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Respiratory and cardiovascular responses to cold stress following repeated cold water immersion

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Muza SR, Young AJ, Sawka MN, Bogart JE, Pandolf KB. Respiratory and cardiovascular responses to cold stress following repeated cold water immersion. Undersea Biomed Res 1988; 15(3):165–178.—The effects of cold acclimation (CA) on the cardiorespiratory responses to cold air and water stress tests (CST) were studied in 7 males before and after a CA program of daily 90-min cold water (18° C) immersions repeated 5 times a wk for 5 consecutive wk. The CST consisted of a 90-min resting exposure to cold air (5° C, 30% relative humidity) or water (18° C) during which rectal temperature, oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), minute ventilation ($\dot{V}E$), heart rate, cardiac output ($\dot{Q}$), and blood pressure (BP) were periodically measured. In cold air following CA, the $\dot{V}O_2$ at 10 min was lower ($P < 0.02$) post- than pre-CA, however, no differences were found in cold water. The $\dot{V}E$ increased ($P < 0.01$) during CST as a function of $\dot{V}CO_2$. The CA did not affect the $\dot{V}E$-$\dot{V}CO_2$ relationship or the pattern of breathing during CST in cold air or water. The CA had no effect on $\dot{Q}$ or (a-$\bar{v}$) O_2 difference, which both increased ($P < 0.01$) during the first 45 min of CST, then remained stable. BP increased significantly during the first cold water exposure, but not during the last cold water immersion. These data indicate that CA attenuated the onset of metabolic heat production during CST in air but did not alter its ultimate magnitude or the relationships between the cardiorespiratory variables and metabolic requirements. Also, the thermoregulatory adjustments associated with CA altered the control of blood pressure during acute cold stress.

hypothermia
ventilatory response
body cooling
cold water
cold air

Respiratory responses to cold stress have received relatively limited study and generally cover only single, acute cold exposures. Generally, minute ventilation increases progressively in response to the increasing metabolic demands of the cold stress (1–4). Resting minute ventilation has been reported to nearly double during an exposure to 8°C air for 2 h (4). In the case of cold water immersion the intensity of this ventilatory response is inversely related to the water temperature (5) and may play an important role in determining whether the victim inhales water. Control of the pattern of breathing during mild hypothermia has not been studied. Also, the effect of mild hypothermia on the chemoreceptor inputs to the control of breathing

is not known. Nor has the respiratory response to cold stress before and after acclimation to cold been investigated.

Cardiovascular responses to cold stress vary depending on the nature of the cold stress (air vs. water, whole body vs. local exposure, ambient temperature, and duration of exposure) and the metabolic and neurophysiologic status of the organism being stressed. Generally, deep hypothermia (core temperature less than 30° C) causes a reduction of arterial blood pressure, heart rate (HR), cardiac output, total peripheral resistance, and oxygen consumption (6, 7). However, in normothermic or moderately hypothermic conscious humans and unanesthetized animals, cold stress elicits quite different cardiovascular responses (1, 2, 8–12). Virtually all of these studies have demonstrated an increased mean arterial blood pressure via increases in either systolic blood pressure (8), diastolic blood pressure (9), or both (1, 2, 10–12). However, HR response to cold stress seems to be more variable and not apparently associated with the nature of the cold stress (i.e., air vs. water) (1–4, 8–10, 12–17). Several studies have measured an increased cardiac output in response to cold stress (1, 3, 8, 12, 13, 17), generally attained via an increased stroke volume (3, 8, 12, 13, 17). The elevated stroke volume has been attributed to an enhancement of venous return via increased peripheral and cutaneous vasoconstriction as the body cools (12, 17). However, calculated total peripheral resistance has been shown to increase (9, 17), decrease (8, 12), or remain constant (13) in response to cold stress.

All of the aforementioned studies of cardiovascular responses to cold stress were one-time acute exposures. Only 2 studies, Budd and Warhaft (10) and Hong et al. (15), investigated the effect of acclimation on cardiovascular responses to cold stress. Both studies limited their observations to BP and/or HR responses. Budd and Warhaft (10) observed that after cold acclimation the systolic blood pressure response to cold stress was attenuated, whereas the bradycardia was enhanced. Likewise, Hong et al. (15) observed enhanced bradycardia during winter diving in the Korean Ama compared to summer diving. No investigators have studied whether the cardiovascular responses to cold are altered during the process of acclimating to cold. One would predict that if the increased cardiac output is serving the increased oxygen demand of the metabolic response to cold stress, as the metabolic response changes with acclimation (18–22) the cardiovascular responses should also change.

