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Enhancement of finger blood flow response of postprandial human subjects to the increase in body temperature during exercise

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Summary. The present study was performed to investigate the effect of food intake on thermoregulatory vasodilatation in seven healthy male volunteers. The changes in oesophageal (T_{oes}) and mean skin temperatures, finger and forearm blood flows (BF), oxygen consumption ($\dot{V}O_2$) and heart rate (f_c) with and without food intake were measured before and during a 40-min exercise at an intensity of 35% maximal O_2 consumption at an ambient temperature of 25° C. Exercise commenced 60 min after food intake. Ingestion of food equivalent to 50.2 kJ·kg body mass^{-1} elevated mean body temperature, BF, $\dot{V}O_2$ and f_c in 60 min. Four subjects responded to exercise with a marked increase in finger BF and with no sweating (non-sweating group), while the other three responded with perspiration over almost the whole skin area and with little change in finger BF. Further analyses were made mainly in the non-sweating group. The postprandial increases in T_{oes}, BF, $\dot{V}O_2$ and f_c were persistent during exercise. The rate of increase in finger BF with the increase in T_{oes} and mean body temperature was significantly greater with food intake than without. However, there was no difference in the response of forearm BF to exercise between the two conditions. These results suggested that food intake enhanced finger BF response to the increase in deep body temperature during exercise. It was also concluded that there was a regional difference in cutaneous vasomotor response to thermal load in the postprandial subjects.

Key words: Dietary-induced thermogenesis - Skin blood flow - Temperature regulation - Heat loss

Introduction

Food ingestion produces various functional changes in metabolism (Welle et al. 1980, 1981; Segal et al. 1987), the autonomic nervous system (LeBlanc et al. 1984; LeBlanc and Brondel 1985; Steffens et al. 1986) and body temperature (Nielsen 1987). In preliminary experiments, we observed that cutaneous vasomotor tone, especially in the palm of the hand, decreased after ingestion of a single meal (Hirai et al. 1989). A similar observation was made by Nielsen (1987) in postprandial human subjects. Takano and Kotani (1989) reported that ingestion of a 700 kcal meal enhanced the cold-induced vasodilatation response of fingers to immersion in ice-cold water. These results seem to show that a small increase in the internal heat load due to the postprandial thermogenesis may affect the skin vascular control mechanisms.

Cutaneous blood flow (BF) increases in proportion to the increase in core temperature during exercise. This thermoregulatory response for dissipating heat accumulated in the body has been shown to be affected by several physiological and environmental factors, such as the state of hydration (Nadel 1980), diurnal (Stephenson et al. 1984) and menstrual (Hirata et al. 1986) cycles, the intensity of exercise (Hirata et al. 1983) and posture (Johnson et al. 1974). According to these reports, the response of cutaneous BF to the increase in core temperature can be altered by modulating the central adrenergic drive to skin vessels and/or by changing the peripheral vascular response to the command. However, little is known about the effect of food intake on the response of skin BF to the increase in body temperature during exercise. The primary objective of the present study was, therefore, to investigate this particular issue.

The skin of the palm of the hand and forearm shows a different anatomy in terms of the vascular structure. Abundant arteriovenous anastomoses exist in cutaneous tissue of the palm of the hand but few are found in the forearm (Sherman 1963). This characteristic vascular system in distal skin areas plays an important role in body temperature regulation, particularly in nonevaporative heat dissipation, by allowing a very high BF (and hence heat flow) through the vessels (Nelms 1963; Hales et al. 1978). Recently, Hirata et al. (1989a, b) have shown that the venous return from the hand

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greatly affects the increase in evaporative and nonevaporative heat losses from the forearm skin surface during an exercise-induced thermal load and suggested that hand BF via the arteriovenous anastomoses is relatively more important than forearm cutaneous BF in thermoregulation. Thus, we measured finger and forearm BF simultaneously to check the possibility of there being differences between the two skin regions in the vasomotor responses to postprandial exercise.

