Acute head-down tilt decreases the postexercise resting threshold for forearm cutaneous vasodilation

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Received 14 January 2000; accepted in final form 21 June 2000

Kenny, Glen P., Dwayne N. Jackson, and Francis D. Reardon. Acute head-down tilt decreases the postexercise resting threshold for forearm cutaneous vasodilation. J Appl Physiol 89: 2306–2311, 2000.—The purpose of this study was to evaluate the role of baroreceptor control on the postexercise threshold for forearm cutaneous vasodilation. On four separate days, six subjects (1 woman) were randomly exposed to 65° head-up tilt and to 15° head-down tilt during a No-Exercise and Exercise treatment protocol. Under each condition, a whole body water-perfused suit was used to regulate mean skin temperature (Tsk) in the following sequence: 1) cooling until the threshold for vasoconstriction was evident; 2) heating (~7.6°C/h) until vasodilation occurred; and 3) cooling until esophageal temperature (Tes) and Tsk returned to baseline values. The Exercise treatment consisted of 15 min of cycling exercise at 70% maximal O2 uptake, followed by 15 min of recovery in the head-up tilt position. The No-Exercise treatment consisted of 30 min resting in the head-up tilt position. After the treatment protocols, subjects were returned to their pretreatment condition, then cooled and warmed again consecutively. The calculated Tes threshold for cutaneous vasodilation increased 0.24°C postexercise during head-up tilt (P < 0.05), whereas no difference was measured during head-down tilt. In contrast, sequential measurements without exercise demonstrated a time-dependent decrease for head-up tilt (0.17°C) and no difference for head-down tilt. Pretreatment thresholds were significantly lower during head-down tilt compared with head-up tilt. We have shown that manipulating postexercise venous pooling by means of head-down tilt, in an effort to reverse its impact on baroreceptor unloading, resulted in a relative lowering of the resting postexercise elevation in the Tes for forearm cutaneous vasodilation.

IN RECENT STUDIES, THE EFFECTS of exercise on the postexercise threshold of cold (i.e., vasoconstriction and shivering) (8, 9) and warm (i.e., cutaneous vasodilation and sweating) (7) thermoregulatory responses have been evaluated during seated upright resting. These studies have indicated that exercise induces a residual effect on thermal control by increasing (~0.3°C) the postexercise resting threshold for cold and warm thermal responses. These data support the previous findings of Thoden et al. (22), who observed that, after 15 min of moderate exercise, skin blood flow and mean skin temperatures (Tsk) returned to baseline values within 15–20 min during postexercise resting, despite a sustained prolonged elevation of esophageal temperature (Tes). Furthermore, in a previous effort to address the mechanism for the postexercise increase in thermal response thresholds, subjects were immersed in warm water (42°C) until Tes increased to levels similar to those induced by 15 min of exercise (10). After exit from the warm water, Tes, Tsk, and skin blood flow all returned to control values within 10 min. Thus it is likely that the postexercise increase in both Tes and the onset threshold for vasodilation are not a consequence of an increased body heat content (10). Although the actual mechanism of the postexercise thermal response remains unclear, recent evidence may favor a baroreceptor-mediated influence.

It is known that active cutaneous vasodilation is normally under baroreceptor control (5). Acute bouts of exercise have been shown to cause postexercise hypotension (3). A decrease in skin blood flow (i.e., vasoconstriction) would tend to maintain venous return and adequate filling pressure in response to the decrease in systemic arterial resistance. This nonthermal factor may influence the elevated threshold for vasconstriction (and vasodilation) during exercise recovery.

Therefore, we propose that the nonthermal baroreceptor response may modify the normal thermoregulatory activity in elevating the threshold for vasodilation during exercise recovery. Within this study, we manipulated postexercise venous pooling by means of head-down tilt, in an effort to reverse its impact on baroreceptor unloading. By doing so, we tested the hypothesis that head-down tilt would result in a relative lowering of the resting postexercise elevation in the Tes for forearm cutaneous vasodilation.

METHODS

Subjects

On approval from the Research Ethics Board, six healthy subjects (5 men, 1 woman) with no history of cardiovascular...
or respiratory disease consented to participate in the study. Subjects were physically active, although none engaged in daily or intensive training programs. Subjects were aged 23 ± 4 (SD) yr, were 1.7 ± 0.1 m tall, and weighed 74 ± 3 kg. The female subject was eumenorrheic with regular, ~28-day-long menstrual cycles. To control for hormonal effects, the female subject was tested twice per month (on days 1 and 4 after the start of her menstrual cycle).

