Toxicology Question of the Week

November 5, 2018

How long should oxygen be administered following carbon monoxide exposure?

**Last month there was a mass CO exposure. Length of time of oxygen administration following the exposure was queried by health care professionals.**

**Background**

Carbon monoxide (CO), a tasteless, colorless, odorless gas, may remain undetectable until injury or death has occurred. (Do you have a CO detector in your house?) Long recognized as a toxin, the assumption was that CO sat on the hemoglobin (Hgb) tetramer in the red cell, taking the place of oxygen and causing hypoxia. Once the CO left the Hgb tetramer, there was nothing more to do. Unknown were the multiple mechanisms of action and the resultant toxicity. Unrecognized was the neuropsychiatric (NP) sequelae (including decreased IQ, memory loss, impaired executive function, psychiatric syndromes) following CO exposure. Still unknown is the cause of the NP sequelae and treatments that prevent or ameliorate it.

**Mechanisms**

CO binds to Hgb to form COHb. The affinity of CO for Hgb is 200 times that of the affinity of oxygen for Hgb. When CO is bound to one of the tetramers of Hgb, it increases the affinity of oxygen at the remaining binding sites preventing the release of oxygen to the peripheral tissues and causing a leftward shift of the oxyhgb dissociation curve. The result is tissue hypoxia.

CO also binds to cytochrome c oxidase, a protein in the mitochondria which alters electron transport. Reactive oxidative species (ROS) are formed causing oxidative stress. CO also binds to myoglobin (affinity 40 greater than that of oxygen) with even greater affinity for cardiac myoglobin. Cardiac dysfunction and arrhythmias may occur.

In the CNS, release of ROS leads to oxidative stress, lipid peroxidation and apoptosis. This, in addition to cytochrome malfunction and hypoxia, may be the cause of the neuropsychiatric sequelae.

The COhgb concentration documents exposure. It doesn’t reflect length of time of exposure, peak COhgb, or degree of exercise during the exposure.

**Treatment**

The problem is that the treatment of CO exposure is controversial with no recognized treatment guidelines. There have been trials of hyperbaric oxygen (HBO) vs normobaric oxygen (NBO) which were inconclusive. Tennessee Poison Center recommends HBO for pregnant women who have been exposed to CO. Fetal demise may occur with CO exposure and pregnant women do well with HBO.

The Tennessee Poison Center (TPC) recommends 12-23 hours of NBO for anyone with a “significant” exposure, even if COhgb is back to normal. Studies have indicated that cytochrome oxidase may malfunction for up to three days following exposure. One studyt demonstrated normobaric oxygen was more efficacious than HBO but these patients received NBO for three days. High risks for NP sequelae include prolonged exposure (even at low COhgb levels), loss of consciousness, age (young and old), hypotension, and symptoms.

CO poisoning is a mandatory reportable condition to be communicated to the Tennessee Department of Health within one week. If you call the Tennessee Poison Center (**1-800-222-1222)**, TPC will assist with the reporting process.

**Additional Information:** <https://www.tn.gov/content/tn/health/cedep/reportable-diseases/carbon-monoxide-poisoning.html>

I am interested in any questions you would like answered in the Question of the Week. Please email me with any suggestion at [donna.seger@vanderbilt.edu](mailto:donna.seger@vanderbilt.edu)

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[www.tnpoisoncenter.org](http://www.tnpoisoncenter.org)

Poison Help Hotline: 1-800-222-1222

The Question of the Week is available on our website: [www.tnpoisoncenter.org](http://www.tnpoisoncenter.org)