Methods

Subjects. Seven healthy males (Table 1) volunteered for the present experiment, giving informed consent. Prior to the test, maximal O_2 consumption ($\dot{V}O_{2max}$) was assessed at the point where there was no change: n O_2 consumption ($\dot{V}O_2$) despite an increasing exercise intensity, using a cycle ergometer (Monark-Crescent AB, Varburg, Sweden) in the upright position. Subsequently, the relationship between exercise intensity and $\dot{V}O_2$ was determined, with the subjects seated in a semi-supine posture in a reclining chair behind the pedals of the ergometer. The exercise intensity as a percentage of $\dot{V}O_{2max}$ was determined from these data for each subject. Prior to participating, they were also familiarized fully with the test procedures and with the equipment affixed to their bodies.

Measurements. Finger and forearm BF were measured every 15 and 30 s, respectively, by venous occlusion plethysmography using temperature-compensated mercury-in-Silastic strain gauges (Whitney 1953; Honda 1962). A strain gauge for measuring finger BF was wound around the middle finger of the right hand at a tension of 10 g and another for temperature compensation was attached to the index finger. A venous occlusion pneumatic cuff, 1.5 cm in width, was placed on the proximal part of the middle finger and inflated to 60 mmHg (8.0 kPa) for 5 s. A double-stranded circumference gauge for measuring forearm BF was placed around the middle of the left forearm. A second double-stranded gauge for temperature compensation was attached next to the active one. The circulation was excluded from the left hand with a pneumatic cuff (5.0 cm width) at the wrist inflated to 210 mmHg (28.0 kPa) for 3 of each 5 min. During this period, venous outflow was occluded by a cuff (12 cm width) at the upper arm inflated to 60 mmHg (8.0 kPa) for 15 s. All pneumatic cuffs were inflated by switch activation of solenoid valves connected to a pressurized air tank.

Oesophageal temperature (T_{oes}) at the level of the left atrium was measured with a thermistor probe introduced via the nose into the oesophagus. Skin temperatures were recorded at seven sites by thermistors held in place with surgical tape. The accuracy of the thermistors (Techno Seven., Yokohama) was within $\pm 0.1°C$. Heart rate (f_c) was obtained from ECG. The $\dot{V}O_2$ was determined from minute ventilation and the fractions of O_2 in the inspired and expired air. The minute ventilation was recorded with a 100-l twin type spirometer (CR-50, Fukuda Irika, Tokyo), expired air being collected through a face mask, and O_2 contents were measured by a polarograph type O_2-analyser (1H21A, SAN-EI Sokki, Tokyo), through which a small part of the expired air (100 ml·min^{-1}) was continuously pumped.

All data except for BF were sampled every 30 s via a computer-based logging system (PC9801VX, NEC, Tokyo). Mean skin ($\overline{T}_{sk}$) and mean body ($\overline{T}_b$) temperatures were computed using the following equations;

$$\overline{T}_{sk} = 0.07\,T_1 + 0.35\,T_2 + 0.14\,T_3 + 0.05\,T_4 + 0.19\,T_5 + 0.13\,T_6 + 0.07\,T_7,$$
$$\overline{T}_b = 0.7\,T_{oes} + 0.3\,\overline{T}_{sk},$$

where T_{1-7} are temperatures of the forehead, trunk, arm, hand, thigh, calf and foot, respectively (Hirata et al. 1986).

Procedure. All tests were conducted during the summer time in a climatic chamber (TBL-6-S, Tabai MFG Co. Ltd., Osaka) at an ambient temperature of $25.0 \pm 0.5°C$ and a relative humidity of $40\% \pm 3\%$. The subjects were instructed to arrive at the laboratory by 0800 hours after an overnight fast. They wore only shorts and rested for 60 min in a chair in an upright position before the start of the experiments.

