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ELSEVIER ERGONOMICS BOOK SERIES

VOLUME 3

Environmental Ergonomics

The Ergonomics of Human Comfort, Health and Performance in the Thermal Environment

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PREFACE

Environmental Ergonomics addresses the problems of maintaining human comfort, activity and health in stressful environments. Its subject areas include thermal environments, illumination, noise and hypo- and hyperbaric environments. Participants at the International Conference on Environmental Ergonomics (ICEE) include research scientists, medical doctors, engineers, administrators, technicians, health care professionals and students from universities, private industry, and governmental research facilities in over 20 countries. The ICEE is currently the world's most distinguished conference in its field.

Since 1982, the ICEE has been held biennially in Europe and North America. We believe the 10th conference in Fukuoka, our first meeting in Asia, provided a stimulus for progress in Environmental Ergonomics in Asian countries.

This book contains papers presented at the 10th International Conference on Environmental Ergonomics held in Fukuoka, Japan, from September 23rd to 27th 2002. There were many excellent papers outside the topic of the thermal environment at the ICEE2002. However, given that the major topics were related to the thermal aspect, we have devoted this book in the Elsevier Ergonomics Book Series to this topic. The thermal environment is one of the major factors which has affected human comfort, health and performance from the age of cave-dwellings to our age of skyscrapers.

We would like to take this opportunity to thank the Organizing Committee and International Program Committee members of ICEE2002 who reviewed the papers. We also thank Shizuka Umezaki, Nobuko Hashiguchi, and Takako Fukazawa who helped with numerous aspects of this publication. In addition, we are indebted to the Elsevier staff members who provided editorial assistance to support this publication successfully.

Fukuoka
October, 2004

Yutaka Tochihara
Tadakatsu Ohnaka

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Section 1 – Physiology

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Regulatory processes of the human body during thermal and work strain

Jürgen Werner*

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Abstract: This chapter focuses on the control processes inherent in the human body when subjected to thermal or exercise stress. The regulatory processes commonly named 'thermoregulation' comprise the interaction, and sometimes the competition, of various control systems, such as the cardiovascular, metabolic, respiratory, osmoregulatory and thermal control systems. Understanding these processes correctly is essential for the estimation and evaluation of physiological strain in environmental ergonomics. Such systems stabilize body temperature in spite of external or internal loads, generally by means of an information loop with negative feedback. A controller network in the central nervous system activates effector mechanisms (such as metabolic heat production, sweat production and vasomotoric action) to an extent that is proportional to the deviation of the controlled variable from its so-called set-point. In hyper- or hypothermia, body temperature deviates substantially from the set-point, mainly because of insufficient effector capacity. The set-point may change periodically (e.g. circadian rhythm) or temporarily, due to interference with the regulation of non-thermal variables (e.g. in states of dehydration or starvation, etc.), or due to pathological, non-thermal influences (e.g. during a fever). The processes of acclimatization may also change the set-point.

Keywords: Thermoregulation, Exercise, Set-point, Control, Feedback

1. The system of temperature regulation

The young man in Fig. 1 is under both thermal and work strain. He is being subjected to the influences of air temperature, air humidity, solar radiation, atmospheric thermal radiation, reflected solar radiation, ground thermal radiation, and also to his running speed and the wind. Important factors affecting the thermal processes are clothing and posture. The processes in the human body, counteracting these stress factors, are evaporation of sweat, respiratory evaporation, conduction, convection via the blood, radiation and metabolic storage. Almost all physiological systems of the body are involved.

In spite of heavy challenges from the environment, the temperature of the human body is kept fairly constant. This is due to a complex control loop, which is presented in schematic form in Fig. 2.

The basic thermoregulatory control loop is composed of two subsystems The first is the 'controlled' system, that is, the human body as a heat transfer system which has to be actively controlled by the second 'controlling' system which consists of various components: thermosensors which are heterogeneously distributed over the body; ascending and descending central information processing; and spatially distributed effector mechanisms which change the net heat gain/loss ratio

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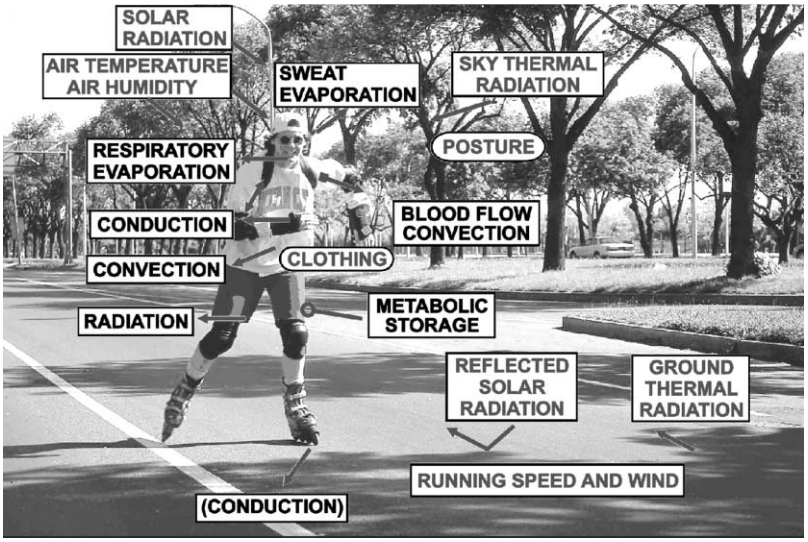

