

INFORMATION TO USERS

This manuscript has been reproduced from the microfilm master. UMI films the text directly from the original or copy submitted. Thus, some thesis and dissertation copies are in typewriter face, while others may be from any type of computer printer.

The quality of this reproduction is dependent upon the quality of the copy submitted. Broken or indistinct print, colored or poor quality illustrations and photographs, print bleedthrough, substandard margins, and improper alignment can adversely affect reproduction.

In the unlikely event that the author did not send UMI a complete manuscript and there are missing pages, these will be noted. Also, if unauthorized copyright material had to be removed, a note will indicate the deletion.

Oversize materials (e.g., maps, drawings, charts) are reproduced by sectioning the original, beginning at the upper left-hand corner and continuing from left to right in equal sections with small overlaps.

Photographs included in the original manuscript have been reproduced xerographically in this copy. Higher quality 6" x 9" black and white photographic prints are available for any photographs or illustrations appearing in this copy for an additional charge. Contact UMI directly to order.

Bell & Howell Information and Learning
300 North Zeeb Road, Ann Arbor, MI 48106-1346 USA
800-521-0600

UMI[®]



Université d'Ottawa • University of Ottawa

**Exercise Intensity and the
Post Exercise Elevation in Esophageal Temperature**

By
Peter C. Niedre
B.Sc., B.Ed., University of Ottawa

Thesis Submitted to the
Faculty of Graduate and Postdoctoral Studies
In Partial Fulfillment of the Degree of
Master of Arts in Human Kinetics

School of Human Kinetics
University of Ottawa
Ottawa, Canada
May, 2000



National Library
of Canada

Acquisitions and
Bibliographic Services

395 Wellington Street
Ottawa ON K1A 0N4
Canada

Bibliothèque nationale
du Canada

Acquisitions et
services bibliographiques

395, rue Wellington
Ottawa ON K1A 0N4
Canada

Your file Votre référence

Our file Notre référence

The author has granted a non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of this thesis in microform, paper or electronic formats.

The author retains ownership of the copyright in this thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without the author's permission.

L'auteur a accordé une licence non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de cette thèse sous la forme de microfiche/film, de reproduction sur papier ou sur format électronique.

L'auteur conserve la propriété du droit d'auteur qui protège cette thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

0-612-57149-1

Canada

ABSTRACT

In order to examine the effects of exercise intensity on the post exercise elevation in esophageal temperature (ΔT_{es}), 8 male subjects performed 15 minutes of treadmill running exercise at 3 different exercise intensities, subthreshold (70% VO_{2max}), threshold (84% VO_{2max}), and suprathreshold (93% VO_{2max}), followed by an upright 45 minute recovery period. In addition, the effects of exercise intensity on the internal temperature threshold for cutaneous vasodilation (T_{dil}), as well as post exercise blood pressure, cutaneous blood flow (SkBF) and cardiovascular conductance (ΔCVC), were examined. Significant differences ($p < 0.05$) in ΔT_{es} were found between the 3 exercise intensities during exercise, as well as between suprathreshold (0.91°C), and threshold (0.56°C)/subthreshold (0.44°C) intensities during the recovery period. The elevated ΔT_{es} remained significantly above preexercise values for the entire recovery period for all 3 exercise intensities. Exercise intensity also demonstrated an effect on T_{dil} during exercise where significant differences were found between the 3 exercise intensities (0.26°C, 0.47°C, 0.84°C for subthreshold, threshold, and suprathreshold intensities respectively). However, no significant correlation was found between T_{dil} , and post exercise ΔT_{es} . Finally, a state of hypotension with respect to pre-exercise was observed during recovery from suprathreshold exercise. This could explain the significantly higher post exercise elevation in ΔT_{es} from subthreshold and threshold exercise.

Acknowledgements

**Dr. James Thoden
Dr. Gord Robertson
Dr. Glen Kenny
Dr. Frank Reardon
Chris Scott
Chris McClure
Alan Farley
Eric Vermander
Sheldon Hancock
Ashley McColgan
Brad Young
Paul Johnson
John Niedre
Ann Niedre
Mark Niedre
Kelly Maloney**

TABLE OF CONTENTS

CHAPTER 1: INTRODUCTION	2
1.1: Purpose:.....	6
1.2: Objectives:.....	7
1.3: Hypothesis:.....	7
CHAPTER 2: REVIEW OF LITERATURE	9
2.1: Heat Dissipation and Heat Exchange Pathways.....	9
2.2: Thermoregulatory Theories.....	10
2.3: Effects of Blood Pressure & Blood Flow Control on Thermoregulation	13
2.4: Post-Exercise Thermal Homeostasis-Hyperthermia	31
CHAPTER 3: METHODOLOGY:	34
3.1: Subjects:.....	34
3.2: Instrumentation:.....	34
3.3: Protocol:.....	35
3.4: Data Analysis:.....	38
CHAPTER 4: RESULTS	43
4.1: Esophageal Temperature.....	43
4.2: Internal Temperature Threshold for Cutaneous Vasodilation (T_{dil})	50
4.3: T_{dil} and Post-Exercise Elevated Plateau in ΔT_{es} Correlation	51
4.4: Weighted Mean Skin Temperature (ΔT_{sk})	52
4.5: Heart Rate and Oxygen Consumption.....	53
4.6: Blood Pressure (SBP, DBP): Mean Arterial Pressure (MAP).....	55
4.7: Skin Blood Flow and Cardiovascular Conductance.....	60
FIGURES AND GRAPHS	62
CHAPTER 5: DISCUSSION	74
5.1: Exercise Intensity and the Post Exercise Elevation in ΔT_{es}	75
5.2: Exercise Intensity and the Internal Temperature for Cutaneous Vasodilation.....	84
5.3: Correlation Between T_{dil} and Post-Exercise T_{es}	89
5.4 Mechanisms Behind the Post Exercise Elevation in T_{es}	92
CHAPTER 6: CONCLUSION	94
APPENDICES	97
REFERENCES	101

Chapter 1: Introduction

A competition exists for blood flow between the active skeletal muscles to supply both their increased metabolic demand and the cutaneous vasculature required to dissipate the increased metabolic heat produced during dynamic exercise (Johnson, 1986, 1992; Kenney and Johnson, 1991). A number of studies have investigated the effects of dynamic exercise on core temperature, blood distribution and flow at the onset, during and following exercise. Few studies, however, have examined the direct relationship between nonthermoregulatory and thermoregulatory control mechanisms of blood distribution during recovery from various dynamic exercise intensities.

Body core temperature (rectal) at rest is maintained in a very narrow range between 36.7 and 37°C (Guyton, 1991). Researchers (Sawka and Wenger, 1988; Gisolfi and Wenger, 1984) have outlined the concept of a thermoregulatory hypothalamic temperature 'set point' (SP_{hyp}) to which the body attempts to adjust temperature by controlling heat production and dissipation mechanisms. An exercise-induced increase in core temperature above SP_{hyp} is interpreted by the body as a 'load error' (Tam et al., 1978; Sawka and Wenger, 1988). Mekjavic et al. (1991) suggested the idea that the regulation of T_c occurs within a neutral temperature or null zone rather than at a set point. Within this 'null zone' (a range of approximately 0.59°C) neither heat production nor dissipation mechanisms occur.

Dynamic exercise initiates vasomotor reflexes that have the effect of redistributing blood flow from inactive tissues to working muscle. At the onset of exercise there is a large redistribution of blood from the cutaneous vasculature of the body as a competition

is induced between the active vasoconstrictor and vasodilator systems (Kellogg et al., 1991a & b).

At the initiation of upright exercise, a decrease occurs in acral and many nonacral skin temperatures, indicative of an exercise-induced reflex vasoconstriction (Thoden et al., 1994). Although several other investigators have observed an initial exercise-induced vasoconstriction at the onset of supine or upright exercise on a bicycle ergometer, (Kellogg et al., 1991a; Nishiyasu et al., 1993; Mack et al., 1995; Rowell and O'Leary, 1990; Kenney and Johnson, 1992; Roberts and Wenger, 1980; Pergola et al., 1993 & 1994), initial vasoconstriction may not be complete enough to lead to a complete drop in skin blood flow. Observations from pilot work in this laboratory using laser Doppler flow have shown an initial increase in skin blood flow at the onset of running exercise, likely as a function of the exercise-driven increase in blood pressure.

A few minutes after the initial reflex vasoconstriction, both core temperature and skin blood flow begin to increase eventually exceeding preexercise levels (Johnson, 1992). Thoden et al. (1994) found that cutaneous vasodilation occurs abruptly approximately 6 -7 minutes after the onset of exercise during moderate workloads (~75% of the subjects maximal aerobic consumption).

The thermoregulatory drive from increasing core temperature for heat dissipation varies with metabolic heat production and thus exercise intensity, which causes a subsequent upward shift from resting values of the internal temperature threshold for active cutaneous vasodilation (T_{dil}) (Taylor et al., 1988; 1990; Mack et al., 1991). A question, however, arises with respect to the mechanisms behind an increase in T_{dil} . In this response, is T_{dil} raised, or is T_{dil} interfered with due to an exercise-induced increase in

blood pressure, thus increasing the thermal drive or temperature change necessary to result in a net cutaneous vasodilation from combined reflex vasoconstriction and vasodilation influences. Observations from this study may provide insight as to whether T_{dil} is raised or if the upward shift is caused by an increased competition between exercise-induced vasoconstriction and thermoregulatory-drive for vasodilation.

During supine recovery from an acute bout of maximal exercise, a hypotensive effect was observed as a rapid drop in diastolic pressure. In addition, persisting peripheral vasodilation occurs despite elevated levels of catecholamines and renin (Piepoli et al., 1993). This group postulated that local vasodilator stimuli overcome sympathetic activity and that the increased sympathetic activity may be associated with the decrease in total vascular resistance. However, since the subjects assumed a supine position immediately following the upright exercise the increased vasoconstrictor tone from an upright posture at rest (Johnson and Park, 1981) was perhaps abolished as the subjects assumed a supine position.

Dynamic exercise intensity also has an effect on the post-exercise cardiovascular responses to exercise (Piepoli et al., 1994). During upright recovery from minimal and moderate upright bicycle ergometer exercise, forearm vascular resistance is elevated, and total peripheral resistance and diastolic pressure do not change during 60 minutes of recovery with respect to pre-exercise values. A persisting cutaneous vasodilation was also observed following maximal exercise, along with a lower diastolic pressure with respect to pre-exercise levels. The investigators speculated that vasodilation in exercising and non-exercising limbs following maximal exercise was responsible for the observed fall in blood

pressure, and that this overcomes, at least in part, sympathetic stimulus for vasoconstriction.

In all of the preceding studies, neither the presence of vasodilator or sympathetic substances, nor the influence of body core temperature was investigated. Furthermore, a thermoregulatory drive to dissipate the increased production of metabolic heat during maximal exercise may play some role in the decreased total peripheral resistance.

A rapid drop in esophageal temperature (T_{es}), followed by a stable plateau which was established within 10 minutes, and remained for 65 minutes of recovery following moderate running exercise (75% VO_{2max}) was observed by Thoden et al., (1994). All nonacral skin temperatures, with the exception of the thigh, continued to rise for 2-3 minutes following cessation of exercise, but stabilized at or just above preexercise levels, 20-30 minutes post exercise. The stabilized skin temperature suggests that cutaneous vasodilation had ceased, or become attenuated. The stable elevated plateau in T_{es} was found to correspond with the esophageal temperature threshold for vasodilation.

These investigators also demonstrated that following moderate intensity exercise, the elevated plateau in T_{es} exists and corresponds to the T_{dil} . This relationship was also observed following 75% and 60% VO_{2max} exercise intensities (Kenny et al 1999), various ambient temperatures and intensities (Kenny et al, 1999), and various durations of exercise (Kenny et al, 1997).

Various studies have been performed examining the relationship between exercise intensity and the threshold for cutaneous dilation, and exercise intensity and post exercise hemodynamics. The effect of exercise intensity on post-exercise esophageal temperature at

exercise intensities higher than 75% VO_{2max} , and how this is related to the core temperature threshold for vasodilation has not yet been investigated. Furthermore, the correspondence between post exercise esophageal temperature and post-exercise hemodynamic parameters has also not been investigated. This study will expand its observations to a higher range of exercise intensities, and attempt to draw more of a relationship between nonthermoregulatory and thermoregulatory responses following exercise.

1.1: Purpose:

To investigate the possibility of post exercise blood pressure underlying the post-exercise elevation in esophageal temperature, this study examined the effects of three different exercise intensities on post-exercise esophageal temperature and its relationship to forearm blood flow and the core temperature threshold for cutaneous vasodilation. The possible mechanisms were assessed through measurements of esophageal (T_{es}) and skin (T_{sk}) temperatures, and the cardiopulmonary variables of heartrate (HR), forearm cutaneous blood flow (SkBF), systolic (SBP), diastolic (DBP) and mean arterial (MAP) blood pressures.

1.2: Objectives:

The specific objectives of this study were to investigate:

- i) the effects of three exercise intensities on the elevated post-exercise plateau in esophageal temperature
- ii) the effects of three exercise intensities on the internal temperature threshold for active cutaneous vasodilation (T_{dil}) to be elicited
- iii) the relationship between the post-exercise elevated plateau in T_{es} and T_{dil} for the three exercise intensities
- iv) the cardiovascular (HR, SkBF, CVC) and hemodynamic (SBP, DBP, MAP) responses during recovery to three exercise intensities and how these responses relate to the established T_{es} plateau

1.3: Hypothesis:

Since T_{dil} has shown to increase in direct relationship to an increase in dynamic exercise intensity above 120 W (Taylor et al., 1988; 1990), and the established post-exercise elevated plateau in T_{es} corresponds with T_{dil} (Thoden et al., 1994), it was hypothesized that a new, significantly different elevated plateau in T_{es} would be established for each exercise intensity. However, examination of pilot work during higher intensity exercise, and findings that cutaneous dilation is rarely observed at higher exercise intensities (Taylor et al, 1988), it was hypothesized that the established T_{es} plateau would not correspond to the internal temperature threshold for active cutaneous vasodilation at each exercise intensity that is studied (i.e., the apparent relationship between the T_{es} plateau will not remain intact at different exercise intensities).

1.4: Implications of the Study:

The main purpose behind this study was to gain a better understanding of the control of thermal homeostasis during and post-exercise as it is influenced by exercise intensity. Furthermore, the results from this study can provide further information on the mechanisms behind the phenomenon of post-exercise hyperthermia, which may allow researchers to gain more knowledge about the body's thermal defense mechanisms against an increase in core temperature.

1.5: Delimitations:

Results obtained from this study can not be generalized beyond a sample of male, physically active and competing athletes. In addition, these results may not be generalized towards women as hormonal changes associated with different phases of the menstrual cycle can cause variations in resting core temperature.

1.6: Limitations

The current study requests subjects to rest for 15 minutes before exercise, exercise at 3 different intensities (70%, 84%, and 93% of the subject's VO_{2max}) for a duration of 15 minutes, as well as recover for an additional 45 minutes. During this time, all surface and core probes as well as blood pressure and blood flow probes remain attached. This protocol may cause some subjects to become frustrated or lose their motivation and retire early from further participation in the experimental sessions.

Chapter 2: Review of Literature

2.1: Heat Dissipation and Heat Exchange Pathways

Heat production is one of the major byproducts of metabolism. Guyton (1991) describes the factors that affect the rate of heat production, or the metabolic rate of the body. The most important factors for this study are: 1) the basal metabolic rate of all the cells in the body, (2) an increase in rate of metabolism caused by muscular activity and (3) extra metabolism caused by an increase in temperature of the body cells themselves.

Two forms of heat dissipation from the skin exist. Sensible heat loss involves dissipation of heat via radiation, conduction and convection. Loss of heat via radiation is loss in the form of infrared heat rays radiating from the body. If the temperature of the body is greater than that of the surroundings, a greater amount of heat is radiated from the body than is radiated from the surroundings to the body. Conduction is a minor form of heat loss, which involves the loss of heat from the body via direct contact to other objects. The rate of heat loss to water via conduction, however, is significantly greater than that lost to other objects. Convection involves the removal of heat from the body by convection air currents across the skin of the body. The heat is conducted to the air and then carried away by convection currents. Small amounts of convection almost always occur around the heated body due to the tendency of the heated air adjacent to the skin to rise.

The loss of heat through the evaporation of sweat is classified as insensible heat loss. When the temperature of the surroundings is greater than that of the skin, thereby preventing the body from losing heat via radiation or conduction, the only means by which the body can lose heat is through sweat.

2.2: Thermoregulatory Theories

The average normal body's core temperature is maintained within a narrow temperature range between 36.7 - 37°C at rest (Guyton, 1991). The regulation of this narrow core temperature range is almost entirely regulated via nervous feedback mechanisms. Most of these nervous feedback mechanisms are regulated through temperature regulating centers within the hypothalamus. The preoptic area-anterior hypothalamus contains heat sensitive neurons which function as temperature sensors, and acts as the thermostatic body temperature control center. The posterior hypothalamus is mainly responsible for the integration of peripheral temperature receptors along with feedback from the anterior hypothalamus-preoptic area. It is here that the signals are integrated to stimulate the major heat-dissipating or heat-conserving mechanisms. When the hypothalamic temperature centers detect that the body's temperature is too high, three major reflex heat loss mechanisms occur: vasodilation, sweating and a decrease in metabolic heat production.

2.2.1: The Hypothalamic Set Point

Sawka and Wenger (1988) describe a concept of heat-dissipation control. The concept of 'load-error' describes the body's heat-dissipating responses to reestablish thermal balance after a sufficient rise in core temperature. Cabanac and Massonnet (1977) immersed subjects in 38°C water until sweating was induced and then quickly transferred the subject to 28°C water until a shivering response occurred. Their results demonstrated the core temperatures at which shivering (heat conservation) and sweating (heat dissipation) mechanisms occur. The body's sweating and shivering mechanisms tended to

coincide at a 'set point'. This increase in core temperature above the hypothalamic set point is interpreted by the body as a 'load error' as identified by Tam et al. (1978); Sawka and Wenger (1988). To compensate for this load error thermal defense mechanisms act to increase the rate of heat loss to attenuate the increase in core temperature.

2.2.2: Thermoregulatory 'Null Zone'

The possibility of the regulation of body core temperature within a neutral temperature zone instead of at a 'set point' was investigated by Mekjavic et. al (1991). Subject's performed bicycle exercise submersed to the chin in 28°C water and then recovered for 100 minutes. During the recovery period, body core temperature dropped such that the heat dissipation mechanism (sweating) ceased and the heat production mechanism (shivering) commenced. These investigators observed a significant difference (0.59°C) between the core temperature at which sweating ceases and where the shivering mechanism commences. These observations indicate that a narrow core temperature range exists in which neither sweating nor shivering mechanisms occur. This temperature range lead the investigators to suggest the idea of a core temperature 'null zone' and not a 'set point' at which neither sweating mechanisms nor shivering thermogenesis occur.

2.2.3: Body Heat Content Regulation

When referring to the regulation of thermal homeostasis in the body, most researchers suggest that the body regulates its temperature centrally through the pre-optic anterior hypothalamic area. The idea that the body regulates its mean heat content rather than regulating body temperature centrally has been identified by researchers (Ivanov,

1976; 1982; 1987; 1990) and hypothesized and reviewed by Webb (1995). Studies exist in which the body's thermal responses are best explained through the concept of total body heat regulation as opposed to local temperature regulation. Generally, these experiments demonstrated that thermal regulatory responses neither appeared nor disappeared until the total body heat content had changed or been restored, as opposed to responding to central or local site temperature changes (Veghte and Webb, 1961; Webb, 1973; Ereth et al., 1992; In: Webb, 1995).

The purpose of heat regulation, according to Webb (1995), is heat balance, which is achieved through continuous changes or processes in heat production, followed by slower changes in heat loss. The physiological effectors that control heat loss to match various levels of heat production are cutaneous vasoconstriction, vasodilation and sweating. Webb (1995) describes the methods of sensing the heat production or 'flow' and the presumed controller of heat loss.

The main method of sensing heat flow (in or out) is the transcutaneous temperature gradient at the surface of the body. Bazett (1951) proposed that these sensors might be responsible for the control of sweating and found evidence that they are located just below the skin surface, and at one or more deeper levels. Many studies (Nielson, 1969; Robinson and Somers, 1971; Webb, 1992; Ivanov et al., 1990; 1987) have provided evidence to support the concept of the transcutaneous temperature gradient that detects both magnitude and direction of heat flow.

As previously mentioned, the control of heat loss is hypothesized not to be controlled centrally within a 'null zone' or at a 'set point' by the hypothalamus (Webb, 1995). Heat loss is controlled to match heat production and control of the outflow of body

heat is control of a continuous process. The heat loss control theory supports the idea that if increased heat outflow is sensed and used as feedback to a heat loss controller, the increased body heat (load error) stays high and in proportion to the load (Webb, 1995).

2.3: Effects of Blood Pressure & Blood Flow Control on Thermoregulation

2.3.1: Control of Blood Pressure and Blood Flow: The Baroreflex:

When blood pressure at rest undergoes a transient increase or decrease due to gravitational or postural stresses, heat stress, or any other stress, a centrally mediated reflex called the baroreflex is elicited to bring blood pressure back to baseline levels. The reflex is initiated by stretch receptors called baroreceptors. Baroreceptors are nerve endings lying in the walls of the arteries and are stimulated when a deformation (a stretch) in the arterial wall occurs. These baroreceptors are located in the walls of almost every large artery of the thoracic and neck regions. Most of the baroreceptors are located in the walls of the carotid sinus and the wall of the aortic arch.

An increase in arterial blood pressure stretches the baroreceptors, causing them to increase their firing rate and transmit these signals into the central nervous system and into the tractus solitarius of the medulla. Secondary signals excite the vagal center and inhibit the vasoconstrictor center of the medulla. This integration in the medulla transmits efferent or feedback signals through the autonomic nervous system to cause a decrease in heart rate and strength of contraction, and a net vasodilation throughout the peripheral circulatory system. A low blood pressure has opposite effects as secondary signals inhibit the vagal center and stimulate the vasoconstrictor center of the medulla to reflexively increase blood pressure back to normal.

2.3.2: Control of Cutaneous Blood Flow at Rest

The control of skin blood flow at rest is generally confined to the influence of local skin and general body warming or cooling, gravitational stresses or postural changes, or any other factor that may cause a change in the hemodynamics of the available cardiac output. The cutaneous circulation is on the efferent limb of several nonthermoregulatory reflexes, including the baroreflex (Kellogg et al., 1990). Both thermoregulatory and non-thermoregulatory control of skin blood flow at rest has been investigated by Johnson (1986); Johnson (1992); Pergola et al. (1994); Pergola et al. (1993); Kellogg et al. (1990); and Wyss et al. (1974).

Kellogg et al. (1990) used local bretylium tosylate iontophoresis to investigate whether the cutaneous vasodilator system is under baroreflex control. Bretylium tosylate iontophoresis blocks or abolishes adrenergic vasoconstrictor control, which can therefore help to isolate any other mechanism of cutaneous vasculature control. Blood flow was measured at both treated and untreated sites as subjects in a water-perfused suit (to control skin temperature) went through periods of normothermia (34-35°C) and hyperthermia (38.0°C to 38.5°C). In addition, lower body negative pressure (LBNP) was applied in both hyperthermia and normothermia to determine if the cutaneous vasodilator system is under baroreceptor reflex control.

During LBNP in hyperthermia, a decrease in cutaneous vascular conductance was observed in the presence of cutaneous vasoconstrictor blockade. Baroreceptor unloading through LBNP directly reduced active vasodilator tone and thereby affected vasoconstriction. Cutaneous vascular conductance recorded at any treated site was exclusively due to the active vasodilator system. The investigators finding that the

cutaneous active vasodilator responds to baroreceptor unloading indicates that vasodilator systems can participate in baroreceptor reflexes.

2.3.2.1: Local Temperature and the Reflex Control of Cutaneous Blood Flow

Reflex control of skin blood flow is mediated through two sympathetic pathways; an adrenergic vasoconstrictor system and a vasodilator system of unknown neurotransmitter. Pergola et al. (1993) examined the role of adrenergic nerve function in the cutaneous vascular response to changes in local skin temperature in the forearm. This was examined using three protocols at rest: i) local bretylium tosylate blockade of vasoconstrictor control, ii) altering background adrenergic tone by changing whole body skin temperature, and, iii) blocking cutaneous nerves by proximal infiltration of local anesthesia. The results from this study suggested several possibilities for the local control of skin blood flow. One relevant to the purposes of this study is that the majority of the vasodilator response to local warming does not require an intact adrenergic system as cutaneous sympathetic nerve block or adrenergic blockage did not reduce or inhibit an increase in skin blood flow during local warming.

