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Postexercise hemodynamics and control of heat loss responses following exercise-induced hyperthermia

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Postexercise hemodynamics and control of heat loss responses following exercise-induced hyperthermia

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B.Sc., University of Ottawa, 2005

THESIS

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ABSTRACT

Purpose: The purpose of this thesis was to investigate the nonthermoregulatory control of cutaneous vascular conductance (CVC) and sweating during recovery from exercise-induced hyperthermia as well as to determine possible sex-related differences during the recovery period. It was hypothesized that an active and passive recovery would maintain mean arterial pressure (MAP), CVC and sweat rate at higher levels than an inactive recovery and result in a faster rate of esophageal temperature (T_{es}) decay. It was also hypothesized that changes in MAP, CVC and sweat rate would be sex dependent. **Methods:** Eighteen participants (9 males, 9 females) were rendered hyperthermic by exercise (i.e. T_{es} = 39.5°C) and recovered in one of three recovery modalities for 60-min: 1) active, 2) inactive or 3) passive. T_{es}, CVC, sweat rate, cardiac output, stroke volume, heart rate, total peripheral resistance, and MAP were recorded at baseline and 2, 5, 12, 20 and every 10-min until the end of recovery. **Results:** Both active and passive recoveries were equally effective in maintaining MAP, CVC and sweat rate at greater levels compared with an inactive recovery ($p \leq 0.05$). A significantly lower T_{es} was subsequently observed during passive recovery at 20-min and for the rest of recovery compared to the active mode ($p \leq 0.05$). Sex did not affect any of the measured variables at any time point during any recovery mode, with the exception of sweat rate which was significantly higher in males throughout the recovery period ($p \leq 0.05$). **Conclusion:** We conclude that despite an important thermal drive, nonthermal input remains an important influence in the modulation of postexercise heat loss responses. Further, action of the muscle pump/mechanoreceptors is the main nonthermal determinant in the postexercise modulation of MAP, CVC and sweat rate irrespective of sex.

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CHAPTER 1

INTRODUCTION

1.0 Introduction

Dynamic exercise can result in altered hemodynamics and heat loss mechanisms during the postexercise period. These alterations appear to be related to the ensuing postexercise hypotension which is thought to be the cause of postexercise syncope and orthostatic intolerance. This is of particular importance in the occurrence and treatment of exercise-induced hyperthermia (EIH). EIH is a serious heat illness resulting from excessive heat storage during exercise, particularly in conditions of high ambient temperature and relative humidity. EIH occurs when the thermoregulatory system is overwhelmed by excessive endogenous heat production and inhibited heat dissipation due to ambient conditions (i.e. high temperature and high relative humidity) (Binkley *et al.*, 2002). EIH is primarily observed in apparently healthy and physically fit populations such as high-profile athletes and military soldiers (Binkley *et al.*, 2002; Carter *et al.*, 2005).

The incidence of morbidity and mortality during EIH is directly related to the duration and intensity of the hyperthermic state. Therefore the strategy of any treatment should be aimed at cooling the body as fast as possible (Proulx *et al.*, 2006). However, studies evaluating different recovery treatments from EIH have been primarily conducted on male subjects. Considering females have different physiological responses to recovery from exercise, and different responses to heat stress, it is important to include females in order to allow for possible comparisons between sexes. As well, to date, the method of choice for the treatment of individuals rendered hyperthermic by exercise relies on cooling methods, such as cold water immersion (Proulx *et al.*, 2003). However, this method may not be the most practical

treatment strategy to employ in a field setting. Therefore, it is important to examine other recovery modalities which have been shown to maintain heat loss responses during recovery from exercise in normothermic individuals, such as active recovery. To our knowledge, no studies have examined different recovery modalities in the treatment of EIH or possible sex-related differences after EIH.

Control of the heat loss responses of sweating and skin blood flow (SkBF) have been studied extensively during short duration moderate intensity exercise performed in thermoneutral ambient conditions. While it is well known that during exercise SkBF and sweating can be initiated via hypothalamic activity, there is evidence that they are also subject to nonthermoregulatory influences (see figure 1) such as central command, baroreflexes, skeletal muscle pump, mechanoreceptors and metaboreceptors (Johnson, 1986; Shibasaki *et al.*, 2003; Shibasaki *et al.*, 2004). Central command involves descending neural signals from the brain which recruit skeletal muscle to contract and activate brainstem centers that control autonomic neural outflow directed to the cardiovascular system (Raven *et al.*, 1997). Baroreflexes are reflex responses caused by baroreceptors. Baroreceptors are mechanically sensitive receptors located in the aortic and carotid sinuses which are sensitive to arterial blood pressure (arterial baroreceptors) and changes in central venous pressure (cardiopulmonary baroreceptors) (Raven *et al.*, 1997). These baroreceptors sense changes in arterial blood pressure or central venous pressure such that reductions or increases in arterial blood pressure or central venous pressure will result in a reflex tachycardia and peripheral vasoconstriction or a bradycardia and peripheral vasodilation respectively (Raven *et al.*, 1997). The skeletal muscle pump is the mechanism by which muscular contractions create intramuscular pressure in order to pump blood from the periphery back to the central circulation and is a major determinant in increasing venous return during exercise (Carter *et al.*, 1999). Muscle

mechanoreceptors and metaboreceptors are neural afferents in skeletal muscle which are sensitive to changes in muscle tension and metabolites in the muscle and may contribute to cardiovascular responses to exercise (Rowell & O'Leary, 1990; O'Leary, 1996). Through efferent and afferent feedback, these nonthermal mechanisms also stimulate the cardiorespiratory centers of the brainstem and therefore also influence the control of mean arterial pressure (MAP). At the cessation of exercise there is a reduction in the nonthermoregulatory activity of central command, skeletal muscle pump, and muscle mechanoreceptor and metaboreceptor feedback. Marked reductions in these inputs are accompanied by a drop in MAP from exercise values which is exacerbated in hyperthermic individuals (Kraning & Gonzalez, 1991). Thus, postexercise SkBF and sweating may only be under baroreceptor influence during an inactive recovery (Carter *et al.*, 2002).

• **Mechanoreceptors:**	- Neural afferents in skeletal muscle - Sensitive to changes in muscle tension
• **Muscle Pump:**	- Effect of muscle contractions - Creates an intramuscular pressure - Important in increasing venous return
• **Metaboreceptors:**	- Neural afferents in skeletal muscle - Sensitive to changes in metabolites in the muscle
• **Central Command:**	- Descending neural signals from motor centers in the brain - Recruit Skeletal muscle to contract - Activate brainstem centers that control autonomic neural outflow directed to the cardiovascular system
• **Baroreceptors:**	- Mechanically sensitive receptors - Baroreceptors (arterial blood pressure) - Cardiopulmonary baroreceptors (central venous pressure)

Figure 1. Nonthermal influences

A number of studies have attempted to differentiate the relative roles of central command, muscle pump, mechanoreceptors and baroreceptors in modulating SkBF and sweating during exercise recovery by the use of different recovery modes in normothermic individuals. The different recovery modes have been used to study nonthermal influences on heat loss responses. During active recovery (loadless pedaling) skeletal muscle pump/mechanoreceptors and central command are activated, during passive cycling skeletal muscle pump/mechanoreceptors are stimulated without the involvement of central command (Nobrega & Araujo, 1993; Nobrega *et al.*, 1994; Williamson *et al.*, 1997; Carter *et al.*, 1999) and during inactive recovery baroreceptors are primarily implicated. While baroreceptors are implicated in each recovery mode, it is believed they are the primary influence on SkBF and sweating in the inactive mode (Carter *et al.*, 2002; Journeay *et al.*, 2004b; Kenny & Journeay, 2005), however the relative role of baroreceptors in individuals rendered hyperthermic remains unresolved. Thus, the relative effects of active, passive and inactive recovery modes, and by extension the roles of central command, skeletal muscle pump/mechanoreceptors and baroreceptors can be evaluated as a means of treatment modality for hyperthermic individuals.

The recovery mode studies (Carter *et al.*, 2002; Journeay *et al.*, 2004b; Wilson *et al.*, 2004; Journeay *et al.*, 2005) similarly suggested that SkBF is at least partially modulated by baroreceptors and skeletal muscle pump/mechanoreceptors. Control of sweating is influenced by such other factors as central command, as well as skeletal muscle pump/mechanoreceptors and baroreceptors. However, to date no studies have examined these responses in individuals rendered hyperthermic (i.e. core temperature greater or equal to 39.5°C) but rather in normothermic individuals (i.e. core temperature less than 38.5°C). Both SkBF and sweating responses are significantly compromised in these conditions and the effect is more pronounced in adverse

environments. To date, it remains to be determined if the combined effects of the exercise induced heat stress and the resultant postexercise hypotension may affect the relative roles of nonthermal influences on SkBF and sweating responses. Also, while these studies have demonstrated that both active and passive recovery modes can preserve skin blood flow and sweating responses, it remains to be determined whether this response can significantly affect core temperature reduction in a prolonged recovery. Journeay et al. (Journeay *et al.*, 2004b; Journeay *et al.*, 2005) and Shibasaki et al. (Shibasaki *et al.*, 2004) employed recovery modes of 15-min and 20-min respectively, and did not report significant changes in core temperature between modes.

There is a possible inherent bias in these studies in that they were either conducted on male subjects exclusively or did not include enough females to determine possible sex-related differences. There have been many reports of reduced tolerance to orthostatic challenge in females compared to males (Convertino, 1998; Gotshall, 2000; Shoemaker *et al.*, 2001; Fu *et al.*, 2004). Carter et al (Carter *et al.*, 2001) suggested that women may be more susceptible to postexercise orthostatic hypotension and that active recovery should reduce that risk. They observed a greater reduction in MAP and less compensatory vasoconstriction in women than in men (Carter *et al.*, 2001). In their study however, the participants performed only 3-min of low intensity exercise. It remains to be determined whether the same response occurs after longer more strenuous exercise performed in hot ambient conditions resulting in EIH. Further, it is unknown whether these observed cardiovascular sex-related differences will affect the relative roles of nonthermal influences on SkBF and sweating responses in individuals rendered hyperthermic.

1.1 Rationale

To date, little is known about the interactions between postexercise cardiovascular adjustments and heat loss responses in individuals rendered hyperthermic by exercise. Further, it remains to be determined if there exists sex-related differences in these responses, as previously shown to occur following short duration exercise (i.e., 15min) performed in thermoneutral conditions (i.e., 24°C) (Journeay *et al.*, 2004b; Journeay *et al.*, 2005). Additionally, the hemodynamic responses to different recovery modes and by extension the role of nonthermal factors on postexercise temperature regulation in males and females has not been examined in individuals rendered hyperthermic. Therefore, the purpose of this study is to investigate the nonthermoregulatory control of cutaneous vascular conductance and sweating during recovery from dynamic exercise in individuals rendered hyperthermic. This will be done with the use of three different recovery modalities: 1) active recovery (loadless pedaling), 2) passive cycling and 3) inactive recovery.

1.2 Hypotheses

1.2.1 General hypothesis of the thesis

It is hypothesized that, considering sex-related differences in thermoregulatory and postexercise thermoregulatory responses, females will demonstrate different physiological responses to recovery from exercise-induced hyperthermia. Also, the different recovery modalities will result in different hemodynamic responses during recovery from exercise.

