Wilderness and Environmental Medicine, **12,** 208–212 (2001)

BRIEF REPORT

Carbon monoxide exposure from cooking in snow caves at high altitude

LINDA E. KEYES, MD; ROBERT S. HAMILTON, MD; JOHN S. ROSE, MD

Women's Health Research Center, University of Colorado Health Sciences Center, Denver, CO (Dr Keyes), Department of Emergency Medicine, Stanford University, Palo Alto, CA (Dr Hamilton), and Division of Emergency Medicine, University of California, Davis, CA (Dr Rose). This work performed while the authors were under the auspices of the Department of Emergency Medicine, Alameda County Medical Center, Oakland, CA (Drs Keyes and Rose), and the Department of Emergency Medicine, University of California, San Diego, CA (Dr Hamilton).

Objective.—To determine the physiological consequences of acute CO exposure from cooking in snow caves at 3200 m. We hypothesized that ambient CO and serum carboxyhemoglobin (COHb) levels would increase and that even low levels of COHb would be associated with symptoms of CO poisoning at high altitude.

Method.—This was a prospective observational study. Twenty-two healthy volunteers age 18 years or older were recruited during a winter camping trip at 3200 m. Subjects filled out symptom questionnaires, and heart rate (HR), oxygen saturation (SaO_2), serum COHb, and ambient CO were all measured before and after cooking inside snow caves.

Results.—Median age of subjects was 32 years, and 87% were male. The median ambient CO level increased by 17 ppm (IQR, 2–27 ppm), $P = .005$. Mean serum COHb level rose from 0.3% (IQR, 0.2%–0.4%) to 1.2% (IQR, 0.7%–2.6%) after cooking, for a difference of 1% (IQR, 0.4%–2.3%), $P < .001$. There were no differences in symptom scores before and after cooking, and there was no significant effect on HR or SaO_2.

Conclusion.—A single exposure to CO at 3200 m increases ambient CO and COHb but not to clinically important levels. Further studies are needed to examine the risks of longer exposures at higher altitudes.

Key words: carbon monoxide, high altitude, carboxyhemoglobin, air pollutants, environmental exposure, mountaineering, heating

Introduction

Mountaineering texts warn of the dangers of carbon monoxide (CO) exposure from cooking inside sealed tents or snow caves. In 1 case report, 2 Swiss mountaineers died on Denali of CO poisoning after cooking in their tightly sealed tent,[1] and in 1985, 2 Denali climbers required rescue after CO exposure induced acute mountain sickness.[2] Anecdotal reports of climbers perishing from CO poisoning on Himalayan peaks circulate frequently in climbing circles. However, the risk of CO exposure from cooking in enclosed spaces at high altitude has not been well studied.

The interaction between CO exposure and the effects of high altitude is not fully understood. Studies on rats breathing a fixed amount of CO demonstrate increased carboxyhemoglobin (COHb) levels with increasing altitude,[3] which suggests a potential for greater toxic effects of CO at higher altitudes. Thus, cooking in snow caves may be even more dangerous at high altitude.

We sought to determine the physiological consequences of acute CO exposure from cooking in snow caves with white gasoline stoves at 3200 m. We hypothesized that at 3200 m above sea level, cooking inside snow caves would increase both ambient CO and serum COHb and that even low levels of COHb would be associated with symptoms of CO poisoning.

Presented at the Society for Academic Emergency Medicine Annual Meeting, Boston, MA, May 19, 1999, and the World Congress on Wilderness Medicine, Whistler, British Columbia, Canada, August 8, 1999.

Corresponding author: Linda E. Keyes, MD, 3510 Endicott Dr, Boulder, CO 80305 (e-mail: Linda.Keyes@UCHSC.edu).

Materials and methods

This was a prospective observational cohort study. Twenty-two healthy volunteer subjects, age 18 years or older, were recruited from a mountain rescue group during their annual snow cave camping trip on Niwot Ridge in Boulder County, Colorado, at 3200 m. Written, informed consent was obtained. The Institutional Review Board at Alameda County Medical Center (ACMC) approved the study protocol. Laboratory and data analysis was performed at the Alameda County Medical Center, Highland Campus, in Oakland, CA.

