Human thermoregulatory responses to cold air are altered by repeated cold water immersion

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YOUNG, ANDREW, J., STEPHEN R. MUZA, MICHAEL N. SAWKA, RICHARD R. GONZALEZ, AND KENT B. PANDOLF. *Human thermoregulatory responses to cold air are altered by repeated cold water immersion.* J. Appl. Physiol. 60(5): 1542–1548, 1986.—The effects of repeated cold water immersion on thermoregulatory responses to cold air were studied in seven males. A cold air stress test (CAST) was performed before and after completion of an acclimation program consisting of daily 90-min cold (18°C) water immersion, repeated 5 times/wk for 5 consecutive wk. The CAST consisted of resting 30 min in a comfortable [24°C, 30% relative humidity (rh)] environment followed by 90 min in cold (5°C, 30% rh) air. Pre- and postacclimation, metabolism (M) increased ($P < 0.01$) by 85% during the first 10 min of CAST and thereafter rose slowly. After acclimation, M was lower ($P < 0.02$) at 10 min of CAST compared with before, but by 30 min M was the same. Therefore, shivering onset may have been delayed following acclimation. After acclimation, rectal temperature (T_{re}) was lower ($P < 0.01$) before and during CAST, and the drop in T_{re} during CAST was greater ($P < 0.01$) than before. Mean weighted skin temperature ($\bar{T}_{sk}$) was lower ($P < 0.01$) following acclimation than before, and acclimation resulted in a larger ($P < 0.02$) T_{re}-to-$\bar{T}_{sk}$ gradient. Plasma norepinephrine increased during both CAST ($P < 0.002$), but the increase was larger ($P < 0.004$) following acclimation. These findings suggest that repeated cold water immersion stimulates development of true cold acclimation in humans as opposed to habituation. The cold acclimation produced appears to be of the insulative type.

cold acclimatization; cold acclimation; hypothermia; thermoregulation; catecholamines; metabolism; core temperature: skin temperature

THE EXISTENCE OF COLD ACCLIMATIZATION[1] in humans has been reasonably well demonstrated. For example, Korean women divers who repeatedly dive in cold water have been reported to have a higher threshold for shivering (lower critical water temperature) than nondivers (16, 19). Also, Australian aborigines and South African bushmen who have lived many years exposed nearly nude to extreme low temperatures at night respond to cold exposure with little or no shivering and lower steady-state core and skin temperature compared with control subjects (13). Nonadapted humans respond to acute cold exposure with marked shivering and elevated metabolic heat production (13, 18). The degree to which persons lacking a lifetime experience of cold stress are able to acclimatize to cold is not known. Furthermore, while cold acclimation procedures have been reported to produce altered responses to cold (3, 11, 12, 14, 17, 20), the extent to which such changes represent habituation (blunted response) as opposed to true acclimation remains unclear.

Three rather different patterns of human cold adaptation have been observed. A metabolic type has been reported to occur by Scholander et al. (20) and Keatinge (11) in which metabolic heat production increased to a greater degree during cold exposure in the adapted than in the nonadapted state. The second type of cold adaptation reported to occur in humans is the hypothermic form. Hypothermic adaptation is characterized by a greater fall in core temperature (3, 7, 14) and a reduction in metabolic heat production (3, 5, 14) during cold stress following adaptation, compared with the nonadapted state. An insulative adaptation has also been reported to occur in humans. With this type of adaptation, body temperature is maintained during cold stress with little or no increase in metabolic heat production (16, 21). Also, skin temperature during cold stress is lowered (16, 19), thereby reducing the thermal gradient between the skin and the environment favoring less heat loss to the environment. Because these studies differed in ambient conditions, clothing, as well as duration and frequency of cold adaptation regimens employed, interstudy comparisons are difficult. However, it seems that subjects acclimated or acclimatized to cold air appear to demonstrate the metabolic (11, 20) or hypothermic (7, 14) type of adaptation but not the insulative. Evidence for insulative adaptations have been obtained in studies of subjects repeatedly exposed to cold water (16, 19, 21).

