

## Carbon Monoxide Poisoning

### Introduction

Carbon monoxide (CO) is an odorless, colorless, nonirritating gas. Toxicity can result from inhalation of excessive amounts of CO, typically produced by incomplete combustion of fossil fuels.<sup>1,3</sup> It is the leading cause of death due to poisoning in the United States. An estimated 10,000 people receive medical attention for this poisoning each year.<sup>2</sup> There are probably thousands of cases more that are never identified or misdiagnosed.

Common sources of CO include car and boat exhaust fumes, smoke from fire, defective or poorly ventilated furnaces, gas powered engine exhaust, home water heaters, or wood stoves. Motor vehicle exhaust is the most common source of fatal CO intoxication, with most deaths associated with stationary vehicles. Deaths in garages frequently occur even with garage doors or windows open for ventilation. Smoke from fire is the second leading cause of CO poisoning and is the most common cause of death in building fires. Many cases of poisoning occur in the winter months. Car exhausts may become blocked with snow and more non-electric heating and cooking devices are used in confined spaces, particularly during winter power outages.

Exposure to methylene chloride, a common ingredient in paint remover, can also cause CO poisoning. It is absorbed through inhalation, ingestion, or dermal contact, and is converted to CO by the liver.

### Pathophysiology

An important mechanism of CO toxicity is displacement of oxygen from hemoglobin binding sites. CO binds to hemoglobin with an affinity 200-300 times that of oxygen. The reduction in blood oxygen content and oxygen delivery to the tissues is the major consequence of CO poisoning. CO binding to hemoglobin also shifts the oxygen-hemoglobin dissociation curve to the left. This decreases oxygen release to the tissues, further contributing to tissue hypoxia. Myocardial depression, peripheral vasodilatation, and ventricular dysrhythmia caused by tissue hypoxia can lead to hypotension, decreased tissue perfusion and further tissue hypoxia.

Other mechanisms of toxicity include decreased cellular respiration due to CO binding with cytochrome a<sup>3</sup>, and myocardial and skeletal dysfunction due to CO binding with myoglobin.

### Clinical Presentation

The acute signs and symptoms that may occur following CO poisoning are shown in Table 1.<sup>1,3</sup> Longer exposures and higher concentrations of CO typically result in more severe symptoms, although there are no guidelines predicting outcome based on duration of exposure.<sup>1</sup> There is some correlation between symptom severity and the peak carboxyhemoglobin (COHb) concentration.<sup>3</sup> Symptoms generally do not occur until COHb concentrations exceed 10%. As the blood concentration increases above 20%, headache, nausea, dizziness, and confusion may develop. COHb concentrations greater than 40% are frequently associated with coma and seizures. COHb concentrations above 60% are often fatal. However, one should not rely on COHb concentrations to predict injury or outcome. Metabolic acidosis is a more reliable prognostic indicator of serious sequelae than COHb concentration.<sup>2</sup>

The magnitude of clinical effects will vary depending on the time since exposure and pre-hospital treatment. Clinical presentation is extremely variable among patients.<sup>1</sup>

Mild cases of CO poisoning may result in respiratory alkalosis, as the respiratory system tries to compensate for decreased oxygen delivery. More severe exposures may lead to metabolic acidosis as lactic acid is produced in hypoxic tissue. Death from acute CO poisoning is usually due to ventricular dysrhythmias secondary to hypoxia.<sup>3</sup>

Neurologic injury following acute CO poisoning may be delayed for several weeks to months after exposure. A wide range of neurologic abnormalities has been reported (Table 2). It is estimated that up to 10% of CO poisoning survivors have persistent gross neuropsychiatric impairment, and a much larger percentage suffer more subtle effects such as personality changes and memory impairment.<sup>3</sup>

**Table 1: Signs and Symptoms of Acute CO Poisoning<sup>1,2,3</sup>**

Severity	Symptoms	Signs
Mild	Asymptomatic Headache Nausea Dizziness	Vomiting
Moderate	Confusion Chest pain Dyspnea Weakness	Tachypnea Tachycardia Cognitive deficits Ataxia <u>Myonecrosis</u>
Severe	Blurred vision Chest pain Heart palpitations Disorientation	Coma Seizures Ventricular dysrhythmias Hypotension Myocardial ischemia