Fig. 1. Environmental factors and responses of the human body.

(for a review, see Refs. (1,2)). The effectors are vasomotor activity, metabolic heat production, sweat production with subsequent evaporation, and behavioral mechanisms (i.e. work and clothing). This system represents a control loop with negative feedback which can compensate for environmental influences like temperature, humidity and air velocity, or cope with the additional heat produced by muscular work.

2. Mechanism for attaining steady states under thermal load

The mechanism for attaining a steady state in the thermoregulatory control loop is explained below, as is the reason why a higher thermal load, whether evoked by environmental conditions, work or clothing, must result in a higher deviation of body temperature from the set-point in spite of negative feedback control. For the moment, let us assume that there is no feedback and thus consider the properties of the two subsystems, controlled and controlling, as open loop systems. The input to the controlled system is net heat gain, HG, and the output is a change in body temperature, T_b, as outlined in Fig. 3A, where we consider only deviations, Δ, from the thermoneutral state, i.e. the state where the effector activities counteracting alterations of body temperature are minimal, and where body temperature is at its so-called set-point.

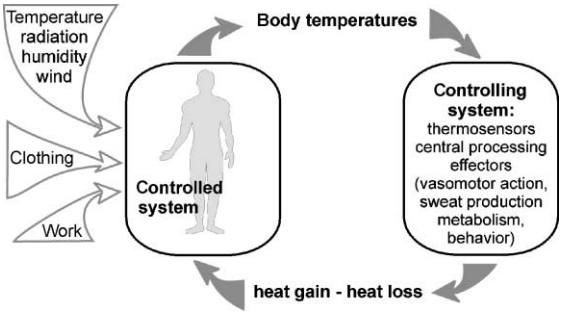

Fig. 2. The basic control of thermoregulation.

In the open-loop controlled system heat gain enhances, and heat loss attenuates, body temperature by an amount that depends on the ambient temperature, T_a. The unbroken line in Fig. 3A shows the thermoneutral ambient temperature ($\Delta T_a = 0$) and the two broken lines represent examples of higher or lower ambient temperatures, which, in the passive system, even with $\Delta HG = 0$, will increase or decrease ΔT_b, as shown by the arrows indicated by ΔT_0. Looking at the controlling system (Fig. 3B) with ΔT_b as the input and ΔHG as the output, an increasing ΔT_b will decrease ΔHG

