AD-A172 509 COLD ACCLIMATION CAN BE INDUCED IN HUMANS BY REPEATED COLD WATER IMMERSION(U) ARMY RESEARCH INST OF ENVIRONMENTAL MEDICINE NATICK MA A J YOUNG ET AL. 1/1

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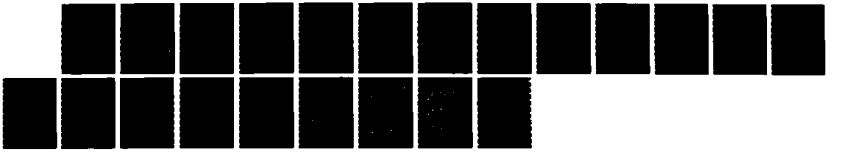

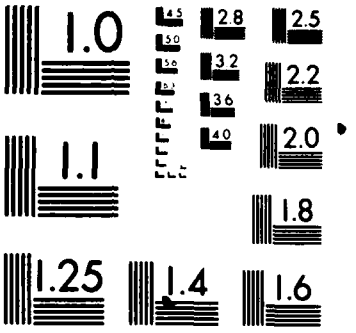

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20. ABSTRACT (Continue on reverse side if necessary and identify by block number)

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 post-acclimation, metabolism (M) increased ($P<0.01$) by 85% during the first 10 min

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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 (T_{sk}) was lower ($P < 0.01$) following acclimation than before, and acclimation resulted in a larger ($P < 0.02$) T_{re} to 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 ACCLIMATION CAN BE INDUCED IN HUMANS BY REPEATED COLD WATER IMMERSION

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INTRODUCTION

Studies of individuals experiencing a lifetime of repeated exposure to cold stress have clearly demonstrated that the process of cold acclimatization does occur in humans. For example, Australian aborigines, South African bushmen and the professional breath-hold divers of the South Korean and Japanese coasts all show different thermoregulatory responses to cold stress than do subjects from populations native to warm or temperate climates (1,2,3). However, concensus is lacking as to whether or not cold acclimation can be induced in humans who lack a lifetime experience of cold stress. Chronic exposure to cold stress (both continuous and repeated intermittent) has been reported to produce adaptations in the responses to cold (4,5,6,7,8). Several of those investigations, however, were "field" studies in which the effects of altered environmental conditions, clothing, or physical activity were not adequately measured, controlled for, or reported (6,7). In addition, it has been suggested that adaptations in physiological responses to cold are really indicative of the development of habituation (decreased responsiveness) and not acclimation (enhanced responsiveness) to cold (8).

As a basis for the present investigation, it was hypothesized that the requisite physiological stimulus to induce development of a measurable degree of cold acclimation in humans is a large and repeated lowering of body temperature as opposed to cold exposure, per se. Previous studies investigating the effects of cold air exposure may have failed to provide sufficient stimulus to induce cold acclimation in their subjects. A greater and more rapid lowering of body temperature can be achieved during cold water immersion than with cold air exposure. Thus cold water immersion would provide a greater thermoregulatory challenge than exposure to cold air. The purpose of this report is to review the results of our recent investigation (9) which was undertaken to determine the

effects of repeated cold water immersion on human thermoregulatory, cardiorespiratory, and body fluid responses to acute cold stress.

METHODS

Seven male Caucasians, native to the continental United States, volunteered as test subjects. None had experienced any significant cold exposure during the six months prior to the beginning of the study. Descriptive characteristics (mean $\pm$ SE) of the subjects were age, 24 $\pm$ 2 yrs; total body mass, 70 $\pm$ 4 kg; body surface area, 1.98 m^2; body fat, 17 $\pm$ 2%; skinfold thickness (14 sites), 11 $\pm$ 2 mm; maximal oxygen uptake, 45 $\pm$ 2 $ml \cdot kg^{-1} \cdot min^{-1}$.

The subjects performed a standardized cold air exposure two days before and two days after completion of the cold acclimation program. The standardized cold air exposure consisted of a 30-min baseline period and a 90-min cold period. During the baseline period, the subject was wrapped in blankets and reclined on a nylon-mesh lounge chair in a comfortable environment (T_a = 24°C, rh=30%). Following the baseline period, the subject stood, entered the environmental chamber (T_a = 5°C, rh=30%), and then reclined for 90-min wearing only swim trunks.

The cold acclimation program consisted of a daily immersion in cold (18°C, stirred) water, repeated five days (generally consecutive) each week for five consecutive weeks. Each immersion was 90-min in duration unless the subject's rectal temperature fell below 35°C in which case that session was terminated early for safety reasons. A total of 24 acclimation sessions were scheduled. During immersion, the subject reclined quietly with the water level adjusted to the bottom of the neck. Only swim trunks were worn during immersion. The first and the last cold water immersion were carried out using the same protocol and data collection procedures as in standardized cold air exposures. During the remaining cold water immersions, only limited data collection was performed

and no baseline period was employed.

