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Natalie McInnis

AUTEUR DE LA THÈSE / AUTHOR OF THESIS

M.Sc. (Human Kinetics)

GRADE / DEGREE

School of Human Kinetics

FACULTÉ, ÉCOLE, DÉPARTEMENT / FACULTY, SCHOOL, DEPARTMENT

Contribution to Nonthermal Baroreceptor Influence on Postexercise Core Temperature Regulation

TITRE DE LA THÈSE / TITLE OF THESIS

Glen Kenny

DIRECTEUR (DIRECTRICE) DE LA THÈSE / THESIS SUPERVISOR

CO-DIRECTEUR (CO-DIRECTRICE) DE LA THÈSE / THESIS CO-SUPERVISOR

EXAMINATEURS (EXAMINATRICES) DE LA THÈSE / THESIS EXAMINERS

Ron Sigal

Denis Prud`homme

Gary W. Slater

Le Doyen de la Faculté des études supérieures et postdoctorales / Dean of the Faculty of Graduate and Postdoctoral Studies

CONTRIBUTION OF NONTHERMAL BARORECEPTOR INFLUENCE ON POSTEXERCISE CORE TEMPERATURE REGULATION

By

NATALIE MCINNIS

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THESIS

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ABSTRACT

Purpose: The objective of the thesis was to examine the role of nonthermal regulatory influences on thermoregulatory responses during the postexercise period. Specifically, *study #1* examined the effect of head down tilt (HDT), and by extension the role of baroreceptors, on prolonged postexercise heat loss and hemodynamic responses in males. *Study #2* examined the effect of 15° HDT on cardiovascular and thermal responses in females. In particular the role of baroreceptors on cutaneous vascular conductance (CVC) and sweating during extended recovery from dynamic exercise. *Study #3* examined the effect of exercise intensity on hemodynamic and thermal responses in females. **Methods**: *Study #1* and *#2*- seven male subjects and seven females subjects respectively, performed the following three experimental protocols: 1) 60 min in the upright-seated (URS) posture followed by 60 min in the 15° head-down tilt position (HDT); 2) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$ peak followed by 60 min recovery in the URS posture; or 3) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$peak followed by 60 min recovery in the 15° HDT position. . *Study #3*- Seven females subjects performed the following 3 experimental protocols on a cycle ergometer: exercise at 1) 85% $\dot{V}O_{2peak}$; 2) 75% $\dot{V}O_{2peak}$; or 3) 55% of their pre-determined $\dot{V}O_{2peak}$ followed by 60 min recovery in the upright-seated position. Mean skin temperature (T_{sk}), esophageal temperature (T_{es}), skin blood flow (SkBF), sweat rate, cardiac output (CO), stroke volume (SV), heart rate (HR), total peripheral resistance (TPR), and mean arterial pressure (MAP) were measured in all three experiments. **Results**: *Study #1*- During recovery from exercise, a significantly greater MAP, SV, CVC and sweat rate and a significantly lower HR were found with HDT in comparison to URS posture ($p \leq 0.05$). Subsequently a significantly lower T_{es} was observed with HDT after 15-min of recovery onwards ($p \leq 0.05$). At the end of 60-min recovery, T_{es} remained significantly elevated above baseline with URS ($p \leq 0.05$), however T_{es} returned to baseline with HDT. *Study #2*- During recovery from exercise, a significantly greater MAP, SV CVC and sweat rate and a significantly lower HR were found with HDT in comparison to URS recovery posture ($P \leq 0.05$). Subsequently a significantly lower T_{es} was observed with HDT from 12-min till the

end of the recovery period ($P \leq 0.05$). At the end of 60-min recovery, T_{es} remained significantly elevated above baseline with URS recovery posture ($P \leq 0.05$), however T_{es} returned to baseline with HDT. *Study #3-* as exercise intensity increased MAP, SV, sweating and CVC significantly decreased and HR significantly increased ($P \leq 0.05$). Subsequently the magnitude and duration of T_{es} also increased with exercise intensity ($P \leq 0.05$). At the end of 60-min recovery, T_{es} remained significantly elevated above baseline following exercise at 85% $\dot{V}O_{2peak}$ and 75% $\dot{V}O_{2peak}$ ($P \leq 0.05$) but returned to baseline values following exercise at 55% $\dot{V}O_{2peak}$ ($P > 0.05$). Conclusion: *Study #1&2-* extended recovery from dynamic exercise in the 15° HDT position attenuates the reduction in CVC and sweating thereby significantly increasing the rate of esophageal temperature decay compared to recovery in the URS posture. *Study #3-* in association with an increase in postexercise hypotension as exercise intensity increases esophageal temperature remains elevated for a prolonged period of time and CVC and sweat rate return to pre-exercise values more rapidly.

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ABBREVIATIONS

BPM	Beats per minute
CO	Cardiac output
CVC	Cutaneous vascular conductance
HR	Heart rate
HDT	Head down tilt
H_{sk}	Heat flux
L	Liters
MAP	Mean arterial pressure
ml	Milliliters
PU	Perfusion units
SkBF	Skin blood flow
SV	Stroke volume
T_{sk}	Skin temperature
T_{es}	Esophageal temperature
URS	Upright-right seated
TPR	Total peripheral resistance
$VO_{2\ max\ or\ peak}$	Maximal Oxygen consumption

PART ONE: EMPIRICAL, THEORETICAL AND METHODOLOGICAL CONSIDERATIONS

CHAPTER 1

INTRODUCTION

1.0 Introduction

During thermoregulation in humans, control of body heat content is in large part dependent upon internal heat exchange between tissues and compartments of the body, and externally between the body surface and the ambient environment. The mechanisms of energy exchange involved in thermal homeostasis include exchange by radiation and conduction internally and externally by radiation, conduction and evaporation. Convective flow of fluid over heated surfaces alters the rate at which heat is exchanged by conduction. Similarly, convective flow of relatively dry air over a wet surface alters the rate of evaporation. Further, convective flow of blood to the skin effects the rate at which heat is exchanged by conduction between the blood and the skin. Therefore, control of skin perfusion directly affects heat exchange with the external environment. Body temperature change is ultimately affected by the rate of heat loss from the surface of the body, therefore assuming a constant ambient temperature and humidity; the ultimate control of heat loss response will be influenced by the body's internal physiological activity.

Thermoregulation at rest, during or after exercise is influenced by the capacity of the cardiovascular system to transport heat to the body's surface via the peripheral circulation. The net exchange of heat between the blood and the skin is determined by temperature gradient and convective flow. In addition, sweating can also provide a gradient favoring net heat loss to the external environment; this gradient is dependent on the humidity of the external environment. During dynamic exercise there is an additional load on the cardiovascular system because muscle and cutaneous blood flow are increased. Elevations in

cutaneous blood flow are primarily driven by thermoregulatory mechanisms which increase the rate of heat loss and to some extent compensate for the rate of heat gain within the body. If the added demand on the limited capacity of the cardiovascular system is not met, any additional adjustments will be at the expense of skin perfusion or central blood volume and consequently mean arterial pressure (MAP) will not be maintained (Gisolfi & Wenger, 1984).

There are a number of cardiovascular mechanisms in place that maintain MAP at an optimal level. Input to the cardiovascular control centers from higher brain centers is coordinated by the autonomic nervous system. These mechanisms along with inputs from thermoregulatory and high brain inputs ultimately determine MAP. Such converging inputs are integrated during dynamic exercise and during post exercise period. In the post exercise period the cardiovascular response is influenced by two opposing mechanisms: 1) those required during thermoregulatory control and 2) those required in the maintenance of MAP subsequent to a reduction in central command and mechanoreceptor stimulation. During recovery from exercise there is an absence of descending central command signals and reduced activation of muscle mechanoreceptors. Therefore, baroreceptor activity is involved in the control of MAP, and also influences the control of skin blood flow and sweating (Carter et al., 2002). This suggests that during the post exercise period, body temperature regulation is secondary to nonthermal influences.

The current understanding of post exercise temperature regulation contains some assumptions about females such that only male subjects have been studied or not enough female subjects were included to determine the mechanisms responsible for gender difference in post exercise period. Given the apparent relationship between post exercise cardiovascular regulation and thermal responses, it is important to examine the effect of gender in light of the

different recovery responses. Carter and colleges (2001) suggested that women may be more susceptible to post exercise orthostatic hypotension and that active recovery from dynamic exercise should diminish that risk. Their study indicates that there is a greater reduction in MAP and less compensatory vasoconstriction in women than in men during recovery from exercise. In addition, a recent study has shown that females appear to demonstrate a difference in the control of cutaneous vascular conductance during recovery (Journeay et al., 2005a). Specifically, it was shown that during passive recovery in females cutaneous vascular conductance is influenced by central command, this was not the case for males when they were studied under similar conditions (Journeay et al., 2004). In addition, Kenny and colleagues (2005) have found that compared to males, females demonstrated a greater magnitude of post exercise hypotension and this was accompanied by a greater elevation in post exercise threshold for cutaneous active vasodilation. Thus the effect of gender differences in cardiovascular regulation should be examined in the context of the possible consequences to post exercise heat loss.

As noted above, current evidence suggests that nonthermoregulatory influence associated with hemodynamic regulation may have a prominent role in heat loss response. This is primarily supported by the apparent link between the thermal response and concurrent cardiovascular changes incurred after a bout of dynamic exercise. Further research is needed to address possible gender differences and the interaction of thermoregulatory and cardiovascular inputs and ultimately the influence of body temperature regulation post exercise. There remains little information regarding the interaction of thermoregulatory and cardiovascular input post exercise. Given the pivotal role of the cardiovascular system in heat dissipation it is essential to describe the changes that typically occur postexercise and evaluate

the possible gender influences on this response. The following research studies will be conducted with the primary aim of characterizing the role of non-thermal baroreceptor activity and post exercise thermal response and to evaluate the possible gender differences. The study will focus on the following three areas:

A) ***15° Head-down tilt reduces postexercise esophageal temperature recovery time while attenuating the reduction in heat loss responses.***

During recovery from dynamic exercise thermoregulatory control is compromised. In addition to a prolonged increase in core temperature, sweating and skin blood flow return to pre-exercise values (Kenny et al., 2002; Mack et al., 1998). During the postexercise period there is also a significant drop in MAP, commonly referred to as postexercise hypotension. Postexercise hypotension results from the combined effects of persistent neural and vascular adjustments as well as the seated posture and lack of muscle pump. Resulting in a significant pooling of blood in the lower extremities and ultimately baroreceptor unloading. The sustained increase in body heat content seen during the postexercise period has been related to non-thermal factors associated with blood pressure regulation.

Head-down tilt (HDT) is known to increase central blood volume by promoting venous return through a reduction in the hydrostatic forces present in the upright-seated posture. Specifically, under resting conditions at tilt angles of less than 30 degrees is thought to engage the cardiopulmonary baroreceptors without changes in systemic MAP and therefore reduces the likelihood of engaging the arterial baroreceptors (19, 21-23, 64, 76). Thus, HDT is a method by which to manipulate postexercise hemodynamic responses and allows for the attenuation of the baroreceptor unloading effect of upright-seated recovery.

Therefore, the purpose of this experiment is to examine the effect of HDT, and by extension the role of baroreceptors, on prolonged postexercise heat loss and hemodynamic responses.

B) *15° Head-down tilt significantly decrease core temperature and attenuates the postexercise reduction in cutaneous vascular conductance and sweat rate in females.*

During recovery from dynamic exercise, significant cardiovascular and thermal adjustments occur. Recovery in the upright-seated posture leads to pooling of blood in the previously active musculature, causing a reduction in cardiac filling (Kilgour et al., 1993) which stimulates baroreceptor unloading (Carter et al., 2002). Increase baroreflex stimulation initiates reflexes that attempt to increase MAP.

Males and females differ in their response to orthstatic stress. When faced with the same orthostatic challenge females will show a greater increase in HR, less of an increase in TPR, lower blood volume, greater decline in CO, and a greater decrease in MAP compared to males (8, 11). The mechanisms responsible for these gender differences are still under investigation. Kenny and colleges (2006) found that along with a greater decrease in postexercise hypotension, there is also a greater elevation in post exercise threshold for cutaneous active vasodilation in females. In addition, by studying the effect of recovery modes Journey et al. (2005) found that cardiopulmonary baroreceptors and central command have a significant effect on cutaneous vascular conductance in females. The control of sweating during the recovery period was less clear. Interestingly, when different recovery modes were studied using males subjects the results showed that central command does not

modulate cutaneous vascular conductance (Journeay et al., 2004). To date there is a lack of information on the mechanisms responsible for these gender differences discussed above.

The present study will use head-down tilt (HDT) to increase central blood volume by promoting venous return. To the best of our knowledge no study has examined the effect of HDT on postexercise heat loss responses in females. HDT angles less then 30 degrees is thought to engage the cardiopulmonary baroreceptors without changes in MAP (19, 21-23, 64, 76). Therefore HDT enables us to manipulate postexercise hemodyanmic responses and allows for the attenuation of the baroreceptor unloading. The purpose of the following study will be to examine the effect of 15° HDT on cardiovascular and thermal responses in females during the postexercise period. In particular the role of baroreceptors on cutaneous vascular conductance and sweating during extended recovery from dynamic exercise.

C) The effect of exercise intensity on postexercise heat loss responses in females

The effect of exercise intensity on postexercise hypotension response remains unclear. Some studies have noticed a significant difference in postexercise hypotension with different exercise intensities (Forjaz et al., 2004; Kenny et al., 2002; Piepoli et al.; 1994) while others have not (MacDonald et al. 1999, Pescatello et al., 1991; Forjaz et al., 1998). Kenny et al. (52) noted that as exercise intensity increases so does postexercise hypotension and the magnitude of the elevation in esophageal temperature during recovery from exercise. This elevation in esophageal temperature is a consequence of a non-thermal baroreceptor-mediated response. Baroreceptors are unloaded which initiates reflexes that aim to increase MAP. These reflexes while beneficial to the reestablishment of MAP seem to have a negative effect on the rate of core temperature recovery.

Females react differently then males to orthostaic challenges and yet many of the studies done in this field of research predominantly use male subjects therefore their findings cannot be generalized to the female population. A recent study has shown that females appear to demonstrate a difference in the control of cutaneous vascular conductance during recovery from dynamic exercise (Journeay et al., 2005a). Specifically, it was shown that during passive recovery in females cutaneous vascular conductance is influenced by central command. Also, Kenny and colleagues (2005) have found that compared to males, females demonstrated a greater magnitude of post exercise hypotension and this was accompanied by a greater elevation in post exercise threshold for cutaneous active vasodilation. A study by Carter et al. (2001) illustrate that there is a greater reduction in MAP and less compensatory vasoconstriction in women than in men during the postexercise period. These studies suggest that males and females may differ in their baroreflex response when faced with the same degree of orthstatic challenge. This may have a significant effect on postexercise thermoregulation.

Therefore the purpose of this study is to examine the effect of exercise intensity on hemodynamic and thermal responses in females. In particular, the role of baroreceptors on cutaneous vascular conductance and sweating during recovery from dynamic exercise at different intensities.

1.1 Hypotheses

General hypothesis of the thesis

It is hypothesized that non-thermal mechanisms that regulate blood pressure may override thermoregulatory responses during recovery from dynamic exercise. Heat loss

responses are compromised in the upright-seated posture secondary to nonthermal baroreceptor influences. In particular, it is thought that when baroreceptors are unloaded reflexes are initiated that attenuate skin blood flow and sweating. As a result the magnitude of the core temperature response is increased during the postexercise period.

Specific hypothesis of the experiments

a) We are testing the hypothesis that recovery from dynamic exercise in the 15°HDT position would augment the heat loss responses of cutaneous vascular conductance and sweating relative to upright-seated recovery in association with an augmented hemodynamic response.

b) It is hypothesized that recovery from exercise in a 15° HDT would offset the decrease in cutaneous vascular conductance and sweating seen during recovery from exercise in the upright-right seated position. Also, this will significantly increase the rate of esophageal temperature recovery time.

c) It is hypothesized that as exercise intensity increases women will show a greater attenuation in the thermoregulatory effector response of skin blood flow and sweating postexercise. This is associated with a greater reduction in postexercise MAP as exercise intensity increases.

1.2 Objectives

a) To examine the effect of promoting an increase in venous return through a reduction in the hydrostatic forces present in the upright posture via 15° head down tilt and its effect on cutaneous vascular conductance and sweating during the postexercise period.

b) To examine the effect cutaneous vascular conductance and sweat rate in females when manipulating postexercise hemodynamic responses using 15° HDT and therefore attenuating baroreceptor unloading seen during recovery in the URS posture.

c) To characterize postexercise hypotension in females and examine baroreceptor unloading in relation to skin blood flow and sweating.

1.3 Significance

These experiments will advance our understanding of non-thermal mechanisms and how they affect hemodyanmics and thermal responses, in particular, the role of baroreceptors on heat loss response. They will also give us information on the thermoregulatory response of females and some incite to why males and females differ in their response to orthostatic stress.

1.4 Limitations/Delimitations

- The results of this experiment will not be generalized to the entire population. This is due to the fact that the volunteers being studied are young, active males and females with no identified health problems.
- These studies will not measure skeletal muscle blood flow. Therefore any conclusions made on muscle blood flow are speculative as a result of manipulation of venous pooling.
- This experiment will not study the possible menstrual cycle effects within women. Although all subjects were in the follicular phase of the menstrual cycle during the testing.

CHAPTER 2

REVIEW OF LITERATURE

Thermoregulation is the process of regulating body temperature. The ability to effectively thermoregulate is limited by the capacity of the cardiovascular system to transport heat to the skin via peripheral circulation. The cardiovascular system is regulated by neural and chemical control systems. Recent research has focused on these controls, specifically the non-thermal mechanisms that control blood pressure. This review of literature will focus on the cardiovascular system and how it affects heat loss response during recovery from dynamic exercise.

2.0 Thermoregulation

The work of Hammel and colleagues (Hammel, 1968; Hammel, 1965; Hammel et al., 1963) and Hardy (1961) showed that signal processing for the regulation of body temperature was based upon a proportional controller with an adjustable set-point. The set-point control model of temperature regulation assumed that deep body temperature is by some means compared to a reference temperature. The preoptic region of the anterior part of the hypothalamus is the main integrating center, which stimulates adjustments in thermoregulatory effector response when changes in temperature are sensed (Stephenson & Kolka, 2003). The mechanisms whereby sensory information is processed and conveyed to the controller, and subsequently transformed into effector signals that stimulate the appropriate output, remain unclear.

Input to the preoptic region is received from temperature receptors in the skin and mucous membranes (peripheral thermoreceptors) and from internal structures (central

thermoreceptors). Central thermoreceptors include the hypothalamus which detects changes in circulating blood temperature. There are two types of thermal sensor distributed across the skin of the body, typically referred to as the warm and cold receptors. Thermal signals from these sensors, as well as from the different core regions, are integrated at the hypothalamus and compared with a central reference temperature referred to as the hypothalamic set-point. When the temperature deviates from this reference point the hypothalamus will coordinate a series of responses to maintain core body temperature in an optimal range using autonomic nervous system responses, neuroendiocrine responses and/or altering behavior.

The control of core temperature is largely dependent upon the internal heat exchange between tissues and different compartments of the body, and the external heat exchange between the body surface and the ambient environment (Webb, 1995). The energy exchange mechanisms employed during thermal homeostasis include internal exchange by radiation and conduction, and external exchange by radiation, conduction and evaporation. Convective flow of fluid over a heated surface alters the rate at which heat is exchanged by conduction. Similarly, convective flow of relatively dry air over a wet surface alters the rate of evaporation. Further, the convective flow of blood to the skin affects the rate at which heat is exchanged by conduction between the blood and the skin. Therefore control of skin perfusion directly affects heat exchange with the external environment. Core temperature change is ultimately influenced by the rate of heat loss from the surface of the body, therefore assuming a constant ambient temperature and humidity, the active control of heat loss responses will be determined by the body's internal physiological activity (Kenny et al., 2003).

The interaction of man with the thermal environment is theoretically demonstrated using the human heat balance equation (Ashrae, 1989):

$$M - W = (K + C + R + E_{SK}) + (C_{RES} + E_{RES}) + S$$

Where: M = rate of metabolic heat production
W = rate of mechanical work (effectively = 0)
K = rate of conductive heat loss
C = rate of convective heat loss from the skin
R = rate of radiative heat loss from the skin
E_{SK} = rate of evaporative heat loss from the skin
C_{RES} = rate of convective heat loss from respiration
E_{RES} = rate of evaporative heat loss from respiration
S = rate of body heat storage (all units $W \cdot m^{-2}$)

During exercise there is an increase in metabolic heat production (M). This increase is mainly due to heat produced as a by-product of skeletal muscle contraction (Saltin et al., 1966; Webb, 1995) Also, during exercise the rate of evaporative heat loss from the skin (E_{SK}) is dependent upon the relative humidity of the environment and can be reduced by insulative and vapour impermeable clothing. Rate of sensible heat loss from the skin through conduction, convection and radiation (K + C + R) is dependent upon the ambient temperature of the environment. When environmental radiant and air temperature is higher then that of skin temperature there is a transfer of heat towards the body and *vise versa*. Respiratory heat loss ($C_{RES} + E_{RES}$) is generally small.

During exercise high values for M not balanced by the avenues of heat loss, rate of body heat storage (S) is positive leading to an overall increase in core temperature. In response to the increase in core temperature, heat loss response mechanisms of are activated. This leads to an increase in sweating and skin blood flow, therefore increasing the rate of whole-body heat loss. Once heat production is equal to the rate of heat dissipation thermal balance is restored and core temperature reaches a steady-state.

Early investigators explained this exercise-induced rise in core temperature above the set-point as a discrepancy between the production and dissipation of heat. This interpretation

has become known as the load error concept and has endured periodic challenges. These challenges suggested that the hypothalamic set-point is reset to a higher value during periods of high metabolic rates (Tam et al., 1978; Jackson et al., 1963; Neilsen, 1938). Principle arguments against an adjustment of the hypothalamic set-point is that during exercise there is a high metabolic rate which elevates and maintains core temperature, in addition there is a simultaneous increase in the rate of heat loss to attenuate this rise in core temperature as noted above (Sawka et al., 1988). There are numerous studies that support the load error concept. For example, phase of the menstrual cycle (Inoue et al., 2005), hydration status (Fortney et al., 1984), and circadian rhythm (Wenger et al., 1978) modify the core temperature at which onset of the heat loss responses of skin vasodilation and sweating are activated.