2.3.3: Control of Blood Flow During Exercise

2.3.3.1: Control of Cutaneous Blood Flow at the Onset of Exercise:

Control of skin blood flow at the onset of exercise has been examined by several investigators (Johnson, 1986, 1992; Kellogg et al., 1990, Kellogg et al., 1991; Mack et al., 1995; Rowell and O'Leary, 1990; Kenney and Johnson, 1992; Roberts and Wenger, 1980;

Pergola et al., 1993, 1994.). At the onset of exercise, there is a large redistribution of blood from the cutaneous vasculature of the body to the core as a competition is induced between the active vasoconstrictor and vasodilator systems (Kellogg et al., 1991). Kenney and Johnson (1992) state that it is clear that dynamic exercise initiates vasomotor reflexes that have the effect of redistributing blood flow from inactive tissues to the working muscles.

Many researchers (Johnson, 1986; Johnson and Park, 1982; Kenney and Johnson, 1992; Rowell and O'Leary, 1990) have documented the effect of the onset of exercise on skin blood flow. At the initiation of exercise, a large redistribution of blood occurs to provide additional blood flow to supply the increased metabolic demands of the exercising muscles. This reflex adaptation to exercise involves reduction in blood flow via vasoconstriction to inactive regions. The skin vasculature is involved in this large redistribution of blood flow at the onset and during exercise in both normothermic and hyperthermic ambient conditions. In contrast to an intact adrenergic constrictor system not being required during local warming at the initiation of exercise, the cutaneous vasoconstrictor reflex requires an intact adrenergic (α_1 receptors) constrictor system both in normothermic and hyperthermic conditions (Kenney and Johnson, 1992).

Kellogg et al. (1991a) investigated the possible mechanisms behind the cutaneous vasoconstriction at the onset of dynamic exercise. Through the use of local (skin area) iontophoresis using bretylium tosylate during exercise, these investigators attempted to determine if this reflex response was due to an exercise-induced increase in sympathetic vasoconstrictor activity, a decrease in active vasodilator activity or both. The investigators based these possibilities on the existence of dual efferent neural control of skin blood flow.

At the initiation of exercise in normothermia, cardiovascular conductance (CVC) (as calculated by laser-Doppler flow/mean arterial pressure) decreased at untreated skin sites and did not change at treated sites. During hyperthermia, they found no significant difference in CVC between treated and untreated sites. During this trial, all sites showed significant vasodilation as CVC increased at both sites. Based on these observations, the researchers concluded that the reflex cutaneous vasoconstrictor response to the initiation of exercise is mediated solely by enhancement of active sympathetic vasoconstrictor tone. Initiation does not attenuate active cutaneous vasodilation. They speculated that this increased active vasoconstrictor tone is the effector of the cutaneous vasoconstriction induced by the initiation of exercise. During heat stress, active vasodilation of cutaneous arterioles compete with the exercise induced increases in active vasoconstriction.

2.3.3.2: Exercise Intensity and the Reflex Vasoconstriction:

Taylor et al. (1990) examined the effects of absolute and relative exercise intensity on the initial skin vasoconstrictor response to dynamic exercise. These investigators had subjects perform various modes (isolating large and small muscle groups) of supine dynamic exercise at a high (80W, due to the mode of exercise, this was the maximum intensity at which a subject could maintain exercise for 6-10 minutes) and low (40W) intensity. Due to the increased vasoconstrictor response (as determined by cutaneous vascular conductance) to the initiation of exercise at the higher intensity, the authors concluded that the degree of vasoconstriction is dependent on absolute exercise intensity. Johnson (1986); Taylor et al. (1984) also found that higher levels of dynamic exercise by

large muscle groups (2 legs) are associated with a greater reflex cutaneous vasoconstriction at the onset of exercise.

2.3.3.3: Exercise and Baroreceptor Control: Central Command

As previously mentioned, the baroreceptors react to an increase in blood pressure by increasing their firing rate to signal to the medulla to inhibit the vasoconstrictor center and stimulate the vagal center to lower blood pressure to baseline levels. An increase in systolic pressure accompanies the onset of exercise due to the contracting muscles and drive to increase stroke volume and heart rate. If the baroreflex was to react to this increase in blood pressure to reduce it, the drive to increase cardiac output and blood flow to the exercising muscles would be hindered.

The rapid rise in blood pressure and increase in heart rate at the initiation of exercise could lead one to conclude that the arterial baroreflex is inactivated during dynamic exercise so that the rise in blood pressure and heart rate are unopposed. Several investigators have examined the function of the baroreflex at the onset and during exercise by either loading (lower body positive pressure or neck pressure) or unloading (LBNP or neck suction) the aortic or carotid baroreceptors (Papelier et al., 1994; Potts et al., 1995a; and 1995b). The concept behind the baroreceptor 'operating or set point' relates to the threshold blood pressure at which the baroreflex occurs. The operating or set point of this reflex during exercise is reset to a higher operating level or point to compensate for the exercise-driven increase in heart rate and blood pressure (Rowell, 1991; 1992).

The concept of central command is outlined by Rowell (1992). Central command is the term for motor command signals originating from subthalamic neurons involved in

locomotion. These signals activate both cardiovascular and skeletal muscle motor systems together during exercise. At the initiation of exercise, the increase in heart rate, cardiac output and blood pressure appears to be mediated by central command by rapid inhibition of vagal tone. The magnitude of central command mediation and the increase in blood pressure is proportional to the number of motor units recruited during muscle contraction. It has been hypothesized that this rapid upward shift of the baroreflex is mediated by central command. Coordination exists between feedforward (central command) and feedback control (baroreflex) in which the effect of central command and the cardiovascular system is changed by adjusting the operating point of the arterial baroreflex.

Gain or sensitivity of the baroreflex is the degree to which the baroreflex acts to decrease or increase blood pressure to normal levels. The effects of exercise on the gain of the baroreflex has been examined by Papelier et al., 1994; Potts et al., 1995a; and 1995b. These investigators consistently found no difference in the sensitivity of the baroreflex between rest and exercise. Furthermore, exercise intensity has no significant effect on the sensitivity of the baroreflex.

Nishiyasu et al. (1993) tested the hypothesis that the forearm vascular resistance (FVR) at the onset of dynamic exercise is modulated by the unloading/activation of baroreflexes. Having subjects perform supine bicycle exercise with or without lower body negative pressure (LBNP), they also attempted to identify the interaction, if any, between reflexes accompanying muscular exercise and baroreflexes. At the onset of moderate exercise (100 W) without LBNP, a significant increase in forearm vascular resistance (FVR) and a subsequent decrease in forearm blood flow (FBF) within the first 2 minutes

of exercise were observed. This increased FVR and decreased FBF was ascribed to a generalized reflex vasoconstriction accompanying muscular exercise. In contrast, the application of -40 mmHg LBNP before the onset of moderate exercise caused a significant decrease in FVR, and a significant gradual increase in stroke volume and FBF within the first minute of exercise. The application of LBNP causes a reduction in the heart's preload before exercise commences, leading to an increase in stroke volume at the onset of exercise, consequently causing forearm vasodilation. This vasodilation is not a thermoregulatory response, but likely a response to the loading of the cardiopulmonary baroreceptors. These observations supported the concept that the FVR response at the onset of exercise is modulated by the influence of the cardiopulmonary baroreflex. The investigators also speculated that the increase FVR response in the first minute of moderate exercise suggests a positive interaction between the cardiopulmonary baroreflex and the generalized exercise-induced reflex.

2.3.4: Cutaneous Blood Flow During Exercise

As dynamic exercise continues, body core temperature (T_c) (as measured through esophageal (T_{es}) or rectal (T_{re}) temperatures) increases and the body must therefore act to dissipate the increase in metabolic heat production. During exercise, skin blood flow increases in proportion to T_c which allows for a transfer of heat from the body core to the skin.

Several investigators (Kellogg et al. 1991b, 1991c; Johnson, 1992; Kenney and Johnson, 1991) have documented that the internal core temperature threshold for cutaneous vasodilation (T_{dil}) is higher during exercise than at rest. Although T_c is rising

during exercise and exceeding the resting temperature threshold for cutaneous vasodilation (T_{dil}), an increase in skin blood flow is attenuated until a higher T_c is reached during exercise (Kenney and Johnson, 1991; Kellogg et al., 1991b). The effect of this upward shift or resetting of T_{dil} is that skin blood flow is lower at any given T_c during exercise than at rest.

Thoden et al. (1994) examined the response of core temperature to upright cycling ergometer exercise during the exercise bout and during recovery. These investigators found that cutaneous vasodilation (as assessed through a marked increase in middle finger skin temperature and an abrupt fall in the temperature gradient between the forearm and middle finger) began to rise abruptly approximately 6 to 7 minutes after the onset of exercise during moderate workloads (~75% of the subjects maximal aerobic consumption). Shortly after this abrupt increase in skin blood flow, the rate of increasing T_{re} begins to decrease as indicated graphically by a decrease in slope.

2.3.4.1: Mechanisms Behind an Increase in Cutaneous Vasodilation

Several investigators, (Mack et al., 1995; Kellogg et al., 1991; Roberts and Wenger, 1980; Pergola et al. 1994), have examined the mechanisms behind the increase in cutaneous vasodilation as T_c increases and a greater competition for available cardiac output arises. Kellogg et al. (1990) state that the active cutaneous vasodilator system is known to function as an efferent limb of thermoregulatory reflexes as well as participating in nonthermoregulatory reflexes such as blood pressure regulation. The competing thermoregulatory drive for increasing skin blood flow does not abolish muscle blood flow during exercise in the heat (Savard et al, 1988). This is likely caused by a redistribution of

blood away from vascular beds other than the skeletal muscle. Rowell (1983) speculates that the distribution of blood towards the cutaneous circulation could lead to a reduction in central blood volume and a decrease in cardiac filling pressure, and therefore stroke volume.

Kellogg et al. (1991b) examined the control of the internal temperature threshold for active cutaneous vasodilation by dynamic exercise. Using bretyllium tosylate iontophoresis to locally block vasoconstriction in two forearm sites, these investigators tried to determine if the shift in T_{dil} is accomplished through the vasoconstrictor system or the cutaneous active vasodilator system. Subjects performed 7 to 10 minutes of supine bicycle ergometer exercise as the T_{dil} was recorded for all skin sites.

These authors observed that the T_{dil} at bretyllium treated and untreated sites were not significantly different. These results lead to the finding that the upward resetting of the T_{dil} during exercise is entirely due to a delay in the onset of active cutaneous vasodilation in contrast to the active vasoconstrictor mediated reflex at the onset of exercise. The authors concluded that the active cutaneous vasodilator system is directly affected by reflex adjustments to dynamic exercise as it is by the baroreceptor reflex. These reflex adjustments increase the core temperature threshold to activate the cutaneous active vasodilator system and therefore delay competition from the cutaneous circulation for blood flow.

Franke et al. (1993) examined the effects of α_1 -mediated vasoconstrictor blockade through the use of prazosin on cardiovascular and thermoregulatory responses to upright heavy cycling exercise in the heat. These investigators had subjects exercise for 30 minutes at 70% of their maximal oxygen consumption in an ambient temperature of 35 C while

under the influence of either 1 mg of the α_1 -receptor blocker prazosin, or a placebo (control). This study found that the α_1 -adrenergic blockade did not affect forearm blood flow, forearm vascular conductance, rectal or skin temperature responses to heavy exercise, and does not improve the rate of heat loss in a hyperthermic environment. These investigators suggested that some mechanism other than α_1 -mediated vasoconstriction are important in the thermoregulatory responses to exercise. They further speculated that the active cutaneous vasodilator system and its control systems (of unknown neurotransmitter) are more likely important in the increase in forearm blood flow during upright exercise.

Mack et al. (1995) investigated the influence of baroreceptor unloading via -40 mmHg LBNP on cutaneous vasodilation and core temperature. These investigators found that baroreceptor unloading increased the esophageal temperature threshold for vasodilation and attenuated thermoregulatory control of skin blood flow. Baroreceptor unloading limited cutaneous vasodilation during dynamic exercise and was immediately reversed following removal of the orthostatic challenge. The researchers speculated that this limitation in cutaneous blood flow indicates a generalized modulation of thermoregulatory reflexes by baroreceptors. The observed responses further support the hypothesis that the interactions between baroreflex control of arterial blood pressure and thermoregulatory reflexes are neurally and centrally mediated; and, this interaction likely occurs somewhere within the central nervous system.

2.3.4.2: Control of Active Cutaneous Vasodilation by Local Skin Temperature

In addition to nonthermoregulatory factors associated with dynamic exercise, cutaneous blood flow is mediated by local thermoregulatory factors such as skin temperatures. Efferent control of cutaneous blood flow is accomplished through 2 sympathetically mediated pathways: an adrenergic and a separate vasodilator system, (Pergola et al., 1994).

To assess the control of reflex skin blood flow (SkBF) by skin temperature (T_{sk}), Pergola et al. (1994) had subjects rest and perform upright cycle ergometer exercise (at an intensity high enough to increase T_c and elicit cutaneous vasodilation) at various skin temperatures; normothermic (30-32° C) and hyperthermic (36-37°C). Exercise commenced at skin temperatures of 30-32°C, followed by an increase to 36-37°C after a decline in the rate of increase in T_{es} . During the normothermic portion of exercise, immediately following the initial reflexive vasoconstriction, T_{sk} begins to rise, which indicates the body is attempting to dissipate heat. No significant difference in CVC was found between the bretylium treated and untreated skin sites. After the increase in skin temperature to a hyperthermic condition, a transient reflex increase in CVC occurred at both control and treated sites, significantly greater than that observed at rest. There was no significant difference found between control and bretylium-treated sites. These observations lead to the conclusion that local or general skin temperatures can play a role in the reflex control of skin blood flow by modulating both cutaneous vasoconstrictor and active vasodilator function. They also concluded that this level of cutaneous vasodilator

activity results from the reflex integration of afferent signals from T_{sk} , T_c , and possibly exercise-induced reflexes.

2.3.5: Effects of Exercise Intensity on Core Temperature Threshold for Dilation

A minimum exercise intensity for the upward resetting of the core temperature threshold for vasodilation has been identified by some researchers (Taylor et al., 1988; 1990; Kenney and Johnson, 1991). Taylor et al.(1988; 1990) identified an absolute power output or exercise intensity during supine bicycle ergometer exercise required to elicit an increase in the T_{dil} . These investigators found that no upward shift in T_{dil} was observed during intensities less than 100 W; however, beyond 100 to 120 W, the core temperature threshold for vasodilation rose or was further attenuated.

The effect of exercise intensity on the core temperature threshold for cutaneous vasodilation and a subsequent increase in active vasodilator activity has been examined by several investigators (Taylor et al., 1988; Kenney and Johnson, 1991; Mack et al., 1993). These investigators have found a characteristic increase in body core temperature with an increase in exercise intensity, thereby causing a subsequent increase in the body core temperature threshold for cutaneous vasodilation.

Taylor et al. (1988) examined the role of exercise intensity in the control of skin blood flow during exercise. The core temperature threshold for cutaneous vasodilation at 5 different exercise intensities was examined through analysis of the cutaneous vascular conductance-esophageal temperature relationship. The CVC threshold was defined as the level of internal temperature at which cutaneous vasodilation commences. Cutaneous

vascular conductance (CVC) was determined as Laser-Doppler flow divided by mean arterial pressure ($100 \times \text{LDF}/\text{MAP}$). Each subject performed supine (to reduce the effects of an increased sympathetic vasoconstriction activity to the skin and increases the T_{dil}) bicycle ergometry exercise was performed at five different intensities from 75 to 200 W.

These investigators found that at an absolute workload of less than 125 W, the internal temperature threshold for cutaneous vasodilation did not rise from resting. At absolute workloads greater than 125 W, the CVC threshold increased with the intensity of exercise and the effect of supine dynamic exercise on T_{dil} was graded with the level of exercise intensity. The highest level of work intensity (200W) resulted in exhaustion within 6.5-8 minutes, and the CVC threshold for vasodilation was never achieved in 3 out of the 4 subjects. At such high exercise intensity, it appears that the reflex vasoconstriction induced by exercise overcomes the thermoregulatory drive to dissipate heat through active vasodilation.

Mack et al. (1994) also investigated the effects of exercise intensity on active cutaneous vasodilation, and the increase in core temperature (T_{es}) required to elicit this response. The relationship between increases in forearm skin vascular conductance (FVC) and T_{es} was examined during indirect heating (legs in 44°C water), 30 minutes of supine bicycle exercise at 75 W (30% $\text{VO}_{2\text{max}}$), and 20 minutes of supine bicycle ergometer exercise at 149 W (60% $\text{VO}_{2\text{max}}$). The T_{dil} during indirect heating and 30% $\text{VO}_{2\text{max}}$ exercise occurred at the same core temperature. The internal temperature threshold for cutaneous vasodilation during 60% $\text{VO}_{2\text{max}}$ exercise was significantly higher than that at rest and during 30% $\text{VO}_{2\text{max}}$. The relationship between exercise intensity and T_{dil} was

non-linear, indicating that a minimum exercise intensity is required to elicit a delay in cutaneous vasodilation.

In both of these studies, supine instead of upright exercise was used to study the thermoregulatory responses to exercise. Supine exercise was used to prevent an increase in sympathetic vasoconstrictor activity at the onset of exercise and a further delay or increase in T_{dil} . Mack et al. (1994) used supine exercise to eliminate the possible confounding influence of changes in baroreceptor loading on sympathetic nervous system activity and skin blood flow at the onset of exercise.

Studies using upright bicycle ergometry exercise have been used to study the internal temperature threshold for cutaneous vasodilation and to compare this to supine exercise, (Johnson and Park, 1981; Roberts and Wenger, 1980). These investigators found a common trend in upright exercise compared to supine. Upright bicycle ergometer exercise consistently decreases the cutaneous blood flow at a given core temperature due to an increase in the internal temperature threshold for cutaneous vasodilation. Cutaneous vasculature is therefore relatively vasoconstricted during heat stress with either dynamic exercise or upright posture, and the greatest decrease in skin blood flow occurs when these two stresses are combined.

2.3.6: Post-Exercise Cardiovascular Responses to Exercise:

After an acute bout of dynamic exercise, an immediate fall in blood pressure occurs and persists sometimes for several hours. Cumming (1972) observed an increase in cardiac output and stroke volume in the first few minutes of recovery after supine submaximal and maximal exercise. Post exercise cardiovascular responses to exercise vary

according to the mode, intensity and ambient temperature at which exercise was performed. Several authors have examined these aspects and how they relate to post exercise hemodynamics (Kilgour et al., 1993, Piepoli et al., 1993; Piepoli et al., 1994).

Piepoli et al. (1993) studied the role of autonomic tone and hemodynamic activity associated with reduced blood pressure after dynamic maximal exercise. On the exercise day, subjects performed an upright maximal bicycle ergometer test with 25 W increments every 5 minutes, followed by lying down supine for 60 minutes of recovery. On the control day, subjects remained in a standing position for 30 minutes, and then laid down for 60 minutes of recovery. A consistent hypotensive effect was observed after exercise due to a persisting vasodilation. Systolic pressure dropped to preexercise values within five minutes post exercise and dropped significantly below preexercise levels at 45 minutes. Diastolic and mean pressure remained below control values during the entire hour of recovery and reached their lowest value five minutes into recovery. A decrease in total peripheral resistance and baroreflex sensitivity (evaluated by the slope of changes in blood pressure against heart rate induced by bolus injections of phenylephrine) was also observed and thought to be associated with sympathetic rather than vagal activation post exercise.

As previously mentioned, immediately following maximal exercise subjects in this study assumed a supine position from an upright one. These investigators observed a significant post-exercise peripheral vasodilation. Investigators who have studied the effects of posture on peripheral vasodilation (Johnson and Park, 1981; Roberts and Wenger, 1980), concluded that upright posture during exercise or at rest causes a significantly greater level of peripheral vasoconstriction than a supine position. Perhaps it is

questionable if these same recovery responses would be observed if the subject had remained in an upright posture.

In a more recent study, Piepoli et al. (1994) studied post-exercise cardiovascular and hemodynamic responses to maximum incremental upright bicycle ergometer exercise while remaining in an upright position. During recovery in an upright position from the incremental (to a maximum workload) exercise, systolic blood pressure remained elevated at 5 minutes post-exercise and fell quickly to preexercise levels thereafter. Diastolic blood pressure was significantly lower than preexercise by five minutes into recovery, and remained for the duration of the recovery. Heart rate remained significantly elevated above baseline values; and, total peripheral resistance and forearm vascular resistance dropped significantly below control values for the entire 60 minutes of recovery. From these results it is evident that a persisting vasodilation remains during recovery from upright exercise.

While studying the effects of thermal stress on cardiovascular response to upright bicycle ergometer exercise, Kilgour et al. (1993) observed differences between heat stressed and non-heat stressed exercise. The thermal stress during exercise caused a significant portion of central blood volume to shift towards the cutaneous vasculature as a significantly lower diastolic blood pressure was observed in heat stress over control. In the control group, TPR returned to preexercise values within five minutes. During recovery after heat stress, the increase in TPR was significantly lower than the control temperature as only approximately half of the pre-exercise values were reached by 15 minutes recovery. This indicates a marked cutaneous vasodilation following exercise in a heat stressed environment compared to a lower level of vasodilator activity in a control temperature.

2.3.6.1: Exercise Intensity and Post-Exercise Cardiovascular Responses

The intensity of dynamic exercise has been found to affect post-exercise cardiovascular responses to the exercise. Cleroux et al., (1992), found that peripheral vasodilation in non-exercising limbs during recovery from dynamic exercise is dependent on the exercise intensity. These investigators observed peripheral vasodilation during the recovery period after greater than 50% maximal exercise; and a peripheral vasoconstriction was observed following less than 50% maximal exercise.

The effects of three levels of dynamic exercise intensity on post-exercise regional skin blood flow and peripheral vascular resistance were studied by Piepoli et al. (1994). Subjects performed upright bicycle ergometer exercise at minimal, moderate and maximal intensities and recovered in an upright posture for 60 minutes. A nonexercising arm was isolated using an armrest to refrain the arm from any movement. During recovery from maximal exercise systolic blood pressure remained significantly elevated at 5 minutes and then quickly fell to preexercise levels. Diastolic pressure dropped significantly below preexercise values by 5 minutes post exercise, and was also significantly lower at 45 and 60 minutes. Post-exercise total peripheral resistance (TPR), and forearm vascular resistance (FVR) were both persistently lower than preexercise values for the entire recovery phase. Systolic and diastolic blood pressure showed no significant drop from preexercise values in recovery from mild and moderate exercise. Furthermore, no significant change was observed between post and preexercise TPR; however, FVR was significantly elevated above preexercise values for the entire 60 minutes of recovery. Heart rate returned rapidly to preexercise values following minimal and moderate exercise; but

remained significantly elevated for entire 60 minutes of recovery following maximal exercise.

The results from this study indicate a relationship between exercise intensity and post exercise cardiovascular and hemodynamic parameters. Maximal (incremental) exercise causes an overall peripheral vasodilation with reductions in TPR and FVR. These results suggest that vasodilation in exercising and nonexercising limbs causes the decrease in blood pressure. The heavy exercise appears to have caused the presence of generalized vasodilator stimulus, which overcomes reflex vasoconstriction. The higher TPR following mild and moderate exercise was speculated as the increase in vasodilation in exercising regions was counterbalanced by a persisting vasoconstriction in non-exercising regions.

