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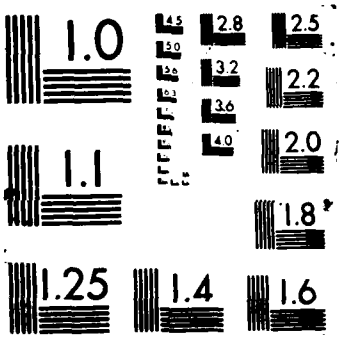
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Exercise Thermoregulation After Prolonged Wakefulness

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The effect of 33 hours wakefulness on the control of forearm cutaneous blood flow and forearm sweating during exercise was studied in three men and three women (follicular phase of the menstrual cycle). Subjects exercised for 30 min at 60% peak $\dot{V}O_2$ while seated behind a cycle ergometer (T_a = 35°C, T_{dp} = 10°C). We measured esophageal temperature (T_{es}), mean skin temperature (T_{sk}), and arm sweating ($\dot{m}_s$) continuously and forearm cutaneous blood flow (FBP) twice each minute by venous occlusion plethysmography. Resting T_{es} was 0.15°C ($P < 0.10$) lower after equilibration to the warm environment following the period of wakefulness. During steady-state exercise, T_{es} was unchanged by sleep loss. The sensitivity of FBF to T_{es} was depressed an average of 30% ($P < 0.05$) following 33 hours of wakefulness with a slight decrease (-0.15°C, $P < 0.05$) in the core temperature threshold for vasodilatory onset. Sleep loss did not alter the T_{es} at which the onset of sweating occurred; but sensitivity of arm sweating to T_{es} tended to be lower, but was not significant. Arm skin temperature was not different between control and sleep loss experiments. Reflex cutaneous vasodilation

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Key words: cutaneous blood flow, sleep loss, sweating, wakefulness

The progressive decline in the resting body (core) temperature associated with extended periods of wakefulness is well known (4,8.10,11). However, the extent to which this decrease in regulated body temperature affects exercise thermoregulation is not well documented. It is known that the circadian rhythm in the regulated core temperature is associated with a similar periodicity in the esophageal temperature threshold for initiation of eccrine sweating and cutaneous vasodilation during exercise (15,16,17).

It has also been suggested, in two widely disparate studies that these heat loss mechanisms may be affected by periods of wakefulness (30 or 50 hours) during exercise in temperate (14) or cold conditions (10). Sawka et al. (14) failed to observe a reduced resting body temperature but reported reduced sweating rate and reduced heat conductance during exercise after wakefulness. Their data showed that the chest conductance sensitivity was also reduced, yet the esophageal temperature threshold for chest conductance tended to be lower after sleep loss. Since heat conductance is an indirect measure of cutaneous blood flow, the seemingly contradictory effect of sleep loss on cutaneous vasodilation may be partially due to the technique used.

The impairment of the heat loss mechanisms during exercise which occurred after sleep loss in both a cold (10) and a temperate (14) environment should be clearly evident during exercise in a warm environment. In this investigation, forearm blood flow was directly measured in a warm environment (35°C) to determine the effect of 33 h of wakefulness on cutaneous vasodilation during exercise. Sweating responses were also measured to determine whether any alteration in the control of thermoregulatory heat loss mechanisms occurred following the expected decrease in resting core temperature after sleep loss.

METHODS

Six healthy adult subjects (Table I) volunteered for the previously approved protocol. The three female subjects were tested during days 3-6 of their menstrual cycle to control for the cyclical variability in heat loss responses. All subjects were familiarized with the experimental techniques before the study. Experiments were conducted during July and August when the subjects should have been naturally heat acclimated.

There were two experiments per subject and all experiments were conducted between 1400 and 1600 hours. Each subject was tested at the same time of day in the control and sleep loss experiments. The order of the control and sleep loss experiments was randomized. The period of sleep loss began at either 0500h for those subjects tested at 1400h or 0800h for those tested at 1500h, thereby requiring each subject to be awake for 33 consecutive hours before testing. Levels of activity and time and composition of meals were controlled during the period of wakefulness, with the last meal being ingested eight hours before the experiment. Subjects were allowed to drink water during this period of time.

