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ORIGINAL RESEARCH

Carbon Monoxide Poisoning in Tents—A Review

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This review discusses the overlooked problem of carbon monoxide (CO) poisoning within small tents. It summarizes previous case reports, reviews the toxicity of CO, and attempts to draw conclusions from experimental work. Finally, practical recommendations are developed on avoiding CO poisoning within tents. The term *carbon monoxide* was used in a search of the Medline database covering the years 1966 to 2003. The results were combined with the terms *atmosphere* or *camps* or *stoves* or *climbs* or *mountains* or *tents* or *poisons.* The resulting articles were reviewed, and those relevant to this problem were obtained. Hard copies were hand searched for further relevant articles until no more citations could be found. Three original articles were impossible to obtain but have been cited to assist others seeking to find them. Other data and articles were obtained from the Ministry of Defence but are unpublished for security reasons.

Key words: carbon monoxide, tent, stove, poison, altitude

History

Laboratory work and computer modeling have predicted that a dangerous level of carbon monoxide (CO) could be reached inside tents within 30 minutes.[1] Numerous anecdotal reports about possible CO poisoning among outdoor enthusiasts also exist.[2] The first literature reports of the dangers posed by CO within tents and snow caves date back to early exploration of higher latitudes in the 1930s. Narrow escapes from episodes of poisoning in the Antarctic, which caused the authors and their companions to collapse, were reported by Amundsen in 1911.[3] Similar episodes in the Arctic were recorded by Steffanson,[4] who reviewed a number of other Arctic accidents in which symptoms of CO poisoning may have been present.

Frequent symptoms of poisoning have been reported within tents and snow caves in Norway[5] and during Antarctic overwintering in crates while using Primus stoves.[6] The author has personal knowledge of a poisoning episode that occurred when a soldier was sitting up inside the door of an open tent while cooking. He lost consciousness and collapsed but rapidly returned to a normal level of consciousness when laid down and dragged to an area of clear ventilation.

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The first reported fatalities from CO poisoning inside tents came from a temperate climate. A 10-year review (1979–1988) of all accidental CO deaths in California revealed that 10 of 136 deaths were associated with camping equipment—7 from lanterns or lamps and 3 from stoves.[7] Other temperate-zone reports include 1 fatality from an alcohol stove inside a campervan in Germany,[8] a propane gas stove inside a large tent in the state of Georgia (United States) that killed an adult and 3 children, and a charcoal grill inside a tent in the same state that killed an adult and child.[9] An average of 30 fatalities per year from 1990 to 1994 as a result of CO poisoning in tents within the United States has been reported.[9,10]

The author has personal experience as the medical attendant during an episode of CO poisoning resulting in the deaths of 2 Royal Marines in Norway, although a third Royal Marine in the same tent survived (Ministry of Defence, unpublished report, 1993). The military 4-man tent had been pitched tactically the night before (dug into the snow, resulting in snow walls higher than the tent, and a camouflage net placed over the top). Subzero temperatures, moderate winds, and light snow characterized the 12 hours after entering the tent before a camping stove was lit to prepare breakfast. There was no need to leave the tent to collect snow during this time, as a snow-storage bag had been prepared the previous night. Other Marines had normal verbal contact with the tent dwellers 100 minutes after the stove was lit and

again 140 minutes after it was lit. In retrospect, the tent inhabitants were noted as sounding incoherent during the second episode of verbal contact, although this was assumed at the time to be a result of sleepiness. When no verbal contact could be established 240 minutes after stove lighting, all 3 men were pulled from the tent unresponsive; only 1 survived despite resuscitation efforts.

Very few literature reports of CO poisoning at altitude exist, and this may be because of the similarity in symptoms with acute mountain sickness (AMS). One of the few reports involves 2 episodes of severe (but nonfatal) poisoning at 5200 m (17 060 feet) on Mount McKinley in 1985. In the first episode, 2 climbers had been melting snow inside an igloo with a white gas stove for an extended period. They suffered headache, insomnia, tachycardia, tachypnoea, and ataxia, all of which improved rapidly when they were moved outside. In the second episode, comments were made about the difficulty of making a differential diagnosis between dehydration, AMS, and CO poisoning.[11]

