Increasing Exercise Duration Does Not Affect the Postexercise Elevation in Esophageal Temperature

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Abstract/Résumé

It has previously been observed that (a) following 15 min of intense exercise, esophageal temperature (Tes) remains elevated at a plateau value equal to that at which active vasodilation had occurred during exercise (i.e., esophageal temperature threshold for cutaneous vasodilation [Thvd]); and (b) exercise/recovery cycles of identical intensity and duration, when sequential, result in progressively higher Tes at the beginning and end of exercise. In the latter case, parallel increases in both the exercise Thvd and postexercise plateau of Tes were noted. This study was conducted to determine if the elevated postexercise Tes is related to increases in whole-body heat content. On separate occasions, 9 subjects completed 3 bouts of treadmill exercise at 70% \( \dot{V}O_2 \text{max} \), 29 °C ambient temperature. Each exercise bout lasted either 15, 30, or 45 min and was followed by 60 min of inactive recovery. Esophageal temperatures were similar at the start of each exercise bout, but the rise in Tes during exercise nearly doubled from 1.0 °C after 15 min of exercise to 1.9 °C after 45 min of exercise. There were no intercondition differences among the exercise Thvd (−0.36 °C above baseline) or postexercise plateau values for Tes (−0.40 °C above baseline). Thus the relationship between the Thvd during exercise and the postexercise Tes did not appear to be dependent on changes in whole-body heat content as produced by endogenous heating during exercise of different duration.

D'après des études, nous savons que (a) après 15 min d'un effort intense, la température œsophagienne (Tes) se maintient à une valeur équivalente à celle associée à la manifesta-

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tion de la vasodilatation active durant l’effort (i.e., le seuil de température œsophagienne pour une vasodilatation cutanée [ThVD]); (b) au cours de séquences effort/repos de durée et d’intensité égales, la TeS augmente graduellement au début et à la fin de l’effort. On note aussi une augmentation parallèle de la ThVD à l’effort et de la TeS après l’effort. Cette étude est entreprise pour vérifier si l’augmentation de la TeS après l’effort est associée à l’augmentation du contenu de chaleur corporelle. À des moments différés, 9 sujets participent à des séances d’exercice sur tapis roulant à 70% du VO₂ max et à une température ambiante de 29 °C. Chaque séance, d’une durée de 15, 30, ou 45 min, est suivie d’une récupération inactive de 60 min. Au début de chacune des séances, la TeS est du même ordre mais la température au cours de l’effort est presque le double, soit 1 °C après 15 min d’effort et 1,9 °C après 45 min. D’une condition à l’autre, il n’y a pas de variation de la ThVD (−0,36 °C au-dessus du niveau de base) et de la TeS en récupération (−0,40 °C au-dessus du niveau de base). Ainsi, la relation entre la ThVD à l’effort et la TeS en récupération ne semble pas être dépendante des variations du contenu de chaleur corporelle obtenues par la production de chaleur au cours d’un effort d’une durée variable.

Introduction

We have previously demonstrated a prolonged (65 min or longer) elevation of esophageal temperature (TeS), compared to preexercise resting values, in humans following dynamic exercise at a thermoneutral temperature (Ta) of 29 °C (Thoden et al., 1994). This TeS value was equal to the threshold esophageal temperature at which active cutaneous vasodilation was initiated during exercise (ThVD). This difference in resting and postexercise TeS was not of a metabolic origin, as oxygen consumption (VO₂) returned to baseline values within 5–10 min of exercise termination. Forearm skin blood flow and skin temperatures (TsK) at all sites, except over the exercised muscles, also decreased to control values within 10–15 min after cessation of exercise. The reduction of TsK and skin blood flow throughout the period of prolonged TeS elevation, following cessation of exercise, is consistent with an exercise-induced elevation of the ThVD that persists into recovery.

