

Hyperbaric Oxygen Therapy in Carbon Monoxide Poisoning

Hyperbaric oxygen (HBO) therapy involves inhalation of 100% oxygen at pressures >1 atmosphere which increases oxygen tension within the body. HBO is used for air embolisms, tissue healing, and carbon monoxide (CO) poisoning (*N Engl J Med.* 2009;360(12):1217-25). CO is formed from incomplete combustion of any carbonaceous compound (ex. fires, gas stoves, generators, etc.). Patients with CO exposures may initially develop non-specific symptoms such as headache, vomiting, and dizziness, or more severe symptoms: EKG changes, loss of consciousness, seizures, and coma.

CO binds to hemoglobin to form carboxyhemoglobin (COHgb) resulting in reduced oxygen carrying capacity, but more importantly, free CO diffuses across membranes and tissues. Free CO interferes with the mitochondrial transport chain, lipid peroxidation, and induces xanthine oxidase resulting in oxidative stress and cell death (*N Engl J Med.* 2009;360(12):1217-25). We can only measure the COHgb level, which is a poor predictor of symptoms and outcomes (*Am J Respir Crit Care Med.* 2007;176:491-497). HBO reduces COHgb half-life from 3-4 hours to 20 minutes. However, the reality is many patients have an undetectable COHgb level by the time they reach the HBO chamber due to time of transport, evaluation, and because the half-life of COHgb on a non-rebreather at normobaric oxygen (NBO) is ~60 min. HBO is therefore used to prevent some of the delayed and persistent neurologic sequelae that manifest as amnesic syndromes, psychosis, dementia, parkinsonism, neuropathies, and others.

There are 6 randomized trials examining HBO in CO poisoning, two that displayed benefit (*Ann Emerg Med.* 1995;25:474-80; *N Engl J Med.* 2002;347(14):1057-67), three reported no difference in outcomes between HBO and NBO (*Lancet.* 1989;2(8660):414-9; *Undersea Hyperb Med* 1996;23 Suppl:7; *Med J Australia.* 1999;170:203-10), and one that showed harm (*Intensive Care Med.* 2011;37(3), 486-492). Among these, only two were large, double-blinded, randomized control trials. One of them displayed a large benefit from HBO reducing the risk of neurologic sequelae by >50%, but it changed the primary outcome mid-trial to include all neurologic sequelae (even non-specific symptoms) (*N Engl J Med.* 2002;347(14):1057-67). The inclusion of all symptoms produced statistically significant results while there was no difference seen in delayed neurologic sequelae alone. The other large study performed displayed no difference in outcomes, but was limited by poor follow-up of only 88/191 of patients (*Med J Australia.* 1999;170:203-10). Furthermore, one study demonstrated no difference between HBO and NBO in non-severe patients but was terminated early due to worsening effects seen in patients with severe toxicity who received two sessions of HBO (*Intensive Care Med.* 2011;37(3), 486-492). A Cochrane review of the literature also failed to determine whether HBO therapy decreases delayed/persistent neurologic sequelae in CO poisoning (*Cochrane.* 2011, Issue 4. Art. No.: CD002041). With that stated, it is important to note that patients with severe symptoms often undergo HBO treatment due to its relatively low risk and potential benefit.

Bottom line: The benefits of HBO in CO poisoning are highly debated. Call the Poison Center for help with individual case assessments.



Did you know?

Although it is still unclear who may benefit, patients are often transported or transferred to specialty centers for HBO therapy.

HBO is often utilized in patients with risk factors for the development of neurologic sequelae or with severe symptoms: loss of consciousness, coma, seizure, COHgb >25%, EKG changes, exposure for ≥ 24 hours, and those >35 years (*Am J Respir Crit Care Med.* 2007;176:491-497). Note that all pregnant women were excluded from the published trials, and any degree of hypoxia induced by CO may be harmful to a fetus, therefore the threshold for pregnant patients to be treated with HBO is lower, at a COHgb >15%.

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