2.4: Post-Exercise Thermal Homeostasis-Hyperthermia

Thoden et al. (1994) investigated the response of esophageal and rectal temperatures to exercise during recovery. Subjects performed running treadmill exercise for 18 minutes at an intensity of approximately 75% of their VO_{2max} and recovered for 65 minutes in a seated/leaning position. These investigators observed a rapid drop in T_{es} followed by a stable elevated plateau above resting levels, which was established within 10 minutes, and remained for the entire 65 minutes of recovery. Rectal temperature (T_{re}) continued to fall gradually throughout the entire 65 minute recovery period. All nonacral skin temperatures, with the exception of the thigh, continued to rise for 2 to 3 minutes following cessation of exercise, but stabilized at or just above preexercise levels, 20 to 30 minutes post exercise. This stabilized skin temperature suggests that cutaneous

vasodilation had ceased, or become attenuated. The higher thigh temperature suggested that a significant amount of metabolic heat produced by the exercise still remained in the exercising muscles and the body was doing nothing to dissipate it after this stable T_{es} had been established. If cutaneous blood flow has fallen (as indicated by a drop in skin temperatures), the vasculature within the exercising muscles is likely vasodilated.

Perhaps the most significant finding along with the elevated plateau of T_{es} is that the temperature corresponded with the T_{es} threshold for cutaneous vasodilation T_{dil} . Once T_{es} had fallen to the threshold for vasodilation, the body appeared to stop actively trying to dissipate heat through both sensible and non-sensible heat loss mechanisms. The investigators postulated that the body had likely entered the 'null zone' of thermoregulation. In contrast, it is also possible that the elevated T_{es} plateau was indicative of a resetting (increase) of the hypothalamic set-point due to the increase in metabolic heat production.

The effects of exercise intensity and ambient temperature on the post exercise elevation in T_{es} and T_{dil} was investigated by Kenny (1997). Subjects performed treadmill running exercise at a variety of ambient temperatures and exercise intensities (cool with light exercise, temperate with heavy exercise, warm with heavy exercise and hot with light exercise). Light and heavy exercise were 45% VO_{2max} and 75% VO_{2max} respectively. The most significant observation from this study was that the relationship between the core temperature threshold for dilation was related to the post exercise elevation in esophageal temperature at every exercise condition. That is, the $T_{dil} - T_{es}$ relationship remains intact over a wide range of exercise intensities. The researchers speculated that the vasodilatory

threshold during recovery is equal to the T_{dil} during exercise since skin blood flow decreases as T_{es} decreases, and SkBF reaches baseline values when T_{es} plateaus. This would have something to do with baroreceptor activity following exercise.

Kenny (1999) also investigated the effects of exercise duration on the post exercise elevation in T_{es} . In this study, subjects performed running exercise for 3 different exercise durations, 15 min, 30 min and 45 min. In this particular study, he found differences in end exercise T_{es} , which was a function of a gradually increasing core temperature. In addition, T_{dil} occurred at essentially the same T_{es} for all 3 exercise intensities. Furthermore, the relationship between T_{dil} and post exercise T_{es} remained intact for all 3 exercise intensities as there was no significant difference between the mean T_{dil} and post exercise elevation in ΔT_{es} . This study concluded that the post exercise elevation in T_{es} is not defined by changes in whole-body heat content as produced by endogenous heating during exercise of different duration. This researcher speculated that the increase may be due to a baroreceptor mediated influence.

Chapter 3: Methodology:

3.1: Subjects:

For this study, a sample of eight university students was used as subjects. Only male subjects were used for the present study to avoid variations among subjects due to body temperature changes associated with the female menstrual cycle. All subjects gave appropriate Physical Activity Readiness Questionnaire (PAR-Q) responses (accepted by the HK-HREC or appropriate screening for exercise by subjects under 30 years of age.), and were thoroughly briefed verbally, and in writing before being asked to sign an information and consent form, (refer to copy of form letters, information and Ethics clearance in appendix I). All subjects were made fully aware of their right to withdraw from the study at any time without repercussion of any kind.

3.2: Instrumentation:

Esophageal temperature (T_{es}) was measured at the level of the left atrium with a probe (Pharmaseal APC 400 series) inserted through the nasal passage, down the esophagus to a distance of one quarter of the subject's height from the nares (Thoden et al., 1994). Surface thermistors (Yellow Springs Instruments model 44004) were applied using hypoallergenic medical adhesive tape (Electroplast) to the following sites on the skin: left forearm, index finger, forehead, chest, subscapular area, thigh, and calf.

The graded incremental maximal aerobic power test and experimental trials were performed on a running treadmill (Quinton Uniwork model 844). The preliminary test, as well as the experimental sessions I, II and III occurred in a thermal control room,

controlled to a temperature of 29°C and relative humidity of approximately 50% by a thermal climatic generator (Cancoil model GLP 0851B).

Oxygen consumption measurements were sampled through a two-way valve breathing system (Collins , Hans-Rudolph) and an automated gas analyzer (Quinton Q-Plex model 1); heart rate(HR) was monitored using a heart rate monitor (Polar Sport-tester, model PE 3000). An electrophygomanometer (Finapres) applied to the left index or middle finger was used to measure blood pressure (BP), and relative changes in forearm cutaneous blood flow (SkBF) were measured noninvasively using laser Doppler (Laser Flow, Blood Perfusion Monitor BPM 403A) applied to the left forearm. The left arm of the subject was placed in a sling to both immobilize it, as well as adjust the height of the Finapres cuff such that it aligned with the level of the heart (approximately the 5th intercostal space).

3.3: Protocol:

3.3.1: Preliminary Session:

All volunteering subjects, on the preliminary screening day, were informed of the procedures, and potential risks involved in participating in the investigation. The signing of a written informed consent form (Appendix I) outlining the experimental protocol approved by the University of Ottawa's Policy on Ethics of Human Research Committee followed.

During the preliminary session, subjects were individually provided with the information pertaining to the nature of the study. Following pertinent verbal & written briefing, as well as signing of the consent form, the subject's weight, resting blood pressure, and height were measured.

To determine the intensity at which the subjects exercised during the thermoregulatory studies, subjects were tested for their anaerobic threshold (using ventilatory threshold as the identifier) using an incremental graded treadmill test as described by Thoden (1991). However, the subjects were pushed to fatigue to help find their maximal oxygen consumption (VO_{2max}), as well as to identify their suprathreshold exercise intensity HR zone. Criteria for attaining VO_{2max} , as outlined by Shi et al. (1993), included 1) a plateau of VO_2 in the last stage of the test, 2) a respiratory exchange ratio of greater than 1.1, 3) no further increase of heart rate (HR) to increase with increasing power output, or 4) voluntary exhaustion.

To avoid complications on the following experimental test days, subjects were trained to insert the esophageal probe under supervision as this has proven to be the most comfortable and effective approach. Furthermore, subjects became oriented with the other equipment during the preliminary session in order to become familiar with the instrumentation.

3.3.2: Experimental Sessions:

At least 48 hours were allowed between the initial test screening day and the first test day to ensure full recovery. Experimental test sessions were separated by at least 48 hours to avoid physiological adaptation. The following protocol was used for each of the three experimental day sessions. Experimental sessions were conducted in the morning following a 24 hour period without prolonged or heavy physical training. In the final 12 hours prior to the test, subjects ate a balanced carbohydrate based meal, and refrained from the consumption of alcohol, caffeine or other stimulants. The morning of the test,

subjects were asked to consume only juice for breakfast, take care to avoid any physical or heat stress prior to the test, and consume 250 ml of water every waking hour up to commencement of the experiment to keep plasma volume stable (Thoden et al 1994).

Subjects wore only running shorts and comfortable running shoes during the testing days. All temperature and blood flow probes, and other equipment were applied while the subject was in the thermal chamber set at an ambient temperature of 29°C and approximately 50% relative humidity. Skin temperature thermistors were applied at the left forearm, finger, forehead, chest, thigh, calf, and subscapular area. The esophageal temperature probe was inserted via the nares and esophagus to a distance of one quarter of the subjects standing body height (Thoden, 1994). In addition, the heart rate monitor and Laser Doppler blood flow probe were applied. Following application of equipment, the subject remained in a leaning/seated position for 15 minutes to allow complete habituation to the new temperature.

The order of trials I, II, and III were assigned randomly to each subject so that an approximately equal number of subjects were exposed in each of the 6 possible orders.

Trial I:

After core and surface temperatures, blood flow, heart rate and blood pressure had become stable (approximately 15 minutes), subjects performed treadmill running exercise at an intensity of approximately 10 to 15% below their pre-determined anaerobic threshold (subthreshold) for a duration of 15 minutes. A duration of fifteen minutes was used to ensure that full thermoregulatory responses occur, Thoden et al. (1994).

Following the 15 minute exercise period, subjects returned to the leaning/seated position and recovered for 45 minutes while data was recorded.

Trial II:

The procedures for trial II follow that of the protocol for trial I with the exception of a change in exercise intensity. The subject performed treadmill running exercise at an intensity equal to their preassessed anaerobic threshold (threshold) for 15 minutes. Following the exercise phase, the subject returned to the seated/leaning position to recover for 45 minutes while recovery data was taken.

Trial III:

This protocol followed the same pre-exercise protocol of Trials I and II. During exercise, the subject ran for 15 minutes at an intensity of 10% above their pre-determined anaerobic threshold (suprathreshold). Again, following exercise, subjects returned to the seated/leaning position as recovery data was recorded for 45 minutes.

3.4: Data Analysis:

Surface and core temperatures, and cutaneous blood flow data were sampled every five seconds and collected (Hewlett Packard, model PC-312, 9000) for the entire duration of the preexercise, exercise, and post exercise phases of the experimental sessions. Heart rate data was also sampled and collected in the same manner. Blood pressure data was sampled every 5 seconds beginning at two minutes preexercise to obtain resting data, for 10 minutes immediately following exercise, and every 5 minutes thereafter until 45 minutes recovery had passed. Blood pressure data was not collected during the exercise

phase, since the purpose of this study was to compare preexercise and recovery blood pressure only, and to parallel studies performed by Piepoli et al, 1993, 1994. Oxygen consumption data was sampled every 30 seconds and collected preexercise, continually during exercise and 10 minutes post-exercise, and at 20, 30 and 40 minutes post-exercise.

All data was imported and pooled into PC worksheet software (Excel). Statistical analyses were performed using the SPSS for Windows statistical package.

A variation tends to exist in resting T_{ca} due to individual differences among subjects. One of the purposes of this study is to examine the effects of exercise intensity on T_{ca} during exercise recovery. Therefore, this study is examining changes in T_{ca} among exercise conditions. Esophageal temperature was expressed as a positive or negative change (Δ) from 0 by taking the mean temperature of the last 10 minutes prior to exercise, and subtracting this number from the rest of the trial data (ΔT_{ca}). A minute average was recorded at each 5 minute interval for ΔT_{ca} .

The 7 skin site temperatures were expressed as the weighted mean skin temperature (T_{sk}), which considers regional differences in temperature distribution. This was calculated using the following formula:

$$T_{sk} = (0.18)T_{fa} + (0.02)T_{fi} + (0.17)T_{th} + (0.13)T_{ca} + (0.22)T_{ch} + (0.06)T_{fh} + (0.22)T_{sa}$$

Where: T_{sk} : Weighted mean skin temperature

T_{fa} : Forearm temperature

T_{fi} : Fingertip temperature

T_{th} : Thigh temperature

T_{ca} : Calf temperature

T_{ch} : Chest temperature

T_{fh} : Forehead temperature

T_{sa} : Subscapular temperature

Calculated similar to ΔT_{es} , the weighted mean skin temperature was also expressed as a change in temperature (ΔT_{sk}). ΔT_{sk} was recorded every 5 minutes, where the minute average around each 5 minute interval was used.

Cutaneous vascular conductance (CVC) was also represented and calculated as $SkBF/MAP$ (Mack et al., 1994), expressed in conductance units of $v/mmHg$, (since $SkBF$ was not calibrated) and expressed as means and standard deviation. Similar to T_{es} and T_{sk} , cardiovascular conductance was expressed as a change from zero (ΔCVC), to represent changes from rest, and alleviate any individual differences.

The weighted mean skin and core temperatures, systolic (SBP) and diastolic (DBP) blood pressure, mean arterial pressure (MAP), heart rate (HR), and oxygen consumption (VO_2 - mL/kg-min), $SkBF$ and ΔCVC were expressed as means and standard deviation of the mean. Since no methods were available to calibrate skin blood flow, this was used to express relative changes within subjects only to help identify T_{dil} , to identify differences between preexercise and post exercise profiles and to calculate CVC..

Comparisons were made between trials I, II and III using a repeated measures ANOVA and *post hoc* tests (Scheffe and Bonferroni) to examine significant differences ($p < 0.05$) between the 3 exercise intensities. Esophageal temperature (ΔT_{es}) and weighted mean skin temperature (ΔT_{sk}) were compared between trials at preexercise, 5, 10, and 15 minutes during exercise; and 5, 10, 20, 30, 40 and 45 minutes post exercise. Comparisons were made for SBP, DBP, & MAP among trials at preexercise, and 1, 2, 3, 4, 5, 10, 15, 20, 25, 30, 35, 40, and 45 minutes post-exercise. Heart rate was assessed among intensities prior to exercise at 5, 10 and 15 minutes during the exercise stage, and every 5 minutes during the recovery period. Finally, VO_2 was compared among trials at 1 minute

pre-exercise; and 5, 10, 20, 30 40, and post-exercise. The preceding times have been chosen in accordance with experiences recorded in the thermal laboratory at the University of Ottawa by Thoden, Reardon, Kenny, and Jette (1994).

One-way ANOVA and *post hoc* tests ($p < 0.05$) were performed between preexercise values and each 5 minute interval during exercise and recovery. These were performed on ΔT_{es} , ΔT_{sk} , HR, SBP, DBP, MAP, and VO_2 SkBF and ΔCVC at the same measurement intervals previously mentioned.

The internal temperature threshold for cutaneous vasodilation (T_{dil}) was determined by using laser-Doppler flowmetry (SkBF). A transient and sustained increase in SkBF during exercise determined where T_{dil} occurred (see fig. 4.11 for example). This (T_{dil}) was reconfirmed by examining T_{ra} during exercise, as represented by a rapid increase in temperature. T_{dil} was adjusted to take into consideration the possible confounding effects of skin temperature (T_{sk}) (Matsukawa et al, 1995). This correction to give the corrected internal temperature threshold for cutaneous vasodilation (T_{corr}) was calculated by first determining T_{sk} at the uncorrected T_{dil} for all 3 exercise intensities. T_{sk} (designated) was the average T_{sk} of the 3 exercise conditions or intensities. Corrected T_{dil} was then calculated using the following equation, which is used to standardize T_{dil} estimation under conditions during which both T_{es} and T_{sk} are changing:

$$T_{\text{corr}} = \Delta T_{\text{dil}} + [\beta / (1 - \beta)] * (T_{\text{sk}} - T_{\text{sk-designated}})$$

Where: $\beta = 0.2$; which is the contribution of T_{sk} to the vasodilation response.

In order to examine the relationship between T_{corr} and the established elevation in ΔT_{ca} (average of 15, 20 and 25 minutes post-exercise). Pearson correlations were performed for each exercise intensity condition separately as well as all 3 intensities pooled together. In addition, t-tests were also used to examine any significant difference between $T_{\text{dil corr}}$ and the elevated ΔT_{ca} . The time at which T_{dil} occurred was also expressed as mean and standard deviation to determine the effects of exercise intensity on the vasodilation response. A one-way ANOVA, as well as the Scheffe and Bonferroni Post-hoc tests were used to examine possible differences between exercise intensities ($p < 0.05$).

Chapter 4: Results

The mean and standard deviation of age and physical characteristics of the subjects are listed in Table 4.1. The mean age of the 8 subjects was 23.88 (± 2.03) years, height was 180.12 (± 6.22) cm and mean mass was 73.08 (± 5.89) kg.

The mean maximal oxygen consumption (mL/kg-min) derived from the maximal incremental test for the subjects was 64.01 (± 8.33) mL/kg-min, and the mean oxygen consumption (mL/kg-min) at ventilatory threshold was 55.2 (± 7.42) mL/kg-min.

Table 4.1: Mean and Standard Deviation of Subject Characteristics (n=8)

	Age (years)	Mass (kg)	Height (cm)	Maximal Oxygen Consump. (mL/kg-min)	Oxygen Consump. at Threshold (mL/kg-min)
Mean/ S.D.	23.88 (± 2.03)	73.08 (± 5.89)	180.12 (± 6.22)	64.01 (± 8.33)	55.2 (± 7.42)

4.1: Esophageal Temperature

As previously mentioned in the methodology, esophageal temperature was corrected to express T_{es} as a change from zero (ΔT_{es}). The mean preexercise T_{es} was determined which gave values of $0.00 \pm 0.04^{\circ}\text{C}$, $0.03 \pm 0.03^{\circ}\text{C}$, and $0.01 \pm 0.03^{\circ}\text{C}$ for subthreshold, threshold and suprathreshold respectively. The values for the mean and standard deviation of exercise and post exercise ΔT_{es} for all three exercise conditions are shown in table 4.2 and are presented graphically in figure 4.1.

At the commencement of exercise, a transient decrease in ΔT_{es} was observed in all 3 exercise conditions, followed by an eventual rise at approximately 2-3 minutes into

exercise. Esophageal temperature continued to rise at a rapid rate until approximately 5 to 7 minutes of exercise (depending on the exercise intensity), where the rate of rise in ΔT_{es} decreased, and a lower positive rate was observed. This change in rate of rise of ΔT_{es} appeared between approximately 1 to 2 minutes following cutaneous vasodilation. Esophageal temperature continued to rise until 1 to 2 minutes into recovery following the termination of exercise, after which a rapid fall in temperature occurred for all 3 exercise conditions.

Analysis of the results using repeated measures ANOVA as well as *post hoc* analysis demonstrated significant main effects in condition and exercise ($p=0.00$). Between subjects effects and within subjects effects were both significant ($p<0.05$), demonstrating a main effect of exercise intensity and ΔT_{es} . More specific results analyzing post-exercise against preexercise ΔT_{es} are shown in the following subsections. Further comparison between exercise intensity groups are also shown.

Esophageal temperature fell at a similar rate among conditions during the first 5 minutes of recovery (Table 4.3) at an average rate of $0.11^{\circ}\text{C}/\text{min}$, $0.15^{\circ}\text{C}/\text{min}$, and $0.17^{\circ}\text{C}/\text{min}$ for subthreshold, threshold, and suprathreshold exercise intensities respectively. Approximately 10 minutes into recovery, the rate of decrease in ΔT_{es} decelerated. During the remaining 30 minutes of recovery, the mean rates of temperature change in ΔT_{es} were $-0.0032^{\circ}\text{C}/\text{min}$ for subthreshold exercise, $-0.0165^{\circ}\text{C}/\text{min}$ for threshold exercise, and $-0.0042^{\circ}\text{C}/\text{min}$ for suprathreshold exercise. Analysis using a one-way ANOVA showed that the rate of decrease in ΔT_{es} from threshold exercise was higher than both subthreshold and suprathreshold intensities ($p<0.05$).

Table 4.2: Mean and Standard Deviation of Esophageal Temperature (ΔT_{es}) for 3 Different Exercise Intensities

Measurement Interval	Subthreshold Exercise ($^{\circ}\text{C}$) (n=8)	Threshold Exercise ($^{\circ}\text{C}$) (n=7)	Suprathreshold Exercise ($^{\circ}\text{C}$) (n=7)
Pre-exercise	0.00 \pm 0.04	0.01 \pm 0.03	0.01 \pm 0.03
Exercise 5	0.20 \pm 0.23 ^{**}	0.38 \pm 0.12 ^{**}	0.37 \pm 0.31 ^{**}
Exercise 10	0.68 \pm 0.31 ^{**}	1.06 \pm 0.21 ^{**}	1.46 \pm 0.35 ^{**}
Exercise 15	1.02 \pm 0.27 ^{**}	1.53 \pm 0.29 ^{**}	2.17 \pm 0.41 ^{**}
Post 5	0.47 \pm 0.17 ^{**}	0.74 \pm 0.28 ^{**}	1.37 \pm 0.22 ^{**}
Post 6	0.47 \pm 0.0034 ^{**}	0.76 \pm 0.002 ^{**}	1.28 \pm 0.02 ^{**}
Post 7	0.49 \pm 0.002 ^{**}	0.74 \pm 0.001 ^{**}	1.20 \pm 0.01 ^{**}
Post 8	0.50 \pm 0.002 ^{**}	0.71 \pm 0.001 ^{**}	1.14 \pm 0.01 ^{**}
Post 9	0.49 \pm 0.0006 ^{**}	0.71 \pm 0.001 ^{**}	1.08 \pm 0.01 ^{**}
Post 10	0.51 \pm 0.23 ^{**}	0.66 \pm 0.22 ^{**}	1.02 \pm 0.18 ^{**}
Post 15	0.47 \pm 0.22 ^{**}	0.64 \pm 0.10 ^{**}	0.96 \pm 0.20 ^{**}
Post 20	0.46 \pm 0.20 ^{**}	0.58 \pm 0.10 ^{**}	0.81 \pm 0.30 ^{**}
Post 25	0.44 \pm 0.17 ^{**}	0.54 \pm 0.08 ^{**}	0.80 \pm 0.29 ^{**}
Post 30	0.42 \pm 0.15 ^{**}	0.50 \pm 0.11 ^{**}	0.77 \pm 0.29 ^{**}
Post 35	0.41 \pm 0.15 ^{**}	0.46 \pm 0.10 ^{**}	0.74 \pm 0.29 ^{**}
Post 40	0.39 \pm 0.14 ^{**}	0.44 \pm 0.10 ^{**}	0.73 \pm 0.28 ^{**}
Post 45	0.40 \pm 0.15 ^{**}	0.40 \pm 0.14 ^{**}	0.73 \pm 0.31 ^{**}

Note: ^{*}: Significantly different @ $p < 0.05$ (pre-post), ^{**}: Significantly different @ $p < 0.05$ (between groups)

4.1.1: Subthreshold Intensity

The mean and standard deviation of ΔT_{es} prior to exercise was 0.00 ± 0.04 $^{\circ}\text{C}$. At the commencement of exercise, a transient decrease in ΔT_{es} was observed until approximately 2 minutes into exercise, when ΔT_{es} began to rise. During the exercise phase, ΔT_{es} rose to 0.20 ± 0.23 $^{\circ}\text{C}$ five minutes into exercise, 0.68 ± 0.31 $^{\circ}\text{C}$ at 10 minutes, and up to 1.02 ± 0.27 $^{\circ}\text{C}$ at the end of exercise. During recovery, ΔT_{es} dropped rapidly to 0.47 ± 0.17 $^{\circ}\text{C}$ by 5 minutes post-exercise, 0.51 ± 0.23 $^{\circ}\text{C}$ at 10 minutes post-exercise, 0.47 ± 0.22 $^{\circ}\text{C}$ at 15 minutes, 0.46 ± 0.20 $^{\circ}\text{C}$, at 20 minutes, and 0.44 ± 0.17 $^{\circ}\text{C}$ at 25 minutes. ΔT_{es} remained relatively stable, and significantly elevated ($p < 0.05$) above pre-exercise values for the remainder of the 45 minute recovery period. The mean and standard

deviation of ΔT_{es} taken at 20, 25, and 30 minutes recovery was 0.44 ± 0.18 °C (Table 4.4). This elevated plateau was found to be significantly different from preexercise values ($p < 0.05$). One-way Anova and *post hoc* analysis (Bonferroni) demonstrated significant differences in ΔT_{es} between preexercise T_{es} and all measurement intervals (exercise and recovery values) ($p = 0.011$) (Table 4.2).

4.1.2: Threshold Exercise Intensity

The mean ΔT_{es} data for threshold exercise is representative of 7 subjects due to failure during one exercise session. The mean and standard deviation of ΔT_{es} was 0.01 ± 0.03 °C prior to the commencement of exercise. Temperature fell rapidly at the onset of exercise similar to subthreshold exercise, and began rising at a rapid pace approximately 2 to 3 minutes into exercise. At five minutes into exercise, ΔT_{es} was 0.38 ± 0.12 °C, which rose to 1.06 ± 0.21 °C by 10 minutes exercise, and was 1.53 ± 0.29 °C by the end of the exercise period. Temperature dropped rapidly approximately 1 to 2 minutes following exercise to 0.74 ± 0.28 °C by 5 minutes into recovery, 0.66 ± 0.22 °C at 10 minutes post-exercise, 0.64 ± 0.10 °C at 15 minutes, 0.58 ± 0.10 °C at 20 minutes, 0.54 ± 0.08 °C at 25 minutes, and 0.50 ± 0.11 °C at 30 minutes post-exercise. A relatively stable elevation in ΔT_{es} was essentially established approximately 5 to 10 minutes into recovery. The mean and standard deviation across 20, 25 and 30 minute time intervals of ΔT_{es} was 0.56 ± 0.10 °C (Table 4.4).