Two male climbers were fatally poisoned (postmortem carboxyhaemoglobin [COHb] levels of 57% and 66%) by a butane stove inside their tent at 4300 m (14 100 feet) on Mount McKinley in 1988. They were discovered with their heads near the vestibule/stove 24 hours after retiring to their tent to cook. The stove was found to be one-quarter turned on, presumably for a low simmer, and the tent was zipped up with all the vents closed because the snowfall had been heavy.[2]

An account of CO poisoning inside a tent on Mount Everest's South Col has recently been reported.[12]

CO toxicity

Basal serum COHb concentration is about 0.7% as a result of haem catabolism,[13] although half of nonsmokers have COHb concentration greater than 1.5%.[14] Carboxyhaemoglobin concentration may reach 4% to 6% in persons with diseases or drugs that increase haemolysis or haem catabolism such as haemolytic anaemia, 8% to 16% after using paint stripper, and up to 20% in heavy smokers.[14] Outside urban air has been noted to have a CO concentration up to 3 ppm.[15]

Carbon monoxide is a chemical asphyxiant gas with a haemoglobin affinity 200 to 250 times greater than that of oxygen (O_2). Carbon monoxide also interferes with cellular oxidation by binding myoglobin, cytochrome oxidase, cytochrome P-450, hydroperoxidases, and other haem proteins. Because it has an affinity for tissues with a high O_2 demand, its main targets are the neurologic system, cardiac tissue, and fetus.[16] The half-life of COHb is 4 to 5 hours at sea level, 80 minutes with 100% O_2 at sea level, and 23.5 minutes with 100% O_2 at a pressure of 3 atmospheres.[17]

Table 1. Predicted relationship of carbon monoxide (CO) concentration to carboxyhaemoglobin (COHb) concentration

CO, ppm	*COHb, %*
50	7
100	15
300	30
500	40
700	50

Carbon monoxide exposure causes a left shift in the O_2 dissociation curve with consequent lowered tissue extraction of O_2 as a result of a decrease in 2,3-diphosphoglycerate levels.[16,18,19]

Probable COHb concentration from a given CO exposure can be calculated by the Coburn-Forster-Kane equation.[20] Although individual cases are highly variable, this rough relationship between atmospheric CO concentration and COHb concentration has been supported with experimental human work (Table 1).[14]

Relating symptoms to exposure can be difficult because it is a function of CO concentration and time. However, mildly reduced maximum physical performance, reduced mean exercise time before exhaustion, and reduced maximum aerobic power can be noted with as little as 1 hour of exposure to 100 ppm (D. Smith, unpublished data).[21] The first consistent subjective and objective evidence of CO toxicity is seen with COHb concentration greater than 15%. The first symptom is a barely perceptible frontal headache, the appearance of which is delayed if the subject is sedentary. The risk of insidious COHb accumulation is therefore less if subjects are active.[17] Table 2 summarizes some of the symptoms and their relationship to exposure (D. Smith, unpublished data).[16,17]

Occupational exposure limits in the UK are set by the health and safety executive to avoid a COHb concentration greater than 5% in healthy nonsmokers. The maximum acceptable exposures are 200 ppm for 15 minutes (short-term exposure limit) and 30 ppm (time-weighted average) for 8 hours.[16] Other sources state that in cases of extreme emergency, 200 ppm could be tolerated virtually indefinitely and certainly up to 24 hours without leading to collapse, which requires levels of 300 ppm (Ministry of Defence, unpublished data). To put these figures into context, 25 ppm is commonly encountered on major roads in urban areas and can reach 100 ppm during weather inversions.[14]

Table 2. Symptomatology and CO Exposure*

	COHb concentration, %	*CO concentration ppm/exposure time*
Asymptomatic	0–20	100/8 h
Mild frontal headache	10–20	100/8 h to 200/4 h
Headache ± tachycardia	20–30	500/2 h
Steady symptom progression		200–1200
Collapse	30–40	300
Coma	50–60	
Chemical asphyxiant actions		>1000
Death in 2 h	60–70	650–1000
Death <1 h	80–90	2000
Death in few min	90–100	4000
Death in 10 min		8000
No symptoms before collapse		10 000
Fatal arrhythmia before high COHb concentration		50 000

*CO indicates carbon monoxide; COHb, carboxyhaemoglobin.