In most previous studies, the respiratory and cardiovascular responses to cold stress were measured at a single time point or derived from data aggregated over time. Consequently, the results do not accurately describe the changing nature of the physiologic responses during the exposure to cold stress. This investigation examined, under controlled laboratory conditions, the effects of cold acclimation on human cardiorespiratory control during acute cold water and air exposure.

METHODS

Subjects and experimental design

The subjects were 7 male Caucasians native to the continental United States. They were fully informed about the requirements and risks of participation. Descriptive characteristics of the subjects were (mean ± SE): age = 24 ± 2 yr; body mass = 70 ± 4 kg; body surface area = 1.98 ± 0.07 m^2; body fat [hydrostatic weighing (23)] =

17.4 ± 1.8%; mean skinfold thickness (14 sites) = 11.4 ± 1.5 mm; maximum aerobic power [treadmill running (24)] = 45.3 ± 1.6 ml · kg^{-1} · min^{-1}. The study was conducted in Natick, MA, during the late fall when seasonal effects of cold exposure were expected to be minimal. For the 9 mo. preceding the study the subjects had not participated in any significant cold weather activities. Subjects abstained from food, beverages, except water, and tobacco for at least 2 h before reporting for any tests. All experimental procedures were performed at the same time of day for a given subject throughout the study.

Subjects completed a standardized cold air stress test (CST) 2 d before and again 2 d after completion of a cold acclimation (CA) program. The CST consisted of a 30-min baseline period spent reclining on a nylon-mesh lounge chair in a comfortable environment [T_a = 24° C, relative humidity (rh) = 30%] while wrapped in blankets. After the baseline period the subject stood and 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 from an indwelling catheter previously placed in the antecubital vein for determination of plasma norepinephrine (NE) and epinephrine (E). Oxygen uptake ($\dot{V}O_2$, l · min^{-1}, STPD), carbon dioxide production ($\dot{V}CO_2$, l · min^{-1}, STPD), minute ventilation ($\dot{V}E$, l · min^{-1}, BTPS), cardiac output ($\dot{Q}$, l · min^{-1}), HR (bpm), and BP (mmHg) were determined once during the baseline period and periodically during the cold air exposure. Rectal (T_{re}) and skin (T_{sk}) temperatures were measured during the last 3 min of the baseline period and at 2-min intervals throughout the cold exposure.

The repeated cold water immersion program consisted of a daily, 90-min immersion in cold (18° C, stirred) water, repeated 5 times a wk for 5 consecutive wk. In general, the acclimation sessions were accomplished on 5 consecutive d each week (Monday to Friday) followed by a 2-d rest. However, on occasion an individual missed a scheduled immersion which was made up on the weekend. Due to a midweek holiday an immersion was missed which could not be rescheduled. Thus, the subjects completed a total of 24 water immersions. The water immersions were performed at the same time of day as the CST in air was performed. For each session, the subject reclined quietly on a nylon-mesh lounge chair while immersed to the neck in the water. Physiologic responses were measured during the first and last cold water immersion sessions according to the same protocol as the cold air exposure, with the exception that the baseline environmental conditions were slightly warmer (T_a = 27° C, rh = 65%). During all cold water immersions, T_{re} was continuously monitored. The cold water immersions were terminated after 90 min or if the 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 ECGs which were radiotelemetered to an oscilloscope-cardiotachometer unit (Hewlett-Packard) for measurement of HR. Ventilatory parameters, $\dot{V}O_2$ and $\dot{V}CO_2$ were measured using an automated open-circuit spirometry system (Sensormedics Horizon MMC). Cardiac output was measured using a semiautomated system (Sensormedics Horizon MMC) employing the CO_2 rebreathing method (25). The (a-$\bar{v}$) O_2 difference was calculated using the Fick principle. Blood pressure was measured by auscultation. 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); mean weighted skin temperature ($\bar{T}_{sk}$) and mean body temperature ($\bar{T}_b$) were calculated (26).

