BRIEF REPORT

Peripheral Responses to Cold: Case Studies From an Arctic Expedition

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Objective.—An Arctic expedition provided an opportunity to examine the interaction between cold injury and peripheral acclimatization. The conditions were similar to those during which acclimatization has been demonstrated, yet they were also conducive to development of peripheral cold injury.

Methods.—Extremity digit temperatures were measured during 30-minute peripheral cold-water (4°C) immersion (CWI) in 2 explorers (R.G. and T.L.) before and after a 109-day Arctic ski trek (average $T_{\text{air}} = -21^{\circ}\text{C}$). This self-supported trek involved carrying heavy backpacks (up to 45 kg) and hauling sleds (~100 kg).

Results.—During the expedition, the explorers' hands did not sustain frostbite, but their feet developed moderate trench foot. Unexpectedly, both men exhibited lower mean finger temperatures during CWI after the expedition in right and left hands (R.G. by 0.9°C and 0.2°C ; T.L. by 1.8°C and 1.1°C), suggesting peripheral acclimatization was impaired. In contrast, mean toe temperatures during CWI were warmer in both right and left feet for T.L. (by 3.6°C and 2.3°C) and in the left foot for R.G. (by 1.3°C) postexpedition. There was no change in R.G.'s right toe mean temperature.

Conclusions.—We speculate that prolonged heavy load carriage may have impaired blood flow or nerve conduction in the hands and inhibited acclimatization. Our data also suggest that despite incidence of moderate trench foot, acclimatization can still occur after resolving this injury.

Key words: cold-induced vasodilation, peripheral blood flow, acclimatization, trench foot, load carriage

Introduction

People who sojourn to cold-weather climates often develop an acclimatization whereby they maintain warmer extremities. However, cold injury may interfere with acclimatization because it is associated with a blunted temperature response to cold. One of the challenges of working in a cold environment is the vasoconstriction that occurs in the extremities, with associated pain, reduced performance, and increased risk of cold injury.

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Cold-exposed extremities initially respond with sympathetically mediated vasoconstriction that lowers skin temperature.^{1,2} After several minutes of continued local cold exposure, a cold-induced vasodilation (CIVD) typically occurs, reflected in a rise in skin temperature. The subsequent increase in blood flow raises the local temperature until vasoconstriction occurs again after several minutes. With ongoing cold exposure, alternating periods of vasoconstriction and vasodilation produce a pattern of increasing and decreasing skin temperature.^{1,3} The CIVD is believed to result when diminished blood flow reduces local tissue temperature, causing reduced norepinephrine release and relaxation of the blood-vessel walls.² Because it is associated with higher mean digit temperature, a strong CIVD response has been suggested to improve manual performance in the cold⁴ and provide protection from peripheral cold injury.⁵

People who experience repeated peripheral cold exposure while their body core remains warm typically develop enhanced CIVD.⁶ Laboratory studies, using protocols of repeated hand immersion in cold water, have

demonstrated higher mean finger temperatures and an earlier onset of CIVD,⁷⁻⁹ as well as less pain.⁸ Field studies from geological workers in Antarctica¹⁰ and from Arctic ski trekkers^{11,12} have also reported higher mean finger temperatures during cold exposure and earlier onset of CIVD, as well as less pain and improved manual dexterity,¹³ after their sojourns as compared with before their sojourns. Because the subjects in those studies were active and wore cold-weather clothing, the primary cold stimulus was repeated local exposure of the extremities, rather than whole-body cooling. The type of exposure is important because the local CIVD response may be modulated by central effects. For example, when core or mean skin temperatures are reduced, the CIVD response is blunted,¹⁴ presumably due to the high overall sympathetic activation associated with whole-body cooling.

Research suggests that peripheral cold injury blunts CIVD, and this effect may persist for several months after the injury.¹⁵ Taylor and Kulungowski¹⁵ reported that 65% of patients with frostbite still had symptoms 6 months after injury, including cold sensitivity, numbness, pain, or hyperesthesia. Likewise, Arvesen et al¹⁶ found delayed onset of CIVD in soldiers who continued to experience cold intolerance 3 to 4 years after they had sustained cold injury to their hands or feet. No study has documented the CIVD response of an individual both before and after cold injury. Although it is often assumed in these studies that a blunted CIVD response is due to cold injury, the comparison is made only with a control group. This assumption overlooks the possibility that the individual may have had a poor CIVD response initially, a trait which might increase susceptibility to peripheral cold injury. In addition, no study has examined the influence of peripheral cold injury on acclimatization of CIVD.