Of note, Thoden et al. (1994) hypothesized that based on the set-point concept of temperature regulation, in the absence of the heat producing events of exercise, core temperature should return to pre-exercise resting levels . In their study, the subjects exercised on a treadmill at 75% $\dot{V}O_{2max}$ for 15min in a thermal controlled room at an ambient temperature of 29°C. In comparison to baseline measurements, their results showed a prolonged increase in esophageal temperature (~0.5-0.6°C) for the duration of the 65-min recovery period. Oxygen consumption, skin temperature and forearm skin blood flow rapidly returned to pre-exercise levels. Therefore they suggested that this reduction in skin temperature and forearm skin blood flow throughout postexercise recovery was an indication of an exercise-induced increase in the threshold for vasodilation during recovery. The postexericse thermoregulatory response will be discussed further below.

2.1 Postexercise Temperature Regulation

As discussed previously, "set-point" control is the most widely used model for explaining physiological changes that occur as a function of heat storage or heat content in the body (Hardy, 1961). Therefore, following this concept one would assume a rapid reestablishment of core temperature in the absence of heat production. While under passive heating conditions, heat balance is quickly restored and core temperature is stabilized to pre-heating values (Kenny et al., 1996). On the other hand, the "set-point" theory is contradicted during recovery from exercise in that the core temperature remains elevated.

Core temperature remains elevated for a prolonged period during recovery from dynamic exercise (Kenny et al., 1999, Kenny et al., 1997, Thoden et al., 1994). This increase in core temperature indicates an inability to effectively dissipate heat during the postexercise period. This difference in core temperature response associated with recovery from exercise and that measured with passive heating appears to be due to a difference in endogenous versus exogenous heating. Despite increasing the core temperature to the same absolute temperature no similar response was observed with exogenous heating (Kenny et al., 1996).

In addition, studies have also shown that sweating (Kenny et al., 2002), skin blood flow and skin temperature (Wilkins et al., 2004, Kenny et al., 2002) return to pre-exercise values despite a persistent elevation in core temperature. Based on the set point theory (Hardy, 1961) we should expect to see an increase in sweating and skin blood flow proportional to an increase in core temperature via a hypothalamic negative feedback loop. Such increases in skin blood flow and sweating would serve to increase heat dissipation, and result in a decrease

in core temperature. It appears that thermoregulation postexercise contrasts that of exogenous heating most likely due to differences in nonthermal factors.

2.2 Nonthermal Influence of Postexercise Temperature Regulation

Recently, Kenny and colleagues (2000) examined the core temperature at which thermoregulatory thresholds for vasodiation and sweating occurred during the postexercise period. They hypothesized that the threshold for cutanous vasodialation and sweating would increase during recovery from exercise. Specifically, their study showed that the core temperature threshold for cutaneous vasodiation increased from 36.56 °C to 37.11 °C during the postexercise period. Similarly, the threshold for sweating increased from 36.79 °C to 37.05 °C. In this study the subjects exercised on a cycle ergometer at 60% of VO_{2max}. The magnitude of the increase in the thresholds has also been shown to vary as a function of exercise intensity, as exercise intensity increases so does the core temperature at which the thresholds are initiated (Kenny et al., 2003a, Kenny et al., 2003b). These studies suggested that dynamic exercise has a residual effect on the control of skin blood flow and sweating however, the mechanisms of altered control remain to be elucidated.

Preliminary evidence suggests that the core temperature elevation during the postexercise period is due to the combination of body heat content and nonthermal factors, such as those associated with blood pressure regulation (Journeay et al., 2004; Jackson et al., 2003; Kenny et al., 2002; Kenny et al., 2000). This hypothesis has evolved from the observation that an increase in the postexercise hypotensive response, induced by exercise of increasing intensity, was shown to 1) result in a relative increase in the onset thresholds for sweating and cutaneous vasodilation, 2) an overall decrease in the rate of heat loss, and 3) a concomitant

increase in the postexercise core temperature recovery time (Kenny et al., 2003; Kenny et al., 2003; Kenny et al., 2002). Thus, in light of the relationship between the cardiovascular system and heat dissipation, it follows that factors determining postexercise cardiovascular status also attenuate postexercise heat loss responses, and consequently affect core temperature regulation.

2.3 Hemodynamics Postexericse

While the hemodynamic response during exercise is well characterized, there is a paucity of information about the recovery period. Mean arterial pressure decreases rapidly during the early stages of recovery as do stroke volume and cardiac output while total peripheral resistance tends to increase towards pre-exercise resting values (Carter et al., 1999). Despite increases in TPR, thoracic impedance values indicate a decrease in central blood volume during inactive recovery compared to that of active recovery modes (Carter et al., 2002; Carter et al., 1999). Studies have shown that decrements in central blood volume during the postexercise period are associated with the increase of blood in the venous system of the lower extremities in the absence of the muscle pump (Halliwill, 2001; Carter et al., 1999; Kilgour et al., 1993). Additionally, persistent muscle vasodilation (Piepoli et al., 1993) also contributes to lower central blood volume and thus reduced cardiac filling (Kilgour et al., 1993). Despite a reduction in pre-load, it has been demonstrated that greater inotropic activity during the first 5-10 minutes of recovery can occur (Kilgour et al., 1990). The combined effects of warm blood stimulating the sinoatrial node (Gorman et al., 1984), and decreased pre-load induced stretch result in an elevated heart rate. The outcome of the increased heart rate and contractility leads to a postexercise cardiac output that is actually equal to or exceeds

pre-exercise values (Kilgour et al., 1993). Although cardiac output is equal to or greater than pre-exercise values, a deficit in peripheral resistance can lead to a decrease in MAP. This postexercise decrease in MAP is now commonly referred to as postexercise hypotension. In short, postexercise hypotension is characterized by a constant drop in systemic vascular resistance that is not completely offset by increase in cardiac output (Halliwill, et al., 1996).

Blood pressure is determined by two main factors: cardiac output and the resistance of the blood vessels to blood flow (Rowell & O'Leary, 1990). This resistance is produced mainly in the arterioles and is known as peripheral vascular resistance. Peripheral vascular resistance is influenced by blood volume. Blood pressure is regulated by neural and endocrine mediated responses. Immediate short-term control of arterial blood pressure is accomplished by negative feedback systems involving a baroreflex mediated response. The parasympathetic and sympathetic branches of the autonomic nervous system sense a deformation in the blood vessel walls. They respond by returning arterial blood pressure to the established central nervous system operating point. A decrease in blood pressure stimulates the baroreceptors (baroreceptor unloading) leading to tachycardia and vasoconstriction. In contrast, an increase in blood pressure leads to a reduction in afferent stimuli from baroreceptors (baroreceptor loading) thereby initiating bradycardia and decrease vasoconstriction tone (Berne & Levy, 2001; Carter et al., 2002).

There are two types of baroreceptors, arterial baroreceptors and cardiopulmonary baroreceptors. Arterial baroreceptors are located in the carotid sinus and in the aortic arch. They alter both cardiac function and vasomotion in response to acute changes in arterial blood pressure. On the other hand, cardiopulmonary baroreceptors are found in the large veins and

in the walls of the atria of the heart (Klabunde, 2004). They play an important role in the regulation of blood volume, which determines mean arterial pressure throughout the body.

2.4 The Role of Nonthermal Factors

Nonthermal factors have been shown to play a significant role in temperature regulation during (Pawelcyzk et al., 1993; Rowell & O'Leary, 1990) and following exercise (Kenny et al., 2002; Journeay et al., 2004; Shibasaki et al., 2004; Wilson et al., 2001). These include mechanoreceptors, metaboreceptors, central command and baroreceptors. In general, muscle mechanoreceptors are stimulated by local pressure and muscle contraction. They inhibit cardiac vagal tone during the onset of exercise thereby rapidly increasing heart rate. Central command is the term given to define the higher brain functions associated with voluntary contraction of muscle. It is responsible for increasing cardiac output and heart rate during exercise. Contraction of the working muscles, known as the skeletal muscle pump, squeezes the veins, therefore facilitating the return of the blood to the heart (Carter et al., 2002). During the postexercise period there is an absence of mechanoreceptor stimulation and descending central command signals, which increases the pooling of blood in the lower extremities.

On the other hand, muscle metaboreceptors are innervated by unmyelinated fibers and are activated by chemicals released during exercise. When an increase in muscle metabolites is detected by chemosensitive afferent fibers, blood pressure is increased and blood flow to the active muscle is increased. These responses lead to the decrease in the concentration of accumulated metabolites (Rowell & O'Leary, 1990). Baroreceptors as described above work by sensing acute changes in arterial blood pressure that causes a mechanical deformation of the blood vessel walls.

2.5 The Relationship Between Postexercise Hemodynamics and Temperature Regulation

There remains little information regarding the interaction of thermoregulatory and cardiovascular input postexercise. Given the pivotal role of the cardiovascular system in heat dissipation demonstrated in recent studies (Kenny et al., 2002; Journeay et al., 2004; Shibasaki et al., 2004; Wilson et al., 2001), it is essential to describe the changes that typically occur postexercise.

The consequences of dynamic exercise include postexercise venous and muscle pooling which is further exacerbated by an upright-seated recovery posture. Thus, during inactive seated recovery pooling of blood would tend to trap heat in the previously active muscle (Piepoli et al., 1993). This effect, in conjunction with a reduction in skin blood flow and sweating, results in a time dependent transfer of heat from muscle to the core. Consequently, there is an observed prolonged elevation in core temperature (Kenny et al., 2002). It has been shown that resistance vessels in skeletal muscle remain dilated after a bout of cycling exercise and the resultant hyperemia persists well into recovery (Piepoli et al., 1993). Such pooling of blood in the lower extremities tends to reduce cardiac filling and unload baroreceptors (Halliwill et al., 2001).

As previously discussed, recent studies have shown that exercise results in a residual increase in the onset threshold for skin blood flow (Kenny et al., 2003a; Kenny et al., 2000) and sweating (Kenny et al., 2003b; Kenny et al., 2000). This effect is also relatively greater during recovery after exercise of increasing intensity (Kenny et al., 2003a; Kenny et al., 2003b). For example, Kenny et al. (2003b) observed an increase in the postexercise core temperature at which sweating occurs. Specifically, a 0.11°C, 0.22°C and 0.33°C increase in

the level of the postexercise onset threshold for sweating above no-exercise resting following light, moderate and intense exercise respectively. In addition, they noted a parallel decrease in MAP as exercise intensity increased. A similar exercise intensity dependent pattern of response was also reported in postexercise cuteneous vasodialtion (Kenny et al., 2000b).

Studies demonstrate that changes in skin blood flow and sweating during exercise can be initiated centrally but they are also subject to nonthermoregulatory controls such as central command, baroreflexes, muscle mechanoreceptors and metaboreceptors (Shibasaki et al., 2003; Pawelcyzk et al., 1993; Johnson et al., 1992; Johnson et al., 1986). At the cessation of exercise however, dramatic changes in MAP occur secondary to changes in input from central command, metaboreceptor and mechanoreceptor feedback. Thus, skin blood flow and sweating may still be subject to nonthermoregulatory baroreceptor control during exercise recovery (Carter et al., 2002). This is plausible given that evidence exists for baroreceptor modulation of skin blood flow and sweating (Crandall et al., 1996; Mack et al., 1995; Kellogg et al., 1990; Johnson et al., 1973). Additionally, the postexercise hemodynamic responses support the hypothesis that during the postexercise period there is greater activity of cardiopulmonary baroreceptors, and possibly arterial baroreceptors depending on the degree of hypotension exhibited.

2.6 Postexercise Skin Blood Flow Response

This section will focus on the control of skin blood flow and how it is modified by nonthermoregulatory factors. Cutaneous circulation is controlled by two groups of sympathetic nerves: sympathetic adrenergic vasoconstrictor nerves and sympathetic vasodilator nerves. The acral region's cutaneous circulation is controlled by sympathetic

adrenergic vasoconstrictor nervous activity, while the non-acral region's cutaneous circulation is controlled by sympathetic adrenergic vasoconstrictor nervous activity and sympathetic vasodilator nervous activity (Kenny & Johnson, 1992; Kellogg et al., 1995; Kellogg et al., 1989). It is well documented during exogenous heating at rest skin blood flow is primarily increased by active vasodilation (Rowell, 1986; Rowell, 1993). Initiation of this response is responsible for 80-95% of the elevation in skin blood flow accompanying heat stress. Furthermore, studies show that the cutaneous vasodilatory system is subject to nonthermoregulatory baroreflex modulation (Mack et al., 2001; Crandall et al., 1996; Kellogg et al., 1990; Johnson, 1986). Baroreceptor unloading has been shown to cause cutaneous vasoconstriction. Specifically, baroreceptor unloading causes a withdrawal of active cutaneous vasodilation (Kellogg et al., 1991).

Recently studies have used different recovery modes to manipulate venous pooling during the postexercise period. Thereby evaluating the role of nonthermal factors on the control of skin blood flow. It has been shown that the manipulation of postexercise hemodynamics by the application of head-down tilt (Kenny et al., 2000) and lower body positive pressure (LBPP) (Jackson et al., 2003; Johnson et al., 1981) alters cutaneous vasomotor responses. For example, Kenny (2000) used the head down tilt method to evaluate the role of baroreceptor control on postexercise threshold for forearm cutaneous vasodilatation. Head down tilt is known to increase central blood volume by promoting venous return through a reduction in the hydrostatic forces present in the upright posture. Specifically, under resting conditions at tilt angles of less than 30 degrees this technique is thought to engage the cardiopulmonary baroreceptors without changes in systemic MAP and therefore reduces the likelihood of engaging the arterial baroreceptors (Goldsmith, 1988;

Goldsmith, 1991; Goldsmith 1985; London, 1983). This study showed that reducing postexercise venous pooling by means of head-down tilt resulted in the lowering of the resting postexercise elevation in esophageal temperature for forearm cutaneous vasodilatation seen during recovery.

In 2003, Jackson and Kenny (2003) used lower body positive pressure to decrease venous pooling postexercise. They found that applying positive pressure to the lower extremities after dynamic exercise decreased the esophageal temperature threshold for cutaneous vasodilation and sweating. They proposed that baroreceptor loading status was responsible for the difference in sweating and cutaneous vascular conductance thresholds. Journeay et al. (2004) used this same technique, with an application of +45mmHg LBPP they also observed a reflex increase in skin blood flow and sweating. Furthermore an increase in the rate of core temperature decay was noted following reversal of postexercise hypotension compared to the no pressure condition. It was postulated that LBPP triggered a nonthermal baroreceptor mediated increase in heat loss responses secondary to a redistribution of blood from the lower extremities to the central circulation.

Changes in postexercise hemodynamic responses, such as an increase in stroke volume and MAP, induced by the application of LBPP or head down tilt were shown to 1) reverse the postexercise increase in the esophageal temperature at which onset of cutaneous vasodilation and sweating occurs (Jackson et al., 2003) add ref, and 2) favor the decay of esophageal temperature to pre-exercise values (Journeay et al., 2004) add ref. Acute reductions in central venous pressure delay or decrease the rise in skin blood flow during heat stress (Mack et al., 2001, Mack et al., 1995). Thus, it is plausible that a baroreceptor mediated peripheral vasoconstriction is elicited by postexercise hypotension. Thus, the

suppressive reflex on skin blood flow likely reflects a central inhibition of vasodilator outflow (Kenny et al., 2003a).

Recent studies have employed active, passive and inactive recovery modes to study the relative roles of nonthermal factors upon postexercise skin blood flow response (Carter et al., 2002, Journeay et al., 2004, Journeay et al., 2005, Shibasaki et al., 2004, Wilson et al., 2004). The principle of using different recovery modes in the study design was to differentiate between the relative roles of central command, mechanoreceptors/skeletal muscle pump, and baroreceptors in the modulation of heat loss responses. During active recovery both mechanoreceptors/skeletal muscle pump and central command are activated. A passive recovery mode is thought to include the skeletal muscle pump without the involvement of central command (Carter et al., 1999). The results from these studies showed that active and passive recoveries are effective in attenuating the fall in sweating, cutaneous vascular conductance and MAP when compared to upright-seated recovery. Specifically, it has been shown that skeletal muscle pump is the main determinate in maintaining cutaneous vascular conductance in males. It is also suggested that skin blood flow is predominantly influenced by cardiopulomonary barorecptors during upright inactive recovery. However, some studies show that arterial baroreceptors also modulate postexercise skin blood flow (Journeay et al., 2004; Journeay et al., 2005).

2.7 Postexercise Sweating Response

There are many factors, thermal and nonthermal that alter the magnitude of sweating. Sweating is initiated when acetylcholine is released from cholinergic sudomotor nerves that innervate sweat glands (Kondo et al., 2001). The exact neurological pathways responsible for

sweating are not entirely understood. Studies have clearly shown that there is a direct relationship between the magnitude of sweating and internal and mean skin temperature (Nadel et al., 1971a; Nadel et al., 1971b). The response of sweating during exercise involves nonthermal factors including central command, baroreceptors, mechanoreceptors, and metaboreceptors (Jackson and Kenny, 2003; Journeay et al., 2004; Kondo et al., 1999; Nadel et al., 1971c Shibasaki et al., 2003). This is in contrast to endogenous heat stress, in which the primary stimulus for sweating is thought to be a factor of thermal origin (Neilsen 1938).

The effects of baroreceptors on the control of sweating are controversial. Kenny et al. (2003b) observed that sweating response during upright recovery was significantly modified by exercise intensity. Specifically, as exercise intensity increased, sweating decreased. In addition, they recorded a greater postexercise hypotension as a function of exercise intensity. Given the postexercise hypotensive response, it was hypothesized that sudomotor activity is also influenced by nonthermal baroreceptor reflex adjustments postexercise. Furthermore, Jackson and Kenny (2003) showed that while the postexercise threshold for sweating was elevated compared with no-exercise control, the postexercise increase in the onset threshold for sweating was reversed with the application of LBPP. While their observations support a baroreceptor modulation of postexercise sweating, other factors such as muscle metaboreflex (94), muscle mechanoreceptors (Fu et al., 2000; Journey et al., 2004; Journeay et al 2005; Shibasaki et al., 2004; Wilson et al., 2004) can not be excluded.

In contrast, Wilson and colleagues (2003) studied the effect active and inactive recovery from exercise and its effects on cutanous vascular conductance and sweating. Subjects were placed in the supine position to minimize the effect of barorecptor unloading. This was an extention of Carter et al. (2002) work that examined the effect of sweating and

cutanous vascular conductance during active and inactive recovery in the upright-seated position. Wilson et al. study confirmed, by comparing their results to Carter and colleagues work (2002) that baroreceptors are responsible for the differences in cutaneous vascular conductance seen during upright-seated recovery when compared to supine recovery. On the other hand, the results showed that there was no difference in sweat rate. Therefore they concluded that baroreceptors are not the mechanism responsible for separation in sweat rate observed between recovery modes in the upright-seated position. Wilson et al. (2003) attributed the difference in sweat to other non-thermal factors such as central command, muscle metabo/mechanoreceptors stimulation.

Of note Carter et al. and Wilson et al. examined this response of sweating only following a 3- min and 5-min recovery period, respecively. More recently however, Journeay et al. (2004) examined the effect of sweating during extended recovery from exercise (60-min). It is well documented that significant cardiovascular and thermoregulatory adjustments occur after five minutes of recovery (Brown et al., 1993; Kilgour et al., 1993; Thoden et al., 1994), which can mask barorecetpor-mediated control of sweating. It is plausible that this is the main reason for these conflicting studies on baroreceptor control of sweating. To date, there remains conflicting information regarding the possible baroreceptor mediated influence upon sweating during the postexercise period, however there is a growing body of literature that supports this concept (35, 41, 42, 56).

2.8 Gender Differences

Most research to date has been done using male participants, or not enough female participants were included to determine gender differences. Therefore our current

understanding of post exercise thermal response contains some inherent bias. It is important that these gender differences be discussed, due to the fact that males and females differ considerably in their response to physiological stressors.

Many studies have shown that males and females differ significantly when subjected to orthostatic stress. Specifically, females are more susceptible to orthostatic intolerance then males. (Covertino, 1998; Gotshall et al., 1991; Gotshall et al., 1994; Shoemaker et al., 2001; White et al., 1996). Orthostatic intolerance is characterized by a compromised venous return, reduced stroke volume and cardiac output, which leads to a decrease in MAP. In 1988, Frey and Hoffler (Frey & Hoffler, 1988) subjected males and females to lower body positive pressure. They observed that females had greater increases in heart rate and males had a concomitant increase in peripheral resistance. Based on these findings, it was postulated that women respond to orthostatic challenges with greater vagal withdrawal, whereas men respond with sympathetic stimulation to the peripheral vasculature as their major response.

Recently Meendering et al. (2005) examined sex-related differences in hemodynamics during head up tilt maneuver in thermoneutral ambient conditions. Females were examined during their early follicular, ovulatry, and mid-luteal phases. Females exhibit more orthostatic intolerance then men, but the degree of orthostatic intolerance did not vary over the course of the menstrual cycle. Meendering et al. observed that the changes in vascular resistance appear to be similar between sexes for a given level of orthostatic stress. Consequently, they suggested that the gender differences were due to anatomical differences. Their results coincide with Fu et al., (1999) who suggested that gender differences exist because females operate on a steeper portion of Frank Starling relationship. Specifically, females have smaller and less distensible left ventricles compared to males.

In 2001 Carter et al. (Carter et al., 2001) compared the responses of women and men during 3 minutes of active and inactive recovery from dynamic exercise (Carter et al., 2001). During upright-seated recovery from dynamic exercise, women exhibited significantly greater decreases in MAP than men. Their results suggest that women are at increased risk of postexercise hypotension. In addition, women showed a greater reduction in cardiac output and did not appear to produce the same degree of compensatory vasoconstriction during recovery compared to men (Carter et al., 2001). Since Carter at al. studied recovery for only a short duration, little is known about possible differences between men and women as well as the competition between cardiovascular and thermoregulatory reflexes during extended recovery. A recent study by Journeay et al. (2005a) has shown that females appear to demonstrate a difference in the control of cutaneous vascular conductance during recovery Specifically, it was shown that during passive recovery cutaneous vascular conductance is influenced by central command whereas nonthermal influence of sweating was similar to males (Carter et al., 2002; Journeay et al., 2005a). In addition, compared to males, females demonstrated a greater magnitude of post exercise hypotension and this was accompanied by a greater elevation in postexercise threshold for cutaneous active vasodilation (Kenny et al., 2005). Thus the effect of gender differences in cardiovascular regulation requires further examination in the context of the possible consequences to post exercise heat loss.

CHAPTER 3

METHODOLOGY

3.0 Population and Sample

A) Seven healthy, physically active males volunteered and gave written consent to participate in this study. Subjects were (mean ± SE) 20 ± 0.8 years old, 182 ± 0.4 cm tall, weighed 78.3 ± 4.2 kg and their mean $\dot{V}O_2$ peak was 56.1 ± 2.6 $ml \cdot kg^{-1} \cdot min^{-1}$.

