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CARBON MONOXIDE POISONING: DEATH ON MOUNT MCKINLEY(US)  
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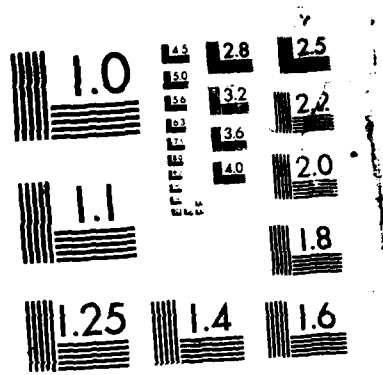
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CARBON MONOXIDE POISONING:  
DEATH ON MOUNT MCKINLEY

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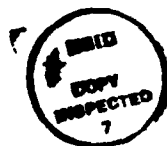
CARBON MONOXIDE POISONING

ABSTRACT

Carbon monoxide (CO) poisoning is a common problem encountered in a wide variety of settings, including both suicide attempts and accidental exposures. Fatal CO exposure occurred in two young, healthy mountain climbers who succumbed to fumes generated by a small cook stove in the enclosed space of their tent at 14,200 feet on Mount McKinley. There is the potential for confusing mild to moderate CO poisoning with the signs and symptoms of altitude illness. Physicians who deal with wilderness and environmental emergencies should be aware of this serious hazard.

KEY WORDS

carbon monoxide  
poisoning  
environmental medicine



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## CARBON MONOXIDE POISONING: DEATH ON MOUNT MCKINLEY

### INTRODUCTION

Carbon monoxide (CO) is a colorless, odorless, tasteless, nonirritating gas, generated by the incomplete combustion of carbon-containing material. It is responsible for thousands of deaths each year, both through suicide and accidental poisoning. We report the accidental deaths of two young, healthy mountain climbers on Mount McKinley, Alaska, who succumbed to CO fumes given off by a butane cook stove used in the enclosed space of their tent. With the increased popularity of mountaineering and backcountry travel, individuals employing similar equipment may be at risk of carbon monoxide poisoning by this unusual means.

### CASE REPORT

A Swiss six man climbing party began their ascent of Mount McKinley after flying to the 7,200 foot Kahiltna Glacier landing site on 29 May 1986. From here, the party split up into two teams. Four members planned to climb the standard, well-traveled West Buttress route, while the other two would attempt the more technically difficult Cassin route.

After an eight day climb up the Kahiltna Glacier, the group of four arrived at the 14,200 foot base camp on the

## CARBON MONOXIDE POISONING

West Buttress. This is a large plateau which contains numerous campsites during the Spring climbing season. The National Park Service/University of Alaska medical/rescue tent is located at this site as well. This facility is well equipped for medical emergencies and was staffed by research emergency physicians throughout the 1986 climbing season.

On 6 June a 23 year old member of the Swiss climbing team was examined by a physician for minor eye irritation. At that time, he appeared well and was tolerating the altitude without undue difficulty.

The following day, the four carried food and equipment to place a cache at the 17000 foot level and returned to base camp. Reportedly, the 23 year old felt quite tired from the day's work while his 22 year old tent companion experienced some mild symptoms of acute mountain sickness (headache and malaise). At approximately 9:20pm, these two climbers told the other members of the party that they were going to cook dinner and then go to sleep, wishing to sleep-in until tomorrow's afternoon. Light snow fell most of the night and into the afternoon of the next day.

As the snow dissipated in the early evening of that following day their companions became concerned about the lack of activity in the tent of the two men. Upon entering the tightly closed tent, both men were found cold and lifeless. A physician and a park ranger were immediately summoned from the nearby medical tent.

## CARBON MONOXIDE POISONING

The following description of the scene is extracted from a statement prepared by the physician on duty at the high altitude medical tent:

"At approximately 7:00pm a Swiss climber appeared, excitedly repeating 'Doctor, doctor! No English.' We followed him to a tent where a second Swiss climber was standing with a forlorn expression. I entered the tent and found two young men who were obviously dead. They were in their sleeping bags, cold, pulseless, apneic and semi-stiff with rigor mortis. Nevertheless, I performed two chest compressions on one of the bodies noting a mucous-like fluid exuding from his nose and mouth. No attempt was made to clear the airway--he was dead. They both were.

I was struck by the peacefulness of their semi-rigid positions. Both were as if they were still asleep. I believe they died in their sleep. I did not note 'cherry red skin color', however some blood pooling was evident. They appeared to have been dead for about 18-24 hours.

I recreated the tent arrangement as I had found it. The victims' heads were near the vestibule, about two feet from their butane stove. The stove was left about 1/4 turn on (probably a low simmer) and the fuel canister was empty. It appeared to me that they were cooking dinner in their tent.