In a preliminary experiment, the postprandial changes in T_{oes} were examined in five subjects. They were fed a commercial balanced food (carbohydrate 41.9%, protein 8.2% and lipid 49.9%; Calorie Mate, Otsuka Pharmaceutical Co., Ltd., Tokyo) over a 10 min period at an amount of 50.2 kJ·kg body mass^{-1} (12 kcal·kg body mass^{-1}) with approximately 400 ml of water, which had been kept at body temperature (37° C). The T_{oes} fell transiently in association with the feeding but was promptly restored to the pre-prandial level. Then T_{oes} started to increase at around 40–60 min after food ingestion and the high T_{oes} was maintained thereafter (Fig. 1). Such an increase in T_{oes} was not observed in the absence of food intake. In two subjects, tympanic temperature was simultaneously recorded using another thermistor probe. The postprandial changes of tympanic temperature were always in parallel with those in T_{oes}, except during and for about 10 min after food intake. We therefore decided that the exercise should start 60 min after the start of food intake and adopted T_{oes} as an indicator of core temperature.

Table 1. Physical characteristics of the subjects

Subjects	Age (years)	Height (cm)	Body mass (kg)	Maximal O_2 consumption (ml·min^{-1}·kg^{-1})
SS	25	170	55	53.5
TT	33	164	69	46.3
AH	37	169	65	35.3
MH	36	164	53	60.3
MT	25	173	69	60.7
MF	21	171	55	68.9
HA	20	172	54	76.7
Mean	28.1	169.0	60.0	57.4
SEM	2.7	1.4	2.8	5.2

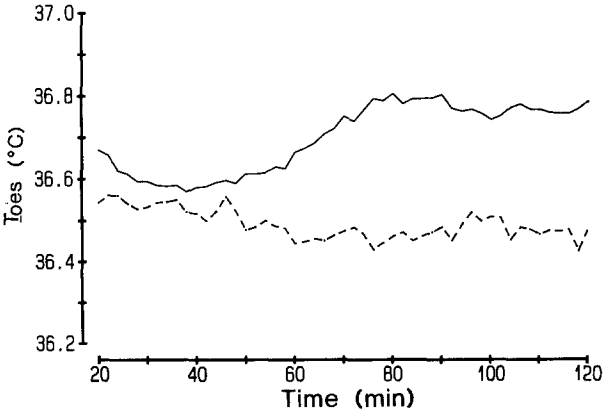

Fig. 1. A representative example of postprandial changes in oesophageal temperature (T_{oes}) (*solid line*) of one subject in non-sweating group. Time on the abscissa is the time after the start of food intake. *Broken line*, changes in T_{oes} without food intake in the same subject

After the food intake as described above, the subjects sat in a semisupine position in a reclining chair behind the pedals of a cycle ergometer. Then, within 10 min a face mask was donned and thermistors and electrodes for ECG recordings were attached to the subjects and measurements commenced. After a 30-min rest, they exercised for 40 min on the ergometer at an intensity of 35% $\dot{V}O_{2max}$. The same measurements were made without feeding. The tests were duplicated in five subjects both with and without food intake on a separate day in a randomized order.

Data analysis and statistics. The duplicated data were averaged in each condition and the mean values were taken as the final data for the subjects. Linear regression lines showing the relationships between finger and forearm BF and T_{oes} were calculated by the method of least squares. The data range used for the regression analysis was limited to the period between the start of the increase in T_{oes} and the point where T_{oes} reached a plateau. The statistical differences between the regression coefficients were assessed by F distribution. The statistical analysis of mean values with time were performed by a one-way ANOVA and the differences of mean values were evaluated by paired and unpaired Student's t-tests where appropriate. The level of significance was taken as $P<0.05$.

