MEDICAL EDUCATION SYSTEMS

Carbon Monoxide

Medical Education Systems, Inc.

TOLL FREE 1-877-295-4719
FAX (619) 295-0252
EMAIL: Info@mededsys.com
www.mededsys.com
P.O Box 81831 San Diego, CA 92138-3939

Carbon Monoxide Poisoning

Learning Objectives

Upon successful completion of this course, you will be able to:

- **Describe what is meant by "carbon monoxide" poisoning**
- **List the possible causes of "carbon monoxide" poisoning**
- **Identify the various treatments for "carbon monoxide" poisoning**
- **List the toxic elements of carbon monoxide**

Treatment Overview

The purpose of oxygen therapy for the treatment of carbon monoxide poisoning is to reduce the amount of carbon monoxide in the blood and restore the oxygen level to normal as quickly as possible.

For hyperbaric oxygen therapy, the affected person lies down on a stretcher that slides into an acrylic tube about 7 ft (2.1 m) long and 25 in. (64 cm) across. The pressure inside the tube is raised, and 100% oxygen is delivered under high pressure. Each treatment session lasts about 90 minutes. After treatment, the chamber is depressurized slowly while the person rests inside.

What To Expect After Treatment

A person usually recovers from carbon monoxide poisoning within a few days. However, it is important to remember that long-term effects may occur days or weeks after carbon monoxide poisoning.

Why It Is Done

Hyperbaric oxygen therapy can be used to reduce carbon monoxide levels in the blood quickly and the symptoms that go with it. The use of hyperbaric oxygen therapy is evaluated on a case-by-case basis. Factors considered include:

- The amount of carbon monoxide in the blood.
- The severity of symptoms, such as whether a person has lost consciousness or appears confused.
- A person's age, the presence of heart or brain disease, and overall health. Infants, small children, older adults, or people with health problems are more easily affected by high amounts of carbon monoxide in the blood, and their symptoms are more severe.
- Pregnancy and whether a pregnant woman has had a significant exposure to carbon monoxide.

Treatments will likely be repeated, depending on the outcome of the first treatment. To date, studies have shown benefits only from multiple treatments.[1]

How Well It Works

A recent study has concluded that three hyperbaric oxygen treatments within a 24-hour period may reduce the risk of cognitive problems, such as lasting damage to memory, attention, and concentration.[2]

In pregnant women who have been exposed to carbon monoxide, hyperbaric oxygen therapy reduces the time necessary to lower carbon monoxide levels in fetal blood, which increases the chances for a healthy baby. The fetus has a higher risk for carbon monoxide poisoning because it takes longer for carbon monoxide to be eliminated from fetal blood than from the mother's blood.[3]

Risks

Risks of hyperbaric oxygen therapy may include ear pain, rupture of the eardrum, sinus discomfort, a bloody nose, and in very rare cases, seizure or problems from too much oxygen.

What To Think About

Hyperbaric oxygen therapy chambers are located only at specialty medical centers or major hospitals.

Hyperbaric oxygen chambers also are used to treat people who have decompression sickness from scuba diving.

Background: Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion of carbonaceous material. Commonly overlooked or misdiagnosed, CO intoxication often presents a significant challenge, as treatment protocols, especially for hyperbaric oxygen therapy, remain controversial because of a paucity of definitive clinical studies.

CO is formed as a by-product of burning organic compounds. Although most fatalities result from fires, stoves, portable heaters, and automobile exhaust cause approximately one third of deaths. These often are associated with malfunctioning or obstructed exhaust systems and suicide attempts. Cigarette smoke is a significant source of CO. Natural gas contains no CO, but improperly vented gas water heaters, kerosene space heaters, charcoal grills, hibachis, and Sterno stoves all emit CO. Other sources of CO exposure include propane-fueled forklifts, gas-powered concrete saws, inhaling spray paint, indoor tractor pulls, and swimming behind a motorboat.

CO intoxication also occurs by inhalation of methylene chloride vapors, a volatile liquid found in degreasers, solvents, and paint removers. Dermal methylene chloride exposure may not result in significant systemic effects but can cause significant dermal burns. Liver metabolizes as much as one third of inhaled methylene chloride to CO. A significant percentage of methylene chloride is stored in the tissues, and continued release results in elevated CO levels for at least twice as long as with direct CO inhalation.