Most investigations of human cold adaptation have employed either intermittent (5, 7, 14) or continuous (3, 20) exposure to cold air to induce acclimation. Some of these studies have been carried out under field conditions where the subjects worked and performed various activities while living outdoors during cold weather (5, 7, 14, 20, 21). In undertaking the present investigation, the assumption was made that the stimulus required to produce a measurable degree of cold acclimation in humans was a repeated reduction of core temperature by a significant amount and not merely cold exposure per se.

[1] Throughout this report the term acclimatization will refer to the physiological adaptation resulting from exposure to changes in the natural environment while acclimation will be used to refer to physiological adaptations resulting from exposure to experimentally induced changes in environmental conditions (2).

Although the subjects in those aforementioned studies were indeed exposed to cold stress, differences in environmental conditions and clothing and the failure to adequately report core temperatures makes it difficult to determine to what extent, if any, core temperatures were reduced during acclimation procedures.

Because heat conduction is greater in cold water compared with cold air, cold water immersion results in greater and more rapid lowering of body temperature than cold air. Thus it was hypothesized that repeated cold-water immersion would present a more intense challenge to the body's thermoregulatory system, resulting in a greater degree of acclimation than possible in cold air. Although one previously reported investigation (17) did study the effects of intermittent cold water immersion on responses to cold air, a limited number of immersions was employed to acclimate the subjects; in addition, subjects were not studied before and after acclimation, but rather responses of a few acclimated subjects were compared with those of a larger group of nonacclimated subjects.

Therefore, the purpose of the present study was to investigate the effects of a program of repeated cold water immersion on human thermoregulatory responses to acute cold air exposure. It was thought that while cold-water immersion would provide the necessary challenge to stimulate a thermoregulatory adaptation, the stress of this environment might be so severe that any adaptations might be masked; however, under the relatively less stressful conditions of cold air exposure, adaptations might be apparent. A repeated-measure design was employed to compare responses obtained in the same individuals before and again after the acclimation program.

METHODS

Subjects and experimental design. Seven male volunteers participated in this study after being completely informed as to the requirements for and risks of participation. Descriptive characteristics (mean ± SE) of the subjects were age 24 ± 2 yr, body mass 79.4 ± 3.7 kg, body surface area (BSA) 1.98 ± 0.07 m^2, body fat (hydrostatic weighing, Ref. 8) 17.4 ± 1.8%, maximal aerobic power ($\dot{V}O_{2\,max}$) determined by a continuous treadmill protocol (15) 45.3 ± 1.6 $ml \cdot kg^{-1} \cdot min^{-1}$, and mean skinfold thickness (14 sites) 11.4 ± 1.5 mm.

The study was completed in Natick, MA, during the late fall when seasonal effects of cold exposure were expected to be minimal. Subjects completed a cold air stress test (CAST) 2 days before and again 2 days after completion of a cold acclimation program. The CAST consisted of a 30-min base-line period spent reclining on a nylon-mesh lounge chair in a comfortable environment [ambient temperature (T_a) = 24°C, relative humidity (rh) = 30%] while wrapped in blankets, following which the subject stood, entered the environmental chamber (T_a = 5°C, rh = 30%), and then reclined for 90 min, wearing only swim trunks. During the last 5 min of both periods, venous blood samples were obtained for determination of plasma norepinephrine (NE) and epinephrine (E). O_2 uptake ($\dot{V}O_2$, $l \cdot min^{-1}$, STPD), carbon dioxide production ($\dot{V}CO_2$, $l \cdot min^{-1}$, STPD), and minute ventilation ($\dot{V}E$, $l \cdot min^{-1}$, BTPS), were determined once during the base-line period and periodically during the cold air exposure. Rectal (T_{re}) and mean weighted skin ($\bar{T}_{sk}$) temperatures were measured during the last 3 min of the base-line period and at 2-min intervals throughout the cold exposure. Both CAST (pre- and postacclimation) were performed at the same time of day to avoid the possible confounding effects of circadian rhythms. Subjects had abstained from smoking, food, and drink (except water) for at least 1 h prior to the CAST.