B) Seven healthy, physically active females volunteered and gave written consent to participate in this study. Subjects were (mean ± SE) Subjects were (mean ± SE) 20.7 ± 0.76 years old, 160.8 ± 12.1 cm tall, weighed 63.6 ± 5.5 kg and their mean $\dot{V}O_{2peak}$ was 42.6 ± 5.3 $ml \cdot kg^{-1} \cdot min^{-1}$

C) Seven healthy, physically active females volunteered and gave written consent to participate in this study Subjects were (mean ± SE) 21 ± 1.5 years old, 162 ± 13 cm tall, weighed 61.7 ± 5.0 kg and their mean $\dot{V}O_{2peak}$ was 43.8 ± 3.1 $ml \cdot kg^{-1} \cdot min^{-1}$.

3.1 Measurement techniques

The following readings were performed in order to study hemodynamic and thermal responses during exercise recovery.

Hemodynamic responses

Mean arterial pressure (MAP) was estimated from the integration of non-invasive recording of blood pressure at the middle digit of the left hand (Finapress 2300, Ohmeda, Madison, WI) and was situated at the third intercostals space (heart level). MAP was verified periodically throughout the protocol by auscultation. Heart rate (HR) was recorded recorded with a polar

heart rate transmitter, recorded continually, and stored on a watch nearby. Skin blood flow was estimated by using laser-Doppler velocimetery (PeriFlux System 5000, main control unit; PF5010 LDPM, Function unit; Perimed AB, Stockholm, Sweden) at the mid-anterior forearm and/or right superior portion of the trapezium. The laser-Doppler flow probes were taped to cleaned skin, in an area that do not appear by visually inspection to be overly vascular and from which consistent readings were noted (Mack, 1998). Cardiac output was estimated using the CO_2 rebreathing technique of Defares (Defares, 1958). A MedGraphics metabolic system will be used to analyze the exponential rise in $P_{ET}CO_2$ as the subject rebreaths 4% CO_2, 35% O_2 and the balance N_2. Studies have also shown that Doppler-derived aortic blood flow measurements correlate with the indirect carbon dioxide rebreathing method (Hadjis et al., 1995).

Thermal responses

Esophageal temperature was measured continuously as an indicator of central body temperature. A pediatric thermocouple probe (Mon-a-therm general purpose, Mallinckrodt Medical, St. Louis, MO) was inserted through the nares and into the esophagus to a depth of approximately one-fourth of the standing height of the subject placing the tip of the thermocouple at the level of the left atrium (Mekjavic & Rempel, 1990). Skin temperature was recorded at 11 sites (Concept Engineering, Old Saybrook, CT, USA, model FR-025-TH44018-6). These are commercially available heat flow disks that can read both heat loss and skin temperature. The area-weighted mean skin temperature ($\bar{T}_{sk}$) was calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%,

anterior calf 9.5%, and posterior calf 9.5% (Hardy and DuBois, 1934). Skin temperature was collected and digitized (Hewlett-Packard data acquisition module, model 3497A) at 5-s intervals, displayed graphically in real time and stored on hard disk (model PC-312, 9000, Hewlett-Packard). Sweat rate was measured using a 3.6 cm^2 ventilated capsule placed over the medial inferior aspect of the trapezius muscle.

3.2 General overview of protocols

A) Seven male subjects performed a total of 3 experimental trials carried out in random order. After instrumentation subjects remained resting for 10-min during which baseline measures are recorded. Subjects were then required to perform one of 3 experimental protocols. These are: 1) 60 min in the upright-seated (URS) posture followed by 60 min in the 15° head-down tilt position (HDT); 2) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_{2peak}$ followed by 60 min recovery in the URS posture; or 3) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_{2peak}$ followed by 60 min recovery in the 15° HDT position. At the end of each experiment, local skin temperature was raised by 42 °C using a heating element. CVC data are presented as a percentage of maximal CVC as determined by local heating (%CVC-peak).

B) The same experimental protocol was repeated in study 2 using females only (n=7).

C) Seven females subjects performed a total of 3 experimental trials carried out in random order. After preparation of the subjects, they remained seated for 10-min while baseline measurements were recorded. This was followed by 15-minutes of exercise on a cycle ergometer at one of the following intensities: 1) 85% $\dot{V}O_{2peak}$, 2) 75% $\dot{V}O_{2peak}$, or 3) 55%

$\dot{V}O_{2\,peak}$. Immediately following the cessation of exercise the subjects were placed in the upright-seated position for one hour. At the end of each experiment, local skin temperature was raised by 42 °C using a heating element. CVC data are presented as a percentage of maximal CVC as determined by local heating (%CVC-peak).

PART TWO: RESULTS OF THE THESIS

15° Head-down tilt reduces postexercise esophageal temperature recovery time while attenuating the reduction in heat loss responses.

Natalie H. McInnis[1], W. Shane Journeay[1,2], Ollie Jay[1], Emily E. Leclair[1] and Glen P. Kenny[1]

[1]Laboratory of Human Bioenergetics and Environmental Physiology, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario, Canada K1N 6N5
and
[2]Toxicology Graduate Program and Department of Veterinary Biomedical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK, S7N 5B4

Address for correspondence and reprint requests:

✉ G.P. Kenny
University of Ottawa
School of Human Kinetics
125 University, Montpetit Hall
Room 367
Ottawa, Ontario, Canada
PO Box 450 Station A
K1N 6N5
(613) 562-5800 ext. 4282
(613) 562-5149 (fax)
e-mail: gkenny@uottawa.ca

Running head: Nonthermal influences on postexercise heat loss

ABSTRACT

The following study examined the effect of 15° head-down tilt (HDT) on postexercise heat loss and hemoynamic responses. We tested the hypothesis that recovery from dynamic exercise in the HDT position would attenuate the reduction in the heat loss responses of cutaneous vascular conductance (CVC) and sweating relative to upright-seated (URS) recovery in association with an augmented hemodynamic response and an increased rate of core temperature decay. Seven male subjects performed the following three experimental protocols: 1) 60 min in the upright-seated (URS) posture followed by 60 min in the 15° head-down tilt position (HDT); 2) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$ peak followed by 60 min recovery in the URS posture; or 3) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$peak followed by 60 min recovery in the 15° HDT position. Mean skin temperature (T_{sk}), esophageal temperature (T_{es}), skin blood flow (SkBF), sweat rate, cardiac output (CO), stroke volume (SV), heart rate (HR), total peripheral resistance (TPR), and mean arterial pressure (MAP) were recorded at baseline, end exercise, 2, 5, 8, 12, 15, 20 and every 5-min until end of recovery (60-min). Without preceding exercise, HDT decreased HR and increased SV ($p \leq 0.05$). During recovery after exercise, a significantly greater MAP, SV, CVC and sweat rate and a significantly lower HR were found with HDT in comparison to URS posture ($p \leq 0.05$). Subsequently a significantly lower T_{es} was observed with HDT after 15-min of recovery onwards ($p \leq 0.05$). At the end of 60-min recovery, T_{es} remained significantly elevated above baseline with URS ($p \leq 0.05$), however T_{es} returned to baseline with HDT. In conclusion, extended recovery from dynamic exercise in the 15° HDT

position attenuates the reduction in CVC and sweating thereby significantly increasing the rate of esophageal temperature decay compared to recovery in the URS posture.

Keywords: skin blood flow, esophageal temperature, nonthermal influences, baroreceptor.

INTRODUCTION

Previous studies have shown that under thermoneutral conditions, core body temperature remains elevated after an acute bout of dynamic exercise (3, 17, 49, 50, 97, 102). Additionally, sweating (53), skin blood flow and skin temperature (53, 102) return to pre-exercise values despite the persistent elevation in core temperature.

Research has been conducted to examine the possible factors contributing to altered temperature regulation postexercise. Studies employing recovery modes have attempted to elucidate the relative roles of nonthermal factors such as central command, mechanoreceptors and baroreceptors in the modulation of heat loss responses during exercise recovery (7, 42, 90, 104). Collectively, the recovery mode studies have determined that attenuating the baroreceptor unloading effect associated with upright inactive recovery, through active or passive recovery, preserves skin blood flow. Central command was did not influence the skin blood flow response. Sweating however can be influenced by central command and mechanoreceptors, albeit the role for baroreceptors remains unresolved.

During upright inactive recovery, there is a growing body of evidence in support of a possible relationship between hemodynamic changes postexercise and heat loss responses. Journeay et al. (41) specifically examined this issue by manipulating postexercise hemodynamics using lower body pressure. They observed a reflex increase in skin blood flow, sweating and heat flux with application of +45mmHg lower body positive pressure (LBPP) in the URS posture. Furthermore an increase in the rate of core temperature decay was noted following reversal of postexercise hypotension by application of positive pressure to the lower extremities. This effect was not seen under lower body negative pressure or no pressure conditions. It was postulated that LBPP triggered a nonthermal baroreceptor

mediated increase in heat loss responses secondary to a redistribution of blood from the lower extremities to the central circulation.

LBPP produces its effects via increased barometric pressure around the lower extremities. This leads to enhanced microvascular compression in the tissues and produces a pressure gradient which tends to increase central blood volume. With the application of sufficient barometric pressure to the lower extremities, this technique may activate both cardiopulmonary and arterial baroreceptors (19). For example, a study showed that application of +45mmHg of LBPP postexercise engaged both the cardiopulmonary and arterial baroreceptors (41). Also it has been suggested that LBPP may activate mechanoreceptors particularly at pressures greater than 20mmHg (19), while others have ruled out mechanoreceptor activation during LBPP (80). One possible limitation of the LBPP technique in thermoregulatory studies is that possible convective air flow to the lower extremities may increase heat loss from the lower extremities. Furthermore, we have previously observed an enhanced rate of core temperature decay with application of LBPP (41), however this may not be a convenient cooling strategy in the treatment of hyperthermic individuals due to the time associated with transfer to the pressure box.

In contrast, head-down tilt (HDT) is known to increase central blood volume by promoting venous return through a reduction in the hydrostatic forces present in the upright posture. Specifically, under resting conditions at tilt angles of less than 30 degrees this technique is thought to engage the cardiopulmonary baroreceptors without changes in systemic MAP and therefore reduces the likelihood of engaging the arterial baroreceptors (19, 21-23, 65, 75). Thus, HDT is another method by which to manipulate postexercise hemodynamic responses and allows for the attenuation of the baroreceptor unloading effect of

upright exercise recovery. Additionally, HDT enables us to intervene immediately postexercise and remove the possible effects of mechanoreceptor activation and/or convective air flow on the lower extremities associated with LBPP.

Thus, the purpose of this experiment was to examine the effect of HDT, and by extension the role of baroreceptors, on prolonged postexercise heat loss and hemodynamic responses. We tested the hypothesis that recovery from dynamic exercise in the HDT position would augment the heat loss responses of cutaneous vascular conductance and sweating relative to upright recovery in association with an augmented hemodynamic response and increased rate of esophageal temperature decay.

METHODS

Subjects

Seven healthy, physically active males volunteered and gave written consent to participate in this study. The study was approved by the Research Ethics Board at the University of Ottawa. Five to seven days before the experiments, peak oxygen consumption ($\dot{V}O_2$ peak) was measured during a progressive cycle ergometer protocol that required the participant to cycle at a cadence of 80 revolutions per minute while the ergometer resistance was increased at 0.5 Kp every 2-mins. The $\dot{V}O_2$ peak data were used to select the submaximal workload for the experimental exercise phase of the study. Subjects were (mean ± SE) 20 ± 0.8 years old, 182 ± 0.4 cm tall, weighed 78.3 ± 4.2 kg and their mean $\dot{V}O_2$ peak was 56.1 ± 2.6 $ml \cdot kg^{-1} \cdot min^{-1}$.

Measurements

Heart rate (HR) was monitored using a Polar coded transmitter, recorded continuously and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy, Finland). Mean arterial pressure (MAP) was estimated from the integration of a non-invasive recording of blood pressure at the middle digit of the left hand (Finapres 2300, Ohmeda, Madison, WI, USA) fixed at heart level (the third intercostal space). The Finapres system is based on the Penaz volume clamp method (dynamic unloaded arterial wall principle). MAP was verified periodically throughout the protocol by auscultation.

Pulmonary $\dot{V}O_2$ was estimated using a metabolic cart (model CPX/D, Medgraphics, St. Paul, MN) during $\dot{V}O_2$ peak assessment preceding the experimental trials. Cardiac output (CO) was estimated using the CPX/D computerized version of the CO_2-rebreathing technique of Defares (14). It has been shown that Doppler-derived aortic blood flow (CO) measurements correlate well with the indirect carbon dioxide rebreathing method (26). The Defares method has also been shown to work well in "unsteady state" testing (33). Stroke volume (SV) was calculated as CO/HR. Total peripheral resistance (TPR) was calculated as MAP/CO.

Skin blood flow (SkBF) was estimated using laser-Doppler velocimetry (PeriFlux System 5000, Main control unit; PF5010 LDPM, Function unit; Perimed AB, Stockholm, Sweden) at the left mid-anterior forearm. The laser-Doppler flow probe (PR 401 Angled Probe, Perimed AB, Stockholm, Sweden) was taped to cleaned skin, in an area which did not appear by visual inspection to be overly vascular and from which consistent readings were noted (67). Cutaneous vascular conductance (CVC) was calculated as the ratio of laser-Doppler flow to MAP. At the end of the experiment, local skin temperature was raised to 42°C using a heating element (PF 5020 Temperature Unit, Perimed AB) that housed the laser-

Doppler flow probe,until peak CVC was measured (~30 min)(96). CVC-peak was determined as a sustained elevated plateau in local SkBF. CVC data are presented as a percentage of maximal CVC as determined by local heating (%CVC-peak). All SkBF measures were taken in the period preceding rebreathing to avoid causing fluctuations in SkBF data at each time point. SkBF measures were recorded from the left mid-anterior forearm such that the arm was level with the heart.

Sweat rate was measured using a 5.0 cm^2 ventilated capsule placed over the medial inferior aspect of the trapezius muscle. Anhydrous compressed air was passed through the capsule and over the skin surface (Brooks 5850, Mass Flow Controller, Emerson electric, Hetfield, Pa, USA). The vapour density of the effluent air was calculated from the relative humidity and temperature measured using the Omega HX93 humidity and temperature sensor (Omega Engineering, Stanford, CT, USA). Sweat rate was defined as the product of the difference in water content between effluent and influent air and the flow rate. The flow rate through the capsule was 1.0 $L \cdot min^{-1}$. The sweat rate value was adjusted for skin surface area under the capsule and expressed in $mg \bullet min^{-1} \bullet cm^{-2}$.

Central body temperature (esophageal temperature, T_{es}) was monitored continuously using a paediatric esophageal temperature probe (Mon-a-therm®, Mallinckrodt Medical, St-Louis, USA) inserted through the nares to a depth one-fourth of the standing height of the subject, whereby the tip of the thermocouple is estimated to be at the level of the left atrium (71). Skin temperature was recorded at 11 sites using 0.3 mm diameter T-type thermocouples integrated into eat-flow sensors (Concept Engineering, Old Saybrook, CT, USA, model FR-025-TH44018-6). The area-weighted mean skin temperature ($\bar{T}_{sk}$) and heat flux ($\bar{H}F_{sk}$) were

calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9.5%, and posterior calf 9.5% (31). Temperature data were collected and digitized (Hewlett-Packard data acquisition module, model 3497A) at 5-s intervals, displayed graphically in real time and stored on hard disk (Hewlett-Packard, model PC-312, 9000).

The 15° head down tilt position was measured using the Unitek Magnetic Polycast® Protractor; model number 3310-036 (Polytech, Empire Level Manufacturing Corporation, Milwaukee, WI).

Experimental protocol

Each subject performed a total of 3 experimental trials carried out in random order. Experiments were separated by a minimum of 48 h during which subjects were instructed to avoid physical activity and excessive stressors such as exposure to hot or cold temperatures, particularly during the period between awakening and experimentation and during transit from home to the laboratory. Trials were performed at the same time of day for each subject to avoid circadian variation in skin and esophageal temperatures. Subjects were asked to fast at least 4 h prior to experimentation, and water ingestion was permitted *ad libitum* during this time. Upon arrival at the laboratory, subjects clothed in shorts and athletic shoes were fitted with the appropriate instruments. All experimental trials were performed at an ambient temperature of 24.0 ± 0.5°C and a relative humidity of 45%.

After instrumentation subjects remained resting for 10-min during which baseline measures were recorded. Subjects were then required to perform one of 3 experimental protocols. These were: 1) 60 min in the upright-seated (URS) posture followed by 60 min in the 15° head-down tilt position (HDT); 2) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$ peak followed by 60 min recovery in the URS posture; or 3) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$ peak followed by 60 min recovery in the 15° HDT position.

At the end of each experiment, peak CVC was determined using a local heating protocol as described above.

Statistical Analyses

A two-way ANOVA with repeated measures was used to analyze the data using the repeated factors of postexercise recovery time (levels: 2, 5, 8, 12, 15 and every 5 min until 60-min) and recovery mode (levels: 15° head-down tilt (HDT) and upright-seated recovery (URS)). The dependent variables employed were the changes from pre-exercise or baseline rest in T_{es}, $\bar{T}_{sk}$, $\bar{H}F_{sk}$, CO, HR, %CVC-peak, MAP, TPR, SV, and sweat rate. All values represent the means and standard deviation for seven subjects. The exercise and no-exercise sessions were analyzed separately to isolate the effect of recovery mode. For ANOVA main effects, Huynh-Feldt corrected statistics are reported where the assumption of sphericity was not met. Pair-wise comparisons were performed using paired sample t-tests. The level of significance was set at an alpha level of 0.05. All analyses were performed using the statistical software package SPSS 12.0 for Windows (SPSS Inc. Chicago, IL, USA).

RESULTS

Esophageal temperature response

Exercise condition. The change in T_{es} at the end of exercise relative to pre-exercise rest was not significantly different between the URS and HDT trials ($p=0.235$), with T_{es} elevated by 1.28°C (SD 0.29) preceding URS, and by 1.18°C (SD 0.25) preceding HDT. During recovery, change in T_{es} from pre-exercise rest became less with postexercise recovery time ($F_{(1.9, 9.4)}=37.0$, $p<0.001$) and was influenced by recovery mode ($F_{(1,5)}=10.1$, $p=0.007$). The interaction between recovery mode and postexercise recovery time for change in T_{es} ($F_{(2.8,14.1)}=4.7$, $p=0.019$) is demonstrated by the significantly lower elevation from pre-exercise rest in T_{es} during HDT in comparison to URS ($p\leq0.05$) after 15-min of postexercise recovery and for the remainder of the 60-min recovery period (Figure 1A). Esophageal temperature remained significantly ($p=0.006$) elevated by 0.22°C (SD 0.12) above pre-exercise rest after 60-min of URS recovery, but was 0.15°C (SD 0.08) *below* pre-exercise rest after 60-min of HDT recovery.

No-exercise condition. Without preceding exercise, change in T_{es} relative to resting values did not significantly change with time ($F_{(2.1, 10.7)}=1.9$, $p=0.198$) nor recovery mode ($F_{(1,5)}=4.7$, $p=0.082$), however a significant interaction was apparent between time and recovery mode ($F_{(1.8, 9.1)}=0.041$) as demonstrated by a small (~0.1°C) but significantly ($p\leq0.05$) lower T_{es} during HDT in comparison to URS between 15-min and 30-min.

Skin temperature, dry heat loss, sweating and %CVC-peak

Exercise condition. Following exercise, the elevations in mean skin temperature from pre-exercise rest became lower as postexercise recovery time progressed ($F_{(1,6)}$=25.8, p=0.002), with a trend for the influence of recovery mode ($F_{(1,6)}$=5.3, p=0.061). The interaction between mode and time ($F_{(2.8, 16.8)}$=5.8, p=0.007) is evidenced by a significantly lower $\bar{T}_{sk}$ after 12-min of HDT recovery and for the remainder of the 60-min recovery period ($p \leq 0.05$). At the end of the postexercise period, $\bar{T}_{sk}$ was 0.49°C (SD 0.53) lower after HDT in comparison to URS. Similarly, the elevations in $\bar{H}F_{sk}$ from pre-exercise rest became lower with postexercise recovery time ($F_{(1.6, 9.4)}$=168.9, p<0.001), however no differences were observed between recovery modes ($F_{(1,6)}$=1.2, p=0.316). Sweat rate was significantly elevated above pre-resting values prior to HDT and URS recovery trials ($p \leq 0.05$) and then became reduced throughout postexercise recovery ($F_{(5.3, 31.8)}$=554.6, p<0.001). Furthermore, sweat rate was influenced by recovery mode ($F_{(1,6)}$=20.3, p=0.004), with a significantly greater sweat rate observed during HDT recovery relative to URS between 8-min and 45-min postexercise ($p \leq 0.05$) (Figure 1B). The elevation in %CVC-peak above pre-exercise rest following exercise prior to both HDT and URS recovery trials became lower with postexercise recovery time ($F_{(2.6, 13.2)}$=17.6, p<0.001). Furthermore %CVC-peak was influenced by recovery mode ($F_{(1,5)}$=49.4, p=0.001) with significantly greater values observed throughout the 60-min postexercise recovery period in the HDT position in comparison to URS ($p \leq 0.05$) (Figure 1C).

No-exercise condition. Without preceding exercise, changes in T_{sk}, HF_{sk}, %CVC-peak and sweat rate relative to resting values were not influenced by time or recovery mode ($p > 0.05$).

Hemodynamic responses

All hemodynamics data for all conditions are summarized in Table 1.