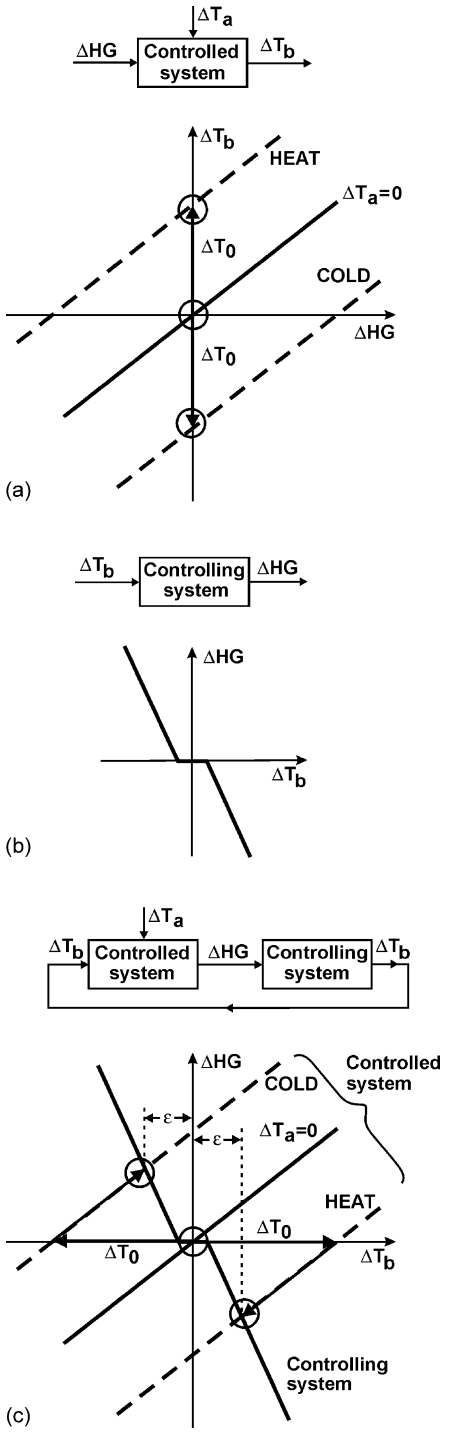

Fig. 3. The mechanism involved in attaining steady states of the body in heat and cold (see text). Abbreviations: Δ = deviation of, T_b = body temperature, T_a = ambient temperature, HG = net heat gain, ΔT_0 = deviation of body temperature without feedback control, ε = deviation of body temperature with feedback control ('load error'). (A) The open-loop characteristics of the controlled system. (B) The open-loop characteristics of the controlling system. (C) Steady states (circles) for $\Delta HG = 0$ attained in the closed control loop for thermoneutral conditions, for cold and heat.

in order to counteract disturbances in the controlled system. ΔHG (see Fig. 3B) is proportional to $-\Delta T_b$, which in the closed control loop (Fig. 3C) is the basis of 'proportional control'. In the closed loop, the controlled and the controlling systems have to interact. The output of the first is the input of the second and vice versa, meaning that a steady state must be compatible with the characteristics of Fig. 3A and B. These are drawn in a common diagram in Fig. 3C, demonstrating that only intersections of the characteristics of the controlled and controlling systems denote possible steady states. Fig. 3C shows the three steady states (circles) for thermoneutral, cold and warm conditions. In the cold and in the heat, this implies deviations, ε, ('load errors') from the set-point. This is an inherent property of 'proportional feedback control'. However, these deviations, ε, are much smaller than the deviations, ΔT_0, without any feedback control (see Fig. 3C).

Only an 'integrating' controller, reacting according to the temporal integral of the input, would provide a vanishing load error, ε. However, such a controller type, frequently applied in technical systems, is obviously not present in physiological autonomic control.

3. Interaction and competition in meshed control loops

It is obvious, e.g. during intense work, that besides environmental factors, vasomotor action and hence skin blood flow/volume and sweat rate determine whether or not a tolerable body temperature is maintained. Fig. 4 presents an overview of the interactions between the physiological systems involved.

The ambient temperature acts on the thermoregulatory centers by changing body temperature. This activates one of three autonomic effector

