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BARORECEPTOR INFLUENCE ON POST-EXERCISE WARM THERMAL RESPONSE THRESHOLDS

by

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ABSTRACT

The role of baroreceptor modulation on the post-exercise esophageal temperature threshold for cutaneous vasodilation (Th_{VD}) and sweating (Th_{SW}) was investigated. Six subjects were randomly exposed to lower body positive pressure (LBPP) and to no lower body positive pressure (NoLBPP) following a No-Exercise (NoEx) and Exercise (Ex) treatment protocol. The Exercise treatment consisted of 15 min of cycling at 70% VO_2max and the No-Exercise treatment consisted of 15 min upright resting. Immediately following either treatment, subjects were positioned within a LBPP box after which a whole body water perfused suit was used to regulate mean skin temperature to assess Th_{VD} and Th_{SW}. Th_{VD} increased 0.34°C post-exercise from the NoEx/NoLBPP condition to the Ex/NoLBPP condition ($P<0.05$). However, the post-exercise increase in Th_{VD} was abolished in the Ex/LBPP condition. No differences in Th_{SW} were measured among the 4 conditions. These data support the hypothesis that the observed elevated post-exercise Th_{VD} is the result of baroreceptor unloading. The similarity in threshold responses for sweating onset suggests that the post-exercise Th_{SW} is not sensitive to baroreceptor loading or unloading.

Key words: baroreflex, LBPP, thermoregulation, skin blood flow, sweating

TABLE OF CONTENTS

APPENDICES

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

bpm or **beats · min^{-1}** - beats per minute

Ex – exercise

HDT – head down tilt

HUT – head up tilt

LBPP - lower body positive pressure

LBNP - lower body negative pressure

MAP - mean arterial pressure

NoEx - no exercise

SkBF - skin blood flow

SP_{hy} - hypothalamic set-point temperature

T_{co} - core temperature

T_{es} - esophageal temperature

$\overline{T}_{sk}$ - mean skin temperature

Th_{SW} - esophageal temperature threshold for the onset of sweating

Th_{VC} - esophageal temperature threshold for the onset of cutaneous vasoconstriction

Th_{VD} - esophageal temperature threshold for the onset of cutaneous vasodilation

VO_2max - maximum aerobic power (maximum oxygen consumption)

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1.0 INTRODUCTION

Temperature regulation in humans involves the integration of many complex systems. Extensive research over the past decades has attempted to explain the mechanisms by which we maintain a homeothermic state. The most widely-accepted theory that exists in the area of thermoregulation is the hypothalamic set point temperature theory. This theory brings forward the notion that thermostatic influence on the cardiovascular system, metabolism, and neuromuscular and sudomotor functions maintain hypothalamic set-point temperature (SP_{hy}). Thus, the observation of an exercise-induced rise in core temperature (T_{co}) above the hypothalamic set point is perceived as a discrepancy between the production and dissipation of metabolic heat. This interpretation has been labeled as the load error (LE) concept and it has been suggested that SP_{hy} is raised (reset) during conditions of high metabolic stress in a similar manner to the fever response (Nielsen 1938; Jackson and Hammel 1963; Nielsen 1966; Tam et al. 1978; Taylor et al. 1984). When the body experiences a hyperthermic load error it is compared to SP_{hy} and the appropriate thermal responses are activated (i.e., cutaneous vasodilation and sweating) to attenuate any further rise in T_{co}. Many thermoregulatory researchers have adopted the set point theory and have utilized it as a basis for human thermoregulatory models (Gagge et al. 1971; Cabanac and Massonet 1977; Stolwijk and Hardy 1977; McIntyre 1980; Bligh 1985). However, there are differing opinions as to which variables influencing the human thermoregulatory system are being controlled. For example, Webb et al. (1978) proposed that rate of heat dissipated from the body is adjusted to balance

metabolic heat production; whereas, Bligh (1978) indicates that core temperature or a combination of temperatures at different anatomical sites are the components being controlled. Other researchers suggest that body heat content is being controlled (Houdas et al. 1972).

Evidence disputing the theory of hypothalamic set point is prevalent in many mammalian thermoregulatory studies. First, it has been well documented that certain non-thermal stimuli (i.e., posture, exercise, lower body negative and positive pressure, certain medications, etc.) can elicit changes in thresholds for vasoconstriction, shivering, vasodilation, and sweating. Experiments carried out in our laboratory have shown that postural change (head-down tilt) significantly decreases the post-exercise resting threshold for forearm cutaneous vasodilation (Kenny et al. 2000). Other studies have shown that exercising at a moderate intensity affects thresholds for vasoconstriction and shivering (Kenny et al. 1998). Kenny et al. (1997) observed that intense exercise increases the post-exercise threshold for sweating and cutaneous vasodilation. Mack et al. (1995), found that the application of lower body negative pressure (baroreceptor unloading) raises the threshold for vasodilation during exercise. Further, it was shown by Nicolaou et al. (1997) that vasoactive drugs such as Clonidine have a modifying effect on thermal responses. Specifically, they measured decreases in vasoconstriction and shivering thresholds, without affecting the threshold for sweating in humans. If the thresholds for vasoconstriction, shivering, vasodilation, and sweating (i.e., thermoregulatory responses) were controlled primarily by a change in core

temperature above or below the SP_{hy}, then non-thermal stimuli would not elicit a thermal response.

It has been suggested that there is a null zone for thermoregulatory response (Mekjavic et al. 1991). This null zone concept brings forth the notion that there is a range of temperatures that the body is thermally insensitive to and as a result temperature regulation remains passive to thermal stimuli within these temperature ranges. Although previous research had suggested the existence of a null zone (Jessen and Ludwig 1971; Mekjavic and Bligh 1989) their results were not sufficiently conclusive to support either the SP_{hy} or null zone concepts.

Thodén et al. (1994) observed that following 15 min of moderate exercise, skin blood flow and mean skin temperatures (T_{sk}) returned to baseline values within 15-20 min post-exercise resting despite a sustained elevation of esophageal temperature (T_{es}). Their results provide a strong argument against SP_{hy} theory. During exercise there is a concurrent increase in the rate of heat loss to attenuate the rate of increase of T_{co} (Sawka and Wenger 1988). Thus, if SP_{hy} theory was accurate, then reducing the metabolic heat load by the cessation of the exercise stimulus should have allowed T_{co} to quickly return to pre-exercise levels.

Furthermore, in an effort to address the mechanism for the post-exercise increase in thermal response thresholds, Kenny et al. (1996_b) immersed subjects in warm water (42 °C) until T_{es} increased to levels corresponding to those induced by 15 min of dynamic exercise. Following exit from the warm water, T_{es}, $\bar{T}_{sk}$, and skin blood flow all returned to control values within 10 min. Thus, it was concluded that the post-exercise increase in both T_{es} and the onset

threshold for vasodilation are not a consequence of an increased body heat content.

Although the actual mechanism of the post-exercise thermal response remains unclear, recent evidence favors a non-thermal, baroreceptor mediated influence. That is, a post-exercise hypotensive effect has been observed following acute bouts of exercise. It has been suggested that the acute post-exercise hypotension is most likely due to a persisting peripheral tissue vasodilation (Halliwill et al. 1996; Headly et al. 1996; Forjaz et al. 1998) needed to dissipate metabolic heat created by the exercise stimulus. However, the contribution of venous blood pooling in the previously active musculature to the hypotensive condition pressure must also be considered.

Reflex control of the cutaneous circulation involves both sympathetic active vasoconstrictor and active vasodilatory systems. The control of skin blood flow has been extensively evaluated and described at rest and during exercise. However, it remains unclear how skin blood flow is controlled during post-exercise recovery. It is known, however, that the cutaneous vasodilator system is under baroreceptor control (Kellogg et al. 1990). Immediately following a bout of dynamic exercise the observed hypotensive condition is most likely due to cutaneous vasodilation needed for heat dissipation and by the persisting venous pooling in the previously active musculature. This possibly results in an unloading of the cardiopulmonary baroreceptors and, in response, a decrease in skin blood flow. This baroreceptor response (i.e., vasoconstriction) would contribute to maintaining adequate left ventricular filling pressure in response to the decrease

in systemic vascular resistance. If cutaneous vasoconstriction is adequate, then mean arterial pressure (MAP) is maintained. However, the venous pooling in the previously active musculature may be great enough that normotensive MAP cannot be reestablished by cutaneous vasoconstriction. In this case, the hypotensive condition prevails long into recovery despite decreases in skin blood flow. This baroreflex response to lower extremity venous pooling has been observed, without exercise, during the application of lower body negative pressure (Crandall et al. 1996).

It is clear that this non-thermal factor may influence the threshold for warm thermal responses during exercise recovery. In that it is plausible that the body strives to maintain blood pressure over maintaining resting thermal homeostasis in the post-exercise period. Thus, blood pressure takes precedent over heat dissipation and we observe higher thresholds for vasodilation and possibly sweating and, consequently, higher resting T_{es}. This notion is supported in that we have shown that manipulating post-exercise venous pooling, by means of head-down tilt, in an effort to reverse its impact on baroreceptor unloading, results in a relative lowering of the resting post-exercise elevation in the esophageal temperature for forearm cutaneous vasodilation (Kenny et al. 2000). It must be noted, however, that the manipulation of posture, by means of head down tilt, may have engaged other physiological systems that could have confounded our results. Further, the lengthy protocol used could have had a confounding effect. Finally, it is well known that head down tilt loads only the low-pressure baroreceptors. The use of lower body positive pressure loads both

cardiopulmonary and arterial baroreceptors, thus providing a stimulus exactly opposing that caused by post-exercise venous pooling. Thus, the use of lower body positive pressure in a posture similar to that of upright cycling was chosen to further and more directly evaluate the baroreceptor influence on warm thermal responses (i.e., vasodilation and sweating).

The objective of this study was to evaluate the influence of venous pooling on post-exercise thermoregulatory thresholds. More specifically, the following study evaluated the effect of a reduction in post-exercise venous pooling by means of lower body positive pressure on the post-exercise esophageal temperature threshold for cutaneous vasodilation (Th_{VD}) and sweating (Th_{SW}).

1.1 Review of Literature

1.1.1 *Basic human body temperature regulation*

The human body tends to maintain core temperature (T_{co}) within 0.6°C of its normal value of 37°C (Johnson 1998). Most models of human thermoregulation accept the hypothalamic set-point theory (SP_{hy}) (Stolwijk and Hardy 1966; McIntyre 1980; Nadel 1983; Johnson and Ruhling 1985; Sawka and Wenger 1988). This theory suggests that thermoregulatory control is based on a negative feedback model. In this model, afferent information is sent from thermoreceptors located at the periphery and in the central nervous system. This afferent information is collected by an "integrative center" located in the hypothalamus, and there compared to a specific hypothalamic set point. If this comparison is outside the target range (known as a load error or LE), then (based on the LE), the "integrative center" coordinates efferent responses involving cutaneous vasomotor activity, sweating, shivering, and chemical thermogenesis. A LE signal is proportional to the difference between the SP_{hy} and the weighted input from both core and surface thermosensitive receptors (Johnson and Ruhling 1985). When the integrated signal is below the temperature set point range, heat loss is reduced by vasoconstriction and inhibition of sweating and shivering increases heat production. When the integrated signal is above the set point range, heat loss is increased through vasodilation and sweating

Three factors contribute to the maintenance of T_{co} near the SP_{hy}: 1) the sensitivity of the effector organ receptor; 2) the gain of the system (receptor and

effector); and 3) the time lag associated with the thermoregulatory threshold response. Gain is commonly known as the level that the output of an effector is modulated for a respective change in the controlled variable. That is, if the gain is low, changes in the LE signal will have a relatively small effect on the output of the effector and the controlled variable and larger LE signals will be needed to change the output of the effector by a given amount (Sawka and Wenger 1988). Therefore, the effect of a low gain is a higher amplitude and a lower frequency of response.

Time lags may have any of several origins. One such is the time needed for afferent information to reach the integrative center. Another involves the amount of time needed for the action potentials propagated from the integrative center to reach the effector and the delay in the response of the effector itself (Sawka and Wenger 1988). The primary effect of the different receptor response times is the way the thermal and non-thermal stimuli are interpreted. This is therefore reflected in the time that it may take to reach certain thermoregulatory thresholds.

As previously discussed, the body maintains homeothermy at a relatively stabilized T_{co} within 0.6°C of its normal value of 37°C (Johnson 1998). Deviations around 37°C are mainly in response to metabolic shifts and environmental stress. In fact, it has been found that fluctuations as high as 1.0°C or greater are common in women during the ovulation period of their menstrual cycle (Sawka and Wenger 1988; Frascarolo et al. 1992). The existence of such fluctuations brings into question the generally accepted concepts of temperature regulation that

contend that the central control mechanism and SP_{hy} can maintain T_{co} within narrow limits. Further, it suggests that these fluctuations are a result of not only thermal, but also non-thermal influences (e.g., metabolic, neuroendocrine, humoral or mechanical) that exhibit direct or indirect affects on the regulation of T_{co}.

