Kentucky Poison Control Center

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Provider Update

Carbon Monoxide

Carbon monoxide (CO) is an insidiously dangerous gas, having no odor, taste, or color. CO is formed from the incomplete combustion of almost any carbon compound. Without a CO detector, it is often very difficult to know it exists in an environment until some harm to a human or animal occurs.

Sources of CO include: boilers, furnaces, generators, cars, trucks, and boats, as well as other gasoline or diesel-powered engines, gas and propane heaters, woodstoves, gas stoves, fireplaces, tobacco smoke, forklifts, and fires. Unusual sources include exposure to methylene chloride, (bubble lights, paint and varnish strippers, degreasing agents, others) which is metabolized to CO and hemolysis, with increased metabolism of hemoglobin.

The fall and winter months are especially dangerous, when the outside temperatures drop and people want to be warm inside their homes. Faulty furnaces and power outages lead people to use generators and gas or propane heaters inside, and as a result, account for increased morbidity and mortality associated with CO exposure. Running a car inside a garage attached to the home is another common source of CO exposure during the colder months of the year. CO poisoning has occurred from running cars while working on them in a garage with inadequate ventilation too. Cracking the garage door at the bottom doesn't allow for CO, which is lighter than air, to escape, and the resulting build up of the gas causes significant morbidity.

The Centers for Disease Control (CDC), through the National Environmental Public Health Tracking Network, reported 1,055 accidental deaths due to CO during the years 2018-2022 across the United States. The state of Kentucky had 16 deaths reported during those years.

Carbon monoxide is rapidly absorbed after inhaling it, and it displaces oxygen on hemoglobin since it has a higher affinity for hemoglobin than oxygen does. There is decreased oxygen delivery to the body as a result. Symptoms associated with breathing in low levels of CO gas are often non-specific, such as headache, nausea, vomiting, and dizziness. These symptoms may be misdiagnosed as influenza, food poisoning, gastroenteritis, or colic in infants. As levels of CO rise, so do the symptoms associated with it. Higher levels may produce cardiac dysrhythmias, myocardial ischemia, chest pain, syncope, confusion, ataxia, tachypnea, weakness, seizures, coma, and even death.

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ALERT ALERT ALERT
Outbreak of Infant Botulism linked to ByHeart infant formula

On November 7, 2025, the Centers for Disease Control (CDC) was notified from the California Department of Public Health's Infant Botulism Treatment and Prevention Program (IBTPP) of a concerning increase in infant botulism in infants consuming ByHeart infant formula. By December 10, 2025 a total of 51 infants across 19 states have been hospitalized and treated with BabyBIG. No deaths have been reported to date.

Clinical signs of infant botulism from Clostridium botulinum neurotoxin include constipation, weakness (notably of gag, cry, suck and swallow), loss of muscle tone, and ultimately, flaccid paralysis. Affected infants have difficulty feeding and often, breathing. In the absence of complications complete recovery from the infection occurs.

BabyBIG, [Botulism Immune Globulin Intravenous (Human)] is indicated in the treatment of infants less than 1 year of age that have been diagnosed with botulism. BabyBIG, approved by the FDA in 2003, is the only botulism immune globulin approved for use in infants.

If a physician has a sick infant and suspects infant botulism as the etiology, there is a 24/7/365 hotline that can be consulted at (510) 231-7600. The on-call physician will determine if BabyBIG is warranted, and the drug can be ordered from an invoice through the IBTPP website. The cost to treat an infant with botulism is currently $69,300.

As a side note, the discovery of infant botulism occurred in 1976. 2026 now marks the 50th year since identification of Clostridium botulinum neurotoxin infection in infants.