Exercise condition. The elevations in CO following exercise of 15.8 L•min^{-1} (SD 2.9) prior to HDT and 16.5 L•min^{-1} (SD 5.1) prior to URS were not significantly different ($p=0.818$). These elevations from rest in CO became lower during postexercise recovery ($F_{(2.7, 13.7)}=151.1$, $p<0.001$), but were not influenced by recovery mode ($F_{(1,5)}=0.005$, $p=0.948$) (Figure 2A). The elevations in SV following exercise of 46.6 ml (SD 26.0) prior to HDT and 51.2 ml (SD 34.5) prior to URS were not significantly different ($p=0.852$). However these elevations from rest in SV became lower with postexercise recovery time ($F_{(3.2, 15.8)}=27.3$, $p<0.001$) and were influenced by recovery mode ($F_{(1,5)}=12.4$, $p=0.017$) with a significantly greater SV observed during HDT recovery throughout the 60-min postexercise recovery period ($p\leq0.05$) (Figure 2B). Following exercise, no significant difference was observed between the elevation in heart rate of 99.1 beats•min^{-1} (SD 11.3) prior to HDT and 97.1 beats•min^{-1} (SD 12.3) prior to URS recovery ($p=0.726$). Heart rate elevations above rest became lower with postexercise recovery time ($F_{(3.8, 23.0)}=297.9$, $p<0.001$) and was influenced by recovery mode ($F_{(1,6)}=34.2$, $p=0.001$) with a significantly lower heart rate elevation observed during HDT relative to URS throughout the 60-min postexercise recovery period ($p\leq0.05$) (Figure 2C). The changes in mean arterial pressure from pre-exercise rest following exercise prior to recovery were not significantly different between HDT and URS ($p=0.376$). However during postexercise recovery, change in MAP was different between recovery modes ($F_{(1,5)}=27.1$, $p=0.003$), and influenced by recovery time ($F_{(2.8, 14.0)}=42.0$, $p=0.001$) with a significantly higher MAP observed during HDT between 2-min and 50-min of postexercise

recovery ($p \leq 0.05$) (Figure 3A). The changes in TPR from rest following exercise of -10.4 $mmHg \bullet L^{-1} \bullet min^{-1}$ (SD 1.7) prior to HDT and -10.5 $mmHg \bullet L^{-1} \bullet min^{-1}$ (SD 2.5) prior to URS were not significantly different ($p=0.834$). However, change in TPR from pre-exercise rest became less throughout postexercise recovery ($F_{(5.2, 25.8)}=18.0$, $p<0.001$) but was not influenced by recovery mode ($F_{(1,5)}=2.2$, $p=0.200$) (Figure 3B).

No-exercise condition. Without preceding exercise, changes in CO, MAP and TPR relative to resting values were not influenced by time or recovery mode ($p>0.05$). The change in HR from resting values was significantly different between recovery modes ($F_{(1,6)}=21.2$, $p=0.004$). Heart rate was not influenced by postexercise recovery time ($F_{(2.0, 12.1)}=2.7$, $p=0.110$) however a significantly lower HR during HDT in comparison to URS was observed throughout postexercise recovery ($p \leq 0.05$). A significant difference between recovery modes was also seen for changes in SV from resting values ($F_{(1,5)}=6.0$, $p=0.050$), with SV greater during HDT relative to URS between 2-min and 50-min of postexercise recovery ($p \leq 0.05$).

Sweat rate and %CVC-peak as a function of esophageal temperature

The change of sweat rate (Figure 4) and cutaneous vascular conductance (Figure 5) as a function of esophageal temperature following exercise show that following exercise the HDT intervention causes a significantly greater %CVC-peak after 5-min of postexercise recovery ($p \leq 0.05$) and a significantly greater sweat rate after 15-min postexercise recovery. The combination of these increased heat loss responses contribute to a significantly lower esophageal temperature after 15-min recovery in the HDT position ($p \leq 0.05$).

DISCUSSION

The most important observation from this study is that the manipulation of postexercise hemodynamics using HDT attenuates the fall of MAP, CVC and sweat rate relative to the upright-seated (URS) posture. The augmented MAP and heat loss responses in the HDT condition were also associated with an enhanced rate of esophageal temperature decay compared to the URS recovery posture. Thus, HDT is an effective technique to attenuate the reduction in heat loss responses and MAP in individuals recovering from dynamic exercise. These findings support previous work that employed LBPP to alter postexercise hemodynamics and heat loss responses (41) and further underscores a baroreceptor mediated attenuation in heat loss responses during URS exercise recovery.

Our observation of an increased rate of esophageal temperature decay in the HDT position is consistent with the elevated heat loss responses of CVC and sweating throughout recovery. In recent studies examining postexercise heat loss responses, it has been shown that there is a significant nonthermal contribution to the control of these responses. Studies employing recovery modes have attempted to elucidate the relative roles of nonthermal factors such as central command, mechanoreceptors and baroreceptors in the modulation of heat loss responses during exercise recovery (7, 42, 90, 104). In *toto* these experiments concluded that during upright inactive recovery CVC is influenced predominantly by cardiopulmonary baroreceptors. Two studies exist however, in males (42) and females (43) where different levels of MAP were observed between recovery modes and therefore the role of the arterial baroreceptor population in heat loss response modulation cannot be ignored. Additional evidence also exists that attenuating the cardiopulmonary and arterial baroreceptor

unloading effect associated with exercise recovery helps preserve the response of CVC (35, 41). Our present study differs from previous work in that we have studied the effect of HDT on prolonged recovery responses, whereas others have employed recovery modes (7, 42, 90, 104) or lower body pressure (41). The present study also supports a cardiopulmonary and arterial baroreceptor mediated influence on postexercise CVC. This is supported by the fact that the cutaneous circulation is on the efferent arm of the baroreflex (13, 38). In our no-exercise condition, 15 degrees of HDT engaged the cardiopulmonary baroreflex as indicated by reflex bradycardia, an increase in SV and no change in MAP. However, we noted a difference in MAP between HDT and the URS posture postexercise. Thus, while 15 degrees of HDT may isolate the cardiopulmonary baroreceptors under resting conditions, we observed that during exercise recovery HDT leads to an altered MAP response as compared to URS recovery posture. This means that between postexercise recovery positions there are different degrees of arterial baroreceptor loading and thus we cannot rule out a possible role for the arterial baroreceptors in the observed CVC response.

The above mentioned recovery mode studies demonstrated that sweat rate during postexercise recovery can be modulated by central command, mechanoreceptors and possibly baroreceptors. Under resting conditions, some researchers did not observe decreases in sweating with baroreceptor unloading (98, 105) while others have observed a reduction in the thermosensitivity with baroreceptor unloading at rest (92) and during exercise (40, 69). There remains conflicting information regarding the possible baroreceptor influence on sweating during the postexercise period. Our data support a role for cardiopulmonary and/or arterial baroreceptors in the modulations of sweat rate as inferred by the hemodynamic responses to HDT. Under conditions of LBPP there may be confounding influences on sweat rate such as

air currents in the chamber or mechanoreceptor activation secondary to increased atmospheric pressure around the limbs. In our present study we employed the HDT technique thereby removing such possible confounders. We observed that when subjects recovered in the HDT position sweat rate declined more slowly than recovery in the URS posture suggesting that baroreceptors are likely involved in the response. In contrast to our previous work whereby applying LBPP stimulated sweat rate after restoring MAP (41), this study commenced the HDT intervention immediately postexercise. A study by Wilson et al. (104) concluded that factors other than thermal or baroreceptor loading status contribute to sweat rate during exercise recovery, however their observations were limited to 5-min postexercise in the supine posture. We first noted a significant difference after 5-min of recovery. Thus, it is possible that a role for baoreceptors in the modulation of postexercise sweat rate might be masked in the initial 5-min period and become more apparent later in recovery. While speculative, our data support this possibility.

HDT tends to cause a shifting of blood headward by counteracting the hydrostatic pooling of blood seen in the URS posture (75). URS inactive recovery is thought to aid in the accumulation of blood in the venous system of the lower extremities in the absence of the muscle pump (6, 27, 61). This hemodynamic consequence may also contribute to postexercise hypotension (27). Our data indicate that without an exercise treatment, there were no significant effects on hemodynamic variables other than a decrease in HR and a concomitant increase in SV in the HDT recovery position relative to the URS posture. This hemodynamic response of SV is consistent with previous observations of resting subjects in the HDT position (23, 65, 75). However, these studies did not show a decrease in heart rate. The difference in our observations may be due to the fact that previous studies compared HDT to

the supine posture and not the URS posture. Conversely, after exercise we observed significant differences in SV, HR, and MAP between HDT and URS subjects. This supports the contention that HDT reduced the baroreceptor unloading effect of exercise recovery, thus underscoring the significant effect of HDT when performed following dynamic exercise.

Our observation of an enhanced rate of esophageal temperature decay in the HDT condition is significant. While recovery mode studies have observed augmented heat loss responses, they have not reported significant changes in core temperature even when performed up to 20-min postexercise (42, 43, 90). Conversely, application of +45mmHg LBPP was effective in increasing the rate of esophageal temperature decline in URS subjects postexercise (41). The present study also reported a greater decrement in esophageal temperature when HDT was applied postexercise. Moreover, HDT may have some practical significance as it is technically easier to perform than transferring subjects to a lower body pressure chamber. A logical extension to this study for future work includes the examination of the responses of severely hyperthermic individuals (i.e. core temperature >39.5°C) to the HDT intervention. This will elucidate the effectiveness of HDT as potentially a more practical alternative for the active cooling methods currently recommended.

We conclude that during recovery from dynamic exercise, heat loss responses are compromised in the URS posture secondary to nonthermal baroreceptors influences. Specifically, application of HDT attenuates the reduction in CVC, sweat rate and MAP typically observed postexercise. The augmented heat loss responses observed under the HDT position also resulted in an increased rate of esophageal temperature decay relative to the URS recovery posture. HDT may therefore be an effective means by which to promote heat

loss in hyperthermic individuals postexercise and provides many technical advantages over LBPP.

ACKNOWLEDGEMENTS

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TABLE LEGEND

Table 1. Mean relative changes from baseline resting in Cardiac Output (CO), Stroke Volume (SV), Heart Rate (HR), Mean Arterial Pressure (MAP) and Total Peripheral Resistance (TPR) during the control trials preceded by no exercise during the control trials preceded by no exercise.

FIGURE LEGENDS

Figure 1. Effect of URS recovery posture (□) and recovery in a 15° HDT recovery position (Δ) following 15-min of cycle ergometry at 75% $\dot{V}O_2$ peak on esophageal temperature (A, top), sweat rate (B, middle), cutaneous vascular conductance (C, bottom). * denotes significant difference between URS recovery posture.

Figure 2. Effect of URS recovery posture (□) and the 15° HDT recovery position (Δ) following 15-min of cycle ergometry at 75% $\dot{V}O_2$ peak on cardiac output (A, top), stroke volume (B, middle), and heart rate (C, bottom). * denotes significant difference between URS recovery.

Figure 3. Effect of URS recovery posture (□) and recovery in a 15° HDT recovery position (Δ) following 15-min of cycle ergometry at 75% $\dot{V}O_2$ peak on total peripheral resistance (A, top) and mean arterial pressure (B, bottom). * denotes significant difference between URS recovery.

Figure 4. The relationship of esophageal temperature with sweat rate during recovery from 15-min of cycle ergometry at 75% $\dot{V}O_2$ peak in the URS posture (filled symbols) and 15° HDT position (clear symbols). A significant difference between recovery positions at a given time point is denoted by an asterisk (*) for sweat rate and a cross (†) for esophageal temperature.

Figure 5. The relationship of esophageal temperature with cutaneous vascular conductance during recovery from 15-min of cycle ergometry at 75% $\dot{V}O_2$ peak in the URS posture (filled symbols) and 15° HDT position (clear symbols). A significant difference between recovery positions at a given time point is denoted by an asterisk (*) for cutaneous vascular conductance and a cross (†) for esophageal temperature.

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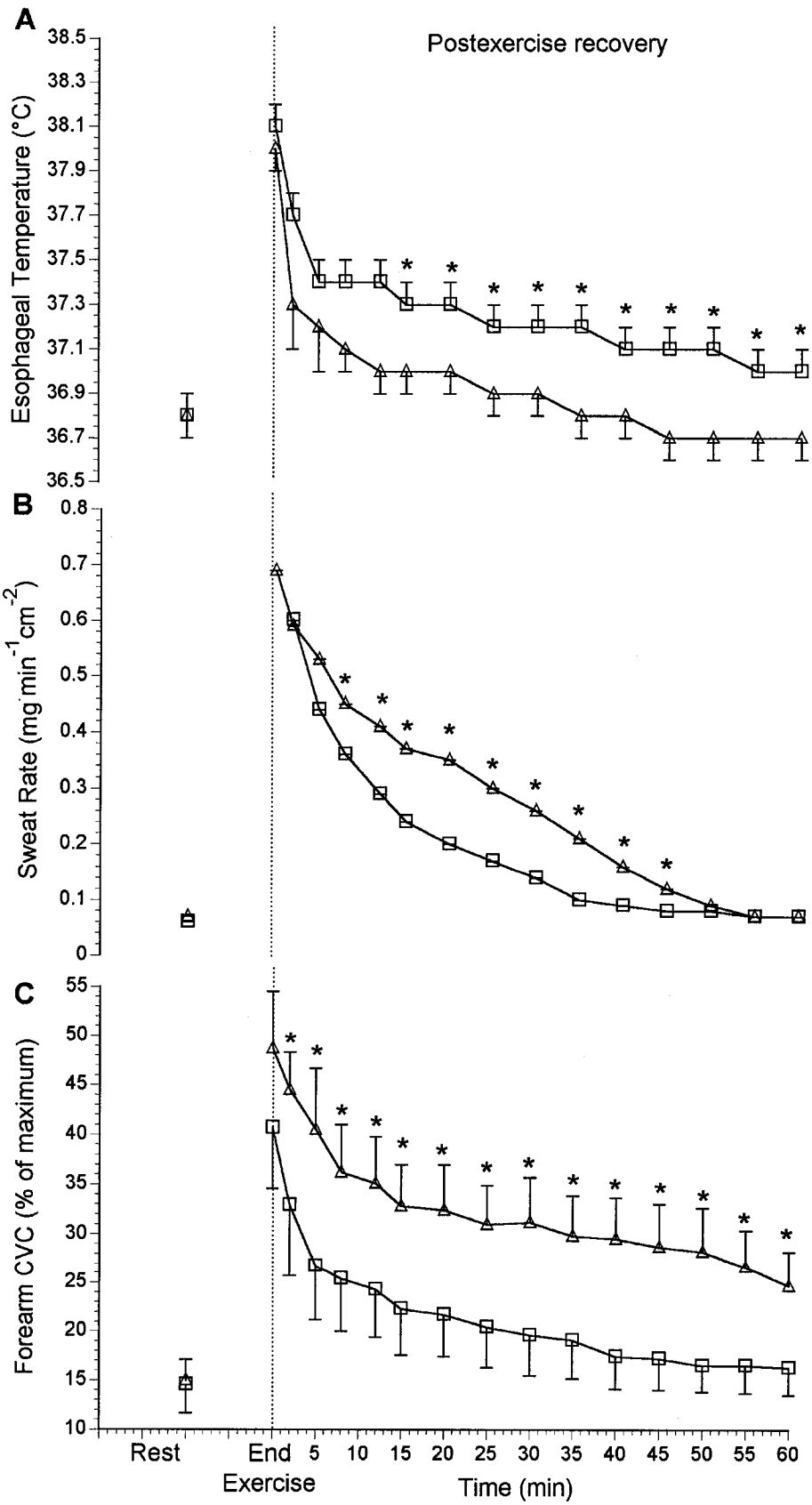
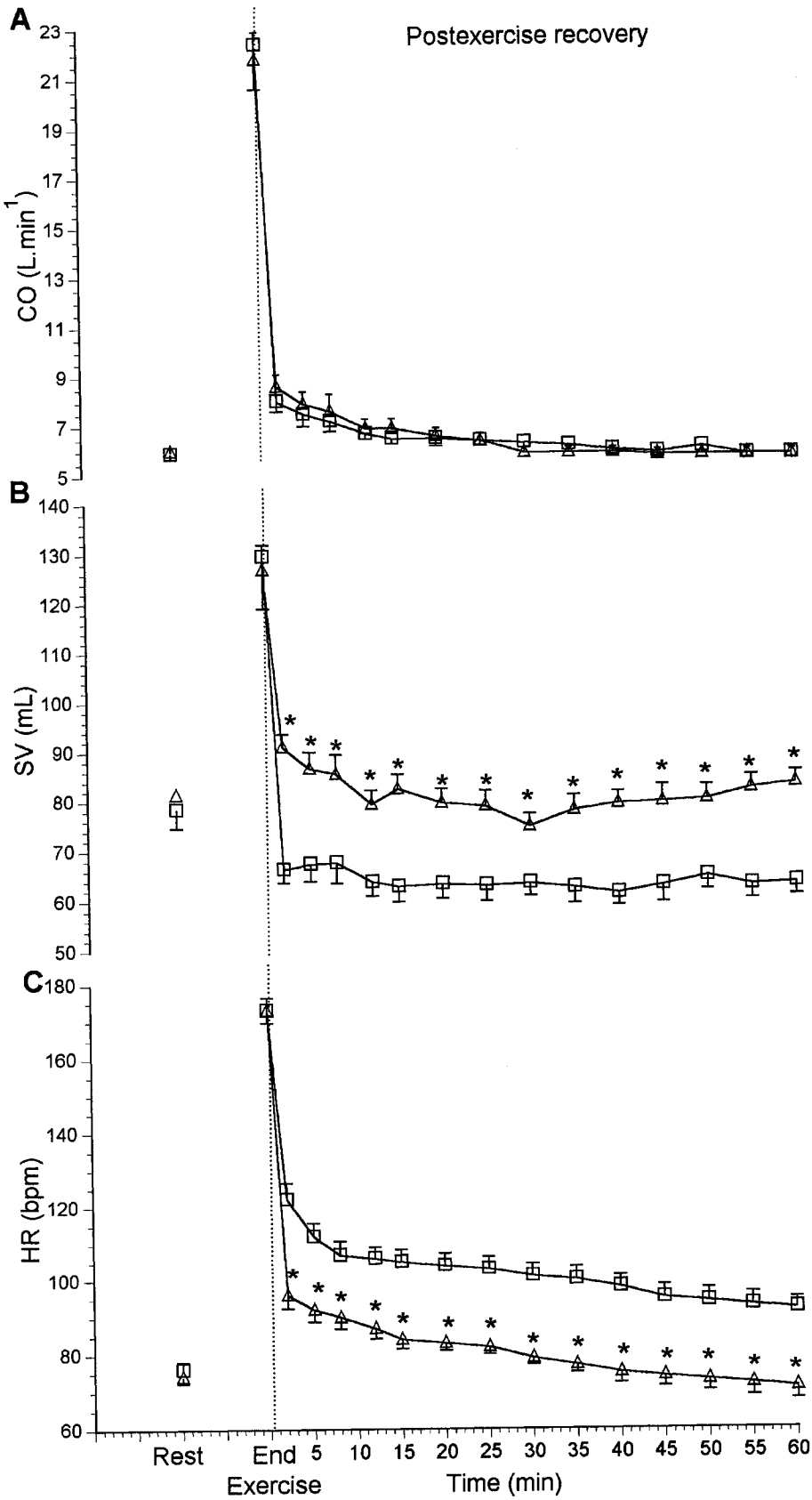
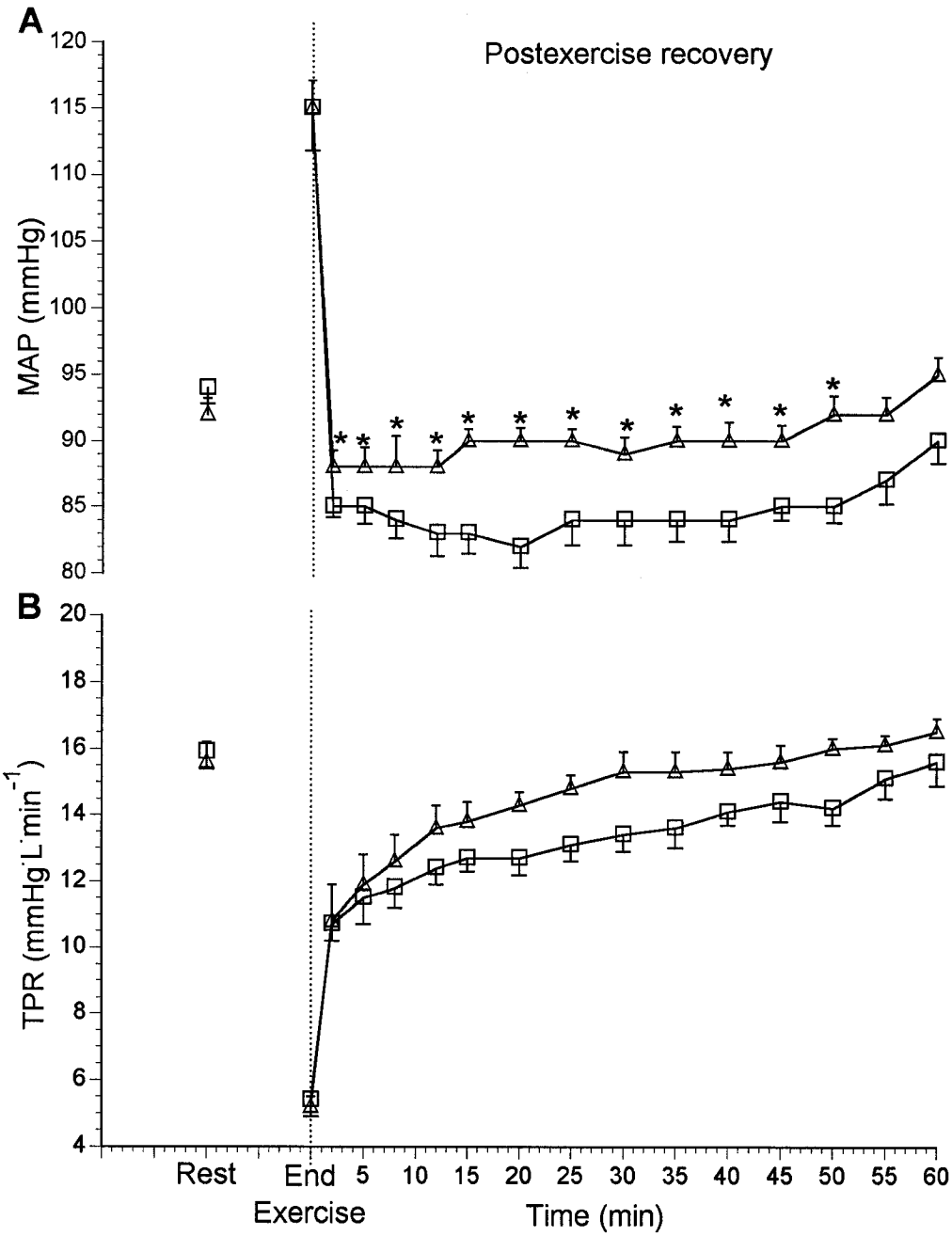
Table 1. Mean relative changes from baseline in Cardiac Output (CO), Stroke Volume (SV), Heart Rate (HR), Mean Arterial Pressure (MAP) and Total Peripheral Resistance (TPR) during the control trials preceded by no exercise.