Previous work—methodology

Previous studies have investigated CO production either inside small experimental models with varying degrees of ventilation or inside tents and snow caves. The main experiments are prospective observational studies, and their methodologies are summarized below.

SNOW CAVE AND TENT MODELS

Henderson and Turner[22] investigated different combustion conditions with a Primus fuel-efficient stove system (as used on Antarctic expeditions) inside a sealed 1000-L iron box. An elaborate arrangement of 3 pans (including 1 ring-shaped pan) completely encircled the stove. All pans were filled with snow, and the stove burned until it became extinguished.

Pugh[6] used a Primus stove inside a 100-L metal drum to compare a free-burning stove with a stove melting ice chips. An inlet pipe forced air in at 300 L/min, and the exhaust gases were collected and analyzed.

Smith studied the CO production of a Coleman Peak stove burning on a low setting in a reduced O_2 atmosphere with no cooking involved. A 200-L Perspex box was linked to an O_2 cylinder to control O_2 levels in 3 bands: 17% to 18%, 18% to 19%, and greater than 19%.

Schwartz et al[10] used an unventilated 400-L cardboard box as a model of a snow cave to compare CO production rates from a Sigg stove for different fuels while heating water for 5 minutes. It provided several benefits: easily controlled experimental conditions, a constant level of ventilation, ease of ventilating to a baseline of 0 ppm, and rapid CO accumulation.

Leigh-Smith et al[23] modified the model of Schwartz et al to allow a minimal degree of ventilation from the bottom of the box. This had the benefit of providing an excess of O_2 for combustion, which allowed an indefinite burn time with no stove self-extinction while retaining the ability to demonstrate varying CO production levels under different conditions.

EXPERIMENTS IN TENTS AND SNOW CAVES

Irving et al[5] compared the COHb concentration of people inside 2000-L impervious tents, 2000-L permeable cotton tents, and snow caves in a series of uncontrolled experiments on Mount Washington (New Hampshire, United States) during the winter. He studied a variety of burn schedules with a kerosene-fuelled Primus stove.

During the 1956 to 1957 Trans Antarctic Expedition, Pugh[6] compared COHb concentration in tent occupants continually melting snow for 2 hours with those merely heating their tents for 3 hours. He used a Primus stove in partially ventilated, double-walled, 2500-L tents made of heavy cotton fabric. He also measured the CO produced while melting ice for 3 hours inside a lightweight single-walled pyramid tent in a laboratory.

Turner et al[24] measured CO concentration during 1- to 2-hour cooking periods with Optimus and MSR stoves in snow caves, tents, and igloos at altitudes from 2000 to 5200 m (6560–17 060 feet) while ascending Mount McKinley in 1988. He also investigated the minimum ventilation necessary to prevent toxic CO concentrations.

Harrigan[25] conducted a series of field and chamber trials inside tents with a Coleman Peak stove at −10°C.

Smith used a Coleman Peak stove and naphtha fuel inside a partially ventilated, 5000-L tent. He compared CO concentration while merely heating the tent with CO concentration during a 20-minute ice melt at different

Table 3. Concentration of CO within tents*

Study	Duration, min	CO, ppm	
		Cooking	Stove alone
Smith	120–390	56	2
Harrigan[25]		50–180	10
Turner et al[24]		50–190	
Keyes et al[26]	60–150	33–95	19

*CO indicates carbon monoxide.

Table 4. Concentration of CO in tent and snow cave model experiments*

Study	CO, ppm	
	Maximum	Minimum
Smith	125	5
Schwartz et al[10]	>80	24
Henderson et al[22]	200	40
Pugh[6]	33	>1
Leigh-Smith et al[23, 29]	53	8

*CO indicates carbon monoxide.

ambient temperatures. Smith's ambient conditions varied from 15°C to a cold chamber at −10°C with the flysheet covered in ice. Test durations were 2 to 6 hours, and during 1 test the researcher was in the tent.

Keyes et al[26] investigated the CO concentration within snow caves and the COHb concentration in 22 healthy volunteers during and after cooking in snow caves at 3200 m (10 500 feet) in Colorado. Unfortunately, many features varied, including snow-cave volumes (5000–12 000 L), times of cooking (60–150 minutes), time after cooking to measurement of CO concentration and COHb concentration (2–109 minutes), type of stove, and size of ventilation hole.

