine, IV fluid hydration
Cyanide poisoning	Somnolence, seizures, coma, shortness of breath	Red blood cell cyanide concentration (abnormal > 2.5 mcg/mL)	Hydroxycobalamin and sodium thiosulfate
Dehydration	Signs of volume down status	Electrolyte abnormalities, concentrated urine, increased Cr in severe cases	Oral or IV rehydration solution
Viral gastroenteritis	Watery diarrhea, abdominal cramping	Not applicable (negative COHb)	Supportive care
Food poisoning	Primarily vomiting with abdominal pain and occasionally fever	Stool culture in severe cases where the specific organism is needed to tailor treatment	Supportive care Antibiotics when indicated
Colic	Tight abdomen, hip flexion when crying, facial flush	Not applicable (negative COHb)	Supportive care

COHb: carboxyhemoglobin; PCR: polymerase chain reaction; NSAIDs: nonsteroidal anti-inflammatory drugs; IV: intravenous; Cr: creatinine

further compromising the oxygen-carrying capacity of hemoglobin.[4,15]

Management

The mainstay of treatment in CO poisoning is the administration of oxygen. One hundred percent oxygen should be provided as soon as possible to the suspected CO-poisoned patient via non-rebreather face mask or endotracheal tube. High concentrations of inhaled oxygen serve to facilitate the dissociation of CO from hemoglobin. The administration of 100% normobaric oxygen reduces the half-life of COHb to 30 to 90 minutes, compared to three to four hours while breathing room air.[15] Although formal guidelines do not exist regarding the termination of treatment, normobaric oxygen therapy should continue until the COHb level has decreased to less than 5% to 10%.[13,15] Additional sources suggest that, given its safety and availability, oxygen should be continued until symptoms resolve.[34]

Hypotension should be treated with intravenous fluids and inotropic support as needed for persistent myocardial depression.[4] In the case of life-threatening dysrhythmias, standard advanced cardiac life support (ACLS) protocols should be followed. Bicarbonate infusion should be avoided for correction of acidemia, since it will further drive the oxyhemoglobin-dissociation curve left, worsening tissue hypoxia.[4]

Although the role of HBOT remains controversial, it should be considered in acute, severe CO poisoning or pregnant patients. Although treatment guidelines are not universally established, HBOT typically is defined as administration of 100% oxygen within a hyperbaric chamber to at least 1.4 atm of absolute pressure.[35] Typical HBOT treatment protocols for CO poisoning use 100% oxygen at 2-3 atm for 120-150 minutes per session. At a partial pressure of 2.5 to 3.0 atm, blood arterial oxygen levels can surpass 1,800 mmHg.[15] The increased levels of dissolved oxygen obtained with HBOT drive COHb dissociation, decreasing the elimination half-life of COHb to approximately 20 minutes.[4] In addition, HBOT reduces CO binding to other heme-containing proteins (such as cytochrome oxidase), prevents lipid peroxidation, and decreases free radical-mediated oxidative damage.[14]

Among the published randomized controlled trials on HBOT, the data are conflicting. A Cochrane review of six trials that included two published only in abstract form did not show any support for HBOT in patients with CO poisoning.[36] However, because of the heterogeneous nature of the trials' methods regarding patient selection, treatment, and outcome measures, the review's conclusions have been challenged.

Conversely, there is one study from the available data from randomized controlled clinical trials on HBOT that fulfills all of the Consolidated Standards for the Reporting of Trials

Table 5. Suggested Indications for HBOT

Strongly Suggested Indications	Relative Indications
• Coma • Seizure • Focal neurological deficits • COHb > 25% • Fetal distress/COHb > 15% in pregnancy • Cardiac ischemia • Prolonged exposure > 24 hours	• Syncope • Altered mental status • Hemodynamic instability • Significant metabolic acidosis (pH < 7.2) • Abnormal cerebellar function • Extremes of age • Equivocal cases > 35 years old