The following review will address the current research within the area of human thermoregulatory control. Further, and more specifically, it will summarize the thermal and non-thermal mechanisms that affect the body at rest, during exercise and post-exercise and how these mechanisms affect blood pressure (BP) regulation, core temperature (T_{co}) regulation, the threshold for cutaneous vasodilation (Th_{VD}), sweating (Th_{SW}), and vasoconstriction (Th_{VC}). Finally, the following review will attempt to integrate the knowledge among these areas to clarify the place of post-exercise hyperthermia within SP_{hy} theory.

1.1.2 *Heat exchange with the environment*

Our bodies are in constant thermal interaction with the environment by means of the skin. The main processes involved in this interaction are radiation, convection, conduction, and evaporation.

The concept of radiation relates to the fact that all surfaces at temperatures above absolute zero give off energy in the form of electromagnetic radiation, and the rate of radiation is proportional to the fourth-power temperature of the surface (Johnson 1998). Radiation involves the transfer of heat from the surface of one object to the surface of another without physical contact. Humans at rest in an

ambient temperature of 21-22 °C can attribute 60% of their heat loss to radiation. Since it is a well-known physical property that temperature gradients flow from hot to cold, then it follows that we lose heat (by radiation) to environments at lower temperatures than our T_{sk}. In ambient temperatures above our T_{sk} we absorb heat from the environment.

Conduction involves the transfer of heat from one material to another through direct molecular contact. As an example, heat generated deep in the body can be conducted through adjacent tissue until it reaches the skin surface. It can then be conducted to the clothing or to the air by direct contact with the skin. Conversely, if the air that surrounds the body is warmer than the skin, heat from the air will be conducted to the skin (Wilmore and Costill 1994).

Convection can be simply defined as the transfer of heat via the movement of a gas (such as air) or a liquid across an object, such as the body. Environmental air is in constant motion. As it circulates and passes over the skin, it takes with it molecules of air that have been warmed (as a result of contact with the skin). The greater the movement of the gas, in this case air, the greater the net heat loss by convection. It follows that the body surface must be warmer than the environmental air in order for this to occur (Wilmore and Costill 1994). There are two common types of convection. The first occurs when air movement is due only to local heating by the body, termed "natural" convection. If external influences such as wind, electric fans, etc. contribute to air movement, the convection is termed "forced". If the skin surface and the environmental air are at the same temperature, then no heat is transferred; if the gradient is such that the air is

warmer than the skin surface, the body will gain heat by convection. The rate in which convection occurs is directly proportional to the exposed surface area of the body and the velocity of the convective flow.

Evaporation accounts for approximately 25% of the heat loss at rest and is the most important means of heat loss when endogenous or metabolic heat is increased and during exposure to extreme ambient temperatures. Energy is required to convert water to a vapor. This energy is referred to as the latent heat of vaporization and is equal to 580 kcal $\cdot L^{-1}$ of water vaporized. From this we can see that the rate of heat loss from the body, by means of evaporation, in kilocalories per hour (J_{Qevap}) is directionally proportional to the rate at which water evaporates form the skin surface and the respiratory passages J_{H2O} in liters per hour (Johnson 1998):

$J_{Qevap} = -(580 \text{ kcal} \cdot L^{-1}) \cdot J_{H2O}$

The body through evaporation, even in low ambient temperatures is constantly losing water. Under sedentary conditions, the basal rate of insensible water loss amounts to about 500ml per day due to loss from the skin surface and by expiration of saturated air, resulting in a net loss of approximately 300 kilocalories per day (Johnson 1998). If the body cannot lose heat by evaporation at the same rate as it may be acquiring it (by radiation, conduction, and/or convection), then body temperature will rise and subsequently the mechanisms for sweating will be employed to further augment evaporative heat loss. However, sweat is only effective for cooling if humidity is low enough for it to evaporate. That is, if the environmental air is already saturated (i.e., very humid), then the

rate of evaporation is greatly reduced, or totally prevented and the sweat remains in a fluid state. Another factor that may hamper evaporation is the lack of air movement, in such a case the air immediately surrounding the body becomes saturated and evaporation is hampered or halted.

Insensible water loss cannot be controlled and it occurs regardless of body temperature. However, regulating the sweat rate can control evaporative sweat loss. At rest, when T_{sk} is low, sweating is generally nonexistent. In contrast, when an unacclimatized individual is exposed to a hot environment he or she may have a maximum sweat rate of approximately 1.5 $L \cdot hr^{-1}$. An acclimatized individual in the same environment may sweat up to and beyond 3.0 $L \cdot hr^{-1}$ (Sawka and Wenger 1988). During maximum sweating a person can lose up to 3.6 $kg \cdot hr^{-1}$ (Taylor 1986).

Sudoriferous glands are also known as sweat glands. Within humans there are two types of sudoriferous glands: eccrine and apocrine. Apocrine or odiferous glands are those located under the arms (i.e., armpits), the dark region around the nipples (i.e., areola), the outer lips of the vulva, and the anal and genital regions. The apocrine glands are larger and exist more deeply than eccrine glands (Van Wynsberghe et al. 1995). Eccrine glands are activated by thermal stress (i.e., heat), but apocrine glands are activated more by psychological stress (i.e., emotional, sexual, etc.). Thus, the eccrine glands are more pertinent to our discussion on thermoregulation. Thermoregulatory sweat in humans is secreted by approximately 1.6 to 4 million eccrine glands (Sawka and Wenger 1988). Eccrine glands are small sweat glands. Although they are distributed over nearly

the entire body surface, there are no sweat glands on the nail beds, margins of the lips, eardrums, inner lips of the vulva, or the tip of the penis (Van Wynsberghe et al. 1995). The number of sweat glands per unit of skin surface area varies considerably between body regions. Eccrine sweat glands are most abundant on the plantar aspect of the feet and least numerous on the back (Sawka and Wenger 1988). These glands are of the coiled tubular type, with the secretory portion embedded in the hypodermis and duct with a hollow, corkscrew configuration running through the dermis to the surface of the epidermis. As the duct reaches the skin surface it tends to straighten out. The colorless aqueous fluid (i.e., sweat) excreted from the eccrine gland is generally composed of water, neutral fats, albumin and urea, lactic acid, sodium and potassium chlorides, and traces of sugar and ascorbic acid (Van Wynsberghe et al. 1995).

According to Sawka and Wenger (1988), the amount of sweat secreted by the eccrine gland is dependent upon the structure and function of the stimulated gland, as well as the sudomotor signal from the CNS. The efferent nerves involved in the sweating reflex originate in the hypothalamus and descend through the brain stem and spinal tract, crossing at various segmental levels and ending in the lateral horn where they synapse on peripheral neurons. The eccrine glands are innervated by post-ganglionic sympathetic fibers, which are nonmyelinated class C fibers that are primarily cholinergic. The eccrine sweat glands respond primarily to thermal stress through sympathetic cholinergic stimulation (Sawka and Wenger 1988). However, it has been shown that the human eccrine glands contain α and β-adrenergic receptors. Thus, as stated by

Sawka and Wenger (1988), it appears that circulating catecholamines (i.e., epinephrine) will elicit a sweat response from eccrine glands. The relative effects of cholinergic and adrenergic controls on sweating is a focus of controversy among researchers. However, it has been proposed that the relative effects on sweat secretion are 4:1:2 for cholinergic, α-adrenergic, and β-adrenergic receptor stimulation, respectively (Sato 1977). Research has shown that sweat composition and volumes are greatly influenced by neural, hormonal, and mechanical factors (Taylor 1986). The anterior hypothalamus elicits sympathetic cholinergic output to stimulate sweating. This is the principal efferent signal for the sweat response. Such output is largely initiated in response to afferent input and is proportional to deep body temperature. There appears to be a linear relationship between a rise in T_{co} and subsequent sweat output (Tam et al. 1978; Astrand and Rodhal 1986; Sawka and Wenger 1988). Further it has been shown that increases in ambient temperature and T_{sk} have elicited increases in the T_{co} to sweat rate relationship (Sawka and Wenger 1988; Bothorel et al. 1991).

The two primary non-behavioral means of heat transfer from the body to the environment is through the release of sweat (which has been discussed at length in this section) and by the control of skin blood flow. The latter will be considered in the following section.

1.1.3 *Thermoregulatory control of skin blood flow at rest*

In a resting human, the response in skin blood flow (SkBF) to heat stress is characterized by an internal temperature threshold, beyond which the rise in SkBF per degree Celsius is fairly steep (Johnson 1986). Some researchers have shown that there is variance among individuals (due to circadian rhythms) with respect to their thresholds and slopes of their T_{co}-SkBF ratio. However, as supported by Johnson (1986), their average values are around 37°C and 20ml·100ml^{-1}· min^{-1}· °C^{-1}, respectively. In thermally neutral environments, skin receives ~5-10 percent of cardiac output, whereas in conditions of heat stress, SkBF can reach 50-70 percent of cardiac output (CO), approaching 8 liters per minute (Johnson and Proppe 1996; Crandall et al. 1996).

Skin blood flow transfers heat by conduction between the deep body tissues and the skin. At low T_{co} and T_{sk} when sweating is not imminent, increases in SkBF allow T_{sk} to approach blood temperature (T_{bl}). Conversely, lowering SkBF allows T_{sk} to approach ambient temperature. Thus, one can note that the body has the ability to control sensible (conduction and radiation) heat loss by adjusting SkBF and subsequently T_{sk} (Sawka and Wenger 1988). If the ambient temperature is high enough to induce a sweating response SkBF will continue to increase. However, there will not be a dramatic increase in T_{sk}, as the evaporation of sweat tends to cool the skin at approximately the same rate as it is being warmed by the increase in SkBF. Thus, as supported by Sawka and Wenger (1988), there is usually little change in skin temperature and sensible heat exchange after the

onset of sweating. Therefore, increased SkBF serves to deliver the heat that is subsequently removed by evaporation (of sweat on the skin surface).

Cutaneous arterioles under tonic control of sympathetic vasoconstrictor fibers and arterioles are also regulated by a unique vasodilator system, which is responsible for 95 to 100 percent of the total increase in skin blood flow during heat stress (Rowell 1986). For the purposes of describing the control of SkBF, the skin surface can be divided into two regions: 1) acral (i.e., hands, feet, nose, and ears) and 2) non-acral (i.e., head, limbs, and trunk). In acral regions, cutaneous arterioles are innervated only through noradrenergic sympathetic nerves (Fox and Edholm 1963, Rowell 1977, Johnson 1986). Thus, it follows that all thermoregulatory and nonthermoregulatory reflexes in the acral skin regions are mediated by adjustments in active vasoconstrictor tone. Non-acral areas contain a much more complex system for controlling SkBF. Efferent neural control of SkBF to these areas is accomplished via two sympathetic neural pathways: a noradrenergic vasoconstrictor system (through postjunctional α_1- and α_2-receptors) and a separate vasodilator system of unknown neurotransmitter (Lewis and Pickering 1931; Grant and Holling 1938; Edholm et al.1957; Roddie et al. 1957; Fox and Edholm 1963; Greenfield 1963; Rowell 1983; Pergola et al. 1994). The complex nature of this system has made it very difficult for researchers to provide solid explanations pertaining to the control of non-acral SkBF.

The amount of SkBF supplied to a given area is mediated by two factors: 1) local factors (i.e., local heating/cooling, pressures, etc.) and 2) reflex control factors (e.g., from increased T_{co} affecting T_{sk} due to internal metabolic heat, from

exercise, etc.). A local factor that affects cutaneous circulation is a direct heating of the blood vessels themselves; however, the mechanisms for the vascular effects of local temperature are not yet well defined (Pergola et al. 1993). As stated by Johnson et al. (1986), local cooling potentiates, and heating weakens, the contractile response of vascular smooth muscle to norepinephrine and other constrictor agonists, apparently by changing the affinity of α_2-adrenergic receptors. Recent research, involving local heating and cooling and observing the subsequent cutaneous vascular responses has suggested a prominent role for postjunctional α_2-adrenoreceptors (Vanhoutte 1980; Flavahan 1991; Pergola et al. 1993). Local cooling has shown to enhance the affinity of α_2-adrenoreceptors for norepinephrine and depress the function of other elements involved in vascular smooth muscle function, with the net effect being a contraction of the cutaneous vascular smooth muscle (Vanhoutte 1980; Flavahan 1991; Pergola et al. 1993). Supportive in vivo studies have shown that α_2-aderenoreceptor blockade prevents the skin vasoconstriction produced by local cooling of the human finger (Ekenvall et al. 1988; Pergola et al. 1993).

As explained by Pergola et al. (1993), evidence from in vitro studies of canine cutaneous veins suggests that the reverse of the previously mentioned mechanism applies to the relaxation of vascular smooth muscle with direct local warming. However, it is further discussed that several studies of human forearm skin (a non-acral region) suggest only a minor role for the adrenergic system in this response. Pergola et al. (1993) has shown that: 1) intact adrenergic nerve terminals and norepinephrine release, but not sympathetic nerve activity per se,

are required for the immediate vasoconstrictor response to local cooling; 2) the stimulus for the release of the norepinephrine is local, possibly an axon reflex; 3) responses to prolonged local cooling involve mostly nonadrenergic mechanisms; and 4) the majority of the vasodilator response to local warming does not require an intact adrenergic system. Again, as mentioned previously, the complexity of this control system prevails.