The cold acclimation program consisted of a daily 90-min immersion in cold (18°C, stirred) water, repeated 5 days/wk for 5 consecutive wk. Subjects wore only swim trunks for immersions. In general, the acclimation sessions were accomplished on 5 consecutive days each week (Monday–Friday) followed by a 2-day rest. However, on several occasions an individual's minor illness required a break in the schedule, and missed sessions were then completed on the weekend. Once during the study, a midweek holiday resulted in cancellation of an acclimation session that could not be rescheduled. Thus the subjects completed a total of 24 acclimation sessions. The acclimation sessions were performed at the same time of day as the CAST was performed. For each acclimation session, the subject reclined quietly on a nylon-mesh lounge chair while immersed to the neck in the water. Prior to and during immersion, T_{re} was continuously monitored and recorded at 10-min intervals. Immersions were terminated after 90 min or if T_{re} fell below 35°C after which the subject was dried and rewarmed.

Experimental procedures. Chest electrodes (CM 5 placement) were used to obtain electrocardiograms, which were radiotelemetered to an oscilloscope-cardiotachometer unit (Hewlett-Packard) for measurement of heart rate. A thermistor inserted 10 cm beyond the anal sphincter was used to measure T_{re}. Skin temperature was measured at three sites using thermocouples taped to the skin (forearm, chest, and calf), and T_{sk} was calculated (4). Respiratory exchange parameters were measured with open-circuit spirometry using an automated system (Sensormedics Horizon MMC). Aerobic metabolism (M) was calculated from the $\dot{V}O_2$ assuming a respiratory quotient (RQ) of 0.82. Venous blood samples were obtained from an indwelling catheter placed in an antecubital vein, kept patent with heparinized saline. Aliquots of plasma were analyzed for NE and E concentration using a radioimmunoassay technique performed by a commercial laboratory (MEDPATH, Teterboro, NJ).

Statistical analyses. Multifactor repeated-measures analysis of variance (ANOVA) was used to determine whether the factors "acclimation" (pre vs. post), or exposure "time" (base line vs. post-CAST for blood parameters or the periodically repeated physiological measurements) had any significant effects. In the event that ANOVA revealed significant main effects or factor interactions, Tukey's critical difference was calculated and used to locate significant differences between means.

RESULTS

Cold acclimation program. The subjects completed a total of 24 cold water immersions. Five of the seven

subjects completed the full 90 min of immersion during every acclimation session. One subject completed the entire 90 min on all but two immersions when he had to be removed from the water at 80 and 85 min, respectively, due to a $T_{re} < 35°C$. One subject completed the full 90 min of the first immersion but thereafter had to be removed from the water at times ranging from 44 to 83 min due to $T_{re} < 35°C$. No particular pattern of increasing or decreasing tolerance time could be discerned in this latter subject. Individual values for initial (preimmersion) and final T_{re} and the change are shown in Table 1 for the 1st and 24th cold water immersion. ANOVA indicated that T_{re} fell ($P < 0.001$) during immersion, but there were no significant differences ($P < 0.22$) in T_{re} responses between the 1st and 24th immersion.

Cold air stress tests. Figure 1*A* shows the (mean ± SE) T_{re} before (0 min) and during (30, 60, and 90 min) the two CAST. During both CAST, a similar pattern of change in T_{re} was observed. During the first 30 min of cold exposures, T_{re} rose, after which it progressively fell (effect of time, $P < 0.001$). However, following the acclimation program, T_{re} was lower ($P < 0.003$) both initially and at all times during the CAST compared with before acclimation. ANOVA also indicated a significant ($P < 0.02$) interaction between the factors time and acclimation, suggesting that there were differences between the two CAST in the temporal pattern of changes in T_{re}. To more closely examine this effect and to adjust for the differences in initial T_{re}, the change in each individual's T_{re} relative to the initial value was calculated for each time interval and these data (mean ± SE) are shown in Fig. 1*B*. The increase in T_{re} during the first 30 min of both CAST was not significantly different and averaged (±SE) 0.25 ± 0.05°C. At 60 min of CAST before acclimation, T_{re} was still higher than the base-line value, whereas at 60 min of CAST following acclimation, T_{re} was not different from the base-line value. By 90 min of CAST before acclimation, the T_{re} had returned to base-line levels, but at 90 min of CAST following acclimation T_{re} was (mean ± SE) 0.28°C ± 0.14 below the initial T_{re}. Thus, following acclimation, T_{re} appeared to fall more rapidly and by a greater amount compared with before acclimation.