The subjects reported to the laboratory dressed in shorts, singlet, shoes and socks. The climatic chamber was already heated to an ambient temperature (T_a) of 35°C, with an average ambient water vapor pressure (P_w) of 1.0 kPa. This environment was chosen both to facilitate evaporative heat loss, and to prevent mean skin temperature (T_{sk}) from varying greatly within an experiment. The central influence on control of the thermoregulatory effectors is best studied in an environment in which T_a approximates T_{sk}. The subject was

weighed, then rested in the chair behind a cycle ergometer, such that the legs of the subject were parallel to the floor. Each subject swallowed a catheter containing a thermocouple, then advanced it to a position at heart level the measurement of core (esophageal) temperature (T_{es}). The subject ingested 200 ml water during this process. Thermocouples were placed on the skin at eight sites (one site being the forearm, where blood flow and sweating rate were measured) and the measured skin temperatures were used to calculate T_{sk}

where T_{sk} = 0.07 (head) + 0.175 (chest) + 0.175 (back) + 0.07 (arm) + 0.07 (forearm) + 0.19 (thigh) +0.20 (calf) +0.05 (hand) (eqn. 1)

An automatic dew-point sensor enclosed in a ventilated capsule (5) was placed on the volar surface of the forearm to determine sweating rate as

$$\dot{m}_s = (AF)\ (\Delta P_w)/R_w \cdot A \cdot T \qquad \text{(eqn 2)}$$

where AF is the air flow through the capsule, ΔP_w is the vapor pressure gradient between ambient air and the capsule, A is the area of the capsule (13.2 cm^2), T is the absolute temperature of dew point, and R_w is the gas constant for water vapor ($0.4618 kPa \cdot \ell \cdot g^{-1} \cdot K^{-1}$)

A mercury-in-silastic strain gauge was placed on the contralateral forearm for measurement of forearm skin blood flow (FBF) by venous occlusion plethysmography (7,18). ECG electrodes were attached to the chest for measurement of heart rate (HR). After thermal equilibration at rest (constancy of core and skin temperatures) metabolic rate was estimated by standard procedures. Sweating rate and FBF were measured frequently after thermal equilibration during rest. The subject began to exercise at approximately 60% peak aerobic power after approximately 30 minutes of rest. FBF was measured every 30 seconds, and T_{es} and T_{sk} were continuously measured during exercise. Dew-point temperature was measured continuously within the

ventilated capsule (air flow = 900 $ml \cdot min^{-1}$) for sweating rate determination, HR was measured every 5 minutes, and metabolism was measured at both 10 and 25 minutes of exercise. Exercise was terminated after 30 min. Heat balance was calculated from partitional calorimetry during both rest and steady-state exercise. Respiratory heat loss was determined as:

$$E_{res} = 0.0023M\ (P_{es} - P_w),\ W \cdot m^{-2} \qquad \text{(eqn. 3)}$$

where: M is metabolic heat production in $W \cdot m^{-2}$ and $P_{es} - P_w$ is the water vapor gradient in kPa between the esophageal and ambient air temperatures. Dry heat loss from respiration was determined as:

$$C_{res} = 0.0014M\ (34 - T_a)\ ,\ W \cdot m^{-2} \qquad \text{(eqn. 4)}$$

The maximal evaporative capacity of the environment was calculated as:

$$E_{max} = h_e\ (P_{s,sk} - P_{s,dp}),\ W \cdot m^{-2} \qquad \text{(eqn. 5)}$$

where: h_e is the evaporative heat transfer coefficient ($W \cdot m^{-2} \cdot Torr^{-1}$) calculated as 16.5 ($h_c$) and $P_{s,sk}$ and $P_{s,dp}$ are the saturated vapor pressures at observed T_{sk} and T_{dp}, respectively.