1.2.2 Specific hypotheses of the thesis

It is hypothesized that females will show greater reductions in mean arterial pressure during recovery from exercise-induced hyperthermia and will show a greater decrease in cutaneous vascular conductance, and sweat rate. It is also hypothesized that an active and passive recovery modality will be effective in maintaining

cutaneous vascular conductance, mean arterial pressure, and sweat rate at a greater level than an inactive recovery irrespective of sex. It is also hypothesized that an active and passive recovery will result in a greater heat loss response and a faster core temperature recovery time.

1.3 Statement of the problem

Although postexercise thermoregulatory responses in normothermic conditions have been extensively studied, little is known about the postexercise thermoregulatory responses during recovery from EIH. As well, it is not known if possible sex-related differences exist during recovery from EIH. Additionally, the hemodynamic responses to active, inactive, and passive recovery and by extension the roles of the skeletal muscle pump/mechanoreceptors, central command, and baroreceptor response in males and females have not been examined in individuals rendered hyperthermic.

1.4 Objectives

To investigate the nonthermoregulatory control of cutaneous vascular conductance and sweating during recovery from dynamic exercise in individuals rendered hyperthermic, by the use of three different recovery modalities: 1) active recovery (loadless pedaling), 2) passive cycling, and 3) inactive recovery and to determine possible sex-related differences during recovery from EIH.

1.5 Relevance

This study will advance our understanding of the different mechanisms that regulate body temperature during recovery from exercise-induced hyperthermia which could lead to effective treatments for individuals suffering from EIH. As well, this study will serve to identify possible sex-related differences during recovery from EIH.

1.6 Delimitations and limitations

There will be an imposed restriction as to the degree of hyperthermia brought about by exercise. Subjects will be limited to a target core temperature (i.e. esophageal) of 39.5°C. Therefore, the results of this study will be limited to individuals suffering mild hyperthermia as compared to severe hyperthermia (i.e. core temperature > 40.0°C). Subjects recruited for the study will be aged between 18 and 35 years physically active and lean individuals. Therefore, the results of this study cannot apply to children, the elderly or to a sedentary or obese population. Females will be tested during the follicular phase of their menstrual cycle and will not be tested in different phases of their menstrual cycle phase; therefore it will not be possible to determine possible effects of menstrual cycle phase on EIH.

CHAPTER 2

REVIEW OF LITERATURE

2.1. Thermoregulation

2.1.1 Control

Central to man's ability of maintaining a fairly constant body temperature despite wide variations in the external environment is that of the set-point control theory (Hardy, 1961; Hammel, 1968; Benzinger, 1969). The set-point control theory can be understood as a closed-loop feedback regulator (Hammel, 1968) that relies on three components: an integrator, a sensor and an effector. The integrator acts as a control unit and receives information from the sensor. As the sensor transmits information from its environment to the integrator, the integrator will, if needed, send information to the effector which in turn will activate the necessary actions to restore homeostasis. The elicited responses from the effector will feedback to the integrator through the sensor, and if the desirable response was obtained, the integrator will cease sending information to the effector which will discontinue its activity. When applied to human thermoregulation, the set-point control theory can be viewed as having an integrator being located in the preoptic/anterior hypothalamus (PO/AH) in the brain (Boulant, 2000) acting as a thermostat. Increases and decreases in body temperature are sensed peripherally by skin thermoreceptors and centrally by thermoreceptors located in the hypothalamus, the nervous system, blood vessels and the abdominal cavity (Brooks *et al.*, 2000). The peripheral and central thermoreceptors transmit information by nerve impulses via the spinal cord to the PO/AH (Brooks *et al.*, 2000) where responses are generated in order to maintain a constant body temperature. Deviations above or below the set-point temperature will initiate heat loss or heat gain mechanisms respectively. If body temperature increases

above that of the central set-point temperature, the PO/AH will initiate thermolytic activity to decrease body temperature by increasing heat dissipation via cutaneous vasodilation and increased sweating rate (Hardy, 1961; Hammel, 1968; Benzinger, 1969). If body temperature decreases below that of the set-point, the PO/AH will initiate thermogenic activity to increase body temperature, by decreasing heat dissipation via cutaneous vasoconstriction and increasing heat production through shivering thermogenesis (Hardy, 1961; Hammel, 1968; Benzinger, 1969). More recently, it was suggested that regulation of body temperature does not evolve around a set-point core temperature, but rather within a zone determined by an upper and lower core temperature limit (Mekjavic *et al.*, 1991; Mekjavic & Eiken, 2006). This theory implies that effectors regulating body temperature become active above or below a certain core temperature threshold which is different for thermogenic and thermolytic mechanisms. For example, if the core temperature threshold for thermogenic activity (i.e. shivering) is ~36.9°C and that of thermolytic activity (i.e. sweating and skin blood flow) is ~37.4°C, when core temperature decreases below 36.9°C, thermogenic mechanisms will be activated and when core temperature rises above 37.4°C thermolytic responses will be activated. On the other hand, when core temperature is situated between ~36.9°C and ~37.4°C, no thermolytic or thermogenic mechanism is stimulated (the "null zone") and body temperature is regulated by vasomotor tone (Mekjavic *et al.*, 1991; Mekjavic & Eiken, 2006).

In contrast to the set-point theory, Webb (Webb, 1995) has recently suggested that thermoregulation does not evolve around controlling body temperature, but rather around body heat content. Although his proposed theory relies on a similar control theory to the one proposed for the set-point theory, the controlled variable in body heat regulation is not body temperature but rather body heat content, which is affected by metabolic heat production and external thermal stimuli (Webb, 1995). In fact,

Webb suggests that changes in body temperature reflect changes in body heat content. Therefore, considering that the body continuously produces heat, adjustments in heat loss are the main effectors in order to control body heat content (Webb, 1995). According to Webb, the controller does not serve to defend a set hypothalamic temperature, but rather responds to changes in outflow and inflow of body heat, with sweating, vasodilation, vasoconstriction and shivering as the main effectors and body heat content, integrated body temperature and heat loss providing the main feedback to the controller (Webb, 1995).

Although many investigations have attempted to elucidate how the human body regulates its body temperature, or even its body heat content, the exact processes behind human thermoregulation are still unclear and remain a matter of debate in the literature.

2.1.2 Heat exchange mechanisms

Although the above explanations of thermoregulatory control theories offer different explanations of how body temperature (or heat) is regulated, it does not give any information on how heat is exchanged between the body and its environment or within the body itself. Human thermoregulation is largely dependent on internal heat exchanges within the body between the different tissues and compartments as well as between the skin surface and the ambient environmental conditions. The mechanisms of energy exchange involved in thermal homeostasis include internal exchanges by convection and conduction and external exchanges by radiation, conduction, convection and evaporation. Conduction is the mechanism by which energy (i.e. heat) is transferred between two surfaces in contact with one another (Keim *et al.*, 2002) depending on the temperature gradient between the two surfaces. Radiation is the mechanism by which heat is transferred between the body and the environment via electromagnetic waves (Keim *et al.*, 2002) depending on the temperature gradient

between the body and the environment. Solar radiation is a good example of a means by which the body can absorb heat by radiation. Evaporation is the mechanism by which heat is lost by evaporation of sweat at the skin surface or through ventilation. Evaporative heat loss depends on the partial pressure of water (P_{H2O}) gradient between the skin surface and the environment (Brooks *et al.*, 2000). Convection is the mechanism by which heat is transferred from a surface to a gas or liquid depending on the temperature gradient and on the rate of motion of the gas (i.e. air) or liquid (i.e. water) and its relative heat storage capacity (Keim *et al.*, 2002). Convection affects the rate of heat flux by conduction and evaporation. The interaction of man with the thermal environment is conceptually demonstrated using the human heat balance equation.

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

Where:
M = rate of metabolic heat production
W = rate of mechanical work
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 $W \cdot m^{-2}$)

2.2. Exercise and thermoregulation

2.2.1 Control and mechanisms

At the onset of exercise, increased metabolic and muscular activity causes an important rise in heat production. This is mainly due to the inefficiency of the metabolic reactions required to provide energy to the working muscles (Gleeson, 1998). In fact, the human mechanical efficiency is situated at approximately 20%, meaning that if 20 kJ is produced for every litre of oxygen consumed, only 4 kJ will be used for mechanical work, and the other 16 kJ will be produced as heat (Gleeson, 1998). Considering that trained individuals can sustain workloads of 800 to 1000 kcal

per hour for a prolonged period of time, the associated heat production with these workloads would be sufficient to increase body temperature 1°C every 5 to 8 minutes if the heat load was stored in the body (Nadel, 1984). Fortunately, under normal ambient conditions, the human thermoregulatory system is capable of matching the heat production with an increased heat loss in order to effectively dissipate excess heat production and avoid hyperthermia.

The increased heat production by the active muscles at the onset of exercise is not immediately matched by an increased heat loss response (Webb, 1995). In fact, much of the heat produced in the early stages of exercise will be stored in the active muscles and muscle temperature will increase (Nadel, 1984). As muscle temperature increases, heat transfer within the body will occur in two ways. First, sympathetic activity increases as muscular contractions increase and blood redistribution towards the working muscles occurs as vasoconstriction in the renal and splanchnic circulation will account for most of the redistribution of blood to the working muscles (Rowell, 1974). Therefore, the increased muscle blood flow will allow for a greater conductive heat exchange between the working muscle and the blood as the convective properties of the blood will increase with increased blood flow through the capillary beds of the muscles (Nadel, 1984). This increased blood flow usually results in a temporary decrease in core temperature (~0.2°C) at the onset of exercise, as cool blood from the periphery mixes with warmer blood from the core (Greenleaf, 1979).

Second, heat is passively transferred from the working muscle to the surrounding tissues and compartments by conduction. As heat is transferred from the working muscles to the blood and to the surrounding tissues, the rise in heat storage of the active muscle will eventually be attenuated and muscle temperature will reach an elevated plateau (Nadel, 1984). As heat is stored in the core, core temperature will rise, causing increased cutaneous vasodilation and skin blood flow (Johnson, 1992;

Charkoudian, 2003). Therefore, the warm blood returning to the core from the active muscles and heated core will be redirected towards the skin permitting transfer of heat by conduction, radiation or evaporation with the surrounding environment. This redistribution of blood to the skin is mainly achieved with an increased cardiac output and the previously mentioned redistribution of blood from the renal and splanchnic circulations (Charkoudian, 2003). When exercise is performed in cool dry ambient conditions, most of the heat at the skin surface will be dissipated by conduction as the temperature gradient between skin temperature and ambient air temperature will favour heat transfer from the skin to the surrounding environment. If air current over the skin surface increases, heat loss by conduction will be favoured by convection. The main avenue of heat loss during exercise, however, is by evaporation at the skin surface and from the respiratory tract (Buskirk, 1977). Evaporation from the skin surface occurs as sweat is secreted from the sweat glands onto the skin surface, which usually occurs 2 to 5 minutes after the onset of exercise (Buskirk, 1977). The evaporation of sweat from the skin surface requires approximately 0.6 kcal per gram of water (Buskirk, 1977; Nadel, 1984). Evaporation of sweat at the skin surface will cool skin temperature cooling the blood at the skin surface returning to the core (Charkoudian, 2003). These heat loss responses will increase as exercise intensity increases and until heat loss matches heat production which is reflected by an elevated plateau in core temperature (Buskirk, 1977; Greenleaf, 1979; Nadel, 1984; Webb, 1995; Charkoudian, 2003). It is important to mention that the elevated plateau in core temperature is not a result of a failure of the thermoregulatory system, but is rather due to the initial aforementioned lag between heat production and heat loss at the initial onset of exercise (Webb, 1995).