Subjects ascended by car to 2300 m and by skis or snowshoes to the study site at 3200 m. Each snow cave had a single entrance and a single ventilation hole made with a ski pole or shovel handle. Snow cave volume was measured. All subjects cooked using white gasoline stoves of various brands and conditions. Subjects completed a questionnaire about demographics, medications, smoking, and 6 symptom variables: headache, nausea/vomiting, dyspnea, dizziness/lightheadedness, fatigue/weakness, and chest pain. Each symptom was measured pre- and postcooking on a 4-point ordinal scale (0 = none, 1 = mild, 2 = moderate, and 3 = severe), giving a score range of 0 to 18. Heart rate (HR) and arterial oxygen saturation were measured using a portable pulse oximeter with a finger probe (Ohmega Inc, Louisville, CO), and venous samples for COHb were drawn. Quantitative measures of ambient CO in parts per million (ppm) were made before and after cooking using a Toxi Plus single sensor gas detector (Biosystems Inc, Middletown, CT). We recorded the number of stoves per cave, burning time, and the time from stove being turned off until venous blood sampling.

Venous samples were stored on ice in heparinized tubes and then analyzed by spectrophotometric assay (Copenhagen Radiometer, Westlake, OH). Previous work has shown that venous COHb samples may be stored for up to 4 months without loss of the original CO saturation[4] and that venous and arterial levels are highly concordant.[5]

The Wilcoxon Signed-rank test was used to compare ambient CO levels, serum COHb levels, symptom scores, HR, and oxygen saturation before and after cooking. Results are expressed as median (interquartile range, IQR). Data analysis was performed using SAS 6.12 (SAS Inc, Cary, NC) and Statview 5 (SAS).

Results

Five campers chose not to participate for personal reasons. Twenty-three subjects were enrolled. One subject was excluded from the final analysis because he left prior to cooking due to a preexisting illness, for a total of 22 subjects. Nineteen (87%) were male. Median age was 32 years (IQR, 23–40). All subjects were nonsmokers. Median altitude of residence was 1687 m (IQR, 1656–1742). One subject was from sea level (31 m). Subject characteristics, pre- and postcooking COHb, HR, oxygen saturation, and symptom scores are listed in Table 1.

Subjects were distributed over 10 different snow caves. Levels of CO measured in snow caves are shown in Table 2. The median ambient CO level increased from 2 ppm (IQR, 2–2 ppm) to 19 ppm (IQR, 2–26 ppm). The median difference before and after cooking was 17 ppm (IQR, 2–27 ppm), $P = .005$. Levels of CO were measured directly over burning stoves in 5 caves. Median CO over burning stoves was 33 ppm (IQR, 8–52 ppm). Median burning time for stoves in all caves was 120 minutes (IQR, 60–150 minutes). Median time the stove was off prior to measuring ambient CO levels was 80 minutes (IQR, 2–109). No subject in cave 3 participated in having blood drawn, but CO measurements were made in this cave. There were no differences in CO levels measured at the cave floor and ceiling.

Effects of cooking in snow caves on COHb level and symptom scores are shown in Table 1. Increased ambient CO after cooking was accompanied by an increase in subjects' median serum COHb level from 0.3% (IQR, 0.2%–0.4%) to 1.2% (IQR, 0.7%–2.6%) after cooking, for a median difference of 1% (IQR, 0.4%–2.3%), $P < .001$. However, there was no change in symptom scores. No subject reported a score of greater than 6 (out of 18) before or after cooking. Cooking inside snow caves also had no effect on HR (precooking 90 bpm IQR [82–99] vs postcooking 81 bpm IQR [74–89]), or arterial oxygen saturation (precooking 90%, IQR [89–92] vs postcooking 89%, IQR [88–90]). The subject with the maximum COHb level postcooking (no. 22, 5.2%) also had the maximum CO level recorded over the stove (95 ppm).

Discussion

Our results demonstrate that both ambient CO and serum COHb rise after cooking in snow caves at 3200 m but not to values that are clinically important. Our study had several limitations that affect the interpretation of our results. The length of stove burning time and the time from cooking to measurement of ambient CO and serum COHb levels were not standardized. This lack of standardization certainly affected the ambient level of CO measured, and, along with different ventilation characteristics of each cave, likely explains the resulting high variability in postcooking CO measurements. Due to the time constraints upon the 2 investigators (L.E.K. and R.S.H.) drawing blood from 22 subjects across 10 caves