Dry heat loss from the body was calculated as:

$$R+C = (h_c + hr)\ (T_{sk} - T_a),\ W \cdot m^{-2} \qquad \text{(eqn. 6)}$$

where h_c is the convective heat transfer coefficient ($W \cdot m^{-2} \cdot Torr^{-1}$), calculated as 8.3 v^{*5}, h_r is the radiative heat transfer coefficient which equalled 4.7 $W \cdot m^{-2} \cdot Torr^{-1}$

Mean body temperature was calculated as:

$$T_b = 0.89\ T_{es} + 0.11\ T_{sk},\ ^{o}C \qquad \text{(eqn. 7)}$$

Body heat storage was calculated as:

$$S = M_{sk} - h\ (T_{sk} - T_a) - wh_e\ (P_{s,sk} - P_w),\ W \cdot m^{-2} \qquad \text{(eqn. 8)}$$

where M_{sk} is the net heat flow calculated as $M_{sk} = M - work - C_{res} - E_{res}$ and h is the combined radiative and convective heat transfer coefficient, and w = skin wettedness.

Skin wettedness was calculated from:

$$w = E_{sk}/E_{max} \qquad \text{(eqn. 9)}$$

where E_{sk} is the evaporative heat loss from the skin, calculated from body weight changes.

The T_{es} thresholds for initiation of cutaneous vasodilation and sweating were calculated for each experiment by analyzing the exercise transient phase of the FBF to T_{es} and $\dot{m}_{sw}$ to T_{es} relationships. The exercise transient phase was defined as the time of exercise during which a rapid increase in T_{es}, sweating rate, and FBF were observed. A regression equation was calculated for each subject during the transient phase for both FBF to T_{es} and $\dot{m}_{sw}$ to T_{es}. At the beginning of exercise, there was a transient decrease in FBF even as T_{es} began to increase. These data, as well as the data collected after T_{es} reached a steady level, were not included in the calculation of the linear regression. The T_{es} threshold for initiation of sweating was calculated from the regression equation at $\dot{m}_{sw}$ = 0.06 $mg \cdot cm^{-2} \cdot min^{-1}$ (2). The T_{es} threshold for cutaneous vasodilation was calculated from the regression equation at the resting control FBF for each subject. All data were analyzed by a one-way ANOVA with repeated measures. All differences reported in the RESULTS are significant at $p<0.05$.

RESULTS

When the subject first reported to the laboratory, T_{es} was measured in a 25°C environment, this T_{es} averaged 0.3°C lower ($p<0.05$) after wakefulness. Likewise, after equilibrating in the 35°C environment, the average T_{es} was still lower by 0.15°C ($p<0.10$) at 33 hours of wakefulness. All other variables were unchanged at rest.

Steady-state exercise partitional calorimetry data are presented in Table 2 for the six subjects. Dry heat exchange was minimized by design. There were no significant differences in T_{es}, T_{sk}, $\dot{m}_s$, whole body sweating (E_{sk}) from weight loss or metabolism between control and sleep loss experiments during exercise. The local sweating (Table 3) and forearm blood flow responses (Table 4) to changing T_{es} are given as the slope and threshold of each relationship for each subject. Figure 1 graphically demonstrates these thermoregulatory responses in a representative subject. No significant alteration in the central control of sweating as determined from the T_{es} threshold for sweating onset occurred following the period of wakefulness. However, five of the six subjects had a lower slope, but subject 1 showed a higher slope after sleep loss so no significant difference was apparent. The onset of cutaneous vasodilation was at a lower T_{es} (0.15°C, $p<0.05$) and the slope was suppressed an average of 30% in the sleep loss experiments. The mean rate of change in T_{es} during the exercise transient was identical in control and sleep loss experiments and averaged $0.11°C \cdot min^{-1}$.

DISCUSSION

A general trend of reduced thermoregulatory response to an exercise-heat stress is apparent following a 33-hour period of sleep loss. The regulation of reflex vasodilation was suppressed, as the slope was decreased (30%) following sleep loss. Paradoxically, the onset of vasodilation occurred at a lower esophageal temperature. These responses are the same as those seen by Sawka et al. (14) using an indirect measure of vasomotor activity. Are these changes indicative of both central (threshold) and peripheral (slope) alterations in the control of heat loss through vasomotor pathways and why do these responses appear to counteract one another? That is, a lowered

temperature at which reflex vasodilation occurs is tied to an attenuated response to increasing esophageal temperature.