1.1.4 *Control of skin blood flow during exercise*

As explained in the review by Johnson (1986), possibly the earliest evidence for a cutaneous vasoconstrictor response to exercise was that of Benedict and Parmenter (1929), who noted a fall in T_{sk} with exercise. This notion of a general exercise induced vasoconstriction of both acral and non-acral regions with the onset of dynamic exercise has been supported in numerous studies (e.g., Blair et al. 1961; Bevegard and Shepherd 1966; Zelis et al. 1969; Johnson and Park 1982; Hirata et al. 1983; Johnson 1990). Plethysmographic measurements confirm a pronounced vasoconstriction in the finger and hand with the onset of dynamic leg exercise (Johnson 1986). The blood flow to the forearm follows a very similar response with the onset of dynamic exercise. That is, forearm blood flow (FaBF) falls in response to the onset of leg exercise (Bevegard and Shepherd 1966; Blair et al. 1961; Johnson 1986). Johnson (1986) explains that FaBF drops off very quickly with the onset of exercise and reverses to a net vasodilation after several minutes of exercise. This later rise in FaBF is assumed to be a response to rising T_{co}. It is well accepted that FaBF, SkBF, and muscle blood flow (MBF)

reduce at the onset of exercise but there are conflicting data concerning the control systems. Zelis et al. (1969) separated the skin and muscle components of the forearm vascular response to exercise with epinephrine iontophoresis. The ambient temperature was 27°C, yielding a level of total FaBF of over 6ml·100ml^{-1}·min^{-1} before exercise. With exercise, SkBF fell by 49-74%. Further, it has been observed that the degree of vasoconstriction in the skin of the torso of the baboon was similar to that of limb skin with exercise. In contrast, Bevegard and Shepherd (1966) reported only small, transient reductions in oxygen content in blood from superficial veins in response to brief exercise (Johnson 1986). Finally, Blair et al. (1961) found that anesthesia of deep forearm nerves abolished the forearm vasoconstrictor response to leg exercise, whereas when a superficial cutaneous nerve anesthetic was administered the net effect was nil. Johnson (1986), concluded that the skin vasculature is a target for the vasoconstrictor responses to leg exercise. However, the cutaneous vasoconstrictor response is dependent on thermal status.

There are two neural control mechanisms thought to be responsible for the autonomic adjustments to exercise. The first originates in the central nervous system (CNS) and has been termed "central command" (Goodwin et al 1972). The other part is integral in skeletal muscle (Coote et al. 1971; McCloskey and Mitchell 1972) and is due to activation of metaboreceptors and mechanoreceptors reflecting the amount of work being performed (Mitchell et al. 1985). It has been shown that activity of the exercising muscle and the resulting metabolism (rather

than effort) determines the cutaneous vasoconstrictor response to the onset of dynamic exercise in humans (Friedman et al. 1991).

When one exercises beyond 5-10 minutes, the subsequent increase in T_{co} initiates a net cutaneous vasodilation (Johnson et al. 1974; Hirata et al. 1983; Johnson 1986). Johnson (1986) explained that although there is a competitive vasoconstrictor effect of exercise on SkBF, the net response is a cutaneous vasodilation that depends in part on thermal conditions (both internal and ambient). Johnson (1977) reported that forearm muscle blood flow does not rise during this period of leg exercise. Blood is shunted from the central circulation to the skin with associated reductions in cardiopulmonary pressures, systemic arterial pressure, and stroke volume (Saltin and Stenberg 1964; Eklund 1967). The progressive increase in SkBF during prolonged exercise occurs without an increase in cardiac output. Consequently, the increase in SkBF must be achieved by redistribution from other regions (Johnson 1986). The increase in SkBF during prolonged exercise may be self-limited. That is, when vasodilation occurs in the skin, cutaneous venous volume increases as a consequence of the higher distending pressures (Rowell 1983; Johnson 1986). Cardiopulmonary pressures fall as a result of the redistribution of blood volume to the skin. This may engage a cardiopulmonary baroreflex that would limit the rise in SkBF (Johnson 1986).

It is very important to note that the threshold for cutaneous vasodilation is increased by exercise through a delay in the activation of the active vasodilator system. Accordingly there is no apparent involvement of the vasoconstrictor system. T_{co} primarily determines the rate of SkBF. Above a T_{co} threshold for

vasodilation the increase in SkBF is proportional to the increase in T_{co}. If T_{sk} is increased, a reduction in the T_{co} threshold for vasodilation occurs. This happens without affecting the slope of the SkBF-T_{co} relationship (Wenger et al. 1975; Sawka and Wenger 1988).

There can be a great difficulty in interpreting changes in temperature regulation. Not only do we see differences in the thresholds from exercise to rest and vice versa but also from the influence of many other factors. Some examples of such factors include: the exercise intensity (Zelis et al 1969; Tam et al. 1978; Hirata et al. 1983; Smolander et al. 1987; Smolander et al. 1991), the type of exercise (Taylor et al. 1990), the exercise posture (Roberts and Wenger 1980; Johnson and Park 1981), the post-exercise posture (Kenny et al. 2000), the duration of the exercise period (Smolander et al. 1987), exercise training (Roberts et al. 1977), heat acclimatization (Roberts et al. 1977), circadian rhythms (Alfoldi et al. 1990; Tobler et al. 1993), dehydration (Thornton and Proppe 1988), and ambient temperature (Wenger et al. 1975; Johnson and Park 1982; Smolander et al. 1987). With so many factors playing a role in the human thermoregulatory response, one can note the complexity and difficulty in interpreting thermal changes within the body.

1.1.5 *Thermoregulatory and hemodynamic responses to postural changes*

In humans, precapillary resistance vessels generally constrict during the transition from supine to upright posture, increasing total peripheral resistance to facilitate maintenance of central blood pressure (Breit et al. 1993). This reaction is mediated centrally by adrenergic activation and vagal withdrawal in response to unloading of arterial and cardiopulmonary baroreceptors. Peripheral vasoconstriction during head-up tilt (HUT) (orthostasis) manifests itself in most regions throughout the body. That is, flow reductions can be noted in the cutaneous, muscular, splanchnic, and renal circulations (Blomqvist and Stone 1984; Rowell 1986). HUT of the human body unloads both cardiopulmonary and sinoaortic baroreceptors and reliably produces an increase in sympathetic activity in normal subjects (Goldsmith and Hasking 1992). This has been demonstrated using both plasma norepinephrine and norepinephrine kinetics (Levine et al. 1983; Davis et al. 1987; Goldsmith and Hasking 1992). Head-down tilt (HDT) loads both cardiopulmonary and sinoaortic baroreceptors by means of central blood volume and pressure increases. Head-down tilt in humans has been shown to cause an increase in FaBF (Goldsmith et al. 1984). This was supported by Breit et al. (1993), who showed that HDT increases loading of baroreceptors, eliciting vasodilation and increased microvascular perfusion.

The previous information serves to present the effect of nonthermal stimuli (such as HUT and HDT) on the major systems involved in

thermoregulatory function. One should note that the control of cutaneous microvascular flow is not only affected by thermoregulatory stimuli (heat, cold) but may be controlled also by nonthermoregulatory controls. The following information will address the effect of lower body pressures (nonthermoregulatory stimuli) on the same systems.

1.1.6 *Thermoregulatory and hemodynamic responses to the application of lower body positive and negative pressure*

Orthostatic stress in humans decreases central venous pressure, stroke volume, and cardiac output. Lower body negative pressure (LBNP) is commonly used to simulate orthostatic stress when negative pressures of – 40 to –50 mmHg are applied to mimic the average increases in distending pressure in the veins and arteries below the heart during upright posture (Escourrou et al. 1993). LBNP at –40 to –50 mmHg has been shown to reduce cardiac filling pressure due to local blood pooling in the legs. The body counteracts the drop in filling pressure (during LBNP) by eliciting baroreflex-mediated vasoconstriction in skeletal muscle and skin (Zoller et al. 1972; Rowell et al. 1973). Regional vasoconstriction during LBNP occurs because arterial pressure must be maintained by a fall in vascular conductance inasmuch as the fall in ventricular filling pressure restricts the ability to raise cardiac output (Escourrou et al. 1993). There is substantial research in agreement that baroreceptor unloading decreases SkBF (Rowell et al. 1973; Mack et al. 1988; Kellogg et al. 1990). This response is similar to the body's response to upright exercise in hot environments. Mack et al.

(1995) completed an experimental protocol that was designed to characterize the interaction between baroreceptor mediated blood pressure regulating reflexes and thermoregulatory control of cutaneous blood flow and sweating. In this study it was found that baroreceptor unloading (LBNP): 1) limited cutaneous vasodilation during dynamic exercise by increasing the T_{co} threshold for vasodilation and attenuating the rate of rise of SkBF per unit increase in T_{co}; and 2) attenuated thermoregulatory control of sweating by reducing the rate of rise in local chest sweat rate per unit increase in T_{co}. In the same study it was also confirmed that the limitations in thermoregulatory function by the application of LBNP were quickly reversed following removal of the orthostatic challenge.

Early work by Bevegard et al. (1977) using lower body positive pressure (LBPP) of 40 mmHg as a means of increasing venous return demonstrated reflex forearm vasodilation via low-pressure (or cardiopulmonary) baroreceptor loading. In the study by Shi et al. (1993) it was found that the hemodynamic responses of the human to progressive increases in LBPP while at supine rest might be a result of two different stimuli. An initial and primary stimulus of LBPP occurs between 0 and 20 Torr (1 Torr = 1 mmHg) LBPP and appears to be a result of translocation of blood volume from the lower body to the thorax. It was found that LBPP above 20mmHg increases blood flow to the forearm (Shi et al. 1993).

1.1.7 *Post-exercise thermoregulation*

Conventional thermoregulatory theory would suggest that an exercise induced elevated T_{co} would diminish upon a reduction in metabolic rate by the

cessation of exercise. That is, if all thermal defenses were to remain active (Thoden et al. 1994). However, it has been shown that increases in esophageal temperature (T_{es}) (by approximately 0.3-0.6 °C from endogenous heat loading (i.e., from moderate exercise) are sustained for approximately 65 minutes after the subject completes exercise (Thoden et al. 1994; Kenny et al. 1996; Kenny et al. 1996_b; Kenny et al. 1997; Kenny et al 1997_b; Kenny et al 1998; Kenny et al. 1998_b; Kenny et al. 2000). Evidence from these studies contradicts the conventional hypothalamic set point theory. Kenny et al (1996_b) showed that the prolonged plateau in T_{es} following moderate exercise is a function of exercise-related factors and not the increase in overall heat content. In their study the thermoregulatory responses to external heating (by warm-water immersion) and endogenous heating (by moderate exercise) were compared. It was found that post-exercise T_{es} was significantly elevated by 0.5 °C above the pre-exercise level and did not statistically differ from the T_{es} at which active skin vessel dilation (Th_{VD}) had occurred during exercise. In contrast, it was found that there was no significant elevation in T_{es} recovery following a single warm-water immersion (Kenny et al. 1996_b). Several studies have reported a post-exercise temperature profile (Johnson and Park 1982; Savard et al. 1988; Cable and Green 1990; Kraning and Gonzalez 1991; Kaciuba-Uscilko et al. 1992). However, none has shown a similar prolonged elevation in T_{co} following exercise; instead they have shown a gradual decay in T_{co} over time. Johnson and Park (1982) did show a similar trend in T_{es} response during intermittent exercise, but their experimental protocol maintained T_{sk} at a constant level for the experiment.

Some author's hold the view that the exercise-induced elevated core temperature above SP_{hy} is a discrepancy between the production and dissipation of heat (i.e., a load error or LE). This interpretation suggests that the hypothalamic thermostat is reset during high metabolic rates in a manner similar to the fever response (Nielsen 1938; Jackson and Hammel 1963; Nielsen 1966; Tam et al 1978; Taylor et al. 1984). The fundamental argument against an adjustment in SP_{hy} is that, unlike fever, the high metabolic rate of exercise is required to both elevate and maintain T_{co} (Sawka and Wenger 1988). Thus, as previously discussed, reducing the metabolic rate by the cessation of exercise should allow T_{co} to return to pre-exercise levels if thermal reflex defenses remain active (Thoden et al. 1994). Contrary to this notion, experiments conducted in our laboratory have noted a post-exercise hyperthermic state in subjects for as long as 65 minutes into recovery.

Despite the fact that there is, at most moderate levels of work, a cardiovascular reserve available to offset increases in T_{co}, accessing this potential does not seem to be a process that is adopted by the system. For example, the ability of the system to increase heat loss by increasing SkBF would seem to be a solution to limit any further rise or to even reduce the accumulation of heat produced from the increased metabolic rate. SkBF for an average man performing a workload equivalent to 50% VO_2max is approximately 2.6 $L \cdot min^{-1}$ (Rowell 1974; Taylor et al. 1984). Rowell (1974) and Taylor et al. (1984) noted that under full vasodilation, blood flow to the surface could be as high as 8 $L \cdot min^{-1}$. The cardiac output at 50% VO_2max represents about 50% of maximum capacity

(Sawka and Wenger 1988) and muscle blood flow at this intensity has been shown to be adequate to maintain tissue oxygenation (Savard et al. 1988), and prevent PtO2 and PvO2 from reaching critical levels (Astrand 1986). In most people, the 5.4 $L \cdot min^{-1}$ difference between SkBF at 50% VO2max and maximum rates is well within the capability of the remaining 50% of cardiac output. One can note that there is adequate cardiac output available to increase SkBF enough to attenuate increases in T_{co} during exercise. However, evidence has shown that thermoregulatory heat loss responses during prolonged exercise match metabolic heat production at a higher T_{co}.