The observation—that during successive exercise/recovery cycles performed at progressively increasing preexercise TeS levels there are parallel increases of (a) the exercise threshold for cutaneous vasodilation during exercise and (b) the postexercise TeS (Kenny et al. 1996a)—suggests a physiological relationship between these latter two parameters. Furthermore, it was demonstrated that the relationship between exercise ThVD and postexercise TeS remained intact over a wide range of ambient temperature (Ta) (20–40 °C)/exercise intensity (45–75% VO₂ max) conditions (Kenny et al. 1996a, 1996b, 1997a; Thoden et al., 1994). This indicates that the hypothetical relationship between ThVD during exercise and the postexercise TeS is not coincidental but of physiological origin. Subsequent studies have confirmed that this increase in postexercise ThVD at rest is comparable to the ThVD during exercise (Kenny et al. 1997b).

The relationship between ThVD during exercise and the postexercise TeS was studied under conditions of increased whole-body heat content (i.e., endogenous heating) produced by (a) increased ambient temperature, (b) increased exercise intensity, (c) elevated preexercise resting TeS, or (d) different combinations of these. However, each of these factors is known to influence ThVD during exercise and the subsequent rate of heat loss (Hirata et al., 1984; Kenny et al., 1996a, 1997b; Nadel
et al., 1979; Saltin et al., 1970). Therefore, in order to determine whether the magnitude of the postexercise elevation in $T_{es}$ is related to the exercise-induced heat load (endogenous heat load) or is defined by factors known to modify the exercise $T_{hvd}$, it is necessary to impose a condition that results in increased whole-body heat content without inducing a concomitant change in $T_{hvd}$.

Therefore to avoid the effects of such factors as ambient temperature, exercise intensity, and preexercise resting $T_{es}$, we compared the relationship between the $T_{hvd}$ during exercise and postexercise $T_{es}$ under standard conditions of $T_a$ of 29 °C after 15, 30, and 45 min of moderate exercise.

**Methods**

**SUBJECTS**

Upon approval of the project by the Human Research Ethics Committee, 7 men and 2 women with no history of cardiovascular or respiratory disease consented to participate in the study. Physically active, nontrained subjects were used in order to avoid any confounding cardiovascular adaptations to prolonged exercise (Johnson and Propppe, 1996). On average, subjects were (mean ± SD) 24 ± 3 years of age, weighed 77 ± 3 kg, were 1.7 ± 0.2 m tall, had a mean body fat content of 13.1 ± 1.7%, and had a maximal aerobic capacity of 45.4 ± 3.0 ml $O_2$ · kg⁻¹ · min⁻¹. Women were eumenorrheic with regular menstrual cycles of approximately 28 days. To control for hormonal effects, the women were studied within 9 days after the start of menstruation (follicular phase).

**INSTRUMENTATION**

Core temperature was measured using an esophageal thermocouple inserted through a nostril to the level of the heart. Skin temperature was monitored at 14 sites, and the area-weighted mean ($T_{ak}$) was calculated by assigning regional percentages as follows: head 6%; upper arm 9%; forearm 6%; hand 2.5%; finger 2%; anterior calf 7.5%; posterior calf 6%; foot 4%; and 9.5% each to the chest, abdomen, upper back, lower back, anterior thigh, and posterior thigh.

Forearm 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 approximately 1 mm² of skin area and is based on the frequency of shift of coherent laser light induced by erythrocytes moving in the cutaneous vessels (Stern et al., 1977). Only relative values were used and no attempt was made to evaluate absolute blood flow.

Oxygen consumption ($VO_2$) was determined by an automated open-circuit method (Quinton, Q-plex) from measurements of expired minute volume and mixed expired gas concentrations sampled from a 10-L fluted mixing box. Temperatures were recorded (Hewlett Packard data acquisition module, Model 3497A) at 5-sec intervals and simultaneously displayed on the computer screen, then stored in spreadsheet format on a hard disk (Hewlett Packard, Model PC-312, 9000).

**EXPERIMENTAL PROTOCOL**

Subjects underwent an incremental maximal $VO_2$ test on a treadmill on the first day. The measured $VO_2$ max was used to establish the work intensity for the sub-
sequent experiment trials. The subjects then participated in 3 trials conducted on separate days either in the morning ($n = 5$) or late afternoon ($n = 4$). Each trial followed a 72-hr period free of heavy or prolonged physical activity and included at least 8 hrs of sleep and a minimum consumption of 0.25 L of water during each waking hour. No food intake was permitted within 2 hours prior to the experimental session. Care was taken to avoid major thermal stimuli or substantial increase of metabolic rate between awakening and the start of the experiment. Subjects completed their 3 experimental trials over a 2-week period.