We did not find a significant depression in the control of thermoregulatory sweating to changing esophageal temperature in the present study. However, examination of the individual data (Table 3) for esophageal temperature threshold and slope of $\dot{m}_s$ to T_{es} shows that in four of our six subjects the sensitivity of arm sweating in the sleep loss experiments was decreased, and a fifth subject showed no change. The average decrease in $\dot{m}_s$ to T_{es} for these four subjects was 29% which is similar to the suppression in FBF. The responses of subject #1 were distinctly different from the other five subjects, although the experiments were identical, and he did not appear to have unique physiological qualities (Table 1). In subject 4, sleep loss did not affect the sensitivity of $\dot{m}_s$ to T_{es}. However, he was routinely spending one day a month without sleep. We feel the responses of these two subjects are correct, however, our data show a trend for an attenuation of $\dot{m}_s$ to T_{es} after sleep loss, which is in agreement with an earlier study.

This general suppression in the local (arm) sweating response is of the same magnitude found by Sawka and colleagues (14), but they used a lower ambient temperature (28°C). However, the reduction in whole body sweating seen in that earlier study (-27%) was not found in the present study (-7%). Perhaps the "priming" of the sweat glands at rest via the increased skin temperature observed at 35°C masked the effect evident at 28°C. That is, when the skin is at a comfortable temperature, the sweating response to a given increase in core temperature should be much less than the sweating response when the skin is warm (3,13). The enhanced signal from skin thermoreceptors in this study may have overridden the sleep deprivation effect of suppressing whole body sweating rate during exercise.

In studies of the circadian variability in heat loss responses, Wenger (17) and later Stephenson (15) demonstrated that esophageal temperature t: ..olds for both vasodilation and sweating tracked the circadian rhythm in body temperature; that is, when core temperature is at its zenith, the onset of the thermoregulatory effectors is at a higher core temperature, and when core temperature is at its lowest, so is the core temperature threshold for the thermoregulatory effectors. Neither of these studies indicated any change in the sensitivity of core temperature to thermoregulatory response. In the present study, the lowered resting core temperature after sleep loss is associated with a lowered or leftward shift in the T_{es} threshold for reflex vasodilation. However, in contrast to those studies of the circadian rhythm on heat loss responses, our data show a clear suppression in the sensitivity of the vasodilatory response to increasing core temperature. It has been documented that the T_{es} threshold for thermoregulatory responses varies substantially over a 24-hour period (15) and the lowering of the threshold temperature for cutaneous vasodilation in the present study is approximately equal to a four-hour difference in the circadian cycle. It is important to note that each pair of experiments on a single subject was conducted at the same time of day in the present study. Could this lowered core temperature be an indication that our subjects had actually experienced a circadian phase advance (12) such that their core temperatures were shifted to a lower temperature than in the afternoon of a control day? A more detailed study would be necessary to address this issue. However, as both thermoregulatory responses did not change in a parallel manner, a threshold change in effector response may not be an adequate explanation.

The general suppression in vasodilation and sweating to changing esophageal temperature seen in our study and the earlier study (14) in terms

of classic thermoregulatory control theory (6) would suggest peripheral alteration in these responses, such as a decrease in neurotransmitter released, a change in sensitivity to the neurotransmitter, or perhaps a decrease in the blood flow to the individual sweat glands (3). However, as Sawka (14) has suggested, perhaps the sudomotor or vasomotor signal from the controller has been altered by the sleep loss period. In that study, the pattern of outflow from the sweat glands was altered in three of their five subjects.

We attempted to minimize the peripheral influence on the control of the thermoregulatory effectors by minimizing changes in T_{sk} during an experiment as $T_{sk} \sim T_a$. However, a peripheral effect was seen as the increase in both FBF and $\dot{m}_s$ to rising T_{es} was attenuated after sleep loss. Additionally, the control of sweating is primarily related to maintenance of body temperature, while skin blood flow is controlled by reflexes involved in arterial pressure regulation, as well as temperature regulation (6). Therefore, changes in the control of cutaneous blood flow associated with sleep loss may not be solely of thermoregulatory origin.