Instrumentation

Core temperature was measured by using an esophageal thermocouple inserted through a nostril to the level of the heart. Skin temperature was monitored at 11 sites by heat flow sensors (Concept Engineering, Old Saybrook, CT), and the area-weighted mean ($T_{sk}$) was calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9.5%, and posterior calf 9.5%.

Forearm skin blood flow was assessed with a laser-Doppler flow probe placed on the midanterior forearm (blood perfusion monitor, TSI, St. Paul, MN). Laser-Doppler flowmetry provides a linear index of skin blood flow from ~1 mm$^2$ of skin area and is based on the frequency of shift of coherent laser light induced by erythrocytes moving in the cutaneous vessels (20). The laser-Doppler flow sensor was taped to the cleaned skin surface at a location that gave a consistent reading. Only relative values were used, and no attempt was made to evaluate absolute blood flow.

Temperatures were collected and digitized (Hewlett-Packard data acquisition module, model 3497A) at 5-s intervals, displayed graphically on the computer screen, and recorded in spreadsheet format on a hard disk (Hewlett-Packard, model PC-312, 9000).

Experimental Protocol

Subjects performed one incremental maximal $O_2$ uptake test on a cycle ergometer on the first day. These data were used to select the work load for the submaximal experimental exercise trials.

Each subject performed a total of four experimental trials that were carried out in a random order. The general phases for each of the four experimental trials are presented in Fig. 1. Briefly, the four trials were as follows: 1) Head-up tilt/Exercise/Head-up tilt, 2) Head-up tilt/No-Exercise/Head-up tilt, 3) Head-down tilt/Exercise/Head-down tilt, and 4) Head-down tilt/No-Exercise/Head-down tilt. Each of the four trials involved three distinct phases that were identified as pretreatment period, treatment period, and posttreatment period.

Each trial was separated by a minimum of a 24-h period without heavy or prolonged physical activity, the last 12 h of which included abstinence from stimulants and alcohol, 8 h of sleep, and a minimum of 0.25 liter of water during each waking hour. On each study day, care was taken to avoid major thermal stimuli or substantial increase of metabolic rate between awakening and the start of the experiment. All trials were conducted during the winter months from November to February. To ensure that the subjects were euhydrated during each experimental trial, they were encouraged to ingest water ad libitum during the application of the instrumentation on experimental days. The experimental trials were conducted either in the morning (3 subjects) or mid-afternoon (3 subjects). This allowed us to consider any changes in thermoregulatory thresholds due to circadian rhythms before and after zenith.

On arrival at the laboratory, subjects, clothed in shorts and running shoes, were instrumented appropriately. They were then outfitted with a water-perfused suit that covered the entire body except the face, fingers, and feet. It should be noted that the water-perfused suit covered the midanterior forearm that was the site for skin blood flow measurement. Spandex pants, a cotton sweatshirt, and a cotton head cover were worn over the water-perfused suit. To determine the relative treatment effect on the threshold for cutaneous forearm vasodilation ($T_{thVD}$), each subject underwent a baseline resting evaluation of $T_{thVD}$ in all four trials (pretreatment warming). Subjects were positioned in a 65° head-up tilt position on a Hang-Up tilt table in a climatically controlled chamber for a minimum period of 15 min at an ambient temperature of 24°C. Baseline resting was then followed by the respective periods described below.

Pretreatment period. In two of the experimental trials, the pretreatment period was carried out with the subject in the 65° head-up tilt position. In the other two experimental trials, the subject was positioned in a 15° head-down tilt. Once the subject was placed in the respective position, cool water (~20°C) was circulated through the water-perfused suit, and ambient temperature was reduced to 14°C until forearm cutaneous vasoconstriction was noted (cooling, ~25 min). Ambient temperature was then increased to 29°C. $T_{sk}$ was increased at a rate of 7.0°C/h (as the water circulating through the suit was progressively increased to 47°C) until forearm cutaneous vasodilation was noted (warming, ~37°C).