TABLE 1. *Changes in rectal temperature during cold-water immersion*

Subj No.	First Cold-Water Immersion			Last Cold-Water Immersion		
	T_{re}, °C			T_{re}, °C		
	Initial	Final	Change	Initial	Final	Change
1	36.9	35.7	−1.2	36.7	34.9*	−1.8
2	36.9	36.8	−0.1	36.6	36.9	+0.3
3	37.0	36.2	−0.8	37.1	36.2	−0.9
5	37.3	36.5	−0.8	37.3	36.6	−0.7
6	37.2	35.9	−1.3	37.2	36.1	−1.1
7	37.4	36.6	−0.8	37.2	35.3	−1.9
8	37.2	36.6	−0.6	37.3	36.2	−1.1
Mean	37.1	36.3	−0.8	37.1	36.0	−1.0
SD	0.2	0.4	0.4	0.3	0.7	0.7
SE	0.1	0.2	0.2	0.1	0.3	0.3

* Immersion terminated after 76 min; all other final values are after 90 min.

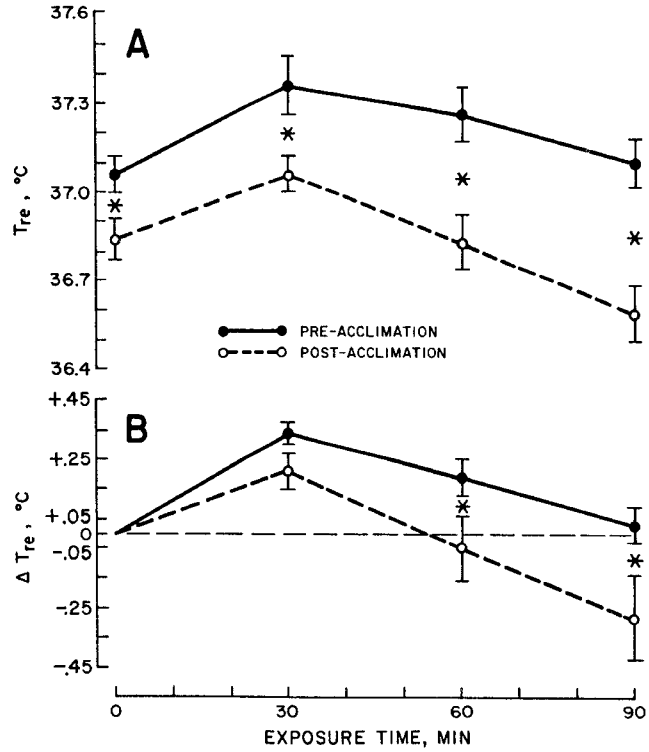

FIG. 1. Rectal temperature (T_{re}) before (0 min) and during 90-min cold air stress test (*A*) and change in T_{re} relative to initial values during cold air stress test (*B*). Values shown are means ± SE; $n = 7$. * Significant ($P < 0.01$) difference pre- vs. postacclimation.

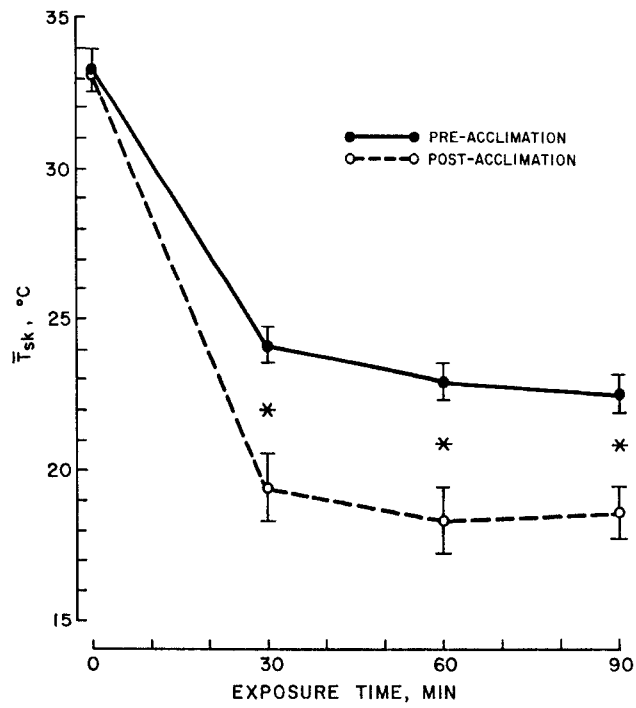

FIG. 2. Mean weighted skin temperature ($\bar{T}_{sk}$) before and during 90-min cold air stress test. Values shown are means ± SE; $n = 7$. * Significant ($P < 0.01$) difference pre- vs. postacclimation.