All cold exposures were completed at the same time of day to avoid the influence of circadian rhythms. Cardiorespiratory measurements were made once during the baseline period and at regular intervals during the cold period of both cold air exposures and the first and last cold water immersion. Rectal (T_{re}) and mean weighted skin (T_{sk}) temperatures were measured at two min intervals during both baseline and cold periods. Venous blood samples were obtained during the last five min of baseline and cold periods for determination of hematocrit and hemoglobin concentration as well as plasma osmolality and concentration of proteins, epinephrine and norepinephrine. Urine production rate was measured during each period and urine aliquots from each period were saved for later analysis of specific gravity, and Na^+ and K^+ concentration.

The data were analyzed using multifactor analysis of variance (ANOVA). When ANOVA indicated significant main or interaction effects for the factors "acclimation" (pre versus post), or "cold exposure" (baseline versus final for blood parameters or the periodically repeated physiological measurements), Tukey's critical difference was calculated and used to determine if differences between means were significant.

RESULTS AND DISCUSSION

As previously stated, it was hypothesized that in order to induce development of a measurable degree of cold acclimation, an individual's core temperature must be repeatedly reduced by a substantial amount for a extended period of time. This was accomplished with the acclimation program used; the subjects sustained a reduction in T_{re} of about 1°C for 90 min on 24 days during a 35 day period. None of the physiological responses to cold water immersion (e.g. thermoregulatory, cardiorespiratory, body fluid responses) were changed by the

acclimation program to indicate development of any particular pattern of cold adaptation. Thus, while cold water immersion provided what was thought to be a sufficient stimulus to induce cold acclimation, adaptations in cold responses were apparently inadequate to offset the severity of the stress imposed by this aqueous environment. The possibility that evidence for cold acclimation would be masked during water immersion by the magnitude of the cold stress had been anticipated and was the reason for inclusion of the cold air exposures in the experimental design.

Figure 1 About here

In contrast to the responses to the cold water, the physiological responses to cold air did show significant adaptations following the acclimation program. Aerobic metabolic rate before and during the two cold air exposures is shown in Figure 1. During the postacclimation cold air exposure, aerobic metabolism was lower ($P < 0.05$) during the first 10 min, but by the 30th min and thereafter there was no difference in pre- and postacclimation metabolic rate. These observations may indicate that the onset of shivering was delayed by acclimation as has been suggested by others (5).

Figure 2 About Here

Figure 2 shows the T_{re} measured before and after 30, 60 and 90 min of the pre-and postacclimation cold air exposure. Before and at all times during the cold air exposure following acclimation, T_{re} was reduced ($P < 0.003$) compared to before acclimation (Fig 2A). Even after correcting for the effect of a lower initial T_{re} in the postacclimation test by calculating individual changes relative

to initial T_{re} (Fig 2B), there was a greater and more rapid fall ($P < 0.01$) in T_{re} during the cold air exposure compared to preacclimation. A greater and more rapid fall in T_{re} during cold air exposure following acclimation has also been observed in other studies of human cold acclimation. This adaptation has been referred to as hypothermic cold acclimation (6) or habituation (8). A reduction in baseline temperature is not usually observed with seasonal cold acclimatization; in fact, other have observed an increase in basal metabolic rate during the winter months (2). However, consistent with a lower baseline temperature, the subjects in the present investigation had slightly lower baseline aerobic metabolic rates (See Figure 1) following acclimation than before, but the difference was not statistically significant. These observations might suggest that cold acclimation results in a resetting of the normally regulated body temperature, but this speculation must be confirmed.

Figure 3 About Here

Figure 3 shows the effect of cold air exposure on $\bar{T}_{sk}$. During the first 30-min of both pre- and postacclimation cold air exposures, $\bar{T}_{sk}$ fell, but thereafter remained plateaued. Figure 3 shows that $\bar{T}_{sk}$ plateaued at about 4°C lower ($P < 0.01$) during the postacclimation cold air exposure compared to preacclimation. This observation is consistent with the observations that persons acclimatized to cold over a lifetime maintain lower skin temperatures during cold exposure than nonacclimatized subjects (2). On the other hand, others have reported that repeated cold water immersion programs had no effect on $\bar{T}_{sk}$ during subsequent cold air exposure (8). However, the failure to observe lower T_{sk} may have been due to a lesser degree of cold acclimation in their subjects

(8). The reduction in $\bar{T}_{sk}$ may reflect reduced skin blood flow due to greater cutaneous vasoconstriction. The increase in plasma norepinephrine (NE) concentration during cold air exposure (Table 1) following acclimation was 2x greater ($P < 0.004$) than the increment in NE before acclimation. Acclimation had no significant effect on plasma epinephrine (E) concentration. Therefore, the increment in plasma NE response is due to enhanced sympathetic nervous activity, not adrenal medullary activity. Enhanced sympathetic responsiveness to cold air could cause more pronounced cutaneous vasoconstriction thereby resulting in the lower $\bar{T}_{sk}$ during the postacclimation cold air exposure. Furthermore, an increased sympathetic responsiveness to cold air stress is clear evidence that repeated cold water immersion induced a true cold acclimation, not habituation.