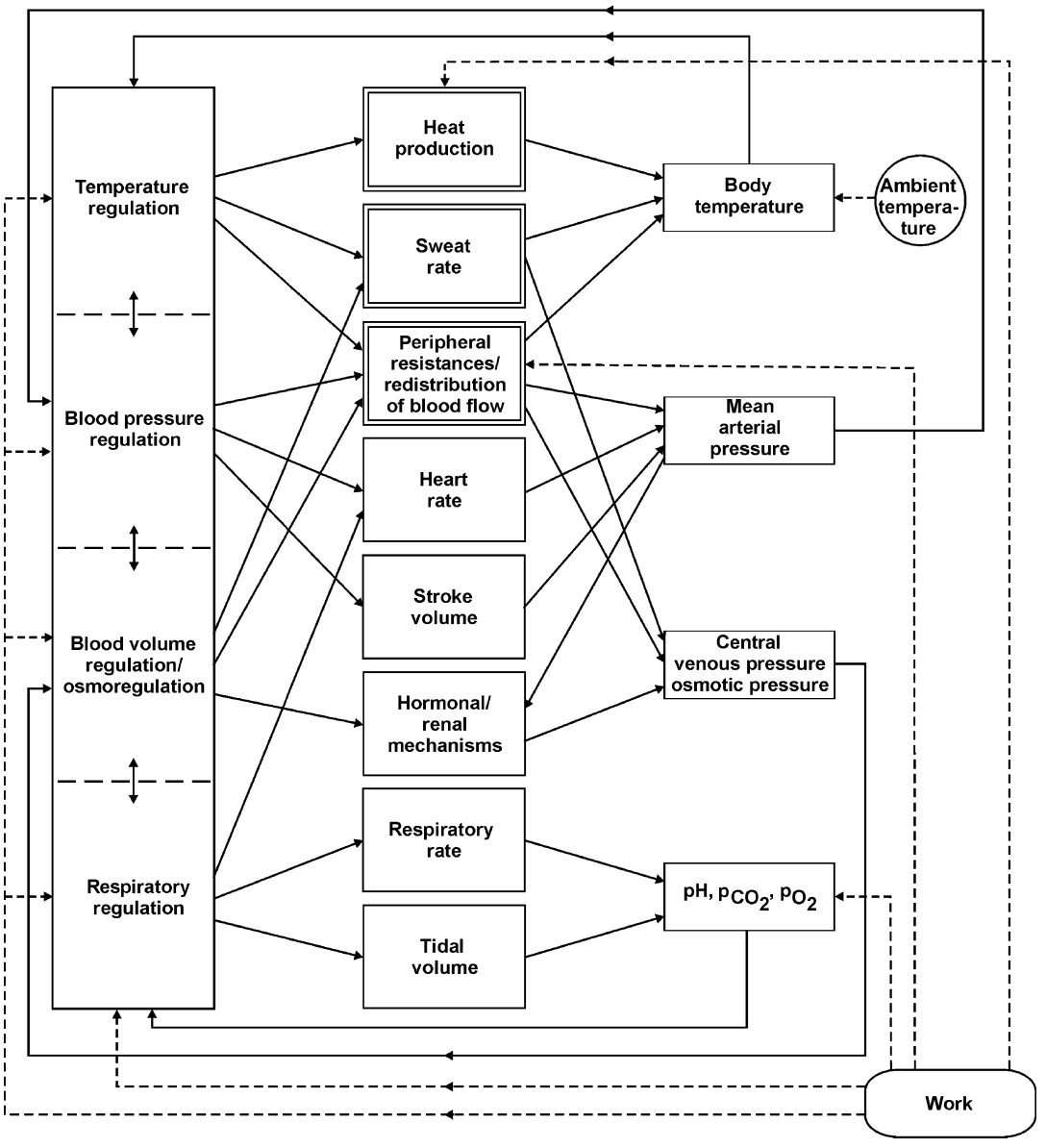

Fig. 4. Schema of interaction of regulatory processes during work.

mechanisms: heat production in the cold, sweat production in the heat, and, in general, changes in peripheral circulatory resistance with redistribution of blood flow (3). On the one hand, the latter effector mechanism may be used at the same time by other regulatory 'centers' (i.e. respiratory regulation, blood volume and osmotic regulation, and blood pressure regulation) and, therefore, may determine or limit thermoregulatory performance. On the other hand, thermoregulatory effector mechanisms may directly disturb the mean arterial pressure and central venous and central osmotic pressure, which again interact considerably with the ongoing process of sweat production.

Work interacts directly with at least two effector mechanisms of thermoregulation, heat production and vasomotor changes, and thereby with three regulated variables: body temperature, mean arterial pressure, and central venous pressure (CVP). Additionally, continuous intense work may disturb variables regulated by respiration. Furthermore, work interacts via proprioceptive sensors with the central control mechanisms of respiration and blood pressure. It is also highly probable that all of the regulatory 'centres', outlined in Fig. 4, communicate with one another. Fig. 4, though extremely schematic and simplified in comparison to reality, shows a complex network of interactions, the most critical and multifold processes being concentrated on the vasomotor activities.

Syncope can occur even in mild heat after prolonged standing, or abrupt stopping after intense work; this is due to competition between orthostatic and thermoregulatory demands impacting upon blood pressure regulation. It is characterized by extreme vasodilatation and a drop in arterial pressure, whilst there may only be a slight increase in body temperature. Generally, the consequences of such a heat syncope are not very harmful, but another heat illness, heat stroke, discussed below, includes the risk of death or, at least, serious permanent disability.

Heat stroke usually occurs when extreme heat stress leads to marked hyperthermia after thermoregulation is subordinated to circulatory and metabolic demands. Then the body temperature may rise beyond tolerable limits, and all regulation will fail (4). The first stage is a tolerable core and skin temperature increase, which as a rule, may be compensated. It is evoked by an increase in heat production and by the redistribution of blood flow. In the second stage, as a result of the body temperature rise, sweat production is enhanced, which together with dehydration increases fluid influx into the muscles, and the higher skin blood flow and volume ultimately leads to a drop of blood (plasma) volume and of CVP. If these stressors are sufficient, low CVP can reduce skin blood flow, and this together with the now lower sweat rate (due to lower blood volume, higher osmolality, local processes, and possibly lower skin blood flow), constitutes a serious impairment of heat loss mechanisms and causes a further increase in core temperature. The rise in body temperature, together with a possible deficiency of substrates in the muscles, might constitute a drive for muscle blood flow, further reducing CVP. Any increase in muscle blood flow, combined with a possible increase in splanchnic blood flow (5), evokes a further decrease in the skin blood flow and consequently, in a third stage, would drive the core temperature to finally reach an intolerably high level, resulting in red blood cell sphering, disseminated intravascular coagulation, coagulative necrosis, cerebral hypoxia, general central nervous dysfunction and death.

Training and heat acclimatization enable people to work longer and at lower body temperatures under conditions of heat stress, if water is available. However, as shown above, if circulatory control gains precedence over temperature regulation, even highly trained workers run the risk of fatal heat stroke, whereas their less fit counterparts often tend to stop work at a lower body temperature or collapse from heat exhaustion.