Figure 2 shows the $\bar{T}_{sk}$ before (0 min) and during (30, 60, and 90 min) the two CAST. Both before and after acclimation, $\bar{T}_{sk}$ fell ($P < 0.001$) during the first 30 min

of cold exposure but did not change during the remainder of the CAST. During the CAST following acclimation, $\bar{T}_{sk}$ averaged 4.7, 4.6, and 3.9°C lower ($P < 0.001$) at 30, 60, and 90 min, respectively, compared with before acclimation. Initial $\bar{T}_{sk}$ were unchanged by acclimation. The gradient from T_{re} to $\bar{T}_{sk}$ during the CAST is shown in Fig. 3. Both before and after acclimation, $T_{re} - \bar{T}_{sk}$ increased ($P < 0.001$) during the first 30 min and thereafter did not change significantly. During the CAST after acclimation, $T_{re} - \bar{T}_{sk}$ was larger than before acclimation ($P < 0.03$) after 30, 60, and 90 min, but $T_{re} - \bar{T}_{sk}$ did not differ initially (0 min).

Aerobic metabolism (M) before and during CAST is shown in Fig. 4*A*. Acclimation had no effect on M prior to cold exposure (0 min). During the first 10 min of cold exposure, there was an increase in M relative to the initial value; M tended to slowly rise during the remainder of the CAST. Following acclimation, M was significantly ($P < 0.02$) lower than before acclimation at 10 min of CAST, but at all subsequent times during CAST, there were no significant differences in M between pre- and postacclimation. When M was corrected for body surface area, the pattern of response was the same, but differences between pre- and postacclimation values at 10 min were smaller and did not achieve statistical significance.

Table 2 shows the changes in plasma NE and E concentration during each CAST. There was no significant difference between base-line (preexposure) NE levels for pre- and postacclimation. Plasma NE increased ($P < 0.002$) during both pre- and postacclimation CAST, but postacclimation, the increase in NE was greater ($P < 0.004$) than preacclimation. Although plasma E levels appeared to follow the same trends as NE, ANOVA indicated that none of the differences between the means achieved statistical significance.

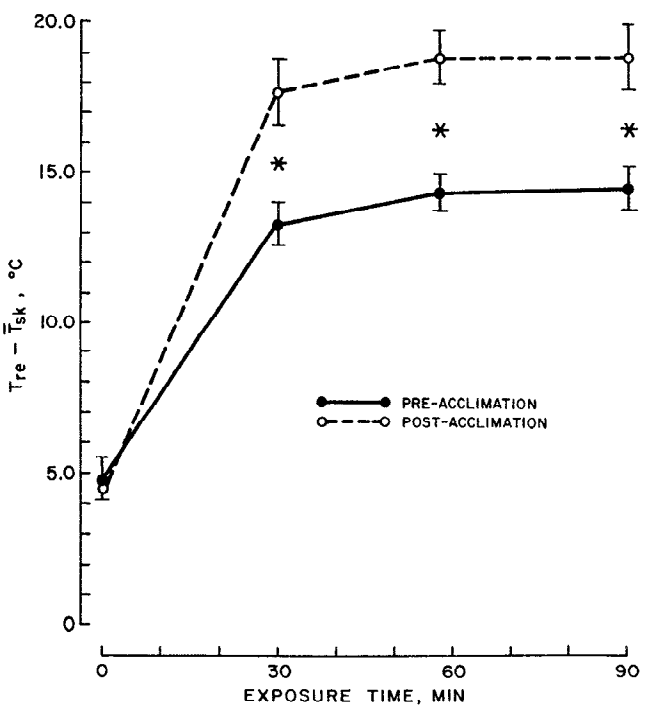

FIG. 3. Gradient between rectal and mean weighted skin temperature ($T_{re} - \bar{T}_{sk}$) before and during 90-min cold air stress test. Values are means ± SE; $n = 7$. * Significant ($P < 0.05$) difference pre- vs. postacclimation.

