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Muscle Temperature Transients and Post-Exercise Esophageal Temperature Elevation

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ABSTRACT

The primary purpose of this research was to study esophageal temperature elevation and quadriceps and triceps intra-muscular temperature gradients during resting recovery from exercise. Eight physically active male participants and 24±4 years of age, rested in a buttocks supported upright posture for a 50±12.5 minutes in ambient conditions of 24.7±1.17°C and 24±4.6% relative humidity. Participants then performed a steady state cycle ergometry exercise at 70% Vo_2 max, until they reached until heat produced by exercise above metabolic rate accumulated to 1000 kJ. This was followed by 120 minutes of resting recovery. Active intra-muscular temperature was measured in the vastus medialis at four depths, with the tip of the probe being at 10 mm from the femur and deep femoral artery and the others sensor at 15, 30 and 45 mm from the tip of the probe. Pre-exercise resting esophageal temperature and quadriceps intra-muscular temperatures were 36.95±0.22°C (T_{es}), 36.31±0.39°C (T_{muq1}), 36.10±0.50°C (T_{muq2}), 35.75±0.84°C (T_{muq3}) and 35.33±0.88°C (T_{muq4}) respectively. Exercise resulted in a 0.94±0.32°C increase in esophageal temperature above pre-exercise resting values. End of exercise intra-muscular quadriceps temperatures were 38.81±0.42°C (T_{muq1}), 38.61±0.60°C (T_{muq2}), 38.13±0.75°C (T_{muq3}) and 37.71±0.96°C (T_{muq4}). Esophageal temperature remained significantly elevated from pre-exercise resting values for 10 minutes following exercise. These results indicate that post-exercise esophageal temperature elevation, following cycle ergometry exercise, is not dependent on a temperature gradient between esophageal temperature and deep muscle temperature from the previously active muscle. Further, a prolonged and sustained post-exercise esophageal temperature was not demonstrated following this cycle ergometry exercise.

RÉSUMÉ

Le but de cette étude expérimentale était d'étudier le gradient de température qui existe entre les températures oesophagienne et intra-musculaire des muscles quadriceps et triceps au repos, durant l'exercice et suite à l'exercice. Huit participants masculins actifs, âgés de 24±4 ans sont demeuré dans une position debout supportés pour une période pré-exercice de 50±12.5 minutes dans des conditions ambiantes de 24.7±1.17°C et 24±4.6% d'humidité relative. Les participants ont ensuite complété un exercice d'ergocycle à 70% de leur Vo_2 max, ceci jusqu'à ce qu'ils atteignent une charge thermique de 1000 kJ. L'exercice fut suivi d'une période de repos de 120 minutes dans les mêmes conditions ambiantes. La température du quadriceps (muscle actif lors de l'exercice) fut prise dans le vaste médial à quatre profondeurs. La première sonde fut placée à 10 mm du fémur et de l'artère fémorale. Les trois autres sondes étaient à 15, 30 et 45 mm de la première sonde. Au repos pré-exercice, la température oesophagienne, la température et du quadriceps étaient de 36.95±0.22°C (T_{es}), 36.31±0.39°C (T_{muq1}), 36.10±0.50°C (T_{muq2}), 35.75±0.84°C (T_{muq3}), et 35.33±0.88°C (T_{muq4}) respectivement. L'exercice a eu comme effet d'augmenter la température oesophagienne de 0.94±0.32°C au-dessus des valeurs du repos. À la fin de l'exercice, les températures intra-musculaires du quadriceps étaient de 38.81±0.42°C (T_{muq1}), 38.61±0.60°C (T_{muq2}), 38.13±0.75°C (T_{muq3}) et 37.71±0.96°C (T_{muq4}). Suite à l'exercice, la température oesophagienne demeura statistiquement différente des valeurs pré-exercice pour 10 minutes. Nos résultats démontrent que l'élévation de la température oesophagienne suite à un exercice en vélo ne dépend pas du gradient de température entre la température oesophagienne et la température intra-musculaire. De plus, suite un exercice à vélo, il n'a pas une élévation prolongée et soutenue de la température oesophagienne.

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LIST OF ABBREVIATIONS

Tco = core temperature

Tes = esophageal temperature

Trec = rectal temperature

T_{muq1} = quadriceps muscle temperature at the tip

T_{muq2} = quadriceps muscle temperature at 15 mm from the tip

T_{muq3} = quadriceps muscle temperature at 30 mm from the tip

T_{muq4} = quadriceps muscle temperature at 45 mm from the tip

T_{mut1} = triceps muscle temperature at the tip

T_{mut2} = triceps muscle temperature at 15 mm from the tip

T_{mut3} = triceps muscle temperature at 30 mm from the tip

Tskn = mean skin temperature

SBF = skin blood flow

SPhy = hypothalamic set point

L.E. = load error

HR = heart rate

E.E. = energy expenditure

B.S.A. = body surface area

B.M.I. = body mass index

Ex = exercise

kJ/min = kiloJoules per minute

kJ = kilojoules

W = watts

kg = kilograms

cm = centimetres

ml = millilitres

L = litres

°C = degrees Celsius

mm = millimetres

Vo_2 = oxygen consumption

Vo_{2max} = maximal oxygen consumption

min. = minutes

RMR = resting metabolic rate

rec. = recovery

ex. = exercise

Wex = work during exercise

Hlex = heat loss during exercise

Hlrec = heal loss during recovery

H.F. = heat flux

n.s. = non significant

Hpemr = heat produced by exercise above metabolic rate

CHAPTER 1

INTRODUCTION

1. Background

During and following dynamic exercise, many physiological mechanisms come into play to maintain thermal homeostasis. A set-point temperature regulatory mechanism has long been accepted as the working model of human core temperature regulation (Hammel, 1968). The integrated thermal input from both peripheral and core regions is compared with a central reference temperature referred to as the hypothalamic set point. Deviation from set-point results in the activation of either heat loss or heat gain thermal responses. However, it has been shown that esophageal temperature (T_{es}) in humans remains elevated for 65 minutes following exercise in different ambient conditions and following different exercise intensities (Kenny et al., 1997a, 1997b; Thoden et al., 1994). Based on the 'load error' concept of the set-point theory, it is expected that in the absence of a heat-producing event such as exercise, there will be a rapid re-establishment of core temperature as exemplified by esophageal temperature. These studies suggest that the thermoregulatory system may be regulating at a new resting thermal equilibrium, defined by the prolonged elevated plateau of esophageal temperature. Early studies based on exercise periods varying from 6 to 60 minutes and work intensities varying between 45% and 100% VO_2 max have graphically presented post-exercise esophageal temperature elevation, but none have specifically addressed this phenomenon (Saltin et al., 1970, 1972). In contrast, some studies (Aikas et al., 1962; Sawka

& Wenger 1988) have reported a rapid post-exercise return of esophageal temperature to pre-exercise values.

The underlying mechanism(s) for the prolonged sustained elevation of core temperature following exercise remain(s) unclear. It has been stated that either exercise has a prolonged residual effect on post-exercise temperature regulation or that the significant increase in whole body heat via exogenous heat load has a modifying effect on core temperature (Kenny et al., 1996c).

With respect to the latter, Kenny et al. (1996b) raised esophageal temperature to a value comparable to a moderate 15-minute exercise by using a total body water immersion in a water bath at 42°C. It was shown that esophageal temperature returned to pre-immersion values within 10-15 minutes following immersion. These results provide preliminary evidence that exercise produces a residual effect on post-exercise temperature regulation and that the post-exercise esophageal temperature elevation may be dependent on the type of heat load applied (i.e., exogenous or endogenous heating). More recently, a post-exercise elevation in core temperature of similar magnitude has been demonstrated with different exercise durations (i.e., 15, 30, 45 minutes) (Kenny et al., 1999a). The magnitude of the post-exercise elevation of esophageal temperature remained unchanged for the three conditions despite significantly greater end-exercise esophageal temperatures following each successive increase in exercise duration. Following cessation of exercise, esophageal temperature decreased to an elevated value that differed only by 0.01 °C between the 15- and 30-minute exercise condition, and by 0.02 °C between the 30- and 45-minute exercise condition. Despite a significantly greater end-exercise esophageal temperature between conditions, these results

demonstrate that exercise *per se*—and possibly not whole body heat content—is responsible for the post-exercise change in thermoregulation (Kenny et al., 1999a).

Kenny et al. (1998a) suggest that it is plausible that the cardiovascular (either central or peripheral), metabolic or baroreceptor control changes during exercise have residual thermal effects that may potentially alter hypothalamic temperature regulation. Recent evidence would favour a baroreceptor mediated influence. Further, it has been suggested that the resultant peripheral vasodilation and hypotension following exercise may cause a pooling of the warmed blood thus entrapping the residual heat of muscle (Brown et al., 1993; Piopeli, 1993). This may result in a time-dependent transfer of the residual heat of muscle to the core.

A few studies have investigated the effect of thermal stress on the temperature profile of muscle tissue during rest and exercise (Aikas et al., 1962; Asmussen & Boje 1946; Saltin & Hermansen, 1966; Saltin et al., 1968, 1970, 1972). To our knowledge, Aikas et al. (1962) and Saltin et al. (1970) were the only researchers to measure muscle temperatures during post-exercise resting. Although they graphically presented quadriceps muscle temperature changes during post-exercise rest, they did not specifically address the post-exercise period. Aikas et al. (1962) showed that esophageal temperature decreases to pre-exercise resting values 15 minutes following exercise at three different intensities (i.e. 900, 1200 and 1500 kp·m/min). Muscle temperature of the quadriceps at the two lower intensities (i.e., 900 and 1200 kp·m/min, respectively) fell below pre-exercise esophageal temperature prior to 15 minutes, but at the higher intensity (i.e., 1500 kp·m/min), muscle temperature remained slightly elevated over esophageal temperature for about 15 minutes. Results from Saltin et al. (1970) also show conflicting data. During moderate intensity exercise (75% VO_2 max) performed in cool temperate ambient temperatures of 10°C and 20°C, they observed an

elevated post-exercise esophageal temperature for 30 minutes. In both these conditions, intra-muscular temperature fell below esophageal temperature at about 20 minutes into the recovery period. In contrast, during moderate intensity exercise (75% VO_2 max) performed in a warmer ambient condition of 30° C, Saltin et al. (1970) observed esophageal temperature return to pre-exercise values 20 minutes post-exercise while muscle temperature remained about 1°C over esophageal temperature for the entire 30 minutes of recovery. More recently, Kenny et al. (1998a) demonstrated that single knee extension produces an increase in the resting contra-lateral muscle temperature comparable to esophageal temperature, which suggests that convective heat transfer to inactive tissue may significantly affect the rate of change in esophageal temperature during and following exercise. A subsequent study evaluated the effect of localized endogenous heating on muscle tissue and core thermal stability following exercise (Kenny et al., 1998d). Despite employing a single muscle group (i.e., quadriceps) during isotonic knee extension using Kin-Com isokinetic apparatus, a post-exercise esophageal temperature remained elevated by ~0.2°C above baseline rest for as long as 20 minutes post-exercise. It was proposed that the transfer of residual heat from previously active musculature might in fact contribute to the sustained elevation in post-exercise esophageal temperature.

Although, Kenny et al. (1998d) examined tissue temperature changes during and following exercise, work intensity was limited to a single muscle group and the heat produced by exercise above metabolic rate generated was insufficient to result in a comparable elevation of esophageal temperature in the order of 0.5 °C as reported by Kenny et al. (1996a, 1999a) and Thoden et al. (1994). These data support the need to further investigate the possible role which heat transfer between tissue compartments may have in modifying the

rate of change in esophageal temperature during and following exercise. Furthermore, in conjunction with core temperature measurement, the evaluation of tissue temperature profile of active and inactive muscles during and following exercise will provide necessary information to evaluate tissue temperature gradients from core to periphery and from periphery to core. This should forward our understanding of post-exercise elevation of esophageal temperature. In this regard, the present study was designed to evaluate the effect of changes in heat gain on core thermal stability during and following dynamic cycle ergometry.

2. Purpose

The purpose of this research was to investigate how (cycle ergometry) exercise-induced increases in local tissue (i.e., quadriceps muscles) temperature influence core (esophageal) temperatures during exercise and subsequent recovery. Secondly, the aim of this study was to investigate time course changes in tissue temperature in both active (quadriceps) and inactive (triceps) muscle tissue in humans at rest, during moderate intensity exercise (70% VO_2 max) until heat produced by exercise above metabolic rate reaches 1000 kJ as produced by cycle ergometry and subsequent recovery.

3. Objectives

The objectives of the present study was to measure the effects of exercise performed in 25°C and 22% relative humidity ambient conditions on: (a) active muscle (i.e., vastus

medialis), inactive muscle (i.e., medial triceps brachii) and core (i.e., esophageal and rectal) temperatures, before, during and following exercise; (b) thigh and forearm cutaneous blood flow before, during and following exercise; and (c) cardiovascular (i.e., heart rate, blood pressure) and metabolic (i.e., energy expenditure) responses during pre-exercise resting, exercise and post-exercise.

4. Hypothesis

As a result of the post-exercise residual heat that remains in the muscle, it is hypothesised that an elevated esophageal temperature will be sustained until active muscle temperature (vastus medialis) decreases to a value equal to esophageal temperature.

5. Limitations

The limitations of the present study include the small sample size and the lack of control of some inter-subject variability. Because of the relatively invasive and uncomfortable testing protocol, subject recruitment was difficult. Further, the intensity of the exercise was such that some participants could not complete the protocol. This resulted in a small sample size, limiting the power of the statistical analyses.

The inter-subject variability that could have influenced our results includes the participants' level of physical activity and their body composition. Both these factors are important in the determination of core temperature responses to exercise. Highly active participants will experience different heat dissipating responses than less active participants, the same way participants with a greater body surface area will respond in a way that is

different from that of participants with a smaller body surface area. A more comprehensive protocol that considers these factors might better predict post-exercise responses.

CHAPTER II

REVIEW OF LITERATURE

1. General Characteristics of Thermal Control

Human temperature regulation is characterized by the effector response of shivering and non-shivering thermogenesis, sweating, peripheral vasomotor adjustments and behaviour. As with the control of many other physiological parameters, thermal homeostasis is the result of a complex organization of physiological and behavioural reflexes through which core temperature (Tco) is regulated within appropriate limits. Body temperature is normally represented as a variable that is controlled by comparing it to a set temperature as defined in the hypothalamus and referred to as the hypothalamic set point (SPhy). A change in Tco from SPhy is controlled by negative feedback. According to Sawka and Wenger (1988), changes in Tco are monitored by thermoreceptors on the skin and thermosensitive neurons in deep body, intra-abdominal, spinal and central nervous system sites that pass information to an integrator located in the hypothalamus which, in turns, compares the input with the SPhy.

It has been established that the thermosensitive neurons in the brain are located in the hypothalamus (Adams, 1963; Fusco et al., 1961; Hammel et al., 1960). Adams (1963) established that the specific thermosensitive sites involved in temperature control were the pre-optic and anterior hypothalamic thermosensitive neurons (POA) and were considered the essential integrators of body temperature. The POA is now accepted as the central site of Tco control and regulation.