Statistical analyses

Multifactor, repeated measures analysis of variance (ANOVA) was used to determine if the factors "exposure" (baseline vs. cold exposure) or "acclimation" (pre vs. post) had significant effects. In the event that ANOVA revealed significant main effects or multifactor interactions, Tukey's critical difference was calculated and used to locate significant differences between means. Single factor regression analysis was performed to compare the changes in cardiorespiratory parameters with changes in metabolic rate and body temperature. Results are presented as the mean ± SE. The results obtained from the thermoregulatory data and vascular fluid responses have been presented and discussed in detail elsewhere (22, 27).

RESULTS

Cold air exposures

The oxygen uptakes and carbon dioxide productions are presented as functions of duration of cold air exposure in Fig. 1. Within the first 10 min of cold exposure before the CA program, the subjects' metabolic rate increased ($P < 0.01$) by approximately 93%. Postacclimation the oxygen uptake was significantly lower than the preacclimation oxygen uptake at 10 min. Carbon dioxide production was not affected by acclimation. As shown in Fig. 1, after CA respiratory exchange ratio was significantly ($P < 0.05$) elevated during the baseline and initial 40 min of cold air exposure compared to the preacclimation values.

In Fig. 2 the minute ventilation, tidal volume, and respiratory frequency responses to cold air exposure as a function of time are presented. These variables were not affected by cold acclimation. The effect of cold acclimation on the relationship between minute ventilation and tidal volume (V_T) during cold air exposure is presented in Fig. 3 Cold acclimation did not alter the pattern of breathing during cold air exposure. Although V_T increased proportionally more than respiratory frequency, in no subject did V_T increase to reach a maximum value (> 50% vital capacity).

Further analysis of the effect of cold stress on the control of breathing is presented in Table 1. Both pre- and postacclimation the minute ventilation was highly correlated ($P < 0.01$) to carbon dioxide production. Cold acclimation did not alter the relationship between minute ventilation and CO_2 production. Postacclimation, the minute ventilation was inversely related to mean body temperature ($P < 0.05$) and mean weighted skin temperature ($P < 0.01$). However, no relationship was observed before or after acclimation between minute ventilation and T_{re} during cold air exposure.

Cardiac output, stroke volume, and HR values as a function of time during cold air exposure are presented in Fig. 4 These variables were not affected by cold acclimation. The effects of cold exposure and acclimation on the control of cardiac output are listed in Table 2. Cardiac output was strongly correlated ($P < 0.01$) to oxygen uptake during cold air exposure pre- and postacclimation. This relationship

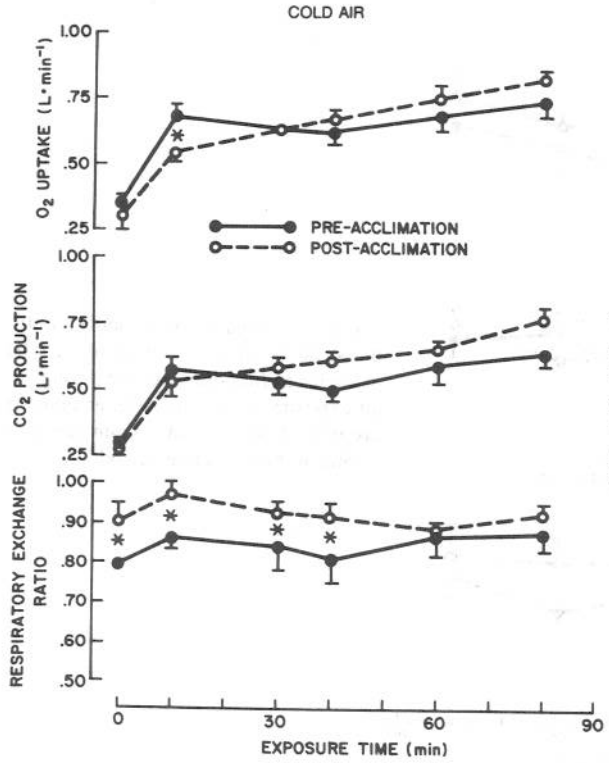

Fig. 1. Mean (±SE) oxygen uptakes, carbon dioxide productions, and respiratory exchange ratios as functions of time during cold air exposure before and after acclimation program. Values at time 0 min measured during 30-min baseline period; *asterisk* indicates significant ($P < 0.05$) difference pre- vs. postacclimation.

was unchanged by the CA program. Before or after CA no significant correlations were obtained between cardiac output and mean body temperature, T_{re}, or mean weighted skin temperature. The calculated (a-$\bar{v}$) O_2 difference increased ($P < 0.01$) from 5.2 ± 0.4 to 9.0 ± 0.7 ml/100 ml of blood during the first cold air exposure and from 4.8 ± 0.3 to 9.6 ± 1.1 ml/100 ml of blood during the postacclimation cold air test. Acclimation did not alter the magnitude of the (a-$\bar{v}$) O_2 difference response to cold air stress.