Table 1 and Figure 4 About Here

The two components which comprise the total insulative shell of the body are the superficial shell (skin and subcutaneous fat) and the subcutaneous muscle shell. The insulation provided by the subcutaneous fat is proportional to its thickness and therefore relatively constant at least for short-time periods such as the duration of the present study. However, the insulation provided by the skin and muscle is in large part determined by the blood flows to these regions and thus can be regulated. Total insulation of the body's shell increases during cold exposure due to a sympathetically mediated cutaneous vasoconstriction and a reduction in skeletal muscle perfusion. The reduction in $\bar{T}_{sk}$ with acclimation enhances insulation in two ways. First, the insulation provided by the skin is increased due to a smaller temperature gradient for heat transfer between the skin and environment. Secondly, the enlarged T_{re}-$\bar{T}_{sk}$ difference (Figure 4)

following acclimation ($P < 0.02$) facilitates the transfer of heat from the body core to the muscle shell while improved skin vasoconstriction would limit the loss of heat from the muscle shell. Thus, acclimation may allow maintenance of a warmer, better perfused muscle shell. The lower T_{re} during cold exposure following acclimation may reflect redistribution of body heat stores from the core to the muscle shell. The development of this type of insulative acclimation would allow better maintenance of muscle function.

Figure 5 and 6 About Here

Cardiorespiratory responses to cold air exposure were not influenced by the acclimation program to any significant degree. For example, cardiac output (Figure 5) and pulmonary ventilation (Figure 6) increase during cold air exposure. However, the increment in cardiac output and ventilation during cold air exposure were not different between pre- and postacclimation. Likewise, body fluid regulation does not appear to be altered by the acclimation program. Plasma volume decreased ($P < 0.05$) and urine flow rate increased ($P < 0.05$) by the same amount (12% and 52%, respectively) during cold air exposure before and after the acclimation program.

In summary, the program of repeated immersion in cold water induced a true acclimation to cold air in nonadapted men. The type of acclimation produced appears to be insulative resulting in lower skin temperature during cold exposure. The physiological mechanism for enhanced insulation is probably an increased sympathetic responsiveness to cold stress which in turn mediates more pronounced and cutaneous vasoconstriction. Frequency, magnitude, and duration of core temperature reduction as opposed to exposure conditions appear to be key determinates of whether cold acclimation or habituation are induced.

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The opinions and/or findings presented in this paper are those of the authors and should not be construed as official Department of the Army position, policy or decision. Human subjects participated in these experiments after giving their free and informal consent. Investigators adhered to AR 70-25 and USAMRDC Regulation 70-25 on the Use of Volunteers in Research.

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Table 1. Changes in plasma catecholamine concentration during 90-min exposure to cold air.

	Norepinephrine, $ng \cdot \ell^{-1}$		Epinephrine, $ng \cdot \ell^{-1}$	
	Preacclimation	Postacclimation	Preacclimation	Postacclimation
Initial	485 ± 90	293 ± 33	62 ± 12	34 ± 5
Final	969 ± 206*	1257 ± 166*	73 ± 17	96 ± 30
Change	484	964†	11	62

Values are means ±SE. * Significant ($P < 0.002$) difference between initial and final.
† Significant ($P < 0.004$) difference between pre- and post-acclimation.

FIGURE LEGENDS

1. Figure 1. Aerobic metabolism before (0-min) and during 90-min exposure to cold (5°C) air shown as absolute value (A) and relative to body surface area (B). Values are means ±SE; n=7. *Significant ($P < 0.05$) difference between pre- and postacclimation.

2. Figure 2. Rectal temperature (T_{re}) before (0-min) and during 90-min exposure to cold (5°C) air (A), and the change in T_{re} during cold exposure relative to the initial value (B). Values shown are means ±SE; n=7. *Significant difference betwen pre- and post acclimation.

3. Figure 3. Mean weighted skin temperature ($\bar{T}_{sk}$) before (0-min) and during 90-min exposure to cold (5°C) air. Values shown are means ±SE; n=7. *Significant difference between pre- and postacclimation.

4. Figure 4. Gradient between rectal and mean weighted skin temperature ($T_{re}-\bar{T}_{sk}$) before (0-min) and during 90-min exposure to cold (5°C) air. Values shown are means ±SE; n=7. *Significant difference between pre- and postacclimation.

5. Figure 5. Heart rate, stroke volume, and cardiac output before (0-min) and during 90-min exposure to cold (5°C) air. Values shown are mean ±SE; n=7.

6. Figure 6. Tidal volume, respiratory frequency, and minute ventilation before (0-min) and during 90-min exposure to cold (5°C) air. Values shown are means ±SE; n=7.