To investigate the potential interaction between cold injury and CIVD acclimatization in the hands and feet, we studied 2 explorers before and after a 109-day expedition across the Arctic Ocean (AO2000). During previous expeditions, including a 2938-km trek from southern to northern Greenland in 1994, these explorers had experienced moderate cold injury (finger frostnip, hand paresthesia, and trench foot). Symptoms associated with these injuries had resolved before beginning the present expedition; therefore, though this study does not allow comparison with a condition of no prior cold injury, the effect of new or recurrent injuries could be observed. Thus, AO2000 presented a potential opportunity to study digit responses to cold before and after cold injury, as well as to examine the interaction between peripheral cold acclimatization and cold injury.

Methods

The US Army Research Institute of Environmental Medicine Scientific and Human Use Review Committees and the Medical Research and Materiel Command Human Subjects Research Review Board approved this study. Investigators adhered to Army Regulation 70-25 and US Army Medical Research and Materiel Command Regulation 70-25 on the use of Volunteers in Research. Written informed consent was obtained from the subjects for their participation in this testing after the purpose, experimental procedures, and known risks of the study had been explained to them. The 2 subjects who participated in this study were experienced with Arctic expeditions.

EXPEDITION

Two 28-year-old Norwegian Navy SEALs (R.G. and T.L.) completed a 2000-km ski trek across the Arctic Ocean via the geographic North Pole, leaving Cape Arctichesky, Siberia, on February 16, 2000, and arriving at Ward Hunt Island, Canada, on June 3, 2000. The expedition was self-supported; they transported all supplies required for the duration of the trek and were not resupplied en route. Each subject started the expedition carrying a 20-kg backpack and hauling two 80- to 100-kg sleds in tandem. After 18 days, the diminished food and fuel supply allowed them each to drop 1 of their sleds, leaving each subject with a single 140- to 150-kg sled and a 20-kg backpack. At 88 days, they dropped the remaining sleds and completed the expedition, each carrying a 40- to 45-kg backpack.

PROTOCOL

Subjects were tested 4 weeks before the expedition began and within 4 days after the expedition ended. Body stature was measured using a stadiometer, and nude body mass was measured on an electronic platform scale. Lean body mass and body fat mass were determined using a dual X-ray absorptiometer (Lunar DPX-L, Lunar Corporation, Madison, WI).

Tests for CIVD were conducted at the same time of day (R.G.: 0700 hours, T.L.: 1000 hours) before and after the expedition. The subjects refrained from alcohol intake and vigorous exercise for 24 hours and from caffeine and tobacco usage for 12 hours before each test. Ambient conditions during the tests were intended to be thermoneutral and were maintained at 27°C with 50% relative humidity. The subjects, wearing shorts and T-shirts, sat in a semisupine posture during the hand tests and lay prone during the foot immersions. The subjects'

movement and talking were minimal during the experiments.

The order of testing was right hand—right foot—left hand—left foot. The fingers were immersed to the second interphalangeal joint, and the feet were positioned such that each toe was fully immersed. Each test began with warm (42°C) water immersion (Neslab Instruments, Newington, NH) designed to abolish vasoconstriction¹⁷ and standardize initial digit temperature, such that any variability associated with initial vasomotor tone or finger heat content was minimized. After 10 minutes in warm water, the subject immediately transferred his hand or foot to cold (4°C) water for 30 minutes. The subjects were asked to rate their digit pain 5 minutes after cold-water immersion (CWI), using a continuous scale with anchors of “no pain” to “intolerable pain.”¹⁸ Pain data are expressed as percentage of maximum. Temperatures were recorded every 6 seconds.

Core temperature (T_{re}) was measured with a rectal thermistor (YSI, Yellow Springs, OH), which the subject placed 10 cm past the anal sphincter. Skin temperature was measured using thermistors (Concept Engineering, Old Saybrook, CT) attached to 4 sites (calf, thigh, chest, and triceps). Mean skin temperature (T_{sk}) was calculated according to the weighting $0.20 \times (\text{calf} + \text{thigh}) + 0.3 \times (\text{chest} + \text{triceps})$.¹⁹ Digit skin temperatures were measured using a type T (24 gauge) thermocouple wire (Pratt and Whitney, East Hartford, CT) attached along the nailbed of each digit.

During the trek, the subjects maintained diaries that included their observations regarding unusual cold exposure (eg, falling through the ice), signs and symptoms of cold injury, and discomfort in their hands and feet. They were questioned about their history of cold injury before the expedition and were debriefed in greater detail after the expedition. Cold injuries were classified according to the signs and symptoms described by Meryman.²⁰

STATISTICAL ANALYSIS

Because there were only 2 subjects in this study, no statistical analysis was conducted. Data at pre- and postexpedition with reference were compared, whenever possible, with previous studies that used similar methodology. Digit temperature data were smoothed using a 3-point running average to reduce noise, and a rise of at least 0.5°C was considered to represent a viable CIVD. These data were examined to determine the initial nadir temperature and the time at which that nadir temperature occurred or the onset of CIVD. Mean data are presented as the average during the last 25 minutes of CWI.