Understanding the control mechanism of Tco has been made more complicated by the fact that regulation of Tco is influenced not only by the POA but also by other thermosensitive sites such as the spinal cord and the thoracic area. There is evidence that cooling and warming the spinal cord via thermodes in the vertebral canal is followed by appropriate thermoregulatory responses; cooling results in shivering and reduced skin blood flow response, while heating results in increased respiratory frequency, increased skin blood flow, and increased sweating rate. In addition, heat loss mechanisms are inhibited by spinal cooling and heat gain mechanisms, by spinal warming. These effects have been shown both in mammals and in birds and have been reviewed by Simon (1974).

Rawson and Quick (1970) found that intra-abdominal heating in sheep resulted in significant thermoregulatory adjustments. Riedel et al. (1973) observed an increase in respiratory rate in rabbits following heating of the dorsal wall of the abdominal cavity. Cooling to 36°C was less effective and led only to a slight decrease in breathing rate; no further decrease was observed when cooling to as low as 10°C. Riedel et al (1973) concluded that warm receptors are the more abundant receptor type. Pierau and Schmid (1990) demonstrated by localization procedures that thermosensitive neurons in the splanchnic region had a significant impact on Tco regulation. They also suggested that skeletal muscle may possess thermoreceptors but additional evidence has not been presented to support their contention.

The importance of deep-body thermal stimulation has been shown to influence Tco regulation (Hori, 1991). However, unlike hypothalamic and spinal cord thermal activity, the role of such stimulation and influence in overall temperature regulation is yet to be determined.

2. Thermoregulatory Theories and Models

Numerous proposed system models of human thermoregulation have been suggested. All models recognize that when the body becomes hot it loses heat by vasodilation and, if required by sweating. If the body becomes cold, heat is preserved by vasoconstriction and, if necessary, generated by shivering.

a. Set-point theory of thermoregulation. The concept that Tco is maintained within a narrow range was introduced by Stolwijk and Hardy (1966). They considered that the changes in thermal response were proportional to the differences between the value of the controlled variable (i.e, Tco or an integrated thermal signal) and the SPhy. This concept has since been described as the Load Error (LE) concept that represents the temperature difference between internal temperature change and SPhy (Sawka & Wenger 1988; Stolwijk & Hardy 1977). Deviations from SPhy produce a corresponding change in the threshold for each thermoregulatory response whereby the responsiveness of the system to a change in Tco is proportional to the LE signal. Johnson and Ruhling (1985) add that the LE signal is not based on a single Tco input but is instead proportional to a weighted temperature input from both core and skin. If the LE is positive (i.e., weighted signal is greater than SPhy), then an increase in heat loss will be activated. Conversely, for small negative values of the LE (i.e., weighted signal is slightly less than SPhy), heat will be conserved, while large negative values of the LE will stimulate the generation of body heat by shivering. It has been suggested that the influence on the SPhy is not limited to skin temperature changes but was in part due to individual or combined inputs from other thermosensitive regions such as the spinal cord and other deep body sites (Hellstrom & Hammel, 1967).

The evidence implicating feedback from multiple sites of central temperature detection and from the skin indicates synergy of feedback paths involved in the regulation of body temperature. For example, if one of the other sites of central temperature detection becomes warmer than hypothalamic temperature, this signal may independently bring about effector responses to increase heat loss.

b. Null zone concept of temperature regulation. The null zone concept accepts that the hypothalamic thresholds for sweating and shivering are significantly different and that within this zone, there is no reflex activity and that thermolytic mechanisms maintain a passive defence towards small fluctuations in Tco.

Mekjavic et al. (1991) investigated the core thresholds for shivering thermogenesis and sweating. Nine male subjects exercised on an underwater cycle ergometer at a work rate equivalent to 50% of their maximum work rate. Throughout an initial 2-min rest period, a 20-min exercise protocol, and the 100-min recovery period, subjects remained immersed to the chin in water maintained at 28°C. The results indicated that the Tco values at which sweating ceases and shivering commences were significantly different regardless of whether Tco was measured within the oesophagus or rectum. They concluded that their results confirmed the existence of a thermoregulatory "null zone" between the threshold Tco for shivering thermogenesis and sweating within which sweating or shivering responses thermal reflexes were not activated. The magnitude of this null zone was determined to be 0.59°C and 0.57°C for esophageal temperature and rectal temperature, respectively. Previous research had suggested the existence of such a null zone (Jessen & Ludwig 1971; Mekjavic & Bligh 1989) but results of different thresholds for sweating and shivering were not statistically significant to support a null zone range.

c. Core shell concept. Webb et al. (1991) proposed the possibility that the primary mechanism of body temperature regulation was not based on the fluctuation of Tco but rather regulated by changes in the rate of heat flow from core to shell and shell to core. Also, Mekjavic and Bligh (1989) showed that the thermolytic mechanism could be triggered without a significant change in Tco by demonstrating that shivering in man occurred immediately upon exposure to cold without a decrease in Tco.

These observations suggest that tissue temperature transients are critical inputs. Webb and co-workers (1991) speculate that the thermoregulatory system may have an inherent ability to monitor changes in heat flow exchange by detecting a transcutaneous temperature gradient, with a specific receptor type that may be located on either side of the compartmental division. Bazett et al. (1950) first suggested the possible role of subcutaneous temperature receptors in detecting heat flow through the skin. Later work by Ivonov et al. (1982) supports the existence of temperature receptors (in rabbits) at different layers in the skin and that some of these subcutaneous receptors (also in rabbits) are connected to hypothalamic neurons.

For resting humans, about 70% of the metabolic heat is produced by internal organs and the viscera within the body core (Stolwijk & Hardy 1966). However, during muscular exercise, up to about 90% of the metabolic heat can be produced by the skeletal muscles. Because of the different sources of metabolic heat during exercise and rest, temperatures measured during exercise within a given body region may change differently relative to other body regions (Aikas et al., 1962; Saltin et al., 1970). For example, during rest in a comfortable environment, skeletal muscle temperature is lower than central blood volume temperature. During exercise, the temperature within the active skeletal muscle rapidly

exceeds central blood volume temperature (Aikas et al., 1962; Saltin & Hermansen 1966; Saltin et al., 1970) while the temperature within the inactive skeletal muscles and other body regions rises more gradually. The resulting different temperatures between body sites may produce differences in threshold control of sudomotor, shivering and vascular conductance demonstrated between rest and exercise (Johnson et al., 1974; Johnson & Park, 1981).

The effects of thermal exchange have also been described in terms of a Tco after-drop and/or after-rise involving a convective exchange of heat from shell to core and vice versa (Savard et al., 1985; Webb, 1992). The phenomenon of an after-drop has been documented in conditions of hypothermia (Bristow et al., 1994; Giesbrecht & Bristow, 1992; Tikuisis et al., 1991). Observations demonstrate that the rate of body core cooling or rewarming can be significantly influenced by compartmental exchange and that changes in peripheral flow have a modifying effect on reflex thresholds and their regulation of Tco.

While the essence of thermoregulatory theory is maintenance of Tco within a narrow range oriented around the SPhy, all models including the null zone and heat exchange concepts describe how the body can maintain an internal temperature near 37°C based on a balance of heat generation and heat exchange processes. If the body did not lose heat to the environment, although there may be heat exchanges within the body, there would be no effective temperature gradient between the body and the environment. However, in most cases, there is an effective temperature gradient between the core and skin and there is a net transfer of heat from the body to the surface where it can be lost to the environment by conduction, convection, radiation and evaporation at the skin surface and the lungs.

3. Heat Exchange Pathways

The human body is constantly exchanging heat with its environment. Whether the body gains or loses heat depends upon the operation of four different mechanisms of heat transfer: radiation, conduction, convection, and evaporation.

Radiation is heat loss in the form of infrared energy. This involves the transfer of heat from the surface of one object to the surface of another, with no physical contact being involved. At rest in a comfortable environment (i.e., ambient temperature 21-22°C), an undressed person will experience heat loss, 60% of which will occur via radiation (Foss & Keteyian, 1998). This is possible because skin temperature will be greater than the temperature of surrounding objects, which causes a net loss of body heat due to the thermal gradient.

Convective cooling occurs if body surface temperature is warmer than environmental air temperature and heat flows from the body to the surrounding air. As this air is heated, it rises and is replaced by the denser, cooler air. Thus, cool air moves continuously up to the body surface, is warmed by body heat, and then flows away, resulting in a net heat loss from the surface. If air movement (velocity) is due only to local heating by the body, the convection is called "natural." If external influences such as wind, electric fans, etc. contribute to the increased velocity of air movement, the convection is termed "forced". The increase in air movement can also be caused by the movement of the body through the air caused by activity. The greater the velocity of the circulation air around the body the greater the heat loss. Heat loss by convection also depends upon the existence of a temperature gradient between the body surface and ambient air. If the surface and air are at the same

temperature, no heat is transferred; if the gradient is reversed with the air warmer than the surface, the body actually gains heat by convection. The rate of convective heat exchange is also in direct proportion to the exposed surface area of the body. Convective heat transfer also occurs within the body to carry heat with circulating blood acting as a transporters from core to shell and from deep layers of muscle tissue to more superficial ones. Recently, Ducharme et al. (1991) conducted extensive research on tissue temperature transients under steady state and during thermal stress (Ducharme et al., 1992; 1994). They demonstrated that convective heat loss could account for 75% of the forearm heat loss during water immersion at 15 to 36 °C. In addition, they demonstrated that convective heat exchange accounted for 85% of the total heat loss to the environment. Further, in warm water immersion (38°C), it was determined that blood acted as a heat sink carrying the heat gained from the environment away from the limb.

Conduction is defined as the transfer of heat from the body to the molecules of cooler objects in direct contact with its surface. Heat loss from conduction is generally associated with contact with the ground or chair.

The final avenue for heat loss is through evaporation which accounts for approximately 25% of the heat loss at rest and is the most important means of heat loss when metabolic rate is increased and under extreme environmental conditions (Taylor, 1986; Sawka & Wenger, 1988). The body loses 0.58 kcal of heat for each gram of water that evaporates despite an environmental temperature greater than that of the skin while the body gains heat by radiation and conduction. If the body cannot lose heat through sweat and evaporation under these circumstances, the body temperature would rise. However, sweat is only effective for cooling if it evaporates. If the humidity is high, the rate of evaporation is greatly reduced

or totally prevented, so that the sweat remains in a fluid state. Effective evaporation is also minimized in conditions where there is a lack of air movement because the air immediately surrounding the body becomes saturated with water vapour.

Evaporation occurs both as a result of visible sweating and insensible water loss. Water evaporates insensibly from the skin and lungs at a rate of about 600 $ml \cdot day^{-1}$ (McArdle et al.,1989).

Insensible water loss cannot be controlled and it occurs regardless of body temperature. However, regulating the rate of sweating can control evaporative sweat loss. Except for insensible water loss (i.e., small amount of extra cellular fluid that continually diffuses through the skin and respiratory surfaces and evaporates unnoticed), sweat rates are essentially zero when the skin temperature is low. In hot weather, an unacclimatized individual (i.e., a person not adapted to maximize the rates of heat loss) has a maximum sweat rate of about 1.5 $L \cdot hr^{-1}$, whereas an acclimatized person can sweat up to 4 $L \cdot hr^{-1}$. During maximum sweating, a person can lose 3.6 $kg \cdot hour^{-1}$ (Taylor, 1986).

There are two principal non-behavioural mechanisms by which the body can control the transfer of heat by radiation, convection, conduction, and evaporation between the surface of the body and the environment. First, the body can alter the temperature of its surface by changing blood flow to the skin. If skin blood vessels are open, warm blood from the core of the body is brought to the surface where the heat is then more easily lost by radiation, conduction, and convection. If the blood vessels to the skin are constricted, heat will be conserved within the inner regions of the body, and less heat will be lost. The second mechanism by which the body can modulate heat transfer between its surface and the environment is through control of sweat secretion.

4. Blood Pressure

When blood pressure at rest undergoes a transient increase or decrease due to postural stress or thermal 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. These baroreceptors are located in the walls of almost every large artery in the thoracic and neck regions. Most baroreceptors are located in the walls of the carotid sinus and the wall of the aortic arch (Schauf et al., 1990). An increase in arterial blood pressure activates these receptors, causing them to increase their firing rate and transmit signals to the central nervous system and then to the medulla. Secondary signals excite the vagal centre and inhibit the vasoconstrictor centre of the medulla. This integration in the medulla transmits efferent signals to cause a net vasodilation throughout the peripheral circulatory system. A low blood pressure has opposite effects as secondary signals inhibit the vagal centre and stimulate the vasoconstriction centre of the medulla to reflexively increase blood pressure back to normal (Fox, 1999).

At the onset of exercise, an increase of blood pressure is due to the contracting muscles and the drive to increase stroke volume and heart rate. If the baroreflex were to react to this increase in blood pressure and try 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 inactive 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 dynamic exercise by either loading (lower body positive pressure) or unloading (lower body negative pressure) the aortic or carotid baroreceptors (Papelier et al., 1994; Potts et al., 1995). The operation of this reflex during exercise is said to be reset to a higher operating level to compensate for the exercise-driven increase in heart rate and blood pressure (Rowell, 1991, 1992). Rowell (1992) outlined a concept of a central command. This 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 during exercise. At the initiation of exercise, the increases in cardiac output and blood pressure appear to be mediated by central command. The magnitude of central command mediation and the increase in blood pressure is proportional to the number of motor units recruited during muscle contractions.

Following exercise, Brown et al. (1993) and Piepoli et al. (1993) have observed a hypotensive state in normotensive participants. This post-exercise hypotension was found to be greater in ambient conditions of 30°C than in neutral 23°C conditions (Hori et al., 1978 cited in Brown et al., 1993). Many explanations have been suggested for the cause of this hypotension. Among these explanations are persisting vasodilation following exercise and/or diminished baroreflex sensitivity (Piepoli et al., 1993). Brown et al.(1993) propose that this post-exercise hypotension is either of neural, cardiovascular and/or metabolic origin.

5. Blood Flow

For purposes of describing the control of skin blood flow (SBF), the skin surface can be divided into acral (hands, feet, nose, and ears) and non-acral regions (head, limbs, and trunk). In acral regions, cutaneous arterioles are activated solely by noradrenergic sympathetic vasoconstrictor nerves (Johnson, 1986). All thermoregulatory and non-thermoregulatory reflexes in acral skin regions are therefore mediated by alterations in active vasoconstrictor tone. The efferent mechanisms that control the non-acral cutaneous vasculature are more complex. Cutaneous arterioles in these areas are controlled by two branches of the sympathetic nervous system (1963; Johnson, 1986). One branch is a noradrenergic active vasoconstrictor system, similar to that found in acral skin areas. The second branch is an active vasodilator system of unknown neurotransmitter and is postulated to have an indirect relationship to sudomotor nerve activity (Johnson, 1986; Love & Shanks, 1962).