The fact that the body does not tend to maintain a T_{co} at near resting levels (during exercise and into recovery) even though it has the capacity to do so suggests that it may be making a compromise or it may be attenuated by a dominating nonthermoregulatory factor. This residual effect, as postulated by Sawka and Wenger (1988), may be due to exercise-related factors such as cardiovascular (either central or peripheral), metabolic, endocrine, or neurochemical (i.e., interlukin-1, α-interferon, dopamine, and thyroid releasing hormone) changes during exercise that have thermal effects that significantly alter hypothalamic temperature regulation. Our laboratory has shown that loading of the arterial and cardiopulmonary baroreceptors by means of head-down tilt decreased the post-exercise resting threshold for forearm vasodilation (Kenny et al. 2000). This suggests a baroreceptor mediated influence on post-exercise resting thermal homeostasis. There is recent evidence that addresses the baroreceptor influence, in that a post-exercise hypotensive effect has been

observed following acute bouts of exercise (Coats et al. 1989; Halliwill et al. 1996). This hypotension exists due to altered baroreflex control of sympathetic outflow and the transduction of sympathetic activity into vascular resistance after dynamic exercise (Halliwill et al. 1996). The post-exercise hypotensive effect has also been observed following dynamic exercise by Wilcox et al. 1982, Kaufman et al. 1987, and Brown et al. 1993 who concluded that it is related to cutaneous cooling in recovery. They suggest that the inverse relationship between T_{sk} and blood pressure underlies a probable peripheral (i.e. lower limb) and/or visceral organ pooling of blood, thus trapping deep body heat and reducing stroke volume by small reductions in venous return. These responses demonstrated a physiological reduction in the capacity for heat loss despite an elevation of T_{co}.

The complexity of the human thermoregulatory system prevails far into exercise recovery. This is clearly outlined in the previously presented information. One can note the many thermal and nonthermal influences that may contribute a hyperthermic state in the recovery phase following dynamic exercise. Consideration must be given to these factors as it has been shown that, in one way or another, they may exhibit modulating effects on warm thermoregulatory response thresholds.

1.2 Research Hypotheses

- This study evaluated the hypothesis that the reversal of post-exercise venous pooling through the application of lower body positive pressure results in a decrease in the post-exercise resting esophageal temperature threshold for cutaneous vasodilation.

- It was also hypothesized that the post-exercise esophageal temperature threshold for sweating decreases with lower body positive pressure application.

1.3 Assumptions

- All subjects adhered to pre-experimental guidelines for sleep, hydration, diet, and physical activity as set forth in their orientation session.
- Subjects did not begin any type of prescription/non-prescription drug use (including cigarette smoking) during the experimental period.
- Subjects did not undergo significant anthropometric changes (i.e., weight gain, or loss) during the experimental period.
- Measuring sweat response at one site on the upper back will give an accurate indication of sweat response in all subjects.
- Esophageal temperature is an accurate indication of body core temperature in humans.

1.4 Delimitations

- The subject pool only included healthy males aged 18 to 35 years, who were not engaged in any type of rigorous and/or regimented training program.

- Smokers, those who were receiving drug therapy, and those involved in regular recreational drug use were excluded from the subject pool.

- Subject selection was limited to those individuals between ~10 - 25% body fat by the hydrostatic weighing technique.

2.0 METHODS

2.1 *Subjects*

With approval from the Faculty of Health Sciences Human Ethics Committee, 6 healthy men were studied after providing written, informed consent. None had any history of cardiovascular or respiratory disease, and although all participants were physically active, none engaged in regimented physical training of any type. Subjects were (mean ± SD) 25 ± 5 years of age, 179.5 ± 5.8 cm tall, and weighed 82.1 ± 7.5 kg.

2.2 *Instrumentation*

Core temperature was measured by means of inserting an esophageal thermocouple, through one nostril, to the level of the heart (approximately one quarter standing height). Skin temperature was measured at 7 sites by heat flow sensors (Concept Engineering, Old Saybrook, CT, USA), and the area-weighted mean $\bar{T}_{sk}$ was calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 19%, anterior thigh 21%, posterior calf 18%. Skin blood flow (SkBF) was measured from the left, mid-anterior forearm at 2 sites separated by a distance of approximately 10 cm (blood perfusion monitor Perimed, PeriFlux System 5000). Laser Doppler skin blood flow sensors were taped to cleaned skin, in an area that did not appear to be superficially vascular, and where consistent readings were noted. Only relative SkBF values were used and no attempt was made to present absolute SkBF. Sweat rate was measured using a ventilated capsule (≈5.0 x 3.5 cm) placed on the

cleaned surface of the upper back and sealed to the skin using a highly adhesive, hypoallergenic dressing. Anhydrous air was passed through the capsule over the skin surface at a rate of $1 L \cdot min^{-1}$. Vapor density of the effluent air was determined based on the relative humidity and temperature of the air measured by an Omega HX93 Humidity and Temperature Sensor (Omega Engineering, Stanford, CT, USA).

Oxygen consumption was measured (breath by breath) using an automated metabolic analyzer (MedGraphics, St-Paul, MN, USA).

Mean arterial pressure (MAP) was continuously recorded from the electrical integration of the pulsatile blood pressure signal obtained from the middle digit (Ohmeda, 2300 Finapres) by the Penaz method, referenced at heart level (at the third intercostal space). Heart rate was measured, at R-R intervals, using a Polar coded transmitter and recorded continuously with a Polar Advantage interface (Polar Electro, Finland).

Core and skin temperatures, sweat rate, and SkBF data were digitized (Hewlett Packard, data-acquisition module, model 3497A) at 10-s intervals, displayed graphically on the computer screen, and recorded in spreadsheet format on a hard disk (Hewlett Packard, model PC-312, 9000). Heart rate data were downloaded and graphed continuously with Polar Precision Performance software and saved to a hard disk. Blood pressure data were recorded continuously at 5 s intervals and recorded to a hard disk.

2.3 *Experimental protocol*

Subjects performed one incremental maximal VO_2 test on a cycle ergometer on the first day. These data were used to select the workload for the submaximal experimental exercise trials.

Each subject performed a total of 4 experimental trials that were carried out in a random order in the morning between 7:00 am and 8:00 am, after a 24 h period without heavy exercise, abstinence from stimulants and alcohol, 8-h of sleep, and a minimum ingestion of 0.25 litres of water per waking hour. To avoid thermal stimuli caused by extraneous metabolic factors, subjects were instructed to fast at least 4 h prior to experimentation; although, water ingestion was encouraged during this time. Furthermore, subjects were to avoid excessive walking or movement in the period between awakening and experimentation.

Upon arrival to the laboratory subjects clothed in shorts and athletic shoes and were instrumented appropriately. They were then fitted with an upper body water-perfused suit (covering the torso, arms and head). Subjects were then placed into the LBPP chamber, in an upright semi-seated position, sealed at the iliac crest.

Each of the four experimental trials commenced with a 15 min baseline-resting period after which subjects either exercised (Ex) or remained resting (NoEx). For the exercise treatment the subjects performed 15 min of cycle ergometer exercise at 70% of their predetermined VO_2max. For the No-Exercise treatment the subjects were instructed to rest in the semi-seated seated upright position for 15 min (see Fig. 1).

To measure the effect of post-exercise venous-pooling on the resting post-exercise Th_{VD} and Th_{SW}, post-treatment resting measurements of Th_{VD} and Th_{SW} were conducted. Immediately following both the NoEx and Ex treatments, subjects either remained (NoEx treatment) or were placed (Ex treatment) in a semi-seated upright position within the pressure chamber sealed at the level of the iliac crest. They were then exposed to either 50 mmHg lower body positive pressure (LBPP) or no lower body positive pressure (NoLBPP) during which time cool water (~20°C) was circulated through the water-perfused suit until forearm cutaneous vasoconstriction was noted (NoEx ~65 min, Ex ~95 min). Mean skin temperature was then increased at a rate of $5.0°C \cdot hr^{-1}$ as the water circulating through the suit was progressively increased to 47°C and cutaneous vasodilation and sweating was noted (~75 min).

The inclusion of the cooling phase prior to whole body heating was not intended to burden or to address thermoregulatory function during cooling. Although it appears that the cooling phase may have added a degree of thermal history, it was included to ensure that all subjects were brought to the same baseline so that true release of vasoconstrictor tone and subsequent vasodilation during whole-body heating could be noted in all trials.

To compare thresholds between conditions in which both esophageal and mean skin temperatures were changing, the following equation (Matsukawa et al. 1995) was used to correct the esophageal temperature [$T_{es(calculated)}$] for a designated skin temperature [$\bar{T}_{sk(designated)}$]:

$$T_{es(calculated)} = T_{es} + [\beta/(1-\beta)][\bar{T}_{sk} - \bar{T}_{sk(designated)}];$$

$\bar{T}_{sk(designated)}$ was set as the average $\bar{T}_{sk}$ at rest (33.5 °C) and β = fractional contribution of the skin to the vasodilation (0.2) (Wenger et al. 1975) and sweating (0.1) (Nadel et al. 1971).

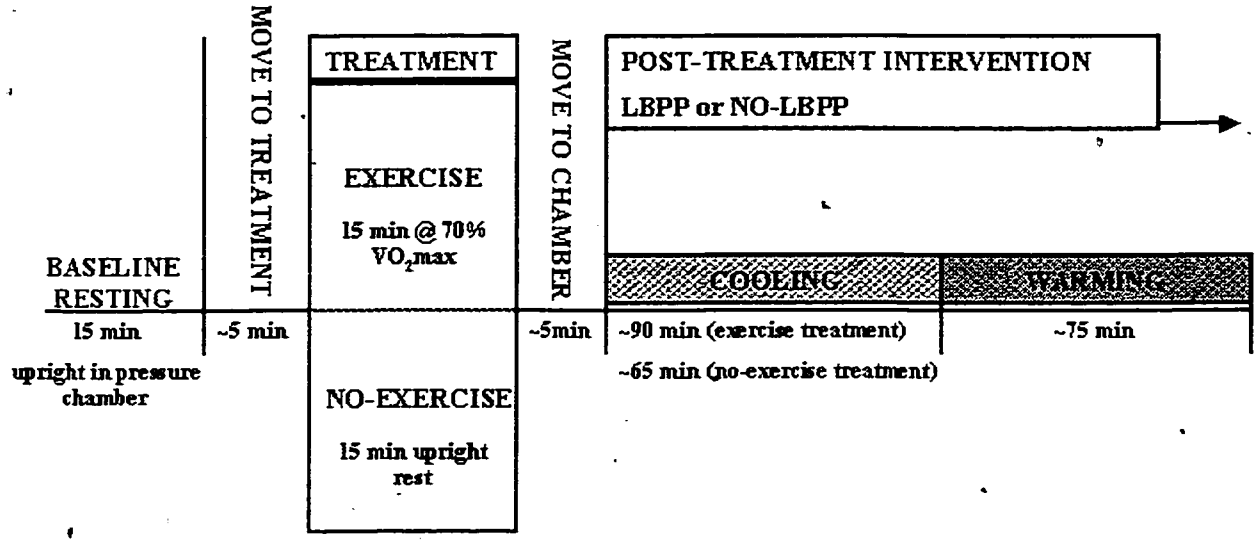

Fig. 1 Timeline of Experimental Events

2.4 *Analysis of Results*

For the purpose of comparison, thermoregulatory response thresholds (for vasodilation and sweating) were identified for each condition as follows: 1) Ex/LBPP; 2) Ex/NoLBPP; 3) NoEx/LBPP; 4) NoEx/NoLBPP. A repeated measures ANOVA was used to test for significant inter-condition differences in $T_{es\ (calculated)}$ at each warm response threshold. In the event of statistical significance ($P < 0.05$), a Tukey's HSD test was used to identify significant differences. The data for warm response thresholds are presented as mean ± SE.

For blood pressure, data were analyzed for significance during baseline resting and post-treatment in each condition using repeated measures T-tests ($P<0.05$). A repeated measures ANOVA was used to test for significant differences in heart rate between Ex/LBPP and Ex/NoLBPP and between NoEx/LBPP and NoEx/NoLBPP. Values were compared during baseline resting (15 min), during the treatment period (last 5 min), and at corresponding 5 min intervals post-treatment for 30 min ($P < 0.05$). Data are presented as mean ± SE.

3.0 RESULTS

There were no inter-condition differences in the rate of warming for the suit perfusate (~19.5 °C·hr^{-1}). Thus, mean skin temperature was increased at the same rate for all subjects in all conditions (~5.0 °C·hr^{-1}). Resting T_{es} and $\bar{T}_{sk}$ were similar for all conditions and remained stable and consistent during the 15 min baseline resting period.

