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Regular paper

Effects of exercise intensity on sweating and skin blood flow responses at the onset of dynamic exercise in mildly heated humans

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Abstract

To investigate quantitatively how sweating and cutaneous blood flow responses at the onset of dynamic exercise are affected by increasing exercise intensity in mildly heated humans, 18 healthy male subjects performed cycle exercise at 30%, 50%, and 70% maximal oxygen uptake for 60 s in a warm environment. The study was conducted in a climatic chamber with a regulated ambient temperature of 35°C and relative humidity of 50%. The subjects rested in the semi-supine position in the chamber for 60 min, and then sweating rate (SR) and skin blood flow were measured during cycle exercise at three different intensities. Changes in the heart rate, rating of perceived exertion, and mean arterial blood pressure were proportional with increasing exercise intensity, while the esophageal and mean skin temperatures were essentially constant throughout the experiment. The SR on the chest, forearm, and thigh, but not that on the palm, increased significantly with increasing exercise intensity ($P < 0.05$). The mean SR value of the chest, forearm, and thigh increased 0.05 $mg \cdot cm^{-2} \cdot min^{-1}$ with an increase in exercise intensity equivalent to 10% of the maximal oxygen uptake. On the other hand, the cutaneous vascular conductance (CVC) on the chest, forearm, and palm decreased significantly with increasing exercise intensity ($P < 0.05$). The mean CVC value of the chest and forearm decreased 5.5%, and the CVC on the palm decreased 8.0%, with an increase in exercise intensity equivalent to 10% of the maximal oxygen uptake. In addition, the reduction in CVC was greater on the palm than on the chest and forearm at all of the exercise intensities ($P < 0.01$). We conclude that non-thermal sweating and cutaneous blood flow responses are exercise intensity-dependent but directionally opposite at the onset of dynamic exercise in mildly heated humans. Furthermore, cutaneous blood flow responses to increased exercise intensity are greater in glabrous skin (palm) than in non-glabrous skin (chest and forearm).

Key words: thermal factors, non-thermal factors, glabrous skin, non-glabrous skin, feed-forward manner

Introduction

Humans have an excellent ability to regulate their internal and skin temperatures by sweating during dynamic exercise. The sweating response during dynamic exercise is controlled by changes in thermal factors, such as internal and skin temperatures (8, 12, 16, 22). However, the initiation of dynamic exercise (7, 19, 31) and brief isometric exercise (3, 17, 28) under warm environmental conditions can increase the sweating rate (SR) without marked changes in the thermal factors. This indicates that sweating responses are due mainly to alterations in non-thermal factors that are associated with mental stress (23, 36), central command (7, 19, 31, 34, 36), and stimulation of mechanosensitive (7, 19, 31) or metabosensitive (3, 18, 28) factors in exercising muscle. Also, it is suggested that the non-thermal sweating response during exercise is a feed-forward mechanism of thermoregulation, since this response precedes changes in the thermal factors (31, 36).

Van Beaumont and Bullard (31) showed for the first time that the SR on the forearm and calf increased without changes in the thermal factors at the onset of dynamic exercise, in situations where sweating was ongoing before the exercise. Furthermore, it has been reported that the SR changes concomitantly with a sinusoidal change in the workload, even when the internal temperature is constant (36). However, neither of these experiments evaluated in a quantitative manner how the non-thermal sweating response at the onset of dynamic exercise changed with increasing exercise intensity, i.e., no information was provided on the relationship between changes in the SR and exercise intensity.

On the other hand, during isometric handgrip exercise for 60 s, the non-thermal SR on the forearm correlated with changes in the relative exercise intensity under mildly hyperthermic conditions, i.e., there was a linear relationship between the SR and exercise intensity from 15% to 45% maximal voluntary contraction (MVC), and the relationship reached almost a plateau at around 45% and 60% MVC (17). In addition, the increases in SR

with raised exercise intensity during the isometric handgrip exercise were associated with changes in the number of active sweat glands (ASG), and not with the level of sweat output per gland (SGO) (20). However, it is not known whether the quantitative effects of exercise intensity on sweating response at the onset of dynamic exercise are similar to those during isometric exercise, since the physiological responses to dynamic and isometric exercise are different (21, 30, 33). It is also unclear how ASG and/or SGO are involved in intensity-dependent sweating responses at the onset of dynamic exercise.

In contrast to the sweating response, cutaneous vascular conductance (CVC), which is also associated with non-thermal factors, decreases at the beginning of dynamic exercise (6, 13, 30). Reflex cutaneous vasoconstriction occurs in the chest, forearm, foot, palm and finger (2, 6, 11, 13, 24, 30, 35), and vasoconstriction is greater in glabrous (hairless) skin than in non-glabrous (hairy) skin at a given exercise intensity (24). Furthermore, there is a relatively high correlation between the reduction in CVC on the forearm and increases in exercise intensity at the onset of dynamic exercise under normothermic conditions (30). However, the potential differences in the effects of exercise intensity on cutaneous vasoconstriction between non-glabrous skin and glabrous skin at the onset of dynamic exercise remain to be verified, since non-glabrous skin is regulated by an active vasodilator system and a noradrenergic vasoconstrictor system, whereas glabrous skin is controlled entirely by a noradrenergic vasoconstrictor system (5, 12).

The purpose of the present study was to investigate quantitatively the effects of changing exercise intensity (non-thermal factors) on sweating response, and to determine whether this quantitative sweating response was due to changes in ASG and/or SGO at the onset of dynamic exercise in humans who were mildly heated. Moreover, we determined the different effects of exercise intensity on cutaneous blood flow response at the onset of dynamic exercise in non-glabrous skin and glabrous skin.