The one-way ANOVA and *post hoc* analysis (Scheffe, Bonferroni) against preexercise values showed results similar to subthreshold exercise. Significant differences

in T_{es} were observed between preexercise measurements and all exercise (with the exception of 5 minutes) and post-exercise measurement intervals ($p=0.031$). Thus, a relatively stable elevation in ΔT_{es} significantly different from preexercise values was observed from approximately 5 minutes, through the remainder of the 45 minutes of recovery.

4.1.3: Suprathreshold Exercise Intensity

The mean and standard deviation ΔT_{es} data is representative of 7 subjects due to 1 subject terminating the exercise phase of the test prematurely due to fatigue. Prior to exercise, ΔT_{es} was $0.01 \pm 0.03^{\circ}\text{C}$, and again dropped at the onset of exercise, indicative of a redistribution of blood away from the periphery. ΔT_{es} appeared to rise later into exercise than during subthreshold and threshold intensities. Five minutes into exercise, the mean and standard deviation of ΔT_{es} was $0.37 \pm 0.31^{\circ}\text{C}$, which rose rapidly to $1.46 \pm 0.35^{\circ}\text{C}$ by 10 minutes, and reached $2.17 \pm 0.41^{\circ}\text{C}$ by the termination of exercise. Approximately 1 minute into recovery, ΔT_{es} began to drop rapidly and was $1.37 \pm 0.22^{\circ}\text{C}$ at 5 minutes post-exercise, $1.02 \pm 0.18^{\circ}\text{C}$ 10 minutes into recovery, $0.96 \pm 0.20^{\circ}\text{C}$ 15 minutes into recovery. At 20 minutes post exercise, ΔT_{es} was $0.81 \pm 0.30^{\circ}\text{C}$, and $0.80 \pm 0.29^{\circ}\text{C}$ at 25 minutes. The mean and standard deviation of ΔT_{es} taken from the average of minute 20, 25, and 30 was $0.91 \pm 0.21^{\circ}\text{C}$, significantly different from preexercise values (Table 4.4, and figure 4.1).

During exercise, one-way ANOVA and *post hoc* analysis demonstrated ΔT_{es} was significantly higher ($p=0.00$) than preexercise values at all 3 exercise stage measurement

intervals. In addition, significant differences were also found in ΔT_{ca} between preexercise values and post exercise ($p=0.05$) at the designated measurement interval times.

4.1.4: Comparisons in ΔT_{ca} Between the 3 Exercise Intensities

The differences among the 3 exercise intensities are presented graphically in figure 4.1. As mentioned in the methodology, repeated measure ANOVAs and *post hoc* analysis was performed to examine a possible difference in ΔT_{ca} among the 3 exercise conditions at each 5 minute interval. These repeated measure ANOVAs were performed at the $p<0.05$ level.

No significant differences in ΔT_{ca} among the 3 exercise conditions were found prior to exercise, as well as 5 minutes into exercise. However, 10 minutes into the exercise phase, significant differences were found between suprathreshold and subthreshold exercise, as well as suprathreshold and threshold exercise ($p=0.05$). Furthermore, at the termination of exercise (15 minutes), *post-hoc* analysis showed significant differences in ΔT_{ca} between all three exercise conditions ($p=0.02$).

During the recovery phase from exercise, ΔT_{ca} dropped rapidly in the first 5 minutes in all 3 exercise conditions, and eventually established a relatively stable elevation by 10 minutes into recovery. Repeated measures ANOVA and one-way ANOVA with *post hoc* demonstrated significant differences in ΔT_{ca} among suprathreshold, and threshold and subthreshold exercise conditions. In measurement intervals 1 to 9 minutes into recovery, *post hoc* analysis using both Bonferroni and Scheffe tests demonstrated significant differences among the three exercise intensities. Following 10 minutes of recovery, however, *post hoc* analysis showed that significant differences were only evident

between the suprathreshold and threshold exercise conditions, as well as the suprathreshold and subthreshold exercise conditions. No significant differences were found in ΔT_{es} between threshold and subthreshold exercise intensities following 10 minutes of recovery ($p < 0.05$). The values of ΔT_{es} from suprathreshold exercise were significantly different from threshold and subthreshold exercise intensities at 5 minutes, 10, 15, 30, 35, 40, and 45 minutes into recovery ($p = 0.04$). Suprathreshold values of ΔT_{es} were not significantly different from threshold during the 20, 25 and 30 minute intervals in recovery, but were statistically different from subthreshold values ($p = 0.01$).

Table 4.3: Mean Post-Exercise Change in ΔT_{es} Across Time Intervals

Measurement Interval(min)	Subthreshold Exercise ($^{\circ}\text{C}/\text{min}$)	Threshold Exercise ($^{\circ}\text{C}/\text{min}$)	Suprathreshold Exercise ($^{\circ}\text{C}/\text{min}$)
Post 0-5	-0.11	-0.15	-0.17
Post 5-10	-0.006	-0.01	-0.068
Post 10-15	-0.006	-0.02**	-0.008
Post 15-20	-0.002	-0.01**	-0.0032
Post 20-25	-0.004	-0.01**	-0.004
Post 25-30	-0.002	-0.01**	-0.004
Post 30-35	-0.004	-0.02**	-0.004
Post 35-40	-0.002	-0.04**	-0.004
Post 40-45	0	-0.03**	-0.002

Note: **: Significantly different between groups @ $P < 0.05$.

Table 4.4: Mean and Standard Deviation of Post-Exercise Elevation in ΔT_{es}

Measurement Interval	Subthreshold Exercise ($^{\circ}\text{C}$) (n=8)	Threshold Exercise ($^{\circ}\text{C}$) (n=7)	Suprathreshold Exercise ($^{\circ}\text{C}$) (n=7)
Pre-Exercise	0.00 \pm 0.04	0.01 \pm 0.03	0.01 \pm 0.03
Post-Exercise Elevated Plateau	0.44 \pm 0.18**	0.56 \pm 0.10** ⁺	0.91 \pm 0.21** ⁺

Note: **: Significantly different @ $p < 0.05$ (Pre-post);
 **: Significantly different between groups @ $p < 0.05$;
 +: Significantly different between groups @ $p < 0.10$ (Subthreshold-threshold).

4.2: Internal Temperature Threshold for Cutaneous Vasodilation (T_{dil})

As previously mentioned in the methodology, the internal temperature threshold for cutaneous vasodilation (T_{dil}) was corrected for the contribution of skin temperature (T_{sk}) to the vasodilation response. The designated T_{sk} was 32.45°C for all 8 subjects and 3 exercise conditions. The contribution of T_{sk} to the vasodilatory response coefficient, β , was 0.2.

The internal temperature threshold for cutaneous vasodilation was determined by examining the profiles of SkBF during exercise. This was reconfirmed (if necessary) through examining T_{fa} profiles as well. At T_{dil} , a rapid, steep increase in SkBF was observed, as well as a rapid increase in T_{fa} in all cases.

The mean and standard deviation of T_{dil} are found in table 4.5, and figure 4.2. The mean and standard deviation of T_{dil} (corrected) for subthreshold exercise was 0.26 ± 0.16°C; 0.47 ± 0.13°C for threshold exercise; and 0.84 ± 0.12°C for suprathreshold exercise. Analysis using a one-way ANOVA, as well as *post hoc* analyses demonstrated significant differences in the mean ($p < 0.05$) of T_{dil} among all 3 exercise conditions.

The time into exercise where cutaneous vasodilation occurred was also examined between the 3 exercise conditions to see the indirect effects of exercise intensity on vasoconstriction. These values can also be found in table 4.6. The mean and standard deviation of time for subthreshold, threshold and suprathreshold exercise were 5.20 ± 1.13 min (5 min., 12 s), 5.80 ± 0.93 min (5 min, 48 s), and 6.93 ± 1.23 min (6 min, 56 s), respectively. One-way ANOVA demonstrated significant differences among the 3 exercise intensities in time at which T_{dil} occurred; and *post-hoc* tests showed that the differences occurred between suprathreshold intensity and subthreshold intensity only ($p < 0.05$).

**Table 4.5: Corrected T_{dil} for all 3 Exercise Intensities
B= 0.2**

	Subthreshold Exercise	Threshold Exercise	Suprathreshold Exercise
Corrected T_{dil} (°C)	0.26 $\pm 0.11^{**}$	0.47 $\pm 0.13^{**}$	0.84 $\pm 0.15^{**}$
Dilation Time (min)	5.20 $\pm 1.13^{**}$	5.80 ± 0.93	6.93 $\pm 1.23^{**}$

Note: ** : Significant difference at $p < 0.05$

4.3: T_{dil} and Post-Exercise Elevated Plateau in ΔT_{es} Correlation

Pearson correlations were performed to examine the relationship between corrected T_{dil} and the post-exercise elevated plateau in ΔT_{es} at all 3 exercise intensities. These values can be seen in table 4.6 and figure 4.3. The post-exercise ΔT_{es} plateau was taken as the average between the 25, 30, and 35 minutes of recovery in each subject. For subthreshold exercise, the mean and standard deviation of ΔT_{es} was $0.44 \pm 0.18^{\circ}\text{C}$, $0.54 \pm 0.10^{\circ}\text{C}$ for threshold exercise, and $0.91 \pm 0.21^{\circ}\text{C}$ for suprathreshold intensity.

The relationship between T_{dil} and post exercise ΔT_{es} for the three conditions were all poorly correlated as no exercise intensity displayed any statistical significant correlation between T_{dil} and post exercise ΔT_{es} . Using independent t-tests, however, demonstrated no statistical significant difference between the mean ΔT_{es} following subthreshold exercise, and T_{dil} for subthreshold ($p=0.04$). The 2 other exercise intensities were significantly different. Furthermore, correlations performed between T_{dil} and ΔT_{es} for the three combined exercise intensities showed poor a statistically significant correlation (0.67) at the $p < 0.05$ level. Independent t-tests performed to measure statistical differences between T_{dil} and post exercise T_{es} demonstrated a significant difference between the 2 intensities.

Table 4.6: Mean Corrected T_{dit} and Post Exercise Elevation in ΔT_{es}

Exercise Condition	Corrected T_{dit} (ΔT_{es} °C)	ΔT_{es} Plateau (°C)	Correlation ($p < 0.05$)
Subthreshold Exercise	0.26 (± 0.16)	0.44 (± 0.18)	-0.0927
Threshold Exercise	0.47 (± 0.13)	0.56 (± 0.10)	0.1812
Suprathreshold Exercise	0.84 (± 0.12)	0.91 (± 0.21)	0.1134
Mean	0.63 (± 0.26)	0.51 (± 0.28)	0.67

4.4: Weighted Mean Skin Temperature (ΔT_{sk})

All mean and standard deviation ΔT_{sk} values are presented in table 4.7 and figure 4.4. Mean and standard deviation of ΔT_{sk} prior to exercise were not significantly different among the three exercise conditions. At the onset of exercise, all T_{sk} fell after achieving a relatively stable state prior to exercise. Approximately 5 - 7 minutes into exercise, ΔT_{sk} began to rise until the end of exercise and continued 2 - 3 minutes into recovery. The time of rise in ΔT_{sk} corresponded to the peak in rise of T_{fa} , and continued to rise several minutes into recovery.

In some instances, T_{sk} data may seem unstable prior to exercise or during recovery. This was accounted for by the movement of air inside the climactic chamber, causing the T_{sk} data to appear wavy. In general, following exercise, after peak ΔT_{sk} had been reached, it began to fall gradually throughout recovery towards preexercise values.

Table 4.7: Mean and Standard Deviation of ΔT_{sk} for 3 Exercise Intensities During Pre-Exercise, Exercise and Recovery

Measurement Interval	Subthreshold Exercise ($\Delta^{\circ}\text{C}$) (n=8)	Threshold Exercise ($\Delta^{\circ}\text{C}$) (n=8)	Suprathreshold Exercise ($\Delta^{\circ}\text{C}$) (n=7)
Pre-exercise	0.02 ± 0.02	0.01±0.01	0.01±0.01
Exercise 5	0.13±0.27	0.08±0.20	0.17±0.12
Exercise 10	0.92±0.64 ^{**}	0.95±0.24 ^{**}	0.67±0.56 ^{**}
Exercise 15	1.60±0.71 ^{**}	1.50±0.44 ^{**}	1.36±0.70 ^{**}
Post 5	1.60±0.69 ^{**}	1.46±0.73 ^{**}	1.96±0.35 ^{**}
Post 10	0.71±0.84 ^{**}	0.58±0.65 ^{**}	1.17±0.63 ^{**}
Post 15	0.90±0.78	0.96±0.60 ^{**}	1.42±0.48 ^{**}
Post 20	0.71±0.84	0.58±0.65	1.17±0.63 ^{**}
Post 25	0.50±0.75	0.42±0.66	0.78±0.47
Post 30	0.48±0.62	0.28±0.54	0.61±0.58
Post 35	0.39±0.56	0.16±0.57	0.28±0.51
Post 40	0.35±0.44	0.07±0.57	0.38±0.45
Post 45	0.28±0.51	0.05±0.41	0.17±0.33

Note: ^{**}: Significantly different at $p < 0.05$ between pre-exercise and measurement interval, [†]: Significantly different at $p < 0.05$ between 3 exercise intensities.

Repeated measures ANOVA and *Post hoc* analysis found no significant differences among exercise conditions at any time interval ($p < 0.05$). There were no main effects of exercise intensity on mean ΔT_{sk} . Mean skin temperature following suprathreshold exercise demonstrated the slowest recovery of the 3 exercise intensities where T_{sk} remained significantly elevated from 10 minutes into the exercise phase until minute 25 of recovery ($p < 0.05$). Recovery from subthreshold and threshold exercise was more rapid than that of suprathreshold exercise as ΔT_{sk} dropped to resting values by 15 minutes and 20 minutes into recovery for subthreshold and threshold respectively ($p < 0.05$).

4.5: Heart Rate and Oxygen Consumption

The mean and standard deviation of heart rate (HR) (bpm) and oxygen consumption (VO_2) ($\text{mL/kg}\cdot\text{min}$) are presented in tables 4.8 and 4.9 and displayed

graphically in figures 4.5 and 4.6 respectively. No significant differences were found among exercise conditions prior to the commencement of exercise in both HR and VO₂. Oxygen consumption and heart rate were not expressed as change from zero since no significant differences were originally found in preexercise data, and only the relationships among preexercise, exercise and recovery data were being examined. Analysis between groups was only to ensure that the exercise intensities were significantly different from each other.

Table 4.8: Mean and Standard Deviation of Heart Rate (bpm) for 3 Exercise Intensities Across Time (min)

Measurement Interval	Subthreshold (bpm)	Threshold (bpm)	Suprathreshold (bpm)
Pre-Exercise	78 ±12	81±18	80±16
Exercise 5	153±12 ^{**}	163±11 ^{**}	173±11 ^{**++}
Exercise 10	164±14 ^{**}	173±13 ^{**}	185±11 ^{**++}
Exercise 15	168±13 ^{**}	180±14 ^{**}	191±8 ^{**++}
Post 5	110±15 ^{**}	117±21 ^{**}	125±16 ^{**}
Post 10	105±14	113±19 ^{**}	121±17 ^{**}
Post 15	100±17	105±20	114±11 ^{**}
Post 20	98±15	101±15	110±10
Post 25	98±16	100±17	113±15
Post 30	95±17	99±17	112±13
Post 35	97±20	96±20	109±14
Post 40	95±16	96±18	108±15
Post 45	93±14	97±19	107±16 ^{**}

Note: ^{**}: Significantly different @ p<0.05 (pre-exercise & recovery)
^{**}: Significantly different between groups at p<0.05.

All mean and standard deviation of heart rate are listed in table 4.8 and presented graphically in figure 4.5. Heart rate increased rapidly during the first few minutes of exercise in all 3 exercise conditions. Following this rapid increase, heart rate continued to rise at a more gradual rate until the termination of exercise. During the first few minutes of recovery, HR demonstrated a rapid decrease towards preexercise values in all 3 exercise intensities. At approximately 15 minutes into recovery, the rate of decrease in HR had

decreased dramatically, and demonstrated a relative elevation in heart rate compared to preexercise values. Statistically significant differences were not found among the 3 exercise conditions in recovery HR. Following 15 minutes recovery from exercise, HR had essentially returned to baseline levels.

Only preexercise and post exercise data were compared with oxygen consumption. In all 3 exercise conditions, VO_2 dropped rapidly towards preexercise values following the termination of exercise. Within the first 10 minutes of recovery, oxygen consumption returned to preexercise values in all 3 exercise conditions (Table 4.9, figure 4.6).

Table 4.9: Mean and Standard Deviation of Oxygen Consumption (mL/kg·min) During Recovery from 3 Exercise Intensities Across Time Intervals

Measurement Interval	Subthreshold (mL/kg·min)	Threshold (mL/kg·min)	Suprathreshold (mL/kg·min)
Pre-Exercise	4.94±0.82	6.13±1.36	6.41±2.11
Post 5	7.34±1.86	9.95±2.40 ^{**}	11.9±3.14 ^{**}
Post 10	6.00±1.58	7.65±2.31	9.03±1.44
Post 20	5.96±1.17	6.11±1.27	6.9±2.2
Post 30	4.8±1.09	5.41±1.24	6.56±2.35
Post 40	4.67±1.09	6.03±1.15	6.74±2.69

Note: ^{**} Significantly different (pre-exercise - measurement interval) @p<0.05

4.6: Blood Pressure (SBP, DBP): Mean Arterial Pressure (MAP)

All mean and standard deviation of blood pressure (MAP, SBP, DBP) data for all 3 exercise intensities are displayed in tables 4.10 through 4.12. Hemodynamic data are also presented graphically in figures 4.7, 4.8, and 4.9. Again, since preexercise and post-exercise data within groups are being examined, blood pressure was not expressed as a

change from zero. Statistically significant differences were not found among the 3 exercise conditions ($p < 0.05$) prior to the commencement of exercise.

At the termination of exercise, blood pressure fell immediately and rapidly towards preexercise levels. Within 5 minutes into recovery, blood pressure had already dropped to preexercise values for all 3 exercise conditions as indicated by MAP, SBP and DBP data.

Table 4.10: Mean and Standard Deviation of Mean Arterial Pressure (mmHg) During Recovery from 3 Exercise Intensities Across Time Intervals

Measurement Interval	Subthreshold (mmHg) (n=8)	Threshold (mmHg) (n=7)	Suprathreshold (mmHg) (n=7)
Preexercise	97±8.7	92±8.4	95±4.2
Post-Exercise(min)			
1 min	91±0.89	97±1.0	93±2.2
2 min	90±0.55	94±0.84	92±1.1
3 min	90±0.55	91±1.2	90±0.84
4 min	92±0.45	95±0.45	85±1.1
5 min	93±8.0	88±9.1	87±7.3
10 min	94±13.3	85±10.4	85±9.5
15 min	88±10	84±6.3	79±9.0 ^{***}
20 min	83±10.2	85±4.9	80±9.4
25 min	80±6.8	84±7.5	82±7.6
30 min	81±11.2	83±6.1	80±8.7
35 min	83±10	85±8.6	81±8.6
40 min	82±10.2	84±10.2	83±8.0
45 min	84±8.4	89±9.7	84±8.9

Note: ^{***} Significantly different at $p < 0.05$ (pre-post).

Table 4.11: Mean and Standard Deviation of Systolic Pressure (mmHg) During Recovery from 3 exercise Intensities Across Time Intervals

Measurement Interval	Subthreshold Exercise (mmHg) (n=8)	Threshold Exercise (mmHg) (n=7)	Suprathreshold exercise (mmHg) (n=7)
Pre-Exercise	134±5.6	129±13.1	132±7.5
Post Exercise			
1 min	133±0.84	134±2.1	130±3.4
2 min	128±0.89	126±1.6	124±2.8
3 min	124±0.55	118±2.3	117±1.5
4 min	126±0.84	122±0.45	110±2.3
5 min	128±4.8	116±18.1	115±18.0
10 min	130±7.4	109±16.5	110±18.1
15 min	121±8.9	111±13.0	104±17.0**
20 min	112±9.7	112±9.0	102±12.4
25 min	109±9.1	111±9.9	109±6.4
30 min	109±14.0	110±8.4	103±15.8**
35 min	116±12.6	113±11.4	102±15.0**
40 min	112±14.7	110±14.3	106±13.8
45 min	114±12.0	117±11.2	107±13.5

Note: ** Significantly different at p<0.05 (pre-post).

Table 4.12: Mean and Standard Deviation of Diastolic Blood Pressure (mmHg) During Recovery from 3 Exercise Intensities Across Time

Measurement Interval	Subthreshold Exercise (mmHg)	Threshold Exercise (mmHg) (n=7)	Suprathreshold exercise (mmHg) (n=7)
Pre-Exercise	79±12.8	74±10.7	76±6.3
Post Exercise			
1 min	70±1.5	78±1.2	75±1.9
2 min	73±2.1	78±0.7	77±0.55
3 min	74±0.55	78±1.5	76±0.84
4 min	75±0.45	82±0.45	73±0.89
5 min	75±10.9	71±6.0	73±2.4
10 min	84±10	73±8.7	73±6.5
15 min	72±11.1	71±5.0	67±6.6
20 min	68±11.3	71±4.7	67±7.4
25 min	65±7.4	70±8.1	69±5.0
30 min	67±10.8	70±7.4	68±5.6
35 min	67±10.5	71±8.5	71±5.5
40 min	67±10.4	71±9.5	72±5.0
45 min	70±10.1	75±9.8	72±8.8

Note: ** Significantly different @p<0.05; * significantly different @p<0.10

4.6.2: Subthreshold Exercise

A rapid decrease in blood pressure was observed in the first minute of recovery, and MAP, DBP, SBP all dropped to preexercise values by five minutes into the recovery phase. Mean arterial blood pressure remained at preexercise levels during 10 and 15 minutes of the recovery phase at 94 ± 13.3 mmHg and 88 ± 10 mmHg, respectively. One – way ANOVA, with *post hoc* analysis demonstrated no significant difference between recovery and preexercise MAP values ($p<0.05$). Mean arterial pressure remained close to preexercise values for the remainder of recovery.

Systolic blood pressure demonstrated similar trends where it fell to preexercise levels by 5 minutes recovery and remained at almost preexercise resting values for the duration of recovery. *Post hoc* analysis did not find SBP significantly lower than preexercise values. Diastolic blood pressure also dropped rapidly following exercise to preexercise levels, and *post hoc* analysis demonstrated no significant difference between preexercise and recovery values.

4.6.3: Threshold Exercise

Threshold exercise demonstrated similar trends to that of subthreshold exercise. Both SBP and DBP, and consequently MAP dropped rapidly towards preexercise levels at the termination of exercise. By 5 minutes into recovery, SBP, DBP and MAP had all dropped back down to preexercise values of 116 ± 18.1 mmHg, 71 ± 6.0 mmHg, and 88 ± 9.1 mmHg respectively ($p<0.05$).

After 5 to 10 minutes recovery, SBP continued to drop to 109 ± 16.5 mmHg, which was not significantly lower than preexercise values ($p<0.05$). Systolic blood pressure

remained at a level significantly equal to preexercise values for the remainder of recovery from exercise. Diastolic blood pressure also dropped to preexercise levels by 5 minutes post-exercise; but, remained at this level for the entire recovery, rather than dropping any further. Finally, MAP recovery from threshold exercise demonstrated similar trends to that of subthreshold exercise, as it fell, and remained significantly equal to preexercise values throughout the remainder of recovery.

4.6.4: Suprathreshold Exercise

The mean and standard deviation of MAP, SBP and DBP are presented in tables 4.10, 4.11, and 4.12, and figures 4.7 and 4.8. Mean arterial pressure dropped rapidly following exercise and reached preexercise levels by 5 minutes into recovery. Mean arterial pressure remained significantly at preexercise values for the remainder of the recovery phase, with the exception of 15 minutes into recovery, where MAP was significantly lower than preexercise values ($p=0.04$).

Systolic blood pressure showed a similar trend to MAP; but, *post hoc* analysis (Bonferroni) showed MAP was significantly lower than preexercise values at 5 minutes ($p=0.05$), 30 minutes ($p=0.03$) and 35 minutes ($p=0.023$) into recovery. *Post hoc* analysis using Scheffe, however, did not show any significant differences between preexercise and recovery SBP values ($p<0.05$). Diastolic blood pressure dropped to preexercise levels within 5 minutes into recovery ($p<0.05$), and remained for the duration of the 45 minute recovery period.