Adapted from Tomaszewski C

**Table 2**

Neurologic impairments from acute CO poisoning
Parkinsonism
Persistent vegetative state
Akinetic mutism
Agnosia
Apraxia
Visual impairment
Amnesic/confabulatory state
Psychosis
Cognitive impairment

## Diagnosis

The most useful diagnostic test following suspected CO poisoning is a blood carboxyhemoglobin (COHb) concentration. This may be measured in either venous or arterial blood. Venous blood concentrations correlate well with arterial COHb concentrations.<sup>4</sup> Carbon monoxide is naturally found in the body as a product of hemoglobin breakdown, therefore normal COHb concentrations can range from 0% to 5%. Smokers have a baseline COHb concentration of up to 10%, with one-pack-per-day smokers averaging a COHb concentration of 6%. COHb concentrations greater than 10% indicate a recent exposure and the need for clinical intervention.<sup>2</sup> CO concentrations may not be a reliable prognostic indicator of either symptoms or outcome. However, they are useful in confirming exposure to excess CO, particularly if taken before oxygen therapy is initiated. Arterial blood gas and basic metabolic panels are used to confirm metabolic acidosis.

The effects of CO are non-specific and may mimic other diseases or conditions making the diagnosis of CO poisoning difficult without a history of an exposure. Patients are commonly misdiagnosed with influenza because symptoms of CO poisoning include headache, nausea, and dizziness.<sup>3</sup> Other common misdiagnoses include gastroenteritis and food poisoning.<sup>2</sup>

CO poisoning should be considered in the differential diagnosis of unexplained headaches, since headaches are the

most common initial symptom.<sup>1,5</sup> A recent study indicates that there are no distinguishing features specific to CO headaches. Headaches associated with CO poisoning are extremely variable in presentation and no specific headache trait aids in the diagnosis of CO poisoning.<sup>5</sup> General feelings of malaise, fatigue, nausea, vomiting, or multiple simultaneous cases should also raise suspicion of CO poisoning. As opposed to viral illnesses, the onset of symptoms for victims of CO poisoning in the same location (e.g. household) occur in close temporal proximity to each other. Patients who may have attempted suicide and present with coma, acidosis, or signs of cardiac ischemia should be screened for CO poisoning as well.<sup>2</sup>

## Treatment

Supplemental oxygen is the most important treatment for CO poisoning.<sup>1,2</sup> High-flow oxygen by nonrebreather face mask or endotracheal tube for several hours should immediately be provided to patients with a strong history or symptoms suggestive of CO exposure.<sup>6</sup> Oxygen administration aids in the dissociation of CO from hemoglobin, reducing the average half-life of COHb from five hours to about 90 minutes.<sup>2</sup>

Treatment decisions should not be made based solely on COHb concentrations, since they do not reliably correlate with degree of injury. Patients should be closely observed and treated with 100% oxygen until all symptoms resolve and the COHb concentration is below 5%.<sup>3</sup> Cardiac monitoring and IV access should be instituted in patients with serious poisoning. Hypotension can be treated with IV fluids, and myocardial depression with inotropic agents. Advanced Cardiac Life Support protocols should be followed for life-threatening dysrhythmias.<sup>2</sup>

Hyperbaric oxygen at 2.5 atmospheres of pressure decreases the half-life of COHb relative to 100% oxygen at normal atmospheric pressure, from about 90 minutes to 20 minutes.<sup>2</sup> It also increases the amount of dissolved oxygen in the blood by about ten fold.<sup>2</sup> Hyperbaric oxygen therapy is considered to be relatively safe.<sup>2</sup> There is a small risk of seizures from central nervous system oxygen toxicity and middle-ear barotrauma may occasionally occur.<sup>8,9</sup> The distance to the nearest hyperbaric-equipped treatment center may be a barrier to the use of hyperbaric oxygen therapy in some areas, and risks of transport to a treatment center must be considered.