Previous work—summary of findings

The findings in different studies, despite lack of standardization and control in some, tend to be consistent. Table 3 summarizes the CO concentration found within tents and snow caves and shows that very similar ranges were detected. A 4-man tent has approximately a 5000-L volume (D. Smith, unpublished data), and the snow cave–tent model studies can be compared if CO concentration is adjusted to this volume (Table 4). Interestingly, despite the use of very different, sometimes elaborate, models in these experiments, the results are in the same range and are similar to those found in tents and snow caves. None of the CO concentrations in these experiments exceeded 300 ppm, the level thought necessary to cause a person to collapse (Ministry of Defence, unpublished data). The reason for deaths despite this observation is discussed below.

Experiments show that in poorly ventilated environments, 2 phases in the rate of CO production occur: An initial slow linear phase is followed by an exponential rate of increase before the flame is extinguished (D. Smith, unpublished data).[27] This pattern is probably caused by low 0_2 and high carbon dioxide levels within the experimental model, which may be caused by either an inadequate air supply to the flame or inadequate exhaust ventilation (D. Smith, unpublished data).[27] These experimental findings could feasibly occur within poorly ventilated snow caves and tents.

Irving et al[5] appear to be the first to mention the prevention of CO accumulation by improved tent ventilation through permeable tent fabric or by wind. They noted up to 18% COHb concentration in occupants of poorly ventilated snow caves and impervious tents. Pugh[6] noted that air and CO diffusion through tent walls might be abolished by snow, ice accumulation, and condensation. He also concluded that a lightweight single-walled pyramid tent had adequate ventilation after demonstrating only 30 ppm CO while melting ice for 3 hours inside this tent in a laboratory. Turner et al[24] have since suggested that 50 cm^2 (about ski-basket size) is the minimum effective vent-hole size for a snow cave and have noted 50 times quicker air-exchange rates (ventilation) in tents than in snow caves. They recorded their highest CO concentration while in snow caves and pointed out the decreased ventilation of tents in zero-wind conditions.

Carbon monoxide production is lowest with a freely burning flame and is increased by cooking or pan contact with the flame (D. Smith, unpublished data).[6,22,23,25–28] Keyes et al[26] found a small but statistically relevant ($P < .0005$) increase in ambient CO concentration (2–19 ppm) during and after cooking in snow caves, whereas Turner et al[24] noted CO concentrations greater than 50 ppm 60% of the time during 1- to 2-hour meal preparation in a variety of tents, igloos, and snow caves. Carbon monoxide production has been shown to be higher while melting snow or ice than when the flame is freely burning (D. Smith, unpublished data).[6]

Elevated COHb concentration has been found with cooking when compared with simply heating the tent or snow cave. Keyes et al[26] detected a small but statistically relevant ($P < .0005$) increase in COHb concentration (0.3%–1.2%) during and after cooking. Pugh[6] found COHb concentration increased from 5% to 10% with cooking, but a continual low level of CO exposure from above-snow vehicles biases these results.

When a blue flame is present, no difference in CO production occurs while heating a pan of ice or water.[23] Elevated CO production seems to occur only when objects placed on the stove disperse the flame, whether the object is a pan, rock,[27] or aluminium block.[28] In Pugh's studies,[6] CO production was not reduced by allowing the ice to melt or bringing the water to near boiling. However, he also seems to partially refute this with the observation that inserting a sheet of asbestos between flame and pan prevents CO production, although this is mentioned only briefly in the article.

Larger pans have been observed to increase the rate of CO production inside tents.[25] Recent work confirms a highly significant ($P < .017$) increase in CO production when pan diameter is increased from 165 to 220 mm while using a camping stove with a maximum blue flame to heat water for 5 minutes.[29]

Carbon monoxide production has been observed to be highest with low flame settings[25]; fatal levels could be reached only experimentally with low flame settings.[27] Irving et al[5] and Leigh-Smith[23] noted that in conditions of poor ventilation, stoves failed to stay lit with a maximum flame but continued to burn and produce CO with a low flame. Further evidence of the dangers of low flames and simmering may come from the low stove setting found after the fatalities on Mount McKinley.[2]

Carbon monoxide production is also markedly raised when a previously blue flame flares yellow, which may be more likely when pans are heated.[23]

Schwartz et al[10] demonstrated significant differences in CO production by different fuels in camping stoves; kerosene produced markedly more CO than did unleaded gasoline or white gas.

