

# Toxicology **FOCUS**

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Toxicology Update for Health Care Providers



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## CARBON MONOXIDE: A CASE REVIEW

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Cold winter months bring an increased use of alternative heating methods such as kerosene heaters, emergency generators, and wood burning stoves which in turn bring an increased risk for exposures to carbon monoxide (CO). CO is a colorless and odorless gas produced by the incomplete combustion of carbon fuels. It is often referred to as the "silent killer" since it is not noticeable and the early effects are often misdiagnosed or missed altogether. In 2014, the Virginia Poison Center has been consulted on 113 cases of CO exposure, 73 of which required evaluation at a health care facility. The following is a representative example:

A 49-year-old male presented to the ED after being found in his car, running in an enclosed garage. On EMS arrival, he was comatose with a RR 4, had flushed skin, and was tachycardic with a HR 118 and BP 123/75. He was placed on 100% oxygen and his respiratory efforts were assisted via bag-valve-mask. He was given 0.5 mg naloxone intra-nasally. On arrival to the ED, his mental status had improved and his carboxy-hemoglobin (COHB) level was 39.5%. Due to the patient's loss of consciousness and COHB of 39.5%, he was flown to a secondary health facility for hyperbaric oxygen (HBO) therapy. The patient was awake and alert with stable vital signs after the initial HBO therapy. Pertinent laboratory studies included a CBC which was significant for a WBC 11.4, HGB 8.5, and HCT 25.8. The metabolic panel was within normal limits. It was also reported that the patient had a purple "grape Kool-Aid® urine color. A repeat COHB level at approximately 4 hours after the initial hyperbaric treatment was 2.3%. A second treatment of hyperbaric oxygen therapy was also done. At a later time, it was noted that he had received hydroxocobalamin (Cyanokit®) either by EMS or the initial health care facility which may have explained the urine color changes. The patient was transferred to psychiatry at 2.5 days post exposure.

CO poisoning causes tissue hypoxia which results from decreased oxygen delivery to tissues, as well as mitochondrial dysfunction and free radical formation. CO has an affinity for hemoglobin approximately 200 to 240 times that of oxygen resulting in decreased oxygen carrying capacity. Organs with high oxygen demand are particularly susceptible to hypoxia, which explains why neurological and cardiac toxicity are most prominent. In addition to decreased oxygen delivery, inhibition of ATP synthesis and increased production of radical oxygen species result in inflammatory responses, neutrophil activation, and oxidative stress. Apoptosis can occur during re-oxygenation, similar to that of reperfusion injuries. Long term effects may include delayed and/or persistent neurological sequelae including impaired mood, short-term memory, attention span and ability to concentrate.

The severity of toxic effects most likely relates to CO dose, which is the product of the CO concentration and the duration of the exposure. Patients with underlying medical problems (particularly neurologic or cardiac) may be at increased risk with any given dose. Venous or arterial COHB levels are essentially equivalent and should be evaluated as soon as possible following exposure. Non-smokers should have a COHB level less than 2-5% and chronic smokers may have a baseline level ranging from 6-10%. Additional laboratory studies for exposed patients should include CMP, lactate, VBG or ABG, serial 12-Lead ECG's, cardiac enzymes and head CT if there is loss of consciousness, focal or persistent neurological findings. 100% oxygen via a non-rebreather mask is recommended or via intubation if the patient is unable to maintain his airway. The effectiveness of hyperbaric oxygen is controversial, with conflicting results in clinical trials, none of which utilized the same HBO treatment algorithm. Suggested indications for the use of HBO therapy include loss of consciousness, cardiovascular instability (hypotension), seizures, COHB level > 25-30%, metabolic acidosis, or the patient is pregnant with a COHB level > 15%. A Virginia Poison Center toxicologist is always available for consultation, particularly with respect to decisions regarding transfer and/or HBO.

Reference: A. Chiew and N. Buckley. *Carbon monoxide poisoning in the 21<sup>st</sup> century.*  
Critical Care (2014), 18: 221

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## Did You Know?