After being appropriately instrumented, and dressed in shorts and running shoes, subjects entered the thermal chamber at an ambient temperature of 29 °C. Exercise began after a minimum habituation period of 1 hour in an upright position (with the subject leaning against a support table) and after which the $T_{es}$ did not vary by more than ±0.05 °C over 10 min. Prior to all exercise periods, the subject stepped on the treadmill and remained relatively still until postural effects on $T_{es}$ had ceased. He or she then performed in random order either 15-, 30-, or 45-min treadmill exercise (70% $\dot{V}O_2$ max) followed by 60 min of recovery in an upright position.

The threshold esophageal temperature for the onset of cutaneous vasodilation ($T_{vdo}$) was defined as a sustained rise in forearm skin blood flow (Kellogg et al., 1991). In order to standardize threshold estimation under conditions during which both esophageal ($T_{es}$) and mean skin ($T_{sk}$) temperatures were changing, the following equation (Matsukawa et al., 1995) was used to correct the esophageal temperature [$T_{es}$(calculated)]) for a designated skin temperature [$T_{sk}$(designated)]:

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

$T_{sk}$(designated) was the average $T_{sk}$ of the three exercise conditions (32.5°C) and $\beta$ was the fractional contribution of the skin to the vasodilation response ($\beta = 0.1$) (Sawka and Wenger, 1988).

**ANALYSIS OF RESULTS**

Preexperimental $T_{es}$, $T_{sk}$, forearm blood flow, and heart rate are presented as an average of the final 10 min of the control period. End-exercise and postexercise values are reported as 1-min averages at end-exercise and 5-min intervals (5, 10, 15 min, etc.) during the postexercise resting period. In order to represent the dynamic nature of $T_{es}$, $T_{sk}$, and forearm blood flow responses, the data were graphed at 5-sec intervals for each condition. Repeated measures ANOVA comparisons of $T_{es}$, $T_{sk}$, forearm blood flow, and heart rate were done at preexercise, the $T_{vdo}$ during exercise, at end-exercise, and at 5-min intervals during postexercise resting for each condition. All data are reported as means unless otherwise stated.

**Results**

**EXERCISE ESOPHAGEAL AND SKIN TEMPERATURE RESPONSES**

All conditions resulted in similar temperature patterns during baseline rest and the first 15 min of exercise. Esophageal temperature during baseline was stable prior to exercise (37.00–37.02 °C) (Figure 1). Exercise $T_{es}$ decreased transiently for about 2 min after exercise began, followed by a steady increase at ~0.16 °C · min⁻¹
Figure 1. Mean (SEM) esophageal temperature (n = 9) during preexercise resting, exercise, and postexercise recovery periods for all 3 exercise conditions: 15 min (○), 30 min (□), and 45 min (▲). Trials were conducted at ambient temp. of 29 °C at 50% rel. humidity.

which decreased to ~0.03 °C · min⁻¹ within 1–1.5 min of the onset of cutaneous vasodilation (i.e., 5.6, 6.17, and 6.0 min of exercise during the 15, 30, and 45 min of exercises, respectively). Esophageal temperatures reached end-exercise values that were significantly greater (p < 0.01) with increases in exercise duration (37.97, 38.46, and 38.90 °C, respectively, for 15, 30, and 45 min).

Baseline Tsk was stable (32.38–32.55 °C) throughout the preexercise resting period. All but the calf skin temperature fell quickly after the onset of exercise and began to increase at or just prior to forearm cutaneous vasodilation. There was a subsequent decrease in Tcs 60–90 sec following the increase in Tsk under all conditions. Mean skin temperature increased for the remainder of exercise to an end-exercise value of 1.56, 2.23, and 2.48°C above baseline resting for the 15-, 30-, and 45-min exercises, respectively (Figure 2).