COHb: carboxyhemoglobin; HBOT: hyperbaric oxygen therapy

(CONSORT) guidelines, including double-blinding, enrollment of all available subjects, and a priori definition of outcomes, and high rates of follow-up.[16] It is a single-center, prospective trial that showed that the incidence of cognitive sequelae was lower among patients who had three hyperbaric oxygen sessions within 24 hours compared to those who were treated with normobaric oxygen (25% vs. 46%; P = 0.007). In addition, the rate of cognitive sequelae at 12 months was substantially reduced in patients who received HBOT compared to patients treated with normobaric oxygen (18% vs. 33%; P = 0.04).[16]

Unfortunately, normobaric oxygen does not appear to offer protection against DNS since it does not reduce cognitive sequelae when compared to patients receiving no supplemental oxygen therapy.[37]

The American College of Emergency Medicine (ACEP) published guidance on HBOT in October 2016 via the Clinical Policy Subcommittee recommendations. The policy emphasizes that while HBOT is a therapeutic option for CO-poisoned patients, its use cannot be mandated given the lack of high-quality evidence. They provide Level B recommendation for HBOT or high-flow normobaric therapy for acute CO-poisoned patients but exclude out-of-hospital emergency care patients, pediatric populations, pregnant patients and fetal exposures, patients with chronic CO poisoning, or those with delayed presentations (more than 24 hours after the end of exposure) from their recommendations.[38] They also state that given the lack of well-designed clinical trials on HBOT, it still is unclear whether HBOT is superior to normobaric oxygen for improving long-term neurocognitive sequelae.[38] These recommendations aim to support the emergency physicians who choose not to refer patients for HBOT, especially when time, financial, or geographic constraints exist.[38] HBOT itself is also not without risk, and possible side effects include decompression sickness, sinus and middle ear barotrauma, seizure, and gas embolism.[15]

In the absence of any universally accepted indications, patients with evidence of end-organ damage, such as syncope, cardiac ischemia, seizure, coma, altered mental status, significant metabolic acidosis, COHb level greater than 25%, and pregnant patients with COHb levels greater than 15% should be considered for HBOT.[29] (*See Table 5.*) The optimal dose and frequency of HBOT therapy has not been established, and ultimately, more randomized controlled trials are needed to confirm efficacy. It is recommended that consultation be sought from a toxicologist or expert in hyperbarics when determining whether HBOT is appropriate therapy.

Additional Aspects

As discussed previously, the risk of neurological and psychiatric sequelae is well characterized in CO poisoning and poses the most serious risks to poisoning victims. However, several additional complications have been associated with acute exposure. A large study from Korea found a significantly increased risk for deep venous thrombosis (DVT) and pulmonary embolism (PE) for 90 days following CO poisoning.[39] The risk was highest in the first month after exposure, with patients being 22 times more likely to experience PEs and 10 times more likely to have DVTs.[39] Accordingly, it is imperative that providers educate patients on the signs and symptoms of blood clots as part of standard CO poisoning return precautions.

CO poisoning also may put patients at an increased risk for stroke compared to age-matched controls for seven years after exposure.[39] The risk of ischemic, hemorrhagic, and subarachnoid hemorrhagic strokes is increased compared to the general population, but ischemic strokes are the most frequent type of cardiovascular accident after CO poisoning.[39] Much like the risk of major clotting events like DVT and PE, the risk of stroke decreases with time after poisoning, with highest risk in the first two years.[39]

Interestingly, evidence also exists for an increased risk for the development of hypothyroidism after CO poisoning, with this risk greatest in the first month after exposure and remaining elevated for at least four years post-exposure compared to the general population.[40] The mechanism for this effect is unclear but has been postulated to be secondary to inflammatory damage, the effects of exogenous CO on the endocrine system's own CO receptors, or autoimmunity because of the association of new-onset hypothyroidism with diabetes mellitus comorbidity.[40]

Ultimately, prevention is the mainstay of addressing CO poisoning as a public health concern. Public education programs have focused on increasing awareness of CO poisoning risks and mitigation, specifically on fuels and devices that emit large amounts of CO. For instance, specific pictogram warning labels were implanted on charcoal briquette packaging to warn against indoor use. Subsequent data showed a 2.5-fold decrease in number of deaths from CO poisoning each year from 1998 to 2007, compared to 1981 to 1997.[29] The U.S. CDC strongly recommend installation of CO residential alarms to aid in early identification of occult indoor CO exposures.[41] These alarms should be located in hallways outside sleeping areas and may be installed at any height since CO diffuses

readily throughout an enclosed space. Importantly, these devices should be checked regularly, such as with daylight saving time, to ensure they are functioning appropriately and do not need battery replacement.