4.7: Skin Blood Flow and Cardiovascular Conductance

The mean and standard deviation of skin blood flow (SkBF) and change in cardiovascular conductance (Δ CVC) prior to, and during recovery from exercise are presented in tables 4.13 and 4.14, and graphically in figures 4.10 and 4.11, respectively. There were no statistical differences ($p < 0.05$) among the 3 intensities in SkBF or Δ CVC prior to exercise.

At the termination of exercise, both SkBF and Δ CVC were significantly elevated above preexercise values ($p < 0.05$), and remained elevated for the remainder of recovery. The graphical trends in SkBF and Δ CVC appear to be similar throughout recovery among the 3 exercise conditions as they tend to remain elevated for approximately 20 minutes into recovery, and then begin to decrease through the remainder of the recovery period. Graphically, SkBF and Δ CVC following subthreshold exercise appear to be higher than that following threshold and suprathreshold exercise for the entire recovery period. However, for the entire recovery period, no significant differences in SkBF and Δ CVC were found among the 3 exercise intensities ($p < 0.05$).

Cardiovascular conductance appear to be similar between threshold and suprathreshold exercise as they both remain fairly stable for the first 20 to 25 minutes of recovery, and then slowly start to fall during the remainder recovery time. Cardiovascular conductance following subthreshold exercise, however, graphically demonstrates a different trend. Approximately 10 to 15 minutes into the recovery period, Δ CVC begins to increase, until approximately 25 minutes, when it then begins to follow the trends of threshold and suprathreshold intensities.

Table 4.13: Mean & Standard Deviation of Laser Doppler Flow (SkBF: v) During Recovery From 3 exercise Intensities Across Time Intervals (n=8)

Measurement Interval	Subthreshold Intensity	Threshold Intensity (n=7)	Suprathreshold Intensity (n=7)
Pre-Exercise	1.12±0.23	1.16± 0.19	1.39± 0.61
Post-Exercise			
5 min	4.46**±1.85	4.14±2.24**	3.74±2.05**
10 min	4.29±2.16**	3.98±2.00**	2.97±1.26**
15 min	4.8±2.50**	3.81±2.18**	3.75±1.13**
20 min	4.32±1.70**	3.93±1.68**	3.61±1.70**
25 min	4.61±1.61**	3.4±2.62**	3.37±1.61**
30 min	4.3 ± 2.36**	3.17±2.76**	3.57±1.82**
35 min	4.02 ±1.86**	3.00±2.39**	3.22 ±1.84**
40 min	3.7 ±1.61**	2.45±1.79**	2.94 ±1.70**
45 min	3.59 ±2.09**	2.25±1.72	3.11±1.72**

Note: **: Significantly different @ p<0.05 between pre/post exercise measurements.

Table 4.14: Mean and Standard Deviation of Cardiovascular Conductance (ΔCVC) During Recovery from 3 Exercise Intensities Across Time Intervals (n=8)

Measurement Interval	Subthreshold Exercise (v/mmHg)	Threshold Exercise (v/mmHg) (n=7)	Suprathreshold Exercise (v/mmHg) (n=7)
Pre-Exercise	0.00± 0.0027	0.00 ± 0.0029	0.00±0.0067
Post 5	0.036±0.027**	0.034±0.022**	0.028±0.021**
Post 10	0.034±0.044**	0.034±0.018**	0.020±0.016**
Post 15	0.043±0.053**	0.033±0.023**	0.033±0.014**
Post 20	0.041±0.061**	0.034±0.020	0.031±0.019**
Post 25	0.046±0.081**	0.028±0.036**	0.027±0.018**
Post 30	0.042±0.075**	0.026±0.035**	0.030±0.020**
Post 35	0.037±0.062**	0.023±0.031**	0.025±0.019**
Post 40	0.034±0.047**	0.017±0.021	0.021±0.018**
Post 45	0.031±0.046**	0.013±0.024	0.022±0.019**

Note: **: Significantly different @p<0.05 for pre/post exercise measurements

FIGURES AND GRAPHS

Fig. 4.1: Graph of Tes (°C) vs. Time (min) for Three Exercise Intensities

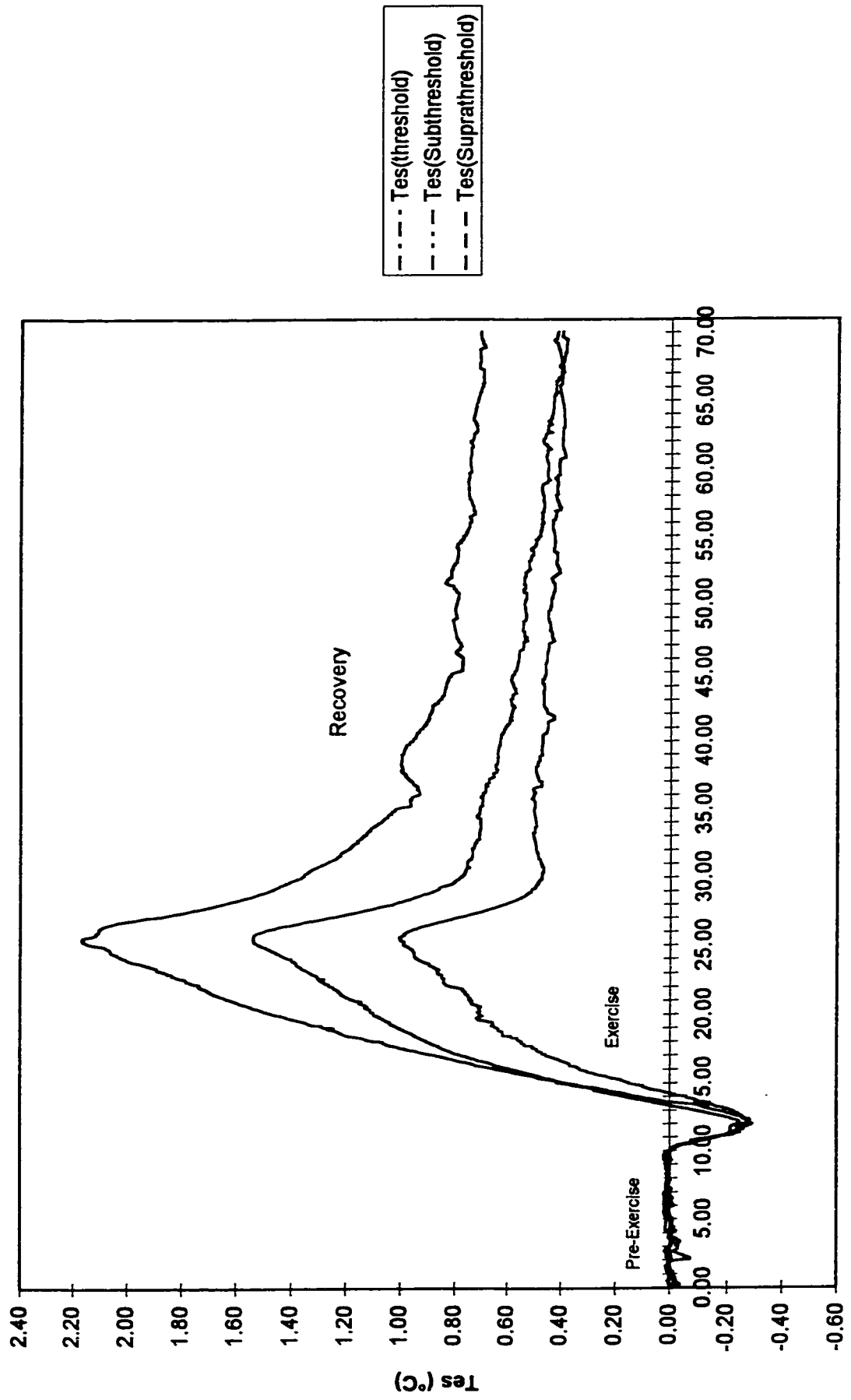


Fig. 4.2: Graph of Corrected Tdii(°C) vs. Post Exercise Tes(°C) for 3 Exercise Intensities

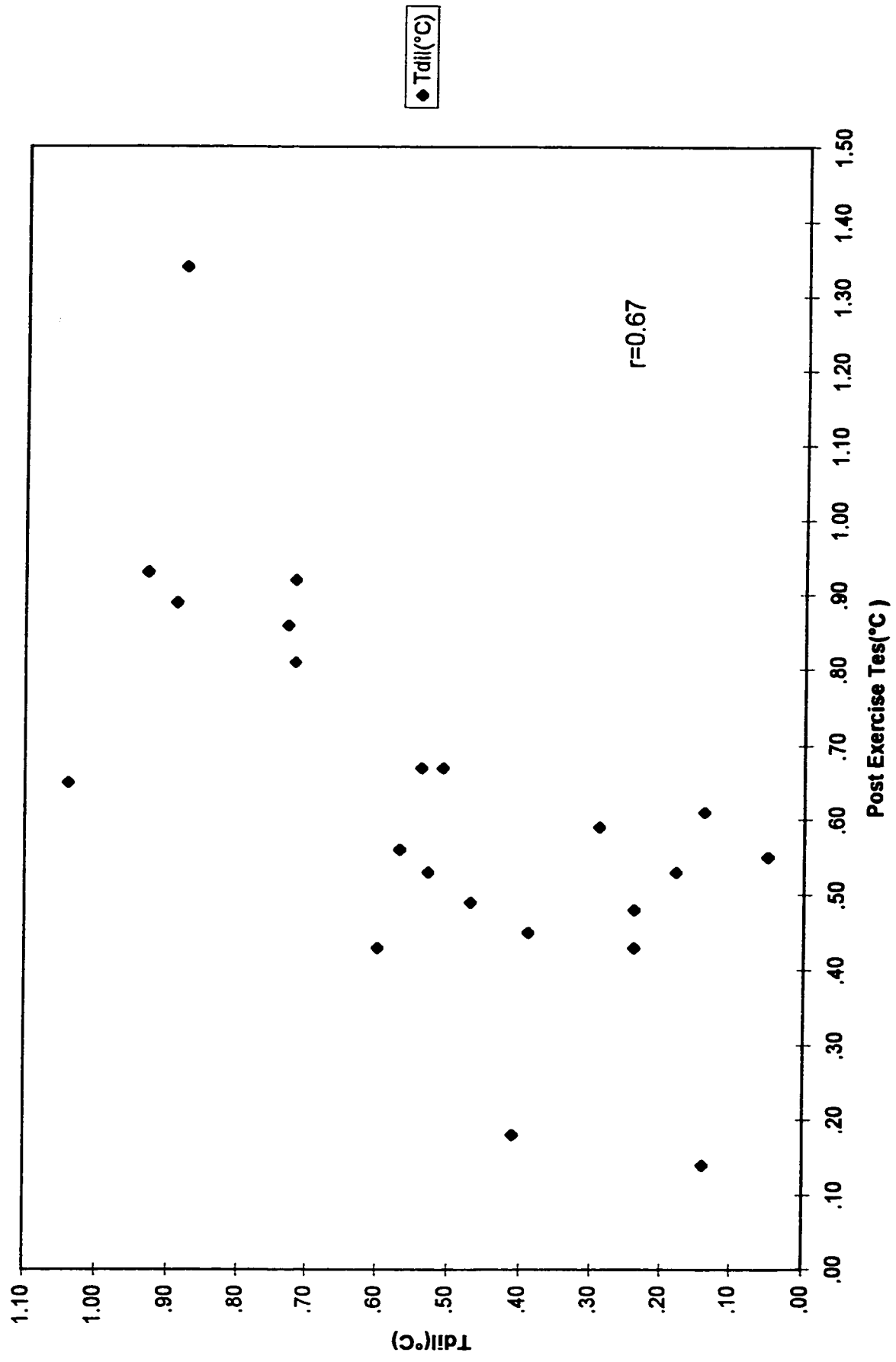


Fig. 4.3: Graph of Corrected Tdil(°C) for 3 Exercise Intensities

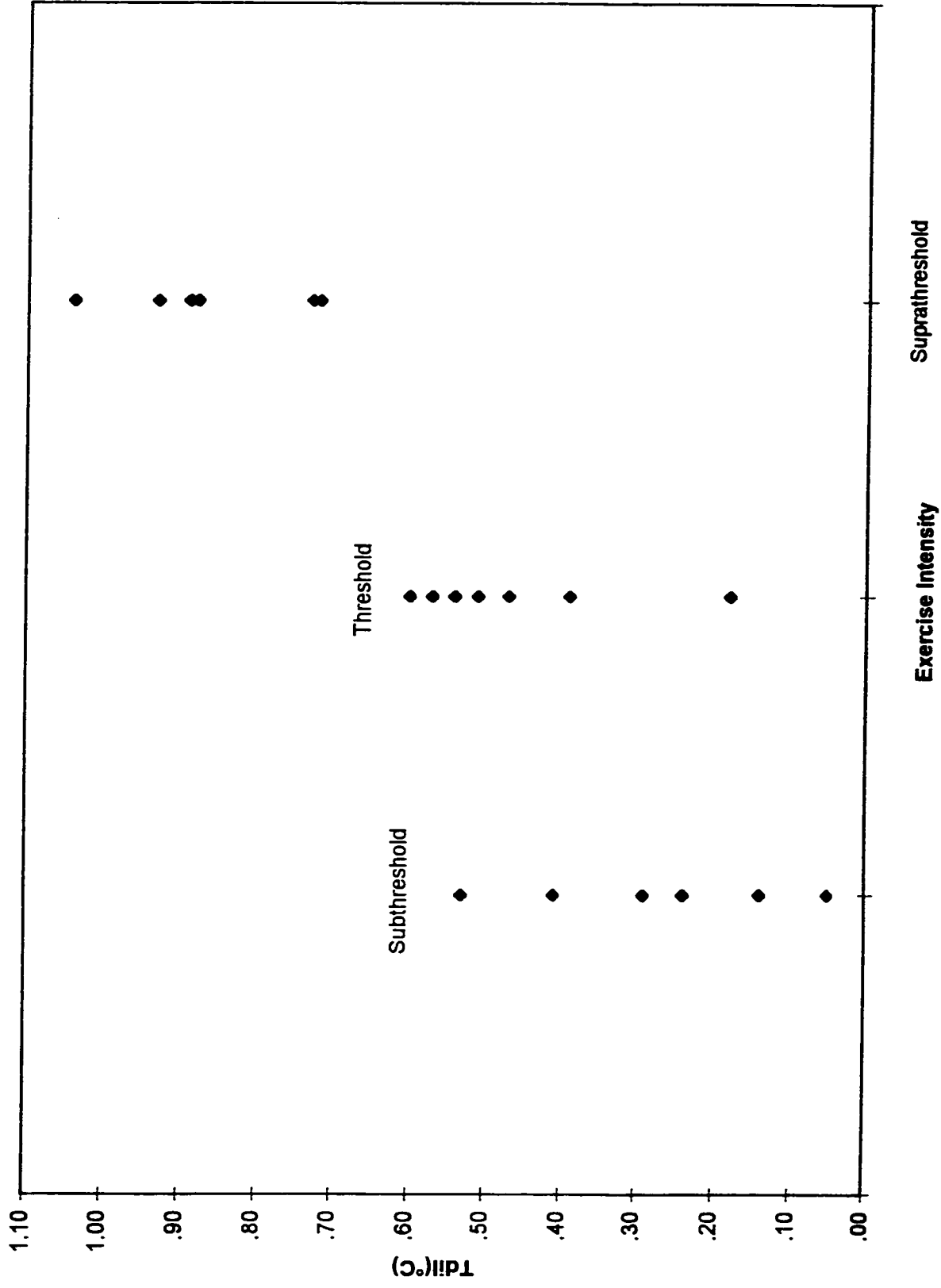


Fig 4.4: Graph of Weighted Mean Tsk(°C) vs. Time (min.) for 3 Exercise Intensities

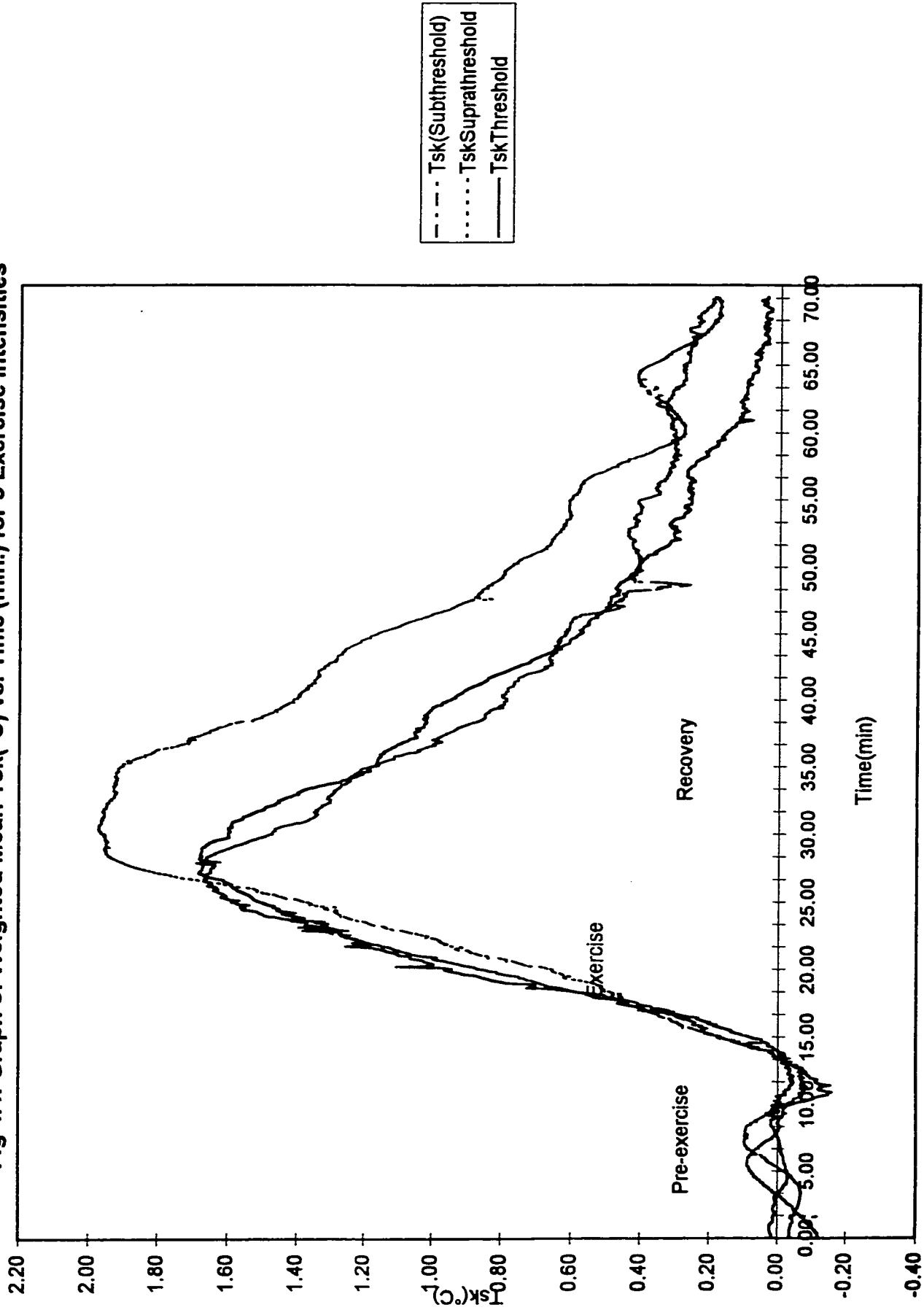


Fig. 4.5: Graph of Mean HR (bpm) vs. Time(min) for 3 Exercise Intensities

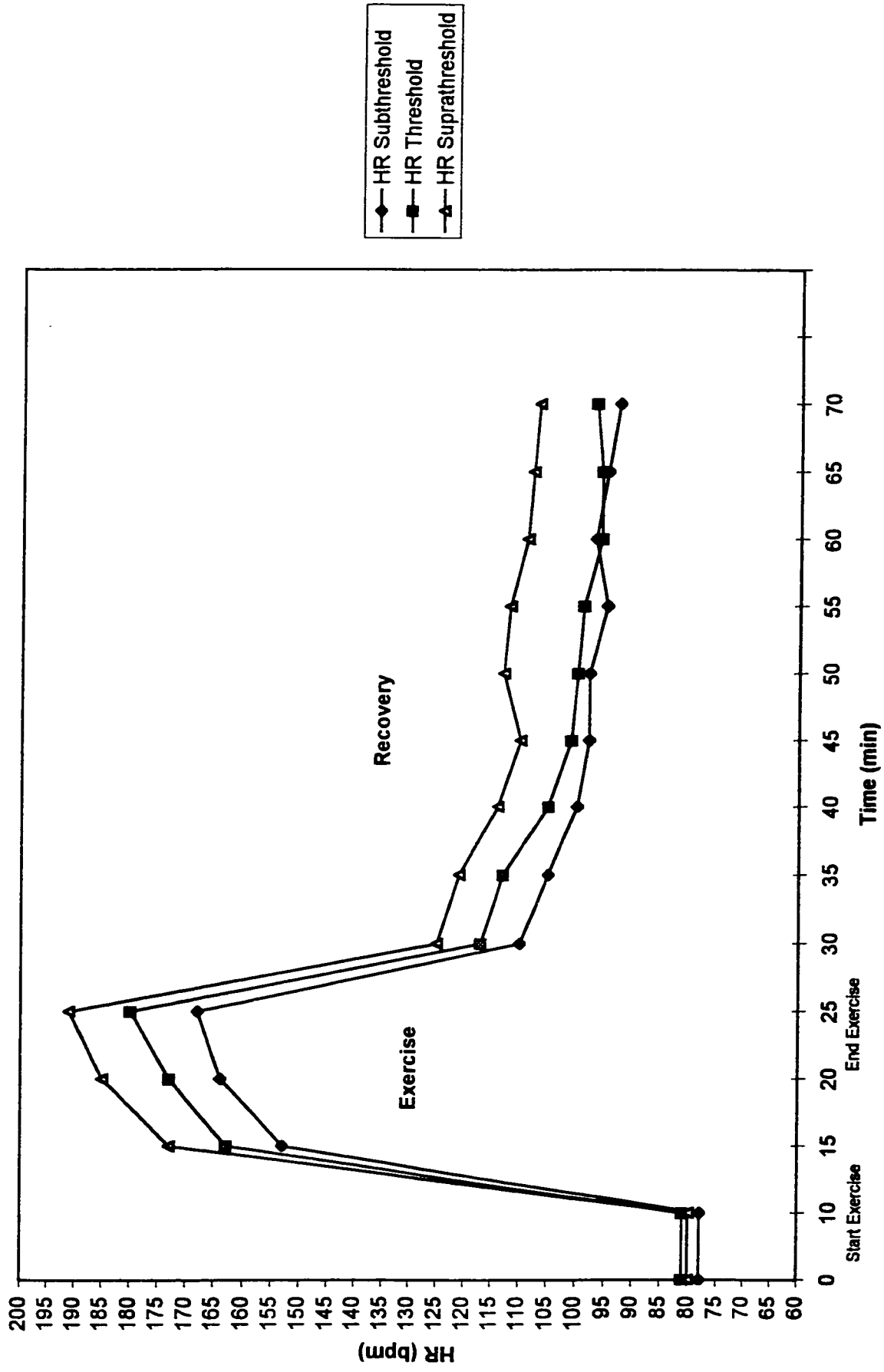


Fig. 4.6: Graph of Oxygen Consumption (ml/kg/min) vs. Time for 3 Exercise Intensities