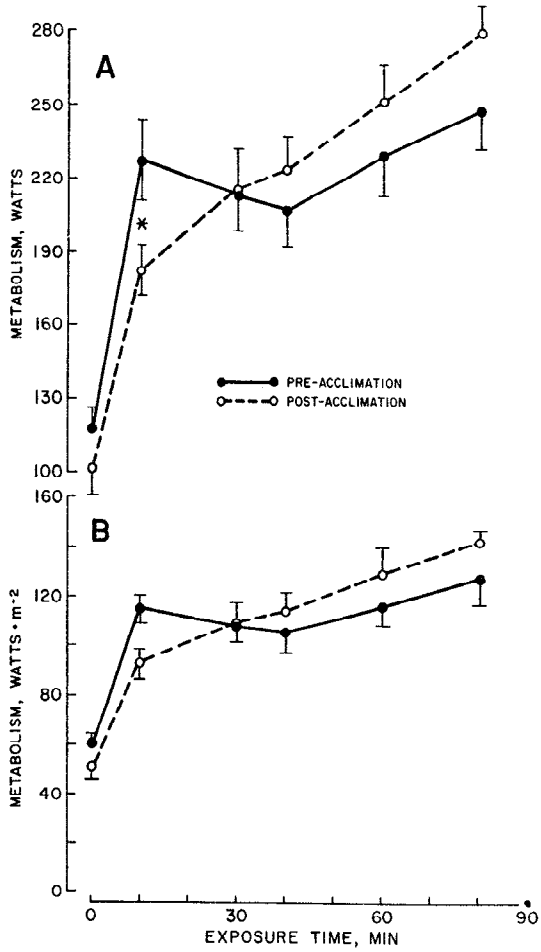

FIG. 4. Aerobic metabolism before and during 90-min cold air stress test shown as absolute value (*A*) and relative to body surface area (*B*). Values are means ± SE; $n = 7$. * Significant ($P < 0.05$) difference pre- vs. postacclimation.

DISCUSSION

Repeated cold water immersion was used to acclimate the subjects, since a more rapid and extensive body cooling can be achieved in water than in air, with little risk of tissue damage. One previous investigation did employ repeated cold (15°C) water immersion to acclimate subjects; however, the immersions were substantially shorter in duration and fewer in number than the acclimation regimen used in the present investigation. Radomski and Boutelier (17) acclimated three men using nine immersions (20–60 min) over a 2-wk period. Responses of these subjects to a cold air (10°C, 60 min) test performed 2 wk later were then compared with those of eight control (nonadapted) subjects. Depending on individual variability in body fat and shivering responses, 20–60 min of immersion may be insufficient to produce a marked fall in core temperature. In the present investigation, subjects sustained a fall in T_{re} of ~1°C on 24 occasions over a 5-wk period. Furthermore, the use of a

TABLE 2. *Effects of acclimation on changes in circulatory (plasma) catecholamines during cold-air stress test*

	Preacclimation			Postacclimation		
	Initial	Final	Change	Initial	Final	Change
Norepinephrine, $ng \cdot l^{-1}$	485±90	969±206*	484	293±33	1,257±166*	964†
Epinephrine, $ng \cdot l^{-1}$	62±12	73±17	11	34±5	96±30	62

Values are means ± SE; $n = 7$. * Significant ($P < 0.002$) difference initial vs. final. † Significant ($P < 0.004$) difference pre- vs. postacclimation.

repeated-measures design enabled the cold air responses of the same individuals to be observed before and soon after the acclimation period.