References:

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History of Carbon Monoxide Poisoning

Carbon monoxide (CO) is a byproduct of the incomplete combustion of carbon-containing materials. It is a colorless, odorless, and tasteless gas, making exposure difficult to detect and particularly dangerous. Hemoglobin binds carbon monoxide with an affinity approximately 240 times greater than oxygen, significantly impairing oxygen delivery. Additionally, myoglobin binds CO with roughly 60 times greater affinity than oxygen, contributing to myocardial depression and hypotension. Clinical manifestations of CO poisoning commonly include headache, nausea, dizziness, dyspnea, tachycardia, and vomiting. Notably, up to 50% of patients with symptomatic exposure may develop long-term complications, including delayed neurologic or neurocognitive sequelae. [1,2]

The first published investigation of carbon monoxide poisoning appeared in 1906 and focused on hematologic changes observed in guinea pigs. This study demonstrated that CO disrupts cellular metabolism by preventing adequate oxygen delivery. In 1916, one of the earliest descriptions of CO poisoning in humans was published, along with initial therapeutic recommendations. The article highlighted occupational exposures in military mining operations, where incomplete detonation of explosives produced high concentrations of CO in confined spaces. Treatment recommendations included administration of oxygen, artificial respiration, and, in some cases, the use of coffee as a stimulant. [1]

Between 1945 and 1980, widespread replacement of residential coal use with natural gas led to a decline in carbon monoxide poisoning rates. During this period, antidepressants and tranquilizers were also introduced, complicating the clinical recognition of CO exposure. In 1970, the Dräger test tube—an early device capable of measuring CO concentration in exhaled breath—was developed. Around this time, long-term psychiatric sequelae associated with CO poisoning were first described. In the late 1970s and early 1980s, public health campaigns were launched to improve recognition of CO poisoning due to high rates of misdiagnosis. These efforts reduced misdiagnosis rates from approximately 30% in the 1970s to 5% by 1980. [1]

Despite advances in detection and prevention, carbon monoxide poisoning remains the leading cause of poisoning-related morbidity and mortality in the United States. In 2022, approximately 1,244 deaths were attributed to CO poisoning, including 624 unintentional fatalities. Annually, an average of 20,636 unintentional, non–fire-related CO exposures are reported in the U.S. Over 40% of these exposures occur during winter months, with approximately 75% taking place in private residences. One-third of unintentional CO-related deaths involve the use of charcoal, wood, or natural gas for heating or cooking. Furthermore, despite emission controls such as catalytic converters, more than half of unintentional CO deaths are associated with motor vehicle exhaust, including exposures involving vehicle occupants, pickup truck beds, boats, and the indoor operation of propane-powered equipment such as ice resurfacers and forklifts.[2,3]

In summary, carbon monoxide is a highly lethal yet often undetectable gas. Although significant progress has been made in prevention through cleaner fuels and improved emission controls, CO poisoning continues to pose a major public health risk, particularly during winter months. Early recognition and prevention remain critical to reducing morbidity and mortality associated with carbon monoxide exposure.

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Clinical Management of Carbon Monoxide

Pathophysiology

CO toxicity is multifactorial. While hemoglobin has approximately 200–250 times greater affinity for CO than oxygen, clinical severity correlates poorly with carboxyhemoglobin (COHgb) levels alone. [3]

CO causes toxicity via:

1. Formation of COHgb → impaired oxygen delivery
2. Leftward shift of the oxyhemoglobin dissociation curve → impaired oxygen unloading
3. Binding to myoglobin → myocardial depression
4. Inhibition of mitochondrial cytochrome oxidase → cellular hypoxia
5. Oxidative stress and inflammatory cascades → delayed neurologic injury [3,4]

Importantly, PaO_2 is typically normal because dissolved oxygen remains unaffected. Smokers may have baseline COHgb levels of 5–10%, whereas nonsmokers typically range from 0–3%. Levels >10% are generally considered abnormal; however, treatment decisions should be clinical, not numeric.

Pharmacokinetics and Oxygen Therapy

CO equilibrates over approximately 4 hours. The elimination half-life CO depends on inspired oxygen concentration:

- Room air (21% FiO_2): ~5–6 hours
- 100% oxygen via non-rebreather: ~60–90 minutes
- Hyperbaric oxygen (HBO) at 2.5 ATA: ~20–30 minutes [3]

Immediate administration of 100% oxygen is the cornerstone of therapy for all suspected cases, regardless of COHgb level. This reduces half-life and improves tissue oxygenation by increasing dissolved oxygen content. [3]

Indications for Hyperbaric Oxygen

HBO remains controversial but is recommended in selected high-risk patients. The strongest randomized trial (Weaver et al., NEJM 2002) demonstrated reduced incidence of cognitive sequelae at 6 weeks in patients treated with HBO.[5] UofL Jewish Hospital and the University of Kentucky are the two hyperbaric chamber facilities in Kentucky that are open 24/7. Propofol can be used for sedation, but we would recommend compounding it in a non-glass Viaflex bag and use the appropriate tubing. Dexamethasone can also be considered, but we recommend consulting with the Kentucky Poison Center (KPC) to discuss nuances. Criteria for use of HBO can be found in the following article.