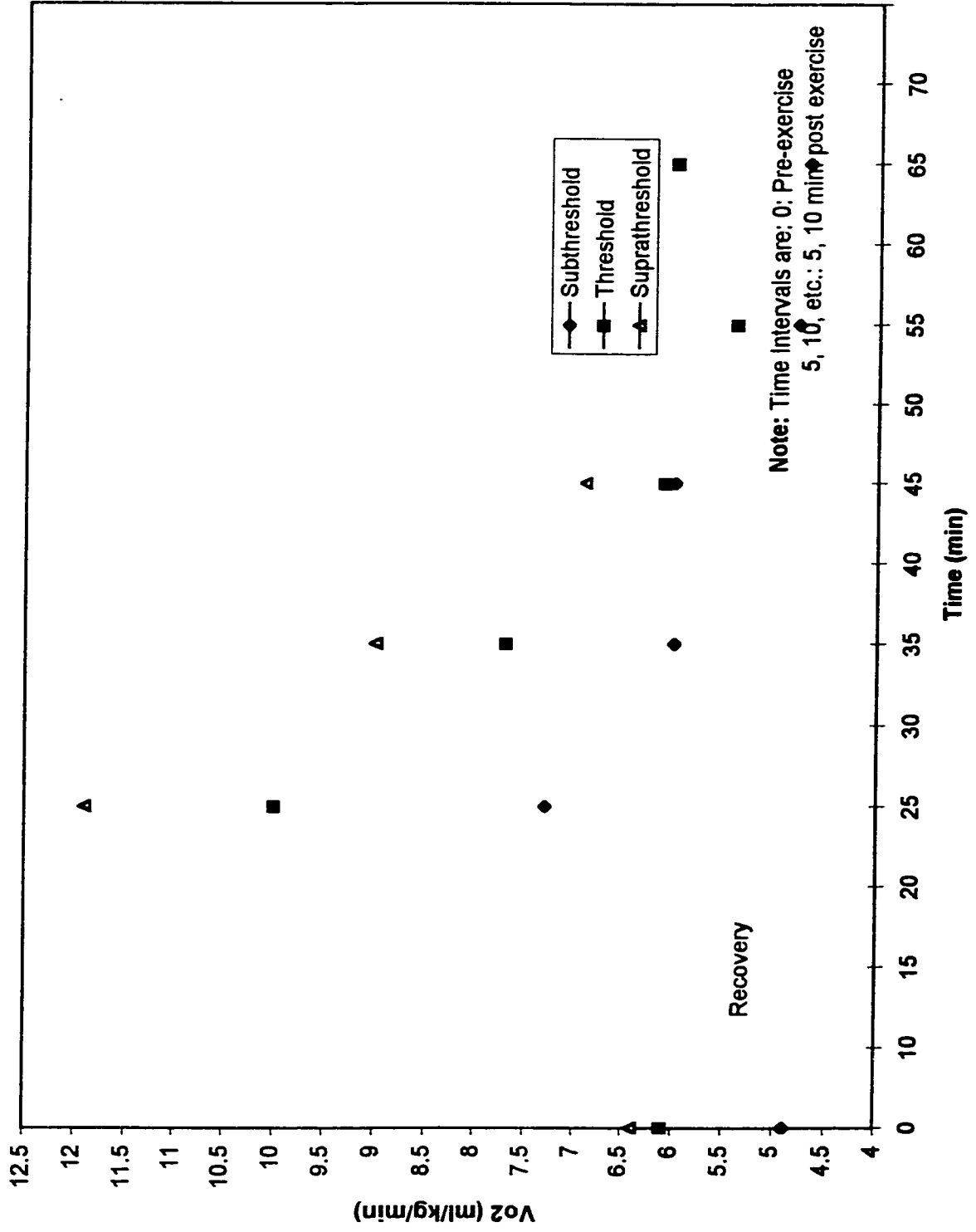


Fig. 4.7: Graph of Systolic Blood Pressure (SBP) and Diastolic Blood Pressure(DBP) (mmHg) vs. Time (min.) for Pre-exercise and Recovery Following 3 Exercise Intensities

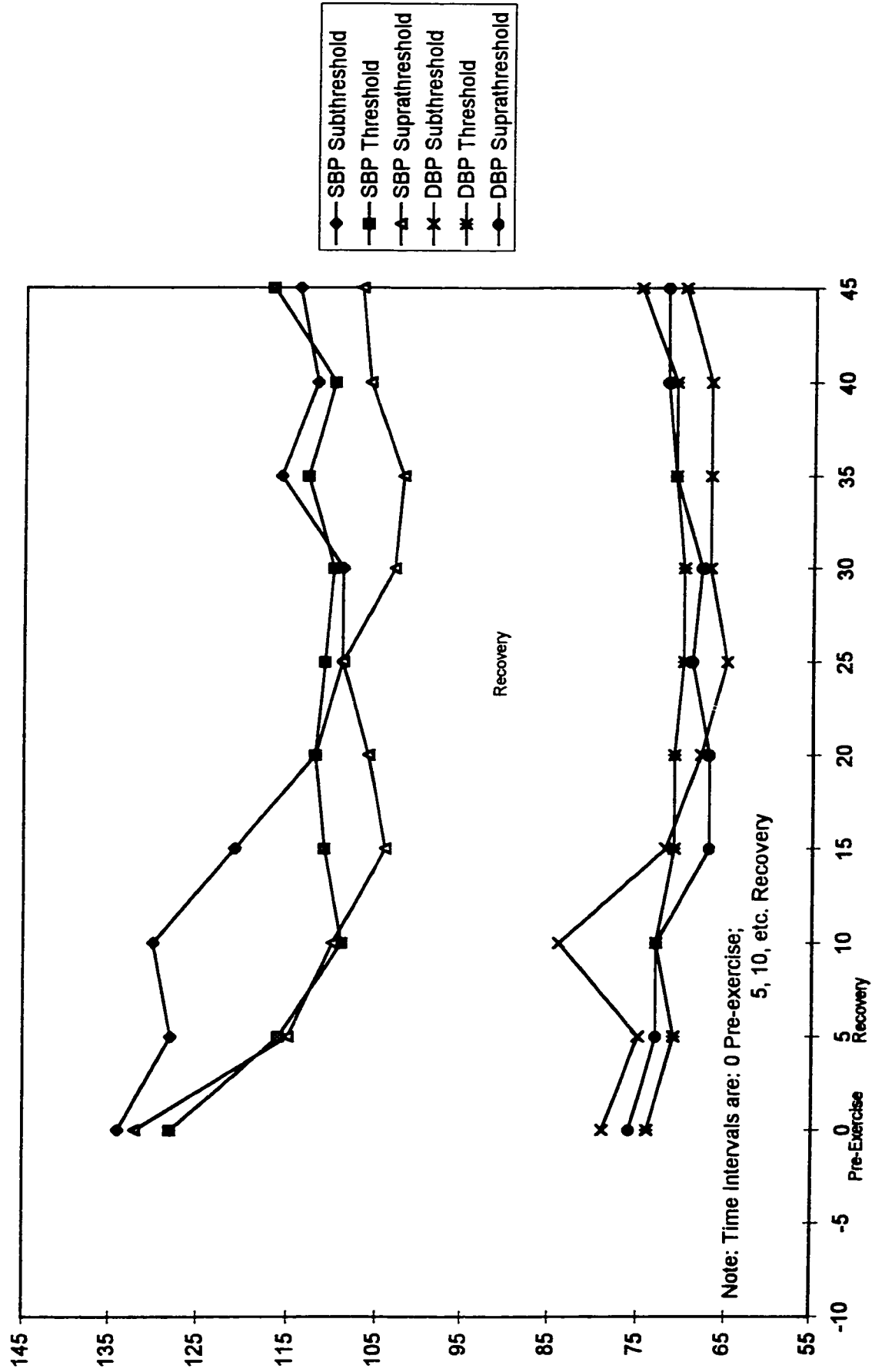


Fig. 4.8: Graph of Mean Arterial Pressure (MAP)(mmHg) vs. Time(min.) for Pre-Exercise and Recovery From 3 Different Exercise Intensities

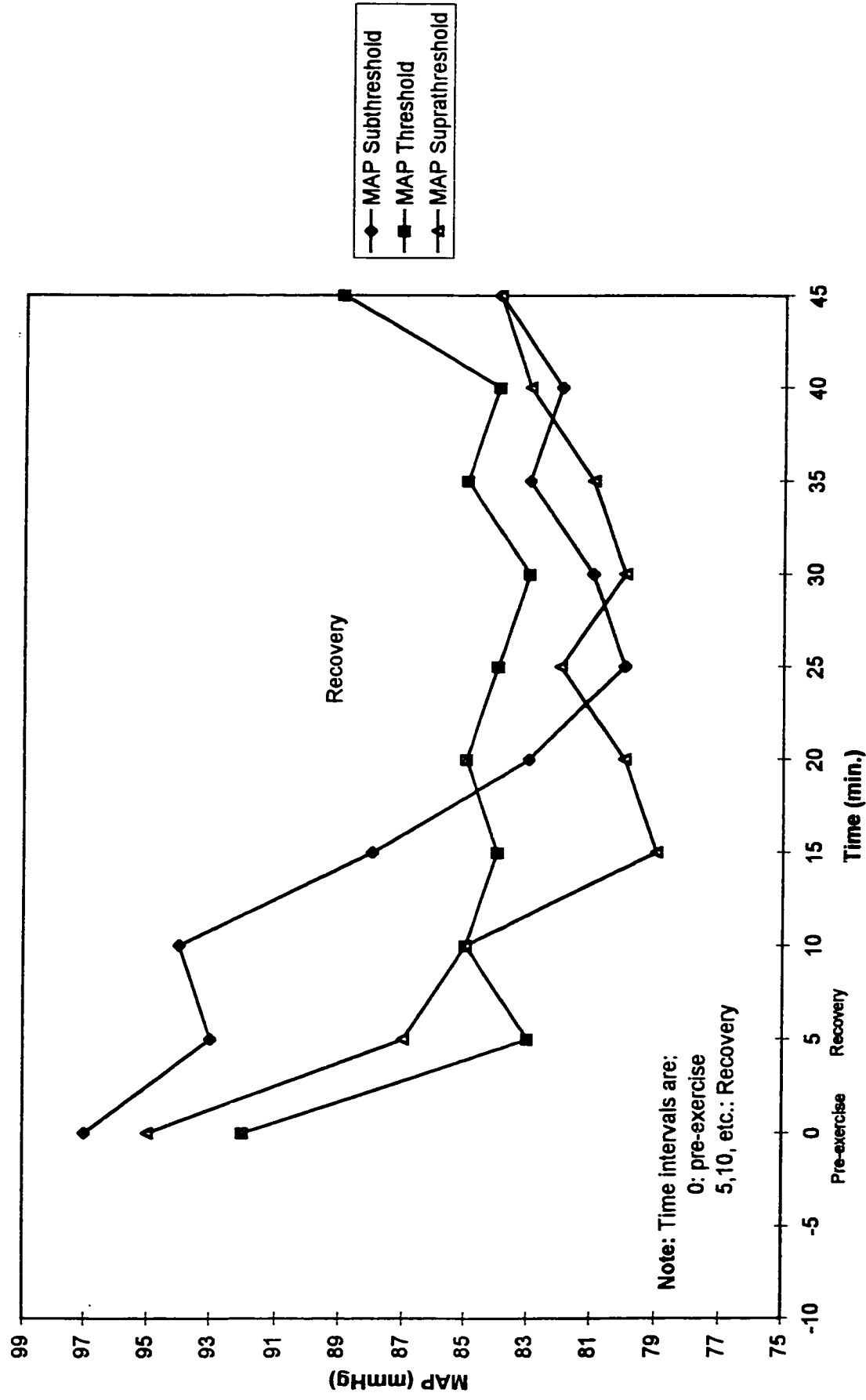


Fig. 4.9: Graph of SkBF vs. Time (min) for 3 Exercise Intensities

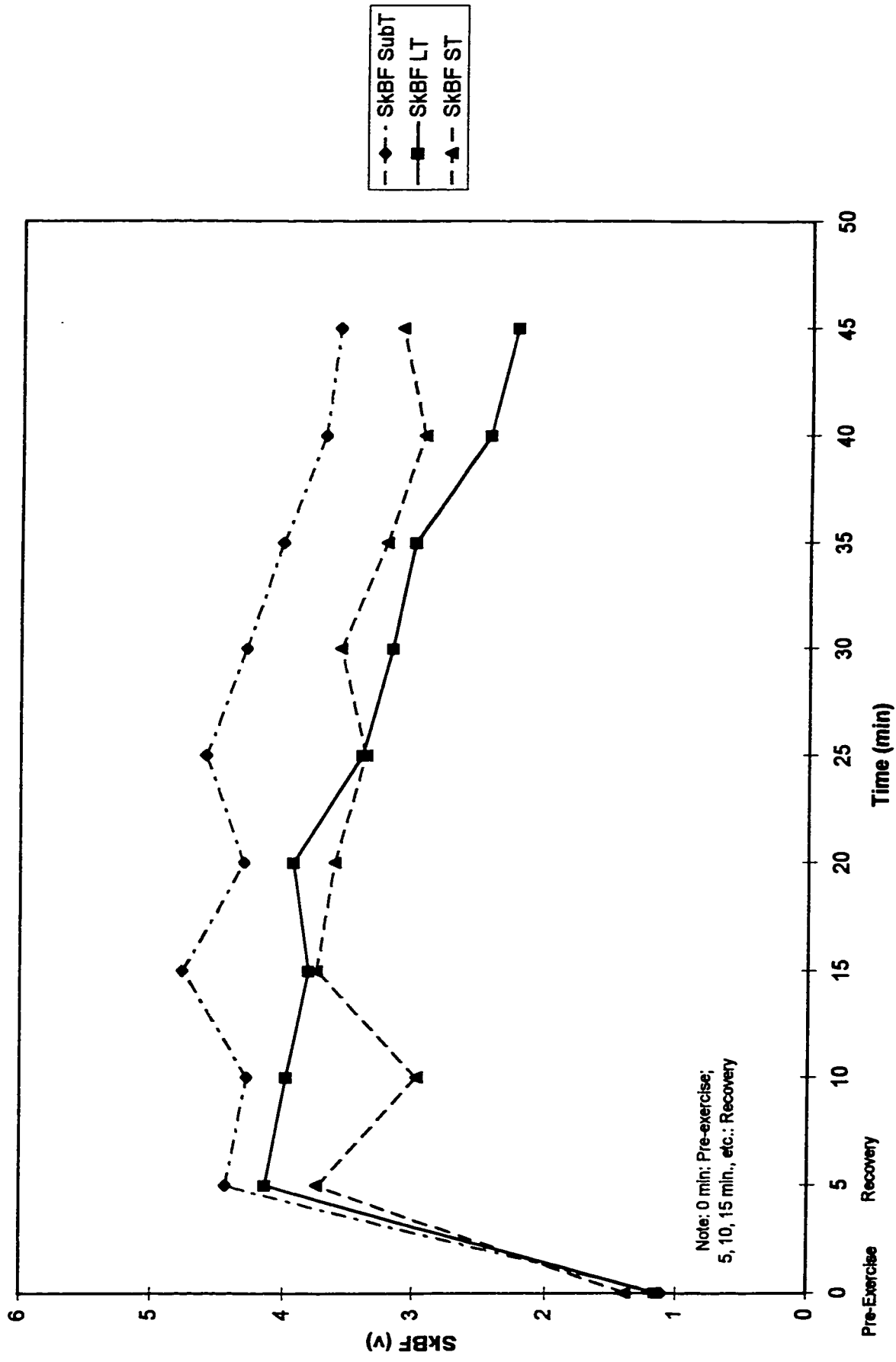


Fig. 4.10: Graph of Delta CVC(v/mmHg) vs. Time (min)

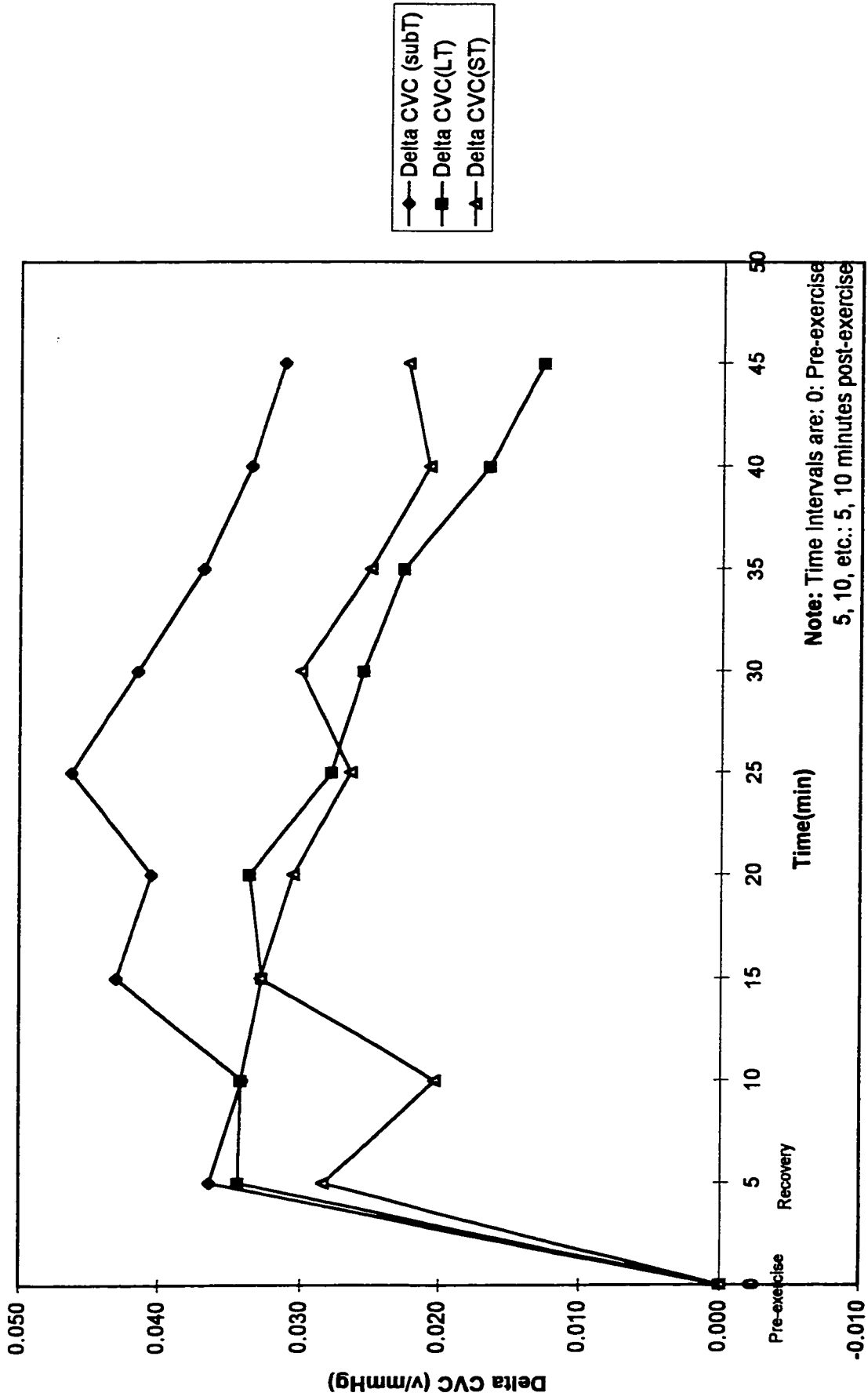
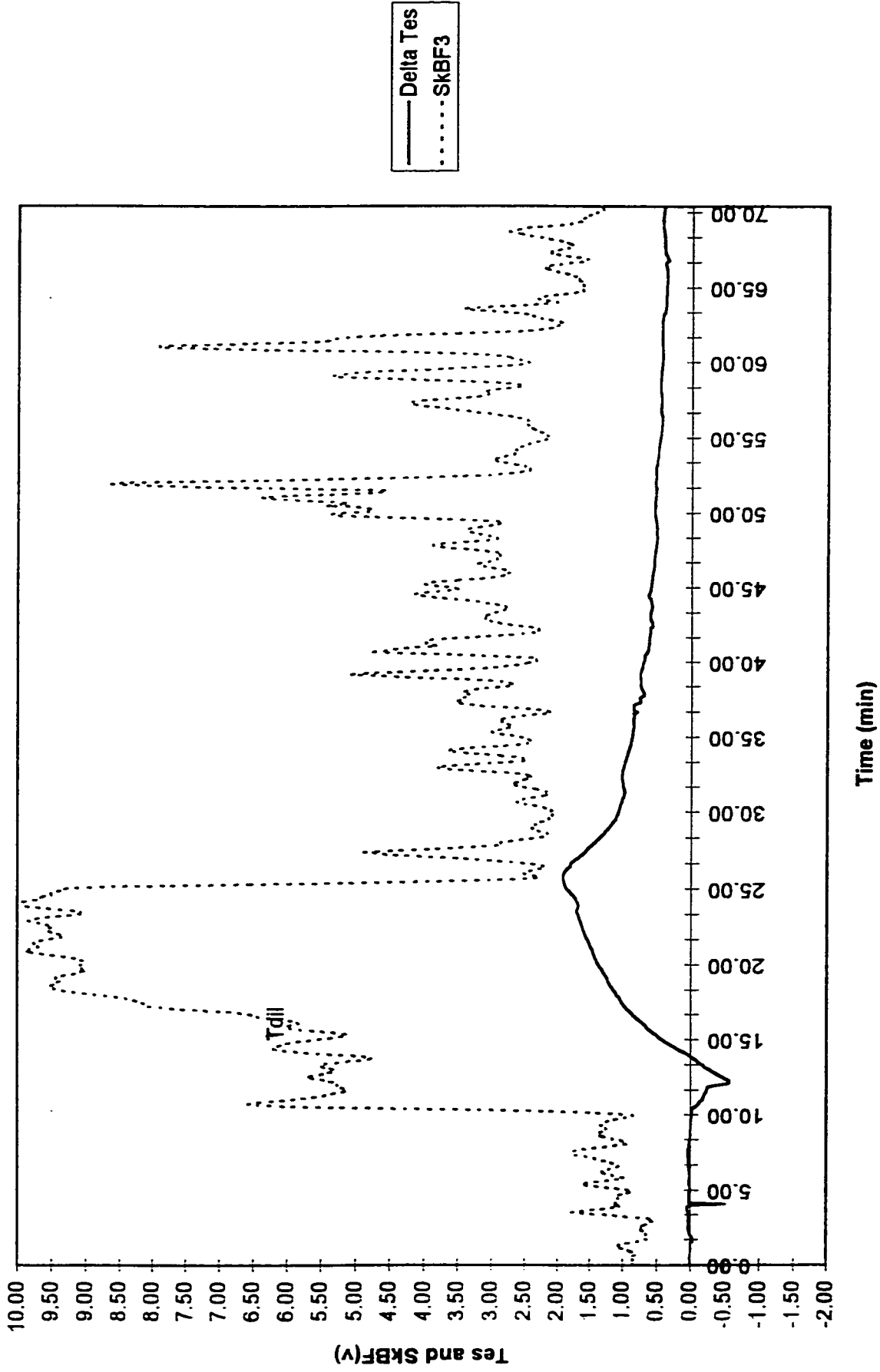


Fig. 4.11: Graph of Tes and SkBF vs. Time (min) for a Single Subject



Chapter 5: Discussion

The purpose of this study was to examine the effects of exercise intensity on the post exercise elevation in oesophageal temperature (ΔT_{es}) and on post exercise thermoregulatory mechanisms. Furthermore, this study investigated the effects of exercise intensity on the relationship between the internal temperature threshold for cutaneous vasodilation (T_{dii}), and the post-exercise elevated plateau in $(\Delta)T_{es}$. Finally, the possibility of the interaction between post exercise blood pressure and the post exercise elevation in ΔT_{es} following 3 different exercise intensities was also investigated. The results from this study have provided new information in the area of thermoregulatory control during, and following exercise, and it might be affected by nonthermoregulatory factors.

The results of this study demonstrate and reconfirm that recovery from dynamic exercise results in a prolonged elevation in ΔT_{es} for 45 minutes of recovery as shown previously by Thoden et al., (1994), Kenny et al., (1997, 1998, 1999), and supported by Johnson and Park (1992). Furthermore, this elevation is intensity dependent, where recovery from running exercise at suprathreshold intensity results in a significantly higher post exercise elevation in ΔT_{es} than both threshold and subthreshold exercise (table 4.2, 4.3, 4.4, fig. 4.1).