Carbon monoxide production has also been noted to increase with lower ambient temperatures while cooking (D. Smith, unpublished data).

Discussion

The 2-phase production of CO in poorly ventilated environments can cause a paradox in which partial but inadequate ventilation of a combustion area, by allowing CO production to continue instead of extinguishing the flame, may actually produce higher CO concentration than would no ventilation at all.[27] When cooking in an airtight room with a Primus stove, the low O_2 has been hypothesized to extinguish the stove before CO concentration reaches dangerous levels.[22] This has been observed in impervious tents.[5] Ventilation clearly has to be adequate because limited ventilation could actually be dangerous.

The ventilation point for CO egress must be as high as possible in the tent or snow cave. The temperature of the combustion gas mixture and the molecular weight of CO, which is slightly lower (28 g/mol) than air (28.9 g/mol), tend to cause CO accumulation toward the roof.[23] A low point for O_2 ingress could establish a continuous flow of gases through the tent. Oxygen can enter the tent or snow cave through permeable walls, snow tunnel, or low ventilation point as a result of diffusion down a concentration gradient, and the CO can escape through a ceiling ventilation port.

A flame-cooling effect by pans has been postulated to be responsible for the increased CO production with cooking: The larger the pan, the greater the cooling effect.[22,25] In view of the absence of any significant difference in CO production when relatively small pans of ice or water are heated, but with a significant increase with larger-diameter pans (whatever the medium inside), this seems unlikely—at least if a blue flame is maintained.[23,29] Flame dispersal may be a more-consistent explanation for the increased CO production with cooking.[23] The difference between the small blue flame of the stove alone and the large, diffuse flame that spreads out across the base of a pan and extends up its sides is quite marked. The finding of higher CO with low flame settings[25] supports this explanation; higher flame settings cause smaller, hotter flames with camping stoves.

In contrast to common advice,[11] using a stove alone for a short period to merely heat a tent appears relatively safe, providing there is nothing touching the flame and it burns with a maximum blue flame.

Yellow flames consistently produce very high CO concentrations,[23] and their presence is a useful visual indicator of potential CO danger for tent occupants. Although there is no direct correlation between the temperature of the medium within the pan and the CO production, the postulated increased incidence of yellow flames combined with the longer time required to boil a pan of ice may increase the risk of CO poisoning when melting snow.[23]

The lack of any experimental CO concentration above 200 ppm despite a number of deaths is indicative of the complexity of CO toxicity for humans, as described by the Coburn-Forster-Kane equation.[20] This equation takes into account exposure duration, atmospheric CO concentration, alveolar ventilation, blood volume, barometric pressure, lung diffusivity for CO, rate of endogenous CO production, and alveolar partial pressure of O_2. The most important of these are exposure duration and atmospheric CO concentration,[14] but because COHb is cumulative, prolonged exposure to low CO concentration is more dangerous than brief exposure to high levels.[30] Camping conditions can easily lead to increases in a number of these factors. Occupant respiration and combustion inside poorly ventilated tents can cause lowering

Table 5. Risk factors for CO poisoning and recommendations for avoidance*

Risk factor for CO poisoning	*Recommendation for avoidance*
Cooking	Avoid prolonged simmering Keep stove highly pressurised Use a maximum blue flame and avoid low flames Use small-diameter pans Use white, pure fuels
Yellow flame	Turn stove off, repressurize, relight Maximum tent ventilation for few min
Inadequate ventilation causing: 1. Lowered O_2 and incomplete combustion 2. CO buildup 3. CO_2 buildup exacerbating incomplete combustion	Ventilation area at least 50 cm^2 Ventilation CO egress port as high as possible Ventilation O_2 ingress port sited low Avoid minimal ventilation paradoxically elevating CO concentration Note higher CO risk in tents in zero-wind conditions
Insidious onset if sedentary Duration of CO exposure Stale air in tents (low O_2)	Beware headache and tachycardia Regular trips outside to unmask symptoms Ventilate tent at regular intervals Ventilation does not have to be continuous
Dehydration	Good hydration
Snow holes worse than tents Altitude Hyperventilation	Attention to above recommendations
Tent icing and snow cover	Attempt to keep tent fabric porous by regular clearing

*CO indicates carbon monoxide; O_2, oxygen; CO_2, carbon dioxide.

of the alveolar partial pressure of O_2 because of a lowering of its partial pressure within the tent. This low O_2 partial pressure within the tent also exacerbates incomplete combustion when the stove is burning. Blood volume may be lowered by dehydration.