Methods

Subjects

Eighteen healthy male subjects were recruited for this study. Their anthropometric data were: age 20.6 ± 1.1 yr, height 1.69 ± 0.01 m, weight 56.7 ± 1.1 kg, and maximal oxygen uptake ($\dot{V}O_{2max}$) 49.9 ± 2.4 ml·kg^{-1}·min^{-1}. The purpose and the procedures of the study were explained to the subjects before their informed consent was obtained. This study was approved by the Human Subjects Committee of our department at Kobe University.

Procedures

Before the experiments, the $\dot{V}O_{2max}$ of each participating subject was measured using a ramped exercise protocol (15~30 W·min^{-1}; pedaling frequency set at 60 rpm) on a cycle ergometer, which was modified so that the subjects sat in a contoured chair behind the pedals (semi-supine position). At least two of the following criteria were used to evaluate the $\dot{V}O_{2max}$ test: (*1*) maximal or near maximal values of rating of perceived exertion (RPE); (*2*) respiratory gas-exchange ratio greater than 1.05; (*3*) heart rate (HR) within 10 beats·min^{-1} of the age-predicted maximal heart rate; and (*4*) inability to maintain a pedaling frequency of 40 rpm.

The main experiments were conducted in an environmental chamber (SR-3000, Nagano Science Co. Ltd, Osaka, Japan) maintained at an ambient temperature of 35°C and a relative humidity of 50% with minimal air movement. We selected these environmental conditions to produce a slight increase in sudomotor activation and skin blood flow (SkBF) with withdrawal of sympathetic vasoconstrictor activation at rest by raising the skin temperature without markedly changing the internal temperature (17, 18). After entering the chamber, each subject rested in the semi-supine position on the modified cycle ergometer for approximately 60 min

until the SR and SkBF levels reached a steady state. During this period, the instruments for measuring various parameters were affixed. After this rest period, the baseline data were recorded for 5 min before the commencement of the cycle exercise. The subjects cycled (60 rpm) at three relative intensities of 30% (58 ± 4.8W), 50% (112 ± 7.2W), and 70% $\dot{V}O_{2max}$ (168 ± 9.6W) for 60 s in random order. We controlled the respiratory frequency at 30 times·min^{-1} during all of the 120 s rest, 60 s cycle exercise, and 120 s recovery periods using an auditory signal, because breathing frequency influences skin sympathetic nerve activity (4). The exercise bouts were separated by rest periods of at least 10 min to make the hemodynamic and thermoregulatory responses return to the baseline levels and to minimize muscle fatigue.

On a different day, the subjects performed the cycle exercise under the same conditions and procedures as the main experiment to measure oxygen uptake ($\dot{V}O_2$) during the exercise.

Measurements

$\dot{V}O_2$ was estimated from the measurements of ventilation volume and expired fraction of oxygen and carbon dioxide production (Aeromonitor AE-300S, Minato Co. Ltd, Osaka, Japan). In the main experiment, the esophageal temperature (Tes), local skin temperature at nine body sites (forehead, chest, abdomen, forearm, hand, palm, thigh, lower leg, and foot), SR on the chest, forearm, thigh, and palm, ASG on the forearm, SkBF on the chest, forearm, and palm, HR, arterial blood pressure (systolic and diastolic pressure), and RPE were measured.

The temperature variables were measured with a copper-constantan thermocouple. One thermocouple with a silicon-lubricated tip was placed at a distance of one-fourth of the standing height from the external nares. The mean skin temperature ($\bar{T}sk$) was estimated according to the method of Hardy and DuBois (9).

The SR on the chest, forearm, thigh, and palm were measured continuously using the ventilated capsule method (17, 22), in which dry nitrogen gas was supplied to four capsules (1.54 cm^2) at a flow rate of 600 $ml \cdot min^{-1}$, and the humidity and temperature of the nitrogen gas flowing out of each capsule were measured using a capacitance hygrometer (HMP 133Y, Vaisala, Helsinki, Finland). The time delay for measuring the SR was 1 s, which was taken into account when calculating the SR.

The number of ASG was measured at a site adjacent to the ventilated capsule on the forearm using the starch-iodine technique (16). The ASG and SGO were identified in 14 subjects during a 10 s window, and the SGO was calculated by dividing the SR by the number of ASG observed over the same period.

The SkBF on the chest, forearm, and palm were monitored continuously using laser-Doppler velocimetry (ALF21, Advance, Tokyo, Japan), which is specific for the skin and is not influenced by the underlying skeletal muscle blood flow (26). The probes for measuring SkBF were located within 1 cm of the ventilated capsules. The CVC on each site of the body was calculated from the ratio of the SkBF to the mean arterial blood pressure (MAP). Although the laser-Doppler flow signal does not provide an absolute mesurement of blood flow, it does provide a reliable index of relative changes in SkBF and CVC from the resting levels before the commencement of each exercise bout.

The various temperature, SR, and SkBF values were recorded every second and stored in a personal computer (PC9801RA, NEC Co. Ltd, Tokyo, Japan) using a data logger (HR2300, Yokogawa Co. Ltd, Tokyo, Japan). We also calculated the mean SR value from three sites of the body (chest, forearm, and thigh) and the mean CVC value from two sites of the body (chest and forearm), to evaluate the responses from non-glabrous regions.

The HR was measured continuously from the R-R interval of the electrocardiogram. The arterial blood pressure from the right radial artery was measured continuously in a

non-invasive manner using arterial tonometry (JENTOW-7700, Colin Co. Ltd, Aichi, Japan). The blood pressure was corrected for systolic and diastolic pressure by brachial auscultation, which was measured with an auto sphygmomanometer during the respective rest periods. The MAP was calculated as the diastolic pressure plus one-third of the pulse pressure. Each subject was asked to rate his perceived exertion on a scale from 6 to 20 (1) as an index of central command (34) at the end of each cycle exercise.