Each subject was eumenorrheic with regular, ~28-day-long menstrual cycles. To control for hormonal effects, the female subject was tested twice per month (on days 1 and 4 after the start of her menstrual cycle).
min). $T_{es}$ and $T_{sk}$ were subsequently decreased to near baseline values by again perfusing the suit with cool water and decreasing ambient temperature to 24°C (cooling, ~30 min).

**Treatment period.** Immediately after the pretreatment period, subjects either exercised (Exercise) or remained resting (No-Exercise). For the Exercise treatment, the subject performed 15 min of cycle ergometer exercise at 70% of predetermined maximal O$_2$ uptake. The subject was then moved back to the tilt table to complete 15 min of resting recovery in the 65° head-up tilt position. For the No-Exercise treatment, the subject was instructed to rest in the 65° head-up tilt position for 30 min.

**Posttreatment period.** To measure the effect of postexercise venous pooling on the resting postexercise $T_{VDD}$, posttreatment resting measurements of $T_{VDD}$ were conducted. Immediately after both the No-Exercise and Exercise treatments, subjects were returned to their original (pretreatment) position on the tilt table. Once the subject was resting in the respective position, the procedure for measuring thermoregulatory thresholds was carried out as that of the pretreatment condition. Specifically, cool water (~20°C) was circulated through the water-perfused suit, and ambient temperature was reduced to 14°C until forearm cutaneous vasodilation was noted (cooling, ~32 min). Ambient temperature was then increased to 29°C, $T_{sk}$ was increased at a rate of 7.0°C/h (as the water circulating through the suit was progressively increased to 47°C), and forearm cutaneous vasodilation was noted (warming, ~37 min).

The inclusion of the cooling phase before whole body heating was not intended to burden or address thermoregulatory function during cooling. Although it appears that the cooling phase may have added a degree of thermal history, it was included to ensure that all subjects were brought to the same baseline so that true release of vasoconstrictor tone and subsequent vasodilation during whole-body heating could be noted in all trials. Thus it can be said that all subjects during all trials were given the same thermal history in the pre- and posttreatment periods.

The esophageal temperature threshold for the onset of cutaneous vasodilation ($T_{VDD}$) was defined as the onset of a sustained rise in forearm blood flow (6). To compare thresholds between conditions in which both $T_{es}$ and $T_{sk}$ were changing, the following equation (12) was used to correct the $T_{es}$ ($T_{es(calculated)}$) for a designated skin temperature ($T_{sk(designated)}$)

$$T_{es(calculated)} = T_{es} + [\beta/(1-\beta)](T_{sk} - T_{sk(designated)})$$

where $T_{sk(designated)}$ was set as the average $T_{sk}$ of pre- and post-No-Exercise warming and pre- and post-Exercise warming for both the head-up tilt and head-down tilt conditions (32°C) and $\beta$ is fractional contribution of the skin to the vasodilation (0.2) (24).

**Analysis of Results**

For the purpose of comparison, thermoregulatory response thresholds were identified during 1) pre- and posttreatment for head-up tilt and head-down tilt warming for the No-Exercise condition and 2) pre- and posttreatment for head-up tilt and head-down tilt warming for the Exercise condition. A three-way mixed ANOVA with two repeated measures [i.e., repeated-measures ANOVA with tilt position (head-up tilt vs. head-down tilt) and treatment period (pre- vs. posttreatment) as the repeated factors] was used to test for significant differences in $T_{sk}$, $T_{es}$, and $T_{es(calculated)}$ at $T_{VDD}$. In the event of statistical significance ($P < 0.05$), a Tukey’s test was used to identify significant differences. Data are presented as means ± SD.

**RESULTS**

There were no differences between pre- and posttreatments of warming for the subject perfusate (~19.5°C/h) in either the No-Exercise and Exercise conditions in the head-up tilt or head-down tilt positions. Thus $T_{sk}$ (~7.0°C/h) and $T_{es}$ (~0.4°C/h) increased at the same respective rates in both treatment conditions. Baseline resting $T_{es}$ and $T_{sk}$ were similar for all conditions and remained stable and consistent during the 15-min period before the initial surface cooling.

**No-Exercise Treatment: Effect of Time on Forearm Vasodilation**

Mean skin and core temperatures at the onset of vasodilation are presented in Table 1. The mean and individual corrected $T_{es}$ at the onset of vasodilation are presented in Fig. 2. The corrected $T_{es}$ threshold for vasodilation during preresting warming was significantly greater for head-up tilt compared with head-down tilt (36.81 ± 0.12°C and 36.66 ± 0.16°C for head-up tilt and head-down tilt warming, respectively) in the No-Exercise condition.