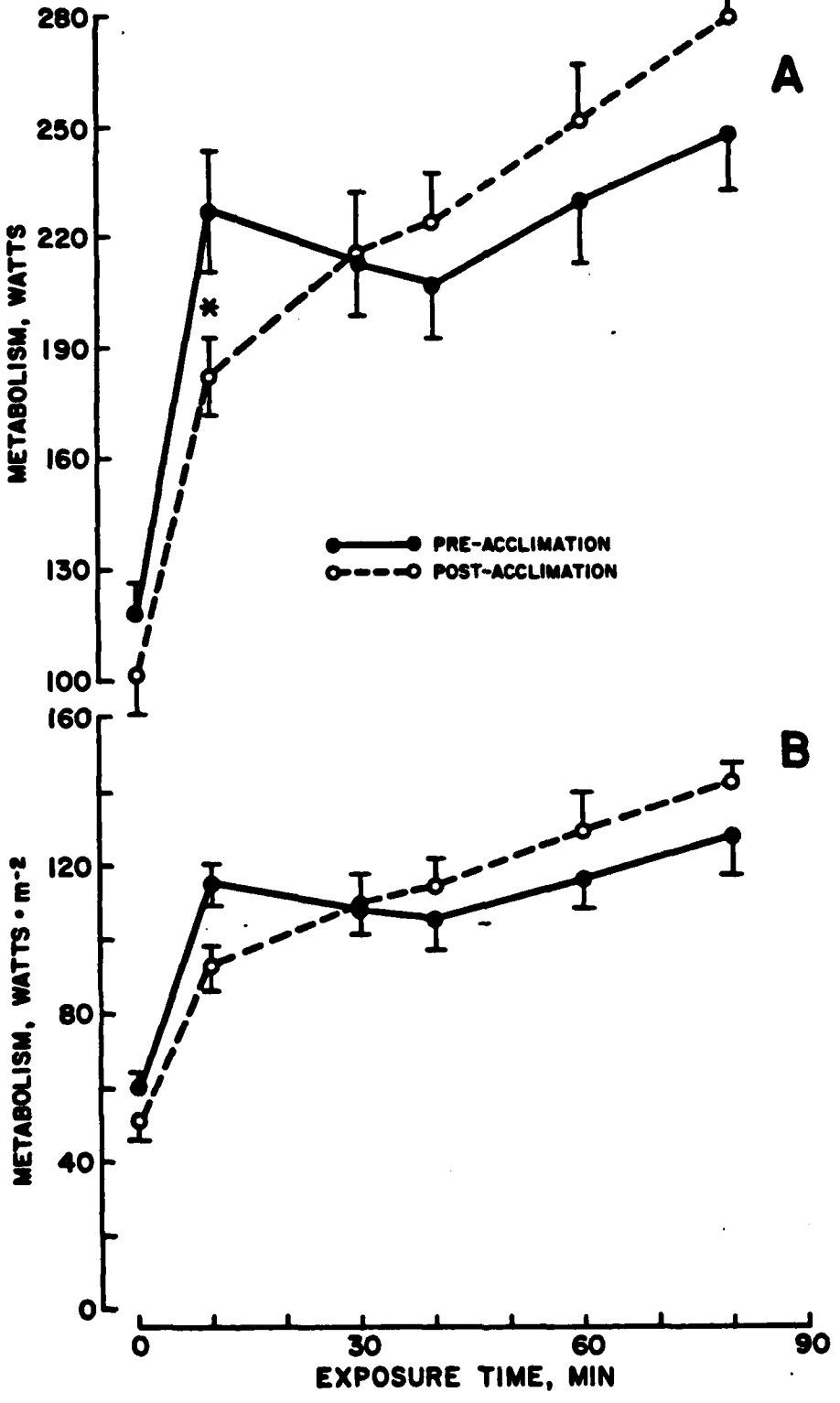

A
B
METABOLISM, WATTS
METABOLISM, WATTS • m⁻²
PRE-ACCLIMATION
POST-ACCLIMATION
EXPOSURE TIME, MIN
280
250
220
190
160
130
100
120
80
40
0
30
60
90
*