The lower T_{es} threshold for cutaneous vasodilation in sleep deprived subjects may in part explain the lower resting T_{es}, seen upon arrival at the laboratory. This lower resting T_{es} and lower threshold for vasodilation could account for the unchanged steady-state T_{es} even though the slopes of $\dot{m}_s$ to T_{es} and FBF to T_{es} were depressed. Perhaps with an increased heat production, caused by a more intense workload, there would be a higher steady-state T_{es} as a result of the depressed sensitivity of the thermoregulatory responses to T_{es}.

In the present study, vasomotor and sudomotor responses to exercise in a warm environment (35°C) were directly measured after 33 hours of wakefulness.

The new observations made were: 1) a lower T_{es} threshold for cutaneous vasodilation; 2) decreased sensitivity of FBF to T_{es} and a trend for decreased $\dot{m}_s$ to T_{es}; and 3) warm skin may attenuate the suppression of whole body sweating rate, which Sawka *et al.* (14) observed in sleep deprived men exercising in a temperate environment (28°C). These results suggest that there is an attenuation of both vasomotor and sudomotor responses to increasing thermal drive after sleep loss and are consistent with and expand previous research (14). The lowered T_{es} threshold for onset of cutaneous vasodilation may partially explain the decreased core temperature after sleep deprivation.

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FIGURE LEGEND

Figure 1. Exercise transient responses for FBF to T_{es} (upper panel) and $\dot{s}_s$ to [illegible] lower panel) for a single subject during control and sleep loss e[illegible]iments.

Table 1. Subject Characteristics

Subject	Sex	$\dot{V}O_2$ peak ($\ell \cdot min^{-1}$)	Age (yr)	Ht (cm)	Wt (kg)	SA (m^2)
1	M	4.44	22	177	82.8	1.99
2	M	4.69	22	180	86.6	2.05
3	F	2.49	21	160	52.0	1.66
4	M	4.09	29	168	74.1	1.82
5	F	2.52	22	159	58.5	1.59
6	F	3.83	27	173	77.5	1.91
X		3.65	23.8	169.5	71.9	1.84
s		1.06	3.3	8.7	13.8	0.18

Table 2. Partitional Calorimetry During Steady-State Exercise in Control and Sleep Loss Environments.

Control

	T_{es} (°C)	T_{sk} (°C)	M ($W \cdot m^{-2}$)	R+C ($W \cdot m^{-2}$)	E_{sk} ($W \cdot m^{-2}$)	E_{res} ($W \cdot m^{-2}$)	C_{res} ($W \cdot m^{-2}$)	S ($W \cdot m^{-2}$)
1	38.05	34.79	432	-8	432	36	-1*	74+
2	37.71	35.66	430	-1	375	35	-1	65
3	37.56	35.20	283	-7	383	22	-1	31
4	37.71	35.23	433	1	444	34	-1	81
5	38.04	35.65	315	5	288	28	-1	54
6	38.26	35.39	360	2	301	31	-1	77
X	37.89	35.32	376	-1	371	31		64
S	(0.27)	(0.33)	(66)	(5)	(65)	(5)		(19)

Sleep Loss

1	38.00	34.78	417	-3	351	35	-1	79
2	37.28	35.34	406	-2	373	32	-1	50
3	37.51	35.45	320	-6	257	25	-1	34
4	37.74	34.83	440	-4	488	34	-1	75
5	38.08	36.12	320	4	288	26	-1	66
6	38.31	35.48	370	-2	301	30	-1	87
X	37.82	35.33	379	-2	343	30		65
s	(0.38)	(0.49)	(51)	(3)	(83)	(4)		(20)

*Rounded to nearest whole number.
+S (storage) resulting from exercise transient. By definition S=0 during thermal steady-state.
The numbers 1 through 6 represent the individual subjects.

Table 3. Slopes and Esophageal Temperature Thresholds for Local (Arm) Sweating in Control and Sleep Loss Experiments.