4. Changes in the set-point of thermoregulation

In hyper- or hypothermia, body temperature deviates substantially from the set-point, mainly because of insufficient effector capacity. The set-point itself may change periodically (e.g. circadian rhythm) or temporarily, due to interference with the

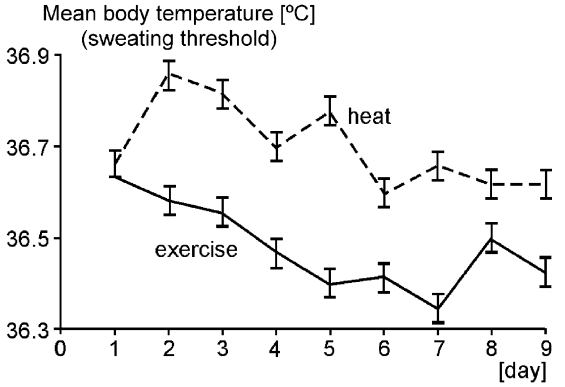

Fig. 5. Decrease of sweating thresholds for mean body temperature evoked either by external heat or by exercise.

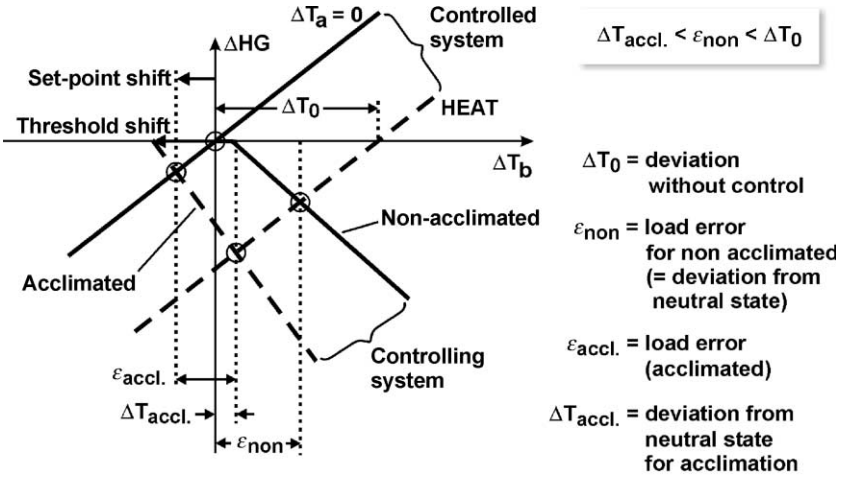

Fig. 6. Different steady-states (circles) and load errors ε in the non-acclimatized and in the acclimatized status due to change of set-point (Abbreviations see Fig. 3).

regulation of non-thermal variables, or due to pathological, non-thermal influences (e.g. during a fever). Also, the processes of acclimatization change the set-point. The change of set-point in these processes is thought to be due to changes in the thermal controller characteristics, particularly changes in thresholds and/or changes of thermo-effector 'gain', (i.e. the slope of the controller characteristic, e.g. Refs. (6,7)). Fig. 5 shows the threshold for the onset of sweating in terms of mean body temperature in the course of acclimatization processes, either evoked by external heat or by exercise (internal heat). The onset of sweating shifts to lower body temperatures, no matter whether the subjects are acclimatized by exercise or by heat stress. Fig. 6 outlines the change of set-point, and hence of load error, ε, due to heat acclimatization that evokes a decrease in the sweating threshold and thus, if physical conditions permit evaporation, a decrease in the heat loss threshold (see broken line, controller characteristic). This causes a change of set-point (denoted by the arrow on the ΔT_b-axis). It implies two different steady states (see circles) for a non-acclimatized and an acclimatized subject, both with $\Delta T_a = 0$ and with heat stress. The deviation, ΔT_{accl}, from the neutral state with acclimatization, is smaller than ε_{non} without acclimatization.

5. Conclusions

Thermoregulation uses proportional feedback control. This implies a 'load error', i.e. in the presence of an external or internal thermal load there is a permanent deviation of body temperature, which is much smaller than that which would be present without feedback control. The set-point is found when the effector activities that counteract alterations in body temperature are minimal.

Thermoregulation is embedded in the main regulatory systems, i.e. respiration, circulation, fluid and osmotic balance, and metabolism. An essential feature of this interaction is the use of common effector mechanisms, particularly vasomotor activity.

This interaction, as well as other important processes, like fever or acclimatization, may involve changes in the set-point of thermoregulation. For example, as a rule, acclimatization reduces the 'load error'.