FIG 1

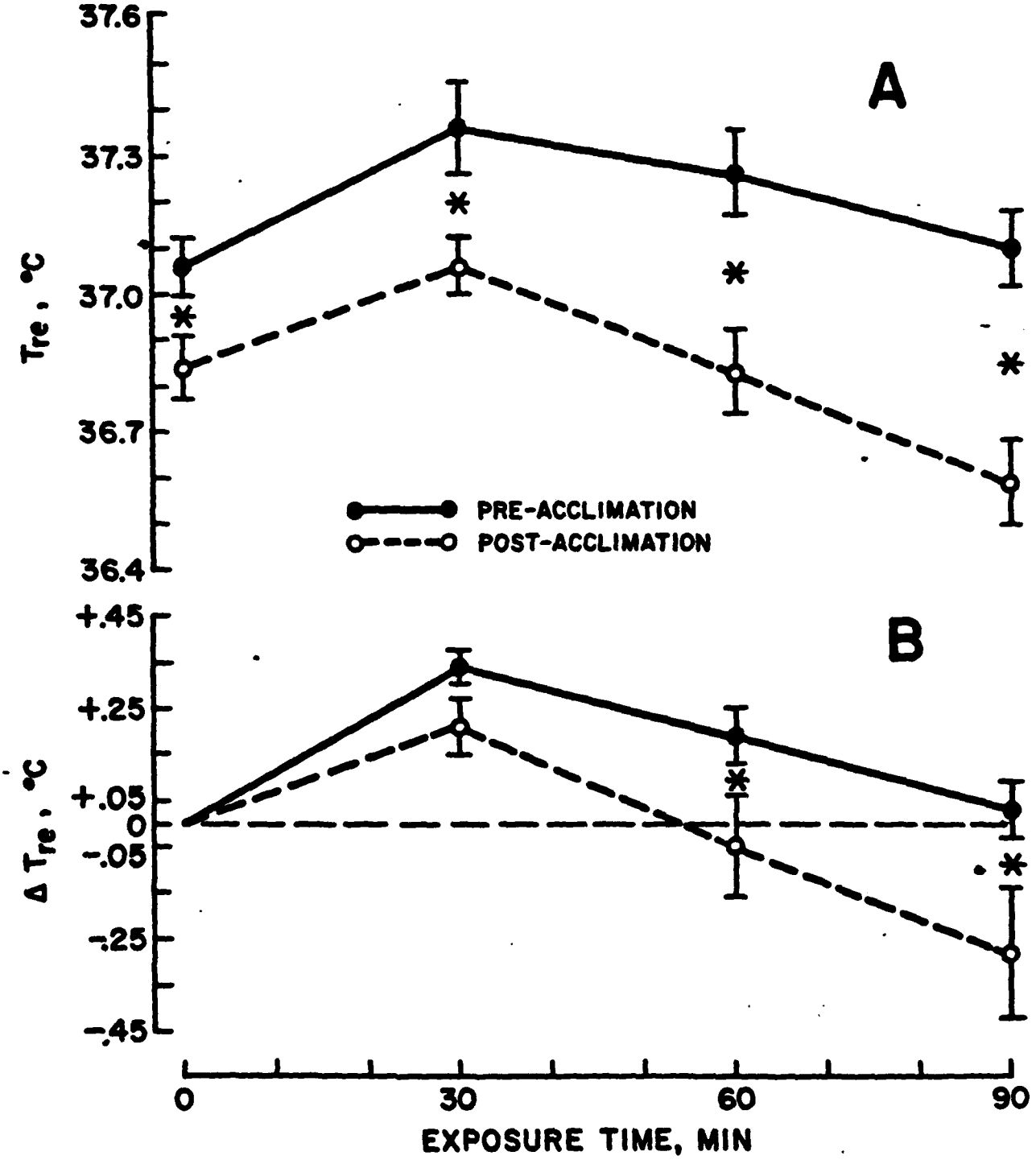

37.6
37.3
37.0
36.7
36.4
Tre , °C
A
PRE-ACCLIMATION
POST-ACCLIMATION
+.45
+.25
+.05
0
-.05
-.25
-.45
Δ Tre , °C
B
0
30
60
90
EXPOSURE TIME, MIN