In the hands, feet, lips, ears, and nose, vasoconstrictor fibres are the predominant in vasomotor activation, and the vasodilation that occurs in these regions during heat exposure is largely the result of withdrawing vasoconstrictor activity. Non-acral cutaneous arterioles are under tonic control of both sympathetic vasoconstrictor fibres and by a unique vasodilator system, which is responsible for 95 to 100 % of the total increase in SBF during heat stress (Kenney & Johnson, 1992). The neurotransmitter of the vasodilator nerves is unknown (Brengelmann et al., 1981). The vasoconstrictor and vasodilator systems can both exert marked effects on the cutaneous vasculature; thus it is often difficult or impossible to identify which system is responsible for a reflex change in SBF.

Dynamic exercise creates a primary drive for redistribution of blood flow away from metabolically inactive tissues (including skin) to active muscle (Kellogg et al., 1991a). However, since the energy expended by contracting muscle is also a source of heat and since SBF accommodates the thermoregulatory dissipation of that heat, there is a direct competition between the thermal and non-thermal reflexes. As a result, the magnitude of SBF during exercise is subject to both vasoconstrictor and vasodilator influences.

With the onset of dynamic exercise, there is a general exercise induced-vasoconstriction of both acral and non-acral regions (Johnson & Park, 1982; Johnson, 1992), which is directly related to the level of exercise (Taylor et al., 1988). 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 participants perform various modes (isolating large and small muscle groups) of supine exercise at high (80W) and low (40W) intensity. Measurement of skin blood flow (SBF) by laser Doppler flowmetry confirmed the initial constrictor response of the cutaneous vasculature at the onset of exercise. Due to the increased vasoconstrictor response at the higher intensity, the authors concluded that the degree of vasoconstriction is dependent on absolute exercise intensity. Johnson (1986) also found that higher levels of dynamic exercise by large muscle groups (2 legs) are associated with a greater reflex of cutaneous vasoconstriction at the onset of exercise.

Studies using upright bicycle ergometry exercise have been used to study core temperature threshold for cutaneous vasodilation and to compare this to supine exercise (Johnson & Park, 1981; Roberts & Wenger, 1980). These investigators found a common trend in upright exercise compared to supine. Upright cycle ergometer exercise consistently decreases the cutaneous blood flow at a given core temperature. 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.

As exercise is extended beyond 5-10 minutes, the rising Tco initiates a net cutaneous vasodilation (Johnson et al., 1974). There is no apparent involvement of the vasoconstrictor system (Johnson & Park, 1974; Kellogg et al., 1991a, 1991b). The threshold for vascular controls of SBF is also known to be influenced by numerous factors such as: whether the exercise is dynamic or static (Taylor et al., 1990), exercise intensity (Smolander et al., 1991), exercise posture (Johnson & Park, 1981; Roberts & Wenger, 1980), duration of the exercise period (Smolander et al., 1987) and ambient temperature (Johnson & Park, 1982; Smolander et al., 1987). With so many factors known to influence threshold control of surface vascular controls, there is a real difficulty associated with interpreting such changes.

6. Post-exercise Homeostasis

Previous studies have reported post-exercise core temperatures (Aikas et al.,1962; Saltin et al., 1970). However, none has shown a prolonged elevation in esophageal temperature following exercise; instead they have shown a gradual decrease in esophageal temperature over time. There are those who have argued that an elevation in Tco during exercise is an indication of a change in SPhy (Nielsen, 1966; Saltin & Hermansen, 1966). Their explanation was that the hypothalamic thermostat is reset during high metabolic rates. The primary arguments against an adjustment of the SPhy are that the high metabolic rate of exercise is required to both elevate and maintain Tco, and that the increase in the rate of heat

loss to attenuate the rate of increase of Tco indicates that the SPhy is lower than the level attained or maintained during the exercise (Sawka & Wenger, 1988). Thus, reducing the metabolic rate by the cessation of exercise should allow core temperature to return to pre-exercise levels if thermal reflex defences remain activated.

Thoden et al. (1994) investigated the response of esophageal and rectal temperatures to exercise during recovery. Following an 18-minute treadmill running exercise at approximately 75% VO_2max, the investigators observed a drop in esophageal temperature followed by a stable plateau which was established within 10 minutes and remained for the entire 65-minute recovery. A significant finding of this study was that the post-exercise esophageal temperature plateau corresponded to the esophageal temperature threshold for vasodilation. It was expected that the exercise-induced elevation of esophageal temperature following cessation of exercise would be gradually eliminated within a short period.

In subsequent studies, it has been demonstrated that successive exercise/recovery cycles performed at progressively increasing pre-exercise esophageal temperature levels resulted in further and parallel increases of the esophageal temperature threshold for cutaneous vasodilation during exercise and of the post-exercise elevation in esophageal temperature (Kenny et al., 1996a). Kenny et al. (1996b) compared the recoveries in esophageal temperature following similar increases in esophageal temperature during endogenous heating (exercise) and exogenous heating (warm water immersion). This experiment was designed to produce comparable changes in the rate and magnitude of the esophageal temperature increase for both conditions. There was no demonstration of an elevated esophageal temperature during post-immersion recovery. There results show that there is a difference in thermoregulatory response depending on the type of heating involved.

Further, Kenny et al. (1999a) showed no differences in the post-exercise sustained elevated plateau in esophageal temperature despite significantly different heat loads via different durations of treadmill running. It has yet to be determined if different heat loads via different exercise intensities produce a similar response.

It has been demonstrated under a number of different conditions such as varying levels of work intensity (Smolander et al., 1991), ambient temperature (Kenney & Johnson, 1992), and skin temperature (Kenney & Johnson, 1992; Taylor et al., 1984) that there are changes in the threshold temperatures for sweating (Johnson, 1992; Sawka & Wenger, 1988) and active vasodilation during exercise (Kellogg et al., 1991; Kenney & Johnson, 1992). Kenny et al. (1997b, 1998b, 1998c) have demonstrated the modification of post-exercise threshold for vasoconstriction and shivering as well as vasodilation and sweating. These thresholds were increased 0.3-0.5°C. These results further support the contention that the prolonged post-exercise increase in esophageal temperature and thermal response thresholds may be a function of some exercise-related residual effects. These results also indicate that exercise has a prolonged effect by increasing both post-exercise warm thermoregulatory responses.

7. Muscle Temperature

Thermal balance in muscle tissue is dictated by a continuous exchange of heat via conduction and convection between the muscle tissue and the blood volume that perfuses the muscle. If no external or internal thermal parameters change, muscle tissue temperature would remain relatively stable. In ambient condition of 29°C and 40% relative humidity, the average resting temperature in human muscle is usually about 34.1°C at a 2cm depth and 34.5°C at a

Table 1

Post-exercise Esophageal Temperature Elevation Comparison

reference	room temp.	humidity	exercise modality	exercise intensity	exercise time	start exercise condition	end-exercise esophageal temperature	post-exercise sustained elevation	duration of elevation	participants
Thoden et al. (1994)	29°C	50%	Treadmill	75% VO_2max	18 min.	±0.05°C over 5 min.	38.22°C	0.5°C	65 min.	5
Kenny et al. (1996)	29°C	50%	Treadmill	70% VO_2max	15 min.	±0.05°C over 10 min	37.21°C	0.48°C	30 min.	9
Kenny et al. (1996C)	29°C	50%	Treadmill	70% VO_2max	15 min.	±0.05°C over 10 min	37.70°C	0.48°C	15 min.	9
Kenny et al. (1997)	A 20°C	A 50%	A Treadmill	A 45%VO_2max	A 18 min	A ±0.1°C over 5min	A 37.38°C	A 0.33°C	A 20 min.	A 5
	B 24°C	B 50%	B Treadmill	B 75% VO_2max	B 18 min	B ±0.1°C over 5 min	B 37.78°C	B 0.59°C	B 20 min.	B 5
	C 29°C	C 50%	C Treadmill	C 75% VO_2max	C 18 min	C ±0.1°C over 5min.	C 38.22°C	C 0.57°C	C 20 min.	C 5
	D 40°C	D 50%	D Treadmill	D 45%VO_2max	D 18 min	D ±0.1°C over 5min	D 37.81°C	D 0.35°C	D 20 min.	D 5
Kenny et al. (1998)	22°C	na	knee extension	60% VO_2max	15 min.	30 min. Rest	37.31°C	0.12 °C	10 min.	6
Kenny et al. (1999)	A 29°C	A 50%	A Treadmill	A 70%VO_2max	A 15 min	A±0.05°Cover 10 min	A 37.97°C	A 0.41°C	A 60 min.	A 9
	B 29°C	B 50%	B Treadmill	B 70% VO_2max	B 30 min	B±0.05°Cover 10 min	B 38.46°C	B 0.42°C	B 60 min.	B 9
	C 29°C	C 50%	C Treadmill	C 70% VO_2max	C 45 min	C±0.05°Cover 10 min	C 38.90°C	C 0.44°C	C 60 min.	C 9
Kenny et al. (2001)	A 29°C	A 50%	A Cycle	VO_2max (20W)	A 25 min	A 15 min. rest	A 38.58	A 0.1°C	A 48 min.	A 6
	B 29°C	B 50%	B Cycle	VO_2max (40W)	B 16 min	B 15 min. rest	B 38.19	B 0.1°C	B 48 min.	B 6
Denis, P. (2002)	25 °C	25%	Cycle	70% VO_2max	22 ± 4 min	±0.1°C over 5 min.	37.89°C	0.43°C	10 min.	8

4cm depth (Webb, 1992). A number of studies have investigated the effect of inactive resting and exercise on intra-muscular temperature but none have focussed on the post-exercise period. Early studies focussed mainly on the possible relationships between changes in muscle temperature and the capacity for work, the effects of endogenous and exogenous heating prior to exercise (Asmussen & Boje, 1946) and blood flow distribution at rest (Barcroft & Edholm, 1946). Subsequent research looked at the rise in muscle temperature during exercise and its possible relationship to core temperature increases and sweat rate (Saltin & Hermansen, 1966; Saltin et al., 1968, 1970).

It has been demonstrated that muscle temperature is not correlated with sweat rate nor is it the primary stimulus for sweating (Nadel et al., 1972; Saltin et al., 1970). More importantly, findings from these early studies showed that muscle tissue temperature rises rapidly within the first 5 minutes of exercise and subsequently reaches a thermal steady state within 10 to15 minutes of exercise, for exercise intensities between 50% and 75% VO_2 max (Aikas et al., 1962; Saltin et al., 1966, 1968). Moreover, it was determined that the rate of rise of muscle tissue temperature during exercise is dependent on the intensity of the exercise (Aikas et al., 1962; Asmussen et al., 1946; Saltin & Hermansen, 1966) and that exercise intensities greater than 50% VO_2max result in muscle tissue temperature increases that are greater than either esophageal temperature or rectal temperature. This creates a temperature gradient between peripheral tissue and core, which is not shown with intensities below 50% VO_2 max. Further, Saltin et al. (1972) also demonstrated that during sub-maximal exercise, muscle temperature reached levels higher then any other tissue in the body but they also demonstrated that muscle temperature was primarily a function of work intensity and secondary to environmental temperature.

As for the importance of muscle temperature and core temperature stability, Saltin et al. (1968) suggested that intra-muscular temperature might have an impact in the regulation of core temperature. They observed that, during three different exercise intensities and in environmental conditions of 20°C and 40% relative humidity, rectal temperature did not increase until muscle temperature was above rectal temperature values. This observation is also supported by Saltin et al. (1970). Given, that it has been demonstrated that rectal temperature is less responsive to dynamic changes in temperature compared to esophageal measures (Saltin & Hermansen, 1966), it would be expected that this same relationship would not exist between muscle temperature and esophageal temperature. However, there is insufficient evidence to support the contention that the relationship between intra-muscular temperature and esophageal temperature exists.

Although only graphically, Aikas et al. (1962) and Saltin et al. (1970) presented data on post-exercise recovery period. It was found that muscle temperature following low intensities exercise fell below pre-exercise esophageal temperature after the 15-minute recovery. However, in the same ambient conditions (24°C and 40% relative humidity) but at higher work intensities, muscle temperature remained elevated over esophageal temperature for about 15 minutes (Aikas et al., 1962). Results from Saltin et al. (1970) also show conflicting data. During exercise at 75% maximal oxygen consumption in ambient temperatures of 10°C and 20°C, they observed an elevated post-exercise esophageal temperature for 30 minutes. In both these ambient temperatures, intra-muscular temperature fell below esophageal temperature before the end of the 30 minutes recovery period. In contrast, during exercise at 75% maximal oxygen consumption in a 30°C environment, Saltin et al. (1970) observed a decreased of esophageal temperature to pre-exercise values within 20

minutes post-exercise while muscle temperature remained about 1°C over esophageal temperature for the entire recovery.

More recently, Kenny et al. (1998a) demonstrated that single knee extension produces an increase in the resting contra-lateral muscle temperature comparable to esophageal temperature, which suggests that convective heat transfer by the blood to inactive tissue may significantly affect the rate of change in esophageal temperature during and following exercise. A subsequent study evaluated the effect of localized endogenous heating on muscle tissue and core thermal stability following exercise (Kenny et al., 1998d). Despite employing a single muscle group (i.e., quadriceps) during exercise (as compared to treadmill running), a post-exercise elevation of esophageal temperature of about 0.2°C above pre-exercise resting values was shown for 20 minutes. Although a more intense exercise would likely have resulted in an increase in the magnitude of the post-exercise esophageal temperature, the results showed that the transfer of residual heat from previously active musculature might contribute to the sustained elevation in post-exercise esophageal temperature.

8. Measurements of Core Temperatures

There are numerous methods available to measure Tco. For example, the rectum, the ear canal or tympanic membrane, and the oesophagus are all sites that have been identified with Tco. Temperatures at these sites can differ by a tenth of a degree or more and variations depend on regional rates of heat production and blood flow (Livingstone et al., 1983). Perfusion of the organ mass determines the time it takes for organ temperature to change in response to a change in arterial blood temperature. The highly perfused hypothalamus

responds rapidly to blood temperature changes as do thermistors when placed in the oesophagus. Conversely, tissues of the lower abdomen respond very slowly to changes in other parts of the body. The benefits and limitations associated with the different measurement sites have been presented in numerous papers (Saltin & Hermansen, 1966).

One difficulty in employing these different measurement sites is determining which temperature provides the ideal measure of Tco. Under steady-state conditions, measurements of Tco are not significantly influenced by thermal input from slowly responding regions, which may take many minutes to equilibrate with the temperature of arterial blood. Under dynamic conditions when Tco is changing, temperatures at various thermosensitive sites can vary substantially and the most accurate representation would be from a site that is able to monitor acute thermal transient changes.