Children riding in the back of enclosed pickup trucks seem to be at particularly high risk. Industrial workers at pulp mills, steel foundries, and plants producing formaldehyde or coke are at risk for exposure, as are personnel at fire scenes and individuals working indoors with combustion engines or combustible gases.

Pathophysiology: CO toxicity causes impaired oxygen delivery and utilization at the cellular level. CO affects several different sites within the body but has its most profound impact on the organs (eg, brain, heart) with the highest oxygen requirement.

Toxicity primarily results from cellular hypoxia caused by impedance of oxygen delivery. CO reversibly binds hemoglobin, resulting in relative anemia. Because it binds hemoglobin 230-270 times more avidly than oxygen, even small concentrations can result in significant levels of carboxyhemoglobin (HbCO).

An ambient CO level of 100 ppm produces an HbCO of 16% at equilibration, which is enough to produce clinical symptoms. Binding of CO to hemoglobin causes an increased binding of oxygen molecules at the 3 other oxygen-binding sites, resulting in a leftward shift in the oxyhemoglobin dissociation curve and decreasing the availability of oxygen to the already hypoxic tissues.

CO binds to cardiac myoglobin with an even greater affinity than to hemoglobin; the resulting myocardial depression and hypotension exacerbates the tissue hypoxia. Decrease in oxygen delivery is insufficient, however, to explain the extent of the toxicity. Clinical status often does not correlate well with HbCO level, leading some to postulate an additional impairment of cellular respiration.

CO binds to cytochromes *c* and P450 but with a much lower affinity than that of oxygen; very low levels of in vitro binding result. Additionally, the patient groups exhibiting neuropsychiatric deficits often are not acutely acidotic.

Studies have indicated that CO may cause brain lipid peroxidation and leukocyte-mediated inflammatory changes in the brain, a process that may be inhibited by hyperbaric oxygen therapy. Following severe intoxication, patients display central nervous system (CNS) pathology, including white matter demyelination. This leads to edema and focal areas of necrosis, typically of the bilateral globus pallidus. Interestingly, the pallidus lesions, as well as the other lesions, are watershed area tissues with relatively low oxygen demand, suggesting elements of hypoperfusion and hypoxia.

Recent studies have demonstrated release of nitric oxide free radical (implicated in the pathophysiology of atherosclerosis) from platelet and vascular endothelium, following exposure to CO concentrations of 100 ppm.

HbCO levels often do not reflect the clinical picture, yet symptoms typically begin with headaches at levels around 10%. Levels of 50-70% may result in seizure, coma, and fatality.

CO is eliminated through the lungs. Half-life of CO at room air temperature is 3-4 hours. One hundred percent oxygen reduces the half-life to 30-90 minutes; hyperbaric oxygen at 2.5 atm with 100% oxygen reduces it to 15-23 minutes.

Frequency:

- **In the US:** Approximately 2 million death certificates are filed yearly in the United States. During the 10-year period from 1979-1988, exactly 56,133 death certificates contained codes addressing CO as a contributing cause; 25,889 (46%) were suicides, 15,523 (28%) involved burns or fires, 210 were deemed homicides, and 11,547 (21%) were categorized as unintentional. Heroin, the second leading cause of poisoning fatality, followed CO with 5948 deaths. In the same period, all other unintentional poisonings resulted in 40,424 deaths.

  Of unintentional fatalities, 57% were associated with automobile exhaust. The next leading identifiable causes are coal, wood, or kerosene stoves and fireplaces; combustion of natural gas from a pipeline; combustion of gasoline, acetylene, or utility gas; and industrial sources.

  Despite population growth and an increased number of cars, the unintentional death rate has declined by 63 deaths per year during the 10-year period from 1979-1988. This was attributed

in part to increased stringency of auto emissions standards, necessitating a longer time to accumulate a toxic level in a given space.

Increasing evidence implicates ambient urban CO levels in rates of angina, arrhythmias, and cardiac arrest. Presuming that the evidence is quantifiable and depending on the true extent, this implies a significant underreporting of CO-associated deaths.