3.1 *Effect of exercise treatment on Th_{VD}, Th_{SW}, MAP, and HR*

Th_{VD} was significantly elevated in the Ex/NoLBPP condition (0.33 - 0.36 °C) relative to the other experimental conditions ($P < 0.05$) (Table 1). There was no difference in Th_{SW} between the Exercise/NoLBPP condition and the other experimental conditions ($P < 0.05$) (Table 2). Post-exercise MAP was significantly lower (~ 4 mmHg) than baseline-resting MAP in the Ex/NoLBPP condition ($P < 0.05$) (Fig. 2). The mean treatment period heart rate was the same for both exercise conditions (166 ± 6 beats·min^{-1}). Post-exercise heart rate remained elevated (≥20 beats·min^{-1}) in the Ex/NoLBPP condition ($P < 0.05$). (Fig. 3)

3.2 *Effect of LBPP on Th_{VD}, Th_{SW}, MAP, and HR*

Post-exercise LBPP application (Ex/LBPP) resulted in a lowering of recovery Th_{VD} (0.34 °C) ($P < 0.05$), when compared to the Ex/NoLBPP condition (Table 1). Th_{SW} was comparable to the other conditions with the post-treatment application of LBPP (Table 2). Post-exercise LBPP increased MAP (~12 mmHg)

from baseline resting values ($P < 0.05$). MAP was significantly elevated (~11 mmHg) with LBPP application in the no-exercise control condition ($P < 0.05$) (Fig.2). At 30 min post-exercise, heart rate fully recovered to resting level with LBPP (70 ± 3 beats·min^{-1}). In the No-Exercise condition heart rate was significantly lowered (~ 13 beats·min^{-1}) with LBPP application ($P < 0.05$). (Fig. 3)

	Exercise	No-Exercise
Onset of Vasodilation	**LBPP**	**LBPP**
Actual $\bar{T}_{sk}$	33.43 ± 0.35 °C	33.84 ± 0.40 °C
Actual T_{es}	36.40 ± 0.07 °C	36.33 ± 0.11 °C †
$T_{es(calculated)}$	36.38 ± 0.10 °C	36.41 ± 0.18 °C
Onset of Vasodilation	**NoLBPP**	**NoLBPP**
Actual $\bar{T}_{sk}$	33.85 ± 0.40 °C	32.87 ± 0.47 °C
Actual T_{es}	36.66 ± 0.14 °C †	36.56 ± 0.10 °C
$T_{es\ (calculated)}$	36.74 ± 0.17 °C *	36.40 ± 0.14 °C

Table 1. Vasodilation threshold values for the 4 experimental conditions. Values are means ± SE; n = 6 subjects. * Indicates a significant difference in $T_{es(calculated)}$ at the onset of vasodilation in the Ex/NoLBPP condition relative to the other 3 conditions ($P < 0.05$). † Indicates a significant difference in *actual* T_{es} values at the onset of vasodilation ($P < 0.05$).

	Exercise	No-Exercise
Onset of Sweating	**LBPP**	**LBPP**
Actual $\bar{T}_{sk}$	35.69 ± 0.22 °C	35.92 ± 0.13 °C
Actual T_{es}	36.61 ± 0.07 °C	36.66 ± 0.05 °C
T_{es} *(calculated)*	36.80 ± 0.09 °C	36.87 ± 0.07 °C
Onset of Sweating	**NoLBPP**	**NoLBPP**
Actual $\bar{T}_{sk}$	35.38 ± 0.32 °C	35.04 ± 0.43 °C
Actual T_{es}	36.79 ± 0.10 °C	36.52 ± 0.26 °C
T_{es} *(calculated)*	36.94 ± 0.13 °C	36.63 ± 0.23 °C

Table 2. Sweat threshold values for the 4 experimental conditions. Values are means ± SE; n = 6 subjects. Note that there were no significant differences in $T_{es\ (calculated)}$, T_{es} or T_{sk} at the onset of sweating between any of the conditions.

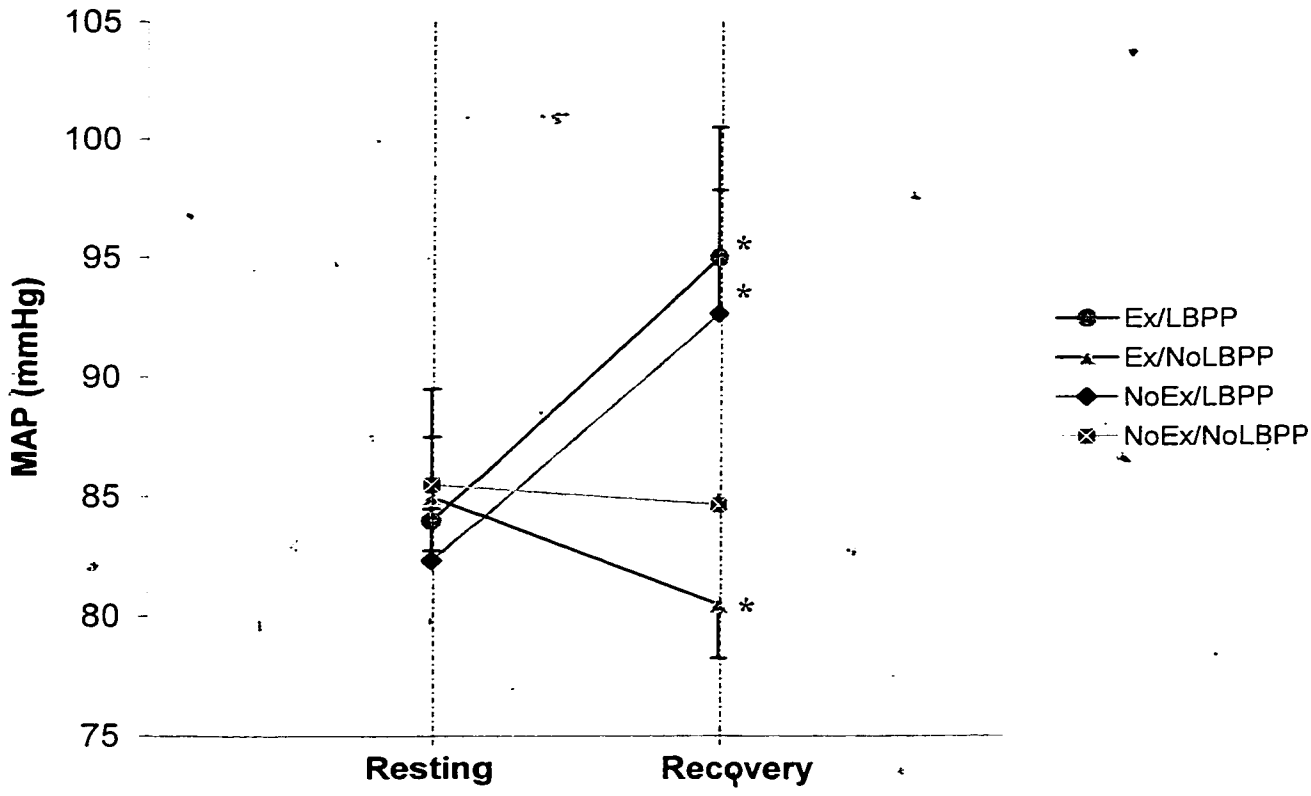

Fig. 2. Mean arterial pressure (MAP) at rest (15 min) and during the first 15 min post-treatment recovery for each experimental condition.
* Indicates significant difference between baseline-resting and post-treatment recovery measures ($P < 0.05$). Error bars represent SE.

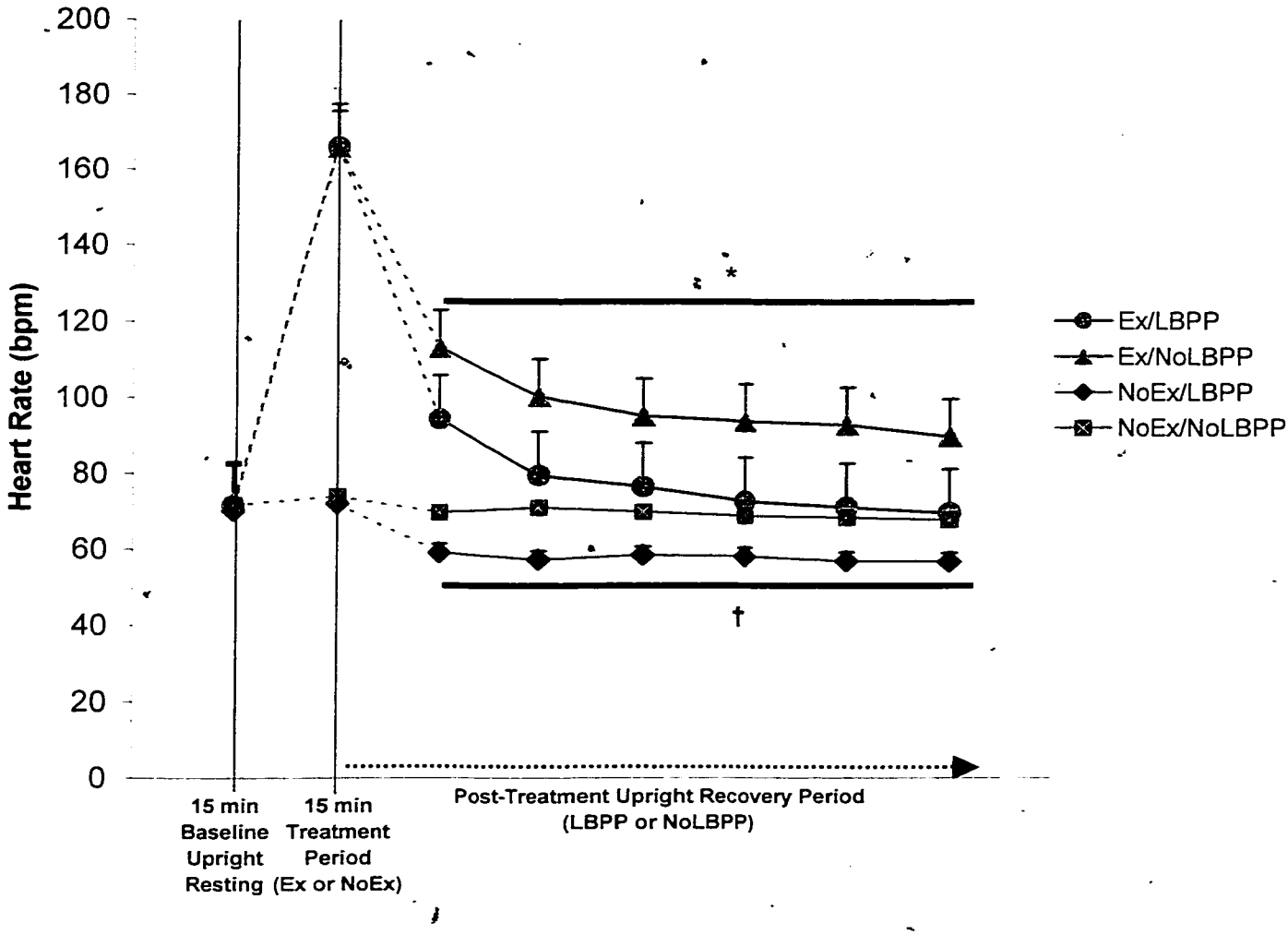

Fig. 3. Effect of the 4 experimental conditions on changes in heart rate during baseline resting, treatment, and 30 min post-treatment. † Indicates significant difference between No-Exercise conditions (NoEx/LBPP and NoEx/NoLBPP) ($P < 0.05$). * Indicates significant difference between Exercise conditions (Ex/LBPP and Ex/NoLBPP)($P < 0.05$). Error bars represent SE.