Table 1. Subject characteristics and data

Subject No.	*Age (yr)*	*Cave No.*	*COHb (% pre)*	*COHb (% post)*	*HR (pre)*	*HR (post)*	*SaO_2 (pre)*	*SaO_2 (post)*	*Sx (pre)*	*Sx (post)*
1	30	1	0.1	0.7	112	90	89	84	0	0
2	32	2	0.2	4.7	62	96	94	88	0	0
3	45	4	0.4	2.7	95	81	87	89	0	0
4	41	4	0.3	2.0	94	72	89	88	1	0
5	30	4	0.5	2.2	92	79	90	94	5	3
6	45	5	0.3	1.1	80	65	90	90	2	1
7	51	5	0.4	2.0	90	77	87	91	0	0
8	31	5	0.3	1.3	96	70	92	91	0	1
9	23	6	0.3	0.7	100	74	90	89	2	1
10	23	6	0.3	0.5	82	57	91	91	1	2
11	20	6	0.2	1.1	61	78	80	93	0	2
12	23	7	0.5	3.4	84	62	87	89	0	1
13	40	7	0.5	1.2	90	75	92	89	1	1
14*	35	7	0.5	3.1	100	89	89	90	1	0
15	34	8	0.2	0.9	106	88	89	89	0	0
16	32	8	0.4	0.5	110	94	85	88	5	5
17	33	8	0.1	0.8	81	82	89	88	5	4
18	31	9	0.2	0.6	70	86	93	86	1	2
19*	42	9	0.4	0.7	102	94	92	87	2	3
20*	22	10	0.2	2.5	85	83	92	89	2	2
21	23	10	0.4	2.6	86	81	94	90	6	1
22	38	11	0.2	5.2	90	100	92	90	3	4

*Female subjects indicated by *; all others male. HR indicates heart rate; Sx, symptom score on questionnaire.

under difficult field conditions, we could not control the timing. Snow cave size, ventilation characteristics, and the type and number of stoves used were left up to the individual campers. Although this added variability to our measurements, it allowed our study to represent a broad variety of true field conditions.

In contrast, the lack of standardized timing to obtaining postexposure serum COHb specimens probably had

Table 2. Snow cave characteristics and data

Cave No.	*Cave volume (L)*	*No. of stoves*	*CO pre (ppm)*	*CO over stove*	*CO post ceiling (ppm)*	*CO post floor (ppm)*	*Burn time (min)*	*Time off (min)*
1	5600	1	2	*	2	3	80	106
2	6000	1	2	*	22	19	60	120
3	8200	*	2	*	38	37	120	*
4	12 000	2	2	7	35	21	150	109
5	7200	1	2	*	8	9	135	0
6	4100	1	1	9	3	28	120	80
7	9500	1	2	33	3	4	60	108
8	4500	3	2	37†	16	10	200	2
9	3000	1	2	*	3	3	60	157
10	6700	1	2	*	22	19	60	0
11	6400	1	*	95	*	*	*	*

*Indicates no measurement made. Burn time is time stove(s) was burning, and time off indicates length of time stove(s) off prior to measurement of postcooking CO levels and COHb.

†Measurement made over 1 stove only.

little effect on measured serum COHb. The half-life of COHb is 4 to 6 hours at sea level and is much longer at 3200 m.[6] Therefore, it is unlikely that the COHb decreased greatly before samples were drawn. It is possible that subjects whose stoves were still burning at the time of COHb sampling had not yet reached their peak levels. Longer exposures might have resulted either in higher COHb levels or more clinical symptoms. None of the residents of the cave with the highest ambient CO (cave 3, peak 38 ppm) participated in the study. Therefore, we may have missed some subjects who had a clinically significant exposure. Although no subject reported adverse symptoms or had a change in symptom score after cooking, we did not perform mental performance or neuropsychological testing, which are more sensitive indicators of CO poisoning. Also, our symptom score has not been validated for CO poisoning.

Turner et al[7] also measured ambient CO, but not COHb, in a tent, igloos, and snow caves on Denali at altitudes between 2000 and 5200 m. These measurements, made during and after meal preparation, showed CO levels as high as 190 ppm in 1 cave. We did not always measure CO while stoves were burning, as was done in Turner's study, which may explain why their CO measurements were higher. Differences in stove type and ventilation are also likely important. The Denali study demonstrated that increasing ventilation hole size increased air exchange and lowered CO.[8] We did not measure the size of our ventilation holes or the time for CO diffusion out of the caves.