The use of hyperbaric oxygen is controversial.<sup>3,7</sup> Some practitioners consider it to be the treatment of choice rather than normobaric oxygen in serious CO poisoning in order to decrease the risk of delayed neurologic sequelae.<sup>2</sup> Controlled-trials have produced conflicting evidence as to the effectiveness of hyperbaric oxygen for preventing neurologic sequelae.<sup>1,3,8</sup> In a recently reported randomized

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control trial, the authors concluded that hyperbaric oxygen did significantly reduce the incidence of delayed neuropsychiatric effects compared with normobaric oxygen.<sup>8</sup>

Indications for hyperbaric oxygen therapy are listed in Table 4. Some practitioners have advocated hyperbaric oxygen therapy for COHb concentrations above 40%, while others more conservatively recommend to treat concentrations above 25%.<sup>2</sup>

**Table 4**

Indications for Hyperbaric Oxygen Treatment
-Loss of consciousness
-Seizures
-Coma
-Altered mental status
-Neurologic symptoms not resolving after several hours of oxygen treatment
-Pregnancy
-Persistent cardiac ischemia
-Age > 50 years and COHb > 25%

## Prevention

CO poisoning is largely preventable. Table 5 lists some recommendations from the CDC for preventing CO exposure.

**Table 5: Preventing CO poisoning<sup>10</sup>**

-Have all fuel-burning venting systems in your home checked by an expert each year
-Never use a gas range or oven for heating
-Never use a portable gas camp stove indoors
-Never cook indoors with charcoal briquettes
-Have all gas appliances checked each year
-Have car exhaust system checked each year
-Never run an automobile in a closed garage

## Summary

CO poisoning is very common and can result in permanent neurologic damage. It should be suspected in cases of unexplained headaches, especially if accompanied by nausea, malaise, or fatigue. Rapid diagnosis and treatment is essential for minimizing the likelihood of permanent sequelae. Treatment with normobaric oxygen versus hyperbaric oxygen is controversial, though recent evidence suggests that hyperbaric oxygen may more effectively decrease delayed neurologic sequelae in serious poisonings. Increased public education may reduce many preventable CO exposures.

CarrieAnn McBeth, Pharm.D.

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## Summer Update Conference

The UPCC held its Train-the-Trainer Update Conference on June 16, 2004 at the Heritage Center, University of Utah and on June 25, 2004 at the Crystal Inn in Cedar City. Conference participants included nurses, pharmacists, health educators, and law enforcement. The Salt Lake conference had 52 attendees and Cedar City had 11 attendees. Topics presented at the conference included a general poison education update, carbon monoxide, toxic native plants, bites and stings, and inhalants and club drugs. Over 95% of the attendees rated the conference effective and useful in their jobs. The UPCC plans to offer an update conference every other year. Suggestions for future topics included pesticides, herbal drugs, elder drug misuse, and use of antidotes.

## Employment Opportunities

The dynamic health care team at the Utah Poison Control Center (UPCC) is seeking nurses with a master's degree preferred or 3 years ER/ICU experience or pharmacists with current pharmacist licensure in the state of Utah. If you have a desire to work in a challenging patient care and academic environment where you are continually learning, the UPCC is the place for you. The UPCC offers a great schedule, excellent benefits, tuition reduction and a fabulous retirement program. For more information, please visit our website at <http://uuhsc.utah.edu/poison/employment>.

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## UPCC Update

The Utah Poison Control Center had a busy spring. A grant application was submitted to the Health Resources and Services Administration (HRSA), Bureau of Maternal and Child Health for the Poison Center Stabilization Program. This application was for 3 years of funding beginning September 1, 2004. This is the second 3-year cycle for federal funding available to poison centers. Funding will be used to support on-site medical direction, quality assurance and outreach education. Outreach education objectives include development and implementation of an outreach education program for older adults, a new plant brochure and a biannual outreach education update conference.

The UPCC also submitted an application to renew its regional certification status. The application is currently under review by the certification committee of the American Association of Poison Control Centers and we anticipate renewal of our certification effective November 2004. Certification renewal occurs every five years.

A number of the UPCC staff attended the North American Congress of Clinical Toxicology Meeting September 9-14, 2004 in Seattle Washington. Three posters were selected for presentation at the meeting.

The UPCC published its 2003 Annual Report this summer, which highlights the activities for 2003. If you would like a printed copy of our annual report, please call the administrative office (801) 587-0600 or email: [poison.control@hsc.utah.edu](mailto:poison.control@hsc.utah.edu). An electronic version is available on the UPCC's website at <http://uuhsc.utah.edu/poison/> follow the links for "About Us" and "Annual Report".

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