Ingestions of 0.5-1 g/kg (or 1 level tablespoon) of table or rock salt can cause a rise in serum sodium of 30.5 mEq/L in a 3 year old, 15 kg child?

Watch out for children being exposed to table and rock salt and de-icing granules.



## Excerpts from the Recent Medical Literature

S. Rutherford Rose, PharmD

### **In vitro release of fentanyl from transdermal patches in gastric and intestinal fluid. Arroyo Plasencia AM, et al. Clinical Toxicology 2014; 52(9):945-7.**

New or used fentanyl transdermal patches may be ingested, either by curious toddlers or as means of abuse by adults. Significant toxicity following patch ingestion has been documented in published cases. This paper describes the amount of fentanyl released from new, undamaged patches (Mylan brand) after being placed in 100 ml of simulated gastric (pH 1.2) or intestinal (pH 6.8) fluid. Each patch contains 7.65 mg of fentanyl, and is designed to release 75 mcg/hr for 3 days through the skin.

Averages of 239 mcg (gastric) and 338 mcg (intestinal) of fentanyl were released from the patches within 5 minutes, and 1,962 mcg (gastric) and 3,139 mcg (intestinal) released over 2 hours. After 3 hours, 26% and 41% of patch contents were released over 3 hours in gastric and intestinal environments, respectively. These amounts considerable exceed the 3% of patch contents released during transdermal use (75 mcg/hr x 3hrs).

These results suggest that intact fentanyl patches if swallowed can release significant amounts of fentanyl. However, with average oral bioavailability of 40-50%, it is uncertain how much of the released fentanyl will be absorbed and ultimately reach the central nervous system. Physical damage to the patch could result in even higher amounts of drug released. While worthy of further study, these data support the observation that significant opioid toxicity can result from fentanyl patch ingestion.



## Nicotine

Michael Emswiler, MD

Nicotine is a well-known alkaloid present in natural tobacco products (cigarettes, cigars, smokeless or chewing tobacco), gum/lozenges/patches used for smoking cessation, and liquid containing nicotine for use in e-cigarettes. The amount of nicotine delivered varies by product and whether the product has been altered or used in an unintended manner. For example, most nicotine in a cigarette is lost through smoke that is not inhaled or nicotine that remains in the cigarette filter, but if a cigarette is ingested it may result in a much larger nicotine exposure. Other examples include nicotine gum that is vigorously chewed, causing faster release of nicotine present in the gum or a nicotine patch that has been altered by crushing, lacerating, or chewing.

Nicotine binds to and stimulates nicotinic receptors that are located in both the parasympathetic and sympathetic nervous system. The effects of low doses of nicotine include increased mental alertness, euphoria, hypertension, tachycardia, tremor, and vasoconstriction. Signs of nicotine overdose may include increased salivation, diaphoresis, nausea, vomiting, diarrhea, headache, dizziness, ataxia, and confusion. Serious signs of nicotine toxicity may include cardiac dysrhythmias, seizures, and muscle fasciculations that may progress to paralysis. The treatment of nicotine toxicity is supportive. There is no antidote for nicotine toxicity and measuring nicotine levels is not useful clinically.

The vast majority of nicotine exposures reported to our poison center continue to be from cigarettes. However, exposures due to e-cigarettes are increasing. These e-cigarettes have a small battery that heats up liquid containing nicotine when an individual inhales through the e-cigarette. Liquid used for refilling e-cigarettes contain a large amount of nicotine and has the potential for serious nicotine toxicity if ingested, inhaled, or absorbed through the skin. Serious toxicity may occur in a child with as little as 1.4 mg/kg. With a nicotine solution containing 18 mg/mL it would take less than one mL to produce serious toxicity in a 10kg child! The first pediatric death related to nicotine toxicity from electronic cigarette nicotine liquid was reported in December, 2014 in Fort Plain, NY.

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