Statistical analysis

The data for the 60 s pre-exercise period and the final 30 s of the exercise period were collected. The changes in SR and CVC were calculated by subtracting the value of the 60 s pre-exercise period from the value of the final 30 s of the exercise period. The data are presented as means ± SEM. Two-way analysis of variance was performed using the Student's paired *t*-test when the *F* values were significant, to compare the data from all of the exercise intensities. Linear regression analysis was performed between SR or CVC and exercise intensity. The slopes of the regression lines were represented as the average of the slopes for each individual. Two-way analysis of variance was also performed to test for regional differences in sweating and cutaneous blood flow responses. A *P* value of less than 0.05 was considered to be statistically significant.

Results

Figure 1 illustrates the average responses of all subjects with respect to Tes, $\bar{T}$sk, HR, MAP, SR on the chest, forearm, thigh, and palm, and CVC on the chest, forearm, and palm during the cycle exercise at 50% $\dot{V}O_{2max}$. The physiological response patterns (time courses) of these variables during the exercise periods were similar, irrespective of the exercise intensity.

[Please insert Figure 1 here]

Tes and $\bar{T}$sk were essentially constant throughout the rest (Tes 36.88 ± 0.05°C and $\bar{T}$sk 35.33 ± 0.08°C) and exercise periods, and did not change significantly with increasing exercise intensity (Fig. 2). The HR increased abruptly just after the initiation of exercise and increased gradually thereafter. After a brief delay, the MAP increased throughout the exercise period. Moreover, the HR and MAP increased proportionally with increasing exercise intensity (Fig. 2; $P < 0.05$). The RPE and $\dot{V}O_2$ during exercise also increased significantly with increasing exercise intensity ($P < 0.05$).

[Please insert Figure 2 here]

The local skin temperature on the chest (0.06 ± 0.03°C) and forearm (0.07 ± 0.03°C) at 70% $\dot{V}O_{2max}$ increased significantly above the pre-exercise values. The skin temperatures on the chest, forearm, thigh, and palm did not change across the exercise intensities, with the exception of the forearm between 30% and 70% $\dot{V}O_{2max}$. The skin temperature was significantly higher on the forearm than on the chest and thigh ($P < 0.01$), and was higher on the chest than on the thigh ($P < 0.05$), at each exercise intensity. The skin temperature was the highest on the palm compared to all the other body sites.

The SR on the palm increased abruptly from the pre-exercise level just after the beginning of exercise, and subsequently reached a steady state at all of the exercise intensities, although the SR values were not significantly different for the various exercise intensities (Fig. 3). The SR on the chest, forearm, and thigh increased rapidly after a period of latency, and increased progressively after somewhat of a plateau during the each exercise period. The time

delays in onset of sweating on the chest and forearm were significantly shorter with increasing exercise intensity, with the exception of exercise that were conducted between 30% and 50% $\dot{V}O_{2max}$ ($P < 0.05$), although that on the thigh was not markedly different across the exercise intensities. The respective time delays for onset of sweating were: 16.4 ± 4.9, 9.6 ± 2.9, and 5.4 ± 1.9 s on the chest, 14.7 ± 4.2, 9.3 ± 2.7, and 5.4 ± 1.6 s on the forearm, and 8.4 ± 2.7, 7.6 ± 2.2, and 7.2 ± 2.1 s on the thigh at 30%, 50%, and 70% $\dot{V}O_{2max}$. The SR on the chest, forearm, and thigh were significantly higher than the pre-exercise values at all of the exercise intensities, and increased linearly with increasing exercise intensity (Fig. 3; $P < 0.05$, respectively). In addition, the slope of the regression line between the SR and relative exercise intensity was significantly steeper on the chest than on the forearm, thigh, and palm (Table 1; $P < 0.05$), and the SR was greater on the chest than on the forearm, thigh, and palm at 50% and 70% $\dot{V}O_{2max}$ ($P < 0.01$). The increase in the mean SR value of the chest, forearm, and thigh was 0.05 $mg \cdot cm^{-2} \cdot min^{-1}$ with an increase in exercise intensity equivalent to 10% $\dot{V}O_{2max}$. Furthermore, significant relationships were found between the SR and RPE on the chest ($r = 0.522$), forearm ($r = 0.570$), thigh ($r = 0.518$), and on the mean value of the chest, forearm, and thigh ($r = 0.571$, $P < 0.01$). The number of ASG on the forearm rose linearly with increasing exercise intensity, as did the SR on the forearm; a similar relationship was not noted for the SGO (Fig. 4; $P < 0.05$).

[Please insert Figure 3, Figure 4, and Table 1 here]

The CVC on the chest, forearm, and palm decreased just after the onset of exercise and reached a plateau during exercise period (Fig. 1). The early reduction in CVC was more pronounced on the palm than on the chest and forearm. The CVC on the chest, forearm, and palm were significantly different from the pre-exercise values at each exercise intensity (except

for the forearm at 30% $\dot{V}O_{2max}$), and decreased proportionally with increasing exercise intensity (Fig. 5; $P < 0.05$, respectively). The slope of the regression line between the CVC and relative exercise intensity tended to dip more sharply on the palm than on the mean value of chest and forearm ($P = 0.072$); the reduction in the mean CVC value of the chest and forearm was 5.5% while that on the palm was 8.0%, with an increase in exercise intensity equivalent to 10% $\dot{V}O_{2max}$ (Table 1). In addition, the slope of the regression line was significantly steeper on the palm than on the chest ($P < 0.05$). The reduction in CVC was significantly greater on the palm than on the chest and forearm at 30%, 50%, and 70% $\dot{V}O_{2max}$ ($P < 0.01$). Moreover, there was a significant correlation between the CVC on the palm and RPE ($r = 0.616$, $P < 0.01$)

[Please insert Figure 5 here]