## CARBON MONOXIDE POISONING

After eating, they left some soup on the stove on low simmer (perhaps also to keep the tent warm in the storm). As they lay in their sleeping bags, exhausted from the day's carry to 17,000 feet, they were overcome by carbon monoxide. Because of the storm, they had the vestibule zipped tight and their ceiling vents closed, leaving a tightly sealed mylar coated bubble. Perhaps if they had only vented their tent this tragedy would not have happened."

On the following day the bodies were evacuated in sleds by skiers who pulled them to the 7,200 foot Kahiltna landing site. From here they were flown to Talkeetna, Alaska, and then to Anchorage. Post mortem examination revealed the cause of death in both individuals to be asphyxiation due to acute carbon monoxide poisoning. Their blood carboxyhemoglobin concentrations were measured at 56.9% and 65.6%, respectively.

### DISCUSSION

Carbon monoxide (CO) has approximately 200-250 times greater affinity for hemoglobin than does oxygen. (1) In very small concentrations, this lethal gas displaces oxygen from hemoglobin, markedly diminishing its oxygen-carrying capacity. (2) At a partial pressure of CO as low as 0.5 mm



## CARBON MONOXIDE POISONING

Hg, the hemoglobin saturation (by CO) is approximately that expected for a partial pressure of 100 mm Hg of oxygen. (3)

Carbon monoxide alters the molecular configuration of hemoglobin, resulting in an unfavorable left shift of the oxyhemoglobin dissociation curve. (4) Additionally, studies by Astrup(5) and Thomas(6) have reported decreased erythrocytic 2,3-diphosphoglycerate (2,3-DPG) concentrations with acute and chronic exposure to CO. Thus, in the presence of carboxyhemoglobin (COHb), the tissue partial pressure of oxygen may be lower than in the case of a depressed oxyhemoglobin saturation (SO<sub>2</sub>) caused solely by hypoxia (Figure). Another major effect of CO may be competition with O<sub>2</sub> for cytochrome A3 oxidase, thereby disrupting cellular respiration, as in cyanide poisoning. (7,8) The resultant profound tissue hypoxia may lead to anaerobic metabolism, manifested by progressive lactic acidosis.

Recently published data refute the validity of the cellular toxicity concept during acute CO poisoning. Halebian, et al found no significant difference in measured O<sub>2</sub> consumption or extraction between dogs subjected to CO poisoning vs nitrogen anoxia. (9) They concluded that CO poisoning is primarily a hypoxic event, in which carboxycytochrome oxidase formation does not play a major role.

At sea level, the blood half-life of CO is between 4-6 hours. Since the carboxyhemoglobin concentration in the

## CARBON MONOXIDE POISONING

blood is cumulative over time, prolonged exposure to low concentrations of CO is more harmful than brief exposure to high concentrations. (10)

Physiological changes which occur at high altitude may potentiate the risk of toxicity from exposure to carbon monoxide. (11) Significant increases in the half-life of COHb may occur because of the environmental reduction in the partial pressure of oxygen (atmospheric PO<sub>2</sub> at 14,000 feet is approximately 90 mm Hg vs. 149 mm Hg at sea level). The clinical effects of CO toxicity are further enhanced at altitude, since CO uptake is inversely proportional to the relative atmospheric PO<sub>2</sub> and uptake of CO is likely to be augmented by increased minute ventilation volumes. (12,13)

At high altitudes, O<sub>2</sub> delivery to the tissues is facilitated by a rise in 2,3-DPG and a right shift of the oxyhemoglobin dissociation curve. (14) The degree to which this beneficial biochemical alteration modifies the unfavorable left shift and decreased 2,3-DPG caused by CO intoxication is not determined. Despite these apparently opposite mechanisms, some studies have shown little difference in oxygen transport between CO hypoxia and hypobaric hypoxia. (15)

The physician must be aware that nonspecific, influenza-like symptoms of mild to moderate CO poisoning may readily be confused with a variety of other conditions. (16) This becomes especially important when the patient may be discharged to return to a potentially toxic environment.

Headache, which is a prominent and consistent symptom in the clinical presentation of CO toxicity, is often accompanied by malaise, dizziness, weakness, mental confusion, nausea and breathlessness. In a high altitude setting, this constellation of symptoms could well be attributed to Acute Mountain Sickness (AMS), a prevalent condition resulting from atmospheric hypoxia. (11) A finding of retinal hemorrhages, common to both AMS and CO poisoning, may add to the confusion. (17,18,19) Some experts in altitude physiology believe that mild degrees of CO poisoning frequently predispose to the development of altitude illness. (Peter Hackett, MD; personal communication)