Due to an oversight, BP was not measured during the first cold air exposure. Following the acclimation program, mean arterial blood pressure did significantly ($P < 0.01$) increase, but only after 45 min of cold exposure (94.4 ± 3.0 and 106.3 ± 2.7 mmHg, at baseline and 80 min exposure, respectively). Total peripheral resistance (TPR) was 0.016 ± 0.002 and 0.012 ± 0.001 mmHg · ml^{-1} · min^{-1} before and after 80 min of cold air exposure. The TPR tended ($P = 0.06$) to be lower than baseline during cold air exposure.

Cold water exposures

Oxygen uptake, minute ventilation, and cardiac output values as a function of time during the first and last (24th) cold water exposures are presented in Fig. 5. These variables were not altered by cold acclimation. Tidal volume and respiratory frequency increased ($P < 0.01$) by 86 and 20%, respectively, preacclimation, and 61 and 36%, respectively, postacclimation. The relationship between minute ventilation and CO_2 production during cold water immersion (Table 1) demonstrated a high correla-

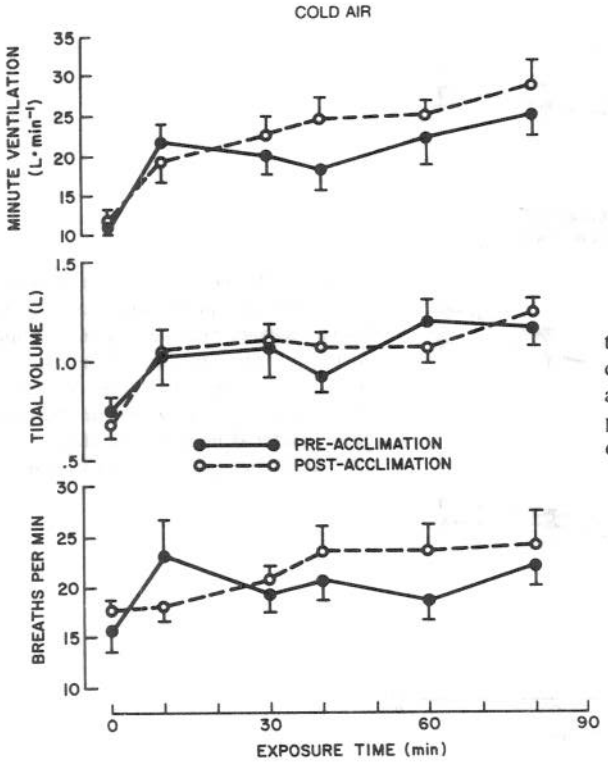

Fig. 2. Mean (±SE) minute ventilations, tidal volumes, and respiratory frequencies as functions of time during cold air exposure before and after acclimation program. Values at time 0 min measured during 30-min baseline period.

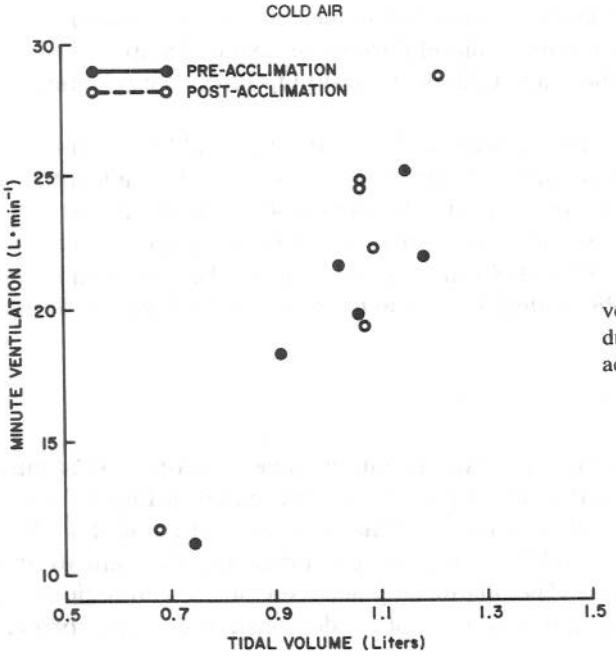

Fig. 3 Plot (group mean) of minute ventilation as a function of tidal volume during cold air exposure before and after acclimation program.