Fig 2

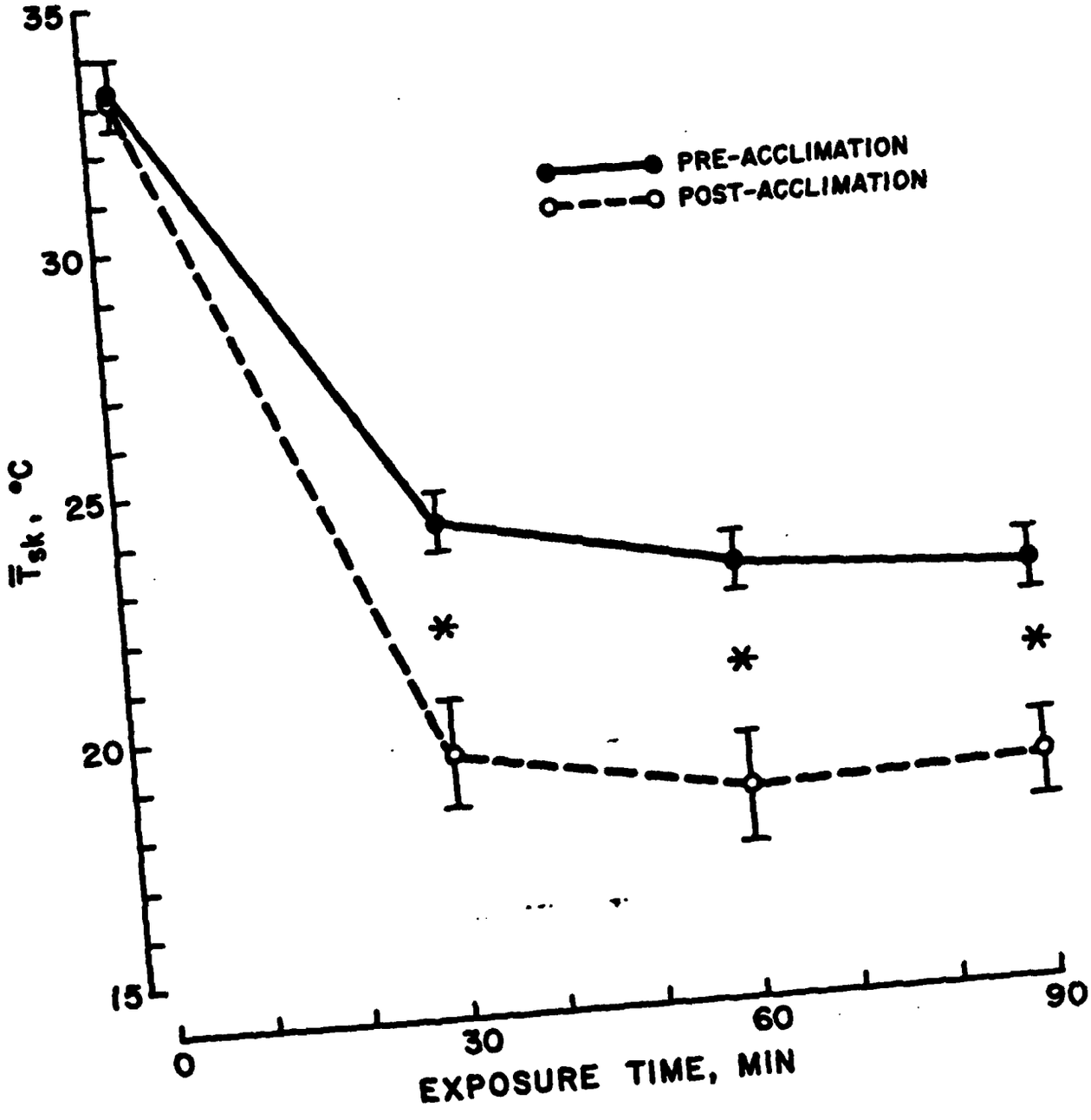

35
30
25
20
15
$\bar{T}_{sk}$, °C
PRE-ACCLIMATION
POST-ACCLIMATION
*
*
*