2.2.2 Exercise in the heat

While thermoregulatory and cardiovascular function are well maintained during exercise in thermoneutral conditions, exercise in the heat creates a competition between the cardiovascular system and the thermoregulatory system as cardiac output ($\dot{Q}$) must be partitioned to meet metabolic demands and heat transport to the skin (Rowell, 1974). Maintenance of $\dot{Q}$ during exercise in the heat is maintained by a redistribution of blood which is reflected by an increased heart rate (HR) due to decreases in stroke volume (SV) and central blood volume (CBV) while metabolic demands are maintained by a widening of the arteriovenous oxygen (A-VO_2) difference (Rowell *et al.*, 1966; Rowell, 1974). If exercise persists, maximal HR is reached as SV decreases, therefore $\dot{Q}$ cannot increase and mean arterial pressure (MAP) decreases (Wyndham, 1973; Rowell, 1974). (Wyndham, 1973; Rowell, 1974). Further, if the heat loss responses of skin blood flow (SkBF) and sweating do not match the heat production brought about by exercise, exercise-induced hyperthermia will occur. However, cessation of exercise removes the exercise stimulus central command and skeletal muscle pump/mechanoreceptors during inactive recovery which, combined with the already decreased SV, CBV and MAP and the widened A-VO_2 difference, will result in a pooling of blood in the periphery resulting in an increased risk of syncope during an upright inactive recovery (Wyndham, 1973).

2.3. Thermoregulatory sex-related differences

Several investigations have attempted to identify possible sex-related differences during exercise thermoregulation and heat stress (Wyndham *et al.*, 1965; Morimoto *et al.*, 1967; Weinman *et al.*, 1967; Fox *et al.*, 1969; Rowell, 1974; Drinkwater *et al.*, 1976; Paolone *et al.*, 1978; Davies, 1979; Avellini *et al.*, 1980; Frye & Kamon, 1981; Horstman & Christensen, 1982). Taken together, these

investigations indicate that women usually sweat less than men, have higher heart rates, and have higher core temperatures (Stephenson & Kolka, 1993). However, most of these studies did not take into account menstrual cycle phase or aerobic capacity (Stephenson & Kolka, 1993; Kaciuba-Uscilko & Grucza, 2001).

Core temperature can fluctuate 0.3 to 0.5°C in women according to the menstrual cycle phase, with core temperature being elevated during the luteal phase (Stephenson & Kolka, 1993; Kaciuba-Uscilko & Grucza, 2001). This elevation in core temperature has been attributed to the rise in progesterone secretion at the beginning of ovulation, as progesterone has a thermal effect (Stephenson & Kolka, 1993; Kaciuba-Uscilko & Grucza, 2001). Also, it has been shown that core temperature thresholds for sweating and cutaneous vasodilation are elevated during the luteal phase compared to the follicular phase during exercise (Stephenson & Kolka, 1985; Kolka & Stephenson, 1989). Despite these differences, recent findings suggest that the menstrual cycle does not seem to alter aerobic capacity, heart rate, rate of perceived exertion and exercise performance and there is no evidence to suggest that women in the luteal phase of the menstrual cycle would be at a greater risk of heat stress while exercising in hot environments (Marsh & Jenkins, 2002). Additionally, orthostatic tolerance during passive heating has been shown to be unaltered by menstrual cycle phase (Meendering *et al.*, 2005). Further, cardiovascular and thermoregulatory responses during recovery from exercise have been shown to be unaffected by menstrual cycle phase, despite differences during pre-exercise measurements (Lynn *et al.*, 2007). Therefore, it seems that although there are cardiovascular and thermoregulatory differences across menstrual cycle phase at rest and during exercise, it seems that these differences do not place women at a greater risk for exercise-induced hyperthermia nor at an increased risk of syncope during recovery from exercise while in the luteal phase of their menstrual cycle.

Women tend to have a lower absolute aerobic capacity compared to men (Kaciuba-Uscilko & Grucza, 2001). Considering that thermoregulatory responses while exposed to humid heat depend mainly on aerobic capacity, women may be at a disadvantage when working at the same workload as their male counterparts as greater heat loads would be generated in females (Kaciuba-Uscilko & Grucza, 2001). Also, possible sex-related differences may be due to anthropometric differences, as women have a 10 to 12% greater surface-area to mass ratio (S:M) (Nunneley, 1978). Since heat exchange is dependent on surface contact, this greater S:M is both an advantage and disadvantage for females. In hot environments, a greater S:M would result in a greater net heat gain from the environment, whereas in cool environments it would result in a greater net heat loss from the skin surface to the environment (Burse, 1979). Therefore, women may absorb more heat from the environment in hot conditions or lose more heat in cool environments. Further, it has been suggested that men are wasteful sweaters, whereas women are more efficient as they sweat less, but have more of their sweat evaporate, rather than simply drip off the skin surface (Wyndham *et al.*, 1965; Avellini *et al.*, 1980; Frye & Kamon, 1981). However, when anthropometric differences are taken into account, most of the sex-related differences in thermoregulatory responses tend to narrow or disappear (Kaciuba-Uscilko & Grucza, 2001). Nonetheless, one finding that persists is that women tend to sweat less, while maintaining a similar core temperature to that of men (Kaciuba-Uscilko & Grucza, 2001).

2.4. Postexercise thermoregulation

In keeping with the set-point control theory, one would expect that after raising core temperature (i.e. during exercise), effectors would be active in returning core temperature to pre-exercise (set-point) values after cessation of exercise. However, it has been shown that exercise results in significant perturbations in

thermoregulatory control during the postexercise period. In fact, these perturbations have been shown to result in a prolonged (up to 60-min) postexercise elevation (~0.4-0.5°C) in core (i.e. esophageal) temperature (Thoden *et al.*, 1994). Thoden et al. (Thoden *et al.*, 1994), demonstrated that this elevation was not of metabolic origin as oxygen consumption had returned to baseline values within 5-10 min and that SkBF values had returned to baseline values within 10-15 min. In fact, the postexercise elevation in core temperature was shown to be equal in magnitude to the vasodilation threshold (Th_{vd}) during exercise, suggesting that thermal reflex vasodilation was significantly reduced before pre-exercise resting core temperature could be re-established (Thoden *et al.*, 1994). Kenny et al. (Kenny *et al.*, 1996b) then demonstrated that the postexercise elevation in core temperature was modulated by pre-exercise core temperature by using successive exercise-recovery cycles and reinforced the observation of a direct relationship between postexercise esophageal temperature (T_{es}) and Th_{vd}. In order to demonstrate that this postexercise elevation in core temperature was due to a residual effect of exercise and not due to an increase in body heat content alone, Kenny et al. (Kenny *et al.*, 1996a) compared postexercise thermoregulatory responses subsequent to 15-min of exercise to 15-min of immersion in 42°C water. The results showed that 15-min of treadmill exercise resulted in an elevated postexercise T_{es} compared to pre-exercise values. This was in contrast to the warm water immersion, which did not produce a postexercise elevation in T_{es}, despite a slightly greater heat gain (Kenny *et al.*, 1996a). This study supported the notion that it was in fact exercise that had a residual effect on postexercise core temperature regulation. Subsequent studies showed that this postexercise elevation in core temperature was independent of ambient temperature (Kenny *et al.*, 1997b), exercise intensity (Kenny *et al.*, 1997b), and exercise duration (Kenny *et al.*, 1999).

Although a sustained elevation in the rate of heat loss would be expected in the face of this postexercise elevation in core temperature, several studies have shown a rapid decrease in sweating (Kenny & Niedre, 2002), SkBF (Thoden *et al.*, 1994; Kenny & Niedre, 2002; Wilkins *et al.*, 2004) and skin temperature (Thoden *et al.*, 1994; Kenny & Niedre, 2002; Wilkins *et al.*, 2004) to pre-exercise baseline values within the early stages of recovery. Further, Kenny and Neidre (Kenny & Niedre, 2002) showed that exercise of increasing intensity was paralleled by an increase in the magnitude of postexercise elevation in esophageal temperature. At higher exercise intensity, they observed 1) a greater decrease in postexercise MAP; 2) an overall decrease in the rate of heat loss; and, 3) an increase in the T_{es} recovery time. More recently, studies have shown that exercise results in a residual increase in the T_{es} threshold at which onset for SkBF (Kenny *et al.*, 2000b) and sweating (Kenny *et al.*, 1997a; Kenny *et al.*, 2000b) are initiated. This effect is also relatively greater during recovery from exercise of increasing intensity (Kenny & Niedre, 2002; Kenny *et al.*, 2003a, 2003b). These findings suggest that the magnitude and duration of postexercise T_{es} elevation is associated with the marked cardiovascular changes that occur after dynamic exercise.

2.4.1 Postexercise cardiovascular changes

During the initial phase of exercise recovery, there are dramatic changes in hemodynamic responses subsequent to a change in input from central command, skeletal muscle pump/mechanoreceptors and metaboreceptors. The hemodynamic changes observed are most pronounced in the first 5-10 minutes after exercise. Upon cessation of dynamic exercise during inactive upright recovery, MAP decreases rapidly during the first 1-2 minutes of recovery (Carter *et al.*, 1999) as do SV and $\dot{Q}$ (Carter *et al.*, 2002). In contrast however, total peripheral resistance (TPR)

tends to increase to pre-exercise levels (Carter *et al.*, 1999). Despite increases in TPR, thoracic impedance values indicate a decrease in central blood volume during upright inactive recovery compared to that of active recovery modes (Carter *et al.*, 2002). It is thought that decrements in central blood volume during upright inactive recovery are associated with the accumulation of blood in the venous system of the lower extremities in the absence of the skeletal muscle pump (Carter *et al.*, 1999; Carter *et al.*, 2002). Additionally, persistent vasodilation in the muscles (Piepoli *et al.*, 1993) contributes to lower central blood volume and thus reduced cardiac filling (Halliwill, 2001) in the upright posture.

2.4.2 Postexercise hypotension

A deficit in peripheral resistance can lead to a decrease in MAP. Postexercise hypotension is thought to be caused by profound changes in the mechanisms that regulate and determine MAP resulting in hypotension that is both vascular and neural in origin (Kenney & Seals, 1993; Halliwill, 2001). It should be noted that the magnitude of the reduction in MAP is greater with the orthostatic influence of upright or standing posture than when the subjects are supine (Kenney & Seals, 1993; Halliwill, 2001). The response also appears to become significantly different from pre-exercise values at approximately 20 minutes in both the supine (Pricher *et al.*, 2004; Wilkins *et al.*, 2004) and upright seated postures (Journeay *et al.*, 2004a). Piepoli et al. (Piepoli *et al.*, 1993) suggested there is persistent peripheral vasodilation that may cause pooling of warm blood and thus trap heat in the previously active muscle when upright. Subsequently, work by Halliwill et al. (Halliwill *et al.*, 1996) noted impaired sympathetic vascular regulation after exercise in the supine position and Pricher et al. (Pricher *et al.*, 2004) reported elevated leg vascular conductance up to 100 minutes postexercise also in the supine posture. Thus, the combined effects of persistent neural and vascular adjustments as well as the

upright seated posture with dependent lower extremities and no skeletal muscle pump results in significant pooling of blood (Coats *et al.*, 1989; Kilgour *et al.*, 1993; Raine *et al.*, 2001). The decrease in cardiac filling during inactive recovery is sensed by cardiopulmonary baroreceptors and if there is significant postexercise hypotension then arterial baroreceptors may also be unloaded. Given the considerable cardiovascular adjustments described above, it is essential that the role of hemodynamic changes be assessed in the evaluation of postexercise thermoregulation.