In addition, examining the effects of exercise intensity on the relationship between T_{dii} and the established post exercise plateau in ΔT_{es} showed contrasting results to findings of Thoden et al., (1994), and Kenny et al., (1997, 1998, 1999). This study's results demonstrated that even though T_{dii} changed positively as a function of increase in exercise intensity, there was very little correspondence and correlation between T_{dii} and the post exercise elevation in ΔT_{es} across the 3 exercise intensities. This was demonstrated through

both Pearson Correlation and t-tests. Finally, blood pressure results indicated that upright running exercise caused a slight state of hypotension relative to preexercise blood pressure values during recovery from suprathreshold intensities, as indicated by significantly lower MAP, and SBP values from preexercise at certain stages in recovery (tables 4.10, 4.11).

This study identified its intensities as percentages of the subject's ventilatory threshold (due to its correlation with lactate threshold), rather than % $\dot{V}O_{2max}$ to better control for variation in metabolic effects (lactate concentration and accumulation, and other metabolic by-products) among subjects in intensities relative to their maximal oxygen consumption. The sample used for this study had a large variation in the subject's $\dot{V}O_{2max}$, and high-end aerobic fitness. For the purpose of this discussion these intensities were recalculated as a percentage of the subject's maximal oxygen consumption (% $\dot{V}O_{2max}$), (see table 4.1). The means and standard deviations of % $\dot{V}O_{2max}$ were 70.5 (± 4.5)%, 84.2 (± 2.7)%, 93.5 (± 1.9)% for subthreshold, threshold and suprathreshold intensities, respectively. Statistical analysis using a one-way ANOVA, and *post hoc* analysis showed significant differences in workload between the 3 exercise intensities ($p < 0.05$).

5.1: Exercise Intensity and the Post Exercise Elevation in ΔT_{ca}

Following treadmill exercise from all 3 exercise intensities, a prolonged post exercise elevation of ΔT_{ca} was observed for the entire 45 minutes of recovery. The qualitative trends in the ΔT_{ca} profiles during, and following exercise for all 3 intensities followed those documented by Thoden et al. (1994), Kenny et al. (1997, 1998, 1999),

Beraznik (1995). As previously mentioned, results demonstrated significant main effects in condition and exercise ($p=0.00$). Between-subjects effects and within-subjects effects were both significant ($p<0.05$), demonstrating a main effect of exercise intensity on ΔT_{es} . Significant differences in the post exercise elevation in ΔT_{es} were found between suprathreshold exercise ($\Delta T_{\text{es}} = 0.91^{\circ}\text{C}$), and threshold ($\Delta T_{\text{es}} = 0.56^{\circ}\text{C}$), and subthreshold exercise ($\Delta T_{\text{es}} = 0.44^{\circ}\text{C}$); but not between threshold and subthreshold exercise.

Despite the large, and significant difference in ΔT_{es} between the 3 exercise intensities immediately following exercise ($1.02\pm 0.27^{\circ}\text{C}$, $1.53\pm 0.29^{\circ}\text{C}$, and $2.17\pm 0.41^{\circ}\text{C}$), there was little difference in ΔT_{es} between the 3 intensities further into recovery. From 10 minutes into recovery to the end of the 45 minute recovery period, ΔT_{es} was essentially equal between threshold and subthreshold exercise conditions ($p<0.05$) as no significant differences were shown. Furthermore, the mean ΔT_{es} following suprathreshold exercise over the last 20 minutes of recovery was only approximately 0.4°C higher than that of threshold, and 0.5°C higher than subthreshold temperatures.

Graphically (fig 4.1), it appears that these 3 exercise conditions should be significantly different, however the relatively large standard deviation in ΔT_{es} for threshold exercise intensity may have an effect on seeing significant differences between the 3 exercise intensities. Perhaps using more subjects would have demonstrated significant differences among groups in the latter stages of recovery. Furthermore, as will be discussed later, perhaps the range of athlete types in this study may demonstrate better or worse heat dissipation capabilities during and following exercise.

The effects of an increase in exercise intensity on end exercise ΔT_{es} can be partly explained by examining the metabolic cost of increasing exercise intensity and various muscle fibre properties. The relationship between the metabolic cost of exercise and heat production in muscles and in the body is well established (Fox and Bowers, 1993). As metabolic cost due to an increase in exercise intensity increases, the amount of heat produced will also increase. At higher exercise intensities ($\%V_{O_{2max}}$), larger amounts of fast twitch muscle fibres are recruited, which possess stronger contractile properties, and thus have a higher metabolic cost for contraction. Fast twitch/glycolytic (FG) and fast oxidative glycolytic muscle fibres, predominantly used during suprathreshold exercise, generate heat at a much faster rate than slow twitch (slow oxidative) fibres, (Fox and Bowers, 1993). This can account for the differences in end-exercise ΔT_{es} .

During higher exercise intensities when these fibres are recruited, an increase in pressure, and a consequent resistance to muscle blood flow exists due to the rapid contractions of the muscles. During high intensity exercise, & anaerobic processes in the muscle, a higher concentration of local vasodilator substances (K^+ , lactate, Ca^+ , H^+ , etc) appear (Rowell & O'Leary, 1990; Piepoli et al., 1994; Kellogg). Following exercise, local vasodilation due to an increase in thermal stress, as well as accumulation of local dilator substances in the exercising muscle may cause a continued hyperaemia, resulting in a reduction of blood pressure. This possibility will be discussed later.

Findings in ΔT_{es} during and at the termination of exercise in this study (i.e., end exercise ΔT_{es} was intensity dependent) are supported by former studies (Saltin & Hermansen, 1966; Smolander et al., 1991; Saltin et al., 1970, 1972; Kenny et al., 1997). Kenny et al. (1997) used upright running exercise at 45% and 70% of subjects' VO_{2max} , at

different ambient temperatures to determine the combined effects of exercise intensity and ambient temperature on the post exercise ΔT_{es} elevation. These investigators found an incremental effect of exercise intensity on the post exercise elevation in ΔT_{es} . An investigation performed by Smolander et al. (1991) demonstrated an incremental effect of exercise intensity on post exercise ΔT_{es} , which further supports this investigation's findings. At the termination of 15 minutes of cycling exercise at 70%, 80%, and 90% $\dot{V}O_{2\text{max}}$, the mean ΔT_{es} were 37.92 °C (ΔT_{es} 0.92°C), 38.57°C (ΔT_{es} 1.57°C), and 38.93°C (ΔT_{es} 1.93°C), respectively. Results in this study demonstrated significant differences among all 3 exercise intensities until approximately 10 to 15 minutes into recovery, after which subthreshold and threshold ΔT_{es} became and remained similar for the duration of recovery.

Similar to the findings of Thoden et al (1994) and Kenny et al. (1997, 1998, 1999), results from this study demonstrate a prolonged elevation in T_{es} , significantly above preexercise levels. Results from this study, however, demonstrate that an actual plateau in ΔT_{es} was not established, as the mean data demonstrates a continuous decay in ΔT_{es} throughout recovery (Table 4.3, fig. 4.1). In the first 5 minutes of recovery, ΔT_{es} dropped at similar rates among all 3 exercise conditions. However, during the last 35 minutes of recovery, ΔT_{es} following threshold exercise decayed at a significantly faster rate than both subthreshold and suprathreshold exercise ($p < 0.05$). Following a rapid drop in ΔT_{es} immediately following exercise, approximately 10 minutes into recovery, the rate of decrease in ΔT_{es} for all 3 intensities decreased. During the remaining 35 minutes of recovery, the mean rates of temperature change in ΔT_{es} were -0.0032°C/min for

subthreshold exercise, $-0.0165^{\circ}\text{C}/\text{min}^{-1}$ for threshold exercise, and $-0.0042^{\circ}\text{C}/\text{min}^{-1}$ for suprathreshold exercise.

As previously demonstrated (Thoden et al., 1994), the post exercise elevation was not of metabolic origin, as both heart rate and oxygen consumption both returned to preexercise values within 10 to 15 minutes following exercise. However, heart rate recovery following suprathreshold exercise was slower than that of subthreshold and threshold intensities (table 4.8, fig. 4.5) as it remained significantly elevated from preexercise values longer. Perhaps this was in response to the low blood pressure (SBP & MAP) following suprathreshold exercise (tables 4.10, 4.11). The possibility of an increase in vasodilator metabolites (lactate, calcium ions, potassium ions, etc.) in the exercising muscles (Piepoli et al., 1994; Rowell & O'Leary, 1990), may be linked to the significantly lower recovery SBP and MAP during various stages of recovery from suprathreshold exercise.

The relationship between cardiovascular and hemodynamic factors, and postexercise T_{es} , especially suprathreshold intensity, is evident when comparing and contrasting these two sets of results. At the termination of exercise blood pressure fell immediately and rapidly towards preexercise levels. This is likely due to the taking away of sympathetic stimulation and perhaps a high muscle core temperature and vasodilator metabolites contributing to muscle capillary dilation (Piepoli et al., 1994; Rowell & O'Leary, 1990). Systolic and mean arterial pressure, demonstrate a state of hypotension with respect to preexercise values at various stages (15 min for MAP, 15, 30 and 35 for SBP) during recovery from suprathreshold exercise (tables 4.9, 4.10, 4.11, fig. 4.7, 4.8). Recovery from subthreshold and threshold intensities did not show a state of hypotension

with respect to preexercise at any time during the recovery period. Graphically, and statistically, recovery from suprathreshold exercise demonstrates a more rapid decline in blood pressure than subthreshold and threshold exercise (fig 4.8, 4.7, tables 4.10, 4.9). The decrease in blood pressure could be caused by a persistent vasodilation within the capillaries of the exercising leg muscles (Piepoli et al., 1994; Rowell & O'Leary, 1990), especially following suprathreshold exercise. Furthermore, the position of the subject (seated/leaning) may have caused further venous pooling in the legs, thereby reducing the amount of venous return to the core.

Several studies (Piepoli et al., 1993; 1994; and Kilgour et al., 1993) have investigated the effects of exercise intensity on post-exercise hemodynamics (SBP, DBP, MAP, FVC). Similar to this study, Piepoli et al. (1993; 1994), demonstrated a state of hypotension following maximal cycling exercise, comparable to blood pressure levels returning to preexercise levels following minimal and moderate intensities. Post exercise diastolic blood pressure, as well as total peripheral resistance and forearm vascular resistance were all persistently lower than preexercise values for the entire recovery phase. These investigators suggested that normal baroreflex vasoconstriction may have been overcome by vasodilator metabolites, or thermoregulatory needs following maximal exercise.

The previous studies (Piepoli et al., 1993; 1994; and Kilgour et al., 1993), as well as results from this study, may provide insight as to why an extended elevation in post exercise T_{es} occurs, as well as the observation of little, or no significant differences in ΔT_{es} between the subthreshold and threshold exercise intensities during the latter stages of recovery. Following suprathreshold exercise, the stimulus for vasodilation in the exercising

muscles may have been strong enough to overcome a normal baroreflex vasoconstriction response to a body's state of hypotension.

During exercise, the baroreflex is reset due to an increase in muscle sympathetic nervous activity (Rowell & O'leary, 1990). Consequently, vasoconstriction is stimulated by central command to increase blood pressure to compensate for the drive for increased blood pressure. If this occurs during exercise, why then following suprathreshold exercise, is a hypotensive effect observed, and why don't baroreceptors recognize this rapid drop in blood pressure? Perhaps the removal of muscle sympathetic nervous activity is integrated through central command to reset the baroreflex back to resting levels. Along with a subsequent strong vasodilation stimulus following maximal exercise (Piepoli et al., 1994; Kilgour et al., 1991), the added effects of a removal of sympathetic stimulation may be primarily responsible for the observed hypotension following suprathreshold exercise, as well as the lack of heat dissipation. This provides explanation as to why ΔT_{es} following suprathreshold exercise was significantly higher than subthreshold and threshold values.

Perhaps, the post exercise elevated SkBF and CVC are in response to baroreceptor unloading, as the cutaneous active vasodilator system responds to the baroreceptor unloading. Kellogg et al, (1993), (1991) demonstrated that vasodilator systems, especially within the periphery, can participate in baroreceptor reflexes. Vasodilation in exercised muscles and cutaneous vasculature (tables 4.13, 4.14, figures 4.9, 4.10) following exercise may be responsible for the observed hypotension, which may have been strong enough to overcome any stimulus for vasoconstriction (Rowell & O'Leary, 1990; Piepoli et al., 1994).

Skin blood flow (SkBF) & Δ CVC (tables 4. 13 & 4.14, fig. 4.9, 4.10)

demonstrated consistent results with blood pressure among the 3 exercise intensities. Although there may not be any statistically significant differences in SkBF and Δ CVC among the 3 exercise intensities following exercise, graphically, differences are apparent. Again, perhaps the use of more subjects would affect the statistical significance of the graphical differences. Following exercise, SkBF and Δ CVC remain significantly elevated over preexercise levels during recovery from all 3 exercise intensities. The degree of hypotension, and the vasodilator effect appears to be more significant following suprathreshold exercise than following threshold and subthreshold intensities. This is evident through examining Δ CVC and SkBF results, where SkBF and Δ CVC appear to be higher following subthreshold exercise, than threshold and suprathreshold intensities.

The higher SkBF and Δ CVC following subthreshold exercise allows for better heat dissipation, and indicates less of a vasodilation stimulus in the working muscles and systemic circulation, to help direct blood towards the cutaneous vasculature (figures 4.7, 4.8, 4.9, 4.10). The lack of hypotension following subthreshold and threshold exercise intensities allows a greater amount of blood flow to the periphery, and a consequently higher SkBF and Δ CVC than following suprathreshold exercise. The stronger vasodilation stimulus following suprathreshold exercise overcomes the reflex vasoconstriction and allows blood pressure to drop below preexercise values. This slight hypotensive effect following suprathreshold exercise lowers total peripheral resistance and does not allow adequate heat transfer and dissipation through cutaneous circulation, thereby establishing a higher post-exercise elevation in T_{es} than subthreshold exercise (table 4.2, fig. 4.1). This speculation is further supported by examining weighted mean skin temperature during

recovery from exercise as well (fig. 4.3). Perhaps using a larger sample size would help to further support these speculations, and graphical results/trends in SkBF and Δ CVC.

Following all 3 exercise intensities, perhaps another explanation for the elevation in post exercise T_{es} is the effects of thermal stress and local vasodilators in the active muscles following exercise, especially suprathreshold intensity. The return of blood pressure in all three exercise intensities to at least preexercise resting levels (below in some stages of suprathreshold recovery), perhaps prevented a significant portion of central blood volume to be directed towards the cutaneous vasculature. In addition, the combined effects of upright posture following exercise & lack of muscle pump activity in the muscle throughout recovery (Kilgour et al., 1991) doesn't allow an effective transfer of blood, and therefore heat from the exercised muscles to the core and then cutaneous vasculature. Furthermore, the hypotensive effect following suprathreshold exercise may be caused by greater accumulation of blood (venous pooling) in the exercised muscles due to a probable greater heat content and accumulation of local vasodilator substances (Piepoli et al., 1994), as well as the seated/leaning posture (Nishiyasu et al., 1998).

Further study of exercising muscle temperature, and local vasodilator metabolites (Rowell & O'Leary, 1990, Piepoli et al., 1994) following exercise would help confirm this idea. Furthermore, future studies examining the effects of loading or unloading baroreceptors through orthostatic manipulation (LBNP or LBPP) would help better explain the relationship between hypotension following exercise and the observed elevation in T_{es} more effectively.

5.2: Exercise Intensity and the Internal Temperature for Cutaneous Vasodilation

Findings from this study demonstrated that corrected T_{dil} is dependent on upright running exercise intensity as an upward shift in T_{dil} is observed for an increase in exercise intensity (tables 4.5, 4.6). A question arises as to whether the increase in T_{dil} is caused by an increase in vasoconstriction at the onset of exercise, a delay in the vasodilation response during activity or by an increased competition between exercise-induced vasoconstriction and the thermoregulatory-drive for vasodilation. Furthermore, at higher exercise intensities (suprathreshold), does an increase in SkBF indicate T_{dil} , or the removal of an attenuation in SkBF due to the higher exercise intensity. The influence of dynamic exercise on thermoregulatory control of SkBF is due, primarily, to a delay in the onset of active cutaneous vasodilation, which is not dependent on activation of sympathetic vasoconstriction (Mack et. al, 1994, Johnson, 1986, Kellogg et al., 1991).

Studies performed pertaining to internal temperature thresholds for cutaneous dilation by other investigators (Taylor et al., 1988, 1990; Smolander et al., 1991; Mack et al., 1994; Kenny et. al, 1997) demonstrate similar results in the effects of exercise intensity on T_{dil} . Investigators (Taylor et al., 1991) from previous studies have shown that changes in T_{dil} are better correlated with relative workload (% VO_2 , or VO_2 (mL/kg-min), etc.), and relative workload with respect to the exercising muscle group, rather than absolute exercise intensity.

Similar to this investigation's findings, Kenny et. al (1997), demonstrated a graded increase in T_{dil} with an increase in workload in treadmill running exercise, further supporting this study. However, these investigators also examined the effects of ambient

temperature, combined with the change of intensity, on T_{dil} , and never made any direct comparisons between exercise intensities at consistent ambient temperatures. Thus, it is difficult to draw comparisons with this present study since this investigation used only one ambient temperature, and three exercise intensities.

Investigations performed by other researchers (Taylor et al. 1988, & Smolander et al. 1991, Mack et al., 1994) demonstrated that exercise intensity affects cutaneous vascular conductance in a graded fashion, and subsequently increases T_{dil} . Taylor et al. (1988) used two-legged and one-legged exercise on a modified bicycle ergometer to study the responses of cardiovascular conductance (CVC) to dynamic exercise. Two-legged dynamic exercise demonstrated that CVC at the initiation of exercise decreases, in a graded manner relative to the absolute workload. Forearm vascular conductance (FVC), however, increased during the first minute of exercise, and also showed a graded effect as FVC was higher in the higher intensity than the lower. This same effect was also observed in graded isometric forearm exercise by other investigators (Vissing, 1998). This increase in FVC at the initiation of exercise was found to be a function of the increase of blood pressure or flow within the forearm muscle only (Taylor et al., 1988). These observations support this investigation's findings of an initial slight increase in SkBF at the initiation of exercise.

Consistent and contrasting results to this study were found by Smolander et al (1991), where during upright cycling exercise, skin blood flow was not significantly influenced by exercise intensity between 50-80% VO_{2max} . Subjects in this study showed a significantly higher increase in T_{cs} during 70% VO_{2max} intensity exercise than at rest,

before T_{dil} was reached. This demonstrated an attenuation in the increase in skin blood flow. However, the subject's skin blood flow response (Smolander et al. 1991) was significantly attenuated at high relative intensities, similar to the findings in this study where the T_{dil} at 84% and 93% of the subjects VO_{2max} were significantly elevated from resting levels. These investigators found the critical intensity for an increase in SkBF to be greater than 80% of the subject's VO_{2max} (Smolander et al., 1991).

Findings from this study, and results from others (Vissing, 1998, Smolander et al., 1991,) provide support to the idea that the delayed vasodilator response, and higher T_{dil} may be due to an increase in vasoconstrictor activity in skin blood vessels during higher exercise intensities. However, it has been shown that an increase in SkBF during exercise is due to an increase in the active vasodilatory response (Kellogg et al., 1991), where at rest, whole body heating increases SkBF by decreasing active vasoconstriction (Pergola et al., 1993). The most significant finding in this study to support this idea is the significant difference between the times at which T_{dil} or the dilation response is reached between subthreshold, threshold and suprathreshold exercise (Table 4.5). Results show that the time into exercise at which the dilation response occurs increases as a function of an increase in exercise intensity. These results demonstrate that an attenuation in T_{dil} or the dilator response occurs as exercise intensity increases, which differs from Kenny et al. (1997), but support findings by other investigators (Kellogg et al. 1991, Smolander et al., 1991, and Vissing 1998).

As previously mentioned, Smolander et al. (1991) found that the SkBF response to dynamic exercise was significantly attenuated at higher exercise intensities (>80% VO_{2max}),

or T_{dil} was not observed in 3 out of 4 subjects (Taylor et al., 1988). These investigators speculated that vasoconstriction in the skin blood vessels persists throughout exercise until T_{ex} is finally high enough to elicit an increase in SkBF due to the vasodilator response. This is further supported by other investigators (Kellogg et al., 1991, Vissing, 1998) who treated skin blood vessels to block sympathetic nervous activity. They concluded that the delay in T_{dil} is due to an increase in vasoconstrictor activity at the onset of exercise, and that exercise affects T_{dil} via the active vasodilator system. They also concluded that the active cutaneous vasodilator system is directly affected by reflex adjustments to dynamic exercise, as it is by baroreflexes during orthostasis, or other changes in blood pressure. The question of how this integration between the active vasodilator system and the vasoconstrictor reflex to exercise needs to be answered.

The first reflex effect of exercise is an initial vasoconstriction regulated via the vasoconstrictor system (Kellogg et al., 1991; Vissing, 1998; Smolander, 1991; Kenney & Johnson, 1992; Rowell & O'Leary, 1990). The reflex effects of the initiation of exercise occurs either within the central or peripheral nervous system, but not at the arteriolar level. This initial reflex is regulated via central command (within the hypothalamus). Recent studies by Vissing (1998) demonstrated that sympathetic nervous system activity (as measured using microneurographic techniques during static exercise) within the cutaneous circulation increases at the initiation of exercise. Furthermore, sympathetic nervous system activity persists longer as exercise intensity increases. Evidence that this reflex is integrated by central command (hypothalamus) was demonstrated in this study as sympathetic nervous activity began before, rather than after the onset of muscle tension

(i.e., the anticipation of exercise) and further increased, instead of decreased, during sustained contraction (Vissing, 1998; Ebert, 1986). The pattern of sympathetic activation to skin produced by exercise suggests that the sympathetic activity is controlled by central command. Although these observations were demonstrated only during static exercise by Vissing (1998) and Ebert (1986) speculations can be made about reflexes during dynamic exercise.

As previously mentioned, investigators found that the initiation of exercise caused a cutaneous vasoconstriction due to an increase in vasoconstrictor activity (Rowell et al., 1990; Kellogg et al., 1991, Kenney & Johnson, 1992. Proof that the active vasodilator system controls the vasodilation response to T_{dil} during exercise is demonstrated indirectly through this study through a sustained increase in $SkBF$ by subjects at their T_{dil} (see fig. 4.11), and directly through others through isolation of certain nerve types (Kellogg et al., 1991; 1993). The delay or increase in T_{dil} during higher exercise intensities in this study is due to an increase in sympathetic activity in the cutaneous circulation, thereby attenuating the vasodilation response, and increasing the core temperature at which the active cutaneous vasodilator system can overcome the initial vasoconstriction. Integration of the reflex vasoconstriction effects of exercise with thermoregulatory reflexes occurs within the hypothalamus and central command of central nervous system (Kellogg et al., 1991).

5.3: Correlation Between T_{dil} and Post-Exercise T_{es}

Another major finding in this study is that these results demonstrated that T_{dil} and the post-exercise ΔT_{es} plateau were not linearly related and were poorly correlated, $r=0.67$ ($p<0.05$). Furthermore, ΔT_{es} was also significantly different from T_{dil} . These findings add argument to findings by Thoden et al (1994), and Kenny et al. (1999) who found a significant correlation or relationship between T_{dil} and post exercise T_{es} at moderate exercise (Thoden et al., 1994), and at varying intensities (45% and 70% $VO_{2,max}$) and ambient temperatures (Kenny et al., 1997).

The observation of a poor correlation between T_{dil} and post-exercise T_{es} may first be explained by the small sample number used in this study. Graphically (Fig. 4.1) there appears to be a relationship between T_{dil} , and the post-exercise elevation in T_{es} . However, the low correlation between T_{dil} and post exercise T_{es} may be observed due to the small sample of subjects. Perhaps with more subjects, this correlation may be greater.

As previously mentioned, in contrast to investigations performed by Kenny et al. (1997, 1999) and Thoden et al. (1994), this study observed a slow, continuous decaying T_{es} throughout the recovery phase of the experiment (tables 4.2, 4.3), rather than an established plateau. In previous investigations (Thoden et al., 1994 and Kenny et al., 1997) a post-exercise plateau in T_{es} was observed for the entire 45 minutes of recovery. Furthermore, T_{es} following exercise at higher intensities in this study (especially 80% and 90% $VO_{2,max}$) saw a more rapid decay. This constant decay in T_{es} following exercise would cause difficulty in finding a correlation between T_{dil} and a post exercise plateau T_{es} .