Disposition and Prognosis

Many patients with mild CO poisoning can be treated with normobaric oxygen in the ED and discharged home once symptoms resolve and a safe environment after discharge is confirmed. If lingering questions persist on safety of the home environment, the patient should be admitted until further investigation can be completed.

Patients with moderate to severe CO poisoning, such as altered mental status, syncope, cardiac ischemia, and dysrhythmias, should be strongly considered for admission. These patients may need further evaluation and monitoring for cardiac and neurologic toxicity due to CO. Other patients may have extensive burns that complicate care or have underlying medical conditions such as coronary artery disease, congestive heart failure, or chronic obstructive pulmonary disease, which may require further specialty consultation and care.

More objective data would benefit decision-makers in equivocal cases, like independent abnormal laboratory values or unusual presentations, but these have yet to be defined. Since strong recommendations for hospitalization criteria do not exist, serum lactate is a reasonable method to stratify risk in CO poisoning. High levels of lactate (greater than 5.7 mmol/L) have a particularly high sensitivity (89%) for predicting poor neurological outcomes.[42] In fact, elevated lactate levels (greater than 1.2 mmol/L) have been shown to be independent predictors of hospitalization in CO poisoning and correlate more closely with elevated troponin-I than COHb does.[42] Perhaps most importantly, lactate is a widely available test in the ED and can be routinely sent in all CO-poisoned patients.

Use of HBOT remains somewhat controversial in the management of CO poisoning because of the lack of clear consensus on clinical variables, including COHb levels that might benefit from HBOT. While the Undersea and Hyperbaric Medical society encourages consideration of HBOT for pregnant patients with COHb greater than 15%, ACEP provides no formal guidance of this population, as described earlier.[15,38] This lack of formal guidance likely is due to conflicting data and the frequent challenges in the initiation of HBOT, including transport, stabilization, and accessibility of HBOT-equipped facilities. In the case of an unstable patient, an individual risk-benefit analysis must be performed when considering interhospital transport. In addition, some facilities may only have access to a monospace hyperbaric chamber, which limits care to the actively unstable patient who might otherwise require frequent interventions.[14]

Patients presenting in the setting of an intentional CO poisoning are at increased risk of death due to suicide.[43] These patients should be admitted for emergent psychiatric evaluation with close monitoring. All patients should follow up after discharge with their primary doctor or appropriate specialist, since the extent and rate of recovery from CO poisoning can be variable. In general, it is difficult to estimate the nature and severity of long-term effects of CO poisoning given the lack of pre-exposure baselines for comparison. However, in one prospective study, 46% of patients initially treated with normobaric oxygen showed signs of neuropsychological sequelae at six weeks.[16] Symptoms can either persist after initial exposure or appear after a several weeks long latency period and can be permanent.[4,14] Suggested late or evolving cognitive impairments because of CO exposure include memory disturbance, depression, anxiety, vestibular problems, motor dysfunction, and inattention.[29]

Summary

CO poisoning is a frequent cause for presentation to the ED and can have significant morbidity and mortality if not promptly recognized and treated. CO toxicity lies in its high affinity to hemoglobin, which displaces oxygen and generates COHb, which impairs cellular metabolism and leads to tissue ischemia. The nonspecific presentation of CO poisoning adds to the diagnostic challenge, with symptoms such as headache, dizziness, nausea, and vomiting, which might easily be mistaken for a benign viral illness. Special attention should be paid to historical clues, such as a suicide attempt, recent inclement weather, or group presentations, which might suggest exposure to non-conventional heating sources or engine exhaust.