2.4.3Nonthermoregulatory influences postexercise

Regulation of body temperature is mainly controlled by thermal factors (Mekjavic & Eiken, 2006), however recent evidence suggests that such nonthermal factors as central command, skeletal muscle pump/mechanoreceptors, metaboreceptors and baroreflexes affect temperature regulation, especially during the postexercise period. Upon the cessation of exercise, dramatic changes in MAP occur secondary to changes in input from central command, metaboreceptor and skeletal muscle pump/mechanoreceptors feedback, therefore SkBF and sweating may only be under nonthermoregulatory baroreceptor control during recovery (Carter *et al.*, 2002). This is possible since evidence exists for a baroreceptor modulation of SkBF and sweating (Johnson *et al.*, 1973; Kellogg *et al.*, 1990; Mack *et al.*, 1995; Crandall *et al.*, 1996). As well, the postexercise hemodynamic response described above may result in a greater unloading of cardiopulmonary baroreceptors, and possibly arterial baroreceptors depending on the degree of hypotension exhibited in the upright posture.

This baroreceptor mediated influence on postexercise heat loss was first shown in a study by Kenny et al. (Kenny *et al.*, 2000a). In this study, the effects of postexercise blood pooling in the upright posture were reversed by the use of head-down tilt. The results showed that head-down tilt following exercise attenuated the

increase in the postexercise Th_{vd} compared to a head-up tilt condition (Kenny *et al.*, 2000a). Therefore, it was concluded that the reversal of peripheral blood pooling, and hence the baroreceptor unloading, attenuated the effect of exercise on the postexercise Th_{vd}. Further assessments of nonthermal influences on postexercise thermoregulatory control by the use of lower body positive pressure (LBPP) revealed that the changes induced by LBPP, such as an increase in $\dot{Q}$ and MAP, were sufficient to not only significantly reduce the postexercise thresholds for cutaneous vasodilation and sweating (Th_{sw}) (Jackson & Kenny, 2003), but also result in an increased whole body heat loss subsequent to an increase in SkBF and sweating (Jackson & Kenny, 2003; Journeay *et al.*, 2004a). Further, it was shown that postexercise elevations in Th_{vd} and Th_{sw} were not observed during extended recovery (i.e. 60-min) due to the restoration of MAP at the end of the extended recovery compared to a short recovery (i.e. 20-min.) (Kenny & Journeay, 2005). Taken together, these studies reinforce the notion that postexercise thermoregulatory function is greatly dependent on hemodynamic status.

In order to assess the relative roles of nonthermal factors on postexercise thermoregulation, a series of recent experiments were undertaken using different recovery modes as a method for controlling nonthermal inputs (Carter *et al.*, 2002; Journeay *et al.*, 2004b; Shibasaki *et al.*, 2004; Wilson *et al.*, 2004; Journeay *et al.*, 2005). The principle of using the modes of active, inactive and passive recovery in the study designs was to delineate the relative roles of central command, skeletal muscle pump/mechanoreceptors, and baroreceptors in the modulation of sweating and SkBF. During active recovery, skeletal muscle pump/mechanoreceptors and central command are activated (Carter *et al.*, 1999). A passive recovery mode is thought to include the skeletal muscle pump/mechanoreceptors without the involvement of

central command (Nobrega & Araujo, 1993; Nobrega *et al.*, 1994). While baroreceptors are implicated in each recovery mode in the upright posture, it is believed that they are the primary input influencing SkBF and sweating in the inactive mode in the upright posture (Carter *et al.*, 2002; Journeay *et al.*, 2004a; Kenny & Journeay, 2005). The results from these studies demonstrate that both active and passive recovery modes are effective in attenuating the fall in cutaneous vascular resistance (CVC), sweat rate, and MAP compared to an inactive recovery mode. This suggests that attenuating the fall in MAP, and thus the baroreceptor unloading effect, through action of the skeletal muscle pump/mechanoreceptors is the main nonthermal determinant in the maintenance of CVC and sweat rate, not central command. As for sweat rate, these studies suggest that central command and skeletal muscle pump/mechanoreceptors are responsible for the nonthermal modulation of sweat rate during the recovery period. However, the data from one study regarding the passive recovery mode shows that sweat rate fell below the active recovery values but remained elevated compared to the inactive recovery mode (Journeay *et al.*, 2004b). Although it had been suggested that baroreceptor influence would be minimal in affecting sweat rate (Wilson *et al.*, 2004), these results suggest that the role of baroreceptors on the modulation of sweat rate cannot be excluded.

2.4.4 Postexercise thermoregulatory sex-related differences

Much of the current research regarding postexercise thermoregulation contains some inherent bias as only male subjects have been studied or not enough females were included to allow for a comparison. Considering the apparent relationship between cardiovascular adjustments during the postexercise period and their influence on postexercise body temperature regulation, it is important to evaluate the possible sex-related differences that could affect restoration of body temperature. The most notable sex-related difference is the fact that women are generally less

tolerant to orthostatic challenges than men (Convertino, 1998; Gotshall, 2000; Shoemaker *et al.*, 2001; Fu *et al.*, 2004). Moreover, Carter et al. (Carter *et al.*, 2001) showed that women had a greater reduction in MAP and less compensatory vasoconstriction than men following 3-min of low intensity exercise. Also, a recent study by Journeay et al. (Journeay *et al.*, 2005) showed that women exhibited a greater reduction in MAP following 20-min of moderate intensity exercise compared to a previous study done on males (Journeay *et al.*, 2004b). Journeay et al. (Journeay *et al.*, 2005) also demonstrated that control of CVC during the postexercise period is different in women than in men. Their results showed that CVC was regulated by central command in females which was not the case in males (Journeay *et al.*, 2005). However, they showed that the control of sweat rate was similar in women as compared to men (Journeay *et al.*, 2005). In another study, women demonstrated a greater reduction in MAP following 15-min of moderate intensity exercise which led to a greater postexercise Th_{vd} compared to males (Kenny *et al.*, 2005). Therefore, it seems that differences in postexercise temperature regulation stem from the fact that women are more susceptible to postexercise hypotension than their male counterparts.

2.5 Summary

In summary, exercise in the heat creates a competition between the cardiovascular and thermoregulatory system such that, at the cessation of exercise, an inactive upright recovery may result in an increased risk of heat syncope. If maintenance of cardiovascular function overrides thermal input after exercise-induced hyperthermia, such is the case in non-hyperthermic individuals, core temperature would remain elevated during the recovery period which would increase the risk of heat related injury. However, if thermal input overrides nonthermal influences, maintenance of cardiovascular function after exercise-induced hyperthermia would be compromised and syncope would likely ensue. Further, females have been shown to

have a greater reduction in mean arterial pressure and orthostatic tolerance following dynamic exercise and have been shown to differ in postexercise heat loss responses. Therefore, they may be at a greater risk of heat syncope following exercise-induced hyperthermia. Also, it is unkown what role, if any, nonthermal stimuli related to central command, baroreflexes, skeletal muscle pump/mechanoreceptors and metaboreceptors play on hemodynamic and heat loss responses during recovery from exercise-induced hyperthermia and if these responses are affected by sex-related differences. Therefore, it is important to evaluate the competing influences of thermal and nonthermal stimuli during recovery from exercise-induced hyperthermia and to determine any possible sex-related differences in heat loss and hemodynamic responses.

PART TWO:

METHODS AND RESULTS OF THE THESIS

Nonthermal control of heat loss responses following exercise-induced hyperthermia

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Running head: Exercise-Induced Hyperthermia and Recovery

ABSTRACT

This study investigated the nonthermoregulatory control of cutaneous vascular conductance (CVC) and sweating during recovery from exercise-induced hyperthermia as well as possible sex-related differences in these responses. Nine males and nine females were rendered hyperthermic by exercise and recovered in one of three recovery modalities for 60-min: 1) active, 2) inactive or 3) passive. CVC, sweat rate, and MAP were recorded at baseline and 2, 5, 12, 20 and every 10-min until the end of recovery. Active and passive recoveries were equally effective in maintaining CVC and sweat rate at greater levels compared with an inactive recovery ($p < 0.001$). Sex did not affect any of the measured variables at any time point in any recovery mode, with the exception of sweat rate ($p < 0.001$). We conclude that despite an important thermal drive, nonthermal input remains an important influence in the modulation of postexercise heat loss responses.

INTRODUCTION

It is well documented that mean arterial pressure (MAP) is reduced below pre-exercise resting values following a single bout of dynamic exercise (Kenney & Seals, 1993; Halliwill, 2001) and that this postexercise hypotension is greater in females as compared to males (Fisher *et al.*, 1999; Carter *et al.*, 2001; Kenny & Jay, 2006). In non hyperthermic individuals, the decreases in MAP following exercise are paralleled by an attenuation of the heat loss responses of skin blood flow and sweating during inactive upright recovery (Thoden *et al.*, 1994; Kenny & Niedre, 2002; Wilkins *et al.*, 2004; Journeay *et al.*, 2006) despite a concomitant prolonged (up to 60-min) elevation (~0.4-0.5°C) in core temperature (i.e. esophageal temperature) above pre-exercise values (Thoden *et al.*, 1994; Kenny & Niedre, 2002; Wilkins *et al.*, 2004). These reductions in heat loss responses during inactive upright recovery have been shown to be related to a nonthermal baroreceptor mediated influence (Journeay *et al.*, 2006), suggesting that nonthermal influences may be overriding thermal stimuli (Crossley *et al.*, 1966). As well, recent evidence suggests that interactions between nonthermal influences and postexercise hemodynamic and heat loss responses are sex-dependant (Kenny & Jay, 2006), which may in part be due to differences in the relative influences of nonthermal factors on postexercise core body temperature regulation (Journeay *et al.*, 2005).

Recently, the relative roles of nonthermal influences on postexercise heat loss responses were characterized by studies using different recovery modalities (Carter *et al.*, 2002; Journeay *et al.*, 2004b; Shibasaki *et al.*, 2004; Journeay *et al.*, 2005). These studies have shown that an active and passive recovery, which reverse the unloading of baroreceptors through the action of the skeletal muscle pump/mechanoreceptors, are both

effective in maintaining heat loss responses at higher levels than an inactive recovery mode. However, it remains to be determined if the level of influence of nonthermal factors on postexercise heat loss responses are comparable with greater levels of hyperthermia. Exercise-induced hyperthermia has been shown to result in decreases in stroke volume and central blood volume (Wyndham, 1973; Rowell, 1974) as well as a greater postexercise hypotension (Kilgour *et al.*, 1993). This may result in a greater arterial and cardiopulmonary baroreceptor unloading which may further exacerbate the postexercise attenuation of heat loss responses despite elevated body temperatures (Kenny & Jay, 2006). This could result in an increased risk of heat related injury and heat syncope associated with compromised thermoregulatory function in an inactive recovery which may be more pronounced in females. To the best of our knowledge, no study has examined postexercise heat loss responses following exercise-induced hyperthermia and it remains unclear if a greater thermal strain would have an overriding influence on nonthermal control of heat loss responses postexercise.