- **Internationally:** Quantifying the global incidence of CO poisoning is impossible because of the transient duration of symptoms in mild intoxication, the ubiquitous and occult nature of exposure, and the tendency of misdiagnosis. In contrast to findings in the United States, one Australian study of suicidal poisonings indicated no decrease following significantly lowered CO emissions from 1970-1996 and revealed no difference between the HbCO levels of occupants in cars with and without catalytic converters.

Race: All ages, ethnic populations, and social groups are affected, yet particular groups are at higher risk.

- For unintentional fatalities, race-specific death rates were 20% higher for blacks.
- Conversely, intentional fatalities demonstrate that race-specific rates for blacks and other minority racial groups are 87% lower than for whites, revealing a cultural partiality to this form of suicide.
- Two recent North American studies examined the incidence of CO toxicity from indoor heating devices used during severe winter storms. Both studies identified a strong association between CO toxicity and US immigrants who are non-English speaking.

Age: Age-specific fatality rates are equivalent for individuals aged 15-74 years; rates increase for persons older than 75 years and decline for persons younger than 15 years.

- Age-adjusted fatality rates are higher in cold and mountainous locations.
- Individuals with pulmonary and cardiovascular disease tolerate CO intoxication poorly; this is particularly evident in those with chronic obstructive pulmonary disease (COPD) who have the additional concern of ventilation-perfusion abnormalities and possible respiratory depressive response to 100% oxygen therapy.
- Neonates and the in utero fetus are more vulnerable to CO toxicity because of the natural leftward shift of the dissociation curve of fetal hemoglobin, a lower baseline PaO_2, and levels of HbCO at equilibration that are 10-15% higher than maternal levels.

Clinical

History: Misdiagnosis commonly occurs because of the vagueness and broad spectrum of complaints; symptoms often are attributed to a viral illness. Specifically inquiring about possible exposures when considering the diagnosis is important. Any of the following should alert suspicion in the winter months, especially in relation to the previously named sources and when more than one patient in a

group or household presents with similar complaints. Symptoms may not correlate well with HbCO levels.

- Acute poisoning
    - Malaise, flulike symptoms, fatigue
    - Dyspnea on exertion
    - Chest pain, palpitations
    - Lethargy
    - Confusion
    - Depression
    - Impulsiveness
    - Distractibility
    - Hallucination, confabulation
    - Agitation
    - Nausea, vomiting, diarrhea
    - Abdominal pain
    - Headache, drowsiness
    - Dizziness, weakness, confusion
    - Visual disturbance, syncope, seizure
    - Fecal and urinary incontinence
    - Memory and gait disturbances
    - Bizarre neurologic symptoms, coma
- Chronic exposures also present with the above symptoms; however, they may present with loss of dentation, gradual-onset neuropsychiatric symptoms, or, simply, recent impairment of cognitive ability.

Physical: Physical examination is of limited value. Inhalation injury or burns should always alert the clinician to the possibility of CO exposure.

- Vital signs
    - Tachycardia
    - Hypertension or hypotension

    - Hyperthermia
    - Marked tachypnea (rare; severe intoxication often associated with mild or no tachypnea)
- Skin: Classic cherry red skin is rare (ie, "When you're cherry red, you're dead"); pallor is present more often.
- Ophthalmologic
    - Flame-shaped retinal hemorrhages
    - Bright red retinal veins (a sensitive early sign)
    - Papilledema
    - Homonymous hemianopsia
- Noncardiogenic pulmonary edema
- Neurologic and/or neuropsychiatric
    - Patients display memory disturbance (most common), including retrograde and anterograde amnesia with amnestic confabulatory states.
    - Patients may experience emotional lability, impaired judgment, and decreased cognitive ability.
    - Other signs include stupor, coma, gait disturbance, movement disorders, and rigidity.
    - Patients display brisk reflexes, apraxia, agnosia, tic disorders, hearing and vestibular dysfunction, blindness, and psychosis.
    - Long-term exposures or severe acute exposures frequently result in long-term neuropsychiatric sequelae. Additionally, some individuals develop delayed neuropsychiatric symptoms, often after severe intoxications associated with coma.
    - After recovery from the initial incident, patients present several days to weeks later with neuropsychiatric symptoms such as those just described. Two thirds of patients eventually recover completely.
    - MRI changes may remain long after clinical recovery. Predicting and preventing long-term complications and delayed encephalopathy have been the object of recent studies, many of which focus on the role of hyperbaric oxygen therapy.