The idea that a single temperature measurement can provide an index of the average internal temperature may not be reasonable. Early on, researchers agreed that the practice of representing average internal temperature as a single measurement was not valid (Bazett et al., 1950). Despite these conclusion, it is common practice to express changes in Tco based on a single measured value or with measurements of body temperature based on a weighted sum of mean skin and core temperatures.

a. Esophageal temperature measurement. Esophageal temperature is obtained by inserting a catheter containing a thermocouple or thermistor through the nasal passage and into the throat and then swallowing it (Mekjavic & Rempel, 1990; Sawka & Wenger, 1988). Most thermal physiologists agree that esophageal temperature is the best practical index of core temperature for humans, based on the findings that esophageal temperature responds rapidly to changes in blood temperature (Shiraki et al., 1986). Shiraki et al. (1986) found that

esophageal temperature quantitatively reflected pulmonary artery (mixed venous blood) temperature with an average difference of 0.1°C and a lag time of about one minute. Several investigators have simultaneously measured esophageal temperature and the temperature of active skeletal muscle during exercise (Aikas et al., 1962; Saltin & Hermansen 1966; Saltin et al., 1970, 1972). The results demonstrated that during exercise esophageal temperature and active muscle temperature achieved steady-state values almost simultaneously after 15 minutes. The rapid response time for esophageal temperature is due to the low heat capacity of the oesophagus and its proximity to the heart, allowing it to be rapidly warmed and cooled by post-respiratory mixed venous blood (Sawka & Wenger, 1988).

b. Rectal temperature measurement. Temperature measurements are uniform within the rectum up to 100-270 mm from the anal sphincter (Nielsen & Nielsen, 1962). Rectal temperature is normally used to represent the temperature of a large mass of deep body tissue. This measure is said to give an "average" internal body temperature; however, it may not be representative of brain temperature and particularly of acute temperature changes (Astrand & Rodhal, 1986; Saltin & Hermansen, 1966). Rectal temperature is generally higher than values measured in arterial blood (Sawka & Wenger, 1988) and other core temperature sites. Steady state value of is generally ~0.2°C higher than simultaneously measured esophageal temperature (Saltin & Hermansen, 1966; Saltin et al., 1970) and they are independent of ambient temperature. Steady-state rectal temperature, however; can provide a good index of body heat storage (Saltin & Hermansen, 1966). The main problem with Trec is that it is slow to respond to changes in blood temperature (Saltin & Hermansen, 1966). During exercise it takes approximately 25-40 minutes to achieve steady-state rectal temperature values (Aikas et al., 1962; Greenleaf & Castle, 1972; Nielsen & Nielsen, 1962;

Saltin et al., 1970, 1972). The reason for the slow response of rectal temperature to thermal transients is probably a low rate of blood flow to the rectum compared to other measurement sites (Aulick et al., 1981). The slow response time makes rectal temperature a poor index for estimating the input to the thermoregulatory controller (Saltin et al., 1970). Despite the limitations, steady-state rectal temperature is widely employed by thermal physiologists because of the ease and comfort in its use. The majority of thermoregulatory theory and modelling as well as the presumed acute responses are based on data from this source.

CHAPTER III

METHODOLOGY

1. Participants

With approval from our Faculty Human Ethics Committee and Ottawa Hospital Research Ethics Board, 12 male volunteers with no history of cardiovascular or respiratory disease participated after providing written and informed consent. Participants were all physically active but did not participate in any form of competitive training program. All participants were subjected to a preliminary session and one experimental session. All experimental trials were conducted from mid February to late March and used a single intervention time series design. Four of the twelve participants did not fully complete the experimental protocol and therefore were not included in any of the means or statistical analysis. Two excluded participants did not complete the experimental trial because of data collection problems, one could not be implanted with intra-muscular probes due to high vasculature and the other participant did not complete the 120 minutes of post-exercise recovery.

Participants ranged in age from 21 to 35 years (mean = 24± 4.5 years), in weight from 68 to 110.6 kg (mean = 83.7± 16.4 kg) and in height from 171 to 190 cm (mean = 177.2± 6.9 cm). Their average body fat (as measured via hydrostatic weighing) and cycle ergometer maximal oxygen consumption were 14.84±7.82 % and 48.6±10.46 $mlO_2 \cdot kg^{-1} \cdot min^{-1}$, respectively. Participants' physical and functional characteristics are presented in Table 2.

Table 2

Individual and Mean Physical and Functional Characteristics of Study Participants

Participant	Age (years)	Weight (kg)	Height (cm)	B.S.A. (m^2)	VO_2 max ($mlO_2 \cdot kg^{-1} \cdot min\text{-}1$)	B.M.I** (kg/m^2)	Body fat* (%)
1	21	84.3	171	1.97	43.73	28.9	13.3
2	22	76.1	182	1.99	45.36	23	10.2
3	35	97	176	2.13	38.73	31.3	26.2
4	25	110.6	190	2.38	35.42	30.6	27
5	24	97.5	182.5	2.19	45.51	29.3	15.9
6	23	68.2	173.5	1.82	65.27	22.7	5.1
7	24	68	172	1.78	60.69	23	10.6
8	21	68	171	1.8	53.86	23.3	11.1
Average (n=8)	24	83.7	177.2	2	48.6	26.5	14.84
stdev (±)	4.5	16.4	6.9	0.21	10.46	3.85	7.82

Note: kg= kilograms, cm= centimetres. The body surface area was estimated from the height and weight of the subject using the following equation [Dubois & Dubois (1916)] B.S.A.= body surface area [B.S.A.= $0.202 \cdot (\text{weight (kg)})^{(0.425)} \cdot (\text{height (cm)})^{(0.725)}$], *% body fat analysis via hydrostatic weighing, **body mass index was calculated according to Professional Fitness and Lifestyle Consultant guidelines [weight (kg)/height $(m)^2$]

2. Methods and Procedures

Body composition, specifically predictions of percentage body fat, was determined by hydrostatic weighing. Hydrostatic weighing determines percent body fat from body density. Density was calculated by dividing mass by volume. Body volume was calculated by subtracting body weight in air from body weight in water (body volume corrected for water temperature). The following equation was used to calculate body density:

$$\delta wb = \frac{Wair}{\frac{Wair - Ww}{\omega w} - RV}$$

Where: δwb = whole body density
W_{air} = weight in air
W_w = weight in water corrected for trapped air volume
ω_w = density of water at T_W
RV = residual volume

Residual volume was calculated as follows (see Boren et al., 1966):

RV = 0.0115·(age) + 0.019·(height in cm) - 2.24

Percent body fat was then extrapolated from the equation of Siri (1956):

$$\%BF = [(4.95 \bullet \delta wb^{-1}) - 4.50] \bullet 100$$

Esophageal temperature was used as an estimate of core temperature in all participants. In 5 of the 8 participants, rectal temperature was also used as an index of deep body abdominal temperature. Esophageal temperature was recorded using an esophageal temperature thermocouple probe inserted through a nostril until it reached a depth that places the tip approximately at the level of the heart. Probe depth was approximated by using the following formula according to standing height (see Mekjavic & Rempel, 1990):

(0.228·(standing height cm)) - 0.194

Rectal temperature was measure between 100mm and 120mm from the anal sphincter using a thermocouple probe (Nielsen & Nielsen, 1962).

Muscle temperature probes where implanted at the Ottawa Hospital General Campus using an aseptic technique as detailed in Kenny et al. (2001). Briefly, the skin, subcutaneous tissue and muscle where all anaesthetized by infiltrating ~2 mL of 1% lidocaine without epinephrine to a maximum depth of 50mm with a 25-gauge needle inserted perpendicular to the skin. Under ultrasound guidance, an 18-gauge, 50mm polyethylene catheter (Cathlon, Critikon Canada, Markham, Ontario) was then inserted into the anaesthetized tract to its full length and the stylet removed. The sterile Teflon coated multi-sensor temperature probe (1 mm diameter, IT-17: 4, type T standard thermocouple, Physitemp Instruments, Inc., Clifton, New Jersey, USA) was then introduced until the end of the probe reaches the end of the catheter. The catheter was withdrawn from the wound and with the multi-sensor probe assembly secured on the skin by a waterproof transparent dressing tape. In large active muscles, there is a wide variation in the tissue temperature measured depending on the proximity of the site of measurement to the surface and to such structures as large arteries and bone (Kenny et al., 2001). In terms of safety, it is necessary to avoid damaging nerves, vessels and bone during the insertion procedure. Typically, in most studies, external surface markers are used to standardize the positioning of intra-muscular probe for all participants. It is possible to measure the final probe positioning by X-ray (Saltin et al., 1968) and magnetic resonance imaging (Binzoni et al., 1995) or to pre-determine the exact site of the muscle temperature measured by computed-tomography scans (Ducharme & Tikuisis, 1992). However, none of these techniques allow for real-time precision guidance of the thermal probe to a specific internal location. This study's technique increases the precision with which

intra-muscular temperature probes can be consistently placed in participants relative to an internal anatomical structure.

Deep muscle temperature was measured in the vastus medialis and triceps medialis. Measurement were made at four depths in the vastus medialis using a single insulated multi sensor probe with thermistors with the tip of the probe at 10mm from the femoral artery and the three other sensors at 15, 30 and 45mm from the tip or approximately 6cm, 4.5cm, 3.0cm and 1.5cm from the skin. The anterior leg implant site was approximately midway between and medial to a line joining the anterior superior iliac spine and base of the patella. The intra muscular probe was placed such that the tip was positioned 10mm equidistant from the deep femoral artery and the diaphysis of the femur. Using a similar probe, measurement where made at the tip and at 15 and 30mm in the triceps medialis or at 4cm, 2.5cm and 1cm from the skin surface. The triceps site was at about two thirds of the way proximally between the acromial process and the olecranion. The probes were inserted under ultrasound guidance to a depth no less than 10mm from the humerus. After the experimental trial, upon removal the probes were inspected for any kinks or folds and were measured for length. It is of note that both probes were not implanted perpendicularly but at a slight angle.

With the resulting ultrasound image for both upper and lower segments probe placement and experimental data it was then possible to determine temperatures across the radial distance of the thigh and the upper arm.

Cutaneous temperature was monitored at 12 sites by heat flow sensors with integral linear thermistors (Concept Engineering, Old Saybrook, CT, model FR-025-TH44018-6) and one site by thermistor all which were placed on the skin surface (see appendix B) and the area-weighted mean skin temperature (T_{skn}) was calculated by assigning the following

regional percentages adapted from Hardy and Dubois (1938) in Mitchell and Wyndham (1968): head 8%, upper arm 9%, forearm 7%, finger 2%, chest 12%, abdomen 9.5%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 7.5 % and posterior calf 6%.

Heat flux (Concept Engineering, Old Saybrook, CT) was also assessed at 11 superficial sites and the area weighted for mean flux was calculated according the following regional percentages adapted from Hardy and Dubois (1938) in Mitchell and Wyndham (1968): head 8%, upper arm 9%, forearm 7%, chest 12%, abdomen 9.5%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 7.5% and posterior calf 6%. The weighed average was then divided by 0.98 to bring the value to 100%.

Blood pressure was measured non-invasively, with finapress automated blood pressure monitor and via auscultation. Automated blood pressure was measured from the electrical integration of the pulsatile blood pressure signal obtained from the middle digit using a Finapress blood pressure monitor (Ohmeda, Louisville CO, USA, Model 2300) referenced at the level of the heart (third intercostal space). Finapress measures where taken for the 15 minutes prior to exercise and the 20 minutes immediately following exercise. Auscultation measures where taken at 20 and 5 minutes prior to exercise, immediately following exercise, and 20, 30 and 60 minutes post-exercise.

Cutaneous blood flow was assessed with a laser-Doppler flowmetry probe placed on both the mid-anterior thigh (TSI, St. Paul Minn., USA, Model BPM 403A) and forearm (Perimed, Periflux, Sweden). The laser-Doppler sensors were taped to a cleaned surface at a location that gave a consistent reading. Muscle blood flow was also measured (Perimed, Periflux, Sweden) in the triceps. The fibre optic probe was inserted in an adjacent position to

the triceps intra-muscular temperature probe. In all blood flow measures only relative values were evaluated and no attempt was made to evaluate absolute blood flow.

Oxygen consumption was measured during pre-exercise resting, exercise and until 65 minutes post-exercise using the open circuit technique. Expired gas was collected through a two-way valve, and analysed for volume and composition using an automated metabolic cart (Medgraphics, Cardiopulmonary Diagnostics System).

The total energy (E_{tot}) expended as a result of exercise, during the period from onset of exercise until the end of exercise, was calculated using the following equation (expressed in kilojoules):

$$E_{tot} = \dot{V}O_2 \cdot \left[\frac{(RER - 0.7)}{0.3} \cdot e_g + \frac{(1 - RER)}{0.3} \cdot e_f \right]$$

Where: e_g = the caloric equivalent in kilocalorie per litre of oxygen for carbohydrates;
e_f = the caloric equivalent in kilocalorie per litre of oxygen for fat; and,
RER = the respiratory exchange ratio.

The total energy expended (E_{TOT}) during the period from the onset of exercise until end of exercise was taken as the average of E_{TOT} for the entire exercise period and then multiplied by the total exercise time. Values calculated were expressed in kJ.

The cycle ergometry mechanical work (W_{mech}) was calculated using the following equation :

$$W_{mech}\ (kJ) = \text{resistance(N)} * \text{rpm} * \text{dist.(m)} * \text{time(min)}$$

Where: W_{mech} (kJ) = mechanical work
rpm = revolutions per minute
dist. = distance travelled for one revolution (6 m)

Given the consistent resistance and revolutions per minute, total work done (W_{tot}) was the product of the mechanical work (W_{mech}) accomplished during each minute and total time of the entire exercise session. These data were also expressed in kJ.

Mechanical efficiency (M.E.) was defined as the total work (W_{tot}) completed during the entire exercise time divided by total energy expenditure (E_{tot}). Thus :

$$M.E. = W_{tot} / E_{tot}$$

The resting metabolic rate (RMR) was calculated from the average rate of oxygen consumption for the period 10 to 5 minutes prior to exercise bout. These values were calculated and expressed in kilojoules using the aforementioned equation.

Whole body heat produced by exercise above metabolic rate (in kilojoules) during exercise was calculated by subtracting resting energy expenditure (RMR) and the energy equivalent of the mechanical work completed from total energy expenditure:

$$\text{Heat produced by exercise above metabolic rate} = E_{tot} - (W_{tot} + RMR)$$

The dry heat loss or the heat lost by radiation, conduction and convection from the skin surface, in excess over pre-exercise resting period, during exercise (Hlex) and during recovery (Hlrec) was estimated by subtracting the area weighted mean heat flux (HF) corrected for body surface area (BSA) during rest from those values recorded during exercise and recovery respectively. Thus:

$$Hlex\ (kJ) = (HF\ (kJ) * BSA)_{ex} - (HF\ (kJ) * BSA)_{rest}$$

$$Hlrec\ (kJ) = (HF\ (kJ) * BSA)_{rec} - (HF\ (kJ) * BSA)_{rest}$$

The body surface area was estimated from the height and weight of the subject using the following equation [Dubois & Dubois (1916)] B.S.A.= 0.202·(weight (kg))$^{0.425}$)·(height (cm)$^{0.725}$).

The dry heat loss during exercise and during recovery was the total dry heat lost during the entire exercise session and 120 minutes of recovery. All heat loss values were converted from watts to kilojoules using 0.06 (1watt = 1 Joule/second) conversion factor.

Heart rate (beats/min) was measured continuously using wireless transmission (Polar vantage nv and Advantage interface System) for the entire trial.

Temperatures where collected and digitized (Hewlett Packard data acquisition module, model 3497A) at 10-s intervals, displayed graphically on the computer screen, and recorded in spreadsheet format on a hard disk (Hewlett Packard, model PC-312, 9000).