Results

Before beginning this expedition, both subjects had intentionally gained body fat in preparation for the extremely high level of energy expenditure. Each lost considerable body weight during the expedition (R.G.: from 91.8 to 78.2 kg, T.L.: from 114.5 to 89.1 kg), primarily as body fat (R.G.: from 19.5% to 5.8%, T.L.: from 24.5% to 5.9%).

The ambient temperature during the 109-day expedition ranged from -39°C at the beginning to -5°C at the end, averaging -21°C , with strong winds. The subjects reported that throughout the expedition they rarely shivered, and they felt whole-body chilling only transiently as they prepared to start skiing each morning and again at the end of the 5- to 10-minute rest period each hour. There were approximately 10 occasions when they felt cold at night, including 5 nights at the end of the expedition when they eliminated some sleeping bag and pad insulation to reduce backpack weight. In spite of these few instances of apparent whole-body cooling, the primary cold stimulus during this expedition was repeated local cooling of the extremities, which occurred frequently throughout the day as they stopped to eat, take photographs, repair equipment, or prepare their camp.

COLD INJURIES

Neither subject sustained frostbite on any digit. Both reported that after the first 2 weeks their hands continuously felt cold, including when the subjects were in their tent, and even as the ambient temperature increased toward the end of the expedition. As they began skiing, it often took 30 to 40 minutes of each 50-minute work period for their hands to feel warm. Upon return to our laboratory, their fingers no longer felt cold, but the subjects reported numbness that persisted for about 3 weeks. During the last 10 days of the expedition, when they carried their remaining supplies in $\sim 45\text{-kg}$ packs, T.L. noted numbness, pain, and decreased function in his right arm.

Both subjects sustained moderate trench foot as a result of using vapor barriers to protect the insulation in their boots and sleeping bags from moisture contamination. Their symptoms included pain, numbness, tingling, aching, and moist, cracking skin. As the ambient temperature increased ($> -20^{\circ}\text{C}$), the temperature inside the tent ($\sim 10^{\circ}\text{C}$ higher than ambient temperature) remained warm enough for moisture to evaporate, and after 8 to 9 weeks they were able to forgo using the vapor barriers in their sleeping bags at night, thus allowing their feet to dry. At this point, their symptoms disap-

peared, their damaged skin sloughed off, and their feet felt warmer thereafter. One subject (R.G.) experienced numbness in his right leg during the last 10 days of the expedition, when the backpack weight increased and he used a tighter waist belt to redistribute the weight from his shoulders to his hips.

TEST RESULTS

At the end of the expedition, both subjects rested, ate, and rehydrated as they traveled from Ward Hunt Island to our laboratory in Natick, MA. Testing on R.G. and T.L. began 48 and 78 hours after finishing the expedition, respectively. The temperature of the warm-water bath postexpedition was $\sim 1.0^{\circ}\text{C}$ higher than during preexpedition testing because of a calibration error, but this difference should not have affected the CVID measurements during CWI.

The temperature responses of the middle digit of each extremity were representative of the other 4 digits; therefore, for simplicity, only middle-digit temperature responses are presented (see the Figure and Table). Meehan²¹ compared finger temperature responses with ice-water immersion in 4 subjects on 5 or 6 separate days and found mean finger temperature to be within a range of $\pm 0.5^{\circ}\text{C}$; therefore, in the present study, a 1.0°C difference pre- to postexpedition likely reflects a significant difference. Using this criterion, no change was observed in finger temperature response for R.G., finger temperatures were lower postexpedition in both of T.L.'s hands, and toe temperatures were higher postexpedition in the feet of both subjects, with the exception of R.G.'s right foot, which showed no change. The higher toe temperatures were associated with higher nadir temperatures and earlier onset times for CVID (see the Table).

Mean core and skin temperatures during CWI of each extremity pre- and postexpedition are shown in the Figure. For R.G., warmer temperatures were observed postexpedition for both core ($\sim 0.3^{\circ}\text{C}$) and mean skin ($\sim 1.0^{\circ}\text{C}$). For T.L., core and mean skin temperatures were similar on both test dates. Digit pain (% of maximum) after 5 minutes of CWI from pre- to postexpedition in all extremities was recorded as the following: for R.G., right hand = 32% to 41%, right foot = 39% to 53%, left hand = 40% to 74%, and left foot = 47% to 63%; for T.L., right hand = 20% to 22%, right foot = 28% to 19%, left hand = 10% to 29%, and left foot = 27% to 30%. Unpublished data from our laboratory indicate that young, healthy subjects report pain 5 minutes after immersion of a single finger in cold water to be 53% of maximum with a standard deviation of 13% within subjects and 26% between subjects. On the basis

of these standard deviations, pain increased significantly only in R.G.'s left hand.