Causes:

- Most unintentional fatalities occur in stationary vehicles from preventable causes such as malfunctioning exhaust systems, inadequately ventilated passenger compartments, operation in an enclosed space, and utilization of auxiliary fuel-burning heaters inside a car or camper.
- Most unintentional automobile-related CO deaths in garages have occurred despite open garage doors or windows, demonstrating the inadequacy of passive ventilation in such situations.

- Colorado state data revealed that sources of 1149 poisonings were residential furnaces (40%), automobile exhaust (24%), and fires (12%).
- Furnaces were determined to be the source in 46% of nonfatal CO poisonings but in only 10% of fatal poisonings. This suggests that the role of home heating appliances is prominent in the large group of underreported nonfatal exposures.
- Most developing countries utilize unvented cookstoves, burning wood, charcoal, animal dung, or agricultural waste. Studies have shown a concurrent rise in HbCO with these types of exposure in developing countries.

Differentials

Acute Respiratory Distress Syndrome
Altitude Illness - Cerebral Syndromes
Depression and Suicide
Diabetic Ketoacidosis
Encephalitis
Gastroenteritis
Headache, Cluster
Headache, Migraine
Headache, Tension
Hypothyroidism and Myxedema Coma
Labyrinthitis
Lactic Acidosis
Meningitis
Methemoglobinemia
Pediatrics, Headache
Pediatrics, Hypoglycemia
Toxicity, Alcohols
Toxicity, Narcotics

Workup

Lab Studies:

- HbCO analysis requires direct spectrophotometric measurement in specific blood gas analyzers. CO can be measured with a handheld analyzer, although less accurately.

- Elevated levels are significant; however, low levels do not rule out exposure, especially if the patient already has received 100% oxygen or if significant time has elapsed since exposure.
- Individuals who chronically smoke may have mildly elevated CO levels as high as 10%. Presence of fetal hemoglobin, as high as 30% at 3 months, may be read as an elevation of HbCO level to 7%.

- Arterial blood gas
  - PaO_2 levels should remain normal. Oxygen saturation is accurate only if directly measured but not if calculated from PaO_2, which is common in many blood gas analyzers.
  - As with pulse oximetry, estimate PCO_2 levels by subtracting the carboxyhemoglobin (HbCO) level from the calculated saturation. PCO_2 level may be normal or slightly decreased. Metabolic acidosis occurs secondary to lactic acidosis from ischemia.
- Troponin, creatinine kinase-MB fraction, myoglobin
  - Myocardial ischemia frequently is associated with CO exposure.
  - Patients with preexisting disease can experience increased exertional angina with HbCO levels of just 5-10%. At high HbCO levels, even young healthy patients develop myocardial depression.
- Creatinine kinase, urine myoglobin: Nontraumatic rhabdomyolysis can result from severe CO toxicity and can lead to acute renal failure.
- Complete blood count
  - Look for mild leukocytosis.
  - Disseminated intravascular coagulation (DIC) and thrombotic thrombocytopenic purpura (TTP) require further hematologic studies.
- Electrolytes and glucose level - Lactic acidosis, hypokalemia, and hyperglycemia with severe intoxication
- BUN and creatinine levels - Acute renal failure secondary to myoglobinuria
- Liver function tests - Mild elevation in fulminant hepatic failure
- Urinalysis - Positive for albumin and glucose in chronic intoxication
- Methemoglobin level - Included in the differential diagnosis of cyanosis with low oxygen saturation but normal PaO_2
- Toxicology screen - For instances of suicide attempt
- Ethanol level - A confounding factor of intentional and unintentional poisonings

- Cyanide level - If cyanide toxicity also is suspected (eg, industrial fire); cyanide exposure suggested by an unexplained metabolic acidosis; rapid determinations rarely are available. Smoke inhalation is the most common cause of acute cyanide poisoning.