3. Experimental Protocol and Procedures

a. Orientation session. During this session, the participants where introduced to the instrumentation and laboratory surroundings. This time was also used to address questions or concerns. Participants where then asked to fill out a participant activity readiness questionnaire (Par-Q) and consent forms. The participant's, weight, height and body fat percentage by hydrostatic weighing where then assessed. Participants then performed an incremental maximal test on a cycle ergometer to determine their maximal oxygen consumption (VO_2 max). The maximal test was carried out using the protocol in Table 3.

Table 3

Cycle Ergometer VO_2 max Protocol

Time (min)	0 - 3	3 – 6	6 - 9	9 – 12	12 - 15	15–18
Revolutions per min	70	70	70	70	70	70
Intensity (kp)	1	1.5	2	2.5	3	3.5

Note: kp= kilopond

Both heart rate and oxygen consumption (VO_2) were measured continuously throughout the test until the voluntary maximum was reached. These data served to select the appropriate cycling intensity for the subsequent experimental trial. On a subsequent day, to confirm work rate and efficiency at the preselected load of 70% VO_2 max, participants were asked to cycle for 10 minutes at this intensity or until they reached 1000 kJ while oxygen consumption and heart rate were monitored. This verification was done to facilitate total heat produced by exercise above metabolic rate estimation given that the participants would be instrumented with muscle probes during experimental trial and to verify if participant could maintain exercise intensity for the required time while fully instrumented. A work intensity of 70% VO_2max was selected because it corresponded to a higher intensity than Kenny et al. (1998a) and similar work intensity as Kenny et al. (1999a). It was important that work intensity be higher than in the Kenny et al.'s (1998a) study because at 60% intensity in single leg knee extension, the researchers were not able to reproduce post-exercise sustained esophageal temperature elevation. Further, with different exercise duration and at 70% work intensity, Kenny et al. (1999a) were able to reproduce a sustained post-exercise esophageal elevation.

Firstly, cycle ergometry exercise was selected as a natural progression to unilateral and bilateral leg extensions (Kenny et al., 1998a ,1998b) to create a higher heat production by

exercise above metabolic rate with the involvement of more muscle mass and without the excessive trauma of treadmill running. Secondly, the higher mechanical efficiency of cycle ergometry serves as a good counter-comparison to the low efficiency (8.13±1.54 %) of leg extensions (Kenny et al., 1998a). Unlike similar studies (Aikas et al., 1962; Saltin et al., 1966, 1968), we tried to better control the total heat produced by exercise above metabolic rate imposed to every participant via the exercise bout. Our target was selected to be 1000 kJ. This target was selected following pilot studies that showed that the average time to attain 1000 kJ was between 18 and 25 minutes, which corresponded to the duration of exercise used to elicit post-exercise esophageal temperature elevation following treadmill exercise. Moreover, this heat produced by exercise above metabolic rate also exceeded the average heat produced by exercise above metabolic rate imposed in the 1998a Kenny et al. study (~300 kJ), which elevated esophageal temperature by only 0.3 °C over baseline resting.

b. Experimental session. The experimental trials where conducted in the morning following a 24h period without heavy or prolonged physical activity. On each study day, care was taken to avoid major thermal stimuli or substantial increase of metabolic rate between awakening and the start of the experiment. Upon arrival to the laboratory, participants were transported to the Ottawa Hospital General Campus for the intramuscular probe implantation and, afterwards, they were returned to the laboratory. The participants were then required to remove excess layer of clothing to be in shorts and running shoes and then were prepared and instrumented appropriately. Participant instrumentation lasted between 45 and 60 minutes. During this time participants remained in a supported standing position in ambient conditions of 25°C and 22% relative humidity.

When at rest, prior to and following exercise, participants remained in a semi-recumbent position. This position is best described as an upright position with participant supported at the buttocks. Participants are seated on the edge of a high cushioned stool with upright backrest. To give back support, a piece of thin foam was rolled (approximately 20cm diameter) and position in the participant mid back. Both feet were firmly positioned on the floor or step and knees kept at a slight bend. Arms were supported at approximately 90 degrees by adjustable harnesses suspended from the laboratory ceiling. This position was chosen for two reasons: (a) it was possible to maintain air flow in the posterior leg and back area; and (b) this position was a better representation of cycling position and allowed for minimal postural change when moving to the exercise segment of the protocol. It was of importance to keep postural changes to a minimum to avoid orthostatic mediated influence to body temperatures. Body temperature changes associated with postural changes have been demonstrated more recently by Tikuisis and Ducharme (1996) and Kenny et al. (2000).

Following instrumentation, participants remained in an upright supported position in ambient conditions of 25°C and 22% relative humidity for 50±12.5 minutes (pre-exercise resting). Participants were prepared to exercise when esophageal temperature did not vary more than ±0.1°C the final 5 minutes of the pre-exercise resting period. Participants were then required to cycle on a Monark stationary cycle ergometer at a work output that elicited 70% of their maximal oxygen consumption for a duration that would result in an accumulation of heat produced by exercise above metabolic rate of 1000 kJ. During exercise, heat produced by exercise above metabolic rate was monitored via laptop computer. Manual data entries for RMR, mechanical work and energy expenditure were entered in a pre-created spreadsheet. The spreadsheet was created in such a way that exercise energy expenditure entered every

minute had RMR and mechanical work subtracted and then was summed to give an approximate heat production by exercise above metabolic rate.

The exercise bout was then followed by 120 minutes of recovery in standing supported position and in ambient conditions of 25 °C and 22% relative humidity.

4. Data Analysis

Values are presented as 1-minute averages at 15, 10 and 5 minutes pre-exercise, at onset of exercise, at 5, 10 and 15 during exercise, at end of the exercise, and at every 5 minutes post-exercise. All means are presented with a standard deviation. Only the first 15 minutes of exercise were average because exercise times varied and every participant has at least 15 minutes of exercise. Single pre-exercise resting data are presented as the average of 10 minutes before move to bike. Statistical analyses for esophageal temperature, mean skin temperature, muscle temperatures(active and inactive), skin blood flow, energy expenditure and mean dry heat loss were performed by one way ANOVA measures to compare these values to exercise and post exercise values every 5 minutes. A *post hoc* Tukey's test at 0.05 significance was then administered. To determine significant differences between esophageal temperature, quadriceps and triceps muscle temperatures, a multivariate analysis was done, followed by a Tukey's *post hoc* test at 0.05 significance. Automated and manual blood pressure results were not considered in the data analysis given their inconsistent nature. There was a high variability in systolic and diastolic measures throughout the sampling times. This variability was caused by not sampling in 2-minute intervals, which caused a high degree of compression in the middle digit.

CHAPTER IV

RESULTS

1. Pre-Exercise Resting

a. Resting temperatures. Pre-exercise resting esophageal (T_{es}), rectal (T_{rec}) and mean skin (T_{skn}) temperatures were 36.95±0.22 °C, 37.21±0.20 °C and 32.46±0.79 °C, respectively (see Figure 1). Mean skin temperature remained significantly ($p<0.05$) below esophageal temperature and non-significantly below active and inactive intra-muscular temperatures for the duration of the pre-exercise rest. During this period, quadriceps muscle temperatures at a depth of 10mm (T_{muq1}), 25mm (T_{muq2}), 40mm (T_{muq3}) and 55mm (T_{muq4}) from the deep femoral artery were 36.30±0.40 °C, 36.08±0.53 °C, 35.72±0.91 °C and 35.33±0.96 °C, respectively (see Figure 1). Triceps brachii muscle temperatures were 35.8±0.70 °C, 35.48±0.88 °C and 34.95±1.17 °C at a depth of 10mm (T_{mut1}), 25mm (T_{mut2}) and 40mm (T_{mut3}) from the humerus. Pre-exercise resting quadriceps muscle temperatures (T_{muq1}, T_{muq2} and T_{muq3}) and the deepest triceps temperature (T_{mut1}) remained non-significantly lower than esophageal temperature for the duration of the pre-exercise period. T_{muq4}, T_{mut2} and T_{mut3} were all significantly ($p< 0.05$) lower than esophageal temperature. The largest gradient between esophageal temperature and active muscle tissue temperature during rest was with T_{muq4}. Gradients between intra-muscular temperatures, from deepest to most superficial, and esophageal temperature were 0.74±0.39, 0.95±0.55, 1.31±0.92 and 1.60±0.95°C, respectively (see Table 4). The temperature gradients between esophageal temperature and inactive muscle tissue from deepest to most superficial were 1.21±0.69, 1.56±0.91 and 2.09±1.17°C (see Table 5) with the most outer site T_{mut3} having the greatest gradient.

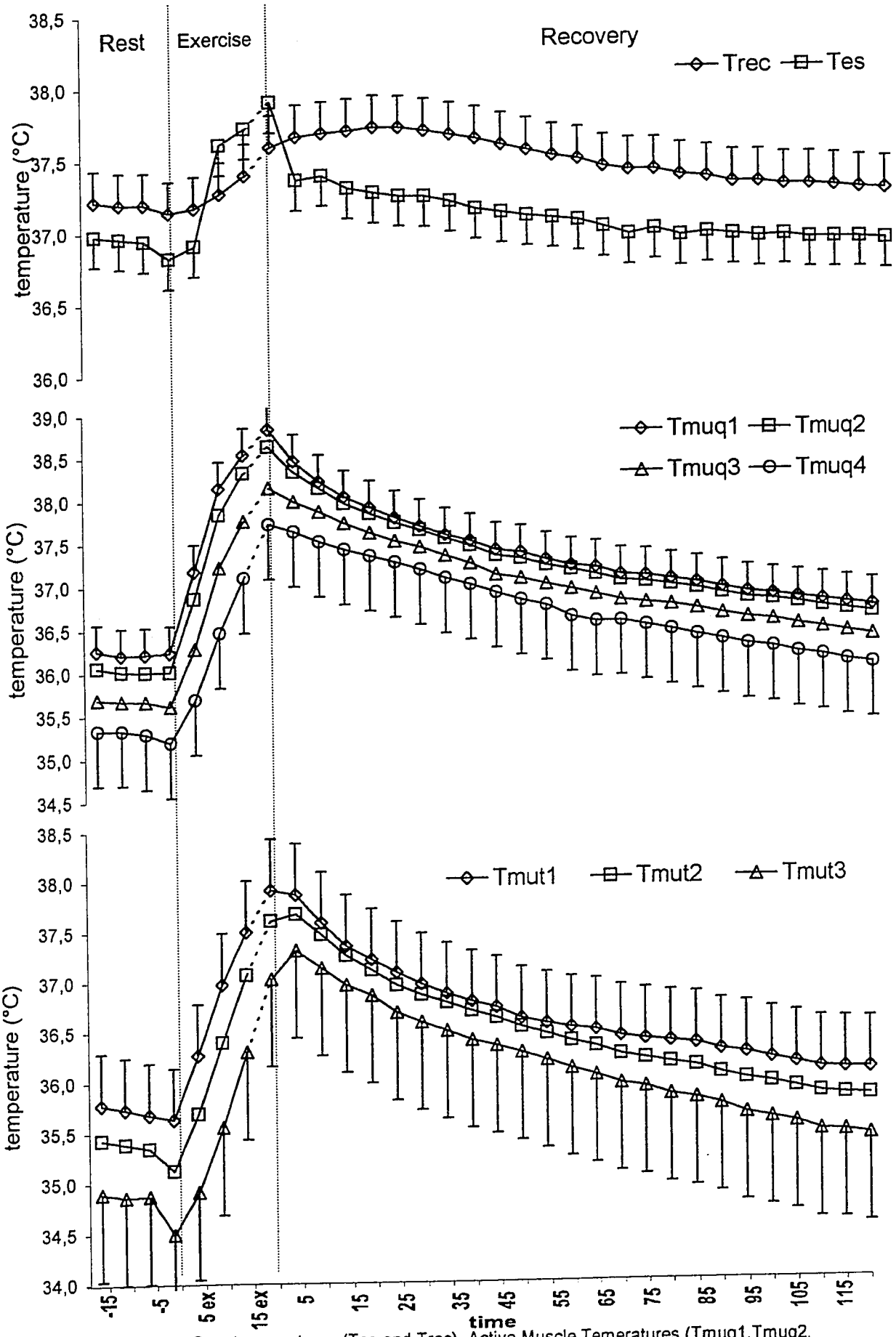


Figure 1 : Mean(+SD) Body Core temperatures (Tes and Trec), Active Muscle Temeratures (Tmuq1,Tmuq2,

Table 4

Mean Esophageal Temperature, Vastus Medialis Muscle Temperatures and Temperature Gradients

	Pre-exercise resting	End exercise	Post-exercise recovery									
			5 min	10 min	15 min	30 min	45 min	60 min	75 min	90 min	105 min	120 min
T_{es} (°C)	36.95	37.90*	37.35*	37.38*	37.29	37.23	37.11	37.05	36.98	36.94	36.91	36.90
Stdev (±)	0.22	0.33	0.21	0.23	0.23	0.19	0.22	0.23	0.21	0.14	0.13	0.20
T_{muq1} (°C)	36.31	38.81*	38.44*†	38.20*†	38.02*	37.67*	37.39*	37.19*	37.06*	36.92*	36.79	36.69
Stdev (±)	0.39	0.42	0.38	0.34	0.32	0.20	0.29	0.30	0.28	0.29	0.28	0.29
T_{muq2} (°C)	36.10	38.61*	38.31*†	38.12*†	37.94*	37.63*	37.32*	37.14*	36.99*	36.85*	36.73	36.61
Stdev (±)	0.50	0.60	0.33	0.30	0.29	0.17	0.20	0.19	0.21	0.23	0.25	0.29
T_{muq3} (°C)	35.75	38.13*	37.97*	37.85*	37.71*	37.43*	37.10*	36.92*	36.75	36.61	36.48	36.35
Stdev (±)	0.84	0.75	0.44	0.39	0.37	0.33	0.41	0.43	0.44	0.43	0.49	0.52
T_{muq4} (°C)	35.33†	37.71*	37.62*	37.50*	37.41*	37.17*	36.89*	36.60*	36.49	36.31	36.15	36.02
Stdev (±)	0.88	0.96	0.70	0.55	0.47	0.43	0.44	0.47	0.50	0.54	0.62	0.63
T_{es}-T_{muq1} (°C)	0.74	-0.85	-1.06	-0.80	-1.00	-0.42	-0.25	-0.13	-0.07	0.02	0.13	0.21
Stdev (±)	0.39	0.27	0.28	0.30	0.21	0.10	0.19	0.22	0.22	0.26	0.27	0.32
T_{es}-T_{muq2} (°C)	0.95	-0.72	-0.96	-0.74	-0.65	-0.40	-0.21	-0.09	-0.01	0.09	0.18	0.29
Stdev (±)	0.55	0.35	0.21	0.23	0.19	0.13	0.21	0.25	0.22	0.24	0.26	0.31
T_{es}-T_{muq3} (°C)	1.31	-0.24	-0.62	-0.47	-0.42	-0.20	0.02	0.13	0.23	0.32	0.43	0.55
Stdev (±)	0.92	0.56	0.39	0.34	0.34	0.35	0.44	0.51	0.45	0.46	0.49	0.51
T_{es}-T_{muq4} (°C)	1.60	0.21	-0.25	-0.12	-0.12	0.06	0.23	0.45	0.49	0.63	0.76	0.88
Stdev (±)	0.95	0.81	0.63	0.49	0.40	0.40	0.44	0.51	0.49	0.56	0.61	0.61