For the pre- and postresting head-up tilt warming, the onset threshold for forearm vasodilation decreased from 36.81 ± 0.12°C to 36.64 ± 0.13°C, respectively ($P < 0.05$). In contrast, the onset thresholds remained

| Table 1. Threshold values for pre- and posttreatment in the No-Exercise and Exercise conditions and for both the head-up and head-down positions |
|---------------------------------|-----------------|-----------------|-----------------|-----------------|
|                                | No-Exercise     | Exercise        |                 |                 |
|                                | Pretreatment    | Posttreatment   | Pretreatment    | Posttreatment   |
| Head-Up Tilt                   |                 |                 |                 |                 |
| Actual $T_{sk}$                | 32.66 ± 0.55    | 32.39 ± 0.54    | 32.55 ± 0.48    | 31.97 ± 0.48    |
| Actual $T_{es}$                | 36.61 ± 0.16†   | 36.60 ± 0.16†   | 36.61 ± 0.10†   | 36.66 ± 0.08†   |
| Head-Down Tilt                 |                 |                 |                 |                 |
| Actual $T_{sk}$                | 31.53 ± 0.45    | 31.78 ± 0.18    | 32.00 ± 0.37    | 32.80 ± 0.73    |
| Actual $T_{es}$                | 36.89 ± 0.08    | 36.69 ± 0.13*   | 36.79 ± 0.13    | 36.93 ± 0.10*   |

Values are means ± SD. $T_{sk}$, mean skin temperature; $T_{es}$, esophageal temperature. *Temperature significantly different from pretreatment for both No-Exercise and Exercise conditions; †head-down tilt temperature significantly different from head-up tilt temperature in the similar treatment period for both the No-Exercise and Exercise conditions.
unchanged between pre- and postresting during the head-down tilt warming (36.66 ± 0.16°C and 36.62 ± 0.16°C, respectively).

Exercise Treatment: Effect of Exercise on Forearm Vasodilation

The vasodilatory thresholds for preexercise warming for head-down tilt were significantly lower than for preexercise warming during head-up tilt (P < 0.05) (Fig. 3). The difference is consistent with that measured during the No-Exercise condition. The onset threshold for skin vasodilation increased from 36.77 ± 0.14°C during preexercise warming to 37.01 ± 0.13°C during postexercise warming in the head-up tilt position (P < 0.05). In contrast, no difference in the onset threshold for vasodilation was measured between pre- and postexercise warming in the head-down tilt position (36.65 ± 0.12°C and 36.64 ± 0.11°C, respectively).

DISCUSSION

The purpose of this study was to evaluate the influence of venous pooling on postexercise thermoregulatory thresholds. More specifically, we evaluated the effect of a reduction in postexercise venous pooling by means of head-down tilt on the postexercise ThVD. The most important observation of this study was the absence of an elevated postexercise ThVD in the head-down tilt condition. It was noted that 1) at rest, the esophageal ThVD was higher in the head-up position (36.81 ± 0.12°C) compared with the head-down position (36.66 ± 0.16°C) (P < 0.05); 2) exercise produced an expected increase in the Tes ThVD in the head-up tilt position (0.24°C) (P < 0.05); and 3) the increase in the postexercise Tes ThVD was attenuated in the head-down tilt position. The increase in the postexercise resting threshold for cutaneous vasodilation confirms a similar increase of warm (i.e., sweating) (7) and cold (i.e., vasoconstriction and shivering) thermal responses (8, 9) measured during upright seating.

The postexercise increase in the threshold for cutaneous vasodilation during head-up tilting was not caused by differences in rate of change of either Tes or Tsk because warming rates were not significantly different for the pre- and postexercise warming period.