Results

Table 2 summarizes the mean values of all parameters measured at rest just before the start of exercise (corresponding to 50–60 min after the start of the meal) with and without food intake. The T_{oes} with food intake was slightly but not significantly higher than without. At 40–50 min after food ingestion, the changes of T_{oes} among the subjects were varied. However, $\overline{T}_b$ and $\overline{T}_{sk}$ were significantly increased by food ingestion, indicative of an accumulation of heat in the body. As previously established, postprandial $\dot{V}O_2$ and f_c were significantly greater than those without food intake. In agreement with our previous observation using thermography (Hirai et al. 1989), finger and forearm BF increased following food ingestion.

Figure 2 shows the mean changes in T_{oes}, $\dot{V}O_2$ and f_c before, during and after exercise with and without food intake. The 40-min exercise transiently lowered T_{oes}, defined as the initial decrease of core temperature, and significantly increased it thereafter. The changes and the amount of the increase in T_{oes} during exercise were not affected by the feeding condition. The $\dot{V}O_2$ and f_c increased sharply at the beginning of exercise and quickly decreased toward pre-exercise levels after the termination of pedalling. The postprandial increase in T_{oes}, $\dot{V}O_2$ and f_c appeared to persist even during and after exercise, making parallel upward shifts of their curves compared to those without food intake.

Table 2. Mean values of all parameters measured at rest with and without food intake

	T_{oes} (°C)	$\overline{T}_b$ (°C)	$\overline{T}_{sk}$ (°C)	$\dot{V}O_2$ (ml·min^{-1})	f_c (min^{-1})	Finger BF (ml·100 ml^{-1}·min^{-1})	Forearm BF (ml·100 ml^{-1}·min^{-1})
				With food intake			
Mean	36.76	35.38*	32.15*	0.192*	66*	25.23	1.39
SEM	0.09	0.05	0.19	0.013	4	4.07	0.11
				Without food intake			
Mean	36.73	35.26	31.85	0.146	56	22.44	1.14
SEM	0.08	0.05	0.23	0.014	3	4.25	0.13

Values of seven subjects obtained from averaged data for 10 min before the start of exercise. * Significantly different from the values without food intake.

T_{oes}, oesophageal temperature; $\overline{T}_b$, mean body temperature; $\overline{T}_{sk}$, mean skin temperature; $\dot{V}O_2$, oxygen consumption; f_c, heart rate; BF, blood flow

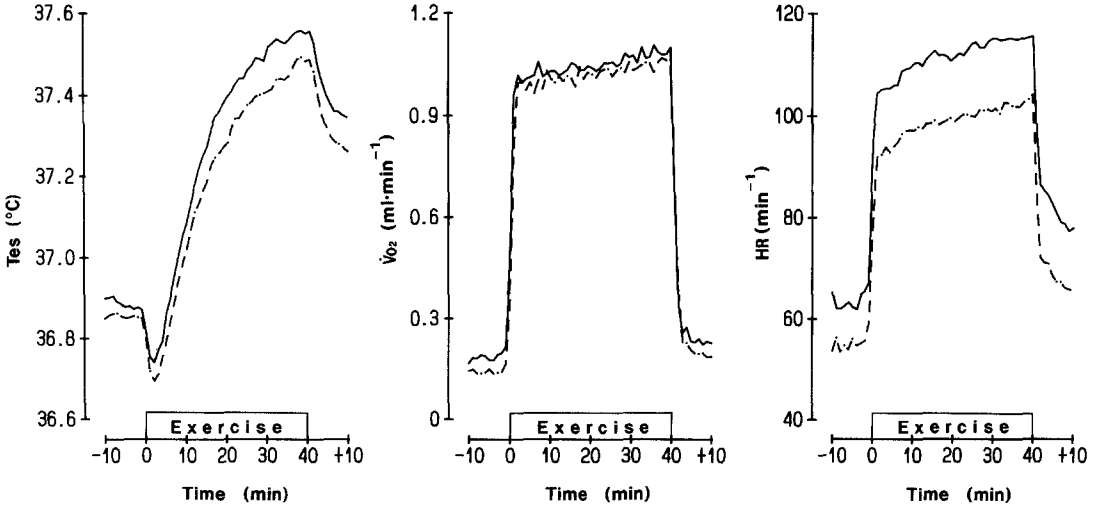

Fig. 2. Mean changes in T_{oes}, $\dot{V}O_2$ and f_c before, during and after exercise. Values are means obtained from averaged data over 2 min (SEMs are not shown to simplify the figure). *Solid line*, with food intake; *broken line*, without food intake; definitions as in Table 2