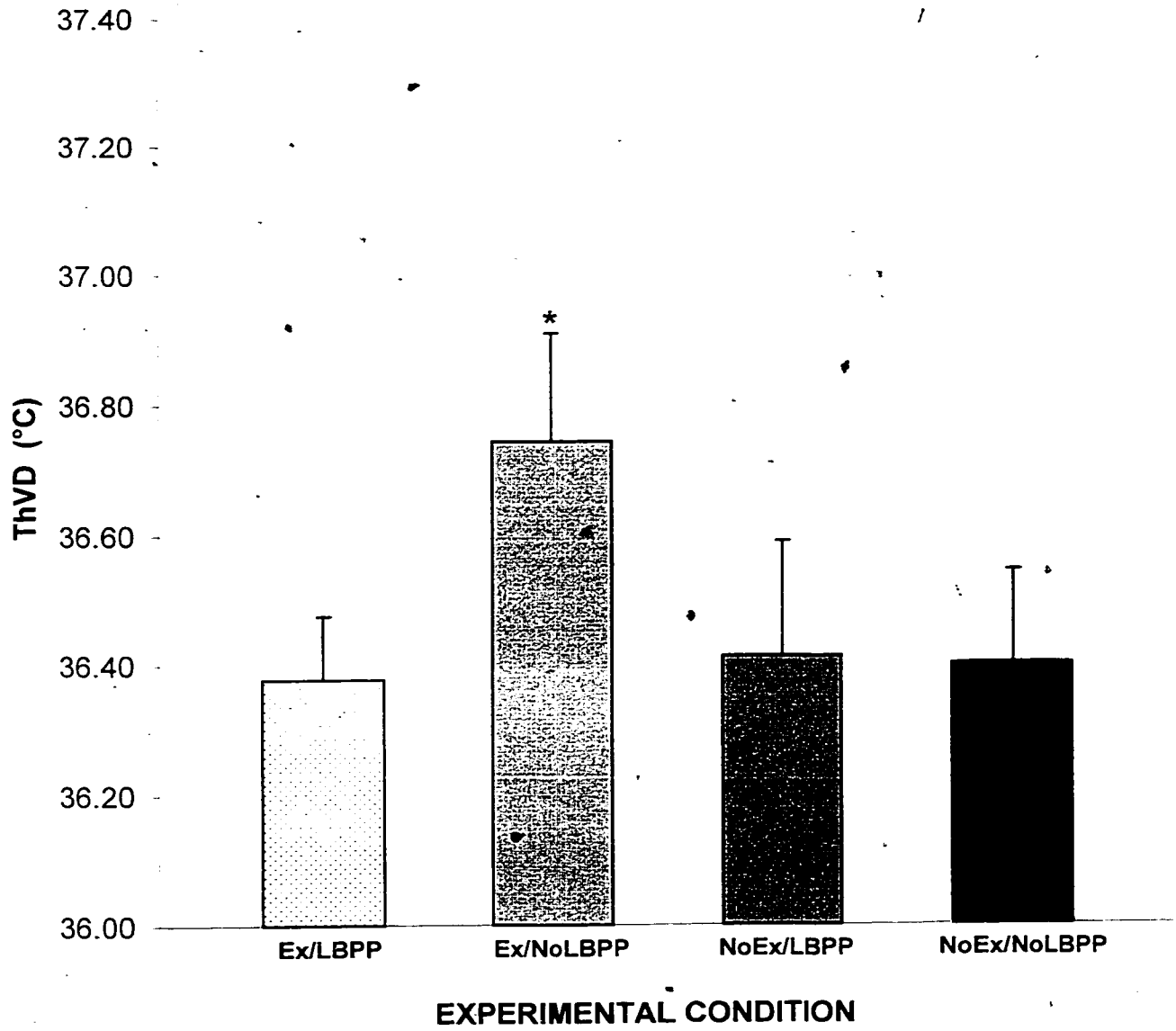

Fig. 4. Comparison of Th_{VD} in the 4 experimental conditions. * Indicates a significant difference between Ex/NoLBPP and the other 3 experimental conditions ($P < 0.05$). Error bars represent SE.

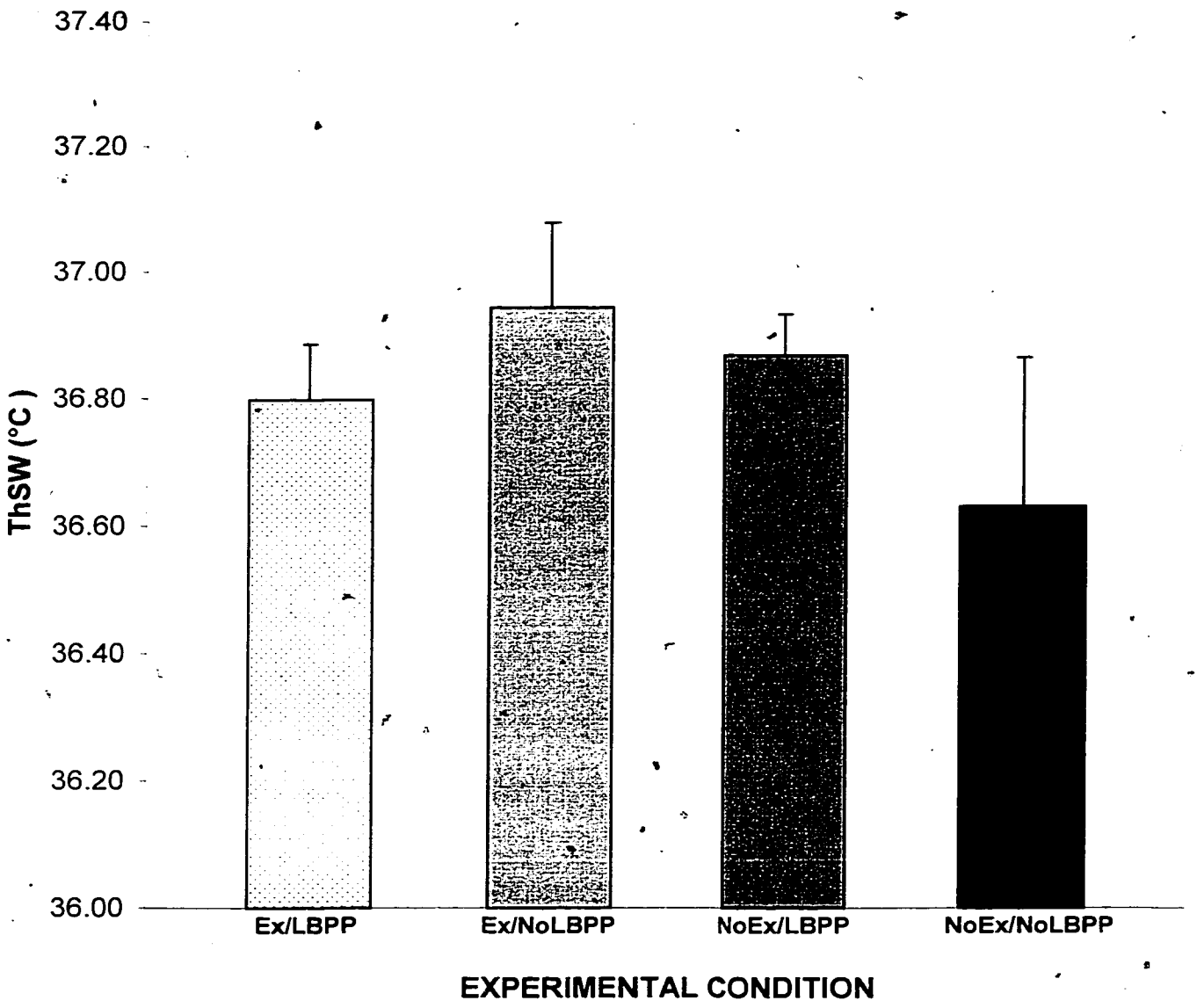

Fig. 5. Comparison of Th_{SW} in the 4 experimental conditions. Note that there were no significant differences in sweat thresholds between experimental conditions. Error bars represent SE.

4.0 DISCUSSION

This study is the first to evaluate the role of baroreceptor control on the post-exercise esophageal temperature thresholds for forearm cutaneous vasodilation (Th_{VD}) and sweating (Th_{SW}). By reversing the impact of post-exercise venous pooling through the application of LBPP it was shown that increases in post-exercise Th_{VD} are sensitive to nonthermoregulatory baroreceptor activity. The results also indicate that post-exercise Th_{SW} is not sensitive to baroreceptor unloading or loading.

The observations within the present study were not a result of differences in rate of change of mean skin temperature, as cooling and warming rates were similar for all subjects in each experimental condition. Further, all experiments were commenced at approximately 8:00 am, thus completing well before zenith. As a result, it can be assumed that the observations were not confounded by circadian shifts in set point.

The results form this study, regarding post-exercise Th_{VD}, are congruent with previous research. Several studies have documented increases in Th_{VD} associated with baroreceptor unloading (through orthostatic stress). Early research involving head-up tilt has proven to evoke cutaneous vasoconstrictor activity in humans (Mosley 1969). Other studies demonstrated similar results with the application of lower-body negative pressure (Beiser et al. 1970, McNamara et al. 1969; Rowell et al. 1973, Prasad et al. 1998, Tripathi and Nadel 1986). The data from this study supports these previous findings. Observations of blood pressure and heart rate data clearly indicate that exercise resulted in post-exercise

orthostatic stress (consistent with baroreceptor unloading). This manifested itself as a significant post-exercise hypotension coupled with elevations in heart rate. These findings are in accordance with other studies that have observed a hypotensive condition in humans following dynamic exercise (Coats et al. 1989, Somers et al. 1987). The present study confirmed that there was an elevation in post-exercise Th_{VD} (0.34 ^{o}C) during the post-exercise recovery period. When post-exercise venous pooling was modified (with the application of LBPP), the residual effect of exercise on Th_{VD} was abolished. That is, application of post-exercise LBPP resulted in Th_{VD} being comparable to that measured at rest.

The observed significant drop in MAP from rest to exercise (85 ± 3 mmHg to 83 ± 3 mmHg, respectively) supports the existence of blood pooling in the previously active musculature. A very likely postulate is that in a response to this post-exercise hypotension, peripheral vasoconstriction was elicited via the cardiopulmonary baroreceptors. The data suggest that peripheral vasoconstriction was not adequate; thus, a persisting post-exercise hypotension was observed. It is clear that nonthermoregulatory modifications (such as this) would have significant thermoregulatory consequences. Thus it is plausible that the increase in post-exercise Th_{VD} was the result of non-thermoregulatory baroreceptor modulation.

Although the mechanism(s) for control of skin blood flow prior to and during exercise have been described (Kellogg et al. 1991_a, Johnson and Park 1981, Smolander et al. 1991, Taylor et al. 1988), post-exercise regulation remains relatively unexamined. However, it has been shown that the cutaneous vasodilator

system is under baroreceptor control (Kellogg et al. 1991_a). It is also known that cutaneous vascular tone is a determinant of blood flow and blood pressure regulation during both exercise and upright posture (Rowell and O'Leary 1990).

Previous research has suggested that nonthermoregulatory baroreceptor modulation is responsible for increases in the threshold for cutaneous vasodilation observed during exercise (Kellogg et al. 1991, Johnson and Park 1981, Smolander et al. 1991, Taylor et al. 1988). This suppressive reflex on cutaneous blood flow is related to the vasoconstrictor influence on cutaneous blood flow observed at the onset of exercise (Smolander et al. 1991, Taylor et al. 1988). The temporary reduction of skin blood flow (at the onset of exercise) is necessary to ensure adequate blood flow to the active skeletal muscle needed to support muscle metabolism and maintain adequate cardiac filling (Rowell and O'Leary 1990). Because the baroreceptor reflex can modulate active vasodilator activity (Kellogg 1990), the suppressive reflex on cutaneous blood flow most likely reflects central inhibition of vasodilator outflow due to baroreceptor unloading.

Thus it would seem that skin blood flow control during and following exercise are subject to significant modifications by baroreceptor reflexes. Because acute reductions in central venous pressure have been shown to delay or decrease the rise in cutaneous blood flow during heat stress (Mack et al. 1988, Morimoto 1990, Nadel et al. 1980), a reasonable postulate is that cardiopulmonary baroreceptors are involved in the post-exercise cutaneous vasoconstriction during resting recovery. Modification of baroreceptor response on cutaneous vascular tone would be manifested either as an activation of

sympathetic adrenergic vasoconstrictor nerves or as a withdrawal of active vasodilator activity (Kellogg et al. 1990).

It is interesting to note that Th_{SW} was not different in any of the conditions. The results from this study demonstrate that baroreceptor unloading caused by post-exercise venous pooling and post-exercise baroreceptor loading via LBPP had no measurable effect on Th_{SW}. In the Ex/NoLBPP condition a significant hypotension was measured for at least 15 min post-exercise and subsequently heart rate remained elevated for greater than 30 min into recovery. This response is consistent with that of baroreceptor unloading (Prasad et al.1998). In the Ex/LBPP condition the residual effects of exercise on MAP and heart rate were reversed. Thus, if the baroreflex had a significant modulating effect on post-exercise Th_{SW}, then we should have observed a significant difference in post-exercise Th_{SW} among two or more of the conditions examined.

The fact that sympathetic nerve recordings from sudomotor nerve fibers show cardiac rhythmicity brings forth the notion that sweat gland activity is modulated by changes in baroreceptor activity (Mack et al. 1995). Kellogg et al. (1991_a and 1991_b), clearly identify the weakness in assuming that sweat gland activity and active cutaneous vasodilator drive are under exacting control systems. Some feel that the thermoregulatory sweat response is too slow to be mediated by a neural reflex and may be due to the action of a circulating agent, possibly arginine vasopressin (Solack et al. 1985). However, the exact control of sudomotor function remains unclear.

Kenny et al. (1997_b) reported an increase in Th_{SW} following dynamic exercise. Their assessment of Th_{SW} involved whole body warm water immersion. It is known that water immersion may modify baroreceptor activity through peripheral displacement of blood volume due to hydrostatic forces (Nielsen et al.1984). Secondly, their post-exercise evaluation of Th_{SW} involved a seated posture, which was different than the exercise posture. It is well known that there is significant baroreceptor activity associated with changes in posture.

In a more recent study, Kenny et al. (2000_b) reevaluated post-exercise resting warm thermoregulatory response thresholds. In an attempt to control for possible baroreceptor interactions a whole body water-perfused suit was used. Again, they demonstrated an increase in post-exercise Th_{SW}. Their reported increase in Th_{SW} was similar to that of Kenny et al. (1997_b), despite possible differences in baroreceptor activity between the studies. This serves to support the findings of the present study, in that changes in baroreceptor activity seems to have no measurable effect on Th_{SW}. Thus, previously reported increases in post-exercise Th_{SW} are not likely due to baroreceptor modulation but due to some other residual exercise related factor.

