J02: Carbon Monoxide

Robert MacMillan

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Introduction

Carbon monoxide (CO) is a colourless, odourless, tasteless, non-irritating gas produced by the incomplete combustion of carbon-containing material such as gasoline, heating fuels, propane, oil, wood, and coal. Unintentional CO poisoning commonly results from inhalation of smoke from house fires, automobile exhaust, flue gas from furnaces or stoves, exhaust gas produced by outboard motors, ice-resurfacing machines, and barbecues used in poorly ventilated areas. Fatalities have been reported in those swimming near the engine exhaust outlets of boats and where gas-powered stoves and generators are misused as an indoor heat source.

Natural gas and propane do not contain CO, but can produce CO if burned without enough air. Methylene chloride, a common ingredient in paint stripper, can be metabolized to CO in the body after exposure.

Tobacco smokers have chronically elevated carboxyhemoglobin levels (see Toxic Dose).

Essentials

- Paramedics and EMRs must contact CliniCall regarding care planning for any suspected or confirmed CO poisoning.
- Remove patients to ambient air prior to assessment.
- Decontamination is not required for patients exposed to CO only. Patients who are removed from a house fire require a dry decontamination (i.e., removal of clothing) as a minimum measure before being loaded in an ambulance.
- High flow oxygen and supportive care is the treatment for all patients exposed to CO.
- ACP resources should be requested, if available, to measure carboxyhemoglobin (COHb) levels.
- Fire departments can measure CO levels inside buildings and this should be accomplished where possible.
- Standard pulse oximetry can be misleading in patients who have been exposed to CO; these devices are only able to detect oxygenated and deoxygenated hemoglobin and not any other form (such as carboxyhemoglobin or methemoglobin).

Additional Treatment Information

- Clinical effects of CO:
  - An elevated COHb level is diagnostic of exposure but may not reflect severity of poisoning or potential for development of delayed neurological sequelae. A more useful method of assessing the level of exposure *may be* to divide clinical signs into categories of severity. The category should be based on early symptoms.
    - Mild: throbbing temporal headache; dizziness; nausea and vomiting; blurred vision
    - Moderate: impaired thinking; confusion; severe headache; syncope or brief loss of consciousness; tachycardia; chest pain; dyspnea; tachypnea; weakness
    - Severe: myocardial ischemia; dysrhythmias; hypotension; cardiac arrest; respiratory failure; seizures; coma

Referral Information

Patients who have COHb levels between 3-10%, and who are symptomatic, require conveyance to hospital. Asymptomatic patients with COHb levels between 3-10% may not need conveyance, provided:

- The patient is with a responsible adult
- There is no history of ischemic heart disease
- The building is cleared of CO
- The patient is not pregnant
- There is no history of syncope

Exposures > 10% require conveyance.

NB: these criteria depend on the ability to measure COHb, which is only available to ACP/CCP units in British Columbia. Point-of-care CO-oximetry can be unreliable, and in-hospital arterial blood gas sampling can reveal significant discrepancies in the amount of COHb in a patient's blood. Paramedics and EMRs/FRs should, on the whole, be biased in favour of conveying patients with potential CO exposure to hospital for observation and additional evaluation.