As previously mentioned, one of the differences between this study and the study performed by Kenny et al. (1997) was the combination of exercise intensity and ambient

temperature used in the latter. In that study, the differences in T_{dil} between intensities were never compared among the same ambient temperature. Furthermore, the exercise intensities in this particular study, 70%, 84%, and 93% VO_{2max} , were equal to or higher than the heavy exercise intensity (70% VO_{2max}) used by Kenny et al. (1997).

The fact that the correlation between T_{dil} and post exercise T_{ca} has been demonstrated in 4 previous studies (Thoden et al., 1994; Kenny et al., 1997, 1998, 1999), provides evidence that this relationship is not coincidental but physiological at moderate exercise intensities. Perhaps at those lower intensities (45% & 70% VO_{2max}), this correlation remains intact until a certain exercise intensity threshold is reached where the vasoconstrictive drive is significantly higher. Further analysis of ΔT_{ca} against T_{dil} in this study showed that there was no significant difference between ΔT_{ca} and T_{dil} at subthreshold exercise intensity ($p < 0.05$), demonstrating a relationship between them, therefore supporting studies by Kenny (1997, 1998, 1999) and Thoden (1994).

The higher vasoconstrictive drive associated with threshold and suprathreshold exercise intensities in this study would cause a significant attenuation in SkBF and subsequent higher core temperature at which the vasodilation response occurs. Studies by Smolander (1991) and Taylor (1988) support this as they didn't observe dilation, or observed a significant attenuation in the cutaneous vasodilation response at exercise intensities higher than 80% VO_{2max} . This attenuation in SkBF due to greater vasoconstrictor activity at higher exercise intensities in this study can account for the poor physiological relationship between T_{dil} and the elevation in ΔT_{ca} following exercise.

This study used a subject group with a fairly wide range of maximal oxygen consumption values (table 4.1). Some of the subjects used in this study were highly trained

in aerobic sports such as mountain biking, distance running, and cross-country skiing while others were body builders. Perhaps differences in plasma volume among aerobic and strength athletes may be able to explain this inconsistency in the values observed for T_{dil} and post exercise T_{es} , especially at the 2 higher exercise intensities. Mack et al. (1994) found differences in T_{dil} between subjects with significantly higher and lower (induced hypovolemia) plasma volumes. This study found that subjects with a higher plasma volume reached T_{dil} at a significantly lower T_{es} than those with acute hypovolemia T_{dil} , demonstrating that T_{dil} and plasma volume were inversely related. Other researchers (Yoshida 1997, Johnson, 1998 and Thomas, 1999) have also demonstrated that higher aerobically fit athletes have a higher plasma volume, which allows T_{dil} to occur at a lower core temperature, which remains intact over a range of exercise intensities. Johnson (1998) and Thomas (1999) suggest that the lowered T_{dil} is caused through the effect of training on the vasodilator system. Studies have shown and are established that a training effect by aerobic training causes an increase in blood and plasma volume (Mier et al., 1996; Shi et al., 1996). These 2 facts can explain some differences among subjects and the nonlinear relationship between T_{dil} and post-exercise T_{es} at higher exercise intensities in this particular study.

Lastly, perhaps some subjects in this study may not have been properly hydrated, even though an attempt was made to control for this in the morning through asking subjects to drink at least 500 mL of liquid prior to the test. Perhaps this method should be altered and level of hydration controlled for earlier. Having subjects follow a hydration protocol earlier than 3 hours prior to the test might further minimize any added effects of dehydration prior to the test on the subject.

5.4 Mechanisms Behind the Post Exercise Elevation in T_{re}

Results from this study provide a connection between nonthermoregulatory and thermoregulatory control of blood flow. As with the onset of exercise, recovery from exercise also induces a competition for blood flow between thermoregulatory needs and control, and nonthermoregulatory needs and control. Recovery hemodynamic data (blood pressure, cardiovascular conductance) following suprathreshold exercise magnifies the mechanism behind the elevation in T_{re} following exercise. Eventhough blood pressure following subthreshold and threshold exercise was not significantly lower than preexercise values, graphically (fig. 4.7, 4.8) it appears to be.

Immediately following exercise, a rapid decrease in blood pressure is observed, which induces a state of hypotension relative to preexercise values (following suprathreshold exercise). Due to the increased heat in the muscle, other local vasodilator substances (lactate, K^+ ions, etc) (Piepoli et al, 1994, Rowell and O'Leary, 1990) as well as posture (Nishiyasu, 1998), dilation is occurring in the muscle vasculature. The lack of muscle pump action and venous return causes venous pooling in the working muscles in the legs during recovery. The slower recovering T_{th} (Thoden et al, 1994) demonstrates a slow transfer of heat from the working muscles.

The lack of active muscle pump and venous return from the legs decreases the amount of central blood volume. As a result of the venous pooling, central blood volume decreases causing blood pressure to decrease as well. The result of this is a decreased flow to the periphery to dissipate heat through dilation. Even if the baroreceptors sense the drop in blood pressure, the amount of vasculature in the worked leg muscles exceeds that

of the cutaneous vasculature in the forearms. There is likely not enough blood flow and blood pressure in the periphery to redistribute the blood flow and increase blood pressure compared to the pooling in the legs. In this competition for blood flow, the dilated vasculature in the legs due to an increase in heat, vasodilator substances, as well as posture overcomes the cutaneous vasculature ability to constrict in an attempt to increase blood pressure.

Chapter 6: Conclusion

This study examined the effects of upright running exercise intensity on the post-exercise elevation of T_{es} . In addition, the effects of exercise intensity on T_{dil} , and the relationship between T_{dil} and the post-exercise established elevation in T_{es} were also examined. Finally, the relationship between blood pressure, $SkBF$ and CVC during recovery and the elevation in T_{es} was also explored. This study provides further evidence that exercise intensity has a graded influence on post-exercise thermoregulatory control. Further more, this study expands observations to a higher range of exercise intensities ($>70\% VO_{2max}$) and attempts to draw more of a relationship between nonthermoregulatory control and thermoregulatory control of blood flow during exercise and recovery from exercise.

Results of this study demonstrated a graded increase in post exercise T_{es} , with an increase in exercise intensity. This elevation never subsided to preexercise levels for the duration of 45 minutes of recovery. This elevation in recovery T_{es} and the differences in T_{es} between the 3 exercise intensities can be explained through post-exercise hemodynamic responses. Local vasodilation within the exercising muscles perhaps overcame any baroreflex vasoconstriction response, thereby causing an observed state of hypotension that occurred at stages of the recovery period following suprathreshold exercise. The graphical but not statistically significant differences in $SkBF$ and ΔCVC perhaps provide further explanation as to why a difference in post exercise T_{es} occurs among the 3 exercise intensities. The lack of hypotension following subthreshold and threshold exercise allows for better heat dissipation through a higher $SkBF$ and ΔCVC .

As previously stated, a larger sample size may demonstrate more significant differences among the 3 exercise intensities in blood pressure, SkBF and CVC, providing further support to these possible mechanisms behind post-exercise thermoregulatory control. Furthermore, future studies could also examine the effects of loading the baroreceptors through orthostatic manipulation during the recovery period. This would provide further evidence about the relationship between cardiovascular responses following exercise, and the elevation in T_{es} .

This study found that T_{dil} was dependent on exercise intensity, which is supported by other investigations (Taylor et al. 1988, & Smolander et. al, 1991 and Mack et al., 1994). It would appear that the delay, or increase in T_{dil} during higher exercise intensities is due to an increase in sympathetic activity in the cutaneous circulation at the onset of exercise, and thus an attenuation in the vasodilation response. This increases the core temperature at which the cutaneous vasodilator system can overcome the initial vasoconstriction.

In contrast to studies performed by Kenny et al. (1997, 1998, 1999) and Thoden et al. (1994) T_{dil} and post exercise T_{es} were poorly correlated throughout the average of the 3 exercise intensities. However, no significant difference was found between post-exercise ΔT_{es} and T_{dil} for subthreshold exercise, suggesting a relationship at this intensity. The poor correlation between T_{dil} and post-exercise T_{es} demonstrated in this study may be the result of the higher exercise intensities causing a significant attenuation in the vasodilation response. In addition, the wide variation in aerobic fitness of the subjects as well as possible effects of plasma volume associated with aerobic fitness and dehydration may have also affected these results. Future studies of the effects of exercise on T_{dil} should

closely investigate blood pressure changes at the onset of exercise as well as at T_{dil} , along with SkBF and CVC to further investigate the on-going competition for blood flow between non-thermoregulatory and thermoregulatory needs.

APPENDICES

Appendix 1: Subject Consent Forms and Information

Investigator: Peter Niedre
Faculty of Health Sciences
School of Human Kinetics
125 University Dr., Rm. 307A
Ottawa, Ont. K1N 6N5
Tel.: 562-9739(H); 562-5800 ext.4244 (lab)

Advisor: Dr.J. Thoden
Faculty of Health Sciences
School of Human Kinetics
125 University Dr.
Ottawa, Ont. K1N 6N5
Tel.: 562-5800 ext. 4283

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

The purpose of this study is to investigate the effects of dynamic exercise intensity on body temperature regulation.

I have read and understood the information presented in the letter of information. I understand that I will be asked to participate in one preliminary and three experimental sessions requiring treadmill running for a duration of twenty minutes. During these tests, I will be connected to skin and esophageal temperature probes, a blood flow probe, heartrate monitor and blood pressure cuff. I understand the minor risks and discomforts associated with the study due to the intensity of exercise, minor irritations associated with the inserted esophageal probe, and general temporary discomfort.

I understand that the anonymity of my data will be maintained at all times. Access to the data will be restricted to the principal investigator and the research advisor. Data will be presented in pooled form, and identified by a specific code.

Finally, I understand that I may withdraw or refuse participation in the study at any time without prejudice or discrimination of any form.

Date: _____ Volunteering Subject: _____

Signature of Volunteer: _____

Signature of Witness: _____ Date: _____

Appendix 1 Continued

Investigator: Peter Niedre
Faculty of Health Sciences
School of Human Kinetics
125 University Dr., Rm. 307A
Ottawa, Ont. K1N 6N5
Tel.: 562-9739(H); 562-5800 ext.4244 (lab)

Advisor: Dr.J. Thoden
Faculty of Health Sciences
School of Human Kinetics
125 University Dr.
Ottawa, Ont. K1N 6N5
Tel.: 562-5800 ext. 4283

This study involves human research and is submitted in partial fulfillment of the degree of masters of arts in movement studies.

The purpose of this research is to investigate the effects of dynamic exercise intensity on body temperature regulation.

As a subject, you will be asked to participate in four sessions, one preliminary or orientation session, and three experimental sessions to be conducted on four separate days.

The preliminary session involves your participation for approximately one hour. During this time, the procedures and technical equipment (skin and esophageal temperature probes, blood flow monitor, heartrate monitor, Q-plex and electrophygomanometer) involved in conducting the experimental sessions will be familiarized to you, and a Physical Activity Readiness Questionnaire (Par-Q) will be answered and signed. Following this orientation, you will be asked to perform an incremental maximal aerobic power test on a running treadmill during which a constant running speed will be kept as the gradient of the treadmill is increased every two minutes until an intensity is reached you can no longer maintain.

The experimental sessions will consist of treadmill running at an ambient temperature of 29° C at three different intensities determined as percentages of your maximal oxygen consumption. These intensities will be 50% (minimal), 70-75% (moderate) of your maximal aerobic power, and a gradual increase (over 15 minutes) up to approximately 90% (maximal) of your maximal aerobic power, which will be maintained for an additional 5 minutes. During these tests you will be connected to skin thermistors (applied at the left forearm, index finger, chest, thigh, calf and back), an esophageal temperature probe, a non-invasive blood flow probe, blood pressure cuff, and heartrate monitor.

There are some minor physical risks associated that you should be made aware of. The study involves performing treadmill running exercise at various intensities. 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 heart or vascular problems. 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 own sterile probe. 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.

There are some minor physical discomforts associated with this study that you should also be aware of. The esophageal probe may cause some minor irritation or discomfort in the nasal passage and throat during, and one or two days following the experimental sessions. Finally, as associated with performing any form of exercise, you may experience some minor muscle soreness for a couple of days following the experimental sessions. Every effort will be made to ensure that these tests are conducted so as to minimize any of these sources of discomfort.

The present study is being conducted to help the scientific community develop a better understanding of body temperature control mechanisms and how this is related to exercise intensity.

Your anonymity in participating in this study is ensured as data is never associated with your name, but with a code. All data will be presented in pooled form, and all raw collected data will be stored in computer memory with specific access codes. Access to the data is restricted to the investigator (myself) and my research advisor (Dr.J. Thoden); and you are welcome to request and discuss the results at any time with myself.

For the entire duration of the four testing days, it is fully understood that you may refuse or withdraw from participation from the study at any time, for any reason, without question or prejudice.

If you have any further questions regarding the nature or protocol of this study, please feel free to contact me, Peter Niedre, at 562-9739(H), or 562-5800 ext.4244(Lab), my research advisor, Dr. Jim Thoden at 562-5800 ext.4283; or, the chair of the human research ethics committee, Dr. R. Proulx at 562-5800 ext. 4251.

References

- Bazett, H. C. (1951). Theory of reflex controls to explain regulation of body temperature at rest and during exercise. Journal of Applied Physiology 4: 245 - 262.
- Crandall CG; Johnson JM; Kosiba WA; Kellogg DL (1996). Baroreceptor control of the cutaneous active vasodilator system. Journal of Applied Physiology 81(5): 2192 - 2198.
- Ebbecke, U. (1917). In: Webb, P. (1995). Physiology of Heat Regulation. Regulatory Integrative and Comprehensive Physiology 37: R838-R850.
- Ebert, T. (1986). Baroreflex responsiveness is maintained during isometric exercise in humans. Journal of Applied Physiology 61: 797-803.
- Franke, W.D.; Hickey, M.S.; Ward, C.W.; Davy, K.P. (1993). Effects of α_1 - receptor blockade on the cardiovascular and thermoregulatory responses to severe exercise in the heat. Journal of Sports Medicine and Physical Fitness 33: 146-151.
- Guyton A.C. (1991) Textbook of Medical Physiology. W.B. Saunders Company: Philadelphia PA; pp797-807.
- Hobbs, S.F.(1982). Central command during exercise: parallel activation of the cardiovascular and motor systems by descending command signals. Circulation, Neurobiology, and Behaviour: 217-231.
- Ivanov, K.P. (1990). The location and function of different skin thermoreceptors. In: Webb, P. (1995). Physiology of Heat Regulation. Regulatory Integrative and Comprehensive Physiology 37: R838-R850.
- Ivanov, K.P.; Dymnikova, L.; Danilova, N. (1987). The hypothalamus neuron's response to the signals from surface and deep skin thermoreceptors. In: Webb, P. (1995). Physiology of Heat Regulation. Regulatory Integrative and Comprehensive Physiology 37: R838-R850.
- Ivanov, K.; Konstantinov, V.; Danilova, N. (1982). Thermoreceptor localization in the deep and surface skin layers. In: Webb, P. (1995). Physiology of Heat Regulation. Regulatory Integrative and Comprehensive Physiology 37: R838-R850.

- Ivanov, K.; Konstantinov, V.; Malovichko, N.; Danilova, N.; Trusova, V. (1976). Physiological mechanisms of skin thermosensitivity. In: Webb, P. (1995). Physiology of Heat Regulation. Regulatory Integrative and Comprehensive Physiology 37: R838-R850.
- Johnson, J.M. (1998). Physical training and the control of skin blood flow. Medicine and Science in Sports and Exercise 30(3): p 382-386.
- Johnson, J.M. (1992). Exercise and the cutaneous circulation. Exercise and Sport Science Reviews 20: 59-97.
- Johnson, J.M. (1986). Non-thermoregulatory control of human skin blood flow. Journal of Applied Physiology 61(5): 1613-1622.
- Kellogg, D.L. Jr.; Johnson, J.M.; Kosiba, W.A. (1991a). Competition between cutaneous active vasoconstriction and active vasodilation during exercise in humans. American Journal of Physiology 261: H1184-H1189.
- Kellogg, D.L. Jr.; Johnson, J.M.; Kosiba, W.A. (1991b). Control of internal temperature threshold for active cutaneous vasodilation by dynamic exercise. Journal of Applied Physiology 71(6):2476-2482.
- Kellogg, D.L. Jr.; Johnson, J.M.; Kosiba, W.A. (1990): baroreflex control of the cutaneous active vasodilator system in humans. Circulation Research 66: 1420 -1426.
- Kenney, W.L.; Johnson, J.M. (1992). Control of skin blood flow during exercise. Medicine and Science in Sports and Exercise 24(3): 303-312.
- Kenny, G., Reardon, F. D., Giesbrecht, G.G., Jette, M., Thoden, J.S. (1997). The effects of ambient temperature and exercise intensity on post-exercise thermal homeostasis. European Journal of Applied Physiology 76(2): 109-115.
- Kenny, G.P.; Chen, A.A.; Nurbakhsh, B.A.; Denis, P.M.; Proulx, C.E.; Giesbrecht, G.G. (1998). Moderate exercise increases the postexercise thresholds for vasoconstriction and shivering. Journal of Applied Physiology 85(4): 1357 – 1361.
- Kenny, GP; Denis, P.M.; Boule, N.G., Proulx, C.E.; Thoden, J.S>, Reardon, F.D. (1999). Increasing exercise duration does not affect the postexercise elevation in esophageal temperature. Canadian Journal of Applied Physiology 24(4): 377 – 386.

- Kilgour, R.D.; Rehel, Gariepy, P.; Rehel, R. (1993). Cardiovascular responses during recovery from exercise and thermal stress. Aviation, Space and Environmental Medicine 64: 224-229.
- Mack, G.; Nishiyasu, T.; Shi, X. (1995). Baroreceptor modulation of cutaneous vasodilator and sudomotor responses to thermal stress in humans. Journal of Physiology 483(2): 537-547.
- Mack, G.W.; Nose, H.; Takamata, A.; Okuno, T.; Morimoto, T. (1994). Influence of exercise intensity and plasma volume on active cutaneous vasodilation in humans. Medicine and Science in Sports and Exercise 26(2): 206-216.
- Matsukawa, T., Kurz, A., Sessler, D.I., Bjorksten, A.R., Merrifield, B., Cheng, C. (1995). Propofol linearly reduces the vasoconstriction and shivering thresholds. Anesthesiology, 82: 1169 – 1180.
- Mekjavic I.B., Sundberg C.J., Linnarsson D. (1991). Core temperature “null zone”. Journal of Applied Physiology 71(4): pp 1289-1295.
- Mier CM; Dmenick, MA; Turner, NS; Wilmore, JH (1996). Changes in stroke volume and maximal aerobic capacity with increased blood volume in men and women. Journal of Applied Physiology 80(4): 1180-1186.
- Nadel, E. F.; Cafarelli, E.; Roberts, M.F.; Wenger, C.B. (1979). Circulatory regulation during exercise in different ambient temperatures. Journal of Applied Physiology 46: (2) 247-251.
- Nishiyasu, T., Nagashima, K., Nadel, E.R., Mack, G.W. (1998). Effects of posture on cardiovascular responses to lower body positive pressure at rest and during dynamic exercise. Journal of Applied Physiology 85(1): 160-167.
- Nishiyasu, T.; Shi, X.; Mack, G.M.; Nadel, E.R. (1993). Forearm vascular responses to baroreceptor unloading at the onset of exercise. Journal of Applied Physiology 75(2): 979-985.
- Papelier, Y.; Escourrou, P.; Gauthier, J.P.; Rowell, L.B. (1994). Carotid baroreflex control of blood pressure and heartrate in men during dynamic exercise. Journal of Applied Physiology 77(2):502-506.
- Pergola, P.E.; Kellogg D.L. Jr.; Johnson, J.M.; Kosiba, W.A. (1994). Reflex control of active cutaneous vasodilation by skin temperature in humans. American Journal of Physiology 35: H1979-1984.

- Pergola, P.E.; Kellogg, D.L. Jr.; Johnson, J.M.; Kosiba, W.A.; Solomon, D.E. (1993). Role of sympathetic nerves in the vascular effects of local temperature in human forearm skin. American Journal of Physiology 34: H785-H792.
- Pergola, P.E.; Kellogg D.L.; Kosiba, W.A.; Johnson, J.M.; (1993). Role of the sympathetic nerves in vascular effects of local temperature in human forearm skin. American Journal of Physiology 265(34): H785 – H792.
- Piepoli, M.; Isea, J.E., Pannarale, G.; Adampoulos, S.; Sleight, P.; Coats, A.J.S. (1994). Load dependence of changes in forearm and peripheral and vascular resistance after acute leg exercise in man. Journal of Physiology 478(2): 357-362.
- Piepoli, M.; Coats, A.J.S.; Adamopoulos, A.; Bernardi, L.; Feng, Y.H.; Conway, J.; Sleight, P. (1993). Persistent peripheral vasodilation and sympathetic activity in hypotension after maximal exercise. Journal of Applied Physiology 75(4): 1807-1814.
- Potts, J.T.; Raven, P.B. (1995). Effect of dynamic exercise on human carotid-cardiac baroreflex latency. American Journal of Physiology 268: H1208-H1214.
- Potts, J.T.; Shi, X.; Raven, P.B. (1995). Cardiopulmonary baroreceptors modulate carotid baroreflex control of heart rate during dynamic exercise in humans. American Journal of Physiology 268: H1567-H1576.
- Roberts, M.F.; Wenger, C.B. (1980). Control of skin blood flow during exercise by thermal reflexes and baroreflexes. Journal of Applied Physiology 48(4): 717-723
- Rowell, L.B. (1992). Reflex control of the circulation during exercise. International Journal of Sports Medicine 13 (S1): S25-S27.
- Rowell, L.B. (1991). Blood pressure regulation during exercise. Annals of Medicine 23: 329-333.
- Rowell, L.B.; O'Leary, D.S. (1990). Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. Journal of Applied Physiology 69(2): 407-418.
- Rowell, L.B. (1974). Human cardiovascular responses to exercise and thermal stress. Physiological Review 54:75-159.

- Sawka M.N., Wenger C.B. (1988) Physiological responses to acute exercise-heat stress. In: Pandolf K.B., Sawka M.D., Gonzalez R.R. (eds) Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. Benchmark, Indianapolis, pp 97-102.
- Shi, X.; Gallagher, K.M.; Smith, S.A.; Bryant, K.H.; Raven, P.B. (1996). Diminished forearm response to central hypervolemic loading in aerobically fit individuals. Medicine and Science in Sports and Exercise 28(11): 1388-1395.
- Smolander, J.; Saalo, J.; Korhonen, O. (1991). Effect of workload on cutaneous vascular response to exercise. Journal of Applied Physiology 71: 1614-1619, 1991.
- Taylor, W.F., Johnson, J.M.; Kosiba, W.A. (1990). Roles of absolute and relative load in skin vasoconstrictor responses to exercise. Journal of Applied Physiology 69(3):1131-1136.
- Taylor, W.F.; Johnson, J.M.; Kosiba, W.A.; Kwan, C.M. (1988). Graded cutaneous vascular responses to dynamic leg exercise. Journal of Applied Physiology 64(5):1803-1809.
- Thoden J., Kenny G., Reardon F., Jette M., Livingstone S. (1994) Disturbance of thermal homeostasis during post-exercise hyperthermia. European Journal of Applied Physiology and Occupational Physiology 68 pp170-176. R838-850.
- Thomas, C.M., Pierzga, J.M., Kenney, W.L. (1999). Aerobic training and cutaneous vasodilation in young and older men. Journal of Applied Physiology 86(5): 1676 – 1686.
- Vissing, S.F. (1996). Differential activation of sympathetic discharge to skin and skeletal muscle in humans. Acta Physiologica Scandinavica. P. 5-32
- Webb, P. (1995). The physiology of heat regulation. Regulatory Integrative Comprehensive Physiology 37:R838 - R850.
- Wyss, C.R.; Brengelmann, G.L.; Johnson, J.M.; Rowell, L.B.; Niederberger, M. (1974). Control of skin blood flow, sweating, and heart rate: role of skin vs. core temperature. Journal of Applied Physiology 36(6): 726-733.
- Yoshida, T.; Nagashima, K.; Nose, H.; Kawabata, T.; Nakai S.; Yorimoto, A.; Morimoto, (1997). Relationship between aerobic power, blood volume, and thermoregulatory responses to exercise-heat stress. Medicine and Science in Sports and Exercise 29(7): 867-873.