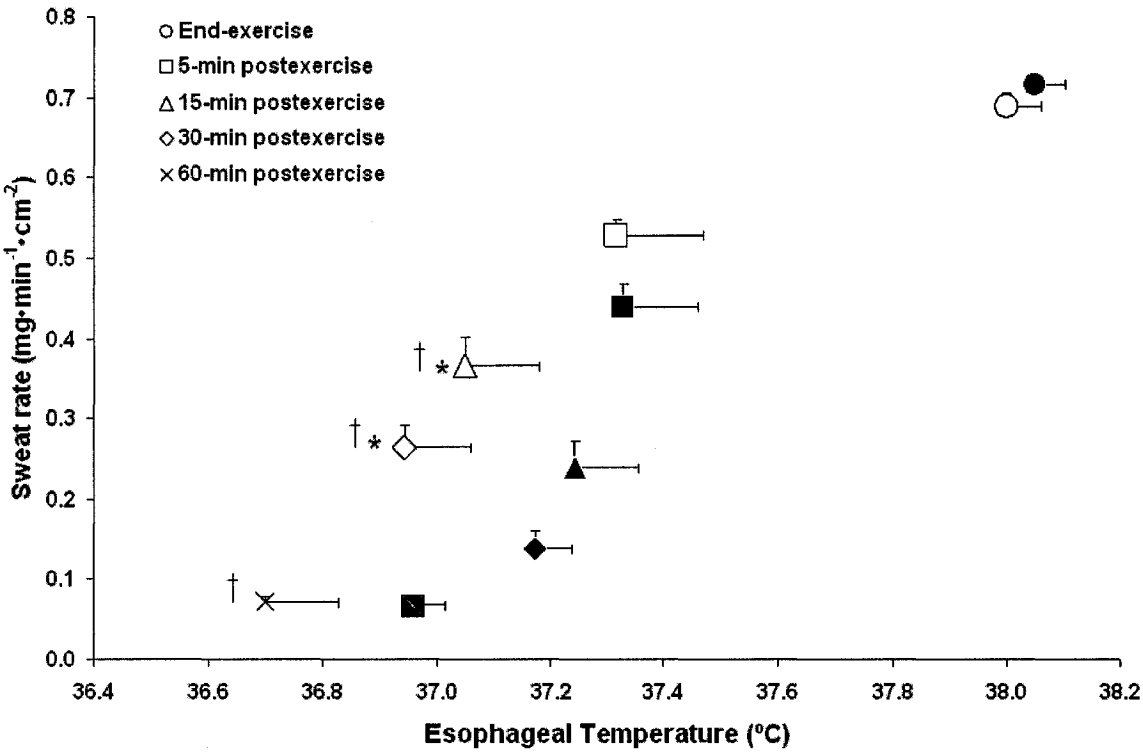
Measure	Position	Baseline	Exercise	2-min	5-min	8-min	12-min	15-min	20-min	25-min	30-min	35-min	40-min	45-min	50-min	55-min	60-min
CO	URS_Ctrl	5.9±0.2	_	0.0±0.6	0.0±0.6	+0.2±0.6	+0.1±0.6	-0.1±0.6	-0.1±0.5	-0.2±0.4	-0.1±0.6	-0.1±0.6	-0.2±0.6	-0.1±0.6	-0.1±0.7	0.0±0.7	0.0±0.7
($L \cdot min^{-1}$)	HDT_Ctrl	6.0±0.2	_	-0.2±0.5	-0.3±0.6	-0.3±0.2	-0.1±0.6	-0.1±0.6	-0.1±0.6	+0.1±0.6	0.0±0.6	-0.1±0.7	0.0±0.6	-0.1±0.6	-0.2±0.6	-0.1±0.6	-0.1±0.7
SV	URS_Ctrl	78.3±3.6	_	-1.2±8.9	-4.3±11.0	-0.8±11.6	-0.5±11.6	-3.5±11.7	-3.7±12.0	-3.9±11.4	-1.7±12.2	-2.0±12.1	-4.3±12.4	-2.3±13.2	-1.2±14.4	+0.6±12.3	-1.2±13.4
(ml)	HDT_Ctrl	81.3±2.9	_	+3.9±8.2*	+4.5±5.3*	+5.4±8.6	+8.1±6.4*	+9.0±8.4*	+9.6±8.1*	+13.0±7.5*	+11.5±7.4*	+12.0±9.3*	+12.7±7.1*	+11.2±8.9*	+15.9±7.7*	+12.8±9.2	+10.6±9.1
HR	URS_Ctrl	76 ± 2	_	+2 ± 10	+4 ± 11	+4 ± 11	+2 ± 10	+2 ± 9	+2 ± 9	+2 ± 10	+1 ± 9	+1 ± 11	+2 ± 10	+2 ± 12	+1 ± 12	0 ± 12	+1 ± 12
($beats \cdot min^{-1}$)	HDT_Ctrl	74 ± 2	_	-5 ± 11*	-6 ± 8*	-7 ± 8*	-7 ± 7*	-7 ± 7*	-8 ± 5*	-8 ± 6*	-9 ± 68	-10 ± 5*	-10 ± 6*	-9 ± 6*	-11 ± 6*	-11 ± 6*	-10 ± 7*
MAP	URS_Ctrl	94 ± 1	_	-1 ± 1	-1 ± 2	-1 ± 2	-1 ± 2	-1 ± 2	-2 ± 2	-2 ± 2	-3 ± 2	-1 ± 2	-2 ± 3	-1 ± 1	-2 ± 1	-1 ± 1	-1 ± 1
(mmHg)	HDT_Ctrl	92 ± 1	_	-1 ± 1	-2 ± 1	-4 ± 1	-3 ± 2	-5 ± 2	-3 ± 1	-2 ± 5	-2 ± 1	-1 ± 1	0 ± 1	-1 ± 4	-2 ± 1	+1 ± 3	0 ± 1
TPR	URS_Ctrl	15.9±0.5	_	-0.4±1.7	0.0±1.7	-0.7±1.4	-0.7±1.2	+0.1±1.3	-0.1±1.1	+0.2±1.1	-0.4±1.6	0.0±1.7	0.0±1.8	0.0±1.7	0.0±1.8	-0.1±2.0	-0.2±1.8
(mmHg)	HDT_Ctrl	15.6±0.6	_	+0.3±1.6	+0.5±2.0	-0.1±2.1	-0.4±1.9	-0.8±1.8	-0.3±2.1	-0.7±2.0	-0.4±1.9	-0.2±2.1	-0.3±2.0	-0.2±2.0	-0.2±1.9	+0.2±1.8	+0.1±2.2

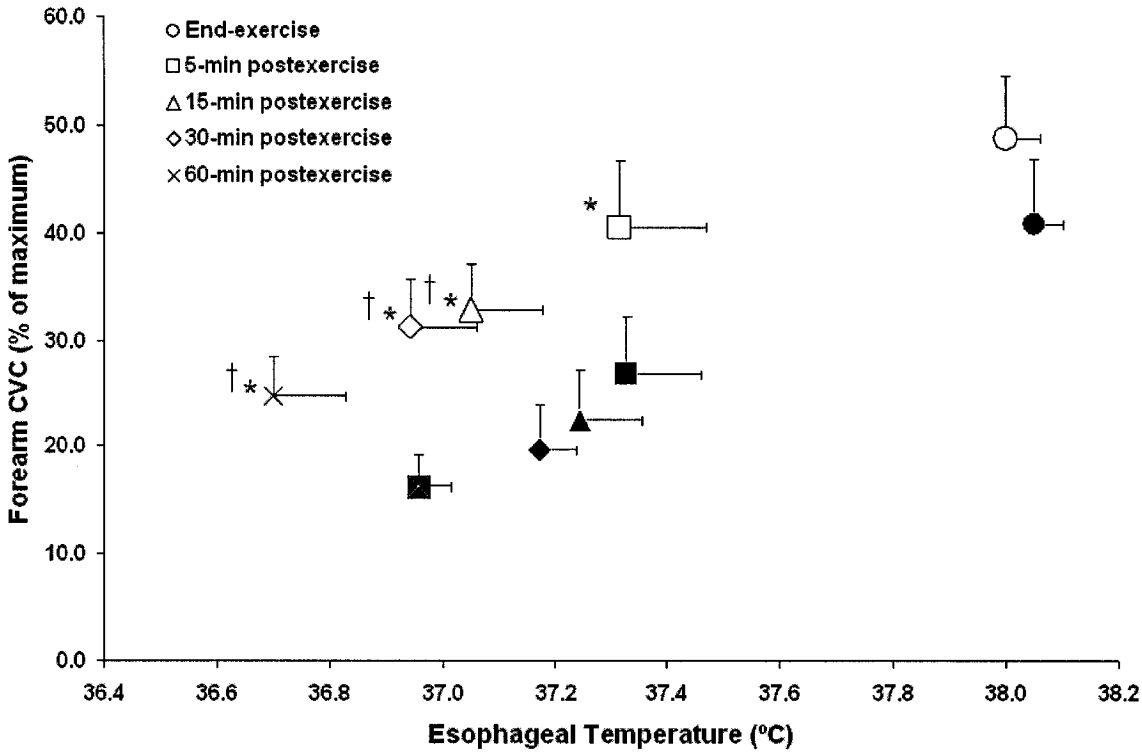
Data reported or n=7, mean and standard deviation (±). URS_Ctrl denotes upright-seated recovery preceded by no exercise; HDT_Ctrl denotes 15° head down tilt recovery preceded by no exercise. Asterisk (*) denotes a within exercise condition difference from upright-seated recovery. All significance at an alpha level of 0.05.


A
Postexercise recovery
Esophageal Temperature (°C)
38.5
38.3
38.1
37.9
37.7
37.5
37.3
37.1
36.9
36.7
36.5
B
Sweat Rate (mg·min-1·cm-2)
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
C
Forearm CVC (% of maximum)
55
50
45
40
35
30
25
20
15
10
Rest
End Exercise
5 10 15 20 25 30 35 40 45 50 55 60
Time (min)


A
Postexercise recovery
CO (L.min-1)
23
21
19
17
15
13
11
9
7
5
B
SV (mL)
140
130
120
110
100
90
80
70
60
50
C
HR (bpm)
180
160
140
120
100
80
60
Rest
End Exercise
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)


A
Postexercise recovery
MAP (mmHg)
120
115
110
105
100
95
90
85
80
B
TPR (mmHg·L·min^{-1})
20
18
16
14
12
10
8
6
4
Rest
End Exercise
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)


End-exercise
5-min postexercise
15-min postexercise
30-min postexercise
60-min postexercise
Sweat rate (mg·min-1·cm-2)
0.0
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
36.4
36.6
36.8
37.0
37.2
37.4
37.6
37.8
38.0
38.2
Esophageal Temperature (°C)


End-exercise
5-min postexercise
15-min postexercise
30-min postexercise
60-min postexercise
Forearm CVC (% of maximum)
60.0
50.0
40.0
30.0
20.0
10.0
0.0
36.4
36.6
36.8
37.0
37.2
37.4
37.6
37.8
38.0
38.2
Esophageal Temperature (°C)

15° Head-down tilt attenuates the postexercise reduction in cutaneous vascular conductance and sweat rate in females.

Natalie H. McInnis[1], W. Shane Journeay[1,2], Ollie Jay[1], Emily E. Leclair[1] and Glen P. Kenny[1]

[1]Laboratory of Human Bioenergetics and Environmental Physiology, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario, Canada K1N 6N5
and
[2]Toxicology Graduate Program and Department of Veterinary Biomedical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, SK, S7N 5B4

Address for correspondence and reprint requests:

✉ G.P. Kenny
University of Ottawa
School of Human Kinetics
125 University, Montpetit Hall
Room 367
Ottawa, Ontario, Canada
PO Box 450 Station A
K1N 6N5
(613) 562-5800 ext. 4282
(613) 562-5149 (fax)
e-mail: gkenny@uottawa.ca

Running head: Nonthermal influences on postexercise heat loss

ABSTRACT

The following study examined the effect of 15° head-down tilt (HDT) on postexercise heat loss and hemodynamic responses in females. We tested the hypothesis that recovery from dynamic exercise in the HDT posture would attenuate the reduction in the heat loss responses of cutaneous vascular conductance (CVC) and sweating relative to upright-seated (URS) recovery in association with an augmented hemodynamic response. Seven females subjects performed the following 3 experimental protocols: 1) 60 min in the URS posture followed by 60 min in the 15° HDT position; 2) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_{2peak}$ followed by 60 min recovery in the URS posture; or 3) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_{2peak}$ followed by 60 min recovery in the 15° HDT position. Mean skin temperature (T_{sk}), esophageal temperature (T_{es}), skin blood flow (SkBF), sweat rate, cardiac output (CO), stroke volume (SV), heart rate (HR), total peripheral resistance (TPR), and MAP were recorded at baseline, end exercise, 2, 5, 8, 12, 15, 20 and every 5-min until end of recovery (60-min). Without preceding exercise, a significant effect of HDT was found for HR and SV ($P \leq 0.05$). During recovery after exercise, a significantly greater MAP, CVC and sweat rate were found with HDT in comparison to URS recovery posture ($P \leq 0.05$). Subsequently a significantly lower T_{es} was observed with HDT from 12-min till the end of the recovery period ($P \leq 0.05$). At the end of 60-min recovery, T_{es} remained significantly elevated above baseline with URS recovery posture ($P \leq 0.05$), however T_{es} returned to baseline with HDT. In conclusion, extended recovery from dynamic exercise in the 15° HDT position attenuates the reduction in CVC and sweating thereby significantly increasing the rate of core temperature decay compared to recovery in the URS posture.

Keywords: skin blood flow, esophageal temperature, nonthermal influences, baroreceptor, head down tilt, females.

INTRODUCTION

An increasing number of studies demonstrate that recovery from dynamic exercise results in significant perturbations of thermoregulatory control. Not only do these perturbations result in a prolonged elevation in postexercise esophageal temperature (54, 97), they are also paralleled by a rapid decrease in sweating (54), skin blood flow (54, 97, 102) and skin temperature (54, 97, 102) to pre-exercise baseline values within the early stages of recovery (~10 to 15-min).

Recovery from exercise is well documented to be associated with significant cardiovascular adjustments. Based on the growing body of evidence supporting a relationship between the cardiovascular system and heat dissipation, it follows that factors determining postexercise cardiovascular status may also attenuate postexercise heat loss responses, and consequently influence core temperature regulation. Following the cessation of exercise, there are changes in the factors that determine mean arterial pressure (MAP) which result in hypotension that is both vascular and neural in origin (27, 47). The magnitude of this decrease in MAP is more pronounced and prolonged following exercise of increasing intensity (15, 54) in the upright seated posture. Postexercise hypotension is thought to occur in part as a result of venous pooling in the previously active musculature (10, 61, 83). It has been shown that resistance vessels in skeletal muscle remain dilated after a bout of cycling exercise and the resultant hyperemia persists well into recovery (82). Such pooling of blood in the lower extremities tends to reduce cardiac filling and unload baroreceptors (27).

Recent studies have employed different recovery interventions to study the relative roles of nonthermal factors such as central command, mechanoreceptors and baroreceptors upon the modulation of postexercise sweating responses. Jackson and Kenny (35) for

example showed that while the postexercise threshold for sweating was elevated compared with no-exercise control, the postexercise increase in the onset threshold for sweating was reversed with the application of LBPP. While their observations support a baroreceptor modulation of postexercise sweating, other factors such as muscle metaboreflex (94), muscle mechanoreceptors (20, 42, 44, 90, 104) and convective heat exchange (i.e., air currents in the chamber) may have contributed to their observations to a certain extent. A more recent study (70) supports the role of cardiopulmonary and/or arterial baroreceptors in the modulation of sweat rate as inferred by the hemodynamic responses to 15° head-down tilt in males. Specifically, the application of head-down tilt attenuates the concurrent reductions in sweat rate and MAP typically observed following exercise.

It has been shown that males and females respond differently to the same orthostatic challenge resulting a greater decrease in postexercise MAP. Furthermore, Kenny et al. (22) showed that the greater postexercise hypotension response observed in females is associated with a greater increase in the onset threshold for skin vasodilation and sweating postexercise. A recent study by Journeay et al. (18) suggests that baroreceptor may play a significant role in modulating postexercise skin blood flow in females albeit their remains inconclusive evidence of their influence on postexericse sweating.

The present study employed 15° head-down tilt (HDT) maneuver to evaluate the hypothesis that recovery from dynamic exercise in the HDT position would augment the heat loss responses of cutaneous vascular conductance and sweating relative to upright recovery in association with an augmented hemodynamic response and increased rate of esophageal temperature decay. We employed a 15° HDT maneuver to study the role of cardiopulmonary baroreceptors on postexericse heat loss. HDT is known to increase central blood volume by

promoting venous return through a reduction in the hydrostatic forces present in the upright posture and is thought to engage primarily the cardiopulmonary baroreceptors without changes in systemic MAP (reduces likelihood of engaging the arterial baroreceptors).

METHODS

Subjects

Seven healthy, physically active females volunteered and gave written consent to participate in this study. The study was approved by the Research Ethics Board at the University of Ottawa. Five to seven days before the experiments, peak oxygen consumption ($\dot{V}O_2$ peak) was measured during a progressive cycle ergometer protocol that required the participant to cycle at a cadence of 80 revolutions per minute while the ergometer resistance was increased at 0.5 Kp every 2-mins. The $\dot{V}O_2$ peak data were used to select the submaximal workload for the experimental exercise phase of the study. Subjects were (mean ± SE) Subjects were (mean ± SE) 20.7 ± 0.76 years old, 160.8 ± 12.1 cm tall, weighed 63.6 ± 5.5 kg and their mean $\dot{V}O_{2peak}$ was 42.6 ± 5.3 $ml \cdot kg^{-1} \cdot min^{-1}$

Measurements

Heart rate (HR) was monitored using a Polar coded transmitter, recorded continuously and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy, Finland). Mean arterial pressure (MAP) was estimated from the integration of a non-invasive recording of blood pressure at the middle digit of the left hand (Finapres 2300, Ohmeda, Madison, WI, USA) fixed at heart level (the third intercostal space). The Finapres system is based on the Penaz volume clamp method (dynamic unloaded arterial wall principle). MAP was verified periodically throughout the protocol by auscultation.

Pulmonary $\dot{V}O_2$ was estimated using a metabolic cart (model CPX/D, Medgraphics, St. Paul, MN) during $\dot{V}O_2$ peak assessment preceding the experimental trials. Cardiac output

(CO) was estimated using the CPX/D computerized version of the CO_2-rebreathing technique of Defares (7). It has been shown that Doppler-derived aortic blood flow (CO) measurements correlate well with the indirect carbon dioxide rebreathing method (26). The Defares method has also been shown to work well in "unsteady state" testing (15). Stroke volume (SV) was calculated as CO/HR. Total peripheral resistance (TPR) was calculated as MAP/CO.

Skin blood flow (SkBF) was estimated using laser-Doppler velocimetry (PeriFlux System 5000, Main control unit; PF5010 LDPM, Function unit; Perimed AB, Stockholm, Sweden) at the left mid-anterior forearm. The laser-Doppler flow probe (PR 401 Angled Probe, Perimed AB, Stockholm, Sweden) was taped to cleaned skin, in an area which did not appear by visual inspection to be overly vascular and from which consistent readings were noted (25). Cutaneous vascular conductance (CVC) was calculated as the ratio of laser-Doppler flow to MAP. At the end of the experiment, local skin temperature was raised to 42°C using a heating element (PF 5020 Temperature Unit, Perimed AB) that housed the laser-Doppler flow probe,until peak CVC was measured (~30 min)(96). CVC-peak was determined as a sustained elevated plateau in local SkBF. CVC data are presented as a percentage of maximal CVC as determined by local heating (%CVC-peak). All SkBF measures were taken in the period preceding rebreathing to avoid causing fluctuations in SkBF data at each time point. SkBF measures were recorded from the left mid-anterior forearm such that the arm was level with the heart.

Sweat rate was measured using a 5.0 cm^2 ventilated capsule placed over the medial inferior aspect of the trapezius muscle. Anhydrous compressed air was passed through the capsule and over the skin surface (Brooks 5850, Mass Flow Controller, Emerson electric, Hetfield, Pa, USA). The vapour density of the effluent air was calculated from the relative

humidity and temperature measured using the Omega HX93 humidity and temperature sensor (Omega Engineering, Stanford, CT, USA). Sweat rate was defined as the product of the difference in water content between effluent and influent air and the flow rate. The flow rate through the capsule was 1.0 $L \cdot min^{-1}$. The sweat rate value was adjusted for skin surface area under the capsule and expressed in $mg \bullet min^{-1} \bullet cm^{-2}$.

Central body temperature (esophageal temperature, T_{es}) was monitored continuously using a paediatric esophageal temperature probe (Mon-a-therm®, Mallinckrodt Medical, St-Louis, USA) inserted through the nares to a depth one-fourth of the standing height of the subject, whereby the tip of the thermocouple is estimated to be at the level of the left atrium (26). Skin temperature was recorded at 11 sites using 0.3 mm diameter T-type thermocouples integrated into eat-flow sensors (Concept Engineering, Old Saybrook, CT, USA, model FR-025-TH44018-6). The area-weighted mean skin temperature ($\bar{T}_{sk}$) and heat flux ($\bar{H}F_{sk}$) were calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9.5%, and posterior calf 9.5%(14). Temperature data were collected and digitized (Hewlett-Packard data acquisition module, model 3497A) at 5-s intervals, displayed graphically in real time and stored on hard disk (Hewlett-Packard, model PC-312, 9000).

The 15° head down tilt position was measured using the Unitek Magnetic Polycast® Protractor; model number 3310-036 (Polytech, Empire Level Manufacturing Corporation, Milwaukee, WI).

Experimental protocol

Each subject performed a total of 3 experimental trials carried out in random order. Experiments were separated by a minimum of 48 h during which subjects were instructed to avoid physical activity and excessive stressors such as exposure to hot or cold temperatures, particularly during the period between awakening and experimentation and during transit from home to the laboratory. Trials were performed at the same time of day for each subject to avoid circadian variation in skin and esophageal temperatures. Subjects were asked to fast at least 4 h prior to experimentation, and water ingestion was permitted *ad libitum* during this time. Upon arrival at the laboratory, subjects clothed in shorts and athletic shoes were fitted with the appropriate instruments. All experimental trials were performed at an ambient temperature of 24.0 ± 0.5°C and a relative humidity of 45%.

After instrumentation subjects remained resting for 10-min during which baseline measures were recorded. Subjects were then required to perform one of 3 experimental protocols. These were: 1) 60 min in the upright-seated (URS) posture followed by 60 min in the 15° head-down tilt position (HDT); 2) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$ peak followed by 60 min recovery in the URS posture; or 3) 15 min of cycle ergometry at 75% of their pre-determined $\dot{V}O_2$ peak followed by 60 min recovery in the 15° HDT position.

At the end of each experiment, peak CVC was determined using a local heating protocol as described above.