TABLE 1
EFFECTS OF COLD EXPOSURE ON CONTROL OF VENTILATION

Variable	Environment	Preacclimation			Postacclimation		
		m	b	r	m	b	r
$\dot{V}E$-$\dot{V}CO_2$	cold air	39.4	−0.8	0.95**	36.5	0.9	0.92**
	cold water	30.9	2.8	0.97**	35.3	0.7	0.94**
$\dot{V}E$-$\bar{T}_b$	cold air	2.1	−50.3	0.13	−4.6	178.0	−0.39*
	cold water	−14.2	489.3	−0.63**	−7.5	268.1	−0.42*
$\dot{V}E$-T_{re}	cold air	0.5	1.2	0.02	−8.1	321.2	−0.28
	cold water	−11.1	428.6	−0.58**	−7.8	308.4	−0.55**
$\dot{V}E$-$\bar{T}_{sk}$	cold air	−0.9	41.7	−0.50**	−0.9	43.0	−0.75**

*$P < 0.05$; **$P < 0.01$; m: slope, b: y-intercept, r: coefficient of correlation.

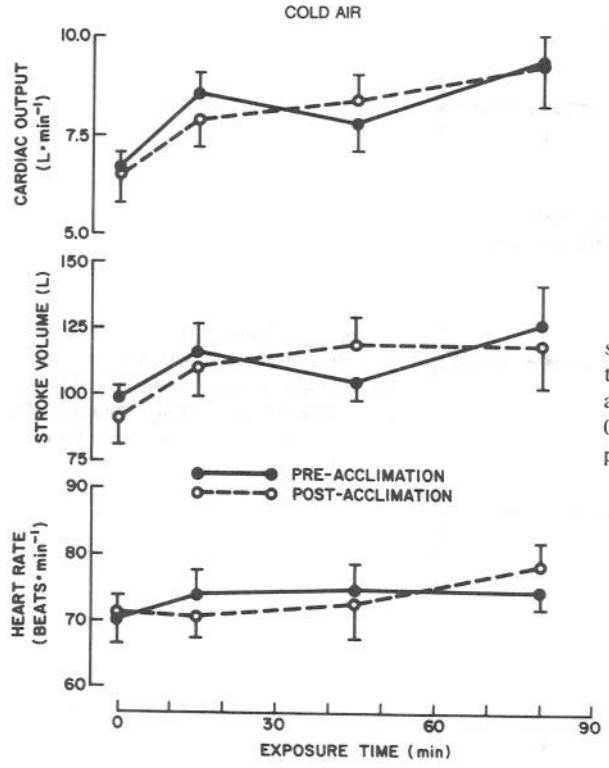

Fig. 4 Mean (±SE) cardiac outputs, stroke volumes, and HRs as functions of time during cold air exposure before and after acclimation program. Values at time 0 min measured during 30-min baseline period.

tion ($P < 0.01$) pre- and postacclimation. Furthermore, cold acclimation resulted in a significant ($P < 0.05$) increase in the slope of the relationship from 30.9 to 35.3 (liter, BTPS/liter, STPD). Inverse relationships between minute ventilation and mean body and rectal temperatures were observed pre- and postacclimation.

Cardiac output increased during the first 45 min of the cold water immersions (Fig. 5). No differences ($P > 0.05$) existed between the 1st and 24th water immersions. Before the last cold water immersion the baseline cardiac output tended to be slightly

TABLE 2
EFFECTS OF COLD EXPOSURE ON CONTROL OF CARDIAC OUTPUT

Variable	Environment	Preacclimation			Postacclimation		
		m	b	r	m	b	r
$\dot{Q}$-$\dot{V}O_2$	cold air	6.4	4.3	0.60**	6.0	4.5	0.60**
	cold water	6.7	4.5	0.78**	3.5	6.4	0.45*
$\dot{Q}$-$\overline{T}_b$	cold air	−0.7	33.6	−0.16	−0.9	39.2	−0.26
	cold water	−4.1	143.1	−0.73**	−2.0	73.5	−0.54**
$\dot{Q}$-T_{re}	cold air	0.2	2.5	0.02	−1.9	78.0	−0.24
	cold water	−3.8	148.9	−0.77**	−1.6	68.3	−0.59**
$\dot{Q}$-$\overline{T}_{sk}$	cold air	−0.2	11.8	−0.33	−0.1	10.8	−0.36

*$P < 0.05$; **$P < 0.01$; m: slope, b: y-intercept, r: coefficient of correlation.