General Information

- CO is readily absorbed after inhalation and crosses the placenta. The elimination half-life of CO is 4-5 hours breathing room air, 1-2 hours breathing 100% oxygen, and approximately 20 minutes with hyperbaric oxygen (2.5 atm).
- Hyperbaric oxygen (HBO) is a therapeutic option for treatment of CO poisoning. HBO produces a 10-fold increase in the amount of oxygen dissolved in blood, increases oxygen delivery to hypoxic tissues, and enhances CO elimination. HBO may also inhibit secondary cell damage (lipid peroxidation, mitochondrial dysfunction).
  - Complications of HBO therapy are infrequent. Ear and sinus pain (common) are managed with decongestants and surgical myringotomy in a small number of patients. Confinement anxiety, pulmonary barotrauma, and oxygen toxicity seizures occur rarely.
  - Despite several prospective trials examining HBO in preventing delayed neurologic sequelae, it remains unclear which patients clearly benefit from HBO or which clearly have no potential for benefit. Each patient must be assessed individually, evaluating potential benefits and risks. The following must be considered: current clinical status; time since exposure; acute vs. chronic exposure; risk of conveyance, travel time to HBO chamber; concomitant diseases; and pregnancy status. Decision may be made in consultation with the poison control centre or the hyperbaric unit.
  - Patients who **may likely** benefit from HBO treatment for CO poisoning include those with:
    - Neurologic signs: altered mental status; coma; cerebellar dysfunction; seizures
    - History of loss of consciousness
    - Pregnant patient with COHb level > 20%
  - Patients who may possibly benefit from HBO treatment for CO poisoning include those with:
    - Myocardial ischemia or cardiac dysrhythmias
    - Metabolic acidosis
    - Older patients
    - Asymptomatic patients with COHb levels > 25%
  - CO poisoning can result in permanent neurologic damage and death. CO poisoning is one of the leading causes of death worldwide; however, death is uncommon in patients who reach medical care. The major goal of treatment is prevention of delayed neurologic sequelae in survivors.
  - The mechanism of toxicity is complex and not fully understood. Toxicity is a result of hypoxia, ischemia, and direct cellular damage. CO binds to heme proteins, impairing normal oxygen function. CO-associated nitric oxide release may enhance oxidative and inflammatory injury to the brain. CO also has a direct effect on cellular respiration by inhibiting the activity of cytochrome oxidase and can provoke a metabolic acidosis.
  - Hemoglobin has an affinity for CO that is 200-250 times greater than its affinity for oxygen. COHb is formed, displacing oxygen from hemoglobin and producing a leftward shift in the oxyhemoglobin dissociation curve resulting in decreased oxygen delivery to tissues and causing hypoxia. Myoglobin is affected similarly, with its CO affinity being 60 times greater. Impaired oxygen delivery may cause myocardial ischemia, resulting in dysrhythmias and systemic hypotension.
  - The toxic dose of CO is highly variable. The degree of poisoning is dependent on the concentration of CO in the inspired air, the duration of exposure, the level of activity among those exposed, and underlying patient health. Infants, patients with pre-existing cardiovascular or lung disease, anemia, and in utero fetuses are more susceptible to CO.
  - Normal COHb blood levels in nonsmokers can be up to 2%. Smokers of 2-3 cigarette packages per day may have COHb levels as high as 7-9%.
  - Exposure to 545 ppm for 10 minutes has produced headache; inhalation of 5000 ppm for 5 minutes has been reported to be fatal.
  - Automobile exhaust may contain up to 100,000 ppm (10%) CO.

Interventions

First Responder

- Have patient(s) removed from exposure
- Decontaminate as required if fire was source of CO
  - → PR05: Patient Decontamination
- Keep the patient warm and protect from further heat loss
- Place the patient in a position of comfort, as permitted by clinical condition
- All patients should receive 100% oxygen through a non-rebreather face mask
  - → A07: Oxygen Administration
- Protect airway and assist ventilations as needed
  - → B01: Airway Management

Emergency Medical Responder – All FR interventions, plus:

CliniCall consultation required to discuss care planning for all patients suspected of carbon monoxide poisoning.

- Monitor vital signs; pulse oximetry is not reliable in patients with CO poisoning

Primary Care Paramedic – All FR and EMR interventions, plus:

- Consider IV access
  - → D03: Vascular Access and Fluid Administration
  - Consider fluid bolus to correct hypoperfusion or hypotension if clinically indicated

Advanced Care Paramedic – All FR, EMR, and PCP interventions, plus:

- Maintain fluid and electrolytes; treat severe acidosis; a slight acidosis may be beneficial by shifting the oxyhemoglobin dissociation curve to the right
  - → D03: Vascular Access
- Avoid alkalosis
- Administer glucose as prophylaxis against hypoglycemia (intracellular glucose may be decreased even in presence of normal or elevated blood glucose)
  - → E01: Hypoglycemia and Hyperglycemia
- Obtain COHb level if patient was exposed to smoke from fire; consider concurrent cyanide toxicity
  - In *mild* exposures, 100% oxygen should continue until patient is asymptomatic and COHb levels are < 5-6%
  - To protect fetus, pregnant patients not receiving HBO may need to continue 100% oxygen 5 times longer than time required to reduce maternal COHb to < 5-6%
  - In *moderate and severely* poisoned patients who are not candidates for HBO, 100% oxygen may need to be continued for 24 hours after symptoms resolve
- Hypotension unresponsive to IV fluids may require vasopressors
- Seizures should be treated with IV benzodiazepines
  - → F02: Seizures
- Treat cerebral and pulmonary edema supportively

Critical Care Paramedic – All FR, EMR, PCP, and ACP interventions, plus:

- Monitor electrolytes, glucose, renal and liver function, creatine kinase
- Monitor ECG, troponin, blood gases and serum lactate levels in symptomatic patients
- Obtain methemoglobin level if patient was exposed to smoke from fire; consider concurrent cyanide toxicity
- Consider transport to a hyperbaric unit.

Evidence Based Practice

Carbon Monoxide

Supportive

- Oxygen

Neutral

- Direct Transport To Hyperbaric Facility

Against

References

1. Alberta Health Services. AHS Medical Control Protocols: Carbon Monoxide Poisoning. 2020. [Link]