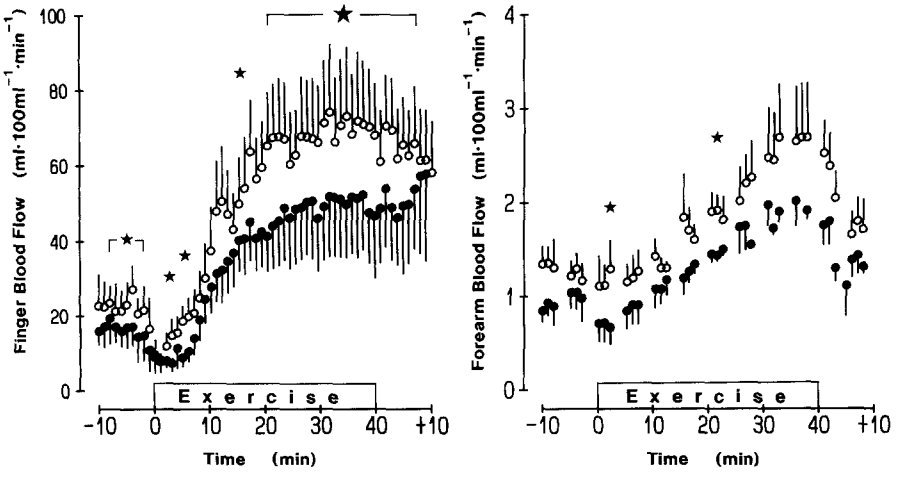

Fig. 3. Mean changes in finger and forearm blood flows before, during and after exercise in the non-sweating subjects. Values are means (with SEM) obtained from averaged data over 2 min. *Open circles*, values with food intake; *closed circles*, values without food intake. ⋆ Significantly different from values without food intake

Four subjects did not sweat during the 40-min exercise period with a large increase in finger BF (mean increase during exercise was $>29\ ml \cdot 100\ ml^{-1} \cdot min^{-1}$), while the other three perspired over almost the whole body skin area and showed little change in finger BF ($<10\ ml \cdot 100\ ml^{-1} \cdot min^{-1}$ on average). The different response of finger BF to exercise was consistent regardless of whether food was ingested or not. Thus we divided the seven volunteers into two groups, four non-sweating and three sweating subjects, and further analyses were made mainly in the non-sweating subjects.

Finger BF greatly increased during exercise in the non-sweating subjects (Fig. 3). Before the start of exercise, finger BF with food intake was slightly higher than that without, but the difference became distinct and significant during exercise, especially for the last 20 min. The amount of the increase in finger BF after 40-min exercise with food intake ($46.0\ ml \cdot 100\ ml^{-1} \cdot min^{-1}$, SEM 13.5) was significantly greater than that without ($29.1\ ml \cdot 100\ ml^{-1} \cdot min^{-1}$, SEM 12.5).

In contrast, in the sweating subjects, mean changes in finger BF during exercise were not statistically significant, although the degrees of exercise-induced increases in T_{oes} and $\dot{V}O_2$ were the same as in the non-sweating group. The 40-min exercise slightly but significantly elevated forearm BF in the non-sweating group both with and without food intake (Fig. 3). The level of forearm BF was consistently higher with food intake than without it before, during, and after exercise. In the sweating group, the increase of forearm BF tended to be greater than that of the non-sweating subjects, which may have depended on the occurrence of sweating in the forearm skin area.