In patients with suspected or confirmed CO exposure, a targeted history and examination should be performed, as well as specific COHb testing via direct CO-oximetry. Treatment with 100% oxygen via non-rebreather mask should be administered as soon as possible and continued until the patient is asymptomatic or the COHb levels decrease below 5% to 10%.[15] HBOT also can further reduce the half-life of COHb and shows promising results in reducing delayed neurological sequelae in high-risk patients or exposures; however, there is no established standard of care for HBOT at this time.

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CME/CE Questions

1. A 28-year-old male who operates the ice resurfacing machine at the nearby ice skating rink presents to the emergency department complaining of a headache and presyncope. His vital signs are currently stable (blood pressure: 110/75 mmHg; heart rate: 99 beats/minute; respiratory rate: 20 breaths/minute; temperature: 37.6°C; O_2 saturation: 99%). Which of the following represents the best next step?
   a. Obtain blood gas for direct carboxyhemoglobin analysis
   b. Start administering 100% oxygen via non-rebreather face mask
   c. Obtain an electrocardiogram and consult cardiology
   d. Immediately initiate hyperbaric oxygen therapy
2. After a 50-year-old male presents with ataxia, headache, dizziness, and nausea, you diagnose carbon monoxide poisoning after blood carboxyhemoglobin testing. A history revealing which of the following professions should prompt concern for a late peak in carboxyhemoglobin level?
   a. Fisherman
   b. Painter
   c. Garage-based valet
   d. Dog walker
3. The differential diagnosis for the most common overall chief complaint in patients with carbon monoxide poisoning should include which of the following?
   a. Viral gastroenteritis
   b. Vestibular neuronitis
   c. Community-acquired pneumonia
   d. Tension headache
4. A 35-year-old is brought in by emergency medical services from home. He was asleep when his carbon monoxide alarm went off, and he still has a headache despite exiting his house hours ago. He takes imipramine for irritable bowel syndrome, drinks two standard drinks daily, and smokes half a pack per day of cigarettes and smokes marijuana occasionally. Which of the following results would be most accurate

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for diagnosis of carbon monoxide exposure?
   a. Direct CO-oximetry of 13%
   b. SpCO reading of 15%
   c. Lactate of 5.65 mmol/L
   d. Injected conjunctivae, hypoxia of 91% on pulse oximetry, and red mucous membranes

5. Which of the following provides the largest contribution to the incidence of carbon monoxide poisoning in U.S. emergency departments?
   a. Mass exposures
   b. Suicide attempts
   c. Malfunctioning furnaces
   d. Vehicle-related exposures (> 50% of deaths, not cases)

6. A 40-year-old female with a past medical history of asthma, migraines without aura, and a 10-year smoking history is brought to the emergency department for evaluation of cyanide and carbon monoxide poisoning after escaping a house fire. Which of the following adjunct treatments should be considered in this patient with carbon monoxide poisoning?
   a. Therapeutic hypothermia
   b. Hydroxocobalamin and sodium thiosulfate
   c. Allopurinol
   d. Sodium nitrite

7. How frequently do delayed neurocognitive sequelae occur after acute, symptomatic carbon monoxide poisoning?
   a. Always
   b. Almost never
   c. In about half of cases
   d. In more than 90% of cases

8. Which of the following medications is contraindicated during the treatment and resuscitation of patients without comorbidities in the setting of severe carbon monoxide poisoning with hypotension, a recent arterial pH of 7.21, and recent runs of ventricular tachycardia seen on telemetry?
   a. Epinephrine
   b. Dobutamine
   c. Sodium bicarbonate
   d. Milrinone

9. During discussion about return precautions, carbon monoxide-poisoned individuals should be advised to look for symptoms of which of the following conditions in particular on discharge because of their increased risk?
   a. Retinal detachment, carotid dissection, sensorineural hearing loss
   b. Deep vein thrombosis, stroke, and hypothyroidism
   c. Orthostatic hypotension, cord compression, neurogenic bladder
   d. Aortic aneurysm, heart block, stroke