Altitude deserves special mention, as the risk of CO toxicity is greater with increasing altitude. Carbon monoxide is thought to have an additive hypoxic effect with altitude for a variety of reasons, including direct additive hypoxic effect of the COHb,[24,31,32] increased CO uptake secondary to altitude hyperventilation,[33] a linear increase in endogenous CO production as a result of secondary polycythemia with increased haemolyis,[34] a greater CO sink because of the polycythemia,[34] and lengthened CO half-life.[35] Increased ventricular ectopy has also been noted with the additive hypoxic effects of altitude and CO exposure,[36] which may represent an added danger to persons exposed to CO at altitude. Predicting COHb concentration at altitude requires modification of the Coburn-Forster-Kane equation to allow for exogenous and endogenous CO production.[37]

Conclusion

Case reports verify that CO poisoning within tents and snow caves is a real and probably overlooked problem. It is potentially an even greater problem at altitude because of the multiplicity of risk factors for CO toxicity. Despite multiple anecdotal reports of climbers perishing from CO poisoning on Himalayan peaks[26] circulating in climbing circles, the danger does not appear to be widely recognized.

Diagnosing CO poisoning in the early stages may be difficult because of the nonspecific nature of symptoms and (at altitude) their similarity to AMS. The masking of symptoms when subjects are sedentary exacerbates the problem, and these are likely to be the occasions when individuals are subjected to the highest CO levels, such as resting and cooking in tents for hours during inclement weather. All attempts must be made to prevent COHb concentration reaching dangerous levels. Some of the evidence on how to do this is well founded; some is fairly poor. Table 5 summarizes the risk factors and provides some recommendations on how to avoid them. Opportunities for research in this interesting and very relevant area are abundant.

Safety could be enhanced by the use of small portable CO detectors. We hope to see no more case reports of healthy, fit young people dying from an entirely preventable cause.