Repeated cold water immersion produced changes in the physiological responses to cold air that are somewhat characteristic of the classical description of hypothermic but not metabolic cold acclimation. Metabolism was lower during the initial 10 min of cold air exposure following acclimation compared with before acclimation, but after the 30th min there was no difference in pre- and postacclimation metabolic rate. Thus, in agreement with some other studies (3, 6), the onset of shivering was probably delayed by acclimation. Eventually, however, shivering and metabolism attained preacclimation levels. Following acclimation, the subjects had lower T_{re} before and throughout the entire period of cold air exposure compared with preacclimation. This might suggest the possibility that the normally regulated temperature had been reset following acclimation. However, even though initial T_{re} was lower for the post- than preacclimation CAST, there was no significant difference in initial T_{re} between the 1st and 24th immersion. This may have resulted from the fact that the air temperature in the immersion pool room was maintained warmer than the conditions used for the base-line measurements for the CAST. The reduction in initial T_{re} for the postacclimation CAST is not commonly observed with seasonal cold acclimatization, which has been reported to result in an increased basal metabolic rate (16). The initial metabolic rate of the subjects in the present investigation was lower for the CAST following acclimation than before, although not significantly so. This small reduction in metabolism may have accounted, in part, for the lower T_{re}. Thus the possibility that cold acclimation results in a downward resetting of the regulated body temperature remains to be confirmed. Even after correcting for the effect of the lower initial T_{re}, it still appears that there was a greater and more rapid fall in T_{re} during cold air exposure after acclimation. These alterations in core temperature responses are in agreement with those of others (7, 14, 17) and are recognized as the hypothermic form of acclimation.

A reduction in $\bar{T}_{sk}$ during cold air exposure was also observed following acclimation. Persons acclimatized to cold over a lifetime have lower skin temperatures during cold exposure than do nonacclimatized individuals. This has been suggested as evidence for the existence of an insulative type of cold acclimatization (16). The present study, however, is the first to clearly demonstrate that insulative adaptations in the physiological responses to cold air can be produced by acclimation procedures accomplished in a relatively short period of time. Although skinfold thickness and body density (hydrostatic weighing) were measured only at the beginning of the study, total body mass was measured before each CAST and weekly during the cold water immersion phase. There was no significant difference in total body mass preacclimation (79.4 ± 3.7 kg) compared with postacclimation (78.6 ± 3.7 kg). Therefore it is unlikely that changes in body composition would account for the reduction in $\bar{T}_{sk}$. The reduction in $\bar{T}_{sk}$ may reflect a greater degree of cutaneous vasoconstriction, at least at the three sites measured. Also, it was interesting to note that following acclimation the steady-state $\bar{T}_{sk}$ during cold exposure averaged 18.6°C, which was nearly equal to the water temperature to which the subjects were acclimated. Radomski and Boutelier (17) failed to observe any change in $\bar{T}_{sk}$ during cold exposure following repeated cold water immersion. This may have been due to a lesser degree of cold acclimation in their subjects due to the relatively less severe acclimation regimen employed. Alternatively, any insulative adaptations that may have occurred in their subjects could have been lost during the 2 wk which elapsed between the completion of acclimation and their cold air testing.

Plasma norepinephrine concentration increased over basal levels during both cold air exposures in agreement with previous investigations (1, 10) of the effects of acute cold exposure on plasma norepinephrine levels. Following acclimation, however, a much greater increase was observed. The effects of acclimation on changes in NE levels during cold exposure observed in the present study are not in agreement with the findings of Radomski and Boutelier (17). Those investigators observed that the subjects who had completed the acclimation procedures exhibited a fall in overnight urinary norepinephrine excretion during a 20-day Arctic sojourn (17). A possible explanation for the difference may be that the overnight urinary excretion of norepinephrine most closely reflects the basal norepinephrine level; in the present study, initial (basal) plasma norepinephrine concentrations were lower than before, although the differences were not statistically different. On the other hand, in agreement with the results of the present study, the control (nonacclimated) subjects in the study of Radomski and Boutelier (17) not only exhibited an increased overnight norepinephrine excretion during the Arctic sojourn, but the increase was greater during the second week of Arctic exposure compared with the first. The larger increase in plasma norepinephrine during cold air exposure following acclimation in the present study indicates a greater degree of sympathetic stimulation, which could cause a

more pronounced cutaneous vasoconstriction, thereby accounting in part for the reduction in $\bar{T}_{sk}$ observed following acclimation.