Note: T_{es}= esophageal temperature, T_{muq1}= muscle temperature 10mm from the femur, T_{muq2}= muscle temperature 25mm from the femur, T_{muq3}= muscle temperature 40mm from the femur, T_{muq4}= muscle temperature 55mm from the femur, *= significantly different from baseline values ($p<0.05$), †=significantly different from esophageal temperature ($p<0.05$). No statistical analysis was done for T_{es}-T_{muq1}, T_{es}-T_{muq2}, T_{es}-T_{muq3} or T_{es}-T_{muq4}

Table 5

Mean Esophageal Temperature, Medial Head of Triceps Muscle Temperature and Temperature Gradients

	Pre-exercise resting	End exercise	Post-exercise recovery									
			5 min	10 min	15 min	30 min	45 min	60 min	75 min	90 min	105 min	120 min
T_{es} (°C)	36.95	37.90*	37.35*	37.38*	37.29	37.23	37.11	37.05	36.98	36.94	36.91	36.90
Stdev (±)	0.22	0.33	0.21	0.23	0.23	0.19	0.22	0.23	0.21	0.14	0.13	0.20
T_{mut1} (°C)	35.82	37.90*	37.86*	37.57*	37.34*	36.95*	36.71	36.50	36.38	36.27	36.13	36.06
Stdev (±)	0.70	0.44	0.45	0.44	0.41	0.37	0.39	0.46	0.44	0.50	0.56	0.59
T_{mut2} (°C)	35.48†	37.60*	37.66*	37.46*	37.25*	36.84*	36.61	36.37	36.11†	36.03 †	35.89 †	35.80†
Stdev (±)	0.89	0.62	0.42	0.39	0.37	0.49	0.66	0.73	0.65	0.66	0.72	0.74
T_{mut3} (°C)	34.95 †	37.02*	37.30*	37.12*	36.96*	36.57*	36.33†	36.10†	35.91†	35.73†	35.54†	35.41†
Stdev (±)	1.18	0.79	0.64	0.53	0.50	0.58	0.78	0.84	0.81	0.86	0.97	1.06
T_{es}-T_{mut1} (°C)	1.21	-0.01	-0.51	-0.19	-0.05	0.28	0.40	0.55	0.60	0.67	0.78	0.83
Stdev (±)	0.69	0.45	0.42	0.38	0.40	0.28	0.31	0.38	0.33	0.45	0.53	0.63
T_{es}-T_{mut2} (°C)	1.56	0.29	-0.31	0.07	0.05	0.39	0.50	0.68	0.80	0.91	1.02	1.10
Stdev (±)	0.91	0.59	0.38	0.31	0.33	0.40	0.58	0.64	0.56	0.62	0.69	0.77
T_{es}-T_{mut3} (°C)	2.09	0.87	0.05	0.26	0.34	0.66	0.78	0.95	1.08	1.21	1.37	1.49
Stdev (±)	1.17	0.76	0.55	0.48	0.41	0.49	0.70	0.74	0.74	0.83	0.95	1.09

Note: T_{es}= esophageal temperature, T_{mut1}= muscle temperature 10mm from humerus, Tmut2= muscle temperature 25mm from humerus, Tmut3= muscle temperature 40mm from humerus, *= significantly different from baseline values ($p<0.05$) †=significantly different from esophageal temperature ($p<0.05$). No statistical analysis was done on temperature differences

b. Heat loss, heart rate and blood flow. The resting mean rate of dry heat loss was 6.04±0.73 kJ/min and resting rate of energy expenditure was 6.08±2.18 kJ/min. The average resting heart rate for the pre-exercise period was 66±5 beats/min. Throughout pre-exercise rest, both cutaneous blood flow sites (anterior forearm and anterior thigh) were stable (see Figure 2). However, there was more variability in the intra-muscular blood flow site. This increased variability is due to probe sensitivity to minor upper limb postural adjustments during resting period.

2. Exercise

a. Heat produced and heat loss response. In setting a specific target for heat production as a function of work intensity, each participant reached approximately the same heat produced by exercise above metabolic rate but had varying exercise times (see Table 6). The average total energy expenditure, total work and mechanical efficiency for the 21.9±4.3 minutes of exercise were 1276±104 kJ, 216.9± 26.8kJ and 17.2±1.3%, respectively (see Table 6). The average total dry skin heat loss produced by exercise was 92.0±22.9 and heat produced by exercise above metabolic rate was 930.85±78.81 kJ (see Table 6).

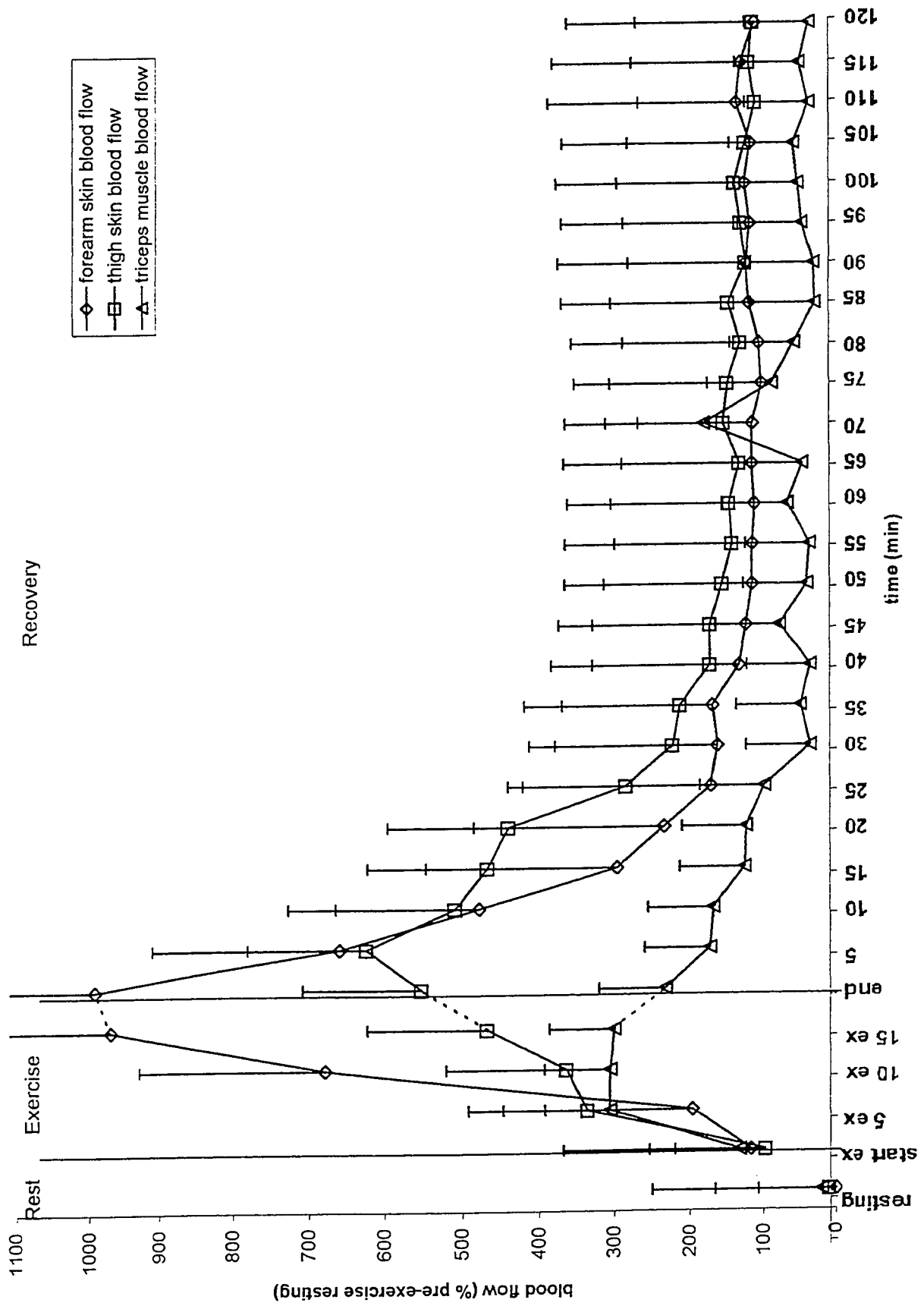


Figure 2: Mean(±SD) Forearm Skin Blood Flow, Thigh Skin Blood Flow and Triceps Muscle Blood Flow, Before, During and After Exercise.

Table 6

Individual and Mean Mechanical Energy, Heat Produced by Exercise above Metabolic Rate and Heat Loss Data

Participant	Exercise time (min)	M.E. (%)	E_{tot} (kJ)	W_{tot} (kJ)	RMR (kJ/min)	Hpemr (kJ)	Hlex (kJ)	Hlrec (kJ)
1	22.5	17.44	1244.2	216.6	5.50	904	107.3	77.2
2	24.5	18.1	1311.7	235.8	4.94	956	87.6	33.7
3	27.5	19.35	1373.5	264.7	6.77	923	141.6	29.8
4	19	16.64	1325.2	219.5	9.90	918	86.1	37.7
5	17	15	1326.3	196.4	7.73	999	76.5	139.1
6	15.5	17.31	1036	179	5.89	766	82.7	74.1
7	23.5	17.43	1308.9	226.2	2.31	1028	86.1	68.2
8	25.5	16.49	1280.9	196.4	5.87	956	68.1	15.4
Mean	21.88	17.22	1275.8	216.9	6.01	931	92.0	59.4
Stdev (±)	4.27	1.27	103.82	26.77	2.22	78.8	22.9	39.5

Note: E_{tot}= total energy expenditure, M.E.= mechanical efficiency, kJ= kilojoules, stdev= standard deviation, Hpemr = heat produced by exercise above metabolic rate, Hlex= dry heat loss during exercise. Hlrec= dry heat loss during 65 minutes recovery, W_{tot}= work during exercise.

b. Temperature response. At the onset of exercise, there was a non-significant decrease in esophageal temperature, all three triceps muscle temperatures, T_{muq4} quadriceps muscle temperature and mean skin temperature. The most pronounced decrease in temperature at onset of exercise was shown to be T_{mut3} in the triceps (-0.46 °C) (see Figure 1). Throughout the exercise period, there was a steady increase in all measured parameters (esophageal temperature, active and inactive muscle temperature and mean skin temperature). Exercise produced a significant ($p< 0.05$) increase of 0.94±0.32°C in esophageal temperature over pre-exercise value. End exercise esophageal and rectal temperatures were 37.90±0.33°C and 37.58±0.26°C, respectively. Also, throughout exercise, temperature gradients between esophageal temperature and muscle temperatures gradually changed. After 15 minutes

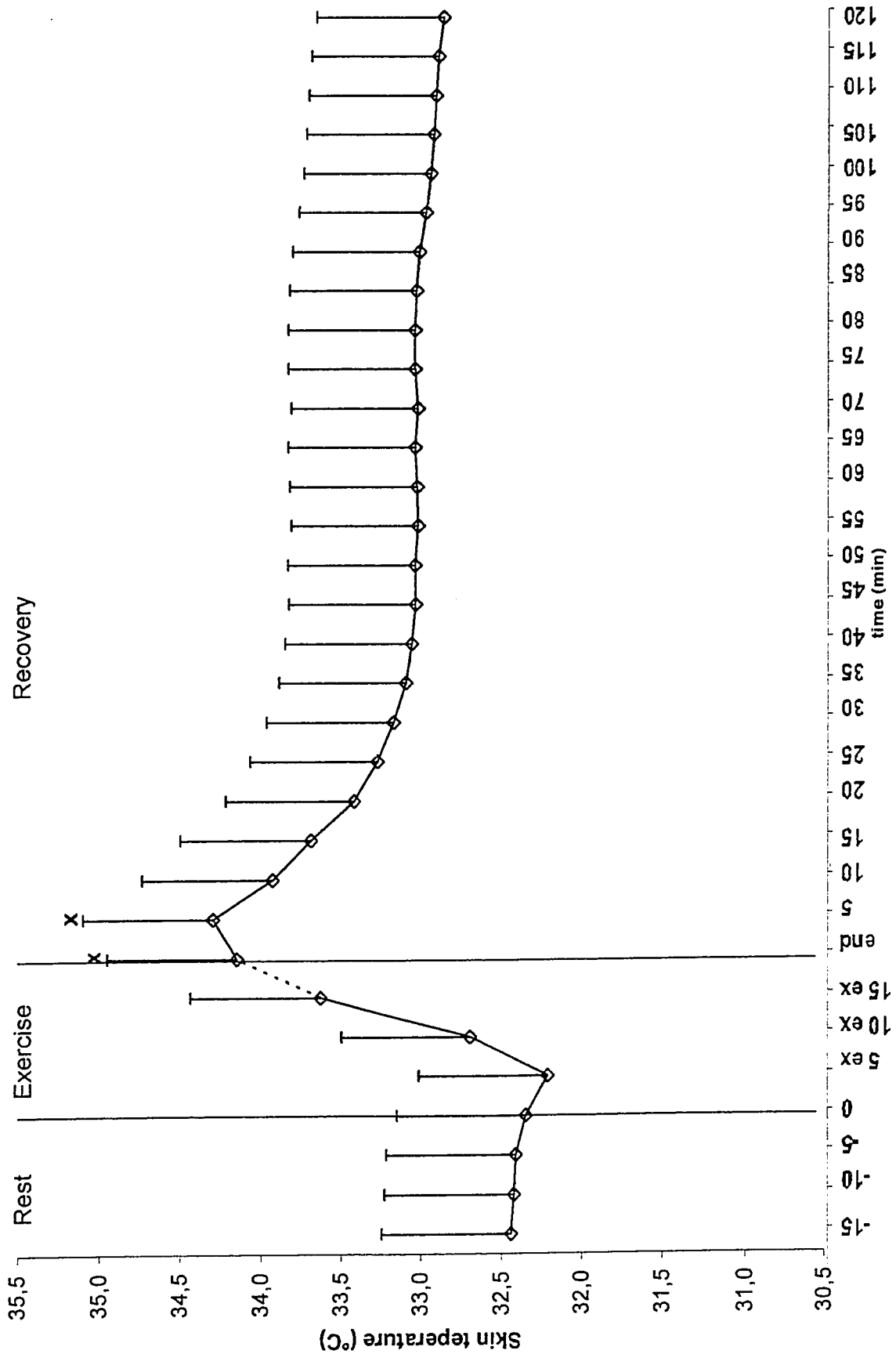


Figure 3: Mean(±SD)Skin Temperatue Before, During and Following Exercise. x= significant difference from pre-exercise($p<0.05$).

of exercise, gradients between esophageal temperature and active muscle temperatures were -0.76±0.25°C, -0.59±0.30°C, -0.03±0.62°C and 0.63±0.97°C from deepest to most superficial. At the same time, gradients between esophageal temperature and inactive muscle tissue sites were 0.22±0.40°C, 0.65±0.66°C and 1.42±0.83°C from deepest to most superficial. At end of exercise, mean skin temperature increased significantly ($p < 0.05$) to 34.14±1.05 °C (see Figure 3) with an elevation of 1.68±0.44°C above pre-exercise resting values.