Contrary to findings by Kenny et al. (1997_b) and Kenny et al. (2000_b), the present study found no post-exercise elevation in Th_{SW}. Since the rate of warming of the skin in the present study (~5.0 $^{\circ}C \cdot h^{-1}$) was comparable to that of Kenny et al. (2000_b), then differences in findings were not due to differences in the rates of warming. However, the contribution of skin temperature to the integrated signal for thermoregulatory response was different between studies. More specifically,

the present study utilized a water perfused suit that only covered the upper body and head. Kenny et al. (2000_b) used a whole body water perfused suit. Thus, within their assessment of Th_{SW} there would have been a greater and more uniform thermoregulatory drive from changes in skin temperature. Human studies have shown similar central to peripheral thermal sensitivity ratios dictating the control of skin blood flow (Wenger et al. 1975) and sweating (Nadel et al. 1971). However, our understanding of the relative sensitivities of specific thermosensitive sites within either the core or cutaneous regions is less defined (Patterson et al. 1998). Some studies involving animals have identified inter-regional differences in cutaneous thermosensitivity (Hales and Hutchinson 1971, Necker 1977). Other studies, to a lesser degree, have addressed inter-regional differences in cutaneous thermosensitivity in humans (Nadel et al 1973, Werner and Heising 1990). However, the relative contributions made by thermosensitive skin sites to the control of human sudomotor function are still relatively undefined. Thus, the differences in the integrated signal may account for differences in Th_{SW} between the present study and those of Kenny et al. (2000_b) and Kenny et al. (1997_b).

5.0 CONCLUSION

These data demonstrate that post-exercise LBPP application results in an esophageal threshold for cutaneous vasodilation that is comparable to that observed at rest. The results from this study also confirm previous conclusions that exercise exerts residual effects on the post-exercise threshold for cutaneous vasodilation. Further, this study presents a plausible explanation for the reported increase in the post-exercise threshold for cutaneous vasodilation. That is, it seems that the non-thermal baroreceptor response to post-exercise hypotension significantly influences cutaneous vasomotor control during exercise recovery. The findings from this study suggest that post-exercise Th_{SW} is not sensitive to baroreceptor activity and therefore supports the notion that sudomotor and vasomotor activity are not governed by the same control systems. Continuing research needs to be conducted to further investigate post-exercise vasomotor and sudomotor control. In addition, future research is needed to address the apparent discrepancies concerning the existence of a post-exercise increase in Th_{SW} and the relative contribution of local skin temperatures to Th_{SW}.

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APPENDIX A

Subject Descriptive Data

Subject	Age (years)	Ht. (cm)	Wt. (kg)	%BF (Hydrostatic)	VO2max (ml/kg/min)	Body Surface Area (m^2)
1	23	177.5	76.5	9.9	40.9	1.94
2	27	176	84	15.7	48	2.03
3	20	188	81.5	8.7	42.6	2.06
4	35	176	94.8	26.2	37.7	2.15
5	19	185.5	73	13.9	41.3	1.94
6	25	174	83	15.2	51.9	2
MEAN	**25**	**179.5**	**82.1**	**14.9**	**43.7**	**2.0**
±SD	±5	±5.8	±7.5	±6.2	±5.2	±0.08

APPENDIX B

Ethics Documents

UNIVERSITY HUMAN RESEARCH ETHICS COMMITTEE
QUESTIONNAIRE ON RESEARCH PROCEDURES CONCERNING RESEARCH CONDUCTED USING HUMAN SUBJECTS
FOR THE HUMAN RESEARCH ETHICS COMMITTEE - FACULTY OF HEALTH SCIENCES

Application submitted by:	*Department*	*Office Address*	*Telephone*
Glen Kenny (Ph.D.)	School of Human Kinetics	125 University Ave. Montpetit Hall, Rm. 376 Ottawa, Ont. K1N 6N5	Office: (613) 562-5800 ext. 4282

1) Title of Research Project:
Baroreceptor influence on post-exercise thermoregulatory responses

To what source of funding is the application being submitted, if any?

2) Who are the subjects (please be as specific as possible)

The sample for this study will consist of physically active, male and female subjects between the ages of 19-45 years of age. Subjects will be physically active in order to minimize the possibility of muscle soreness and pre-mature fatigue frequently experienced by sedentary subjects following exercise. Subjects will not exceed the age of 45 to avoid potential medical problems (cardiovascular diseases, etc.) which is associated with an increase in age.

3) Number of subjects involved in the study

Ten subjects will be asked to volunteer for each portion of main study program.

4) How will the subjects be recruited for this study?

Subjects will be recruited by direct communication with university students and staff members.

5) How will you obtain the informed consent of the subject(s) (and where if applicable of parents/guardian)?

After an expression of interest in volunteering for the study, a personal preliminary session will be held with each subject. During this time, the attached information letter and consent to be a subject form will be presented to the subject and they will be verbally instructed of the objectives and procedures of the study. In addition, subjects will be introduced to the measurement instruments to be used in the study. It is hoped that the subject will hold an interest in continuing in the study at this point, but assurance will be given to the subject that he/she may withdraw from the study at any time without repercussions of any kind. If the subject still shows an interest in participating in the study, the attached consent form will be presented for completion and signature.

6) Specify the level (none, low, moderate, or high) and describe the nature of risk (legal, physical, psychological, or social) associated with each major procedure with human subjects in this research. Justify the choice of this procedure(s) and state how you propose to minimize the risk.

There are some physical risks, although no social or psychological risks, associated with the subjects participation in this study. Performing exercise always carries the potential for minor soreness associated with the fatigue of muscles following moderate or near maximal intensity work. Since the volunteering subjects chosen will be physically active and satisfy the university ethics committee form for exercise (PAR-Q), the risk of injury from moderate to heavy exercise is minimal. All exercise, except the initial test, will be performed at less than maximum and control of its termination remains with the subject.

The esophageal probes may cause minor irritation to the nasal passage and esophagus. The esophageal probe is an extensively used method of measuring core temperature by researchers (see current ethics clearance for "*Investigation of Exercise and Ambient Temperatures as Influences on Thermoregulatory Reflexes in Humans*" granted to J. S. Thoden for studies incorporating nearly identical protocols), and is required to compare results with the current and past literature on post-exercise hyperthermia. Over 700 similar exercise protocols have been conducted in our laboratory with no medical problems or serious health related issues have been reported.

The use of lower body negative pressure (LBNP) and lower body positive pressure (LBPP) as a means of eliciting a local blood pooling effect (in the legs, as displayed during exercise) and as a means of increasing venous return (comparable to the effect of bed-rest) to the lower body respectively, is common practice in research today (20, 21, 22, 23). Since the use of lower body negative pressure will only induce mild hypotension (i.e., low blood pressure), the cardiovascular and physical risks are minimal. Since there is a possibility of the hypotensive subject feeling light headed, he/she will remain in the seated position throughout the application of LBNP, so as to minimize any chance of injury due to loss of coordination or dizziness. The application of LBPP will only induce a mild hypertension for a short period of time (until the body reestablishes homeostasis); thus this risk is also low.

Finally, minor skin irritation may occur due to the method of applying the surface temperature thermistors to the skin but is reduced through the use of special hypoallergenic adhesive tape. All hygienic, safety and experimental procedures associated with standard pulmonary and exercise laboratories are in place. All instruments utilized in this study will be properly sterilized and the laboratory will be under aseptic conditions.

7) Specify the level (none, low, moderate, or high) and describe the nature of discomfort (legal, physical, psychological, or social) associated with each major procedure with human subjects in this research. Justify the choice of this procedure(s) and state how you propose to minimize the discomfort.

No legal or psychological discomforts are associated with the procedures of this study. To avoid psychological discomfort to the subject, only those involved in the data collection will be allowed (i.e., investigators only) to be present during experimental sessions and to view data. Some minor physical discomfort may be experienced caused by the esophageal probe being in place for a duration for over 2 hours, as well as dryness in the throat as the subject is asked not to swallow excessively for the duration of the testing in order to maintain probe stability. The subject will be given water in such cases where the throat becomes sore. Low physical discomfort may also be associated with the exercise phases of the test. This is minimized by the use of physically active subjects.

8) Specify the method(s) by which you plan to ensure the anonymity of the subjects and the confidentiality of the data. If you are not using pooled data, indicate clearly how anonymity of subjects will be protected. Where subjects are interviewed, state whether the interviewees will be quoted and if so, how anonymity will be ensured. If interviewee(s) are not to remain anonymous, how will permission to quote be obtained?

Anonymity and confidentiality of the subject's data will be ensured through several methods. All data collected will be stored in numerical form, with a specific code separating one set of subject's data from others. All data will be presented in a pooled form and individual subject names will not be used in any form of presentation. Use of the data will be restricted to the investigators. All data stored on diskettes will be locked in a cabinet and access to the data on computer will be restricted by a security access code. Data that has no potential for secondary analysis will be destroyed upon completion of the project.

9) Briefly outline what the subjects will be required to do. Indicate the number of sessions required per subject and the length of each session. Submit a copy of protocols, questionnaires or other relevant materials to be administered to subjects. Do not submit mechanical apparatus. - where scientific instruments are to be used which involve covert or overt physical contact (e.g. electrodes, sensory devices), provide a clear description of the apparatus and its function.

Please see attached documents for a complete description of the specific research trials. A description of the equipment to be used in certain portions of the experimental trial is identified below:

A rigid, sealed chamber in which the subject will be placed (sealed at hips and feet) will provide negative and positive pressure to the lower body. Pressures will be provided via a standard vacuum (for negative pressure) and compressed air (for positive pressure). A minimal amount of both negative and positive pressure will be applied (between –50mmHg and +50mmHg respectively) in order to elicit the appropriate vasomotor response. Again, it must be noted that such vasomotor responses are no different than those elicited during exercise and during recovery.

Heat flux from the various superficial regions will be continuously monitored during the experiment with 10 waterproofed heat flux transducers placed on the skin surface. In addition, skin temperature will be measured by thermistors, (~2-3 mm^2) positioned directly to the skin with sterile medical adhesive tape. Esophageal temperature is measured with a sterile, thin (~ 3 mm), smooth, flexible probe that is inserted through the nostril, to the esophagus and swallowed down to the level of the right atrium of the heart (equal to approximately one quarter of the subject's height from the nares).

Blood pressure is measured using a Finapress (model 2300) blood pressure monitor. This monitor measures arterial blood pressure in the finger using a noninvasive technique. A finger cuff containing photoelectric components for measuring a blood plethysmograph and a bladder for applying pressure to the finger, is wrapped around the patient's finger and connected to the Patient Interface Module. Blood flow is measured with a sensor applied to the skin that emits laser pulses, which bounce off red blood cells, and cutaneous blood flow velocity. Oxygen consumption is measured by an automated metabolic cart (Medgraphics). The subject is required to wear a small mouthpiece (pneumotach) from which a sample line will run to the metabolic cart for breath to breath analysis of expired air.

I agree to abide by the guidelines, ethical principles and code of ethics adopted by the UHREC and its subcommittees, and where applicable, by those of the granting agency to which this proposal is being submitted, and by those of my profession or discipline as well as those of the facility or institution in which the research is undertaken. I am aware of my personal responsibility to be familiar with those standards. I further agree to notify the UHREC and the Human Research Ethics Committee of the Faculty of Health Sciences of any substantive changes in the use of human subjects in this research and to comply with the requests by UHREC or its subcommittee for other information/documentation during the life of this research.

SIGNATURE

	*DAY*_____ *MONTH* _____ *YEAR*_____
	*DAY*_____ *MONTH* _____ *YEAR*_____

INFORMATION SHEET

Baroreceptor influence on post-exercise warm thermoregulatory response thresholds

Investigator(s):
Dwayne Jackson (B.Sc.)
Glen K. Kenny (Ph.D.)
University of Ottawa,
School of Human Kinetics
562-5800 ext. 4282 or 4244

Faculty of Health Sciences
Research Ethics Committee Chair:
Dr. R. Proulx
Faculty of Health Sciences
Human Research Ethics Committee
Tel. 562-5800 ext.4251

The purpose of this research project is to investigate mechanism responsible for the post-exercise increase of warm thermoregulatory responses. We previously demonstrated that following dynamic exercise, there is an increase in both the warm and cold thermal responses. This increase may have an important impact in understanding how exercise changes the body's ability to dissipate heat. The present study will improve our understanding of the physiological mechanism responsible for the post-exercise changes in skin blood flow control and sweating response.

As a subject, you will be asked to participate in one preliminary session, and four experimental sessions to be conducted on separate days at a minimum interval of 48 hours. The preliminary sessions involve your participation for approximately one-hour on each of two occasions. During this time, the procedures will be reviewed and the experimental equipment (i.e., skin and esophageal temperature probes, blood flow monitor, heart rate monitor, etc.) to be used in the experiment will be described to you. You will then be asked to complete a Physical Activity Readiness Questionnaire (Par-Q form). Following the orientation period, you will be asked to perform an incremental maximal aerobic power test on a cycle ergometer. You will ride at a cadence of 60 rpm while the resistance will be increased 0.5 kp every two minutes until such time as you can no longer maintain the required load. These data will be used to select the workload for the submaximal exercise during two of the experimental trials.

The risks and discomforts of such tests include nausea, dizziness and sometimes fainting. The incidence of cardiac arrest during maximal exercise testing is normally less than 1 in 10,000 tests. The "Guidelines for Graded Exercise Testing and Exercise Prescription" (American College of Sports Medicine) indicate that maximum exercise testing is not contraindicated for men under 40 yr. of age and women under 50 yr. of age, with no symptoms or risk factors for cardiovascular disease. Nonetheless these tests will be terminated at any time if you so indicate.