Discussion

This study was the first to evaluate CVID responses in the fingers and toes before and after a severe cold-weather challenge, with the objective of examining the interaction between acclimatization and cold injury in the extremities. Although there were only 2 subjects, and testing pre- and postexpedition did not represent a controlled study, the results offer new considerations for extended operations in cold environments. Our findings suggest the following: 1) prolonged heavy load carriage over rough terrain may inhibit acclimatization of CVID in the hands; and 2) moderate trench foot, after being resolved with proper care, does not prevent subsequent acclimatization.

Both laboratory⁷⁻⁹ and field¹⁰⁻¹² studies have demonstrated enhanced CVID after extremities are repeatedly exposed to cold without concurrent whole-body cooling. This type of acclimatization is typically accompanied by decreased pain.^{8,13} In the present study, neither subject demonstrated enhanced CVID in the fingers, and both reported that their hands felt cold and painful throughout the expedition, even toward the end as ambient temperature rose to -5°C . The lack of acclimatization was unexpected and the explanation is unclear.

One possibility is that the magnitude of cold, due to low temperatures and convective cooling from constant wind, induced a strong sympathetic activation that blunted the CVID response and inhibited acclimatization. However, the cold-weather clothing and high metabolic rates during this expedition would be expected to maintain body temperature in spite of these conditions, creating conditions under which CVID would typically occur. Indeed, previous studies have demonstrated acclimatization after a similar expedition under colder conditions (-52°C to -12°C).¹³ In contrast, in the present study both subjects' hands remained cold even when the subjects were inside their tent and out of the wind. Thus, it seems unlikely that the environmental conditions were too severe for acclimatization to occur. This scenario suggests that some other mechanism may be responsible for the blunted CVID in the hands.

A second possibility is that, although neither subject sustained frostbite, the subjects' symptoms (cold and painful fingers during the expedition and numbness continuing 3 weeks postexpedition) represented a nonfreezing cold injury. Nonfreezing cold injury is associated with local nerve or blood vessel damage²² and blunted CVID^{15,16}; therefore, it may also inhibit the development of acclimatization. The subjects in the present

Nailbed temperature (°C) response during 10-minute warm-water and 30-minute cold-water immersions, preexpedition (solid line) and postexpedition (dotted line), for each extremity of both subjects (R.G. and T.L.). Mean rectal (T_{re}) and skin (T_{sk}) temperatures during each test are also presented.

Mean temperature during cold-water immersion, and nadir temperature and onset time of initial cold-induced vasodilation, pre- and postexpedition.*

	Mean (°C)		Nadir (°C)		Onset (min)	
	Pre	Post	Pre	Post	Pre	Post
R. G.						
RH	6.9	6.0	5.4	5.8	4.0	4.6
RF	5.2	5.2	4.9	4.7	9.2	13.4
LH	6.5	6.3	5.7	5.6	4.6	3.9
LF	4.9	6.2	4.6	4.8	6.9	5.9
T. L.						
RH	9.5	7.7	6.6	9.3	3.6	4.0
RF	5.1	8.7	4.5	8.0	12.5	5.8
LH	8.1	7.0	6.4	6.3	4.4	3.5
LF	4.7	7.0	4.3	5.6	11.9	5.2

*R. G. and T. L. indicate subjects; RH, right hand; RF, right foot; LH, left hand; LF, left foot.

study may have been more susceptible to cold injury because of previous frostbite. On an expedition 2 years earlier, each had sustained frostbite injury to the thumb and index fingers of their right hands. The data from the present study show no difference in response between these previously injured digits and noninjured fingers. For example, the mean skin temperature of the index finger (R.G., preexpedition: 7.2°C, postexpedition: 5.5°C; T.L., preexpedition: 7.6°C, postexpedition: 7.8°C), which previously had been frostbitten, was similar to the mean temperature of the fourth finger (R.G., preexpedition: 7.7°C, postexpedition: 5.6°C; T.L., preexpedition: 8.1°C, postexpedition: 8.0°C), which had never been frostbitten. Although prior cold injury does not appear to be responsible for the lack of acclimatization, the development of a nonfreezing cold injury cannot be ruled out.