Statistical Analyses

A two-way ANOVA with repeated measures was used to analyze the data using the repeated factors of postexercise recovery time (levels: 2, 5, 8, 12, 15 and every 5 min until 60-min) and recovery mode (levels: 15° head-down tilt (HDT) and upright-seated recovery (URS)). The dependent variables employed were the changes from pre-exercise or baseline rest in T_{es}, $\bar{T}_{sk}$, $\bar{H}F_{sk}$, CO, HR, %CVC-peak, MAP, TPR, SV, and sweat rate. All values represent the means and standard deviation for seven subjects. The exercise and no-exercise sessions were analyzed separately to isolate the effect of recovery mode. For ANOVA main effects, Huynh-Feldt corrected statistics are reported where the assumption of sphericity was not met. Pair-wise comparisons were performed using paired sample t-tests. The level of significance was set at an alpha level of 0.05. All analyses were performed using the statistical software package SPSS 12.0 for Windows (SPSS Inc. Chicago, IL, USA).

RESULTS

Esophageal temperature response

Exercise condition. The change in T_{es} at the end of exercise relative to pre-exercise rest was not significantly different between the URS and HDT trials ($p=0.457$), with T_{es} elevated by 1.14 °C (SD 0.40) preceding URS, and by 1.08 °C (SD 0.37) preceding HDT. During recovery, change in T_{es} from pre-exercise rest became less with postexercise recovery time ($F_{(2.2, 20.7)}=68.22$, $p<0.001$) and was influenced by recovery mode ($F_{(1,5)}=7.831$, $p=0.038$). T_{es} was significantly higher from 12-min to 60-min postexercise recovery in the URS posture compared to the HDT position (Figure 2). Esophageal temperature remained significantly ($p=0.002$) elevated by 0.17°C (SD 0.15) above pre-exercise rest after 60-min of URS recovery, but returned to baseline after 60-min of HDT recovery.

No-exercise condition. Without preceding exercise, change in T_{es} relative to resting values did not significantly change with time ($F_{(1,5)}$=2.18, p=0.199) nor recovery mode ($F_{(1,5)}$=1.51, p=0.274).

Skin temperature, dry heat loss, CVC_{peak} and sweating

Exercise condition. Following exercise, the elevations in mean skin temperature from pre-exercise rest became lower as postexercise recovery time progressed ($F_{(2.1,8.6)}$=4.42, p=0.04), however no difference was observed for recovery mode ($F_{(1,6)}$=1.4, p=0.287). Similarly, the elevations in HF_{sk} from pre-exercise rest became lower with postexercise recovery time ($F_{(1,5)}$=76.8, p=0.005) and were influenced by recovery mode ($F_{(1,6)}$=9.9, p=0.026). HF_{sk} was significantly higher in the HDT position from 8-min to 60-min during postexercise recovery. The elevation in CVC_{peak} above pre-exercise rest following exercise prior to both HDT and URS recovery trials became lower with postexercise recovery time ($F_{(1,5)}$=34.39, p=0.002). Furthermore CVC_{peak} was influenced by recovery mode ($F_{(1,5)}$=23.88, p=0.005) with significantly greater values observed throughout the 60-min postexercise recovery period in the HDT position in comparison to URS posture ($p \leq 0.05$). Sweat rate was significantly elevated above pre-resting values prior to HDT and URS recovery trials ($p \leq 0.05$) and then became reduced throughout postexercise recovery ($F_{(4.5,22.8)}$=135.12, $p<0.001$). Furthermore, sweat rate was influenced by recovery mode ($F_{(1,5)}$=8.4, p=0.034), with a significantly greater sweat rate observed during HDT recovery relative to URS between 12-min and 45-min postexercise ($p \leq 0.05$).

No-exercise condition. Without preceding exercise, changes in T_{sk}, HF_{sk}, CVC_{peak} and sweat rate relative to resting values were not influenced by time or recovery mode ($p>0.05$).

Hemodynamic responses

Exercise condition. The elevations in CO following exercise of 9.4 $L \cdot min^{-1}$ (SD 2.5) prior to HDT and 9.5 $L \cdot min^{-1}$ (SD 1.7) prior to URS were not significantly different ($p=0.882$). These elevations from rest in CO became lower during postexercise recovery ($F_{(1,6)}=20.89$, $p<0.001$), but were not influenced by recovery mode ($F_{(1,5)}=0.752$, $p=0.426$). The elevations in SV following exercise of 16.3 ml (SD 13.2) prior to HDT and 16 ml (SD 8.8) prior to URS were not significantly different ($p=0.479$). These elevations from rest in SV were not significantly affected by recovery time ($F_{(1, 5)}=27.3$, $p<0.001$) on the other hand it was influenced by recovery mode ($F_{(1, 5)}=12.4$, $p=0.017$) with a significantly higher SV observed during HDT recovery from 5-min till the end of the postexercise recovery period ($p \leq 0.05$). Following exercise, no significant difference was observed between the elevation in heart rate of 104 $beats \cdot min^{-1}$ (SD 9.6) prior to HDT and 100 $beats \cdot min^{-1}$ (SD 10.0) prior to URS recovery ($p=0.304$). Heart rate elevations above rest became lower with postexercise recovery time ($F_{(6.7,33.2)}=82.1$, $p<0.001$) and were influenced by recovery mode ($F_{(1,5)}=36.5$, $p=0.004$) with a significantly lower heart rate elevation observed during HDT relative to URS throughout the 60-min postexercise recovery period ($p \leq 0.05$). The changes in MAP from pre-exercise rest following exercise prior to recovery were not significantly different between HDT and URS ($p=0.597$). However during postexercise recovery, change in MAP were different between recovery modes ($F_{(1, 5)}=36.6$, $p=0.002$), and influenced by recovery time ($F_{(1, 5)}=10.2$, $p=0.024$) with a significantly higher MAP observed during HDT throughout postexercise recovery ($p \leq 0.05$). The changes in TPR from rest following exercise of -8.84 $mmHg \cdot L^{-1} \cdot min^{-1}$ (SD 2.1) prior to HDT and -8.93 $mmHg \cdot L^{-1} \cdot min^{-1}$ (SD 1.9) prior to URS were not significantly different ($p=1.00$). However, change in TPR from pre-exercise rest

became less throughout postexercise recovery ($F_{(1,5)}$=22.8, p=0.005) and was also influenced by recovery mode ($F_{(1,5)}$=16.3, p=0.01) with a significantly higher TPR during recovery in the HDT position from 35-min to 60-min postexercise recovery.

No-exercise condition. Without preceding exercise, changes in CO, MAP and TPR relative to resting values were not influenced by time or recovery mode ($p>0.05$). However a change in HR from resting values was observed during HDT ($F_{(1,6)}$=7.97, p=0.037), but this was not influenced by postexercise recovery time ($F_{(1,6)}$=0.45, p=0.534) with a significantly lower HR during HDT observed at 5-min till the end of the postexercise recovery period ($p\leq0.05$). Also, SV was significantly higher during HDT from 30-min to 55-min compared to URS posture ($F_{(1,5)}$=11.1, p=0.045), but was not affected by postexercise recovery time ($F_{(1,5)}$=40.41, p=0.567). There was also a significant interaction between recovery mode and time ($F_{(1,5)}$=5.18, p=0.03)

DISCUSSION

The most important observation from this study is that extended recovery from dynamic exercise in the 15° head down tilt (HDT) position attenuates the fall in MAP, and increases CVC and sweating. The higher sustained skin blood flow and sweating response was paralleled by a greater rate of core temperature decay. Thus, HDT is an effective technique to attenuate the reduction in heat loss responses in females recovering from dynamic exercise. These findings support a recent study done by Journeay et al. (17) who used LBPP to manipulate venous pooling during recovery from dynamic exercise in males. They observed a baroreceptor-mediated attenuation in heat loss responses of both CVC and sweating.

Effect of Tilt at Rest

The present study employed HDT to induce a shift in blood volume from the lower extremities to the upper body. To the best of our knowledge, no previous studies have examined the effect of 15° HDT on hemodynamic and thermal responses in females. Previous studies on males have shown that in normotensive individuals at rest an angle of 15° is sufficient to increase venous return and thereby increase intracardiac pressure and stimulate the cardiopulmonary baroreceptors (27). Stimulating the cardiopulmonary baroreceptors causes an increase in SV and a decrease in TPR. In contrast we showed that HDT resulted in a significant increase in HR and SV with no significant difference in TPR. The difference may be due to the fact that we compared our findings to URS posture and Nagaya et al. (27) compared their data to the supine posture. It has been suggested that a change in body

position from the URS posture to the supine posture will decrease cardiac sympathetic nerve activity and increase vagus nerve activity (28, 30).

Effect of URS posture on Hemodynamics Responses

Hydrostatic forces present in the URS posture increase orthostatic stress during recovery from exercise. Therefore, there is a greater increase in venous pooling, reducing central blood volume, which in turn leads to a greater decrease in MAP (13). Our postexercise hemodynamic responses in the URS posture were consistent with previous observations (17-19, 20). Our results showed that MAP was significantly lower compared to baseline values in the URS posture following one hour of recovery. Also, during this period we observed a sustained increase in HR, TPR and a decrease in SV, which is indicative of an increase baroreceptor stimulation (baroreceptor unloading).

Effect of HDT on Hemondynamics Responses

Our results illustrate that hemodynamics during 15° HDT in resting conditions and 15° HDT during recovery from dynamic exercise are significantly different. For example, there is a significant effect on MAP and TPR in the HDT position following exercise that is not seen during HDT at rest. These differences may be due to the fact that venous pooling in the lower extremities is significantly increased following exercise. It has been suggested that postexercise hyperemia in the lower limbs may be more profound in females then males (1). During 15° HDT at rest central venous pressure increases by approximately 2.16 mmHg (27) we can assume that since venous pooling is increased during recovery from exercise that the same degree of tilt will envoke a greater increase in central venous pressure. Therefore it is

possible that during recovery from dynamic exercise not only are cardiopulmonary baroreceptors being loaded but arterial baroreceptors may be affected as well.

Effect of URS Posture on Heat Loss Responses

Following cessation of exercise, the decrease in MAP associated with the pooling of blood in the lower limbs stimulates an increase in baroreceptor activity resulting in baroreflex mediated increase in vasoconstrictor tone and heart rate. Previous studies have suggested that during recovery from exercise in the upright posture, sweating and skin blood flow are influenced by baroreceptors (4, 18, 19). A study by Carter et al. (3) showed that females have a greater decrease in MAP and less of a decrease in TPR compared to males during the postexercise period. Their findings suggest that females may have an attenuated baroreflex mediated response on postexericse heat loss response as compared to their male counterparts. Our results indicate that during recovery in the upright seated posture sweating and skin blood flow returns to pre-exercise values despite a sustained increase in core temperature. Conversely, when the subjects are placed in the HDT position, the level of postexercise hypotension and the magnitude of the postexercise elevation in core temperature are reduced as compared to the upright seated position.

Effect of HDT on Heat Loss Responses

Based on our postexercise hemodynamic data, recovery from exercise in the 15° HDT position is effective in reversing the unloading of the baroreceptors and thereby attenuating the reduction of MAP. We showed that HDT significantly decreased HR and increased SV compared to the URS posture. Of particular importance is the fact that altering the

postexercise hemodynamic response using a head-down tilt maneuver significantly modified the postexercise themoregulatory responses. Following recovery in the HDT position, we observed a significant increase in the rate of core temperature decay compared to upright seated posture. This increase in core temperature decay is likely due to the increase in sweat rate and skin blood flow recorded during recovery in the HDT position. Previous studies have shown that attenuating unloading of the baroreceptors will preserve CVC during recovery from exercise in males (16, 18) but to date there are contradictory findings on how baroreceptors effect sweat rate (2, 18, 29, 31). Our findings support the former and extend these observations to females.

Jackson and Kenny (16) have shown that applying LBPP during recovery from exercise will decrease the postexercise resting threshold for sweating. More recently, Journeay et al (19) studied the effect of recovery mode, active passive and inactive, on sweating and skin blood flow their findings suggested that CVC and sweating are influenced by cardiopulmonary baroreceptors. Our data support these findings, albeit an influence of arterial baroreceptors in the modulation of postexercise skin blood flow and sweating can not discounted. Specifically, we did note a significant increase in MAP throughout recovery from exercise in the HDT position.

Summary

Recovery from exercise in the HDT position augments the hemodyanmic response of MAP and SV and heat loss responses of cutaneous vascular conductance and sweating relative to the URS posture. Specifically HDT caused a significant increase in CVC and sweat rate, which lead to an increase in the rate of core temperature decay. These findings

have potential application for hyperthermic females. Placing hyperthermic individuals in a HDT position will maintain blood pressure and significantly increase the rate of heat loss.

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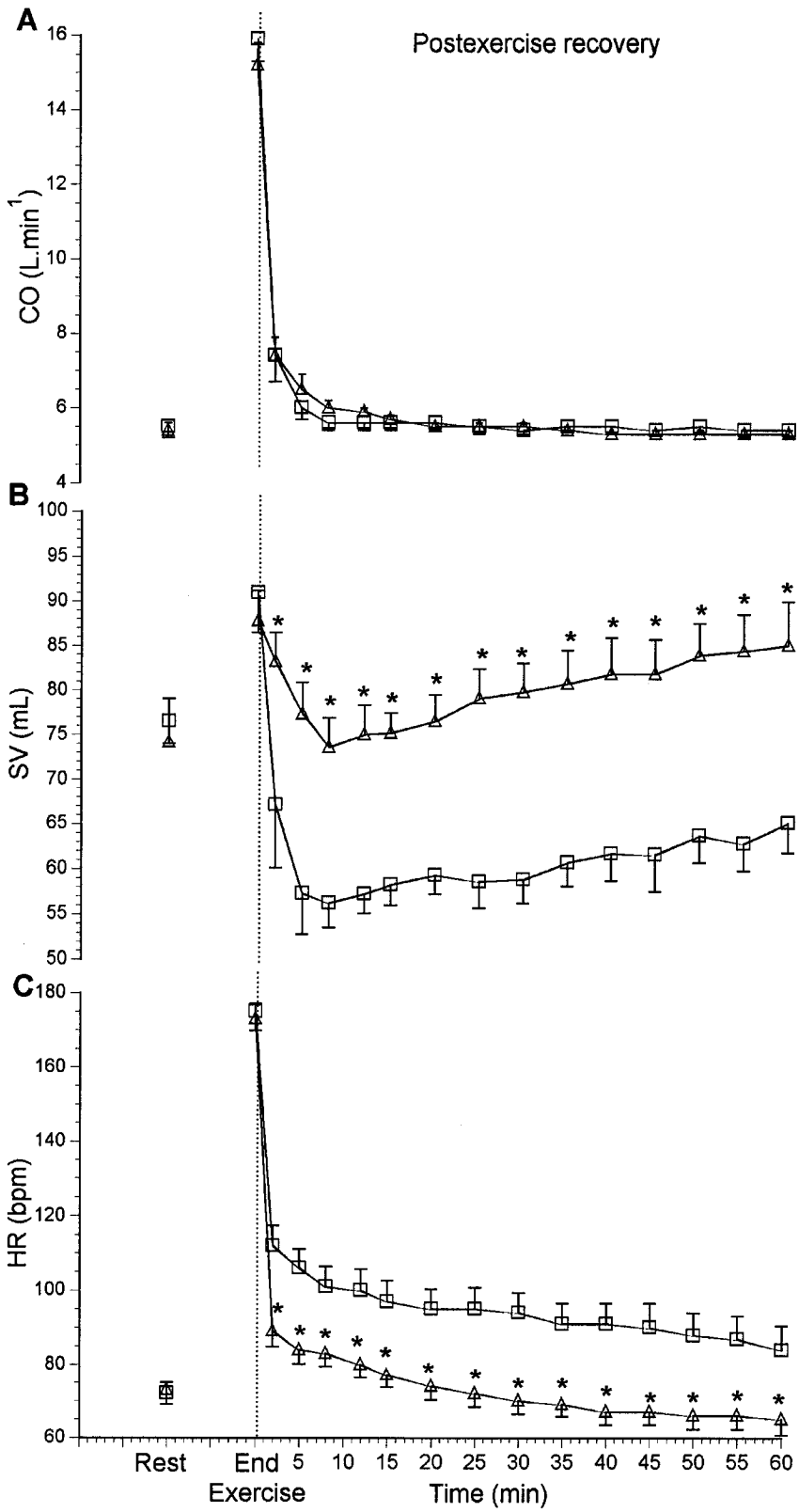
FIGURE CAPTIONS

Figure 1. Effect of URS posture (□) and recovery in a 15° HDT recovery position (Δ) following 15 min of moderate intensity cycle ergometry on A) cardiac output, B) stroke volume, and C) heart rate . Data expressed as means ±SE. * denotes significant difference between inactive seated recovery.

Figure 2. Effect of URS posture (□) and recovery in a 15° HDT recovery position (Δ) following 15 min of moderate intensity cycle ergometry on A) total peripheral resistance and B) mean arterial pressure. Data expressed as means ±SE. * denotes significant difference between URS posture.

Figure 3. Effect of URS recovery posture (□) and recovery in a 15° HDT recovery position (Δ) following 15 min of moderate intensity cycle ergometry on A) esophageal temperature , B) sweat rate , C) cutaneous vascular conductance. Data expressed as means ±SE. * denotes significant difference between URS posture.


A
Postexercise recovery
CO (L.min^{-1})
16
14
12
10
8
6
4
B
SV (mL)
100
95
90
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80
75
70
65
60
55
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C
HR (bpm)
180
160
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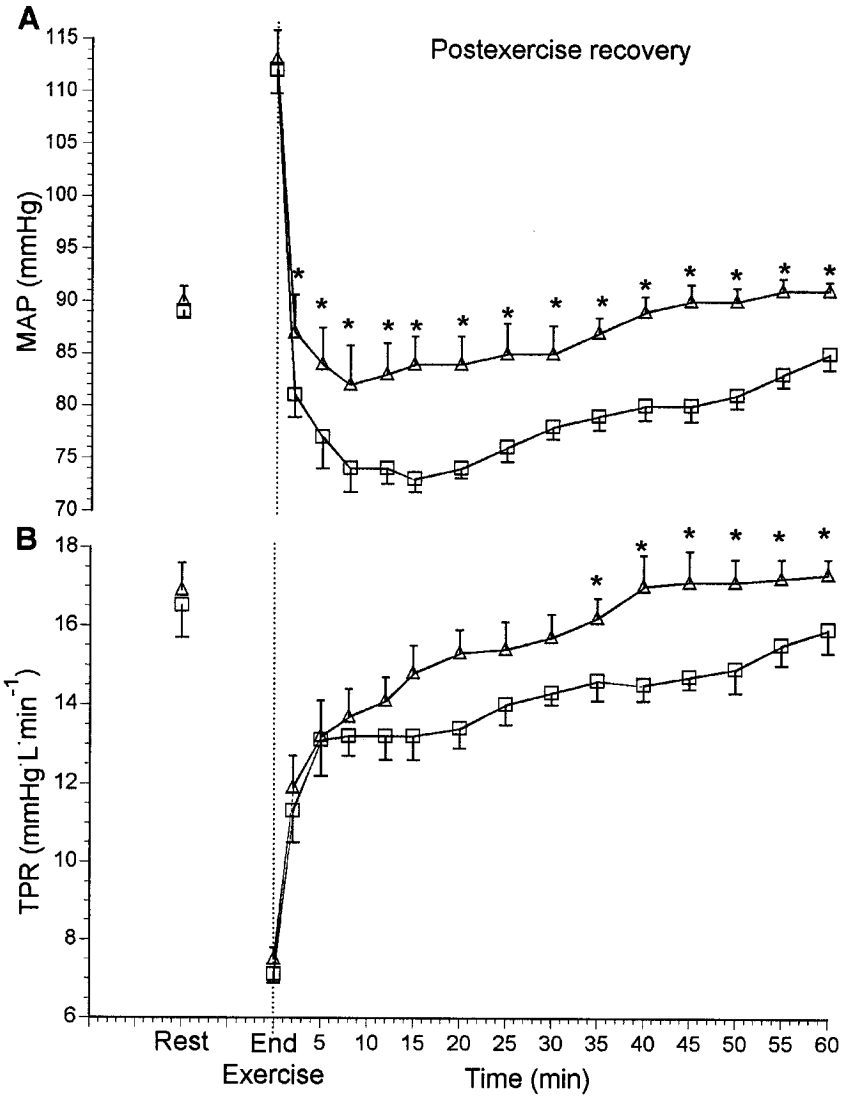
Rest
End Exercise
5 10 15 20 25 30 35 40 45 50 55 60
Time (min)


A
Postexercise recovery
MAP (mmHg)
115
110
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100
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90
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75
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B
TPR (mmHg·L·min^{-1})
18
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14
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8
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Rest
End Exercise
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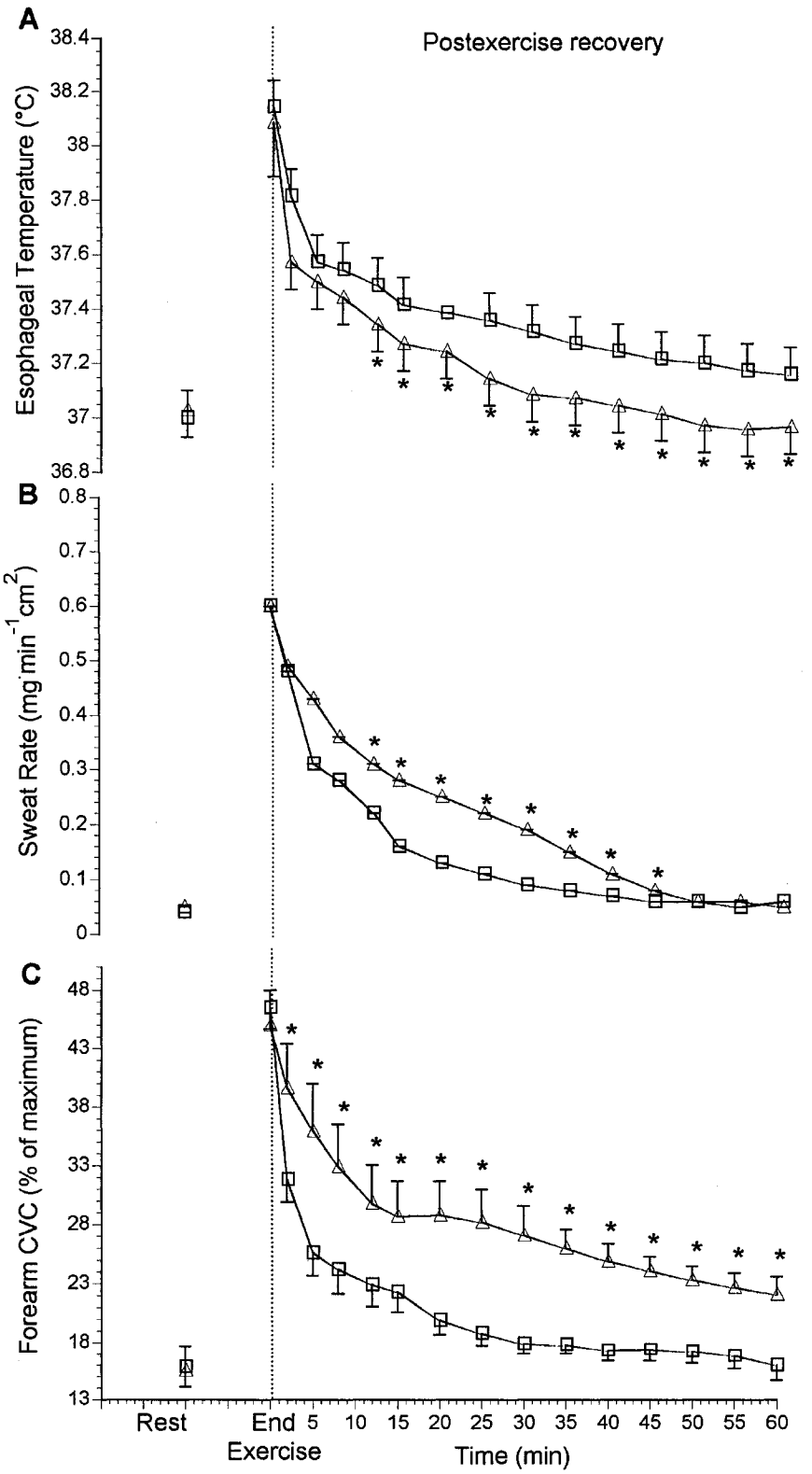
60
Time (min)


A
Postexercise recovery
Esophageal Temperature (°C)
38.4
38.2
38
37.8
37.6
37.4
37.2
37
36.8
B
Sweat Rate (mg·min-1·cm2)
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
C
Forearm CVC (% of maximum)
48
43
38
33
28
23
18
13
Rest
End Exercise
5 10 15 20 25 30 35 40 45 50 55 60
Time (min)

THE EFFECT OF EXERCISE INTENSITY ON POSTEXERCISE HEAT LOSS RESPONSE IN FEMALES

Natalie H. McInnis[1], W. Shane Journeay[1,2], Ollie Jay[1], Emily E. Leclair[1] and Glen P. Kenny[1]

[1]Laboratory of Human Bioenergetics and Environmental Physiology, School of Human Kinetics, Faculty of Health Science, University of Ottawa, Ottawa, Ontario, Canada K1N 6N5;
and,
[2]Toxicology Program and Department of Veterinary Biomedical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, SK, S7N 5B4.