Imaging Studies:

- Chest radiography
    - Obtain a chest radiograph with significant intoxications, pulmonary symptoms, or if hyperbaric oxygen is to be used.
    - Findings usually are normal.
    - Changes such as ground-glass appearance, perihilar haze, peribronchial cuffing, and intra-alveolar edema imply a worse prognosis than normal findings.
- CT scan
    - Obtain a CT scan of the head with severe intoxication or change in mental status that does not resolve rapidly.
    - Assess cerebral edema and focal lesions; most are typically low-density lesions of the basal ganglia.
    - Positive CT scan findings generally predict neurologic complications.
    - In one study, 53% of patients hospitalized for acute CO intoxication had abnormal CT scan findings; all of these patients had neurologic sequelae. Of those patients with negative scan results, only 11% had neurologic sequelae.
    - MRI is more accurate than CT scans for focal lesions and white matter demyelination and is often used for follow-up care.
    - Serial CT scans may be necessary, especially with mental status deterioration.
    - A recent report describes the evolution of acute hydrocephalus in a child poisoned with CO, documented by serial CT scans.

Other Tests:

- Electrocardiogram
    - Sinus tachycardia is the most common abnormality.
    - Arrhythmias may be secondary to hypoxia, ischemia, or infarction.
    - Even low HbCO levels can have severe impact on patients with cardiovascular disease.
- Neuropsychologic testing
    - Formal neuropsychologic testing of concentration, fine motor function, and problem solving consistently reveal subtle deficits in even mildly poisoned patients.

- Abridged versions of these tests, more applicable to the emergency department (ED) setting, have been developed as possible means to assess the risk of delayed neurologic sequelae, to assess the need for hyperbaric oxygen therapy, and to determine the success of hyperbaric therapy in preventing delayed sequelae.
- These tests are used in some institutions, but studies prospectively confirming the conclusions are lacking.
- Abridged tests can be performed in about 30 minutes by a well-trained examiner.
- Recent research indicates a specific link to deficits in context-aided memory; such specific testing has been proposed as a tool for measuring the severity of neurologic involvement in the ED.

Treatment

Prehospital Care:

- Promptly remove from continued exposure and immediately institute oxygen therapy with a nonrebreather mask.
- Perform intubation for the comatose patient or, if necessary, for airway protection.
- Institute cardiac monitoring. Pulse oximetry, although not useful in detecting HbCO, is still important because a low saturation causes an even greater apprehension in this setting.
- Give notification for comatose or unstable patients because rapid or direct transfer to a hyperbaric center may be indicated.
- If possible, obtain ambient CO measurements from fire department or utility company personnel, when present.
- Early blood samples may provide much more accurate correlation between HbCO and clinical status; however, do not delay oxygen administration to acquire them.
- Obtain an estimate of exposure time, if possible.
- Avoid exertion to limit tissue oxygen demand.

Emergency Department Care:

- Cardiac monitor: Sudden death has occurred in patients with severe arteriosclerotic disease at HbCO levels of only 20%.
- Pulse oximetry: HbCO absorbs light almost identically to that of oxyhemoglobin. Although a linear drop in oxyhemoglobin occurs as HbCO level rises, pulse oximetry will not reflect it.

Pulse oximetry gap, the difference between the saturation as measured by pulse oximetry and one measured directly, is equal to the HbCO level.

- Continue 100% oxygen therapy until the patient is asymptomatic and HbCO levels are below 10%. In patients with cardiovascular or pulmonary compromise, lower thresholds of 2% have been suggested.
- Calculate a gross estimate of the necessary duration of therapy using the initial level and half-life of 30-90 minutes at 100% oxygen. Complicated issues of treatment of fetomaternal poisoning are discussed in Special Concerns.
    - In uncomplicated intoxications, venous HbCO levels and oxygen therapy are likely sufficient. Evaluate patients with significant cardiovascular disease and initial HbCO levels above 15% for myocardial ischemia and infarction.
    - Consider immediate transfer of patients with levels above 40% or cardiovascular or neurologic impairment to a hyperbaric facility, if feasible. Persistent impairment after 4 hours of normobaric oxygen therapy necessitates transfer to a hyperbaric center.
- Serial neurologic examinations, including funduscopy, CT scans, and, possibly, MRI, are important in detecting the development of cerebral edema. Cerebral edema requires intracranial pressure (ICP) and invasive blood pressure monitoring to further guide therapy. Head elevation, mannitol, and moderate hyperventilation to 28-30 mm Hg PCO_2 are indicated in the initial absence of ICP monitoring. Glucocorticoids have not been proven efficacious, yet the negative aspects of their use in severe cases are limited.
    - Do not aggressively treat acidosis with a pH above 7.15 because it results in a rightward shift in the oxyhemoglobin dissociation curve, increasing tissue oxygen availability. Acidosis generally improves with oxygen therapy.
    - In patients who fail to improve clinically, consider other toxic inhalants or thermal inhalation injury. Be aware that the nitrites used in cyanide kits cause methemoglobinemia, shifting the dissociation curve leftward and further inhibiting oxygen delivery at the tissue level. Combined intoxications of cyanide and CO may be treated with sodium thiosulfate 12.5 g intravenously to prevent the leftward shift.
    - Admit patients to a monitored setting and evaluate acid-base status if HbCO levels are 30-40% or above 25% with associated symptoms.