Therefore, the following study evaluated the effects of hyperthermia on heat loss responses postexercise and the relative influence of nonthermal stimuli and sex-related differences. It was hypothesized that both active and passive recoveries would be more effective in attenuating the fall in MAP, cutaneous vascular resistance and sweat rate. It was also hypothesized that females would demonstrate a more pronounced reduction in MAP paralleled by a greater decrease in the heat loss responses of skin blood flow and sweating.

METHODS

Participants

Following approval of the experimental protocol from the University of Ottawa Research Ethics Committee, 18 (9 males, 9 females) healthy and physically active participants volunteered. Participant characteristics are presented in table 1. To control for hormonal effects, estradiol and progesterone measurements verified that all female participants were tested during the follicular phase of their menstrual cycle.

Instrumentation

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.

Sweat rate was measured using a 5.0 cm^2 ventilated capsule placed over the upper trapezius muscle. Ambient air passed trough the capsule and over the skin surface. The vapour density of the influent air was calculated from its relative humidity and temperature using a 373 Dew Point Mirror (RH systems, Albuquerque, NM) while the vapour density of the effluent air was calculated using an Omega HX93 humidity and temperature sensor (Omega Engineering, Stanford, CT). Sweat rate was calculated from the difference in water content between effluent and influent air and the flow rate. The flow rate through the capsule was ~0.7 $L \cdot min^{-1}$. The sweat rate value was normalized for skin surface under the capsule and thereby expressed in $mg \cdot min^{-1} \cdot cm^{-2}$.

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 also verified periodically throughout the protocol by manual auscultation from a clinical sphygmomanometer (Baumanometer® Standby® Model, Copiague, N.Y.) and a stethoscope (AMG medical Inc., Montreal, Qc.). Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were determined as the first and fifth Korotkoff sounds respectively. MAP was calculated as DBP + 1/3(SBP-DBP).

Skin blood flow (SkBF) was estimated using laser-Doppler velocimetry (PeriFlux System 5000, main control unit; PF5010 LDPM, function unit; Perimed, Stockholm, Sweden) at the left midanterior forearm which was resting at heart level. The laser-Doppler probe (PR 401 angled probe, Perimed) was taped to cleaned skin, in an area that did not appear by visual inspection to be overly vascular and from which consistent readings were noted (Mack, 1998). At the end of the baseline period, local skin temperature was raised using a heating element (PF 5020 temperature unit, Perimed) which houses the laser-Doppler flow probe to elevate local skin temperature to 44°C. Peak SkBF was determined as a sustained elevated plateau in local SkBF (~30-min) (Taylor *et al.*, 1984). Cutaneous vascular conductance (CVC) was calculated as the ratio of laser-Doppler flow to MAP. CVC data will be presented as a percentage of peak CVC ($\%CVC_{peak}$).

Cardiac output ($\dot{Q}$) was estimated using a metabolic cart (model CPX/D, Medgraphics, St. Paul, MN) and the CPX/D computerized version of the CO_2-rebreathing technique of Defares (Defares, 1958). It has been shown that Doppler-derived aortic blood flow measurements of $\dot{Q}$ correlate well with the indirect carbon dioxide

rebreathing method (Hadjis *et al.*, 1995). The Defares method has also been shown to work well in "unsteady state" testing (Inman *et al.*, 1985). Each measure took approximately 20-25 seconds to perform. Subjects performed one rebreathing protocol per designated time-point. Stroke volume was calculated as $\dot{Q}$/HR. Total peripheral resistance was calculated as MAP/$\dot{Q}$.

Heart rate (HR) was monitored continuously using a Polar coded transmitter, and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy, Kempele, Finland).

Experimental Protocol

Each participant took part in one preliminary session and three experimental trials separated by a minimum of 48 hours and carried out in balanced order. Participants were asked to abstain from alcohol, caffeine, and intense or prolonged physical activities for at least 12 hours prior to all sessions. Participants were asked to ensure they were properly hydrated by drinking at least 100 ml of water every waking hour prior to the experimental trials. All trials were performed at the same time of the day for each subject. During the preliminary session, body composition was assessed by hydrostatic weighing and participants were asked to perform a maximal exercise test on a treadmill to determine peak oxygen consumption ($\dot{V}O_{2peak}$)

The experimental timeline is represented in figure 1. The experimental sessions began with an instrumentation period. Once all the equipment and probes were in place and functioning, baseline data was collected at an ambient temperature of (average ± SD) 31.2 ± 1.0°C and relative humidity (RH) of 33 ± 6% for 15min. Following baseline data

collection, CVC_{peak} was determined as described previously (CVC_{peak} period). Participants then entered a thermal chamber set at 41.8 ± 0.3°C and 19 ± 4%RH. There was a 5-min habituation period in order for their core body temperature to stabilize. Participants then exercised on a treadmill for 30 ± 6min (at ~70% $\dot{V}O_{2peak}$) until T_{es} reached 39.5°C.

Following exercise, participants exited the thermal chamber and transferred (2.5 ± 0.6-min) to an ambient temperature of 31.2 ± 1.0°C and RH of 33 ± 6% where they began a 60-min recovery period in one of three recovery modalities: active, passive, or inactive. The active recovery consisted of loadless pedaling at 60 rpm on a cycle ergometer. During the inactive recovery, participants remained upright seated and motionless, and during the passive recovery they remained seated while a second person pedaled a cycle ergometer in tandem such that they were driven through the full range of the pedaling motion at 60 rpm in a passive manner. Participants were informed at the beginning of each trial which recovery modality would be used during the post-exercise period.

Statistical Analyses

For the continuous measures of core temperatures, sweat rate, and HR a 60-s average value was determined. MAP, $\dot{Q}$, SV, and TPR were recorded 10, 20, and 30-min into CVC_{peak} and throughout the postexercise period. A three-way mixed model analysis of variance was used to analyze the dependant variables using the repeatable factors of time (levels: 2, 5, 12, 20, 30, 40, 50 and 60-min.) and recovery mode (levels: active, inactive, and passive) and the non-repeatable factor of sex (levels: males and females). Exercise time was analyzed using a two-way mixed model analysis of variance with the repeated factor of time and the non-repeated factor of sex. Differences were considered significant

when $p \leq 0.05$. Pair-wise comparisons were performed using one-tailed paired sample t-tests. The level of significance was set at 0.05 and the alpha level was adjusted during multiple comparisons so as to maintain the rate of type I error at 5% during Bonferroni post-hoc analysis ($p \leq 0.05 \cdot n^{-1}$; n = number of comparisons). All analyses were performed using the statistical software package SPSS 15.0 for Windows (SPSS Inc. Chicago, IL, USA).

RESULTS

Exercise time

Exercise time taken to reach the experimental withdrawal criteria (T_{es} = 39.5°C) was not different between recovery mode trials ($F_{(2,32)}$=0.329, p=0.722) nor different between sexes ($F_{(1,16)}$=0.339, p=0.569).

Hemodynamic responses

Heart Rate (HR). The elevations in HR during recovery from exercise-induced hyperthermia became lower with time ($F_{(2.9,46.0)}$=312.0, p<0.001), and were influenced by recovery mode ($F_{(2,32)}$=40.8, p<0.001) (Fig. 2a). However, changes in HR were not affected by sex ($F_{(1,16)}$=0.1, p=0.952) (Fig. 2b), at any time during any recovery modality ($F_{(5.8,93.3)}$=0.918 p=0.484).

Stroke Volume (SV). The decreases from baseline values in SV during exercise-induced hyperthermia recovery became gradually greater with time ($F_{(2.8,45.1)}$=24.2, p<0.001). These reductions in SV were affected by recovery mode ($F_{(2,32)}$=15.1, p<0.001) and were most prominent during the inactive modality (Fig. 2c). Changes in SV were not different between sex ($F_{(1,16)}$=0.1, p=0.913) (Fig. 2d), at any time point during all three recovery modes ($F_{(6.3,100.7)}$=0.982, p=0.444).

Cardiac Output ($\dot{Q}$). The elevations in $\dot{Q}$ from rest became lower throughout recovery from exercise-induced hyperthermia ($F_{(2.4,38.6)}$=152.4, p<0.001). Furthermore, $\dot{Q}$ remained highest during the active recovery condition ($F_{(2,32)}$=53.3, p<0.001) (Fig. 2e) and changes in $\dot{Q}$ were not affected by sex ($F_{(1,16)}$=2.4, p=0.142) (Fig. 2f) throughout active, inactive and passive recovery ($F_{(3.9,63.1)}$=0.916, p=0.459).

Mean Arterial Pressure (MAP). Changes in MAP from pre-exercise rest due to exercise-induced hyperthermia were influenced by recovery time ($F_{(1.9,3.7)}$ =22.7, p<0.001) and mode ($F_{(2,32)}$ =15.3, p<0.001). The magnitude of postexercise hypotension was significantly less during the active and passive modalities relative to inactive after 12-min and for the remainder of the recovery period (Fig. 3a). Changes in MAP were not significantly different between sex ($F_{(1,16)}$ =1.1, p=0.312) (Fig. 3b), at any point irrespective of recovery modality ($F_{(3.7,59.7)}$ =0.617, p=0.641).

Total Peripheral Resistance (TPR). Reductions from resting TPR as a consequence of exercise-induced hyperthermia became less as a function of time ($F_{(3.3,53.3)}$=163.2 p<0.001). However, differences between recovery modes ($F_{(2,32)}$=22.2 p<0.001) became greater with time ($F_{(5.7,91.5)}$=6.9 p<0.001) (Fig. 3c). Changes in TPR were not affected by sex ($F_{(2,32)}$=22.2 p<0.001) (Fig. 3d), at any time during all three recovery modes ($F_{(5.7,91.5)}$=1.4 p=0.244).

Thermal responses

Cutaneous Vascular Conductance. The elevation in % of CVC_{peak} above resting values became lower with recovery time ($F_{(2.1,33.2)}$=345.2, p<0.001) and was influenced by recovery mode ($F_{(2,32)}$=12.5, p<0.001). Percentage of CVC_{peak} was significantly

greater during the active and passive modalities relative to inactive after 12-min and for the remainder of the recovery period (Fig. 4a). Sex did not affect changes in % of CVC_{peak} ($F_{(1,16)}$=3.4, p=0.141) (Fig. 4b), at any time point for any recovery mode ($F_{(3.6,58.1)}$=1.3, p=0.295).

Sweat Rate. Sweat rate became significantly reduced throughout recovery ($F_{(1.9,30.7)}$=165.8, p<0.001) and was influenced by recovery mode ($F_{(2,32)}$=3.8, p=0.034). The latter was evidenced by a tendency for sweat rate to be higher in the active and passive recoveries at 30, and 40-min (0.05<p<0.10), while being significantly greater at 50 and 60-min into the recovery period compared to inactive recovery (Fig. 4c). Sweat rate was significantly greater for males compared to females throughout recovery from exercise-induced hyperthermia ($F_{(1,16)}$=12.3, p=0.003) (Fig. 4d). However, the differences in sweat rate between sex were the same irrespective of recovery mode ($F_{(4.1,65.7)}$=1.0, p=0.402).