10. Which of the following patients might be considered for hyperbaric oxygen therapy after discussion with your local toxicologist or hyperbaric expert?
   a. A 26-year-old male with palpitations
   b. A 52-year-old female with chest pain and negative troponin result
   c. A 32-year-old pregnant female with COHb 20% on CO-oximetry
   d. A 45-year-old male with fatigue and normal neurologic exam

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Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

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Carbon Monoxide Exposure: Evaluation and Management

Sources Implicated in Carbon Monoxide Poisoning

Man-Made	Metabolic Exposure	Natural Sources
• Malfunctioning furnaces • Portable generators • Gas camping stoves • Charcoal briquettes • Building fires • Automobile exhaust • Boat engine exhaust • Propane-powered forklifts • Ice resurfacing machines • Kerosene heaters • Waterpipe (hookah)	• Methylene chloride • Paint stripper	• Forest fires • Volcanic eruptions

Elimination Half-Life of Carbon Monoxide

Treatment	Half-Life
• Room air • 100% normobaric oxygen • Hyperbaric oxygen therapy (2.5 atm)	• 3-4 hours • 30-90 minutes • 15-23 minutes

Clinical Signs and Symptoms of Carbon Monoxide Poisoning

Mild-Moderate	Moderate-Severe
• Headache • Fatigue • Nausea • Vomiting • Dizziness • Blurred vision • Confusion • Chest pain • Shortness of breath • Weakness	• Cardiac ischemia • Non-cardiogenic pulmonary edema • Syncope • Dysrhythmias • Syncope • Seizure • Hypotension • Altered mental status • Coma • Cardiac arrest

Differential Diagnosis

Diagnosis	Differentiating Clinical Features	Differentiating Laboratory Features	Treatment
Carbon monoxide poisoning	Headache, nausea, vomiting, lethargy, history of exposure	COHb level > 5% in nonsmokers and > 10% in smokers	100% oxygen therapy, hyperbaric oxygen
Influenza	Myalgias, fever, rhinorrhea	Positive PCR for influenza virus	Oseltamivir (early initiation), supportive care
Viral upper respiratory infection	Rhinorrhea, eye discharge, sinus/lung congestion	Respiratory viral pathogen panel (rarely indicated)	Supportive care
Tension headache	Similar headache as carbon monoxide poisoning in the absence of other symptoms	Not applicable (negative COHb)	NSAIDs, acetaminophen, caffeine, IV fluid hydration
Cyanide poisoning	Somnolence, seizures, coma, shortness of breath	Red blood cell cyanide concentration (abnormal > 2.5 mcg/mL)	Hydroxycobalamin and sodium thiosulfate
Dehydration	Signs of volume down status	Electrolyte abnormalities, concentrated urine, increased Cr in severe cases	Oral or IV rehydration solution
Viral gastroenteritis	Watery diarrhea, abdominal cramping	Not applicable (negative COHb)	Supportive care
Food poisoning	Primarily vomiting with abdominal pain and occasionally fever	Stool culture in severe cases where the specific organism is needed to tailor treatment	Supportive care Antibiotics when indicated
Colic	Tight abdomen, hip flexion when crying, facial flush	Not applicable (negative COHb)	Supportive care

COHb: carboxyhemoglobin; PCR: polymerase chain reaction; NSAIDs: nonsteroidal anti-inflammatory drugs; IV: intravenous; Cr: creatinine

Suggested Indications for HBOT	
Strongly Suggested Indications	**Relative Indications**
• Coma • Seizure • Focal neurological deficits • COHb > 25% • Fetal distress/COHb > 15% in pregnancy • Cardiac ischemia • Prolonged exposure > 24 hours	• Syncope • Altered mental status • Hemodynamic instability • Significant metabolic acidosis (pH < 7.2) • Abnormal cerebellar function • Extremes of age • Equivocal cases > 35 years old

COHb: carboxyhemoglobin; HBOT: hyperbaric oxygen therapy

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