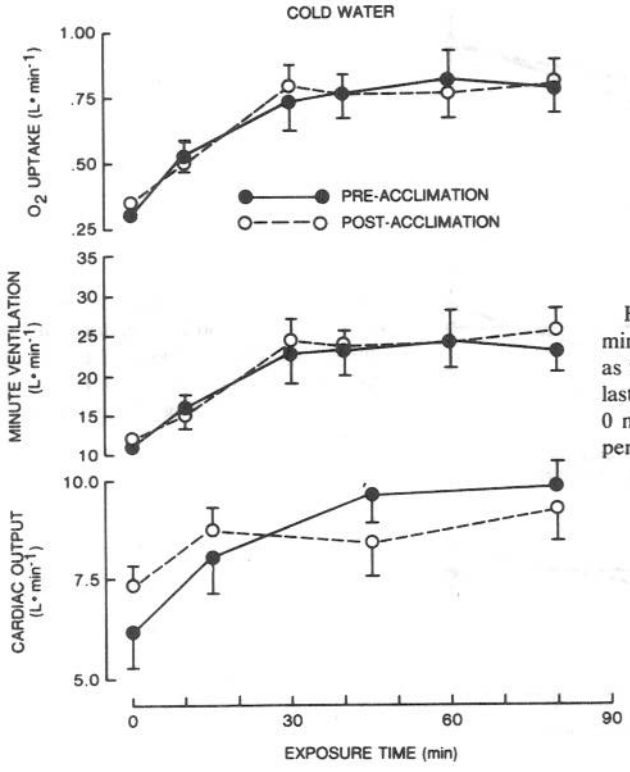

Fig. 5. Mean (± SE) oxygen uptakes, minute ventilations, and cardiac outputs as functions of time during the first and last cold water immersions. Values at time 0 min measured during 30-min baseline period.

higher ($P = 0.1$) than the respective preacclimation value. Consequently, although the cardiac output response during the cold water exposure was unchanged pre- vs. postacclimation, the percent change was diminished. Increased cardiac output was almost solely due to increased stroke volumes, since heart rates were not significantly increased. As previously described for the cold air exposures, a strong correlation was observed between cardiac output and oxygen uptake during the cold water

immersions (Table 2). However, unlike the cold air exposures, significant inverse correlations were obtained between cardiac output and mean body and T_{re} during the 1st and 24th water immersions. Cold acclimation did not significantly alter these relationships. The calculated (a-$\bar{v}$) O_2 difference were 5.0 ± 0.7 and 4.8 ± 0.3 ml/100 ml of blood for the pre- and postacclimation baseline periods. During cold water immersion the (a-$\bar{V}$) O_2 difference increased ($P < 0.01$) to 9.0 ± 0.6 and 9.4 ± 0.9 ml/100 ml of blood pre- and postacclimation, respectively.

The mean arterial blood pressure responses to cold water immersion as a function of time are presented in Fig. 6. During the 1st immersion, mean arterial blood pressure significantly increased from baseline due to increases of both systolic and diastolic BP. There was a significant acclimation effect ($P < 0.01$) with lower values postacclimation. Both pre- and postacclimation TPR did not systematically change during cold water immersion. Baseline TPR was 0.016 ± 0.003 and 0.012 ± 0.001 mmHg · $ml^{-1} \cdot min^{-1}$ pre- and postacclimation, respectively. During the cold water immersion, TPR at 80 min exposure was 0.011 ± 0.001 and 0.010 mmHg · $ml^{-1} \cdot min^{-1}$ before and after acclimation, respectively.