High Altitude Pulmonary Edema (HAPE) and High Altitude Cerebral Edema (HACE) are more serious forms of altitude illness. Cerebral and/or pulmonary edema is also a frequent complication of severe CO poisoning. (20,21) Intense headache, common to both altitude illness and CO poisoning, has been attributed to cerebral edema and increased intracranial pressure caused by fluid transudation across hypoxic capillaries. (12,22,23) In animal studies, CO poisoning has been shown to adversely affect cerebral venous flow, based on marked stagnation of venous circulation and on a precipitous rise in cerebral spinal fluid pressure. (20) The increased intracranial pressure of CO toxic states may have both a cytotoxic and a vasogenic etiology, which has been cited as the underlying mechanism of altitude illness as well. (23,24,25) Tissue hypoxia may be the common

denominator for these clinical manifestations. It appears reasonable that CO toxicity may interact synergistically with hypobaric hypoxia to complicate or precipitate varying degrees of altitude illness.

Commonly noted sources of CO include: automobile exhaust; smoke from fires, stoves and charcoal burners; cigar, cigarette and pipe smoke; and space heaters and furnaces. There are numerous anecdotal reports regarding possible CO poisoning among outdoor explorers and enthusiasts; however we know of no other proven deaths by this means. Despite existent warnings concerning the dangers of stove use in tents, CO may be a greater hazard than is generally appreciated by mountaineers, backpackers and others who employ tightly sealed tents and portable cook stoves in pursuit of outdoor adventure. (Warren Bowman, MD; personal communication) In light of this risk, it would seem prudent for tent and stove manufacturers to more clearly print a warning regarding the potential for carbon monoxide poisoning on their merchandise.

#### SUMMARY

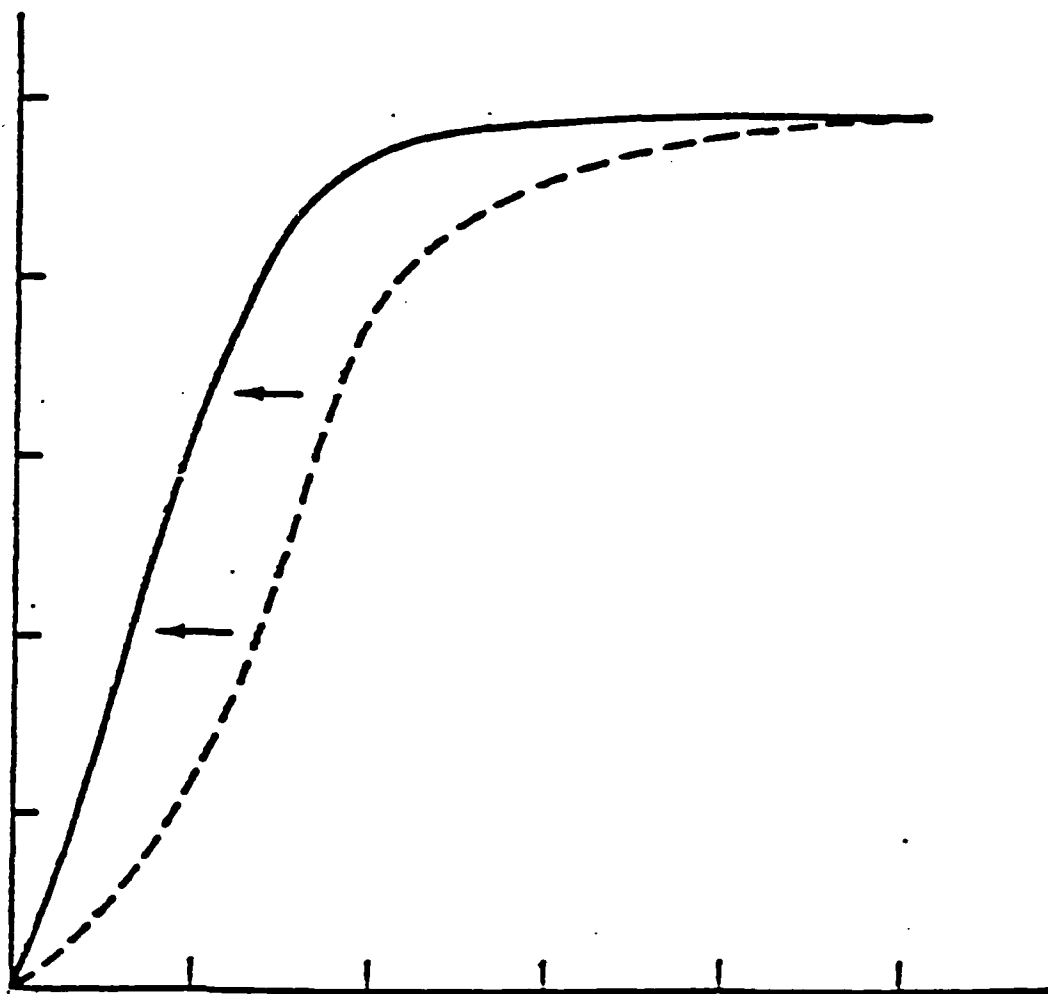
One does not expect death due to toxic fumes in a pristine wilderness setting, yet this case illustrates the extreme hazard that can occur when CO is allowed to accumulate in any closed space. Physicians who care for individuals pursuing similar outdoor activities have a two-fold

responsibility: to provide information in order to prevent accidental poisoning and to consider this problem in the differential diagnosis of persons complaining of flu-like symptoms in a high altitude environment.