References

1. Cohen MA. Air pollution exposures to campers inside of tents: A study of the use of camping stoves and lanterns. Proceedings International Specialty Conference Indoor Air Quality Cold Climates. Ottawa, Ontario, Canada; May 1985.
2. Foutch RG, Henrichs W. Carbon monoxide poisoning at high altitudes. *Am J Emerg Med.* 1988;6:596–598.
3. Byrd R. *Alone*. New York, NY: Adventure Library; 1938.
4. Stefansson W. *Unsolved Mysteries of the Arctic.* New York, NY: Macmillan; 1939.
5. Irving L, Scholander P, Edwards G. Experiments on carbon monoxide poisoning in tents and snow houses. *J Ind Hyg Toxicol.* 1942;24:213.
6. Pugh L. Carbon monoxide hazard in Antarctica. *BMJ.* 1959;34(5116):192–196.
7. Girman JR, Chang YL, Hayward SB, et al. Causes of unintentional deaths from carbon monoxide poisonings in California. *West J Med.* 1998;168:158–165.
8. Rupp W, Nadjem H, Thoma KH. Alcohol stove as a source of CO poisoning in a camper [in German]. *Arch Kriminol.* 2000;206:8–13.
9. Centers for Disease Control and Prevention. Carbon monoxide poisoning deaths associated with camping–Georgia, March 1999. *JAMA.* 1999;282:1326.
10. Schwartz RB, Ledrick DJ, Lindman AL. A comparison of carbon monoxide levels during the use of a multi-fuel camp stove. *Wilderness Environ Med.* 2001;12:236–238.
11. Seibert R. Climbs and expeditions—Alaska. *Am Alpine J.* 1986;28:139–142.
12. Krakauer J. *Into Thin Air—A Personal Account of the Everest Disaster.* Chatham, UK: Pan Books; 1997.
13. Coburn RF, Blakemore WS, Forster RE. Endogenous carbon monoxide production in man. *J Clin Invest.* 1963;42: 1172–1178.
14. Stewart RD. The effect of carbon monoxide on humans. *Ann Rev Pharm.* 1975;15:409–423.
15. Dubois L, Zdrojewski A, Monkman JL. The analysis of carbon monoxide in urban air at the ppm level, and the normal carbon monoxide value. *J Air Pollut Control Assoc.* 1966;16:135–139.
16. Health and Safety Executive, UK Government. Carbon monoxide: health hazards and precautionary measures. *Guidance Note.* 2nd ed. Sudbury, Ontario, Canada: HSE Books; 1998.
17. Stewart RD, Peterson JE, Baretta ED, et al. Experimental human exposure to carbon monoxide. *Arch Environ Health.* 1970;21:154–164.
18. Astrup P. Intraerythrocytic 2,3-diphosphoglycerate and carbon monoxide exposure. *Ann N Y Acad Sci.* 1970;174: 252–254.
19. Thomas MF, Penney DG. Hematologic responses to carbon monoxide and altitude: a comparative study. *J Appl Physiol.* 1977;43:365–369.
20. Coburn RF, Forster RE, Kane PB. Considerations of the physiological variables that determine the blood carboxyhaemoglobin concentration in man. *J Clin Invest.* 1965; 44:1899–1910.
21. Rylander R, Vesterlund J. Carbon monoxide criteria. With reference to effects on the heart, central nervous system and fetus. *Scand J Work Environ Health.* 1981;7(suppl 1): 1–39.
22. Henderson Y, Turner J. Carbon monoxide as a hazard of polar exploration. *Nature.* 1940;145:92–95.
23. Leigh-Smith S, Watt I, McFadyen A, et al. Comparison of carbon monoxide levels during heating of ice and water to boiling point with a camping stove. *Wilderness Environ Med.* 2004;15:164–170.
24. Turner WA, Cohen MA, Moore S, et al. Carbon monoxide exposure in mountaineers on Denali. *Alaska Med.* 1988; 30:85–90.
25. Harrigan M. *A Study of Carbon Monoxide Exposure Amongst Troops During Arctic Training* [Master's thesis], 1992.
26. Keyes LE, Hamilton RS, Rose JS. Carbon monoxide exposure from cooking in snow caves at high altitude. *Wilderness Environ Med.* 2001;12:208–212.
27. Westerlung K, von Ubisch H. Carbon monoxide from small camping appliances and from stoves without chimney connection. *Nordisk Hygienisk Tidskrift.* 1972;53:26–33.
28. Prescher KE. Occurrence of carbon monoxide, carbon dioxide and nitrogen oxides during the use of gas stoves [in German]. *Schriftenr Ver Wasser Boden Lufthyg.* 1982;53: 191–198.
29. Leigh-Smith S, Stevenson R, Watt M, et al. Comparison of carbon monoxide levels during heating of water to boiling point with a camping stove using different diameter pans. *Wilderness Environ Med.* 2004;15:164–170.
30. Sokal JA, Kralkowska E. The relationship between exposure duration, carboxyhemoglobin, blood glucose, pyruvate and lactate and the severity of intoxication in 39 cases of acute carbon monoxide poisoning in man. *Arch Toxicol.* 1985;57:196–199.
31. Vollmer E, King G, Birren J. The effects of carbon monoxide on three types of performance at simulated altitudes of 10000 and 15000 feet. *J Exp Psychol.* 1946;36:244–251.
32. *Altitude as a factor in air pollution.* Research Triangle Park, NC: Environmental Protection Agency; 1978. U.S. EPA No. 600/9-78-015. Monograph.
33. Forbes WH, Sargent F, Houghton FJW. The rate of carbon monoxide uptake by normal men. *Am J Physiol.* 1945;143: 594–608.
34. McGrath JJ. Effects of altitude on endogenous carboxyhemoglobin levels. *J Toxicol Environ Health.* 1992;35: 127–133.
35. Ellenhorn M. Diagnosis and treatment of human poisoning. In: Ellenhorn MJ, Schonwald S, Ordog G, Wasserperger J, eds. *Ellenhorn's Medical Toxicology.* Baltimore, MD: Williams and Wilkins; 1997.
36. Leaf DA, Kleinman MT. Urban ectopy in the mountains: carbon monoxide exposure at high altitude. *Arch Environ Health.* 1996;51:283–290.
37. Collier C, Goldsmith J. Interactions of carbon monoxide at altitude. *Atmos Environ.* 1983;17:723–728.