Address for correspondence and reprint requests:

G.P. Kenny
University of Ottawa
School of Human Kinetics
125 University, Montpetit Hall
Room 376
Ottawa, Ontario, Canada
PO Box 450 Station A
K1N 6N5
(613) 562-5800
(613) 562-5149 (fax)
e-mail: gkenny@uottawa.ca

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ABSTRACT

The following study examined the effect of exercise intensity on postexercise heat loss and hemodynamic responses in females. We hypothesized that in association with a greater decrease in mean arterial pressure (MAP) during recovery from exercise at higher intensities the duration and magnitude of esophageal temperature will increase. In association with a decrease in MAP, skin blood flow and sweat rate will be reduced. On three separate occasions, seven females subjects performed cycled at 1) 55% $\dot{V}O_{2peak}$;; 2) 75% $\dot{V}O_{2peak}$; or 3) 85% of their pre-determined $\dot{V}O_{2peak}$ followed by 60 min recovery in the upright-seated position. Mean skin temperature (T_{sk}), esophageal temperature (T_{es}), skin blood flow (SkBF), sweat rate, cardiac output (CO), stroke volume (SV), heart rate (HR), total peripheral resistance (TPR), and MAP were recorded at baseline, end exercise, 2, 5, 8, 12, 15, 20 and every 5-min until end of recovery (60-min). As exercise intensity increased MAP, SV sweating and CVC significantly decreased and HR significantly increased ($p \leq 0.05$). Subsequently the magnitude and duration of T_{es} also increased with exercise intensity ($p \leq 0.05$). At the end of 60-min recovery, T_{es} remained significantly elevated above baseline following exercise at 85% $\dot{V}O_{2peak}$ and 75% $\dot{V}O_{2peak}$ ($p \leq 0.05$) but returned to baseline values following exercise at 55% $\dot{V}O_{2peak}$ ($p > 0.05$). We conclude that the exercise intensity dependent decrease in postexercise MAP was associated with a decrease in the magnitude of CVC and sweating response and a concomitant increase in core temperature recovery time.

Keywords: skin blood flow, esophageal temperature, nonthermal influences, baroreceptor, exercise, females.

INTRODUCTION

Following cessation of exercise, there are changes in the factors that determine MAP which result in hypotension that is both vascular and neural in origin (27, 47). Mean arterial pressure decreases rapidly during the early stages of recovery as do stroke volume and cardiac output while total peripheral resistance tends to increase towards pre-exercise resting values (6). The magnitude of this decrease in MAP is more pronounced and prolonged subsequent to exercise of increasing intensity (15) and is greater with the orthostatic influence of upright posture (27, 47, 83).

Postexercise hypotension is thought to occur in part as a result of venous pooling in the previously active musculature (10, 61, 83). It has been shown that resistance vessels in skeletal muscle remain dilated after a bout of cycling exercise and the resultant hyperemia persists well into recovery (82). Such pooling of blood in the lower extremities tends to reduce cardiac filling and unload baroreceptors (27). Changes in hemodynamic response, such as an increase in stroke volume and MAP, induced by the application of positive pressure to the lower limbs in the upright position postexercise were shown to 1) reverse the postexercise increase in the esophageal temperature at which onset of cutaneous vasodilation and sweating occurs (35), and 2) favor the decay of esophageal temperature to pre-exercise values (41). Thus, the same conditions that give rise to these cardiovascular phenomena postexercise appear to attenuate heat loss during the postexercise period, eliciting a prolonged elevation in esophageal temperature.

The studies examining postexercise control of cutaneous circulation and sweating have primarily been conducted in men. To date, it remains to be determined how possible differences in the postexercise cardiovascular response, induced by exercise of increasing

intensity, between men and women may influence control of the cutaneous circulation and sweating. The study of differences between men and women in the control of skin blood flow and sweating is particularly important when one considers that there is increasing evidence demonstrating differences in postexercise blood pressure regulation in women as compared to men in the upright seated position. Studies show that women have a reduced tolerance to orthostatic challenges at rest (11, 18, 25, 91, 101), and/or an attenuated responsiveness in mechanisms that regulate arterial pressure (11, 25). Furthermore, these studies show a difference in the blood pressure response postexercise between men and women (5) as well as a relationship between postexercise blood pressure and thermal response thresholds in men (55). Therefore, it is a reasonable postulate that women will show a greater attenuation in skin blood flow and sweating in association with a greater reduction in postexercise mean arterial pressure than men.

Thus, the purpose of this study was to examine the effect of exercise intensity on hemodynamic and thermal responses in females. In particular, the role of baroreceptors on CVC and sweating during the postexercise period.

METHODS

Subjects

Seven healthy, physically active females volunteered and gave written consent to participate in this study. The study was approved by the Research Ethics Board at the University of Ottawa. Five to seven days before the experiments, peak oxygen consumption ($\dot{V}O_2$ peak) was measured during a progressive cycle ergometer protocol that required the participant to cycle at a cadence of 80 revolutions per minute while the ergometer resistance was increased at 0.5 Kp every 2-mins. The $\dot{V}O_{2peak}$ data were used to select the submaximal workload for the experimental exercise phase of the study. Subjects were (mean ± SE) 21 ± 1.5 years old, 162 ± 13 cm tall, weighed 61.7 ± 5.0 kg and their mean $\dot{V}O_{2peak}$ was 43.8 ± 3.1 $ml \cdot kg^{-1} \cdot min^{-1}$.

Measurements

Heart rate (HR) was monitored using a Polar coded transmitter, recorded continuously and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy, Finland). Mean arterial pressure (MAP) was estimated from the integration of a non-invasive recording of blood pressure at the middle digit of the left hand (Finapres 2300, Ohmeda, Madison, WI, USA) fixed at heart level (the third intercostal space). The Finapres system is based on the Penaz volume clamp method (dynamic unloaded arterial wall principle). MAP was verified periodically throughout the protocol by auscultation.

Pulmonary $\dot{V}O_2$ was estimated using a metabolic cart (model CPX/D, Medgraphics, St. Paul, MN) during $\dot{V}O_{2\,peak}$ assessment preceding the experimental trials. Cardiac output (CO) was estimated using the CPX/D computerized version of the CO_2-rebreathing technique

of Defares (5). It has been shown that Doppler-derived aortic blood flow (CO) measurements correlate well with the indirect carbon dioxide rebreathing method (8). The Defares method has also been shown to work well in "unsteady state" testing (5). Stroke volume (SV) was calculated as CO/HR. Total peripheral resistance (TPR) was calculated as MAP/CO.

Skin blood flow (SkBF) was estimated using laser-Doppler velocimetry (PeriFlux System 5000, Main control unit; PF5010 LDPM, Function unit; Perimed AB, Stockholm, Sweden) at the left mid-anterior forearm. The laser-Doppler flow probe (PR 401 Angled Probe, Perimed AB, Stockholm, Sweden) was taped to cleaned skin, in an area which did not appear by visual inspection to be overly vascular and from which consistent readings were noted (67). Cutaneous vascular conductance (CVC) was calculated as the ratio of laser-Doppler flow to MAP. At the end of the experiment, local skin temperature was raised to 42°C using a heating element (PF 5020 Temperature Unit, Perimed AB) that housed the laser-Doppler flow probe,until peak CVC was measured (~30 min)(96). CVC-peak was determined as a sustained elevated plateau in local SkBF. CVC data are presented as a percentage of maximal CVC as determined by local heating (%CVC-peak). All SkBF measures were taken in the period preceding rebreathing to avoid causing fluctuations in SkBF data at each time point. SkBF measures were recorded from the left mid-anterior forearm such that the arm was level with the heart.

Sweat rate was measured using a 5.0 cm^2 ventilated capsule placed over the medial inferior aspect of the trapezius muscle. Anhydrous compressed air was passed through the capsule and over the skin surface (Brooks 5850, Mass Flow Controller, Emerson electric, Hetfield, Pa, USA). The vapour density of the effluent air was calculated from the relative humidity and temperature measured using the Omega HX93 humidity and temperature sensor

(Omega Engineering, Stanford, CT, USA). Sweat rate was defined as the product of the difference in water content between effluent and influent air and the flow rate. The flow rate through the capsule was 1.0 $L \cdot min^{-1}$. The sweat rate value was adjusted for skin surface area under the capsule and expressed in $mg \cdot min^{-1} \cdot cm^{-2}$.

Central body temperature (esophageal temperature, T_{es}) was monitored continuously using a pediatric esophageal temperature probe (Mon-a-therm®, Mallinckrodt Medical, St-Louis, USA) inserted through the nares to a depth one-fourth of the standing height of the subject, whereby the tip of the thermocouple is estimated to be at the level of the left atrium (17). Skin temperature was recorded at 11 sites (Concept Engineering, Old Saybrook, CT, USA, model FR-025-TH44018-6). The area-weighted mean skin temperature ($\bar{T}_{sk}$) was estimated by calculating the weighted mean value, using the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9.5%, and posterior calf 9.5% (11). Temperature data were collected and digitized (Hewlett-Packard data acquisition module, model 3497A) at 5-s intervals, displayed graphically in real time and stored on hard disk (Hewlett-Packard, model PC-312, 9000).

EXPERIMENTAL PROTOCOL

Each subject performed a total of 3 experimental trials carried out in random order. Experiments were separated by a minimum of 48-hours during which subjects were instructed to avoid physical activity and excessive stressors such as exposure to hot or cold temperatures, particularly during the period between awakening and experimentation and during transit from home to the laboratory. Trials were performed at the same time of day for

each subject to avoid circadian variation in skin and oesophageal temperatures. They were asked to fast at least 4 h prior to experimentation, and water ingestion was permitted *ad libitum* during this time. Upon arrival at the laboratory, subjects clothed in shorts, a sports bra and athletic shoes were fitted with the appropriate instruments. All experimental trials were performed at an ambient temperature of 24.0 ± 0.5 °C and a relative humidity of 45%.

After preparation of the subjects they were asked to remain seated for 10-min while baseline measurements were recorded this is followed by 15-minutes of exercise on a cycle ergometer at one of the following intensities: 1) 85% $\dot{V}O_{2\,peak}$, 2) 75% $\dot{V}O_{2\,peak}$, or 3) 55% $\dot{V}O_{2\,peak}$. Immediately following the cessation of exercise the subjects were placed in the upright-seated position for one hour.

At the end of the experiment, peak CVC was determined using a local heating protocol as described above.

DATA ANALYSIS

Statistical Analyses

A two-way ANOVA with repeated measures was used to analyze the data using the repeated factors of postexercise recovery time (levels: 2, 5, 8, 12, 15 and every 5 min until 60-min) and exercise intensity (levels: 85%, 75% and 55%). The dependent variables employed were the changes from pre-exercise or baseline rest in T_{es}, $\bar{T}_{sk}$, $\bar{H}F_{sk}$, CO, HR, %CVC-peak, MAP, TPR, SV, and sweat rate. All values represent the means and standard deviation for seven subjects. For ANOVA main effects, Huynh-Feldt corrected statistics are reported where the assumption of sphericity was not met. Pair-wise comparisons were performed using paired sample t-tests. The level of significance was set at an alpha level of 0.05. All analyses were

performed using the statistical software package SPSS 12.0 for Windows (SPSS Inc. Chicago, IL, USA).

RESULTS

Esophageal temperature response

Exercise condition. The change in T_{es} at the end of exercise relative to pre-exercise rest was significantly different between 85% $\dot{V}O_{2\,peak}$ and 75% $\dot{V}O_{2\,peak}$ ($p=0.001$), 75% $\dot{V}O_{2\,peak}$ and 55% $\dot{V}O_{2\,peak}$ ($p=0.001$) and 85% and 55% $\dot{V}O_{2\,peak}$ ($p<0.001$) trials. With T_{es} elevated by 1.47°C (SD 0.36) preceding 85% $\dot{V}O_{2\,peak}$, by 1.1°C (SD 0.29) preceding 75% $\dot{V}O_{2\,peak}$, and 0.6°C (SD 0.1) preceding exercise at 55% $\dot{V}O_{2\,peak}$. During recovery, change in T_{es} from pre-exercise rest became less with postexercise recovery time ($F_{(2.6,15.3)}=88.17$, $p<0.001$) and was influenced by exercise intensity ($F_{(1.5,8.8)}=26.61$, $p<0.001$). T_{es} was significantly higher throughout recovery following exercise at 85% $\dot{V}O_{2\,peak}$ compared to exercise at 55% $\dot{V}O_{2\,peak}$ ($p<0.05$) and was significantly higher then 75% $\dot{V}O_{2\,peak}$ ($p<0.05$) from 12-min postexercise for the remainder of the recovery period. Also, T_{es} was significantly elevated following exercise at 85% $\dot{V}O_{2\,peak}$ from 12-min to 35-min compared to recovery from exercise at 75% $\dot{V}O_{2\,peak}$ ($p<0.05$) (Figure 3). Subsequent to exercise at 85% $\dot{V}O_{2\,peak}$ and 75% $\dot{V}O_{2\,peak}$ esophageal temperature remained significantly elevated by 0.34°C (SD 0.11, $p<0.001$) and by 0.21°C (SD 0.13, $p=0.006$) above pre-exercise rest after 60-min of

recovery, but had returned to baseline by the end of the recovery period proceeding exercise at 55% $\dot{V}O_{2\ peak}$.

Skin temperature, dry heat loss, CVC_{peak} and sweating

Exercise condition. Following exercise, the elevations in mean skin temperature from pre-exercise rest became lower as postexercise recovery time progressed ($F_{(1,6)}$=17.39, p=0.006); however no difference was observed for exercise intensity ($F_{(1.8,12)}$=0.026, p=0.0.963). There was also an interaction observed between time and intensity ($F_{(6.5,39.1)}$=3.5, p=0.006). A significantly higher T_{sk} was observed at 5-min and 8-min following exercise at 85% $\dot{V}O_{2\ peak}$ compared to exercise at 55% $\dot{V}O_{2\ peak}$ ($p<0.05$). There was also a significantly higher T_{sk} observed at 2-min and 5-min following exercise at 75% $\dot{V}O_{2\ peak}$ compared to exercise at 55% $\dot{V}O_{2\ peak}$ ($p<0.05$). Similarly, the elevations in HF_{sk} from pre-exercise rest became lower with postexercise recovery time ($F_{(1.3,8.0)}$=65.16, p, 0.001), but there was no influenced of exercise intensity ($F_{(1.5,9.0)}$=0.752, p=0.463). The elevation in CVC_{peak} above pre-exercise rest following exercise of 85% $\dot{V}O_{2\ peak}$, 75% $\dot{V}O_{2\ peak}$ and 55% $\dot{V}O_{2\ peak}$ became lower with postexercise recovery time ($F_{(1,6)}$=9.71, p=0.021). Furthermore CVC_{peak} was influenced by exercise intensity ($F_{(1.7,10.2)}$=52.02, $p<0.001$) with significantly greater values observed from 20-min to 50-min during recovery after exercise at 55% $\dot{V}O_{2\ peak}$ compared to recovery following exercise at 85% $\dot{V}O_{2\ peak}$ ($p\leq0.05$). Sweat rate was significantly elevated above pre-resting values prior to 85% $\dot{V}O_{2\ peak}$, 75% $\dot{V}O_{2\ peak}$ and 55% $\dot{V}O_{2\ peak}$ recovery trials ($p\leq0.05$) and then became reduced throughout postexercise recovery

($F_{(2.5,15.8)}$=129.49, p<0.001). Furthermore, sweat rate was influenced by exercise intensity ($F_{(1.6,9.7)}$=14.9, p=0.002), with a significantly greater sweat rate observed after exercise at 55% $\dot{V}O_{2\,peak}$ from 8-min to 45-min compared to exercise at 85% $\dot{V}O_{2\,peak}$ (p<0.05) and 25-min to 30-min compared to recovery preceding 75% $\dot{V}O_{2\,peak}$ (p<0.05). Sweat rates were also significantly higher following exercise at 75% $\dot{V}O_{2\,peak}$ from 5-min to 40-min compared to 85% $\dot{V}O_{2\,peak}$ (p<0.05).

Hemodynamic responses

Exercise condition. The elevations in CO following exercise of 85% $\dot{V}O_{2\,peak}$ is 11.1 L•min^{-1} (SD 2.3), 75% VO_{2peak} is 10.2 L•min^{-1} (SD 1.7), and 55% $\dot{V}O_{2\,peak}$ 7.6 L•min^{-1} (SD 1.6). CO prior to exercise at 55% $\dot{V}O_{2\,peak}$ is significantly different from exercise at 85% $\dot{V}O_{2\,peak}$ and 75% $\dot{V}O_{2\,peak}$ (p<0.001), on the other hand there is no significant difference between CO at 85% and 75% $\dot{V}O_{2\,peak}$ (p>0.05). These elevations from rest in CO became lower during postexercise recovery ($F_{(1,6)}$=24.5, p=0.003), and showed a trend for exercise intensity ($F_{(1,6)}$=5.64, p=0.055). Preceding exercise of 55% $\dot{V}O_{2\,peak}$ a significant difference was seen at 2-min compared to 75% $\dot{V}O_{2\,peak}$ (p<0.05) and at 2-min and 5-min when compared to 85% $\dot{V}O_{2\,peak}$ (p<0.05). The elevations in SV following exercise at 85% $\dot{V}O_{2\,peak}$ is 25.2 ml (SD 8.97), 75% $\dot{V}O_{2\,peak}$ is 21 ml (SD 8.2), and 14.8 ml (SD 6.98) at 55% $\dot{V}O_{2\,peak}$, only 85% $\dot{V}O_{2\,peak}$ and 55% $\dot{V}O_{2\,peak}$ were significantly different (p=0.002). These elevations from rest in SV

were significantly affected by recovery time ($F_{(1,6)}$=6.74, p=0.041) and also influenced by exercise intensity ($F_{(1.8,10.7)}$=14.26, p<0.001) with a significantly higher SV observed throughout recovery preceding exercise at 55% $\dot{V}O_{2\,peak}$ compared to 85% $\dot{V}O_{2\,peak}$ and at 12-min, 15-min, 30-min and 40-min to 60-min compared to 75% $\dot{V}O_{2\,peak}$ (p<0.05). Also, recovery from exercise at 75% $\dot{V}O_{2\,peak}$ showed a significantly higher SV at 5-min and 12-min then recovery following 85% $\dot{V}O_{2\,peak}$ (p<0.05). Following exercise, significant difference was observed between the elevation in heart rate of 103 beats•min^{-1} (SD 10.6) at 85% $\dot{V}O_{2\,peak}$, 94.6 beats•min^{-1} (SD 94.6) at 75% $\dot{V}O_{2\,peak}$, and 78.1beats•min^{-1} (SD 9.0) at 55% $\dot{V}O_{2\,peak}$ (p<0.05). Heart rate elevations above rest became lower with postexercise recovery time ($F_{(1.9,11.2)}$=91.5, p<0.001) and was influenced by exercise intensity ($F_{(1.9,11.6)}$=14.37, p=0.001) with a significantly lower heart rate elevation observed throughout exercise recovery preceding exercise at 55% $\dot{V}O_{2\,peak}$ relative to exercise at 85% $\dot{V}O_{2\,peak}$ (p≤0.05) and from 2-min to 55-min relative to 75% $\dot{V}O_{2\,peak}$ (p≤0.05). Heart rate was also significantly higher during recovery from exercise after 85% $\dot{V}O_{2\,peak}$ compared to 75% $\dot{V}O_{2\,peak}$ from 25-min to 60-min (p≤0.05). The changes in MAP from pre-exercise rest following exercise were significantly different between all three exercise intensities (p<0.05). However during postexercise recovery, change in MAP was different between all three exercise intensities ($F_{(1.7,10.2)}$=52.02, p<0.001), and influenced by recovery time ($F_{(1,6)}$=32.67, p=0.001) with a significantly higher MAP observed preceding exercise at 55% $\dot{V}O_{2\,peak}$ throughout postexercise recovery compared to 85% $\dot{V}O_{2\,peak}$ and it is also higher from 5-min to 45-min

compared to recovery from 75% VO2peak ($p \leq 0.05$). The changes in TPR from rest following exercise of 85% $\dot{V}O_{2\,peak}$ –8.59 $mmHg \cdot L^{-1} \cdot min^{-1}$ (SD 5.1) and –8.39 $mmHg \cdot L^{-1} \cdot min^{-1}$ (SD 4.5) following exercise of 75% $\dot{V}O_{2\,peak}$ and –7.7 $mmHg \cdot L^{-1} \cdot min^{-1}$ (SD 3.7) following exercise of 55% $\dot{V}O_{2\,peak}$ were not significantly different ($p > 0.05$). However, change in TPR from pre-exercise rest became less throughout postexercise recovery ($F_{(1,6)}=21.5$, $p=0.004$) and was also influenced by exercise intensity ($F_{(1,6)}=8.55$, $p=0.026$) with a significantly higher TPR during recovery in from 55% $\dot{V}O_{2\,peak}$ from 2-min to 8-min and 20-min to 45-min compared postexercise recovery following 85% $\dot{V}O_{2\,peak}$, and at 2-min and 25-min to 40-min compared to recovery.