0
30
60
90
EXPOSURE TIME, MIN

Fig 3

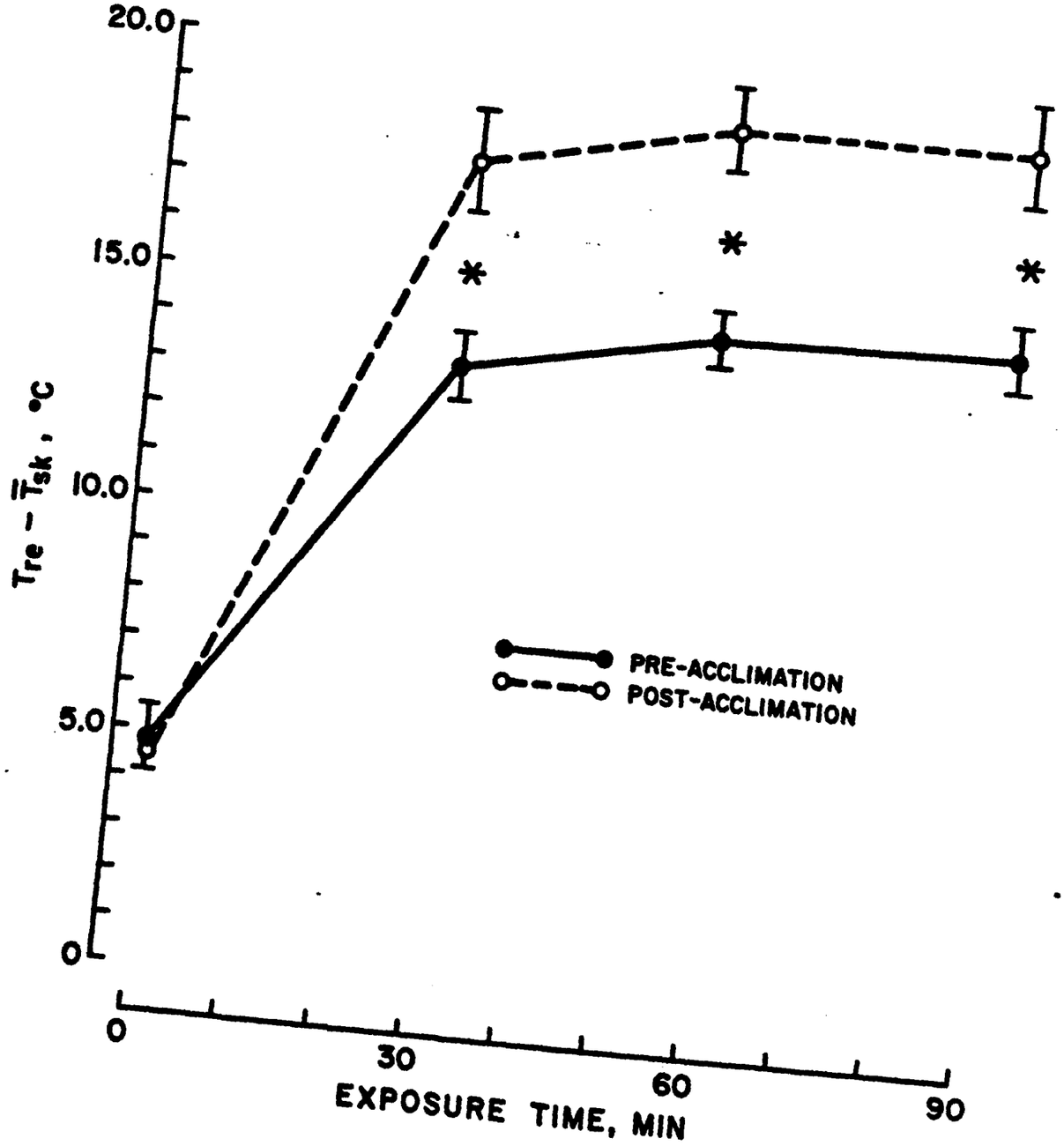
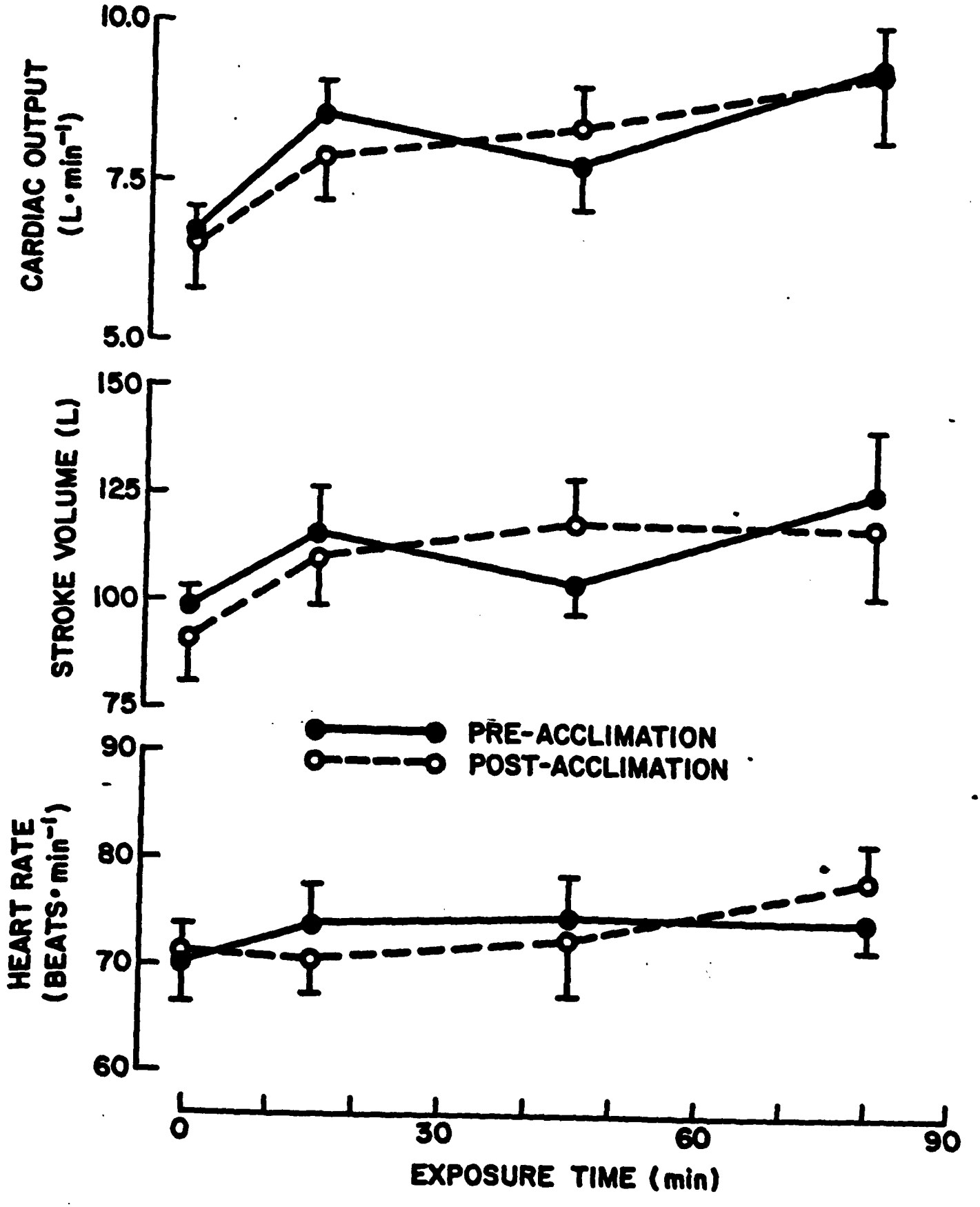