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The interrelation of thermal and nonthermal reflexes in the control of postexercise heat loss responses☆

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Abstract: The role of baroreceptor modulation on the postexercise esophageal temperature threshold for cutaneous vasodilation (Th_{VD}) and sweating (Th_{SW}) was investigated. Five subjects, fitted with a water-perfused, upper body suit, performed a total of four experimental trials that were carried out in a random order. Each of the four experimental trials commenced with a 15-min baseline rest period, after which subjects either exercised (Exercise) or remained resting (No-Exercise) in a temperature-controlled chamber (25°C). For the Exercise condition the subjects performed 15 min of upright cycling at 70% of their predetermined $\dot{V}O_{2max}$. For the No-Exercise condition the subjects were instructed to rest in a semi-seated, upright position for 15 min. Immediately following both the No-Exercise and Exercise conditions subjects were placed in a semi-seated, upright position within a specially designed pressure chamber sealed at the level of the iliac crest. They were then exposed to either 50 mmHg lower body positive pressure (LBPP) or no lower body positive pressure (No-LBPP). During this time cool water (~20°C) was circulated through the water-perfused suit until forearm cutaneous vasoconstriction was noted. Mean skin temperature was then progressively increased to 47°C by increasing the temperature of the water circulating through the suit at a rate of 4.2 ± 0.8°C h^{-1} and cutaneous vasodilation and sweating was noted (~80 min). To compare thresholds between conditions in which both esophageal and mean skin temperatures were changing, the following equation was used to correct the T_{es} [$T_{es(calculated)}$] for a designated skin temperature [$\bar{T}_{sk(designated)}$]: $T_{es(calculated)} = T_{es} + [\beta/(1 - \beta)][\bar{T}_{sk} - \bar{T}_{sk(designated)}]$, where β is the fractional contribution of the skin to the vasodilation ($\beta = 0.2$) and sweating response ($\beta = 0.1$). Th_{VD} and Th_{SW} increased by 0.42 and 0.25°C, respectively, postexercise from the No-Exercise/No-LBPP condition to the Exercise/No-LBPP condition ($p < 0.05$). The postexercise increase in Th_{VD} and Th_{SW} was abolished in the Exercise/LBPP ($p < 0.05$). The parallel response observed in Th_{VD} and Th_{SW} postexercise, with and without the application of LBPP, may support a possible functional link between sweating and active vasodilation. More importantly, these data support the hypothesis that the elevated postexercise Th_{VD} and Th_{SW} observed are the result of baroreceptor unloading.

Keywords: Skin blood flow, Sweating, Blood pressure, Hypotension, Baroreceptor

1. Introduction

Dynamic exercise is known to cause postexercise hypotension (1). Although the exact mechanism(s) responsible for postexercise hypotension remain(s) undetermined, it has been shown that acute reductions in central venous pressure delay or decrease the rise in skin blood flow (SkBF) (2) and sweating during heat stress (3). Thus, it is possible

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that the postexercise increase in Th_{VD} and Th_{SW} (4) is related to the fact that the control of SkBF and sudomotor activity following exercise is subject to significant modulation by nonthermoregulatory baroreceptor reflex. In a recent study, it was shown that the nonthermal baroreceptor response to postexercise venous blood pooling, by means of head-down tilt, significantly influences cutaneous vasomotor control during exercise recovery (sweating response was not measured) (5). However, due to the fact that mild head-down tilt does not seem to modify arterial blood pressure, it is thought that in this case only cardiopulmonary baroreceptors are loaded. On the other hand, typical hypotension associated with postexercise venous pooling tends to unload both cardiopulmonary and sinoaortic baroreceptors. Furthermore, the postural manipulation model may stimulate several other reflexes (i.e. vestibular, etc.) that may tend to distort the primary baroreceptor response. Thus, using a specially designed upright lower body positive pressure (LBPP) chamber, we evaluated the hypothesis that reversing postexercise venous pooling with LBPP would result in a relative lowering of the resting threshold for Th_{VD} and Th_{SW}. Since postexercise hypotension is most commonly reported after a bout of upright dynamic exercise, we felt that this upright model would be most suitable.

2. Methods

With approval from the Faculty of Health Sciences Human Ethics Committee, five healthy men participated in the study. Although all participants were physically active, none was engaged in regimented physical training of any type. Subjects were (mean ± SD) 26 ± 5 years of age, 185.5 ± 6.38 cm tall, and weighed 84.1 ± 7.8 kg.

Esophageal temperature (T_{es}) was measured by means of inserting an esophageal thermocouple, through one nostril, to the level of the heart. Skin temperature was measured at seven sites by heat flow sensors and the area-weighted mean $\bar{T}_{sk}$ was calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 19%, anterior thigh 21%, and posterior calf 18%.