Keep in mind that pregnancy warrants special attention because fetal hemoglobin has even greater affinity for CO, and fetal elimination is prolonged. [4]

Delayed Neurologic Sequelae

DNS may occur days to weeks after exposue and include memory impairment, personality changes, gait disturbance, and executive dysfunction. Incidence ranges from 10–40%. [4,5] Risk factors include older age, prolonged exposure, and loss of consciousness. ED physicians should counsel patients and arrange follow-up for neurologic reassessment within 2–4 weeks if symptomatic.

Special Consideration: Methylene Chloride

Methylene chloride (paint strippers) is hepatically metabolized via CYP2E1 to CO and can produce delayed and prolonged COHgb elevation. Its elimination half-life (~13 hours) is substantially longer than inhaled CO, requiring extended oxygen therapy and observation. [3]

Practical ED Approach

1. Administer 100% oxygen immediately.
2. Obtain COHgb level (venous sample acceptable).
3. Perform ECG and troponin in moderate–severe cases.
4. Assess acid-base status and lactate.
5. Consult hyperbaric center early for high-risk features.
6. Provide clear discharge instructions regarding delayed symptoms.

COHgb level does not reliably predict clinical severity. Management decisions should prioritize symptoms, comorbidities, and exposure history over absolute numbers.

For consultation, contact the Kentucky Poison Center 24/7 at 1-800-222-1222.

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Using Hyperbaric Oxygen for CO Poisoning

Hyperbaric oxygen (HBO) treatment is often recommended for significant carbon monoxide (CO) exposures. 100% oxygen at ambient pressure reduces the half-life of CO from 320 minutes to 85 minutes, but at 2.5 atmospheres absolute, it is reduced to 20 minutes. HBO increases the amount of dissolved oxygen in the body by a factor of 10, ensuring adequate supply of metabolic needs in the absence of hemoglobin. HBO has been shown in animal models to prevent brain lipid peroxidation. The implication for humans is that HBO therapy is a valid modality to help protect against neurological injury from CO poisoning if delivered early enough after an exposure.

The optimal timing for HBO and number of treatments needed is still unclear, but treating patients within 24 hours of exposure, administrating 3 treatments has been shown to improve cognitive outcomes. The earlier in the poisoning that HBO is used, the better the outcome for the patient in most all cases.
Table 1 (below) shows criteria for HBO therapy.

Table 1: Recommended Indications for Hyperbaric Oxygen Therapy

Syncope (transient loss of consciousness)
Coma
Seizure
Altered mental status (GCS < 15) or confusion
Carboxyhemoglobin > 25%; independent of signs/symptoms
Abnormal cerebellar function
Pregnancy with CO> 15% and/or fetal distress
Equivocal cases with age > 35 years and prolonged exposure (> 24 hr's)

There are special considerations for the treatment of pregnant women and children with HBO. Pregnant females are excluded from trials, so information regarding efficacy is drawn from actual exposures and subsequent treatment and outcome. The absorption and elimination of CO is slower in the fetal circulation than in maternal circulation. There is also a precipitous drop in fetal arterial oxygen content when the mother has been poisoned with CO. The threshold for use of HBO in these patients is lower than a non-pregnant person, as indicated in the table. Fetal distress may include an increased heart rate.

Children appear to be more sensitive to the effects of CO at levels lower than that of adults, placing them at higher risk for poisoning. Children generally have increased respiratory and metabolic rates. Younger children may have unusual presentation, sometimes having just vomiting or an isolated seizure. Most children will show nausea, lethargy, or headache following a CO exposure. The presence of fetal hemoglobin or increased bilirubin may lead to falsely elevated CO readings on lab instrumentation. It is recommended to look at lactate as a reliable marker in children.

Consult the local poison center (800) 222-1222 for additional assistance in these cases.

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