Esophageal temperature (T_{es}). The change in T_{es} from pre-exercise rest became less with recovery time ($F_{(4.4,70.7)}$ =824.4, p<0.001). The rate of decay of T_{es} became significantly different due to recovery mode ($F_{(4.1,65.8)}$ =98.8, p<0.001) with T_{es} becoming significantly lower during passive recovery compared to active recovery at 20-min postexercise and for the remainder of recovery. T_{es} tended (0.05<p<0.10) to be lower relative to inactive recovery at 60-min postexercise (Fig. 5a). Sex did not affect changes in T_{es} ($F_{(1,16)}$=0.6, p=0.445) (Fig. 5b), at any time point during recovery for all three recovery modes ($F_{(4.1,65.8)}$=1.1, p=0.350).

DISCUSSION

The most important finding from this study was that nonthermal input modulated the heat loss responses of cutaneous vascular conductance (CVC) and sweating despite an important thermal drive (i.e. exercise-induced hyperthermia). Furthermore, sex did not affect the relative changes from baseline in any of the measured variables, except for sweat rate, during all three recoveries studied. Both active and passive recoveries were effective in maintaining mean arterial pressure (MAP), CVC and sweat rate at greater levels than an inactive recovery during recovery from exercise-induced hyperthermia. Since an active recovery includes the involvement of central command and the skeletal muscle pump/mechanoreceptors, while a passive recovery only involves the action of the skeletal muscle pump/mechanoreceptors without the descending input from central command, these results suggest that action of the skeletal muscle pump/mechanoreceptors is the main nonthermal determinant in the maintenance of MAP, CVC and sweat rate during the recovery period in individuals rendered hyperthermic by exercise. Another important finding was that esophageal temperature became significantly lower during passive recovery compared to the active modality and it tended to be lower compared to an inactive recovery.

Our results differ from previous reports (Thoden *et al.*, 1994; Kenny & Niedre, 2002; Journeay *et al.*, 2004b; Shibasaki *et al.*, 2004; Wilkins *et al.*, 2004; Journeay *et al.*, 2005; Journeay *et al.*, 2006) which show that CVC and sweat rate return to near baseline values in the early stages of inactive recovery following exercise in non-heat stressed individuals. We show that CVC and sweat rate remain elevated throughout an inactive recovery in a mildly hyperthermic individual (i.e. T_{es} = 39.5°C). Interestingly, the higher

levels of CVC and sweat rate were measured despite a comparable magnitude of decrease in MAP as compared to previous studies in non-hyperthermic individuals (Thoden *et al.*, 1994; Kenny & Niedre, 2002; Journeay *et al.*, 2004b; Wilkins *et al.*, 2004; Journeay *et al.*, 2005; Journeay *et al.*, 2006). Previous work had suggested that baroreflexes would predominate over thermal responses (Crossley *et al.*, 1966). However, our results suggest that, although nonthermal influence of baroreceptors on modulation of CVC and sweating during recovery from exercise-induced hyperthermia is still present, the increased thermal drive is sufficient to override the baroreflex-mediated attenuation of CVC and sweat rate. This is consistent with studies which report that orthostatic tolerance is significantly reduced with passive heat stress (Lind *et al.*, 1968; Wilson *et al.*, 2002; Meendering *et al.*, 2005).

Results from the present study show that nonthermal input during recovery from exercise-induced hyperthermia affect hemodynamic and CVC responses independent of sex. This is in contrast to previous work, in non-heat stressed individuals, that showed that postexercise CVC was affected by sex-related differences (Marchand *et al.*, 2001; Journeay *et al.*, 2005). Journeay et al. (Journeay *et al.*, 2005) reported that central command was the main nonthermal determinant in the maintenance of postexercise CVC in females, based on the observation that CVC during the passive recovery mode fell below those in the active mode while remaining above the inactive values. However, it would appear that during recovery from an exercise-induced heat stress condition, the maintenance of CVC during the recovery period is dependant on skeletal muscle pump/mechanoreceptors irrespective of sex. Also, our observation that sex did not affect the magnitude and temporal pattern of response of any of the hemodynamic variables is

in agreement with recent findings in non-heat stressed individuals (Fisher *et al.*, 1999; Lynn *et al.*, 2007). However, although sex did not affect the pattern of response in MAP in any of the recovery modes, it is important to note that the absolute change in MAP was significantly lower in females than in males. This is consistent with previous reports (Fisher *et al.*, 1999; Carter *et al.*, 2001; Kenny *et al.*, 2005; Kenny & Jay, 2006) which show that females tend to have a greater postexercise hypotension than their male counterparts suggesting females may be at a greater risk of postexertional syncope with greater levels of hyperthermia (i.e. $T_{es} > 39.5°C$). Our findings confirm previous reports that nonthermal control of sweating is independent of sex (Journeay *et al.*, 2005). While sweat rate for males was elevated compared to values measured for females in the present study, this was an expected finding as females have consistently been shown to have lower sweat rates than males (Kaciuba-Uscilko & Grucza, 2001). This is further supported by recent data from our laboratory using whole-body direct calorimetry (Gagnon *et al.*, 2006) which shows that females had a lower rate of latent heat loss compared to males during recovery from exercise.

Our findings in individuals rendered hyperthermic by exercise show that nonthermal influences modulate the postexercise heat loss responses of CVC and sweating despite an important thermal drive. This is evidenced by the fact that both active and passive recoveries were equally effective in maintaining CVC at higher levels as compared to an inactive recovery. This is consistent with our hypothesis and previous reports (Carter *et al.*, 2002; Journeay *et al.*, 2004b) in moderately heated individuals ($T_{es} \leq 38.0°C$). Recent evidence supports the notion that CVC is modulated by baroreceptors during recovery from exercise. Studies using either lower body positive pressure

(Journeay *et al.*, 2004a) or head-down tilt (McInnis *et al.*, 2006) show that attenuation of the postexercise decrease in MAP results in the maintenance of CVC above pre-exercise values. Further, the higher levels of CVC measured during active and passive recoveries is thought to be the consequence of the action of skeletal muscle pump/mechanoreceptors which has been shown to maintain MAP and stroke volume (SV) above inactive values (Carter *et al.*, 1999; Carter *et al.*, 2002; Journeay *et al.*, 2004b; Journeay *et al.*, 2005). The lower CVC values observed during inactive recovery could be related to significantly lower MAP and SV, which is likely the consequence of a lowered central blood volume (Carter *et al.*, 1999), which resulted in greater arterial and cardiopulmonary baroreceptor unloading.

Increasing evidence supports that sudomotor control may be mediated by nonthermal baroreflex activity during recovery from exercise (Jackson & Kenny, 2003; Kenny *et al.*, 2003; Journeay *et al.*, 2004a; Journeay *et al.*, 2004b). Our observations support a baroreceptor mediated influence on sweat rate during recovery from exercise as both an active and passive recovery mode maintained sweat rate values at a higher level than an inactive recovery. As with CVC, results from the present investigation support that attenuation of the decreases in MAP and SV, and therefore the baroreceptor unloading associated with an inactive upright recovery, results in the maintenance of sweat rate above inactive values (Journeay *et al.*, 2004a; Journeay *et al.*, 2004b). However, our observations, which are based on individuals rendered hyperthermic following exercise in the heat, differ from reports employing a short duration exercise bout. We showed that both an active and passive recovery were effective in maintaining sweat rate at greater levels during the recovery period as compared to an inactive

recovery. This suggests that skeletal muscle pump/mechanoreceptors were important in maintaining sweat rates (Shibasaki *et al.*, 2004). However, this is in contrast to a previous study by Journeay et al. (Journeay *et al.*, 2004b) which reported that sweat rates became significantly different between all three recovery modes at 8-min into the recovery period. They suggested there may be a differential control of sweating such that central command and skeletal muscle pump/mechanoreceptors would affect sweat rate postexercise. However, from the results of the present study, the fact that a passive recovery was as effective as an active recovery in maintaining sweat rate above inactive values, despite the absence of input from central command, would suggest that control of sweating during recovery from exercise-induced hyperthermia is similar to that of CVC.

Previous studies have shown that unloading of baroreceptors by use of lower body positive pressure (Journeay *et al.*, 2004a) and head-down tilt (McInnis *et al.*, 2006) resulted in a greater esophageal temperature decay. From these studies, it could be hypothesized that maintenance of MAP and the heat loss responses of CVC and sweating through action of skeletal muscle pump/mechanoreceptors during active recovery would result in a lower esophageal temperature. We showed that esophageal temperature became significantly lower during passive recovery compared to active recovery and tended to be lower than an inactive recovery. In contrast, previous studies report no significant differences in esophageal temperature using similar recovery modes in non-heat stressed individuals (Carter *et al.*, 2002; Journeay *et al.*, 2004b; Shibasaki *et al.*, 2004; Wilson *et al.*, 2004; Journeay *et al.*, 2005). It is possible that the different responses may be explained by the fact that: 1) previous studies did not allow sufficient recovery time to observe differences in esophageal temperature between recovery modes or 2) the

greater heat loss response during active recovery is a function of a greater heat load. In previous recovery mode studies (2, 21, 22), the longest recovery period previously reported was 20-min in duration (22). We showed differences in esophageal temperature only after 20-min of recovery. Further, recent data from our laboratory using direct calorimetry (Gagnon *et al.*, 2006) has shown that although an active recovery maintains evaporative and dry heat loss responses at a higher level than an inactive recovery, it does produce a higher heat load (Shibasaki *et al.*, 2004). However, despite the higher heat load, we showed that the relative increase in evaporative and dry heat loss during active recovery was sufficient to reduce esophageal temperature to levels similar to those measured during an inactive recovery (Gagnon *et al.*, 2006). Therefore, it is reasonable to postulate that a passive recovery results in a more rapid decay of esophageal temperature in hyperthermic individuals due to the fact that it is as effective in maintaining heat loss responses as an active recovery, while producing a lower heat load (Nobrega *et al.*, 1994).

In summary, thermal stimuli seems to override nonthermal input during recovery following exercise-induced hyperthermia and this response seems to be unaffected by sex-related differences in postexercise cardiovascular and thermoregulatory responses. However, nonthermal input from the maintenance of the action of the skeletal muscle pump/mechanoreceptors, through active and passive recoveries, does play a role in the maintenance of postexercise MAP, CVC, and sweat rate in both hyperthermic males and females. Furthermore, a passive recovery, which maintains MAP, CVC and sweat rate as effectively as an active recovery but with a possibly lower heat load, contributes to a faster esophageal temperature decay compared to an active recovery. Therefore, from a

practical point of view, a passive recovery may be an effective treatment modality for the treatment of individuals suffering from exercise-induced hyperthermia or for individuals performing repeated work/rest cycles in adverse environments.