The relationships between finger and forearm BF and T_{oes} during the 40-min exercise were assessed in the non-sweating group with and without food intake. Figure 4 illustrates mean changes of BF during exercise as a function of T_{oes}. The rate of increase in finger BF with the increase in T_{oes} was obviously greater after food intake, and the slope of the regression line showing the relationship between finger BF and T_{oes} with food intake ($81.1\ ml \cdot 100\ ml^{-1} \cdot min^{-1} \cdot °C^{-1}$) was significantly steeper than that without ($63.9\ ml \cdot 100\ ml^{-1} \cdot min^{-1} \cdot °C^{-1}$). When the regression analysis was made on the relationship between finger BF and $\overline{T}_b$, the result was similar. In addition, we computed linear regression lines for individuals. Since there were great interindividual variations of finger BF response to exercise, e.g. in subject SS, finger BF promptly and greatly increased after the onset of exercise but this was not the case in subject MH, the regression coefficients were somewhat varied. Regardless of the magnitude of finger BF re-

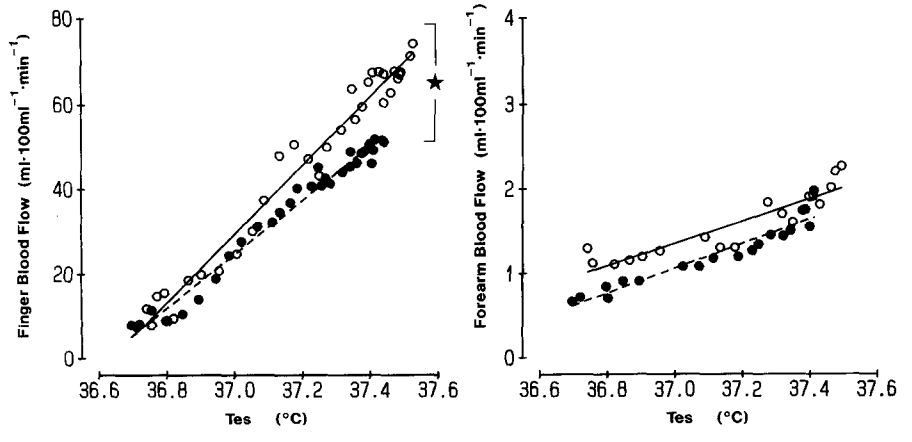

Fig. 4. Changes in finger (*left panel*) and forearm (*right panel*) blood flows (BF) as a function of oesophageal temperature (T_{oes}) during exercise in the non-sweating subjects. Values are means obtained from averaged data over 2 min. *Symbols* are the same as in Fig. 3. *Solid* and *broken lines* indicate linear regression lines showing the relationships between BF and T_{oes} with and without food intake, respectively. ⋆ Slopes significantly different

Table 3. Slopes of regression lines for individual showing the relationship between oesophageal temperature and finger blood flow with and without food intake

Subjects	With food intake ($ml \cdot 100\,ml^{-1} \cdot min^{-1} \cdot °C^{-1}$)	Without food intake ($ml \cdot 100\,ml^{-1} \cdot min^{-1} \cdot °C^{-1}$)
SS	147.5	145.4
TT	98.6	76.9
AH	57.6	31.8
MH	28.2	14.7
Mean	83.0	67.2
SEM	25.9	29.2

sponse, however, the slopes of the relationship between finger BF and T_{oes} were consistently steeper with food intake than without (Table 3).

The threshold T_{oes} for the initiation of the finger vasomotor response, which was determined by extrapolation to the value at which finger BF would have become 0, was not affected by food intake (36.60° C, SEM 0.16 and 36.48° C, SEM 0.15 with and without food intake, respectively). In contrast, there were no particular differences in the rate of increase in forearm BF with the increase in T_{oes} between conditions (1.3 and 1.5 $ml \cdot 100\,ml^{-1} \cdot min^{-1} \cdot °C^{-1}$ with and without food intake, respectively).