Discussion

Three new observations of non-thermal sweating and cutaneous vascular responses at the onset of dynamic exercise in mildly heated humans emerge from this study: (*1*) significant increases occur in the SR from non-glabrous skin, but not from glabrous skin, with an increase in exercise intensity from 30% to 70% $\dot{V}O_{2max}$, in the absence of marked changes in Tes and $\bar{T}sk$ across these exercise intensities. The mean SR value of the three body sites (chest, forearm, and thigh) increased quantitatively 0.05 $mg \cdot cm^{-2} \cdot min^{-1}$ with an increase in exercise intensity equivalent to 10% $\dot{V}O_{2max}$; (*2*) the exercise intensity-dependent non-thermal sweating response is due to changes in ASG but not SGO; and (*3*) the intensity-dependent cutaneous blood flow responses were different for glabrous skin and non-glabrous skin at the onset of the exercise. The mean CVC value of the two body sites (chest and forearm) and the CVC on the palm decreased 5.5% and 8.0%, respectively, with an increase in exercise intensity equivalent

to 10% $\dot{V}O_{2max}$, and the slope of the regression line between the CVC and relative exercise intensity on the palm tended to be steeper than that on the mean value of chest and forearm. These results suggest that the exercise intensity-dependent responses (linear relationship) of non-thermal sweating and cutaneous blood flow show directionally opposite responses. Furthermore, the cutaneous vasoconstrictor responses were more pronounced in glabrous skin than in non-glabrous skin with increasing exercise intensity.

In this study, we used mildly heated conditions to induce slight increases in sweating rate and cutaneous blood flow without markedly increasing the internal temperature, and to clearly investigate non-thermal sweating and cutaneous blood flow responses at the onset of dynamic exercise, since it was reported previously that increases in SR and decreases in CVC by non-thermal factors during exercise were attenuated by sufficient hyperthermia (11, 15). On the other hand, the skin temperatures on the chest and forearm rose significantly by 0.06°C and 0.07°C, respectively, above the pre-exercise values at 70% $\dot{V}O_{2max}$, without marked changes in Tes and $\bar{T}$sk throughout the experiment (Fig. 2). Although local skin temperature influences sweating (22) and cutaneous blood flow (12) responses, the observed increases in skin temperature on the chest and forearm were too low to have any physiological impact on thermoregulatory reflexes. Thus, the environmental conditions selected in this study were appropriate for an investigation of non-thermal sweating and cutaneous blood flow responses at the onset of dynamic exercise.

It has been reported that an increase in muscle temperature, which activates thermoreceptors in deep veins and muscles, may facilitate the sweating response at the onset of dynamic exercise in warm environments (7). However, it is currently uncertain whether thermoreceptors actually exist in deep veins and muscles. Assuming that local thermoreceptors exist, doubts remain as to whether their activation would affect sweating and cutaneous blood flow responses (32), since changes in the SR and CVC showed opposite

responses in this study (Fig. 3 and 5). These results suggest that thermal factors, which include muscle temperature, may not importantly affect sweating and cutaneous blood flow responses in this study.

Although the SR on the forearm due to non-thermal factors reached a plateau at around 45% to 60% MVC during the isometric handgrip exercise (17), we observed a linear relationship between the non-thermal SR on the forearm and exercise intensity from 30% to 70% $\dot{V}O_{2max}$. Also, the SR on the palm during dynamic exercise, which reflects psychological effects, did not show a clearly graded response to increasing exercise intensity, while that on the palm during the handgrip exercise depended on exercise intensity (17). In addition, the RPE and MAP at 45% and 60% MVC were greater than those at 70% $\dot{V}O_{2max}$. Thus, the differences in exercise intensity-dependent responses of non-thermal sweating between dynamic and isometric exercise may be due to the degree of physical strain, even when relative exercise intensity levels are compared.

As shown by Figure 1, the SR on the palm increased rapidly just after the onset of dynamic exercise, but the SR on the chest, forearm, and thigh increased gradually at all of the exercise intensities. These patterns of sweating responses are consistent with those observed during isometric handgrip exercise (17). Furthermore, in this study, we found that the intensity-dependent response of non-thermal sweating was due to increases in ASG but not in SGO at the onset of dynamic exercise, as well as during the isometric handgrip exercise (20). The result suggests that the effect of non-thermal factors on changes in the SR from non-glabrous skin may induce the recruitment of ASG during both dynamic and isometric exercise routines.

It has been reported that the sweating response differs from one region of the body to another during dynamic exercise, and that the SR is greater on the chest than on the forearm and thigh (10, 22, 29). In this study, the slope of the regression line between the SR and relative

exercise intensity was significantly greater on the chest than on the forearm, thigh, and palm. Furthermore, the SR was significantly greater on the chest than on the forearm, thigh, and palm at 50% and 70% $\dot{V}O_{2max}$. The regional differences in sweating responses might be modulated by regional differences in ASG rather than differences in local skin temperature, since the skin temperatures on the chest, forearm, thigh, and palm did not differ with exercise intensity, and the change in SR on the forearm was due to ASG rather than SGO (Fig. 4). These results suggest that there are regional differences in non-thermal sweating responses at the onset of dynamic exercise, not only between non-glabrous and glabrous regions but also across non-glabrous regions with increasing exercise intensity.