POSTEXERCISE ESOPHAGEAL AND SKIN TEMPERATURE RESPONSES

Upon cessation of exercise, Tcs subsequently decreased and reached an elevated plateau of 37.5 °C within 5, 15, and 30 min of exercise cessation for the 15-, 30-, and 45-min exercise conditions, respectively. Subsequently, esophageal temperature decreased over the remainder of 60 min of recovery to 37.41, 37.42, and 37.44 °C for 15-, 30-, and 45-min exercises, respectively. Mean skin temperature continued to rise for 2–3 min after cessation of exercise and returned to or near preexercise values within 20–30 min postexercise for all conditions. This pattern was followed by all but the thigh and calf sites, which were over the primary muscles for this
Figure 2. Mean (SEM) skin temperature ($n = 9$) during preexercise resting, exercise, and postexercise recovery periods for all 3 exercise conditions: 15 min (○), 30 min (□), and 45 min (■). Trials were conducted at ambient temp. of 29 °C at 50% rel. humidity.

exercise. Laser Doppler measurements throughout showed that forearm blood flow returned to baseline values within 10–25 min for all conditions (Figure 3).

CUTANEOUS VASODILATION AND POSTEXERCISE $T_{es}$

All postexercise $T_{es}$ values at the end of recovery (60 min postexercise) were significantly elevated above the initial baseline preexercise $T_{es}$ by 0.40, 0.42, and 0.42 °C for the 15-, 30-, and 45-min exercises, respectively ($p < 0.05$). The $T_{es}$ thresholds for forearm cutaneous vasodilation were 37.36, 37.34, and 37.39 °C (i.e., 0.36, 0.34, and 0.37 °C above baseline resting) and occurred within 5.6, 6.17, and 6.0 min of exercise during the 15-, 30-, and 45-min exercises, respectively. There were no significant differences between the exercise $T_{bvd}$ and postexercise $T_{es}$ for any conditions.

HEART RATE RESPONSE

Preexercise resting heart rate was similar for all conditions (Figure 4). At the start of exercise, heart rates for all conditions showed the same abrupt and then more gradual increase until exercise termination. End-exercise heart rates were significantly different between conditions (i.e., 161, 175, and 184 beats · min⁻¹ for 15-, 30-, and 45-min exercise, respectively) ($p < 0.05$). Heart rate decreased rapidly within 30 sec of end-exercise but remained elevated above resting values for the duration of the 60 min for all conditions (79, 90, and 97 beats · min⁻¹ for 15-, 30-, and 45-min exercises at 60 min of recovery).
Figure 3. Mean (SEM) forearm skin blood flow ($n = 9$) as measured by laser-Doppler flowmetry during preexercise resting, exercise, and postexercise recovery periods for all 3 exercise conditions: 15 min (○), 30 min (□), and 45 min (▲). Trials were conducted at ambient temp. of 29 °C at 50% rel. humidity.

Figure 4. Mean (SEM) heart rate response ($n = 9$) during preexercise resting, end-exercise, and postexercise recovery periods for all 3 exercise conditions: 15 min (○), 30 min (□), and 45 min (▲). Trials were conducted at ambient temp. of 29 °C at 50% rel. humidity.
Discussion

The primary observation of this experiment was that the magnitude of the postexercise resting elevation of $T_{es}$ over preexercise values did not differ between the three 15-, 30-, and 45-min exercise conditions. This was true despite the significantly greater end-exercise $T_{es}$ for each successive increase in exercise duration. In addition, the same relationship between exercise $Th_{v0}$ and postexercise elevation in $T_{es}$ that we have previously demonstrated for a range of ambient temperatures, exercise intensities, and preexercise resting temperatures (Kenny et al., 1996a; 1996b; 1997a) was evident even with significantly higher core temperatures (i.e., whole-body heat content) generated by the longer exercise bouts.

It is well documented that during exercise, the threshold for cutaneous vasodilation increases above preexercise resting values (Johnson and Park, 1981; Kellogg et al., 1991; Smolander et al., 1991; Taylor et al., 1988). The magnitude of the increase is dependent upon ambient temperature (Nadel et al., 1979), exercise intensity (Smolander et al., 1991; Taylor et al., 1988), and preexercise resting core temperature (Kenny et al., 1996a). Cutaneous vasodilation is significantly delayed or absent at intensities beyond 80% $VO_2$ max. Since all of our exercise conditions were conducted at the same ambient temperature, exercise intensity (70% $VO_2$ max), and preexercise resting core temperature, it was to be expected that the exercise $Th_{vd}$ would be similar for all three exercise conditions.