SkBF was measured from the left mid-anterior forearm at two sites, separated by a distance of approximately 10 cm, by laser-Doppler flowmetry (Perimed, PeriFlux System 5000). Sweat rate was measured using a ventilated capsule ($\approx 5.0 \times 3.5$ cm) placed on the upper back. Sweat rate was the product of the difference in water content between effluent and influent air, and the flow rate.

Mean arterial pressure (MAP) was continuously recorded from the electrical integration of the pulsatile blood pressure signal obtained from the middle digit (Ohmeda, Finapres 2300). Heart rate (HR) was measured, beat-by-beat, using a Polar coded transmitter and recorded continuously with a Polar Advantage interface (Polar Electro, Finland).

Subjects performed one incremental maximal oxygen consumption ($\dot{V}O_{2max}$) test on a cycle ergometer on the first day. These data were used to select the workload for the submaximal experimental exercise trials. Each subject performed a total of four experimental trials that were carried out in a random order and commenced between 7:00 and 8:00 am. Upon arrival at the laboratory, subjects were clothed in shorts and athletic shoes and instrumented appropriately. They were then fitted with an upper body water-perfused suit (covering the torso, arms, and head). Subjects were then placed into the LBPP chamber, in an upright, semi-seated position, sealed at the iliac crest.

Each of the four experimental trials commenced with a 15-min baseline resting period after which subjects either exercised (Exercise) or remained resting (No-Exercise) in a thermally controlled room (25°C). For the Exercise treatment the subjects performed 15 min of upright cycling at 70% of their predetermined $\dot{V}O_{2max}$. For the No-Exercise treatment the subjects were instructed to rest in a semi-seated, upright position for 15 min. To measure the effect of postexercise venous pooling on the resting postexercise T_{es} threshold for cutaneous vasodilation (Th_{VD}) and sweating (Th_{SW}), post-treatment resting measurements of Th_{VD} and Th_{SW} were conducted. Immediately following both the No-Exercise and Exercise treatments, subjects either remained (No-Exercise treatment) or were placed

(Exercise treatment) in a semi-seated upright position within the pressure chamber sealed at the level of the iliac crest. They were then exposed to either 50 mmHg LBPP or no lower body positive pressure (No-LBPP). During this time cool water (~20°C) was circulated through the water-perfused suit until forearm cutaneous vasoconstriction was noted (No-Exercise ~69 min, Exercise ~95 min). Mean skin temperature was then increased at a rate of 4.2 ± 0.8°C h^{-1} as the water circulating through the suit was progressively increased to 47°C and cutaneous vasodilation and sweating were noted (~80 min).

To compare thresholds between conditions in which both esophageal and mean skin temperatures were changing, the following equation was used to correct the T_{es} [$T_{es(calculated)}$] for a designated skin temperature [$\bar{T}_{sk(designated)}$]: $T_{es(calculated)} = T_{es} + [\beta/(1 - \beta)]\ [\bar{T}_{sk} - \bar{T}_{sk(designated)}]$ (4,6). β = fractional contribution of the skin to the vasodilation ($\beta = 0.2$) (7) and sweating response ($\beta = 0.1$) (8). For the purpose of comparison, the thermoregulatory response thresholds for cutaneous vasodilation and sweating were identified for each condition as follows: (a) Exercise/LBPP; (b) Exercise/No-LBPP; (c) No-Exercise/LBPP; and (d) No-Exercise/No-LBPP.

3. Results

Resting T_{es} and $\bar{T}_{sk}$ were similar for all conditions and remained stable and consistent during the 15-min baseline resting period. The T_{es} required to elicit cutaneous vasodilation (Th_{VD}) and sweating (Th_{SW}) for Exercise/No-LBPP was significantly elevated from the No-Exercise/No-LBPP by 0.42 ± 0.15°C and 0.25 ± 0.12°C, respectively ($p < 0.05$) (Tables 1 and 2). The application of LBPP following exercise resulted in a relative lowering of Th_{VD} and Th_{SW} by 0.44 ± 0.10°C and 0.23 ± 0.09°C, respectively. No differences were measured between the two No-Exercise conditions for either Th_{VD} or Th_{SW}.

Resting MAP and HR were similar for all conditions. Postexercise MAP (80 ± 2 mmHg) was significantly lower than baseline resting MAP (85 ± 2 mmHg) in the Exercise/No-LBPP condition ($p < 0.05$), while no differences were noted for the No-Exercise/No-LBPP conditions (i.e. 85 and 84 mmHg for resting and post-treatment, respectively). MAP was significantly elevated with LBPP application in the No-Exercise (82–93 mmHg) and Exercise (83–95 mmHg) conditions ($p < 0.05$). The mean exercise heart rate was the same for both Exercise conditions (166 ± 6 beats·min^{-1}). Postexercise heart rate remained elevated (≧ 20 beats·min^{-1}) in the Exercise/No-LBPP condition for the duration of the experimental trial ($p < 0.05$). Heart rate returned to baseline resting values within 30 min postexercise with the application of LBPP (70 ± 3 beats·min^{-1}). In the No-Exercise condition heart rate was significantly lowered below baseline values (12 ± 1 beats·min^{-1}) with LBPP application ($p < 0.05$).