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Table 1. Participant characteristics

Subject	Sex	Age (yrs)	Height (cm)	Weight (kg)	$VO2_{peak}$ ($ml \cdot min^{-1}$)	$VO2_{peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	BFP (%)
1	M	20	179	89.1	4976	56.0	22
2	M	25	173	82	4990	60.8	19
4	M	35	185	91.5	5194	56.7	14
6	M	19	173	65.1	4244	65.0	13
8	M	26	177	67.6	4354	59.7	14
9	M	25	163	74.5	4227	56.8	21
10	M	19	178	80.3	5065	63.1	14
13	M	19	172	71.1	4166	58.5	19
15	M	18	179	66.8	3951	59.3	12
Average		23	175	76.4	4574	59.5	17
SD		6	6	9.8	473	3.0	4

Subject	Sex	Age (yrs)	Height (cm)	Weight (kg)	$VO2_{peak}$ ($ml \cdot min^{-1}$)	$VO2_{peak}$ ($ml \cdot kg^{-1} \cdot min^{-1}$)	BFP (%)
3	F	33	170	74.2	3428	46.1	27
5	F	30	174	70.7	3532	49.9	28
7	F	31	167	55.3	2865	51.8	17
11	F	23	165	52	3052	58.5	20
12	F	22	159	57.2	2653	46.4	20
14	F	21	163	62.4	3498	55.9	22
16	F	25	169	67.5	3080	45.6	24
17	F	24	163	54.2	2793	51.7	19
18	F	23	169	67.6	3556	52.6	21
Average		26	167	62.3	3162	50.9	22
SD		4	5	8.0	350	4.5	4

* BFP = Body fat percentage

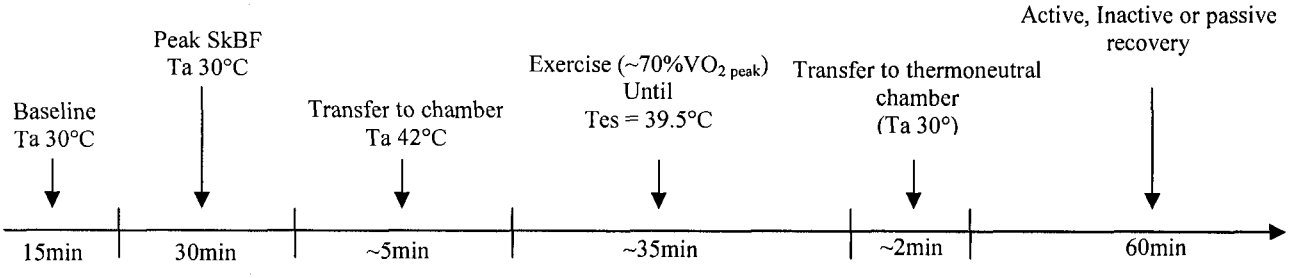

Figure 1. Experimental timeline

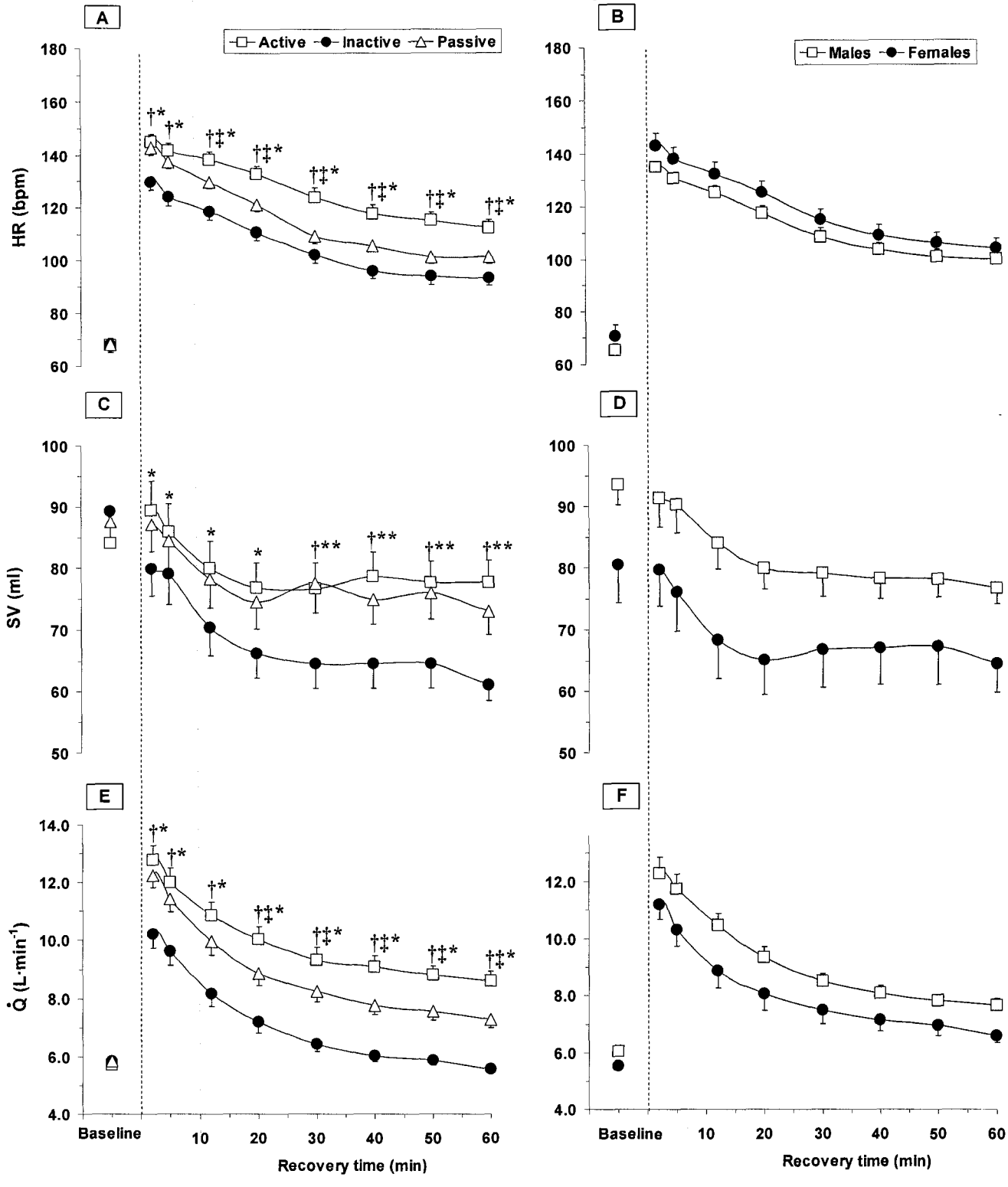

Figure 2. Effect of active (□), inactive (•), and passive (Δ) recovery after exercise-induced hyperthermia on A. Heart rate (HR), C. Stroke volume (SV), and E. Cardiac output ($\dot{Q}$). *Significant difference between active and inactive recoveries. † Significant difference between passive and inactive recoveries. ‡Significant difference between active and passive recoveries. Effect of sex, as a function of time for B. HR, D. SV, and F. $\dot{Q}$. Values from all three recovery modes were pooled for males (□) and females (•). Values are means ± SE for 18 subjects.

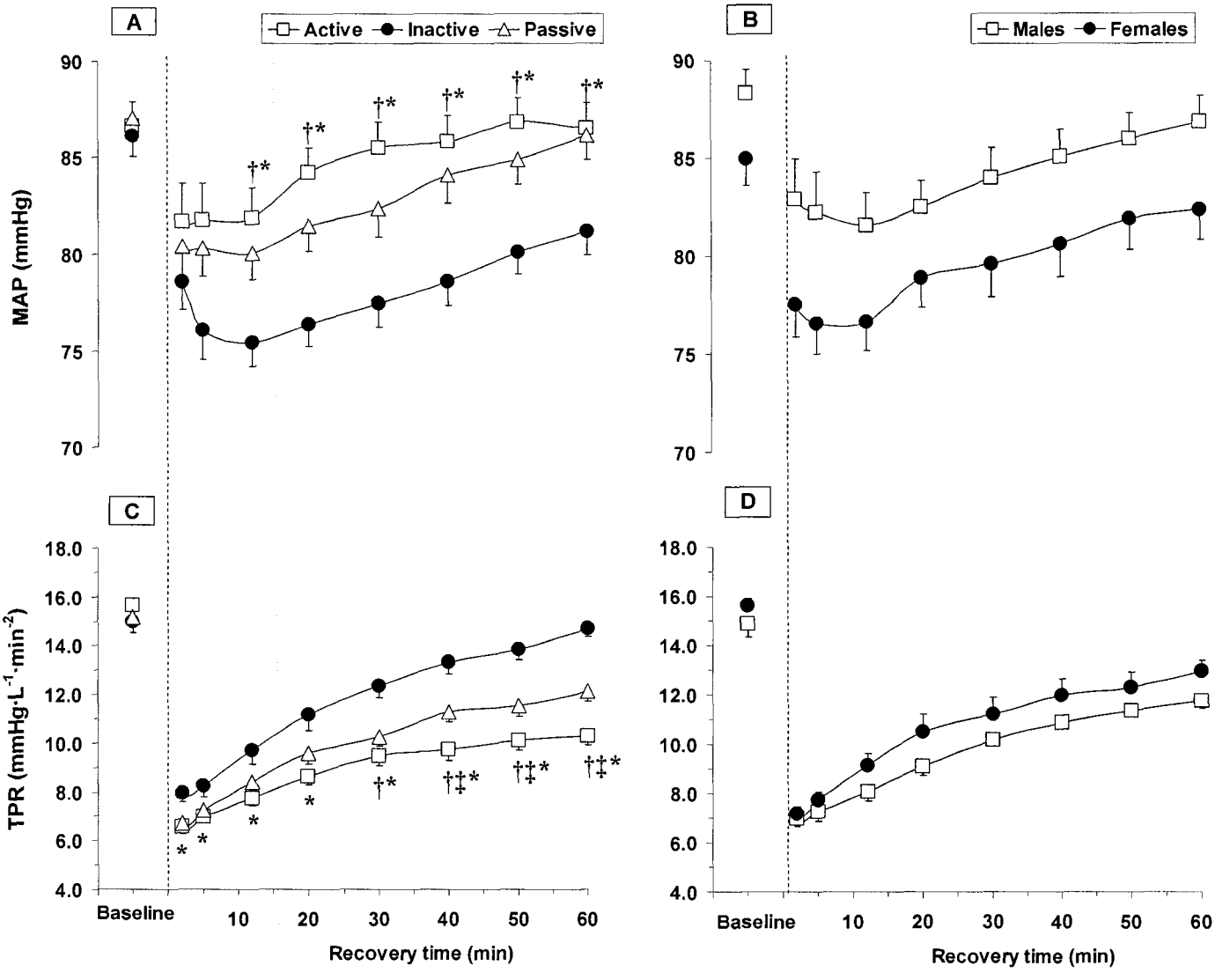

Figure 3. Effect of active (□), inactive (•), and passive (Δ) recovery after exercise-induced hyperthermia on A. mean arterial pressure (MAP), and C. total peripheral resistance (TPR). *Significant difference between active and inactive recoveries. † Significant difference between passive and inactive recoveries. ‡Significant difference between active and passive recoveries. Effect of sex, as a function of time for B. MAP, and D. TPR. Values from all three recovery modes were pooled for males (□) and females (•). Values are means ± SE for 18 subjects.