	Control		Sleep Loss	
	Slope	T_{es} Threshold	Slope	T_{es} Threshold
1	1.49	36.44	3.02	36.91
2	2.10	36.64	1.18	36.70
3	2.61	36.72	2.22	36.75
4	1.35	36.30	1.35	36.46
5	1.07	36.78	0.46	36.99
6	1.42	36.91	1.20	36.68
X	1.67	36.63	1.57	36.75
s	(0.57)	(0.23)	(0.90)	(0.19)

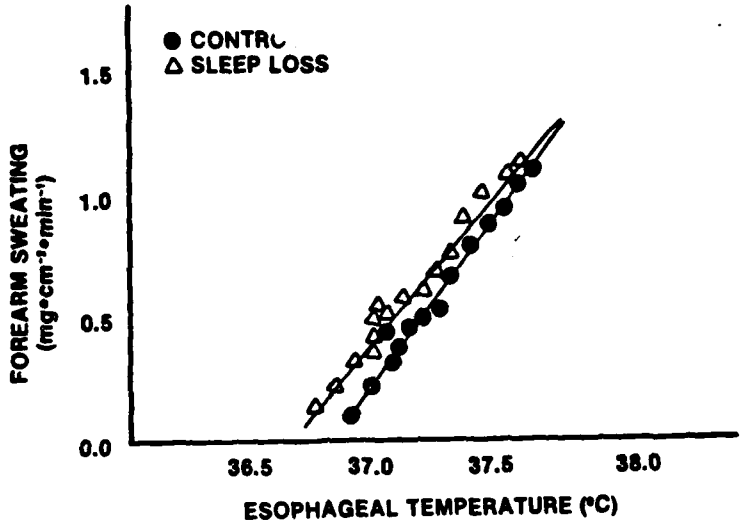
The numbers 1 through 6 represent the individual subjects.

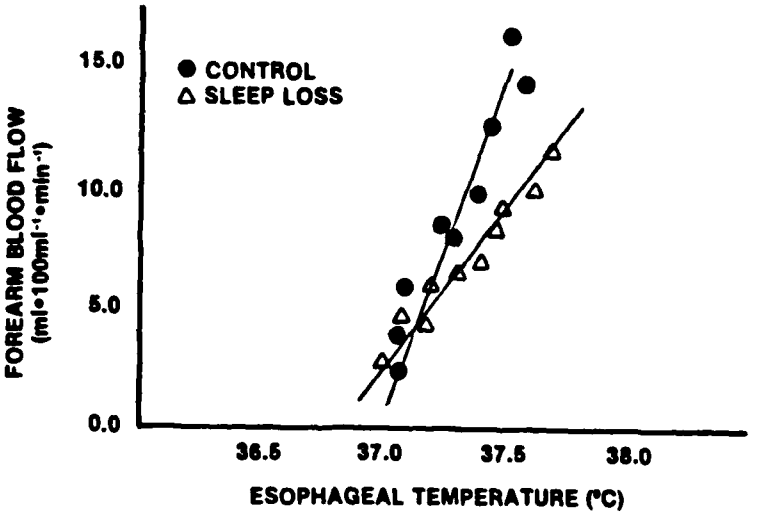
Table 4. Slopes and Esophageal Temperature Thresholds for Cutaneous (Arm) Blood Flow in Control and Sleep Loss Experiments

	Control		Sleep Loss	
	Slope	Threshold	Slope	Threshold
1	25.14	37.00	20.63	36.86
2	33.54	36.89	23.93	36.56
3	32.81	36.99	13.39	36.74
4	22.50	36.75	17.46	36.67
5	23.50	37.47	17.89	37.42
6	29.55	37.13	17.32	37.14
X	27.84	37.04	18.44*	36.90*
s	(4.79)	(0.25)	(3.55)	(0.32)

The numbers 1 through 6 represent the individual subjects.

*Different from control experiments $p<0.05$


● CONTRO
△ SLEEP LOSS
1.5
1.0
0.5
0.0
FOREARM SWEATING
(mg•cm⁻²•min⁻¹)
36.5
37.0
37.5
38.0
ESOPHAGEAL TEMPERATURE (°C)


● CONTROL
△ SLEEP LOSS
15.0
10.0
5.0
0.0
FOREARM BLOOD FLOW
(ml•100ml⁻¹•min⁻¹)
36.5
37.0
37.5
38.0
ESOPHAGEAL TEMPERATURE (°C)

END

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