The threshold limit value for CO in industrial exposures is 35 ppm for an 8-hour workday and a maximum COHb of 5%.[6] A concentration of 200 ppm is considered the ceiling level to which a worker may be exposed transiently without raising COHb level.[6] The ambient CO in some caves reached levels that may have resulted in exposures above these limits (caves 3, 8, and 11), and 1 subject (no. 22) had a COHb that exceeded the industrial standard.

We did not observe any signs of CO toxicity in our study. In general, symptoms of CO toxicity are not thought to manifest until COHb levels rise above 10%.[6] The maximum we measured was 5.2%. For perspective, the average smoker has a COHb level around 2%.[9] However, our findings should be interpreted with caution. Rylander and Vesterlund[10] noted in their review of the cardiovascular effects of CO that in healthy nonsmokers, a 1-hour exposure of 100 ppm was associated with a COHb of 3.95% and a decreased mean exercise time until exhaustion. They also cited a similar study in which maximum aerobic power was decreased at COHb levels of 2.5% after a short exposure to 50 ppm.[10] Exercise tolerance and aerobic power are important concerns for mountaineers, and some of our subjects achieved COHb levels similar to these. Another investigation at 2100 m demonstrated a doubling of exercise-induced ventricular ectopy in men with coronary artery disease exposed to CO and a mean COHb level of 3.91%.[8] These studies suggest that lower levels of COHb may have important effects that we did not measure.

Another theoretical concern about CO exposure in high altitude snow caves is that the increased half-life of CO at altitude may allow for cumulative increases in COHb over multiple stove burns during the course of a trip. This pertains especially when mountaineers may be ''holed up'' in closed shelters waiting for bad weather to clear. The half-life of COHb depends on the partial pressure of oxygen and decreases as the partial pressure of oxygen rises.[6] Conversely, COHb will persist longer at high altitude where the partial pressure of oxygen is lower. We could not examine the possibility of cumulative increases in COHb in the single exposure design of our study. Our results also may not apply to similar exposures at higher altitudes, where people are more hypoxemic at baseline. In these conditions, the additional reduction in oxygen saturation due to COHb could potentially compromise adequate tissue delivery of oxygen and lead to illness.

Our results demonstrate that ambient CO and COHb rise after cooking in snow caves at high altitude. However, this exposure did not result in measurable symptoms of toxicity. Therefore, we conclude that a single exposure after cooking in a snow cave at 3200 m or lower is probably safe. Further studies with larger sample sizes are needed to determine the safety of cooking in enclosed spaces at higher altitudes, with longer cooking times and multiple exposures.

Acknowledgments

The authors wish to thank Christopher J. DiGiano, PhD, for his technical assistance during the study and E. John Gallagher, MD, for his critical review of the manuscript and assistance with statistical analysis. The authors thank the members of the Rocky Mountain Rescue Group for their participation.

References

1. Foutch RG, Henrichs W. Carbon monoxide poisoning at high altitudes. *Am J Emerg Med.* 1988;6:596–598.
2. Seibert R. Climbs and expeditions—Alaska. *Am Alpine J.* 1986;28:139–142.
3. McGrath JJ. Effects of altitude on endogenous carboxyhemoglobin levels. *J Toxicol Environ Health.* 1992;35:2, 127–133.

4. Ocak A, Valentour JC, Blanke RV. The effects of storage conditions on stability of carbon monoxide in post mortem blood. *J Ann Toxicol.* 1985;9:202–206.
5. Touger M, Gallagher EJ, Tyrell J. Relationship between venous and arterial carboxyhemoglobin levels in patients with suspected carbon monoxide poisoning. *Ann Emerg Med.* 1995;25:481–483.
6. Ellenhorn MJ. *Ellenhorn's Medical Toxicology, Diagnosis and Treatment of Human Poisoning*. Baltimore, MD: Williams and Wilkins; 1997.
7. Turner WA, Cohen MA, Moore S, Spengler JD, Hackett PH. Carbon monoxide exposure in mountaineers on Denali. *Alaska Med.* 1988;30:85–90.
8. Leaf DA, Kleinman MT. Urban ectopy in the mountains: carbon monoxide exposure at high altitude. *Arch Environ Health.* 1996;51:283–290.
9. Wald NJ, Idle M, Boreham J, Bailey A. Carbon monoxide in breath in relation to smoking and carboxyhaemoglobin levels. *Thorax.* 1981;36:366–369.
10. 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.