DISCUSSION

The most important observation of this study that in relation to an exercise-induced decrease in the magnitude of the postexercise MAP, we showed a parallel decrease in the level of skin blood flow and sweating. We also noted that the duration of the postexercise attenuation of skin blood flow and sweating was more enduring with increasing exercise intensity. Furthermore, the magnitude of the postexericse esophageal temperature elevation was greater with increasing exercise intensity. These findings support the recent observations by Kenny et al. (15) who showed that the magnitude of the esophageal (and muscle tissue) temperature elevation during the postexercise period is dependent upon exercise intensity and consequently the magnitude of postexercise hypotension in males. Our observations lend further support to the concept of a non-thermal baroreceptor-mediated attenuation in heat loss responses during recovery in the upright position.

Exercise intensity is known to influence the magnitude of the postexercise postexercise hypotension and heat loss responses (6, 15, 19). A study by Forjaz et al. (15) demonstrated that postexercise hypotension is greater and longer after more intense exercise. They noted that while following light exercise, SV increases and HR returns to baseline resting values, SV is reduced and HR remains elevated after more intense exercise bouts. In the following study, we did not see a significant difference in CO between the three intensities after the first 5-min of recovery reflecting the compensatory changes in SV and HR. These findings support the concept of greater deactivation of the arterial barorelfex during recovery from exercise at higher intensities.

This study further supports a nonthermal contribution to the control of skin blood flow and sweat rate during the postexercise period. Upright dynamic exercise provides not only an

endogenous thermal challenge, but also a postexercise orthostatic challenge. That is, the high degree of blood pooling in the previously active musculature provides a significant additional decrease in central blood volume. This effect has been noted previously in the form of a postexercise hypotensive period as discussed above. Previous studies have shown that acute reductions in central venous pressure delay or decrease the rise in skin blood flow and sweating that occur with an elevation in core temperature (15). Our data demonstrate that the increase in the postexercise hypotensive response, induced by exercise of increasing intensity, was paralleled by an increase in both the magnitude of the postexercise elevation in esophageal temperature and the level of skin blood flow and sweat rate. The observed postexercise hypotension is most likely to have been caused by a decrease in total peripheral resistance occurring immediately following exercise cessation. The acute decrease in total peripheral resistance is due in part to a sustained elevated skin blood flow in the early minutes of exercise recovery and a persisting venous pooling in the previously active musculature. The resultant decrease in central venous pressure results in an unloading of the cardiopulmonary and sinoaortic baroreceptors, which evokes a non-thermal baroreceptor-mediated response aimed at reducing skin blood flow in order to increase total peripheral resistance (and therefore central venous pressure). However, the postexercise hypotension condition would be manifested for a prolonged period during postexercise resting if, despite cutaneous vasoconstriction, muscle tissue vasodilation remained. This non-thermal baroreflex response to lower extremity venous pooling has been observed in the absence of exercise during the application of lower-body negative pressure (Crandall et al. 1996).

Our observations, in conjunction with recent observations of a baroreceptor mediated influence on postexercise skin blood flow and sweating provides further evidence to suggest

that postexercise temperature regulation may be preceded by the need to regulate postexercise blood pressure. Thus, in the face of a sustained postexercise hypotension, it is possible that the magnitude of the esophageal temperature elevation is defined by the residual heat load of the previously musculature. This is supported by the recent observations by Kenny et al. (2006), that postexercise resting muscle temperature remains elevated above esophageal temperature for a prolonged period postexercise and the magnitude of the elevation is exercise intensity dependent.

The postexercise hemodynamic response, as represented by heart rate and mean arterial pressure, is consistent with postexercise hypotension (10, 52, 66). There have been many reports of reduced tolerance in the face of an orthostatic challenge in women as compared to men (11, 18, 25, 91, 101). This response is subsequent to a reduced mean arterial pressure and reduced compensatory vasoconstriction in women than in men (2). Implicit in the evidence that women have reduced tolerance to orthostatic challenge is the observation that women regulate blood pressure via different mechanisms (5, 88). Moreover it has been shown that women have less effective baroreflex buffering of arterial blood pressure as compared to men (9). It has also been shown that females appear to have an attenuated responsiveness in the mechanisms that regulate arterial pressure (11, 25). Although this study did not specifically examine sex-related differences, we noted that the magnitude of decrease in MAP postexercise were similar to previous reports comparing males and females. It is plausible therefore, that given the orthostatic effect of the upright seated posture during exercise recovery employed in our study in combination with the decreased responsiveness of cardiovascular mechanisms to regulate arterial pressure in women that this may have resulted in the greater postexercise hypotension observed in this study.

We conclude that following cessation of exercise heat loss responses are compromised secondary to nonthermal baroreceptors influences. Specifically, at higher exercise intensities there is a greater decrease in MAP which is associated with a greater increase in esophageal temperature during the postexercise period. In addition, CVC and sweating return to pre-exercise values quicker during recovery from exercise at higher intensities despite a sustained increase in esophageal temperature. Future studies should be conducted to examine possible sex-related differences in the magnitude of this response.

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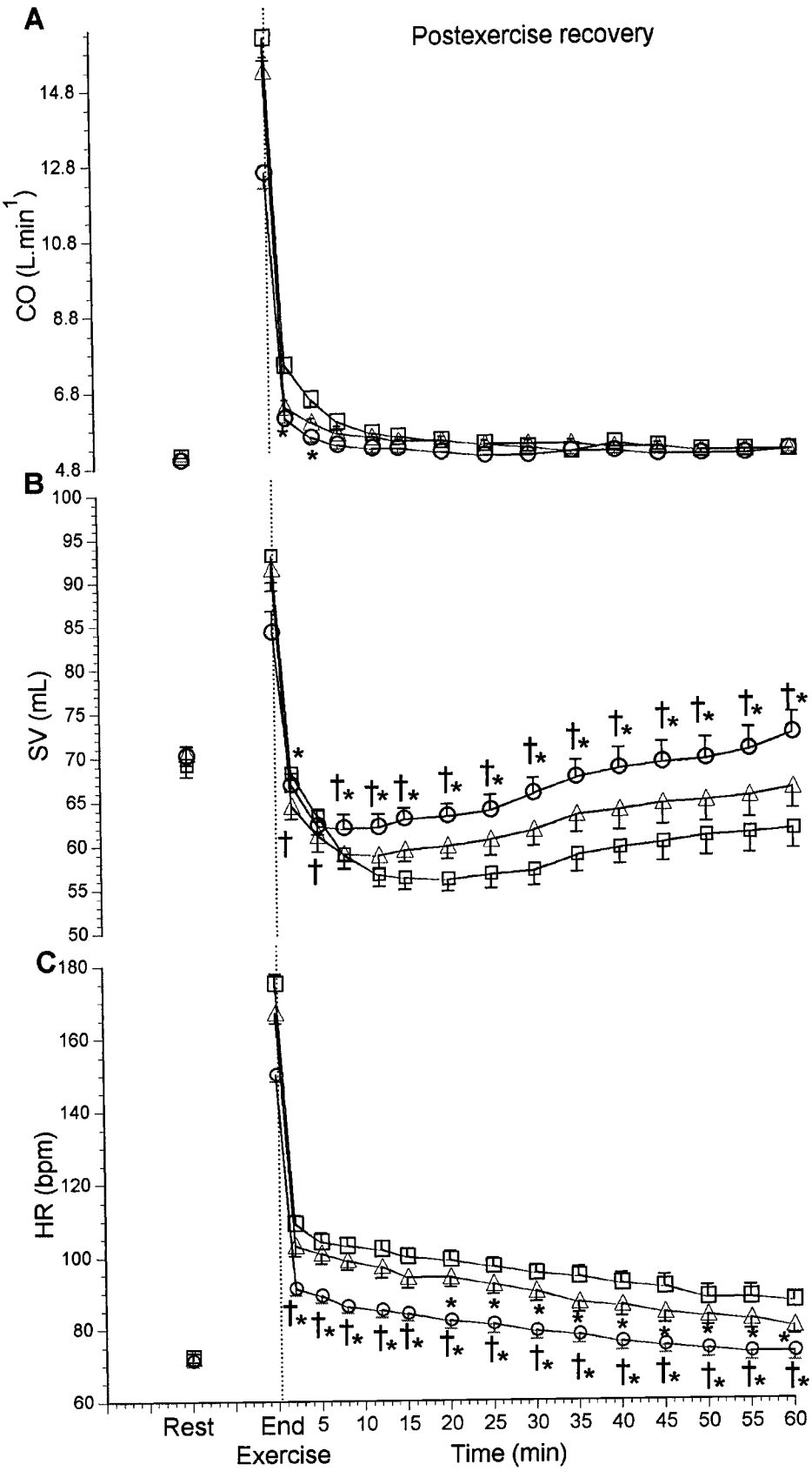
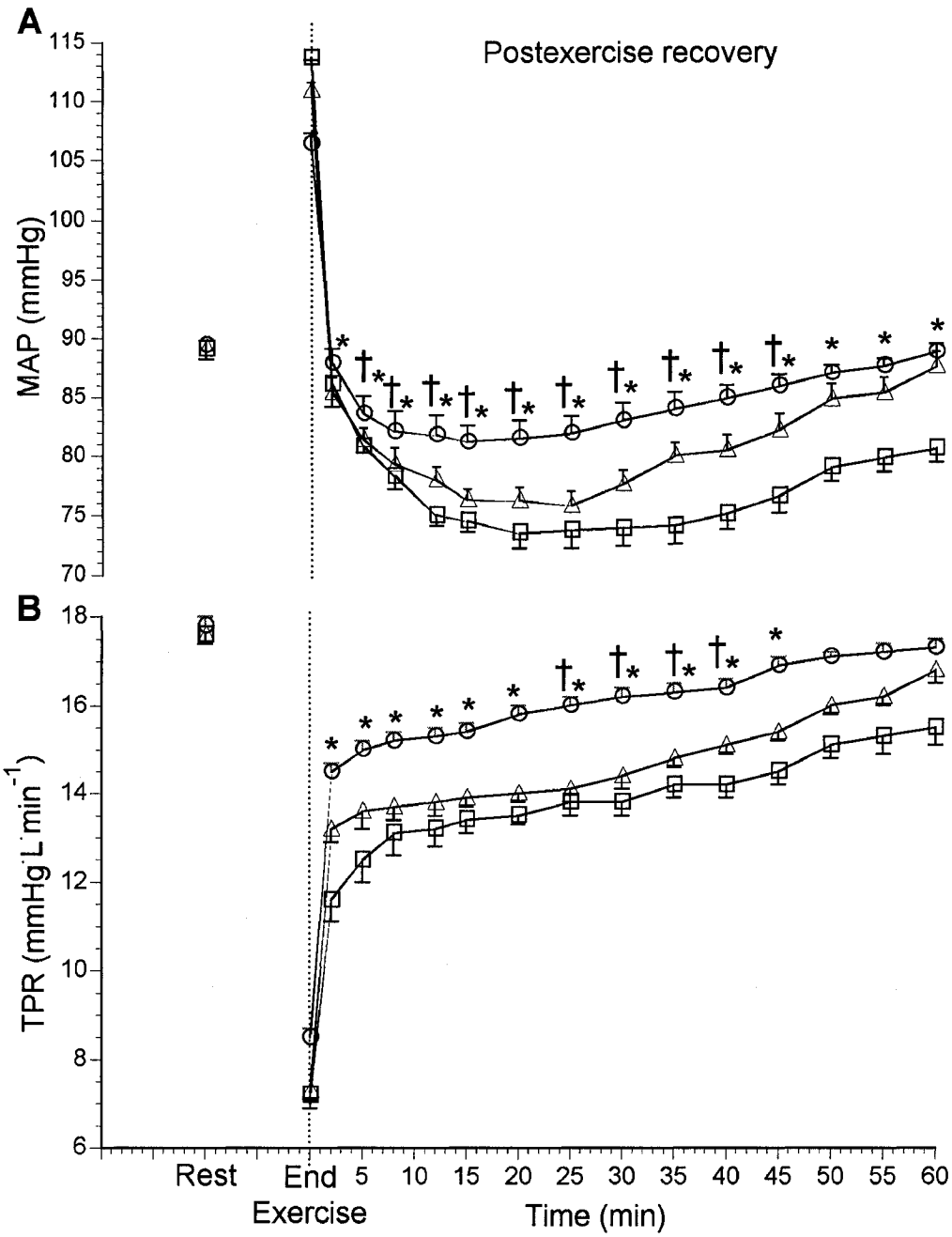
FIGURES

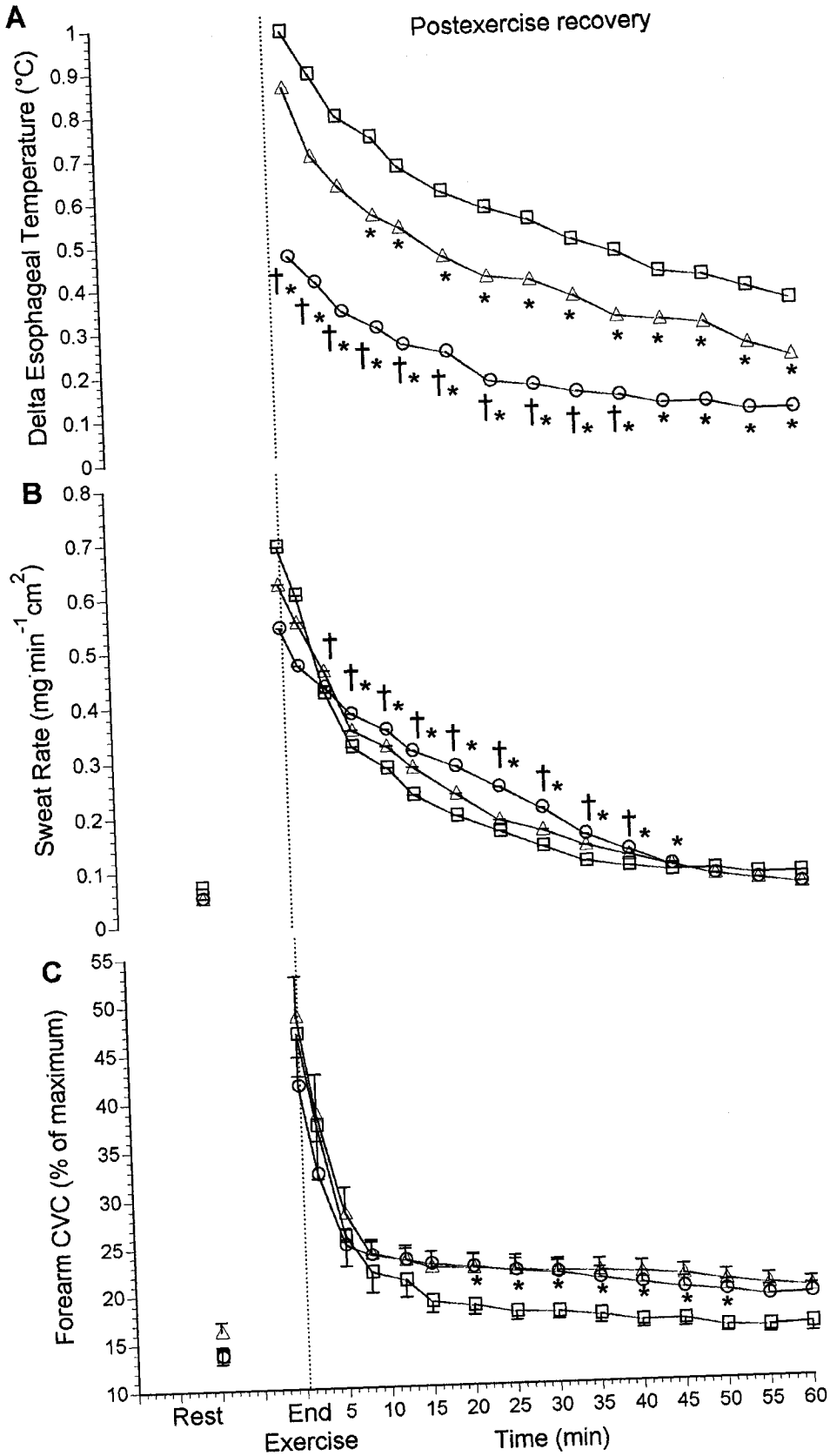
Figure 1. Recovery in the URS position following exercise on a cycle ergometer at 85% $\dot{V}O_{2peak}$ (□), 75% $\dot{V}O_{2peak}$ (Δ) and 55% $\dot{V}O_{2peak}$ (○) on A) cardiac output, B) stroke volume and C) heart rate. Data expressed as means ±SE. * denotes significant difference from 55% $\dot{V}O_{2peak}$ † denotes significant difference from 75% $\dot{V}O_{2peak}$.

Figure 2. Recovery in the URS position following exercise on a cycle ergometer at 85% $\dot{V}O_{2peak}$ (□), 75% $\dot{V}O_{2peak}$ (Δ) and 55% $\dot{V}O_{2peak}$ (○) on A) mean arterial pressure and B) total peripheral resistance. Data expressed as means ±SE. * denotes significant difference from 55% $\dot{V}O_{2peak}$ † denotes significant difference from 75% $\dot{V}O_{2peak}$.

Figure3. Recovery in the URS position following exercise on a cycle ergometer at 85% $\dot{V}O_{2peak}$ (□), 75% $\dot{V}O_{2peak}$ (Δ) and 55% $\dot{V}O_{2peak}$ (○) on A) esophageal temperature, B) sweat rate and C) cutaneous vascular conductance. Data expressed as means ±SE. * denotes significant difference from 55% $\dot{V}O_{2peak}$ † denotes significant difference from 75% $\dot{V}O_{2peak}$.


A
Postexercise recovery
CO (L.min-1)
14.8
12.8
10.8
8.8
6.8
4.8
B
SV (mL)
100
95
90
85
80
75
70
65
60
55
50
C
HR (bpm)
180
160
140
120
100
80
60
Rest
End Exercise
5 10 15 20 25 30 35 40 45 50 55 60
Time (min)


A
Postexercise recovery
MAP (mmHg)
115
110
105
100
95
90
85
80
75
70
B
TPR (mmHg·L·min^{-1})
18
16
14
12
10
8
6
Rest
End Exercise
5
10
15
20
25
30
35
40
45
50
55
60
Time (min)


A
Postexercise recovery
Delta Esophageal Temperature (°C)
B
Sweat Rate (mg·min-1·cm2)
C
Forearm CVC (% of maximum)
Rest
End Exercise
Time (min)

PART THREE:

DISCUSSION OF THE ARTICLES

7.0 DISCUSION

When individuals are faced with heat stress the primary mechanism for heat dissipation are increases in sweating and skin blood flow. During recovery from exercise these mechanisms are compromised and core temperature remains elevated for a prolonged period of time. Recent studies have suggested that this increase in esophageal temperature is associated with cardiovascular changes that occur after dynamic exercise.

During recovery from exercise there is a marked decrease in MAP, commonly referred to as postexercise hypotension. Postexercise hypotension results from the combined effects of persistent neural and vascular adjustments as well as the seated posture. A decrease in MAP during the postexercise period unloads barorecpetors, which initiates reflexes that attempt to reestablish blood pressure. The focus of this thesis was to determine if theses reflexes have a significant effect on postexercise heat loss response.

There seems to be an overall consensus that barorecptors are capable of modulating cutaneous vascular conductance, which is reasonable due to the fact that skin blood flow is on the efferent arm of the baroreflex. On the other hand, influence of baroreceptors on sweat rate remains controversial. However, the results from the three studies examined in this thesis support a barorecpetor influence on sweat rate. The controversy with sweat rate could lie in the duration of the recovery period. Previous studies (Shibasaki et al., 2004; Wilson et al., 2003) have ruled out barorecpetors as a possible influence on sweat rate but they only studied the response for a short duration the present study focused on extended recovery from dynamic exercise.

The first study investigated the effect of head down tilt on postexercise heat loss and hemodyanmic responses. Specifically, the effect of increasing venous return and thereby

attenuating baroreceptor unloading associated with recovery from exercise in the upright-seated position. The results of this study illustrate that HDT attenuates the reduction in MAP, CVC and sweating thereby significantly increasing the rate of esophageal temperature recovery. This study further supports a baroreceptor-mediated attenuation in heat loss responses during the upright-seated position.

The following two studies evaluate postexercise heat loss and hemodyanmic responses in females. Previous studies have shown that males and females react differently to the same degree of orthostatic stress. Carter and colleges (2001) found that females had a greater drop in stroke volume and less of an increase in total peripheral resistance compared to males even though females had a greater drop in MAP. Therefore males and females may differ in their baroreflex response to orthostatic stress. Despite these differences females have not received a lot of attention in this field. The following two studies look exclusively at females in an attempt to characterize the effect of baroreceptors on postexercise heat loss.

The second study in this set of work evaluated the contribution of barorecpetors to the control of skin blood flow and sweating during the postexercise period. The HDT technique was used to reduce the hydrostatic forces present in the upright-seated position. Results showed a significant increase in CVC, SV, MAP and sweat rate and a significant decrease in HR compared to recovery in the upright-seated position. Consequently, esophageal temperature returned to baseline values quicker during recovery in the head down tilt position. Thus this study concluded that heat loss responses are compromised in the upright-seated position secondary to nonthermal baroreceptor influences.

The final article examined exercise intensity and its effect on postexercise heat loss in females. The most important observation of this study is that as exercise intensity increases

CVC and sweat rate decrease, as a result the magnitude of esophageal temperature increases. We suggested that this relationship is due to increasing degrees of baroreceptor unloading during recovery from exercise at higher intensities. This concept is support by the hemodyanmic data during recovery, as exercise intensity increased SV and MAP significantly decreased and HR significantly increased. As a consequence CVC and sweat rate are attenuated and the magnitude of the core temperature elevation is increased.

These studies further support the notion that nonthermal influences involved in blood pressure regulation have a significant effect on postexercise heat loss. Specifically, baroreceptors play an important role in the preservation of CVC and sweating during recovery from dynamic exercise which significantly effects esophageal temperature recovery time.

PART FOUR: REFERENCES

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