You will perform a total of 4 experimental trials that will be carried out in a random order in the morning between 7:00 am and 8:00 am, after a 24 h period without heavy exercise, abstinence from stimulants and alcohol, 8-h of sleep, and a minimum ingestion of 0.25 liters of water per waking hour. To avoid thermal stimuli caused by extraneous metabolic factors, you are asked to fast at least 4 h prior to experimentation; however, water ingestion is encouraged during this time. Further, you are asked to avoid excessive walking or movement in the period between awakening and experimentation.

Upon arrival to the laboratory you will be clothed in shorts and athletic shoes and instrumented appropriately. You will then be fitted with an upper body water-perfused suit (covering the torso, arms and head) and placed in the pressure chamber, in a semi-seated posture, sealed at the iliac crest. Instrumentation will include a number of skin probes applied to various anatomical locations on the body. These probes will be taped to the skin with hypoallergenic tape. They provide an indication of skin temperature and surface heat loss. Some hair may need to be shaved in order to secure the probes adequately to the skin surface. Also there may be some discomfort upon removing the tape.

In order to monitor the core or central body temperature, an esophageal temperature probe will be inserted through the nostril to a depth so that the tip of this probe lies in the esophagus at the level of the heart. There may be some mild discomfort and mild reflex gagging from the swallowing of this probe. This sensation soon passes however. This probe is much smaller than the probe usually used to record esophageal temperature in conscious and unconscious patients.

Blood pressure will be measured using a Finapres (model 2300) blood pressure monitor. This measure of arterial blood pressure is a non-invasive technique. A finger cuff containing a blood plethysmograph and a small pressure bladder will be applied to the finger and connected to the Patient Interface Module.

A flexible laser probe will be taped to the mid-forearm and will remain there throughout the experiment in order to estimate forearm skin blood flow. This application of this measurement device is completely non-invasive and thus does not result in any risk or discomfort. In order to avoid movement artifacts on both blood flow and blood pressure readings, a cushioned harness will support your arm.

Sweat rate will be measured using a ventilated capsule (≈5.0 x 3.5 cm) placed on your upper back. Anhydrous compressed air will be passed through the capsule over the skin surface at a rate of 1 $L \cdot min^{-1}$. There is no discomfort associated with this technique.

Each of the four experimental trials will commence with a 15 min baseline-resting period after which you will either exercise (Ex) or remain resting (NoEx). For the exercise treatment you will perform 15 min of cycle ergometer exercise at 70% of your predetermined VO_2max. For the No-Exercise treatment you will be instructed to rest in the semi-seated upright position for 15 min.

Immediately following both the No-Exercise and Exercise treatments, you will either remain or be placed in the upright semi-seated position within a pressure box sealed at the level of the iliac crest. At such time you will receive an application of either 50 mmHg lower body positive pressure (LBPP) or no-lower body positive pressure. Simultaneously, cool water (~20 °C) will be circulated through the water-perfused suit until forearm cutaneous vasoconstriction is noted. The water circulating through the suit

will then be progressively increased to 47 °C until cutaneous vasodilation and sweating are noted.

Application of lower body positive pressure (LBPP) produces an increase in venous return. The effect is comparable to extended head-down bed-rest. The application of LBPP will only induce a mild hypertension for a short period of time (until the body reestablishes homeostasis).

You should be aware that there are some minor physical risks associated with any form of exercise. This study involves performing treadmill-running exercise. There are essentially no risks for young, healthy, active people while performing the submaximal intensities. When performing maximal intensity exercise, there is a very minor possibility of cardiovascular dysfunction. However, no such incident has occurred in this laboratory during almost 30 years of operation. No risk of infection is present with the use of esophageal probes as each subject has his/her own sterile probe that is disposed of once you have completed all tests. All tests will be conducted under standardized conditions for human exercise experiments as laid out by the Canadian Society for Exercise Physiology and the American College of Sports Medicine. Every effort will be made to ensure that these tests are conducted so as to minimize any of these sources of discomfort.

Anonymity is ensured to the extent possible, in that all data will be stored in computer memory under specific alphanumeric access codes known only to the researchers and presented only as pooled group data. Access to the data is thus restricted to the investigators. You are encouraged to request and discuss the results at any time.

For the duration of the project you may refuse to participate and/or withdraw your consent at any time, for any reason, without consequence prejudice to you. If you have any further questions regarding the nature or protocol of this study, please feel free to contact me, Dr. Glen Kenny at 562-5800 ext. 4282.

Once you have read this information form, please feel free to ask any questions. In order that we can confirm that you understand and have read all of the above information, please sign below. (Remember, you have the right to ask questions and stop the test at any time).

Subject:_____ Date:_____

Witness:_____

CONSENT FORM

Baroreceptor influence on post-exercise warm thermoregulatory response thresholds

Dwayne Jackson (B.Sc.),
Glen K. Kenny (Ph.D.),
University of Ottawa,
School of Human Kinetics
562-5800 ext. 4282 or 4244

I understand that the purpose of this research project is to investigate mechanism responsible for the post-exercise increase in the resting threshold for cutaneous vasodilation and sweating. We previously demonstrated that following dynamic exercise, there is an increase in the vasodilation threshold (and vasoconstriction). This increase may have an important impact in understand how exercise changes the body's ability to dissipate heat.

I have read and understood the information presented in the information sheet and I understand the risks involved with both the maximal and experimental exercise protocols and the instrumentation that will be used in the testing procedure. I have filled out a "Par Q and You: Physical Activity Readiness Questionnaire. I understand that I will be asked to participate in one preliminary test during which time I will be required to exercise maximally.

I understand that I will be required to participate in four experimental sessions, two of which will involve cycle ergometer exercise at 70% of my previously established maximal aerobic power. The experimental trials will be randomly assigned and will be as follows: 1) Ex/LBPP, 2) Ex/NoLBPP, 3) NoEx/LBPP, 4) NoEx/NoLBPP carried out at ~24° C in thermoneutral conditions. During these tests, I will be connected to skin, and esophageal probes, a Laser-Doppler flowmeter, a ventilated capsule, a portable blood pressure monitor and an automated metabolic cart.

I understand that there are minor risks and discomforts associated with these procedures due to the nature and intensity of exercise and the minor irritations associated with the inserted esophageal and temperature probes.

I understand that the data is strictly confidential and will be maintained secure at all times. Access to the data will be restricted to authorize personnel. The raw data will be identified by specific codes and scrubbed of all personal identifiers. In any written reports or publications, I will not be identified as only pooled, group data will be presented.

I will receive no direct benefit from having participated in this study. However, the investigators may learn more about temperature regulation during and following exercise in humans.

I have consulted with the principle investigators and my questions have been answered. If I have any other questions I may call them at (613) 562-5800 ext. 4282 or ext. 4244

I have been given a copy of this consent form and the information sheet. I realize that my participation in this research is voluntary. **I may decline to participate in any part of the study and may withdraw at any time without prejudice or discrimination of any form.**

Volunteering Subject:_____ Date:_____

Signature of Witness:_____ Date:_____

Signature of Researcher:_____ Date:_____

APPENDIX C

Pressure Chamber Specifications

Construction of the upright, lower body positive pressure (LBPP) and lower body negative pressure (LBNP) chamber.

The structural integrity of the lower body positive and negative pressure chamber was accomplished through a frame constructed of 1.5" x 1.5" angled aircraft aluminum. The enclosure for the pressure chamber was made entirely of 0.5" transparent acrylic plastic, except for the floor and backside, where 5/8" laminated pressboard was used. Two braces, bolted to each surface, accomplish reinforcement for the acrylic, where the frame does not offer support. Stainless steel bolts (1/4" bolts and acorn nuts with 4" spacings) were used throughout the chamber's assembly.

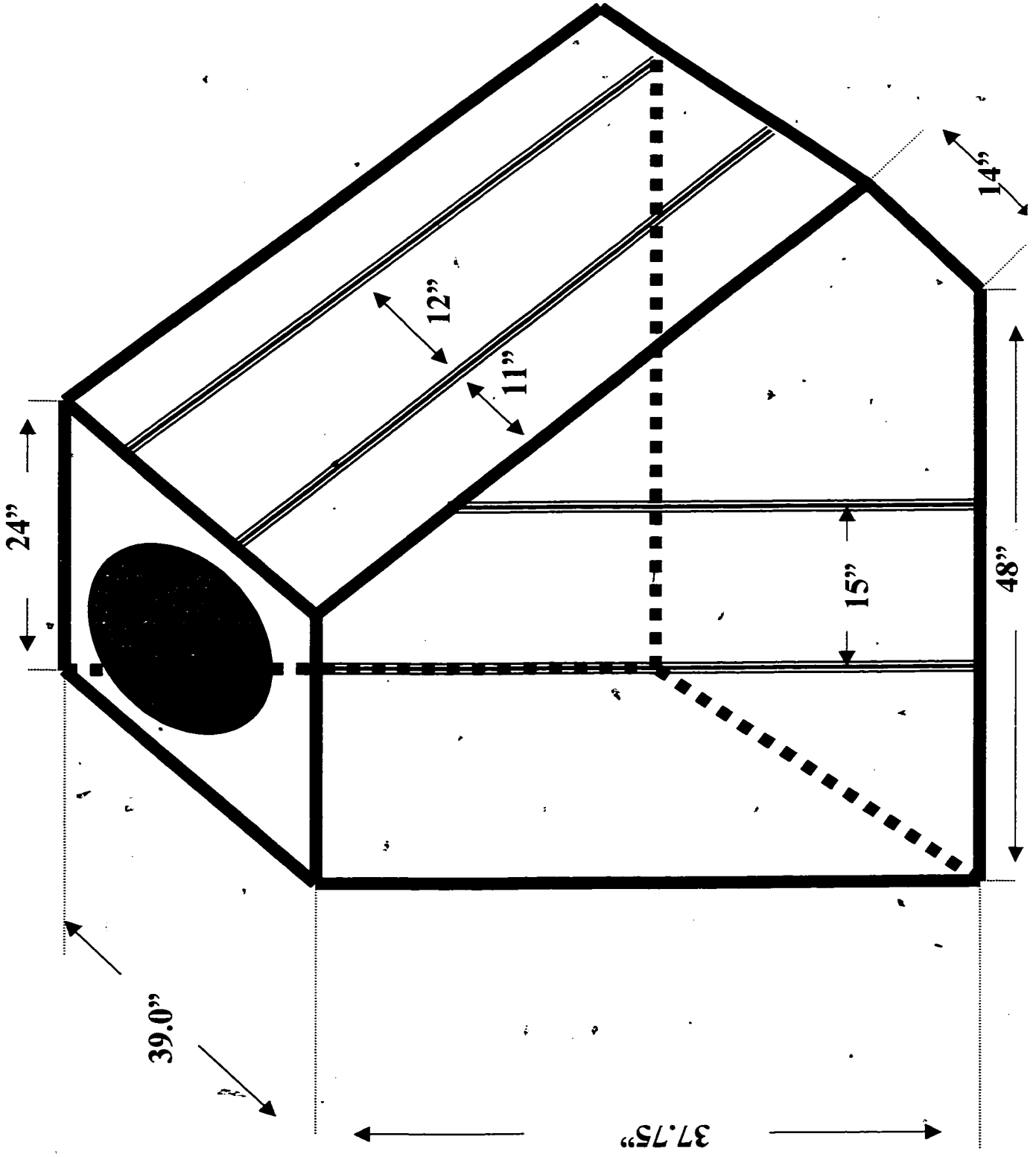
The pressure is applied via an industrial vacuum/blower system (Ridgid, WD1200, 4.5 Hp). The precise control of the internal pressure level is accomplished through a rheostat (Leviton mfg, 1000W). This mechanism modifies voltage output to the motor on the vacuum/blower. Since the current draw, by the vacuum, is large and over extended periods of experimental time, a heat sink was incorporated into the rheostat to avoid over heating. Within the pressure chamber, a baffle system ensures that, during pressure application, the rush or draw of air in or out will produce minimal or no air current. Instantaneous monitoring of internal chamber pressure is accomplished by a calibrated mercury monometer (LBPP) and a calibrated mechanical diaphragm style monometer (LBNP).

Placement of subjects within the chamber is controlled by an adjustable, semi-seated positioning system. When properly set up, intra-subject posture remains virtually identical throughout experimental trials. Grip tape covers the chamber's floor, to ensure that the subject's feet do not slip during experimentation. In order to seal the subject into

the chamber, a neoprene sealing system was developed. After instrumentation, the subject is placed into the specially developed neoprene skirt (which is mounted to the chamber's lid) and is subsequently placed into the chamber. The lid is fastened to the chamber with 15 quick release latches. These fasteners ensure rapid placement and removal of the subject during experimental phases. The design of the neoprene sealing system and chamber will accommodate subjects of varying standing heights, weights, and body types.

Specifcations

- Overall Height: 37.75"
- Overall Width: 39"
- Overall Length: 48"
- Maximum LBNP: -70 mmHg
- Maximum LBPP: +70 mmHg
- Frame: 1.5" X 1.5" angled aluminum
- Enclosure: 0.5" acrylic plastic (transparent) and 5/8" laminated pressboard (back and floor)


14"
12"
11"
24"
15"
48"
39.0"
37.75"