20.0
15.0
10.0
5.0
0
Tre − T̄sk , °C
*
*
*
PRE-ACCLIMATION
POST-ACCLIMATION
0
30
60
90
EXPOSURE TIME, MIN

Fig 4


CARDIAC OUTPUT (L·min⁻¹)
10.0
7.5
5.0
STROKE VOLUME (L)
150
125
100
75
PRE-ACCLIMATION
POST-ACCLIMATION
HEART RATE (BEATS·min⁻¹)
90
80
70
60
0
30
60
90
EXPOSURE TIME (min)

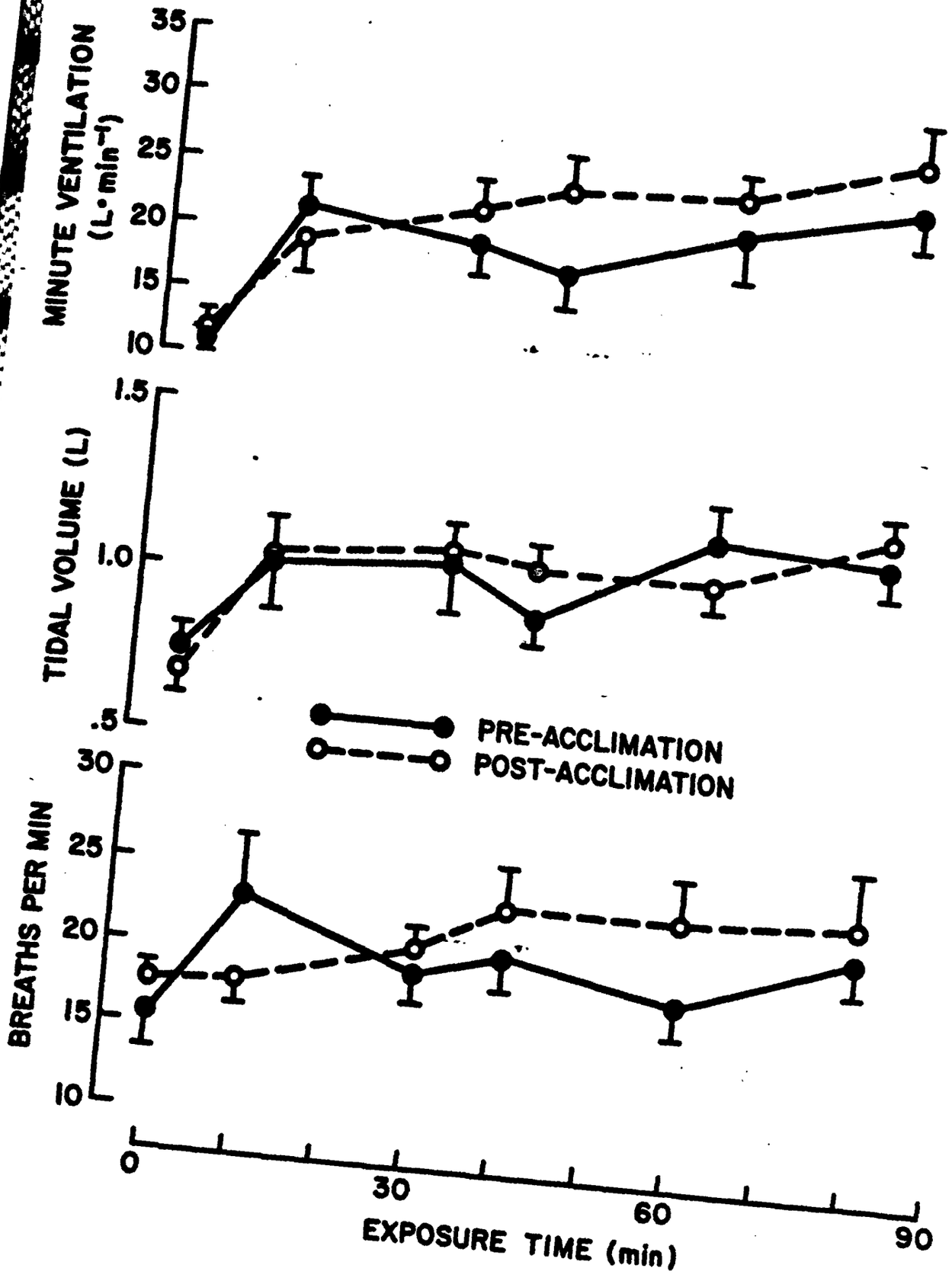

MINUTE VENTILATION (L·min⁻¹)
35
30
25
20
15
10
TIDAL VOLUME (L)
1.5
1.0
.5
PRE-ACCLIMATION
POST-ACCLIMATION
BREATHS PER MIN
30
25
20
15
10
0
30
60
90
EXPOSURE TIME (min)

FIG 6

END

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