Poison Facts:

Medium Chemicals: Carbon Monoxide

Properties of the Chemical

Carbon monoxide (CO) is an odorless, tasteless and colorless gas that is produced when there is incomplete combustion of carbon-containing fuels such as coal, petroleum peat and natural gas. Automobile exhaust accounts for more than half of all man-made carbon monoxide. Carbon monoxide is produced in industrial processes involving carbon-containing fuels and is found in homes with malfunctioning water heaters, furnaces or other appliances that use natural gas. Cigarette smoke may contain as much as 5 percent carbon monoxide, and secondhand smoke contains as much as 4.5 percent carbon monoxide.

Uses of the Chemical

Carbon monoxide is a reducing agent in blast furnaces and is used in the purification of metals, as well as in the production of acetic acid, formic acid, methyl formate, N, N-Dimethylformamide, acrylic acid, propanoic acid and phosgene. A large variety of chemicals, ranging from saturated hydrocarbons to oxygenated compounds such as methanol, are produced using carbon monoxide.

Absorption, Distribution, Metabolism and Excretion (ADME)

Carbon monoxide is absorbed through the lungs, and the concentration of carboxyhemoglobin will depend on the concentrations of inspired carbon monoxide, the duration of exposure, degree of pulmonary ventilation and the concentration of carboxyhemoglobin originally present. In addition to its reaction with hemoglobin, carbon monoxide combines with myoglobin, cytochromes and metalloenzymes such as cytochrome c oxidase and cytochrome P-450. The binding of carbon monoxide to hemoglobin produces carboxyhemoglobin and decreases the oxygen-carrying capacity of blood. This appears to be the principal mechanism of action underlying the induction of toxic effects of low-level carbon monoxide exposures. The precise mechanisms by which toxic effects are induced are not fully understood but likely include the induction of a hypoxic state in many tissues of diverse organ systems. COHb is fully dissociable, and, once acute exposure is terminated, the carbon monoxide will be excreted through the lungs. Only a very small amount of carbon monoxide is oxidized into carbon dioxide.

Clinical Effects of Acute Exposure

Carbon monoxide is a gas that is absorbed through the lungs. Although many organ systems are affected, including the eyes and skin, these effects are secondary to the effects of inhalation exposure. Inhalation exposures produce a variety of symptoms, depending on the concentration of CO and the duration of exposure.

- **Mild exposure:** (10 to 20 percent) Causes headache, fatigue, lightheadedness and dizziness.

- **Moderate exposure:** (20 to 40 percent) Causes severe headache, weakness, dizziness nausea, vomiting, syncope, tachycardia and tachypnea, followed by bradycardia and bradypnea, diminished vision, diminished manual dexterity, impaired judgment, confusion, drowsiness, hallucinations, tinnitus and cardiovascular toxicity.
- **Severe exposure:** (40 percent and above) Causes syncope, seizures, skin bullae, incontinence of urine and feces, dysrhythmias, cardiorespiratory depression, coma and death.

In-Field Treatment Prior to Arrival at a Health Care Facility

Move the patient to fresh air. Administer oxygen if it is available. Giving mouth-to-mouth resuscitation does not place the provider at risk of CO poisoning.

Special note to first responders:

Wear a positive-pressure Self-Contained Breathing Apparatus (SCBA).

Treatment of Exposures in a Health Care Facility

Administer 100 percent oxygen by a tight-fitting face mask to reduce the biological half-life of the CO. Use assisted ventilation as necessary. Draw a Carboxyhemoglobin level (COHb) when the patient is first seen and repeat at 2- to 4-hour intervals until patient is asymptomatic. Obtain a COHb level prior to oxygen therapy, as long as this does not delay treatment. COHb levels above 25 percent (0.25) are usually considered toxic, although COHb levels do not correlate well with clinical severity; patients may have significant toxicity with mildly elevated or even normal COHb levels, particularly if levels were obtained after oxygen therapy was initiated. Toxicity is best determined by the patient's cardiovascular and mental status, arterial pH and bicarbonate levels.

Admit all patients with neurological symptoms, chest pain, abnormal EKGs, metabolic acidosis or carboxyhemoglobin levels greater than 20 percent. Hyperbaric oxygen should be considered with certain groups of patients (i.e. severely symptomatic and/or pregnant patients).

For more poison prevention and first aid information, call the

Poison Control Center

Serving the Residents of Kansas

Toll-free Hotline

1-800-222-1222

THE UNIVERSITY OF KANSAS HOSPITAL

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