CATHOLIC HEALTH INITIATIVES
Franciscan Health System
St. Joseph Medical Center
Hyperbaric Medicine Service

Pressure Points

Quality Solutions... Healthy Returns

Carbon Monoxide Poisoning

The Role of Hyperbaric Oxygen Treatment

Case History

At 0730, W.H., a 26 year old white male, awoke in a trailer house after approximately seven hours of sleep in an unventilated room with a propane fueled space heater. He was disoriented with a severe headache, nausea, and vomiting. He had to drive to a neighbor's house to access the 911 system and returned to the trailer with the neighbor where they extricated C.H., the patient's unresponsive 48 year old father. EMS personnel provided high flow oxygen administration for both patients during transport that was subsequently converted to 100% oxygen by a tight fitting mask in the emergency room. C.H. had a carboxyhemoglobin level of 23%. He remained unresponsive and was intubated and ventilated when subsequently transferred to the hyperbaric chamber with his son at 1421.

Carbon monoxide (CO) poisoning, whether accidental or intentional, is the leading cause of poisoning death in the United States. While CO poisoning is typically associated with smoke inhalation in fires and inhalation of automobile exhaust, exposure can also occur from improperly vented gas heaters and stoves, propane powered industrial equipment, and other industrial processes such as steel and paint production. CO is colorless, odorless, tasteless, and non irritating. It is a cellular asphyxiant producing a "respiratory" hypoxia by displacing oxygen from hemoglobin and disrupting the mitochondrial cytochrome electron transport system **(1, 2)**.

"Carbon monoxide (CO) poisoning, whether accidental or intentional, is the leading cause of poisoning death in the United States."

CO bind reversibly to hemoglobin (Hb) producing carboxyhemoglobin (COHb) as **CO + Hb = COHb** with 200-250 times greater affinity for Hb than oxygen. Oxygen transport to tissues is decreased as oxygen is displaced from Hb during uptake of CO. CO also causes a left shift in the oxyhemoglobin dissociation curve and ultimately decreases cardiac output through depression of myocardial contractility and cardiac output decreasing blood flow to critical organs further reducing oxygen availability. CO binds to mitochrondrial electron transport proteins such as cytochrome oxidase and also to myoglobin producing directly toxic effects. The net effect is cellular hypoxia from reduced oxygen availability and decreased oxygen utilization at the subcellular level. There is also evidence suggesting that CO exposure may produce post-ischemic reperfusion injury in the central nervous system **(3)**.

It is important to note that COHb levels are poor predictors of patient outcomes because they have not been shown to correlate well with total CO exposure or end organ dysfunction. The most accurate predictors of adverse outcome are loss of consciousness, even brief, and neurological or other end organ dysfunction. Most disturbing is the incidence of delayed neurologic injuries. Between 23 and 47 percent of patients show impairments of concentration, attention, language, learning, memory, or motor function, or depression, dementia, or psychosis developing between 2 and 28 days following poisoning **(4)**.

Treatment of CO poisoning has focused primarily on administering supplemental oxygen to the patient to increase the rate of displacement of CO from hemoglobin and increase oxygen delivery to tissues. The published randomized clinical trials prior to 2001 of hyperbaric oxygen treatment in CO poisoning have been reviewed **(5)**. In 2002, Weaver **(6)** published in the NEJM the most carefully controlled randomized, prospective, double blind clinical trial to date. The trial involved 76 patients in each arm of the trial, treatment within 24 hours of CO exposure, sham treatments in the control group, a treatment profile similar to common hyperbaric oxygen treatment experience, explicit definitions of cognitive sequelae, and a very high degree of late follow up (73/76 in the hyperbaric group, 71/76 in the control group). Cognitive sequelae at six weeks were less frequent in the hyperbaric oxygen treated group (25 percent) compared to the normobaric oxygen treated group (46.1 percent). Thus for every six patients treated, one case of delayed neurologic sequelae could be avoided.

Pressure Points

Adverse effects of hyperbaric oxygen treatment include otologic barotrauma and oxygen induced seizure that has an incidence of 1:10,000 exposures although no seizures occurred in the Weaver study.