Consultations:

- Hyperbaric oxygen therapy
    - Locate the nearest hyperbaric oxygen center by contacting the Divers Alert Network (DAN) at Duke University at (919) 684-2948.

- Hyperbaric oxygen therapy (HBO) currently rests at the center of controversy surrounding management of CO poisoning. Increased elimination of HbCO clearly occurs. Certain studies proclaim major reductions in delayed neurologic sequelae, cerebral edema, pathologic central nervous system (CNS) changes, and reduced cytochrome oxidase impairment.

- Presently, universal treatment criteria do not exist; however, a survey of directors of North American HBO facilities with 85% responding demonstrates some consensus. The most common selection criteria (regardless of HbCO level) include the following: coma (98%), transient loss of consciousness (77%), ischemic ECG changes (91%), focal neurologic deficits (94%), and abnormal neuropsychiatric testing (91%). Ninety-two percent of HBO facility directors use HBO for headache, nausea, and HbCO levels above 40%; yet only 62% have a specific minimum HbCO level in asymptomatic patients. One half of the centers place a time limit on delay of treatment in patients with transient loss of consciousness alone.

- HBO at 3 atm raises the amount of oxygen dissolved in the serum to 6.8%, enough to sustain cerebral metabolism. Elimination half-life is reduced to 15-23 minutes. Elimination half-life of CO from methylene chloride intoxication of 13 hours at room air temperature is reduced to 5.8 hours. Chambers are small monoplace hulls, allowing space for a single patient in a supine position who can be viewed through a window at the head, or they are acrylic walled and allow full visualization. Many of these monoplace chambers allow for care of critically ill patients, including intravenous lines, arterial lines, and ventilator. Others are large multiplace chambers that permit ventilation equipment and allow medical teams to accompany the patient.

- Treatment regimens usually involve 100% oxygen at 2.4-3 atm for 90-120 minutes. Re-treatment, although controversial, may be performed for acutely and chronically persistent symptoms. One study suggests that degree of acidosis can predict the need for re-treatment.

- Complications of therapy include decompression sickness, sinus and middle ear barotrauma, seizure, progression of pneumothorax to tension pneumothorax, gas embolism, reversible visual refractive changes, and complications related to transport of unstable patients.

- For treatment of complications from therapy, decongestants are useful, prophylactic myringotomy is common and a requirement for intubated patients, and chest tube placement is mandatory with pneumothorax. Exercise caution in patients who have experienced chest compressions, central venous catheterization, intubation, and positive pressure ventilation. Seizures are most often secondary to oxygen toxicity and do not mandate anticonvulsant therapy or discontinuation of HBO therapy.

- In multiplace chambers, seizure therapy consists of removing the oxygen mask. In monoplace chambers, decompression lowers oxygen concentration. It is crucial not to do this during the tonic phase of the seizure because it may cause pulmonary barotrauma secondary to gas expansion in the lungs.

- A 10-year retrospective study found that transfer to an HBO facility did not need to be delayed for concern of cardiac arrest, respiratory arrest, myocardial infarction, or

worsening mental status if they had not occurred during initial resuscitation; however, hypotension, dysrhythmia, seizure, emesis, and agitation were of concern in transit as well as in initial resuscitation.