We observed that the reduction in CVC was significantly greater on the palm than on the chest and forearm at all of the exercise intensities. Yamazaki et al. (35) noted that the amplitude of fluctuation in CVC during dynamic exercise, during which the workload was modified in a sinusoidal manner, was greater on the palm than on the forearm and dorsal hand, due to differences in the baseline CVC levels. However, in this study, the regional differences in cutaneous blood flow response were probably not due to differences in the pre-exercise CVC levels, since the changes in CVC were normalized to 100% of the resting levels before the commencement of exercise bout. It has been reported that the cutaneous vascular response in non-glabrous skin is regulated by both the noradrenergic vasoconstrictor and active vasodilator systems, whereas that in glabrous skin is regulated entirely by the noradrenergic vasoconstrictor system (5, 12). The decrease in CVC at the initiation of dynamic exercise is attributed entirely to enhanced active vasoconstrictor tone (13). On the other hand, it is possible that the active vasodilator system, which is activated mainly under hyperthermic conditions (14), was not activated under our experimental conditions, because we noted that the Tes did not increase markedly throughout the experiment. Therefore, the observed differences between non-glabrous and glabrous skin might be due to differences in sympathetic

innervation of these skin regions. It is also possible that the presence or absence of arteriovenous anastomoses (AVA) affects the cutaneous blood flow response, because the change in SkBF is much greater through AVA in the palm (12, 25, 35). Thus, the cutaneous blood flow response from glabrous skin is more sensitive to changes in exercise intensity at the onset of dynamic exercise than that from non-glabrous skin.

The exercise intensity-dependent responses of non-thermal sweating and cutaneous blood flow did not result from psychic excitement, because the SR on the palm did not change with increasing exercise intensity. In this study, the RPE may be an index of central command (34), which correlated significantly with the SR on the chest, forearm, and thigh and with the CVC on the palm. Therefore, we suggest that the SR from non-glabrous skin and the CVC from glabrous skin may be determined by the magnitude of the central command with increasing exercise intensity. However, the effects of central command on non-thermal SR might be weaker at the onset of dynamic exercise than during the isometric handgrip exercise, because the correlation coefficient between the SR on the forearm and RPE was smaller in this study ($r = 0.570$) than that reported ($r = 0.728$) in the previous study of Kondo et al. (17).

We cannot rule out the possibility that peripheral excitatory factors, such as the mechanoreflex (6, 7, 19, 31) and metaboreflex (3, 6, 18, 28) of contracting skeletal muscle, modulate the exercise intensity-dependent response of non-thermal sweating. In this study, the SR from non-glabrous skin increased rapidly following a period of latency, and increased progressively after leveling off in the latter half of the dynamic exercise period (Fig. 1). The sweating response just after the initiation of dynamic exercise may be modulated by the muscle mechanoreflex as well as by the central command, because the muscle metaboreceptors are probably not stimulated during the initial stages of mild or moderate dynamic exercise (33). On the other hand, the progressive increase in the SR during the latter half of the exercise period, which is greater with increasing exercise intensity, may support the involvement of

muscle metaboreflex.

In this study, the time delays of sweating onset on the chest and forearm were shorter with increasing exercise intensity, which is consistent with the results of the isometric handgrip exercise (17). The exercise intensity-dependent time delays may not be attributed entirely to the central command and the muscle mechanoreflex, because the time delays ranged from 5.4 s to 16.4 s, which represent slow responses to the effects of these two factors. Since it has been suggested that acetylcholinesterase is capable of modulating the SR when sudomotor activity is low, but is less effective when sudomotor activity is high (27), the time delay may also depend on the level of SR.

In conclusion, the sweating response from non-glabrous skin (chest, forearm, and thigh) increased linearly with increasing exercise intensity from 30% to 70% $\dot{V}O_{2max}$, whereas glabrous skin (palm) did not show a clearly graded response. The intensity-dependent response of non-thermal sweating is due to changes in ASG but not SGO. On the other hand, there was a negative correlation between CVC and relative exercise intensity, with the slope being steeper in glabrous skin (palm) than in non-glabrous skin (chest and forearm). These responses occur early during dynamic exercise, before there are any changes in body temperatures, indicating their non-thermal nature.

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Table 1. The slope of the regression line between sweating rate (SR) or cutaneous vascular conductance (CVC) and relative exercise intensity (30%, 50%, and 70% maximal oxygen uptake: $\dot{V}O_{2max}$).

	Slope of regression line	
Variables	SR ($mg \cdot cm^{-2} \cdot min^{-1} \cdot \%\dot{V}O_{2max}^{-1}$)	CVC ($\% \cdot \%\dot{V}O_{2max}^{-1}$)
Chest	0.007 ± 0.001	-0.43 ± 0.15
Forearm	0.004 ± 0.001 #	-0.59 ± 0.21
Thigh	0.004 ± 0.001 #	————
Palm	0.002 ± 0.001 #	-0.80 ± 0.16 #
Mean	0.005 ± 0.001	-0.55 ± 0.15

Values are means ± SEM of 18 subjects. Mean: the mean SR value of the chest, forearm, and thigh and the mean CVC value of the chest and forearm. # $P < 0.05$, compared to the chest.

Figure legends

Figure 1. Typical changes in esophageal temperature (Tes), mean skin temperature ($\bar{T}$sk), heart rate (HR), mean arterial blood pressure (MAP), sweating rate (SR) on the chest, forearm, thigh, and palm, and cutaneous vascular conductance (CVC) on the chest, forearm, and palm during cycle exercise at 50% maximal oxygen uptake ($\dot{V}O_{2max}$) for 60 s. Values are means ± SEM of 18 subjects.

Figure 2. Changes in esophageal temperature (Tes), mean skin temperature ($\bar{T}$sk), heart rate (HR), rating of perceived exertion (RPE), and mean arterial blood pressure (MAP) with increasing exercise intensity (30%, 50%, and 70% maximal oxygen uptake: $\dot{V}O_{2max}$). Values are means ± SEM of 18 subjects. # $P < 0.05$, compared with resting value. * $P < 0.05$, between exercise intensities.

Figure 3. Changes in sweating rate (SR) on the chest, forearm, thigh, palm, and mean value of the chest, forearm, and thigh with increasing exercise intensity (30%, 50%, and 70% maximal oxygen uptake: $\dot{V}O_{2max}$). Values are means ± SEM of 18 subjects. # $P < 0.05$, compared with resting value. * $P < 0.05$, between exercise intensities.