DISCUSSION

We have previously discussed (22) the thermoregulatory adaptations that occurred as a result of the cold acclimation program. Cold acclimation similar to the insulative type exhibited by the Korean breath-hold divers, before the use of wet suits became common practice (21), resulted from the repeated cold water immersion. During the postacclimation cold air exposure the subjects' skin temperatures were significantly lower, indicative of insulative cold acclimation. We attributed the lower $\overline{T}_{sk}$ during postacclimation cold exposure to greater cutaneous vasoconstriction mediated by an augmented sympathetic nervous stimulation. A larger increment in circulating NE during the postacclimation cold exposure was observed and taken as evidence for an

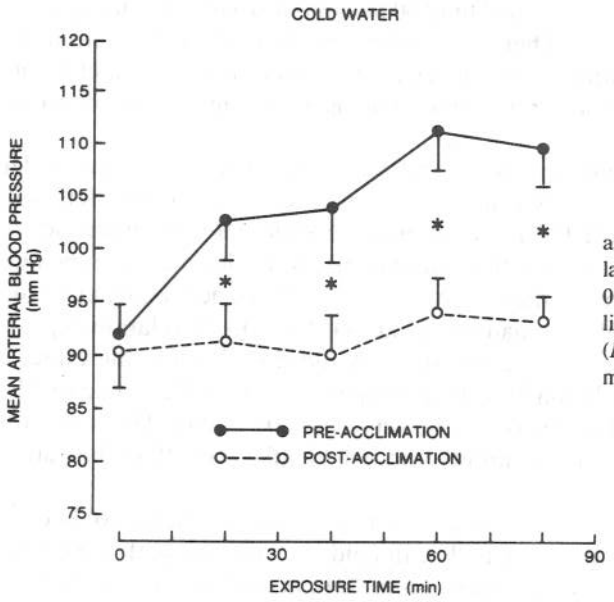

Fig. 6. Mean arterial BP (mean ± SE) as a function of time during the first and last cold water immersions. Values at time 0 min measured during the 30-min baseline period; *asterisk* indicates significant ($P < 0.05$) difference pre- vs. postacclimation.

augmented sympathetic nervous activity. We have also reported (27) that insulative cold acclimation, produced by our repeated cold water immersion program, had no measurable effect on body fluid regulation.

Results of the present investigation indicate that insulative cold acclimation produced by repeated, short duration cold water immersion had relatively minor effects on the respiratory and cardiovascular responses to acute cold stress. Generally, when plotted as a function of time, minute ventilation and cardiac output during cold air or water exposure were not significantly altered by the cold acclimation program. When plotted as functions of various body temperatures, minute ventilation and cardiac output exhibited varying degrees of correlation, which were not altered by cold acclimation. However, the BP response to cold water immersion was significantly altered by acclimation.

In agreement with previous studies (1–4), $\dot{V}E$ increased during the cold exposure proportionally to the metabolic demand. For exercise of low-to-moderate intensity (< anaerobic threshold), $\dot{V}E$ changes as a function of $\dot{V}CO_2$ rather than $\dot{V}O_2$ (28). This close relationship is evident in Table 1 during both the cold air and water exposures. Before acclimation, the $\dot{V}E$-$\dot{V}CO_2$ relationship was steeper ($P < 0.05$) during the cold air compared to the cold water exposures. After the acclimation program, the $\dot{V}E$-$\dot{V}CO_2$ relationship during cold air exposure was not altered. However, during the postacclimation cold water exposure the slope increased. Consequently, the $\dot{V}E$-$\dot{V}CO_2$ relationship was similar during the postacclimation cold air and water exposures. Whether the change in the $\dot{V}E$-$\dot{V}CO_2$ relationship during the cold water immersion represents a physiologic adaptation is uncertain. Keatinge and Evans (29) reported diminished ventilatory responses to cold stress after repeated cold water immersions. However, their cold exposures lasted only 20 min and their measurements of $\dot{V}E$ may have included the hyperventilation produced upon immersion into the cold water. Cooper et al. (5) reported that repeated immersion in cold water (12.0–12.7° C) for 5 d attenuated the initial ventilatory response. We did not measure $\dot{V}E$ until after 9 min of cold exposure, by which time the initial hyperventilation response was over. Furthermore, our measurements continued until 80 min of cold exposure during which time $\dot{V}E$ was proportional to $\dot{V}CO_2$. Therefore, following the initial hyperventilatory response, the control of ventilation does not seem to be altered by cold acclimation given that the $\dot{V}E$-$\dot{V}CO_2$ relationship remained unchanged during cold air exposure pre- vs. postacclimation.