Quadriceps muscle temperatures T_{muq1}, T_{muq2}, T_{muq3} and T_{muq4} were 38.81±0.42°C, 38.61±0.60°C, 38.13±0.75°C and 37.71±0.96°C, respectively, and triceps muscle temperatures T_{mut1}, T_{mut2} and T_{mut3} were 37.90±0.44°C, 37.60±0.62°C and 37.02±0.79°C, respectively, at the end of exercise. All end of exercise muscle temperatures were significantly ($p < 0.05$) elevated from their pre-exercise resting values. Prior to exercise the temperature gradient between esophageal temperature and T_{muq1} was 0.64 °C. At end of exercise, this gradient was reversed with T_{muq1} being 0.91°C above esophageal temperature (see Figure 4). All end of exercise temperature gradients between esophageal temperature and quadriceps and triceps muscle temperatures are presented in Tables 4 and 5.

During exercise, the average rate of increase in esophageal temperature was 0.0018±0.0002 °C/kJ for the first 400kJ. This rate then decreased to 0.0006±0.0004°C/kJ at the end of exercise (see Figure 5). The average rate in the active muscle from the onset of exercise to 400kJ was 0.0043±0.0008 °C/kJ, 0.0043±0.0006 °C/kJ, 0.0038±0.0006 °C/kJ and 0.0031±0.0004 °C/kJ at T_{muq1}, T_{muq2}, T_{muq3} and T_{muq4} respectively. After 400kJ the average rate of in the quadriceps was 0.0014±0.0006 °C/kJ, 0.0015±0.0008 °C/kJ, 0.0019±0.0005 °C/kJ and 0.0023±0.0006 °C/kJ at T_{muq1}, T_{muq2}, T_{muq3} and T_{muq4} respectively (see Figure 6). The rate in inactive muscle tissue was constant throughout the exercise: these rates were

found to be 0.0030±0.0002 °C/kJ, 0.0028±0.0003 °C/kJ and 0.0023±0.0004 °C/kJ at T_{mut1}, T_{mut2} and T_{mut3}, respectively (see Figure 7). The temperature rate increase expressed in °C/min for esophageal temperature, active and passive muscle temperatures are presented in table 7.

Table 7

Mean Esophageal Temperature, Active and Passive Muscle Temperature Rates of Increase during Exercise

	0-5 min	5-10 min	10-15 min
T_{es} °C/min	0.02	0.14	0.02
Stdev (±)	0.03	0.04	0.04
T_{muq1} °C/min	0.19	0.19	0.08
Stdev (±)	0.09	0.06	0.06
T_{muq2} °C/min	0.17	0.20	0.10
Stdev (±)	0.12	0.07	0.06
T_{muq3} °C/min	0.13	0.19	0.11
Stdev (±)	0.12	0.08	0.09
T_{muq4} °C/min	0.10	0.15	0.13
Stdev (±)	0.12	0.09	0.09
T_{mut1} °C/min	0.13	0.14	0.10
Stdev (±)	0.08	0.04	0.05
T_{mut2} °C/min	0.11	0.14	0.13
Stdev (±)	0.08	0.04	0.06
T_{mut3} °C/min	0.08	0.13	0.15
Stdev (±)	0.06	0.05	0.07

Note: All temperature rates of increase are expressed in °C/min, T_{es} = esophageal temperature T_{muq1}= muscle temperature 10mm from the femur, T_{muq2}= muscle temperature 25mm from the femur, T_{muq3}= muscle temperature 40mm from the femur, T_{muq4}= muscle temperature 55mm from the femur, T_{mut1}= muscle temperature 10mm from humerus, T_{mut2}= muscle temperature 25mm from humerus, T_{mut3}= muscle temperature 40mm from humerus.

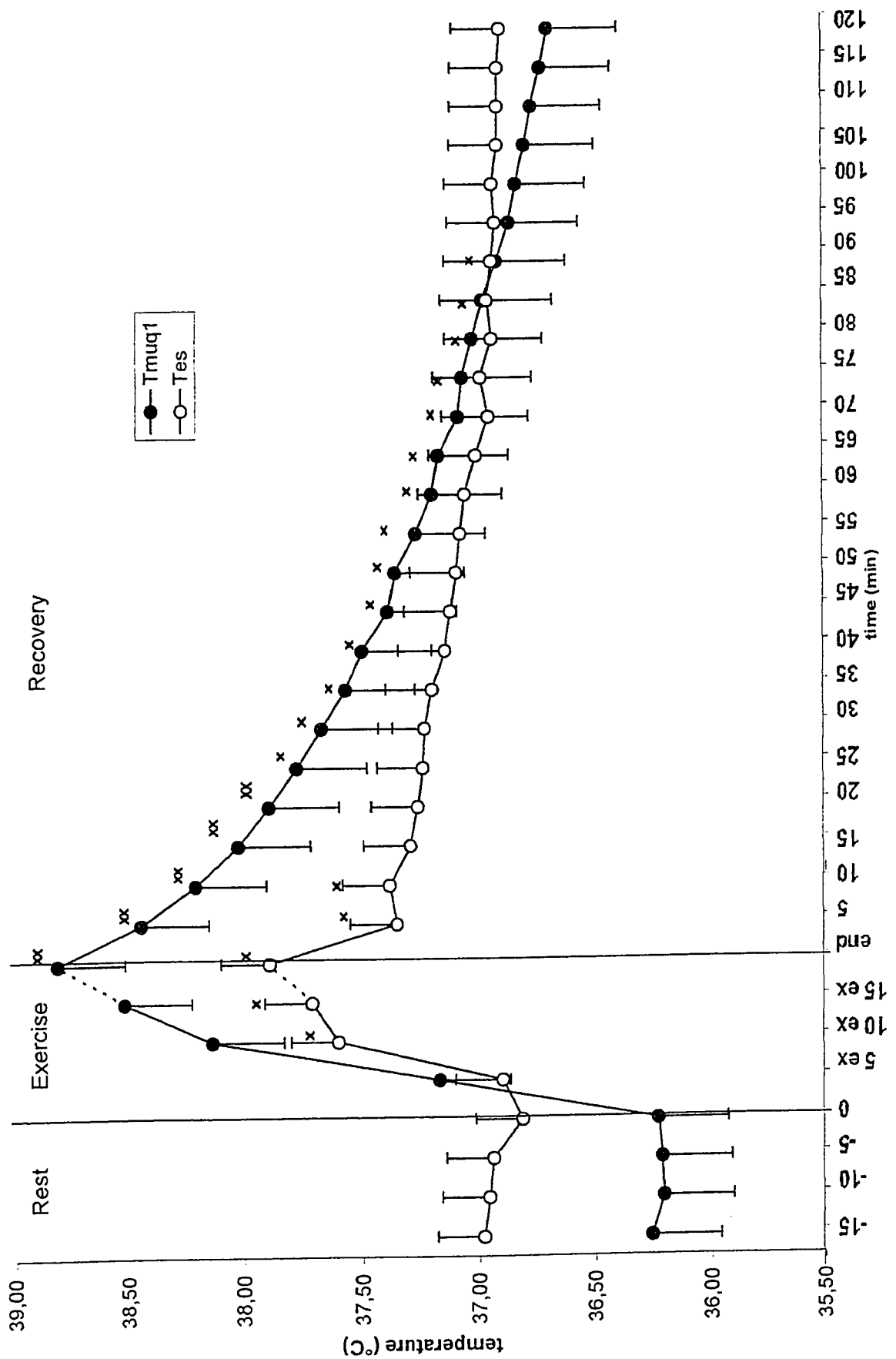
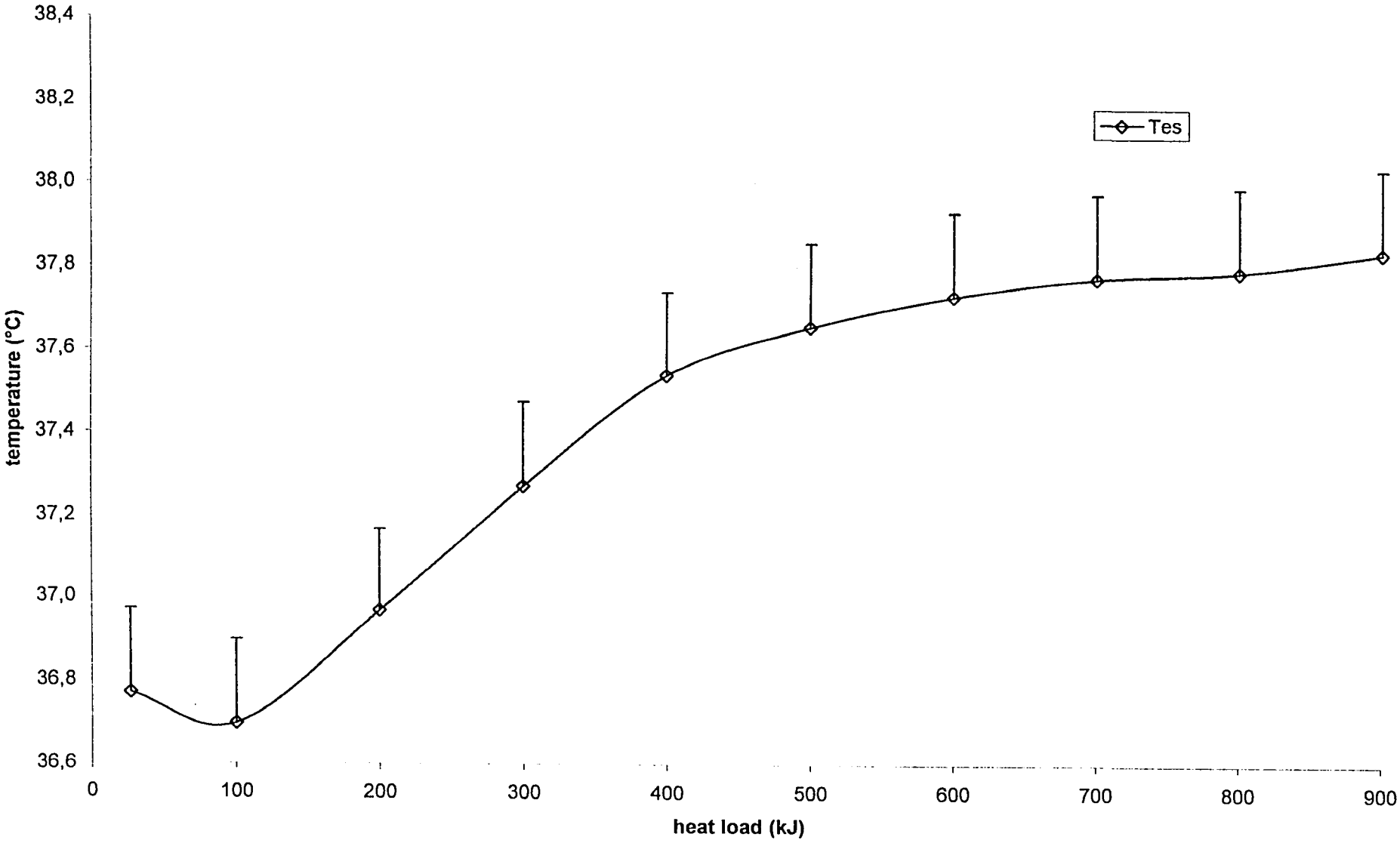


Figure 4: Mean (±SD) Esophageal Temperature (Tes) and Deep Quadriceps Temperature (Tmuq1) Before, During and After Exercise. X= significant difference from baseline ($p<0.05$) XX= also significantly different form Tes



Figures 5: Mean (±SD) Esophageal Temperature Profile During Exercise According to Cumulative Heat Produced by Exercise above Metabolic Rate

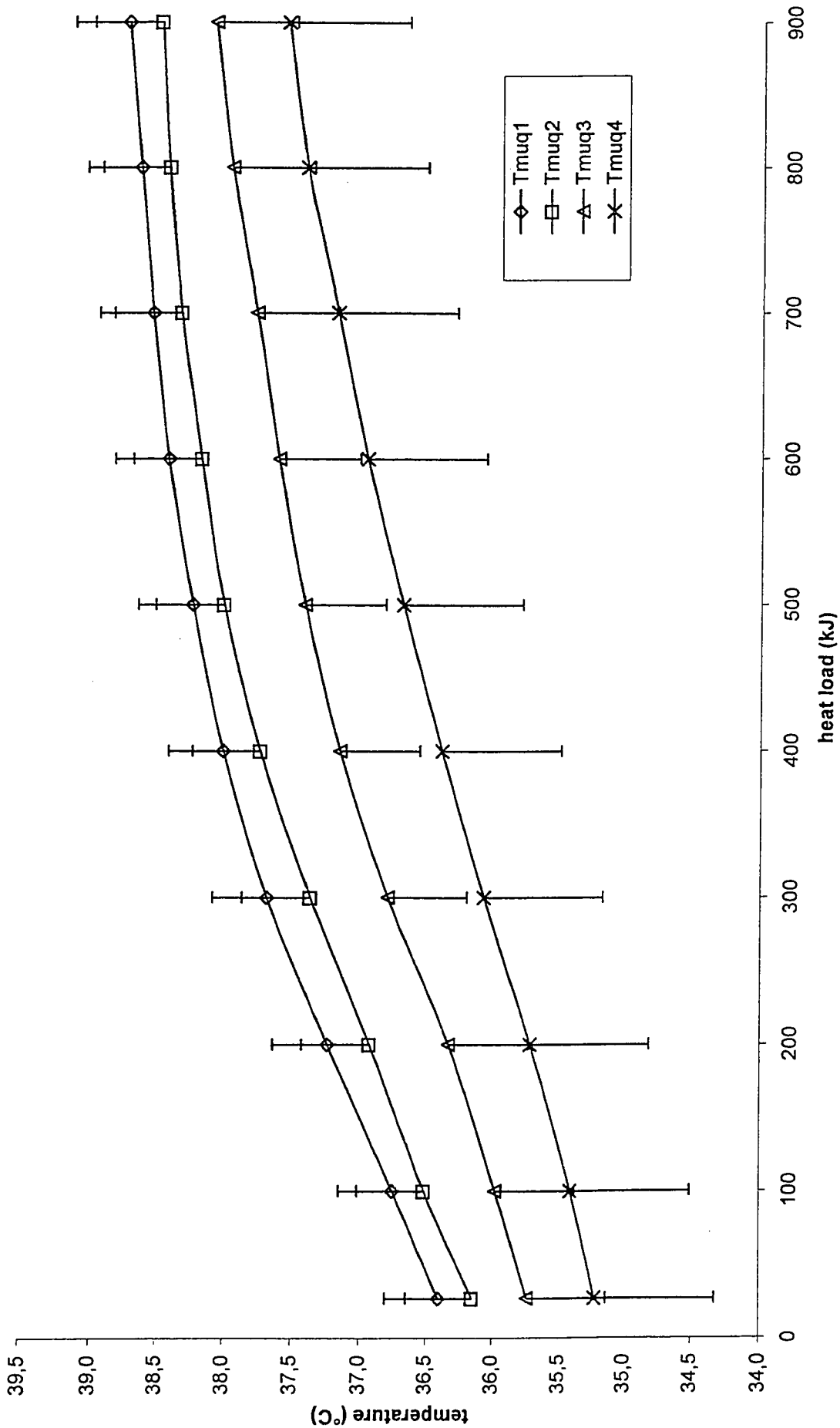


Figure 6: Mean (±SD) Active Muscle Temperature Profile During Exercise According to Cumulative Heat Produced by Exercise above Metabolic Rate