- A portable hyperbaric chamber (Gamow Bag) has been used for several years for in-the-field treatment of high-altitude pulmonary edema. Fragility of the fabric limits pressure to 2 psi. A new portable product intended for use in traditional HBO (Chamber-lite 15) allows pressures of 15 psi (2 atm). A study demonstrated the device's safety in the laboratory, and reports of field use are pending.

Follow-up Care

Further Inpatient Care:

- Admitted patients generally require monitored settings, telemetry beds, or cardiac care unit/medical intensive care unit (CCU/MICU) beds for more severe cases.
- Patients with cerebral edema may be most appropriately treated in a neurosurgical ICU setting; this may dictate transfer to another facility. Admission to a toxicology service is helpful in these cases.

Further Outpatient Care:

- Asymptomatic patients with HbCO levels below 10% may be discharged.
- Arrange early follow-up care with a medical toxicologist or hyperbaricist experienced in CO poisoning.

Prognosis:

- Variability of clinical severity, laboratory values, and outcome limits prognostic accuracy.
- Cardiac arrest, coma, metabolic acidosis, and high HbCO levels are associated with poor outcome.
- Abnormal findings on CT scan are associated with persistent neurologic impairment.
- Neuropsychiatric testing may have prognostic efficacy in determining delayed sequelae

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Patient Education:

- Discuss the possibility of delayed neurologic complications, although they are much more common in admitted patients.
- Suggest minimizing physical activity for 2-4 weeks.

- Advise patient to stop smoking.
- For excellent patient education resources, visit eMedicine's Poisoning Center. Also, see eMedicine's patient education article Carbon Monoxide Poisoning.

Miscellaneous

Medical/Legal Pitfalls:

- Failure to accurately diagnose is the principle concern. A level of clinical aggression is necessary in patients with cardiovascular disease and pregnant patients. Explaining the possibility of delayed neurologic sequelae is important.
- Failure to transfer a person with moderate intoxication to a HBO facility may be of more concern if continued research validates such use.
- Failure to contact all parties who still may be at risk from exposure could allow for further CO toxicity.

Special Concerns:

- Fetomaternal poisoning
    - A pregnant CO-poisoned patient represents a particular quandary for the emergency physician. Although the mother may appear well with seemingly nontoxic levels, the developing fetus is at increased risk. Additionally, new research suggests a correlation between pre-term labor in the third trimester and CO exposure.
    - With a relatively small amount of scientific data support, conservative thought dictates treatment for any pregnant patient with evidence of CO exposure. CO displaces the oxygen-hemoglobin dissociation curve to the left. Fetal oxyhemoglobin dissociation curve lies further to the left than normal adult hemoglobin.
    - In the pregnant patient, a significant lag time exists for uptake and elimination of CO between the mother and fetus.
    - Fetal HbCO levels indicate little change during the first hour of maternal intoxication, yet they increase slowly over the first 24 hours. The peak actually may exceed maternal HbCO levels.
    - The fetus is particularly vulnerable with increased accumulation in fetal blood 10-15% higher than maternal blood and lower PaO_2 levels (20-30 mm Hg compared with 100 mm Hg in adults). It is important to realize that acute nonlethal maternal intoxication may result in fetal demise. After intoxication, during the washout phase at room air temperature, fetal HbCO half-life is 7-9 hours.
    - Fetal HbCO half-life with pure hyperbaric oxygen treatment is not accurately known; however, with maternal normobaric oxygen therapy, the fetal HbCO half-life can be reduced to 3-4 hours.

    - Because fetal hemoglobin constitutes 20-30% of the total at 3 months, neonates are at particularly greater risk than their infant and toddler counterparts.

- Carbon monoxide (CO) detectors: Home CO detectors with audible alarms are available. One study of 911 calls for suspected CO poisoning showed that 80% of calls for alarming detectors resulted in verifiable ambient CO levels; the mean concentration of CO was 18.6 ppm in homes tested for alarming detectors but was 96.6 ppm in those homes tested following calls for suspicious symptomatology.
- COPD: Clinician must remain aware of the potential for suppression of the respiratory drive in a chronically hypoxic patient. This should not result in the withholding of oxygen therapy in the already compromised patient.

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Carbon Monoxide Examination

Select the *best* answer to each of the following items. Mark your responses on the Answer form.