The pattern of breathing during the cold air and water exposures were similar. The $\dot{V}E$ increased as a function of both VT and respiratory frequency. Consistent with previous studies (5, 30), VT tended to increase proportionally more than frequency. Cold acclimation did not appear to alter the pattern of breathing during cold exposure. As seen in Fig. 3, $\dot{V}E$ was attained with similar increases of VT and frequency before and after acclimation. Hey et al. (28) had reported that the $\dot{V}E$-VT relationship was altered when body temperature was increased. They did not examine the effect of body cooling on the $\dot{V}E$-VT relationship. Our temperature data (22) indicate that during the postacclimation cold air exposure the subjects were cooler. However, this small decrease in body temperature apparently did not significantly affect the pattern of breathing.

During the cold air exposures before acclimation, $\dot{V}E$ was not related to $\overline{T}_b$ or T_{re} but was moderately correlated inversely to $\overline{T}_{sk}$. In cold air, subjects with the coolest skin had the highest ventilatory response. Postacclimation, in cold air $\dot{V}E$ was inversely

related to $\overline{T}_b$ and $\overline{T}_{sk}$ but not to T_{re}. During cold water immersion pre- and postacclimation, $\dot{V}E$ demonstrated weak-to-moderate degrees of inverse correlations to $\overline{T}_b$ and T_{re}. The evolution of significant correlations between $\dot{V}E$ and $\overline{T}_b$ or $\overline{T}_{sk}$ during cold air stress probably results from a greater and more rapid decrease of T_b and $\overline{T}_{sk}$ following acclimation. A previous study (4) of these relationships during acute cold stress demonstrated a weak but significant inverse relationship between $\dot{V}E$ and T_{re}. No relationship was reported between $\dot{V}E$ and $\overline{T}_{sk}$. The earlier study evaluated these relationships at discrete times during the acute cold air (7.7° C) exposure. In the present study, the relationships between $\dot{V}E$ and various body temperatures were calculated using data collected from 10 to 80 min of exposure. This different method of assimilating the data may account for the different results obtained. Except for the initial ventilatory response to cold exposure (5), it is likely that temperature receptors in the skin and hypothalamus do not uniquely influence the steady-state ventilatory response to cold stress. Rather, these inputs stimulate metabolic and circulatory adjustments in attempts to maintain thermoregulatory homeostasis. The ventilatory response is then governed by the metabolic requirements. With the exception of the initial 10 min of cold air exposure, acclimation did not alter metabolic responses to cold air or cold water, thus ventilatory responses were not altered.

Upon exposure to the cold, oxygen delivery in response to the increased metabolic demands was provided by significant increases in $(a\text{-}\overline{v})$ O_2 difference and cardiac output. Similar to the ventilatory responses, cardiac output increased proportionally to the metabolic demand. Numerous studies (1, 3, 8, 12, 13, 17) have reported an increase of cardiac output during acute cold exposure. In cold air and water the increased cardiac output was mainly achieved by larger stroke volumes, since heart rate was slightly elevated only in the cold air. This is consistent with findings of several investigators (3, 8, 12, 13, 17). The cold acclimation program did not alter the relationship between cardiac output and metabolic rate nor the stroke volume and HR responses. Both before and after acclimation in cold air or water the cardiac output, stroke volume, and HR responses were not significantly different.

When plotted as a function of various body temperatures, cardiac output demonstrated significant negative correlations only during cold water immersion (Table 2). These correlations suggest that the cooler a subject's body, the greater the cardiac output. Again, instead of suggesting that body temperature per se uniquely governs the cardiac output response to cold stress, the present study along with several prior studies (1, 3, 8, 12, 13, 17) supports the hypothesis that the cardiac output response is primarily directed by increased metabolism resulting from thermoregulatory adjustments attempting to maintain body temperature. Since metabolism was essentially unchanged by acclimation, there was no change in cardiac output.

Consistent with previous studies (1, 2, 8, 11, 12), before acclimation mean arterial BP increased via elevations in both the systolic and diastolic components during the cold water immersions. After acclimation, the mean arterial BP was not systematically altered during the cold water exposure. Budd and Warhaft (10) had observed that after cold acclimation induced by exposure to cold while working in Antarctica, the systolic BP response to cold stress was attenuated, but the diastolic pressure response was not altered. In the present study, during the last cold water immersion mean arterial BP did not increase, although cardiac output was increasing and total peripheral resistance was not significantly decreasing over time. We have proposed (22) that postacclimation the subjects maintained a warmer and more highly perfused

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