Table1. gives a summary of the current recommendations for treatment of CO poisoning with hyperbaric oxygen treatment.

Case History

C.H. and W.H. completed a CO hyperbaric oxygen treatment table that administered 50 minutes of 100% oxygen breathing at 3.0 ATA and 100 minutes of 100% oxygen breathing at 2 ATA will full recovery of W.H. and with significant improvement of C.H. such that he could breathe spontaneously, move all extremities purposefully to command, and respond to verbal commands and questioning. W.H. required no additional treatment and was released after follow up without evidence of neuropsychological deficit or sequelae. C.H. required nine additional hyperbaric oxygen treatments of 90 minutes of 100% oxygen breathing at 2.4 ATA leading ultimately to full recovery, also without neuropsychological deficit or sequelae.

References

1. Raub JA, et al. Carbon monoxide poisoning - a public health perspective. **Toxicology** 2000; 145:1-14.
2. Gorman DF, et al. A longitudinal study of 100 consecutive admissions for carbon monoxide poisoning to the Royal Adelaide Hospital. **Anaesth Intens Care** 1992; 20:335-349.
3. Thom S. Editorial: Hyperbaric-oxygen therapy for acute carbon monoxide poisoning. **NEJM** 2002; 347:1105-1106.
4. Thom SR, et al. Delayed neuropsychologic sequelae after carbon monoxide poisoning: Prevention by treatment with hyperbaric oxygen. **Ann Emerg Med** 1995; 25:474-480.
5. Hampson, NB, et al. Carbon monoxide poisoning: interpretation of randomized clinical trials and unresolved treatment issues. **Undersea Hyper Med** 2001; 28:157-164.
6. Weaver L. et al. Hyperbaric oxygen for acute carbon monoxide poisoning. **NEJM** 2002; 347:1057-1067.

Table 1. Carbon Monoxide Poisoning Treatment Recommendations

1. Remove the patient from the source of CO poisoning while avoiding significant exposure to potential rescuers.
2. Provide 100% oxygen administration by tight flow face mask or endotracheal tube with a high flow delivery system.
3. Verify carbon monoxide poisoning by an arterial or venous carboxyhemoglobin level although the level measured may not contribute to the decision to refer the patient for hyperbaric oxygen treatment.
4. In minimally symptomatic patients without loss of consciousness or other focal neurological abnormalities, myocardial ischemia, or other evidence of end organ hypoxia or myoglobinuria provide 100% oxygen breathing as above for a minimum of two hours. Any new symptoms or failure of all symptoms to resolve should at this point prompt consideration of hyperbaric oxygen treatment.
5. Refer for emergency hyperbaric oxygen treatment any patient with any of the following:
   - Any history of loss of consciousness
   - Any evidence of end organ dysfunction due to hypoxia
   - A concomitant lactic acidosis (consider concomitant cyanide poisoning if in the setting of smoke inhalation)
   - Persistence or progression of symptoms with surface oxygen administration
   - Carboxyhemoglobin level greater than 25 percent
6. Make the decision to refer for hyperbaric oxygen treatment based on symptoms rather than initial carboxyhemoglobin level (except with very high levels of COHb).
7. Provide hyperbaric oxygen treatment as soon after extrication from the source of poisoning possible for best results.
8. More than one treatment may be required for optimal results.
9. Provide for follow up of all CO patients to monitor for late neurological and psychological sequelae.

Pressure Points

A Publication of Praxis Clinical Services
Robert A Warriner, III, M.D., FACA, FCCP
Chief Medical Officer

17772 E. 17th Street, Suite 205
Tustin, California 92780
888-345-3300
www.PraxisUSA.com

CATHOLIC HEALTH INITIATIVES
Franciscan Health System
St. Joseph Medical Center

Hyperbaric Medicine Service

1717 South "J" Street • Tacoma, WA 98401
Ph: (253) 426-6630 • Fax: (253) 426-6545
www.fhshealth.org/location/sjmc.asp