The body's insulative shell can be subdivided into two regions, the superficial shell (skin + subcutaneous fat) and the subcutaneous muscle shell, both of which contribute to the total shell insulation (16). Insulation provided by the fat is proportional to the thickness of the layer (16, 23) and can be assumed to remain constant. Therefore the total amount of insulation, as well as the relative contribution of skin and muscle, is determined by the amount of blood flow to each region and the ratio of flows between the regions. During cold exposure, total shell insulation increases due to sympathetically induced cutaneous vasoconstriction augmented by reduced skeletal muscle perfusion. In cooled skeletal muscle, perfusion could be reduced because of a temperature effect on local blood flow control (e.g., altered myogenic activity and increased blood viscosity; Ref. 22). Other possibilities are that in cooled muscle the sympathetic activation is not overridden by local control; or finally, there may be a local metabolic (Q_{10}) effect. The reduction in $\bar{T}_{sk}$ response to cold air observed in the present study has two important implications for insulation. As previously discussed, the more pronounced increments in plasma norepinephrine following acclimation strongly suggest that a more intense vasoconstrictor response would govern the lower $\bar{T}_{sk}$, and improved insulation results from a smaller thermal gradient between skin and ambient air, thereby favoring less heat loss to the environment. Second, the reduction in $\bar{T}_{sk}$ during cold exposure following acclimation was larger than the concomitant reduction in T_{re} responses. Therefore (see Fig. 3), the rectal-to-skin temperature gradient was increased following acclimation.

The increased T_{re}-$\bar{T}_{sk}$ gradient would favor improved heat transfer from the core to the subcutaneous muscle shell, whereas enhanced superficial shell insulation would prevent or limit less heat loss from the muscle shell. Additionally, it has been suggested (9) that cold-acclimatized persons have a more efficient countercurrent heat exchange system in the limbs enabling improved conservation of heat. Due to these conditions, muscle cooling might not be as pronounced following acclimation and perfusion could be improved. Thus the lower T_{re} during cold air exposure following acclimation may reflect a redistribution of body heat stores from the core areas to the muscle shell. Maintenance of a warmer and more highly perfused muscle shell at the expense of a cooler superficial shell would be advantageous for optimal metabolic and contractile function of the skeletal muscle.

It has been suggested that cold adaptation in humans represents a habituation process in which the repeated exposure to cold results in reduced response to the stimulus (17). This conjecture was based on the assumption that the autonomic (sympathetic) response to cold is less following the adaptation process as evidenced by a reduction in overnight urinary catecholamine excretion. However, changes in plasma norepinephrine levels during the cold stress provide a more accurate estimate of sympathetic nervous response than provided by urinary excretion data. The results of the present study clearly show that plasma norepinephrine concentration during acute cold air exposure increases to a much greater extent following the program of repeated cold water immersion. This greater sympathetic response substantiates the fact that the cold adaptation observed in the present study represents a true form of acclimation, as opposed to habituation, characterized by improved cutaneous insulation and probable redistribution of body heat from core to subcutaneous muscle shell. Future studies should measure the degree and distribution of peripheral blood flow and temperatures at a variety of depths below the skin in order to confirm that the subcutaneous muscle shell is indeed warmer and better perfused following acclimation.

The existence of an insulative type of cold acclimation does not necessarily refute the existence of the metabolic or hypothermic type of cold acclimation in humans. Skreslet and Aarefjord (21) have suggested that the different types of cold acclimation do not represent mutually exclusive physiological states but, rather, different successive stages in the development of complete cold adaptation. Although based on somewhat inconsistent data obtained from only three subjects, they hypothesized that cold acclimation develops progressively beginning with responses to cold characteristics of the metabolic type, proceeds through an intermediate phase during which cold responses are of the hypothermic type, and results finally in the development of the insulative type of acclimation. For cold acclimation to confer some physiological advantage, the eventual development of insulative adaptations is required. Maintenance of body temperature by shivering and elevated metabolic heat production is relatively inefficient from the standpoint of conservation of metabolic energy stores. The transition from the metabolic type of cold response to the hypothermic type undoubtedly involves some degree of habituation or conditioning. The development of the insulative adaptation with more complete cutaneous vasoconstriction (superficial shell) would enable greater transfer of heat to the subcutaneous muscle shell without increasing loss of heat between skin and environment. Muscle temperature and function during cold stress could therefore be improved with acclimation without resorting to energy wasteful elevations in metabolism.

In summary, this study has demonstrated that *1*) repeated cold water immersion produces acclimation to cold air in humans and *2*) the cold acclimation is primarily of the insulative type in that skin temperatures are lower, probably due to greater sympathetic nervous system activation mediating stronger cutaneous vasoconstriction. The degree of cold acclimation achieved is probably related to the intensity (frequency and duration of reduction in core temperature) of the acclimation procedures employed.

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