Figure 4. Changes in the number of active sweat glands (ASG) and sweat output per gland (SGO) on the forearm with increasing exercise intensity (30%, 50%, and 70% maximal oxygen uptake: $\dot{V}o_{2max}$). Values are means ± SEM of 14 subjects. * $P < 0.05$, between exercise intensities.

Figure 5. Changes in cutaneous vascular conductance (CVC) on the chest, forearm, palm, and mean value of the chest and forearm with increasing exercise intensity (30%, 50%, and 70% maximal oxygen uptake: $\dot{V}o_{2max}$). Values are means ± SEM of 18 subjects. # $P < 0.05$, compared with resting value. * $P < 0.05$, between exercise intensities.

Figure 1

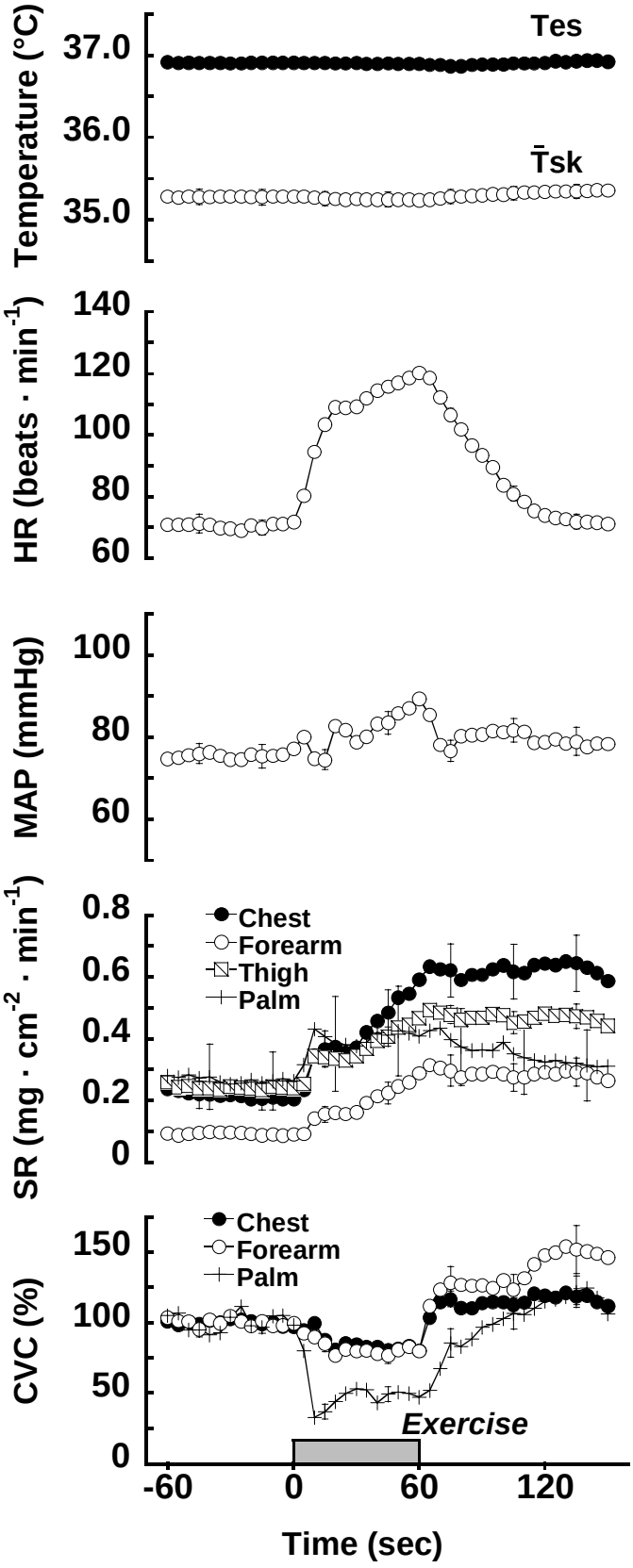

Tes
T̄sk
Temperature (°C)
37.0
36.0
35.0
HR (beats · min-1)
140
120
100
80
60
MAP (mmHg)
100
80
60
SR (mg · cm-2 · min-1)
0.8
0.6
0.4
0.2
0
Chest
Forearm
Thigh
Palm
CVC (%)
150
100
50
0
Chest
Forearm
Palm
Exercise
-60
0
60
120
Time (sec)