In the sweating group, finger BF did not increase during the exercise in this study. Moreover, the change in forearm BF may have been influenced by sudomotor activity and may not have reflected correctly the vasomotor drive from the central thermoregulatory centre. Therefore we did not perform regression analyses between BF and T_{oes} using these data.

Discussion

The present results clearly showed that the postprandial increase in T_{oes}, $\dot{V}O_2$ and f_c persisted even during exercise. These observations confirm earlier reports showing that the increases of $\dot{V}O_2$ and f_c after food intake and exercise were additive in human subjects (Welle 1984; Kelbæk et al. 1987; Segal et al. 1987; Nichols et al. 1988) and were comparable with a finding that, in rats, postprandial thermogenesis was constant, regardless of the intensity of locomotor activity (Nagasaka and Shido 1989). Similarly, the increase of $\overline{T}_{sk}$ and forearm BF with food intake appeared to be supplementary to those during exercise. However, the amount of the increase in finger BF after the 40-min exercise period was significantly greater in the postprandial condition than in the fasting state (Fig. 3).

The characteristic gain constants of finger and forearm BF were determined by plotting BF against body core temperatures as the central thermal drive to skin vasodilatation. As shown in Fig. 4, the slope of the relationship between finger BF and T_{oes} was steeper in the subjects who were fed. The observation was, of course, limited when the subjects responded to the exercise-induced hyperthermia with a great increase in finger BF (non-sweating group). The results indicate that the finger BF response to a thermal load was facilitated in the postprandial subjects and suggest the modification of finger vasomotor control mechanism by food intake. This was not the case in forearm BF. There may have been some differences between finger and the forearm in the vasodilator response to postprandial exercise, indicative of regional differences in skin BF control mechanisms.

There are rich arteriovenous anastomoses in certain regions of human skin, such as fingers, ear lobes, eye lids and lips (Sherman 1963). It is recognized that these special vascular structures play an important role in heat dissipation and the BF is controlled by central thermoregulatory drive mainly dependent on changes in deep body temperature (Hales et al. 1978). Furthermore, Hirata et al. (1989a) have suggested that, when there is no evaporative heat loss, the venous return through the hand arteriovenous anastomoses contributes markedly to heat loss from the forearm skin during exercise. According to their estimation, 89% of nonevaporative heat loss from the forearm depended on the hand BF at an ambient temperature of 20° C. In the present non-sweating subjects, the vasodilator response to exercise-induced heat load occurred predominantly in the finger and only a little in the forearm (Fig. 3). Moreover, the nonevaporative heat loss response appeared to be modulated by food intake only when the relationship between finger BF and T_{oes} was assessed. It would, therefore, seem to be that finger BF is more appropriate than forearm BF as a parameter for the gain or sensitivity of the nonevaporative heat loss mechanism to certain thermal stimuli.

With food intake, a small amount of heat accumulates in the body and, subsequently, core and $\overline{T}_{sk}$ increase slightly (Nielsen 1987; Hirai et al. 1989). Takano and Kotani (1989) have shown that cold-induced vasodilatation of the finger was enhanced in postprandial women subjects, probably through the activation of central thermoregulatory mechanisms which can detect a small increase in heat content of the body. In this present study, the magnitude of the heat load during exercise at a constant intensity in the fed subjects should have been greater than without food, which would have denoted a greater central thermoregulatory drive to the cutaneous vessels for increasing BF in the postprandial subjects. When the changes of finger BF were assessed as a function of $\overline{T}_b$, however, the response of finger BF with food intake to the increase in $\overline{T}_b$ was similarly greater. Since $\overline{T}_b$ is thought to reflect total thermal input from the body to the thermoregulatory centre, the enhanced finger BF response to exercise in the postprandial subjects may not be explained simply by the increased central thermoregulatory drive due to the large heat load in the body.