Following cessation of exercise, $T_{es}$ decreased to an elevated value that differed only by 0.01 and 0.02 °C between 15- and 30-min exercise and 30- and 45-min exercise conditions, respectively. This was the case despite a large difference in corresponding end-exercise $T_{es}$ of 0.49 and 0.44 °C. Similarly, there were no statistical differences between exercise $Th_{vd}$ and postexercise $T_{es}$ elevation within the three exercise conditions.

We have previously demonstrated that successive exercise/recovery cycles (15 min exercise followed by 30 min recovery) performed at progressively increasing preexercise esophageal temperature levels resulted in parallel increases of exercise $Th_{vd}$ and postexercise elevation in $T_{es}$ (Kenny et al., 1996a). End-exercise $T_{es}$ attained values ~1.1, 1.5, and 1.7 °C above baseline resting for the first, second, and third bouts, respectively. This was comparable to the ~0.9, 1.5, and 1.9 °C increase in end-exercise $T_{es}$ following the 15-, 30-, and 45-min exercises, respectively, in this study. Yet, unlike the parallel increase in exercise $Th_{vd}$ and postexercise $T_{es}$ measured during the successive exercise/recovery cycle, no comparable increase in either the exercise $Th_{vd}$ or postexercise $T_{es}$ were observed in this study. This supports our previous conclusions that postexercise elevation in $T_{es}$ is physiologically related to exercise $Th_{vd}$.

In addition, it would seem that the relationship between exercise $Th_{vd}$ and the postexercise $T_{es}$ response is defined by factors that modify the exercise threshold for cutaneous vasodilation such as exercise intensity (Smolander et al., 1991; Taylor et al., 1988), ambient temperature (Kenny et al., 1997a; Nadel et al., 1979), preexercise resting esophageal temperature (Kenny et al., 1996a), or a combination of these, and is not related to an increase in whole-body heat content as defined by a significant increase in the end-exercise core temperature.

The actual mechanism of the postexercise increase in $T_{es}$ remains unclear. It is possible that any number of factors such as those of neural, cardiovascular (ei-
ther central or peripheral), or humoral origin may be responsible. Recent evidence favors a baroreceptor-mediated influence because it is known that the cutaneous vasodilator system is under baroreceptor control (Kellogg et al., 1990). Acute bouts of exercise have been shown to cause postexercise hypotension (Coats et al., 1989), likely due to a decrease in baroreflex sensitivity (Somers et al., 1987). We have previously shown an elevated postexercise threshold for vasoconstriction (Kenny et al., 1998b) and vasodilation (Kenny et al., 1998a) that was consistent with our observations of a reduction of skin temperatures and skin blood flow back to baseline in the face of a sustained elevated $T_{es}$ (Thoden et al., 1994).

If a decrease in skin blood flow (vasoconstriction) helps maintain adequate cardiac filling pressure in response to the decrease in systemic vascular resistance, it is plausible that the increase in postexercise $T_{es}$ threshold for cutaneous vasodilation, and the resultant postexercise sustained elevation in $T_{es}$, may result from a strong non-thermoregulatory drive to maintain postexercise blood pressure. Our demonstration of a sustained elevated recovery heart rate for all three exercise conditions is consistent with a cardiovascular response aimed at maintaining cardiac output in the face of a sustained peripheral vasodilation of the previously active musculature.

In conclusion, the data show that increases in whole-body content produced by progressively longer exercise bouts (15, 30, and 45 min) did not affect the magnitude of postexercise $T_{es}$ elevation. In addition, the similarity between the exercise $T_{tho}$ and postexercise elevation in esophageal temperature reinforces our previous conclusions of a physiological relationship between exercise and postexercise thermal regulation. These data support our hypothesis that postexercise elevation in $T_{es}$ is not defined by the changes in whole-body heat content as produced by endogenous heating during exercise of different duration.

References


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