Brengelmann et al. (2) have demonstrated a time-dependent decrease in sweating threshold in experiments of >2-h duration. Thus it is thought that the prolonged protocol used in this study in and of itself probably did not contribute to the observed postexercise elevated threshold for cutaneous vasodilation (in the head-up tilt position). The observed time-dependent decrease in the threshold for cutaneous vasodilation under resting conditions [i.e., 0.17 and 0.04°C for head-up tilt (P < 0.05) and head-down tilt (not significant), respectively] is most likely and at best an underestimation of the exercise-induced increase in the threshold for cutaneous vasodilation. The circadian

![Fig. 2. Mean (● connected by solid line) and individual core corrected thresholds for vasodilation during pre- and posttreatment warming for the No-Exercise condition in both the head-up tilt (top) and head-down tilt (bottom) positions (n = 6). Bars, SD; *significantly different from preresting warming (P < 0.05).](http://jap.physiology.org/)

![Fig. 3. Mean (● connected by solid line) and individual core corrected thresholds for vasodilation during pre- and posttreatment warming for the Exercise condition in both the head-up tilt (top) and head-down tilt (bottom) positions (n = 6). *Significantly different from preresting warming (P < 0.05).](http://jap.physiology.org/)
effect on set-point shift and therefore the time of day at which the tests were conducted may have had some bearing on the observed results. However, the results of those experimental trials conducted at zenith in early afternoon were comparable to those conducted in early morning.

It has been demonstrated that baroreceptor unloading increases the threshold for cutaneous vasodilation. Specifically, Mosley (15) showed that there is cutaneous vasoconstrictor activity in response to head-up tilting. Similar results have been demonstrated during lower body negative pressure (1, 13, 18, 23). This modification has been demonstrated during passive heating at rest (4) and by exercise (11). Our data are consistent with these findings in that we showed a lower pretreatment threshold for vasodilation during head-down tilting for both No-Exercise (0.15°C) and Exercise (0.12°C) conditions, respectively, compared with head-up tilting (Figs. 2 and 3). Thus it is clear that, with the lack of peripheral pooling after exercise in the head-down tilt position, the exercise effect on the \( T_{es} \cdot T_{VD} \) is attenuated. These data suggest that baroreceptor unloading due to peripheral blood pooling in the postexercise period contributes to the postexercise increase in the \( T_{es} \cdot T_{VD} \).

Although the mechanism(s) for thermoregulatory control of skin blood flow before and during exercise have been described (4, 6, 19, 21), postexercise regulation remains unclear. It is known, however, that the cutaneous vasodilator system is under baroreceptor control (6). Cutaneous vascular tone is a determinant of blood flow and blood pressure regulation during both exercise and upright posture (17). Acute bouts of exercise have been shown to cause postexercise hypotension. This is likely due to the persisting blood pooling in the previously active musculature. The resultant baroreceptor unloading elicits peripheral vasoconstriction in an attempt to maintain blood pressure. If peripheral vasoconstriction is not adequate to overcome the hypotension caused by blood pooling, then the hypotensive condition persists. It can be noted, however, that, in an attempt to preserve blood pressure (via peripheral vasoconstriction), the body’s ability to dissipate accumulated heat is diminished. It is plausible, therefore, that the increase in the postexercise threshold for cutaneous vasodilation is the result of a strong nonthermoregulatory drive (i.e., baroreceptor reflex) to maintain normal postexercise blood pressure.

Thus it would seem that skin blood flow control after exercise is subject to significant modifications by nonthermoregulatory baroreceptor reflexes. Because acute reductions in central venous pressure have been shown to delay or decrease the rise in cutaneous blood flow during heat stress (11, 14, 16), a reasonable postulate is that cardiopulmonary baroreceptors are involved in the postexercise cutaneous vasoconstriction during postexercise resting. Modification of baroreceptor response on cutaneous vascular tone would be manifested either as an activation of sympathetic adrenergic vasoconstrictor nerves or as a withdrawal of active vasodilator activity (5).

We have shown that manipulating postexercise venous pooling, by means of head-down tilt, in an effort to reverse its impact on baroreceptor unloading, resulted in a relative lowering of the resting postexercise elevation in the \( T_{es} \) for forearm cutaneous vasodilation. Thus it would seem that the nonthermal factor of baroreceptor control significantly influences cutaneous vasomotor control during exercise recovery. Further studies are required to examine the mechanism of control and to determine how nonthermoregulatory reflexes, such as baroreceptor control, may possibly modify postexercise warm and cold thermal responses.

We acknowledge Dr. Gordon Giesbrecht of the University of Manitoba for assistance in this project and the technical assistance of Carolyn Proulx.

This research was supported by Natural Science and Engineering Research Council (Canada).

REFERENCES