Figure 2

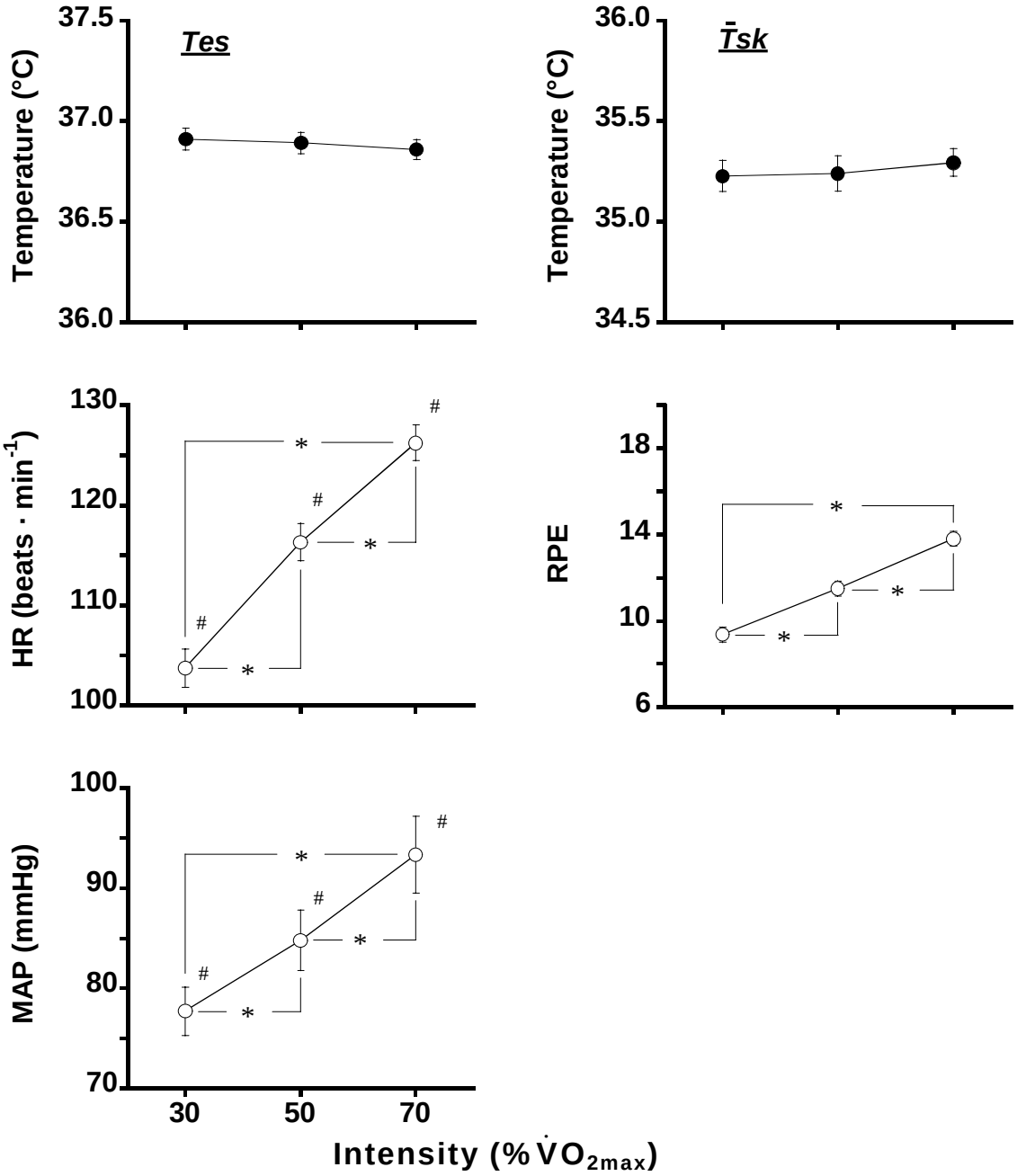

Tes
Temperature (°C)
37.5
37.0
36.5
36.0
T̄sk
Temperature (°C)
36.0
35.5
35.0
34.5
HR (beats · min-1)
130
120
110
100
RPE
18
14
10
6
MAP (mmHg)
100
90
80
70
30
50
70
Intensity (% V̇O2max)

Figure 3

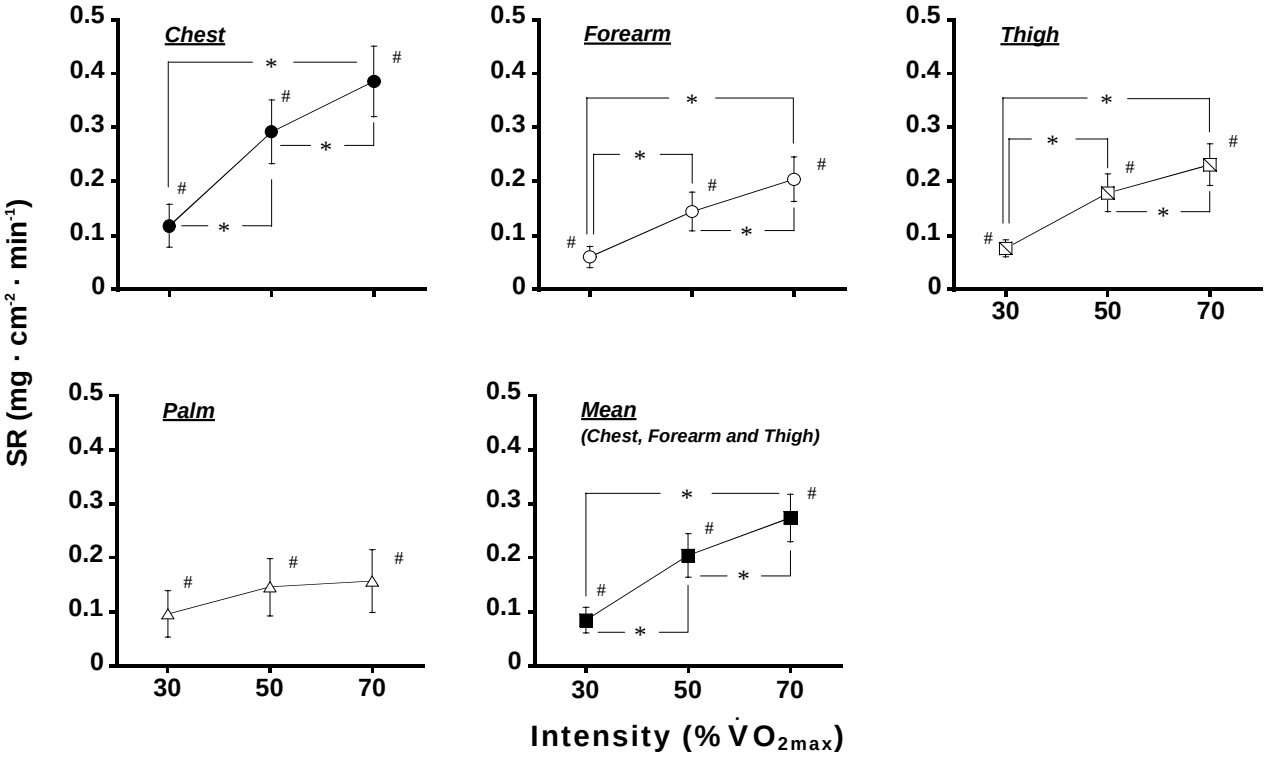

Chest
Forearm
Thigh
Palm
Mean
(Chest, Forearm and Thigh)
SR (mg · cm-2 · min-1)
Intensity (% V̇O2max)
0.5
0.4
0.3
0.2
0.1
0
30
50
70
*
#

