Letters to the Editor

Carbon Monoxide Toxicity at High Altitude

To the Editor:

“All sickness at altitude is altitude sickness until proven otherwise” is an often-quoted adage; however, it is not always true. Carbon monoxide (CO) poisoning is a potentially fatal situation that can imitate acute mountain sickness (AMS). Dr Roscoe and colleagues prospectively examined the relationship among CO exposure at altitude, elevated carboxyhemoglobin (COHb) levels, and AMS incidence in climbers on Mt McKinley. Although a trend was seen in climbers at 4300 m, the percentage who met criteria for AMS (13.7%) or tested positive for CO exposure (12.5%) was too small to show statistical significance. Those descending the mountain, who had likely spent more cumulative time inhaling fumes, showed significantly higher CO levels. Although the results are not definitive, the paper highlights the practicality of having a high index of suspicion in an environment with potential for CO exposure, which may either precipitate AMS or cause toxicity that mimics it.

Carbon monoxide is the commonest form of accidental poisoning in the United States, accounting for 40 000 emergency department visits and 800 deaths annually. Carbon monoxide has an affinity for hemoglobin 240 times that of oxygen, thereby impairing oxygen delivery to tissues and resulting in end-organ ischemia. Direct damage is caused by anoxic injury, and lipid peroxidation results in a secondary reperfusion injury. At sea level, the half-life of COHb is 320 minutes on room air, 60 minutes on 100% oxygen, and 23 minutes with hyperbaric oxygen at 2.5 to 3.0 atm of pressure. But at higher altitudes, the reduction of partial atmospheric pressure, right shift of the oxyhemoglobin dissociation curve, and increased minute ventilations may potentiate clinical toxicity from CO. Furthermore, the expedition environment may lead to prolonged exposures to low concentrations of CO that accumulate and are potentially more harmful than a brief high-concentration exposure.

Clinically, CO is a great imitator of AMS, presenting with nonspecific “flu-like” symptoms that are familiar to anyone who has been to high altitude—headache, nausea, weakness, and dizziness. Severe CO toxicity can present identically to high-altitude cerebral edema with progressive confusion, ataxia, and loss of consciousness or high-altitude pulmonary edema with dyspnea, hypoxia, and a noncardiogenic pulmonary edema. Furthermore, the hypoxic stress caused by CO strands a victim at a “physiologically higher” altitude, which may precipitate AMS.

The outdoor literature is full of anecdotal reports of toxic CO exposure occurring in both tents and snow caves, ranging from the temperate climates of California to the arctic circle of Norway, as extensively reviewed by Leigh-Smith. Poorly ventilated spaces, low-flame camping stoves, and larger cooking-pot diameters have all been shown to increase CO levels. However, details of CO poisonings at high altitude are scarce and likely underreported because of the similarities between toxicity symptoms and AMS, as well as the lack of definitive diagnostic testing.

The cornerstone of treatment for CO poisoning is supplemental oxygen. Oxygen at either normal or hyperbaric pressures hastens the dissociation of bound CO in the blood and tissues, as well as theoretically decreasing the secondary cerebral reperfusion injury. Weaver et al designed a prospective trial that clearly demonstrated the superiority of hyperbaric oxygen to normobaric oxygen in reducing the cognitive sequelae (memory, attention, concentration, or affect problems) at 6 weeks and 12 months. This neurologic syndrome lasting 1 month or longer appears to occur in 25% to 50% of patients who have lost consciousness or have COHb levels of 25% or greater. Carboxyhemoglobin levels correlate poorly with clinical toxicity and outcome, and the therapeutic mechanisms of hyperbaric oxygen lead some researchers with ready access to hyperbaric chambers to advocate this treatment for all symptomatic CO exposures.

Wilderness medicine is distinguished from hospital-based medicine by both the remoteness of location as well as the limited availability of diagnostic resources. It is unreasonable to expect a portable CO-oximeter to be available at base camp for the expedition doctor; therefore, it becomes paramount to make clinical decisions based on a thorough history and physical examination. If the diagnosis of CO exposure is considered, do not allow the victim to return to a potentially toxic environment. Most high-altitude doctors will have access to supplemental oxygen. Although supplemental oxygen is not usually used in the treatment of mild to moderate AMS, a low threshold for early application in CO poisoning will rapidly reverse the symptoms.