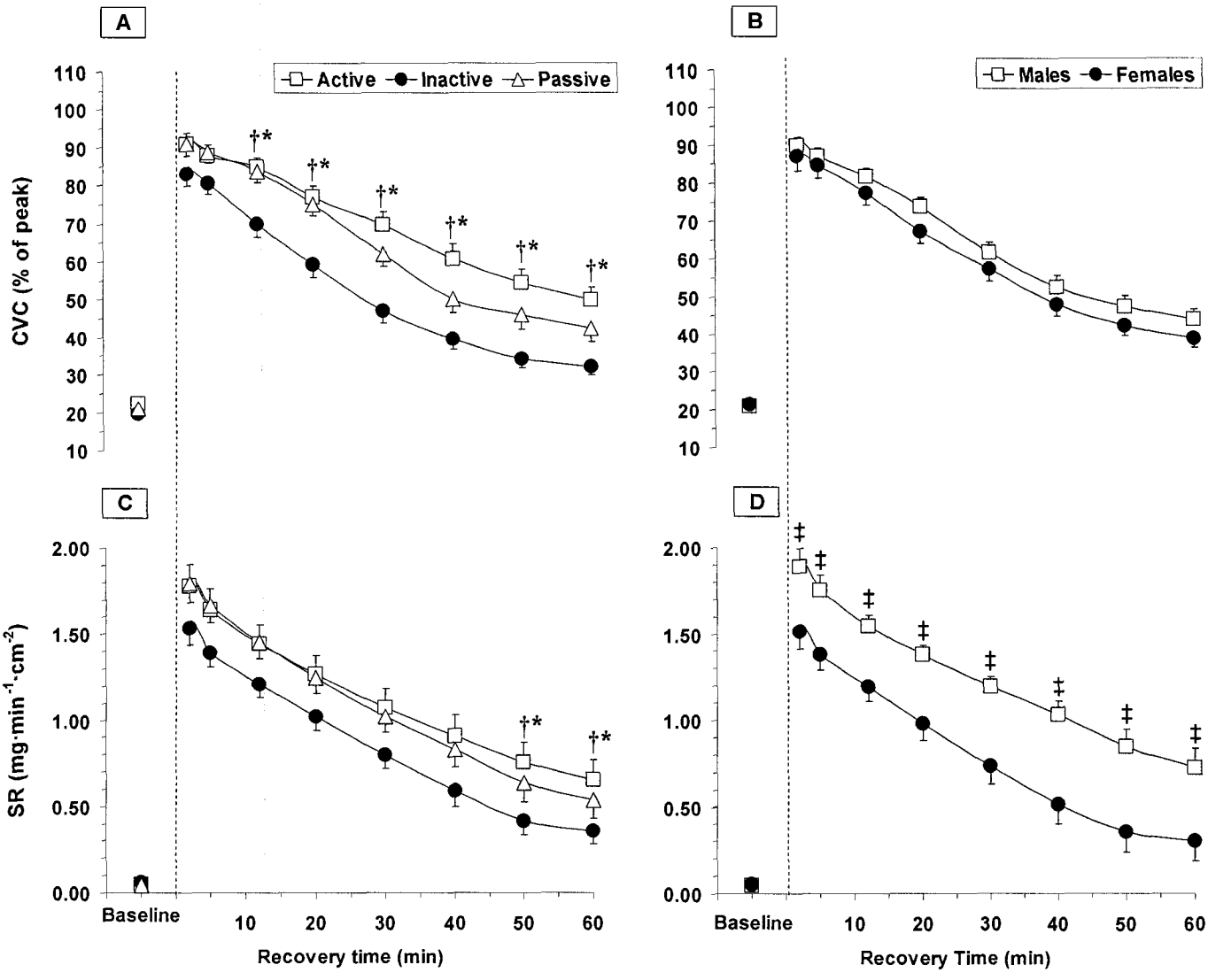

Figure 4. Effect of active (□), inactive (•), and passive (Δ) recovery after exercise-induced hyperthermia on A. percentage of peak cutaneous vascular conductance (%CVC_{peak}), and C. sweat rate (SR). *Significant difference between active and inactive recoveries. † Significant difference between passive and inactive recoveries. Effect of sex, as a function of time for B. %CVC_{peak} , and D. SR. ‡Siginificant difference between males and females. Values from all three recovery modes were pooled for males (□) and females (•). Values are means ± SE for 18 subjects.

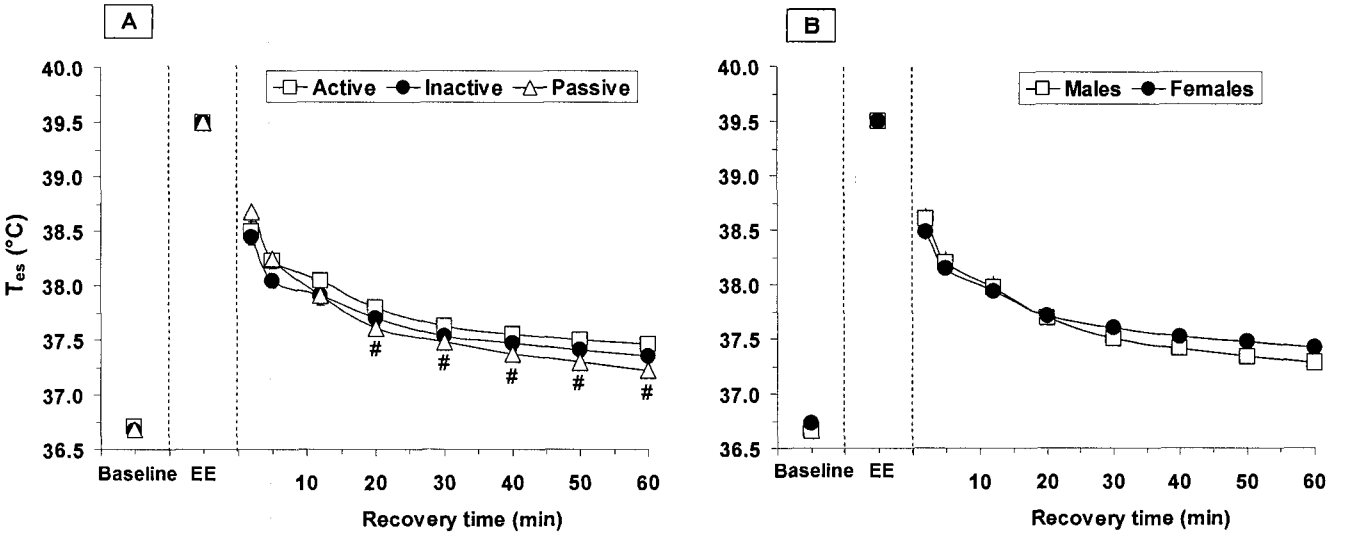

Figure 5. Effect of active (□), inactive (•), and passive (Δ) recovery after exercise-induced hyperthermia on A. esophageal temperature (T_{es}). #Significant difference between passive and active recoveries. Effect of sex, as a function of time for B. T_{es} .Values from all three recovery modes were pooled for males (□) and females (•). Values are means ± SE for 18 subjects.

PART THREE:

DISCUSSION AND CONCLUSION OF THE THESIS

4.0 Discussion

The purpose of this thesis was to examine the nonthermoregulatory control of skin blood flow (SkBF) and sweating during recovery from exercise-induced hyperthermia and to determine if changes in the control of heat loss and hemodynamic responses were sex dependent. This was achieved by using three different recovery modalities which have been used previously to delineate the relative influences of nonthermal stimuli on the control of postexercise SkBF and sweating in individuals not rendered hyperthermic. The mechanisms of the three recovery modes were as follow: 1) active recovery (loadless pedaling), during which central command and skeletal muscle pump/mechanoreceptors are involved, 2) passive recovery (assisted pedaling) which involves the action of the skeletal muscle pump/mechanoreceptors without the descending input from central command, and 3) inactive (upright seated) recovery where baroreceptors are primarily implicated. While baroreceptors are implicated in each recovery mode, it is thought that they are the main influence during the inactive recovery mode. By employing these three recovery modes, it was therefore possible to examine the relative influences of central command, skeletal muscle pump/mechanoreceptors, and baroreceptors following exercise-induced hyperthermia.

The most important finding of the present thesis is that, despite an important thermal drive (i.e. exercise-induced hyperthermia) nonthermal influences affected heat loss responses during recovery from exercise-induced hyperthermia and these responses were independent of sex. This was evidenced by the measurements of higher cutaneous vascular conductance (CVC) and sweat rates during both active and passive recoveries compared to an inactive recovery in both males and females. This suggests that nonthermal control of postexercise CVC and sweat rate in individuals rendered mildly hyperthermic by exercise in the heat is mainly determined by action

of the skeletal muscle pump/mechanoreceptors. Of note, although the action of the skeletal muscle pump/mechanoreceptors maintained CVC and sweat rate to higher levels, CVC and sweat rate did not return to baseline values within the early stages of the recovery period. This is contrast to previous studies examining the influences of nonthermal influences in the postexercise control of CVC and sweat rate in individuals not rendered hyperthermic. This suggests that, although nonthermal influences are important in the maintenance of hemodynamic and heat loss responses during a greater thermal strain (i.e. exercise-induced hyperthermia), thermal stimuli seem to override the influence of nonthermal input to maintain heat loss responses at the expense of maintaining blood pressure. This was evidenced by the greater magnitude of postexercise decrease in mean arterial pressure observed in the inactive recovery mode. This suggests that individuals may be at a greater risk of postexertional syncope during an inactive recovery after prolonged exercise in the heat. Although many participants experienced pre-syncopal symptoms (i.e. dizziness, light headedness etc.) during inactive recovery, none of them experienced such symptoms during either active or passive recovery. Furthermore, none of the measured variables during recovery from exercise-induced hyperthermia were affected by sex-related differences for the exception of sweat rate which was higher in males throughout recovery. This is in contrast to previous work done in non-heat stressed individuals which suggested that nonthermal control of postxercise CVC was different between males and females. Therefore, under hyperthermic conditions, it seems that nonthermal modulation of heat loss responses is not affected by sex-related differences, as thermal stimuli seems to override nonthermal inputs from central command, skeletal muscle pump/mechanoreceptors, and baroreceptors.

5.0 Conclusion and perspectives

In summary, input form nonthermal stimuli affects postexercise control of SkBF and sweating despite a high thermal drive (i.e. exercise-induced hyperthermia). Maintenance of the action of the skeletal muscle pump/mechanoreceptors seems to be the main nonthermal determinant in the postexercise maintenance of MAP, CVC and sweat rate in individuals rendered hyperthermic in both males and females. Our findings may have practical implications in the treatment of individuals suffering from exercise induced hyperthermia which is known to occur in athletes, military personnel and occupational workers who are required to perform demanding tasks in hot ambient conditions. In these conditions, a passive recovery could help restore normal hemodynamic and thermoregulatory function in the clinically hyperthermic individual. Further, our results are of particular importance for individuals performing intermittent bouts of work/rest cycles in adverse environments (i.e. high ambient temperature and humidity) as an active or passive recovery would maintain heat loss responses and possibly reduce increases in core temperature over time, thus lowering the risk of developing a hyperthermic condition and heat-related injuries.

This thesis adds important knowledge to the combined thermal and cardiovascular interactions during recovery from exercise and extends our understanding of these controls in individuals rendered mildly hyperthermic. Future research on postexercise hemodynamic and heat loss responses in individuals rendered hyperthermic should focus on examining the dynamics of heat exchange during and after exercise-induced hyperthermia, the effects of hydration status, the effect of recovery modes on repeated work/rest cycles and possibly the effect of different phases of the menstrual cycle. Further, the effects of active and passive recoveries on whole body thermal status by the use of whole-body direct calorimeter and muscle temperatures could provide more insights into the beneficial effects of the

higher levels of heat loss responses of skin blood flow and sweating during active and passive recoveries.

PART FOUR:

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