Figure 4

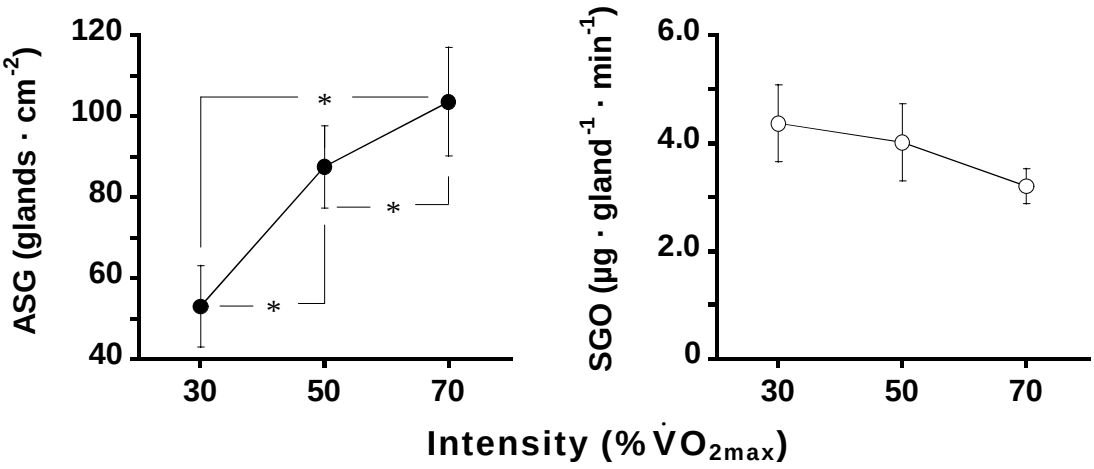
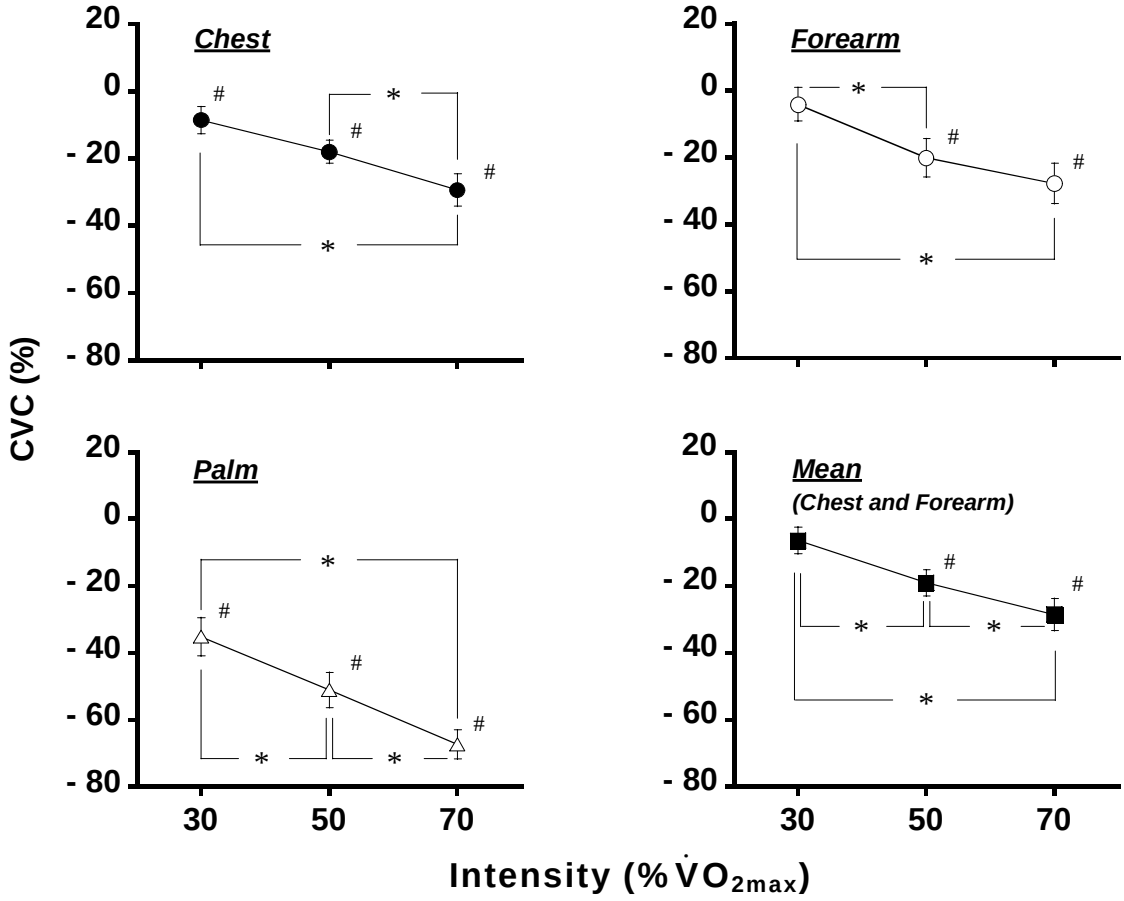

ASG (glands · cm-2)
120
100
80
60
40
*
*
*
SGO (µg · gland-1 · min-1)
6.0
4.0
2.0
0
30
50
70
30
50
70
Intensity (% V̇O2max)

Figure 5


Chest
Forearm
Palm
Mean
(Chest and Forearm)
CVC (%)
20
0
- 20
- 40
- 60
- 80
30
50
70
Intensity (% V̇O2max)