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CARBON MONOXIDE POISONING



CARBON MONOXIDE POISONING

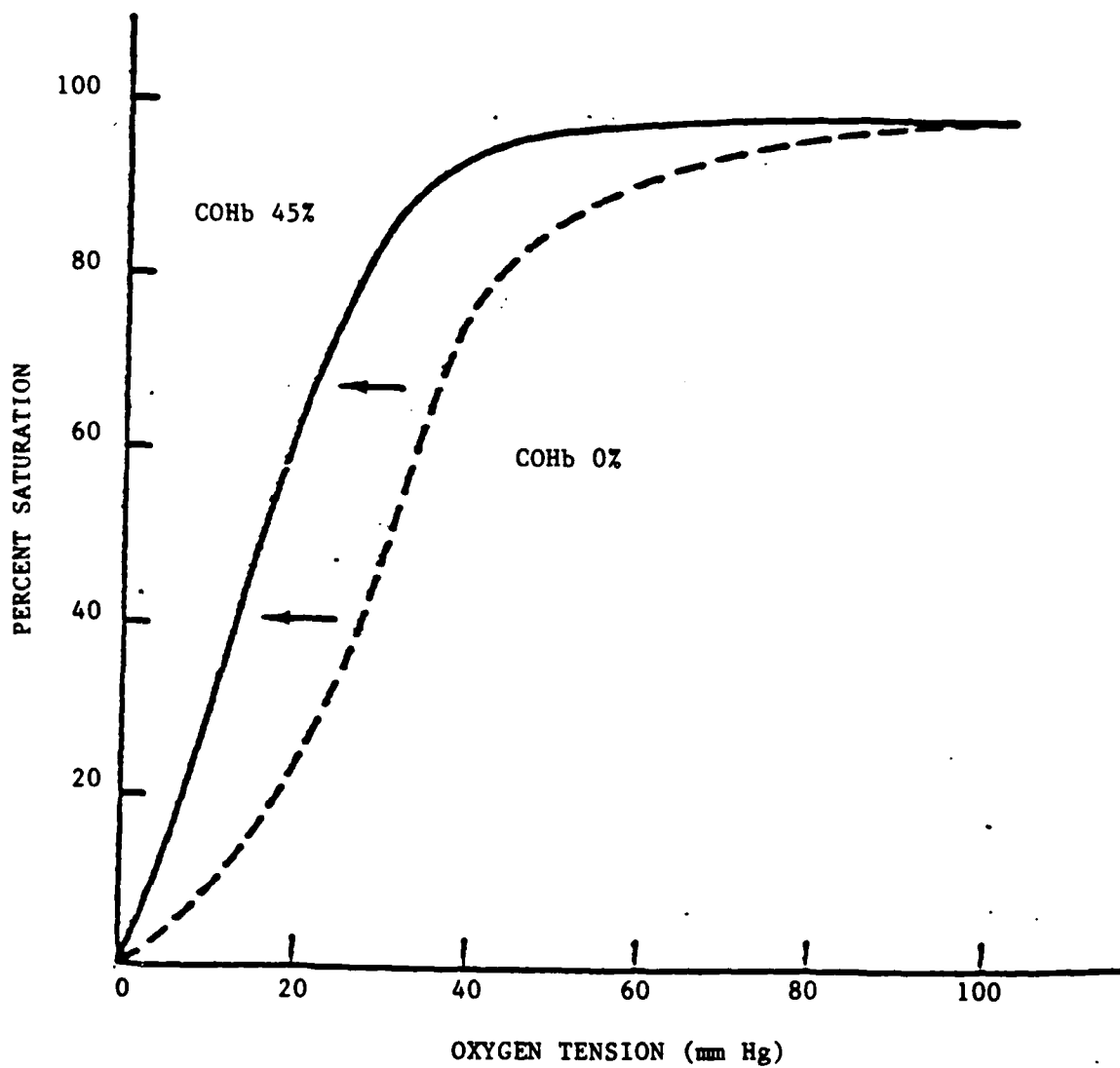


FIGURE. Carboxyhemoglobin (COHb) effect on oxyhemoglobin dissociation curve.

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