4. Discussion

The postexercise increase in Th_{VD} (0.42°C) and Th_{SW} (0.25°C) observed here is similar to previous

Table 1
Mean (± SE) threshold values for cutaneous vasodilation.

	No-Exercise			Exercise		
	T_{sk} (°C)	T_{es} (°C)	$T_{es(calculated)}$ (°C)	T_{sk} (°C)	T_{es} (°C)	$T_{es(calculated)}$ (°C)
No-LBPP	33.77 (0.14)	36.49 (0.09)	36.40 (0.12)	34.64 (0.35)	36.69[a] (0.10)	36.82[a] (0.15)
LBPP	33.84 (0.40)	36.33 (0.11)	36.26 (0.18)	34.07 (0.33)	36.38[b] (0.07)	36.38[b] (0.10)

Note: $\bar{T}_{sk(designated)}$ was set as the average $\bar{T}_{sk}$ at rest for all conditions (34.1°C).
[a]Significant difference from the No-Exercise/No-LBPP condition ($p < 0.05$).
[b]Significant difference from No-LBPP for the respective treatment conditions ($p < 0.05$).

Table 2
Mean (± SE) threshold values for sweating.

	No-exercise			Exercise		
	T_{sk} (°C)	T_{es} (°C)	$T_{es(calculated)}$ (°C)	T_{sk} (°C)	T_{es} (°C)	$T_{es(calculated)}$ (°C)
No-LBPP	34.65 (0.15)	36.71 (0.13)	36.62 (0.14)	35.43 (0.27)	36.87[a](0.23)	36.87[a] (0.12)
LBPP	35.85[b] (0.13)	36.63 (0.07)	36.68 (0.08)	35.71 (0.23)	36.60[b] (0.07)	36.64[b] (0.09)

Note: $\bar{T}_{sk(designated)}$ was set as the average $\bar{T}_{sk}$ at rest for all conditions (35.4°C).
[a]Significance differences from the No-Exercise/No-LBPP condition ($p < 0.05$).
[b]Significant difference from No-LBPP for the respective treatment conditions ($p < 0.05$).

findings of a postexercise increase in warm response thresholds (4). The most important finding within the present investigation is that postexercise upright LBPP application resulted in a decrease of Th_{VD} (0.44°C) and Th_{SW} (0.23°C) compared to that observed during normal resting conditions. The observed decrease in Th_{VD} of 0.16°C (n.s.) in the No-Exercise trial with the application of LBPP is consistent with the decrease (~0.1–0.2°C) in Th_{VD} in response to postural changes (i.e. an upright to supine position) (9). No differences were measured in Th_{SW}.

Cutaneous circulation is considered to be primarily an efferent arm of thermoregulatory reflexes, but it is also known to respond to several nonthermoregulatory demands (10), including baroreceptor control (3,11). Several studies have documented increases in Th_{VD} associated with baroreceptor unloading in resting conditions. Early research involving head-up tilt has proven to evoke cutaneous vasoconstrictor activity (12). Other studies demonstrated similar results with the application of lower-body negative pressure (LBNP) (3,13). As the results presented here demonstrate, modification of postexercise venous pooling by LBPP results in a relative lowering of the resting postexercise Th_{VD}. Thus, it would seem that SkBF control during and following exercise are subject to significant baroreceptor-mediated modifications. Because acute reductions in central venous pressure have been shown to delay or decrease the rise in SkBF during heat stress (2), it is reasonable to postulate that baroreceptors are involved in postexercise cutaneous vasoconstriction during postexercise recovery in an attempt to maintain normal postexercise blood pressure. This baroreceptor response on cutaneous vascular tone would be manifested either as an activation of sympathetic adrenergic vasoconstrictor nerves or as a withdrawal of active vasodilator activity (14).

Sympathetic nerve recordings from sudomotor fibers show cardiac rhythmicity indicating that changes in blood pressure may act to modify sweat gland activity (15). Solack *et al*. (16) showed that local sweat rate was attenuated during application of LBNP during resting. The postexercise increase in Th_{SW}, paralleled by a postexercise hypotension, observed here is consistent with the findings of Mack *et al*. (3). They reported a greater increase in Th_{SW} during exercise with baroreceptor unloading by LBNP. The reversal of the postexercise increase in Th_{SW} with LBPP supports an important role of baroreceptor modulation on postexercise sweating. Furthermore, the observed parallel response of Th_{VD} and Th_{SW} postexercise, with and without the application of LBPP, supports an important functional link between sweating and active vasodilation.

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