A notable difference between the present study and earlier expeditions^{12,13} is that these subjects hauled ~150-kg sleds and carried ~20-kg backpacks, whereas the earlier expeditions were resupplied and the subjects therefore carried lighter loads. For example, Etienne hauled a 50-kg sled and was resupplied 5 times between Canada and the North Pole.¹³ Thus, a third possibility for the lack of acclimatization in the hands is that carrying heavy loads and the associated constant pressure of the shoulder straps on the brachial plexus region may have impaired sensory function. Such "rucksack paralysis" has been demonstrated to reduce hand strength and tactile sensitivity.^{23,24} These symptoms have been noted to worsen with increasing pack weight, load-carriage du-

ration, and roughness of terrain,²³ and as little as 10 kg of pack weight is sufficient to cause tissue irritation.²⁵ A possible contributing factor is the extreme body-weight loss during this expedition, as emaciation has been suggested to contribute to "pack palsy."²⁶ Although speculative, it is conceivable that the prolonged backpack use may have caused circulatory or neural impairment in the hands, disrupting the development of CIVD. The subjects reported that their hands began to feel cold and painful only after the first 2 weeks of the expedition, during which time these symptoms may have developed. The numbness that developed in T.L.'s right arm during the 10 days of the expedition also supports a role for heavy backpacks to impair sensory function.

In contrast with the fingers, there was evidence of acclimatization in both of T.L.'s feet and in R.G.'s left foot. Both field^{12,27} and laboratory²⁸ studies have demonstrated local acclimatization in the feet, similar to that observed in the hands; however, in the present study such acclimatization was unexpected because both subjects sustained trench foot early in the expedition. Ungley and Blackwood²⁹ reported that the mildest cases of trench foot may resolve in 2 to 5 weeks, though most cases proceed through several stages of recovery lasting 3 to 12 months. Despite the subjects' continued exposure to cold damp feet during the daytime ski trek, the resolution of this injury underscores the importance of allowing the feet to dry to whatever extent possible, thus limiting the severity of the injury and allowing a more rapid recovery.

The acclimatization in the feet was associated with both a higher nadir temperature and an earlier onset of CIVD. A higher nadir temperature suggests a diminished initial sympathetic response, perhaps due to less available norepinephrine or reduced affinity of the α_2 -adrenoceptors for norepinephrine, whereas an earlier onset of CIVD may reflect greater sensitivity to the reduced blood flow and local tissue cooling, resulting in earlier relaxation of blood vessels. Unfortunately, no measurements of sympathetic activity were made during this study that might elucidate the mechanism for the changes in CIVD response due to acclimatization. The lack of adaptation in R.G.'s right foot, which experienced numbness toward the end of the expedition as a result of heavy load carriage, may be related to a similar neural impairment as we speculated for the hands.

Meyer and Webster³⁰ described 2 basic patterns of response to peripheral cold exposure: hunting, which refers to the classic cyclic oscillations in blood flow and skin temperature, and proportional control, which describes a sustained moderate blood flow with little cyclic variation after the initial vasoconstriction. In the Figure, an example of a classic hunting response can be seen in

T.L.'s right hand at postexpedition, whereas at preexpedition this digit shows more of the proportional control pattern. Whether one pattern is more advantageous than the other for maintaining dexterity or reducing the risk of cold injury is not known. An interesting anomaly is the "shoulder" in the temperature response postexpedition of T.L.'s right hand. The initial nadir temperature occurs during that shoulder (9.3°C at 4 minutes), though the next minimum temperature occurs at 6.8°C after 8.5 minutes. Such a response has been observed occasionally in our laboratory, but it is unclear why this occurs. There appears to be an interruption of the initial sympathetic response to cold, which resumes after several minutes to reach a more typical nadir temperature. The large variability that is inherent in this response can be seen by the varied patterns in the Figure, even within the same individual. It is this variability that creates a challenge for analyzing CIVD cycling patterns or evaluating their effectiveness for hand function.

A unique speculative concept from this study proposes that carrying heavy backpacks for extended periods may impair blood flow or nerve conduction in the fingers, thereby blunting the CIVD response and inhibiting peripheral acclimatization. We are unaware of any studies that have examined whether there are changes in blood flow to the hands while carrying heavy loads, but the results of this study suggest that future research in this area is warranted to determine whether this may be a factor in injuries related to prolonged load carriage. This study also demonstrates enhanced CIVD in the toes despite experiencing mild-moderate trench foot early in the expedition, suggesting the severity of cold injury was limited and rapid recovery ensued due to appropriate foot hygiene. These are important considerations for physically active individuals exposed to extreme cold for extended periods.

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