1. The purpose of oxygen therapy for the treatment of carbon monoxide poisoning is to reduce the amount of carbon monoxide in the blood and restore the oxygen level to normal as quickly as possible.

a. True
b. False

2. Hyperbaric oxygen therapy can be used to reduce carbon monoxide levels in the blood quickly and the symptoms that go with it. The use of hyperbaric oxygen therapy is evaluated on a case-by-case basis. Factors considered include:

a. the amount of carbon monoxide in the blood
b. the severity of symptoms, such as whether a person has lost consciousness or appears confused
c. A person's age, the presence of heart or brain disease, and overall health. Infants, small children, older adults, or people with health problems are more easily affected by high amounts of carbon monoxide in the blood, and their symptoms are more severe.
d. All of the above

3. Risks of hyperbaric oxygen therapy may include _____, and in very rare cases, seizure or problems from too much oxygen.

a. ear pain
b. sinus discomfort
c. a bloody nose
d. All the above

4. Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion of carbonaceous material. Commonly overlooked or misdiagnosed, CO intoxication often presents a significant challenge, as treatment protocols, especially for hyperbaric oxygen therapy, remain controversial because of a paucity of definitive clinical studies.

a. True
b. False

5. CO toxicity causes impaired oxygen delivery and utilization at the _____. CO affects several different sites within the body but has its most profound impact on the organs (eg, brain, heart) with the highest oxygen requirement.

a. surface level
b. cellular level
c. subdural level
d. None of the above

6. CO is eliminated through the lungs. Half-life of CO at room air temperature is 3-4 hours. One hundred percent oxygen reduces the half-life to _____ minutes; hyperbaric oxygen at 2.5 atm with 100% oxygen reduces it to 15-23 minutes.

a. 10-15
b. 15-25
c. 25-35
d. 30-90

7. Approximately 2 million death certificates are filed yearly in the United States. During the 10-year period from 1979-1988, exactly 56,133 death certificates contained codes addressing CO as a contributing cause; 25,889 (46%) were suicides, 15,523 (28%) involved burns or fires, 210 were deemed homicides, and 11,547 (21%) were categorized as unintentional.

a. True
b. False

8. Increasing evidence implicates ambient urban CO levels in rates of _____. Presuming that the evidence is quantifiable and depending on the true extent, this implies a significant underreporting of CO-associated deaths.

a. angina
b. arrhythmias
c. cardiac arrest
d. All of the above

9. Individuals with pulmonary and cardiovascular disease tolerate CO intoxication_____; this is particularly evident in those with chronic obstructive pulmonary disease (COPD) who have the additional concern of ventilation-perfusion abnormalities and possible respiratory depressive response to 100% oxygen therapy.

a. quite well
b. moderately
c. poorly
d. None of the above

10. After recovery from the initial incident, patients present several days to weeks later with neuropsychiatric symptoms such as those just described. _____ of patients eventually recover completely.

a. 15%
b. 25%
c. two thirds
d. all

11. Most unintentional fatalities from carbon monoxide poisoning occur in stationary vehicles from preventable causes such as _____, and utilization of auxiliary fuel-burning heaters inside a car or camper.

a. malfunctioning exhaust systems
b. inadequately ventilated passenger compartments
c. operation in an enclosed space
d. All of the above

12. If cyanide toxicity also is suspected (eg, industrial fire); cyanide exposure suggested by an unexplained metabolic acidosis; rapid determinations rarely are available. Smoke inhalation is the most common cause of acute cyanide poisoning.

a. True
b. False

13. In one study, ___% of patients hospitalized for acute CO intoxication had abnormal CT scan findings; all of these patients had neurologic sequelae. Of those patients with negative scan results, only 11% had neurologic sequelae.

a. 26
b. 37
c. 53
d. None of the above

14. HbCO absorbs light almost identically to that of oxyhemoglobin. Although a linear drop in oxyhemoglobin occurs as HbCO level rises, pulse oximetry will not reflect it. Pulse oximetry gap, the difference between the saturation as measured by pulse oximetry and one measured directly, is equal to the HbCO level.

a. True

b. False

15. Presently, universal treatment criteria _____; however, a survey of directors of North American HBO facilities with 85% responding demonstrates some consensus.

a. are well established
b. are still controversial
c. are being changed
d. do not exist

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PO BOX 81831, San Diego, CA, 92138-3939
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