Local BF may be physically influenced by systemic blood pressure through the altering of the perfusion pressure in the region. There are several reports showing systemic haemodynamic changes following food intake. Kelbæk et al. (1987) reported that a substantial meal produced a marked increase in stroke volume, f_c

and, as a result, cardiac output, which was persistent during moderate exercise. In this case, however, mean arterial blood pressure measured both at rest and during exercise was not affected by food intake. Similar observations have been made by Jones et al. (1965) in human subjects and Fronek and Stahlgren (1968) in conscious dogs. Although we could not monitor blood pressure, these reports suggest there would be little contribution by a change in perfusion pressure to the great increase in finger BF during exercise following food intake.

Concomitantly with food ingestion or digestion, various humoral factors are secreted to control gastro-intestinal functions and the metabolism of energy materials (Steffens et al. 1986; Nielsen 1987). Several gastrointestinal hormones, such as cholecystokinin and bombesin, are known to exist in the central nervous system in high concentrations and have been thought to be potent modulators of food intake by inducing satiety in animals (Wood et al. 1981; Morley et al. 1985). The polypeptides have also been suggested as possible neurotransmitters or neuromodulators in temperature regulation in mammals (Shido et al. 1989). In human subjects, the concentration of plasma neurotensin was observed to increase fourfold 45 min after food intake (Mashford et al. 1978), and the same substance has been shown to produce hypothermia in rats in a thermoneutral environment through increasing nonevaporative heat loss (cutaneous BF) and depressing metabolic heat production (Shido and Nagasaka 1985). Thus a strong interaction may exist between temperature regulation and food intake or control systems in digestion. At present, we do not know the underlying mechanisms which modify the heat loss response after a single meal and, to clarify this particular issue, further intensive studies need to be made.

Nielsen (1987) has observed that rectal temperature had significantly increased after the first hour following the initiation of feeding of a 885-kcal meal. A similar result was obtained by Takano and Kotani (1989), who showed that T_{oes} increased 90 min after food intake but not after 30 min. In agreement with these two reports, we also observed that T_{oes} of the postprandial subjects [640–830 kcal (2675–3469 kJ)] a meal did not differ from that without food intake at 50 min after eating. However, it should be noted that $\dot{V}O_2$ and $\bar{T}_{sk}$ had already increased by that time (Table 2). It seems that a heat loss response can occur with little change in core temperature during internal heat loading due to food intake.

We divided the subjects into two groups according to the occurrence of sweating during exercise. Three subjects responsed to the exercise-induced increase in T_{oes} predominantly with perspiration, showing the activation of the evaporative heat loss mechanism, and the other four responded with large increases in finger BF without sweating. It is clear that the sweating response in the three subjects was not attributed to a water load or to other events associated with food intake, since the response was similar when fasting. Nadel et al. (1974) have shown that training by exercise in the heat lowers the threshold core temperature for the onset of sweating and enhances the sweating capacity during exercise. Our three sweating subjects were well-trained in the field and showed significantly higher $\dot{V}O_{2max}$ ($68.8\ ml \cdot min^{-1} \cdot kg^{-1}$, SEM 4.6) than did the non-sweating subjects ($48.9\ ml \cdot min^{-1} \cdot kg^{-1}$, SEM 5.3). Since the present experiment was performed during the summer time, the three subjects may also have been acclimatized to heat, which resulted in a more rapid onset of sweating during exercise. Although the three subjects perspired greatly, the changes in T_{oes} during the 40-min exercise did not differ from those in the non-sweating subjects with the large increase in cutaneous BF. It is, therefore, probable that the evaporative and nonevaporative heat loss mechanisms are complementary and the choice of the avenue of heat loss response during a moderate heat load depends on the condition of physical fitness or temperature acclimatization of the subjects.

In conclusion, the finger BF response to the increase in core temperature during exercise was facilitated in postprandial subjects when there was no perspiration. However, this was not the case, or was at least obscure in forearm BF, implying that there was a regional difference in cutaneous vasomotor response to moderate exercise at this ambient temperature.

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