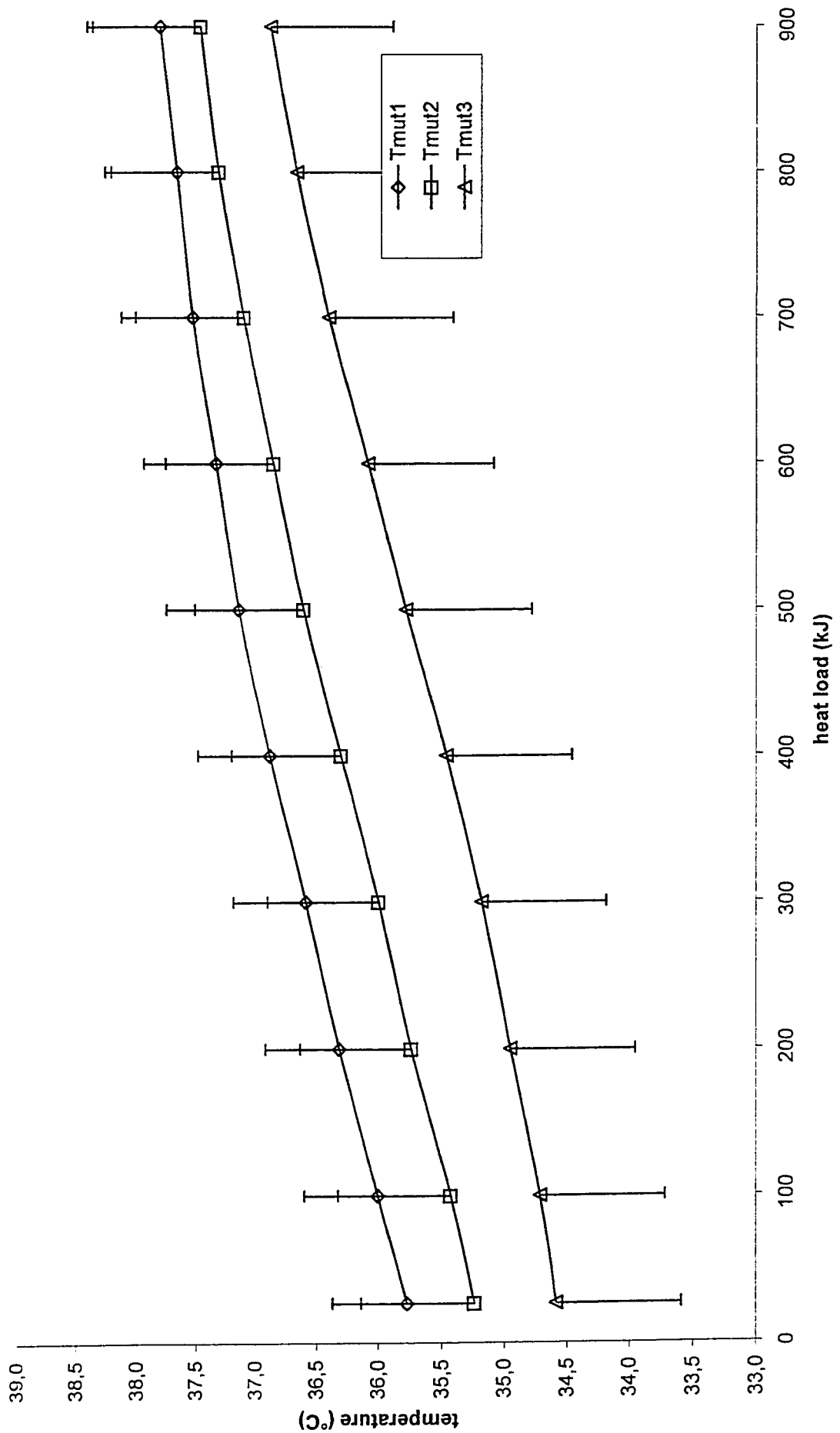


Figure 7: Mean (±SD) Passive Muscle Temperature Profile During Exercise According to Cumulative Heat Produced by Exercise above Metabolic Rate

c. Heart rate and blood flow response. Heart rate increased significantly ($p<0.05$) to 139±12 beats/min within 5 minutes of onset of cycle ergometry. Heart rate showed a gradual but slower increase over the duration of exercise to 152±11 beats/min at 10 minutes exercise. Average end of exercise heart rate was 158±10 beats/min (see Figure 8). Within the first 5 minutes, anterior thigh cutaneous blood flow and anterior forearm cutaneous blood flow were elevated about 300% over pre-exercise resting values. Throughout exercise, there was a continued increase in these parameters with anterior forearm and anterior thigh reaching level of 1000 % and 550 % respectively over their pre-exercise resting values at end exercise (see Figure 2). At end exercise, thigh cutaneous blood flow was significantly ($p<0.05$) elevated over pre-exercise values. Inactive intra-muscular blood flow responded in a slightly different manner. Within the first 5 minutes of exercise, there was a sharp rise in flow (305%) and this was followed by a sustained flow for the duration of exercise (see Figure 2).

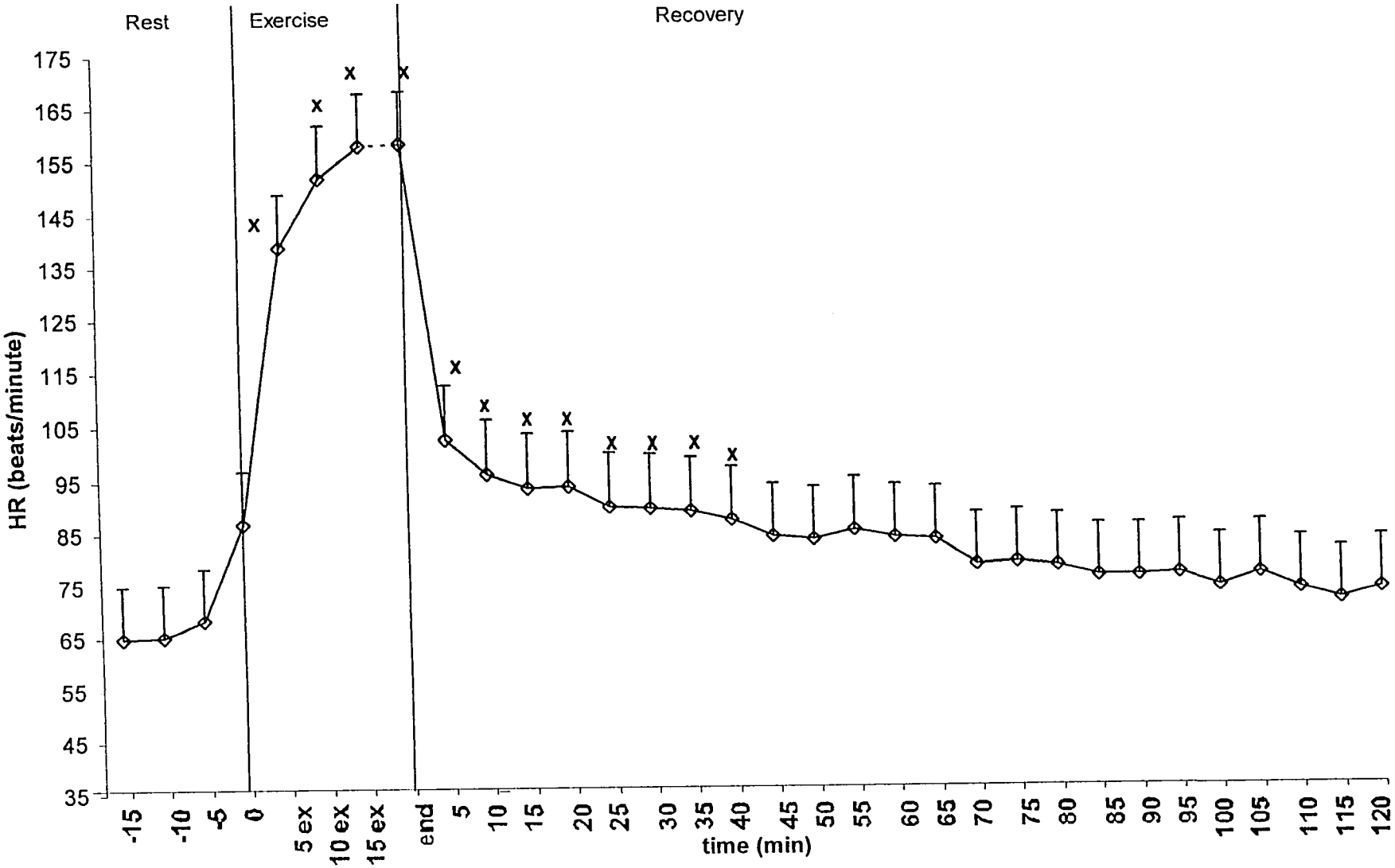


Figure 8: Mean (±SD) Heart Rate (HR) Response Before, During and Following Exercise. X= significance from pre-exercise ($p<0.05$)

3. Post-Exercise Resting Recovery

a. Temperature response. Following exercise, esophageal temperature dropped rapidly for the first few minutes and remained significantly elevated from pre-exercise resting values for 10 minutes following exercise (see Figure 4). In contrast, rectal temperature continued to rise and reached a maximum increase of 0.5 °C, 20 minutes following exercise but there where no statistical differences observed in rectal temperature change. Active muscle temperatures decreased when exercise ended and remained significantly ($p< 0.05$) elevated from pre-exercise resting values for 90 (T_{muq1}), 115 (T_{muq2}), 85 (T_{muq3}) and 80 (T_{muq4}) minutes. At the end of the 120 minutes recovery, active muscle temperatures were 36.69±0.29°C, 36.61±0.29 °C, 36.35±0.52 °C and 36.02±0.63°C (T_{muq1}, T_{muq2}, T_{muq3} and T_{muq4}), respectively but non-significantly elevated from pre-exercise resting values (see Figure 1). Following exercise, T_{mut3} was below end of exercise esophageal temperature while T_{mut1} and T_{mut2} remained above. For 5 minutes post-exercise, T_{mut1} decreased while T_{mut2} and T_{mut3} continued to increase slightly (0.07±0.34 °C, 0.28±0.31°C respectively) (see Figure 1). Ten minutes into the recovery period, triceps muscle temperatures decreased steadily. Triceps muscle temperatures remained significantly ($p< 0.05$) elevated from their pre-exercise values for 40 (T_{mut1}), 35 (T_{mut2}) and 30 (T_{mut3}) minutes, respectively. At the end of the 120-minute recovery period, triceps muscle temperatures were non significantly elevated from their pre-exercise resting values (see Figure 1). Following 5 minutes of recovery, temperature gradients between esophageal temperature and both quadriceps and triceps temperatures continued to increase given the rapid drop in esophageal temperature (see Figures 9 and 10). This was followed by gradual cooling for the duration of the recovery. Following 120 minutes of

recovery, none of the 4- and 3- point gradients for quadriceps and triceps muscles were re-established (see Table 8).

Table 8

Mean Muscle Temperature Gradients for the Quadriceps and Triceps Muscles

	Pre-exercise resting	End exercise	Post-exercise recovery									
			5 min	10 min	15 min	30 min	45 min	60 min	75 min	90 min	105 min	120 min
Tmuq1-Tmuq2	0.21	0.05	0.06	0.01	0.01	0.00	0.04	0.04	0.07	0.08	0.08	0.11
Stdev (±)	0.05	0.16	0.17	0.16	0.15	0.14	0.19	0.24	0.17	0.15	0.16	0.19
Tmuq1-Tmuq3	0.55	0.49	0.36	0.26	0.22	0.20	0.26	0.27	0.30	0.31	0.32	0.36
Stdev (±)	0.09	0.32	0.47	0.41	0.43	0.39	0.44	0.51	0.48	0.45	0.50	0.52
Tmuq1-Tmuq4	0.91	0.85	0.63	0.57	0.49	0.41	0.44	0.52	0.54	0.59	0.61	0.64
Stdev (±)	0.1	0.51	0.54	0.48	0.42	0.42	0.40	0.49	0.46	0.50	0.58	0.57
Tmuq2-Tmuq3	0.35	0.48	0.35	0.27	0.23	0.20	0.22	0.22	0.24	0.23	0.25	0.26
Stdev (±)	0.05	0.27	0.34	0.30	0.29	0.27	0.29	0.32	0.34	0.33	0.36	0.35
Tmuq2-Tmuq4	0.77	0.92	0.70	0.63	0.53	0.46	0.43	0.51	0.50	0.54	0.58	0.59
Stdev (±)	0.07	0.54	0.51	0.42	0.36	0.36	0.30	0.38	0.38	0.42	0.48	0.45
Tmuq3-Tmuq4	0.42	0.45	0.38	0.35	0.30	0.27	0.21	0.29	0.26	0.31	0.33	0.33
Stdev (±)	0.07	0.31	0.36	0.21	0.26	0.27	0.17	0.24	0.24	0.21	0.20	0.21
Tmut1-Tmut2	0.34	0.31	0.19	0.12	0.10	0.11	0.10	0.14	0.19	0.24	0.24	0.26
Stdev (±)	0.29	0.40	0.29	0.24	0.22	0.24	0.34	0.34	0.34	0.31	0.37	0.37
Tmut1-Tmut3	0.87	0.89	0.56	0.45	0.39	0.38	0.38	0.41	0.47	0.54	0.60	0.66
Stdev (±)	0.56	0.76	0.66	0.56	0.50	0.42	0.50	0.47	0.51	0.50	0.60	0.64
Tmut2-Tmut3	0.54	0.58	0.36	0.33	0.29	0.27	0.28	0.27	0.28	0.30	0.35	0.39
Stdev (±)	0.28	0.45	0.47	0.40	0.34	0.25	0.23	0.22	0.24	0.27	0.29	0.29
Tmuq1-Tmut1	0.36	0.97	0.64	0.67	0.71	0.71	0.63	0.61	0.60	0.54	0.53	0.49
Stdev (±)	0.61	0.50	0.49	0.44	0.41	0.34	0.41	0.49	0.35	0.35	0.35	0.34

Note: All temperature differences are expressed in °C, T_{muq1}= muscle temperature 10mm from the femur, T_{muq2}= muscle temperature 25mm from the femur, T_{muq3}= muscle temperature 40mm from the femur, T_{muq4}= muscle temperature 55mm from the femur, Tmut1= muscle temperature 10mm from humerus, T_{mut2}= muscle temperature 25mm from humerus, T_{mut3}= muscle temperature 40mm from humerus.

Mean skin temperature continued to rise for 5 minutes following cessation of exercise. Following the 5-minute post-exercise rise, mean skin temperature decreased steadily until the

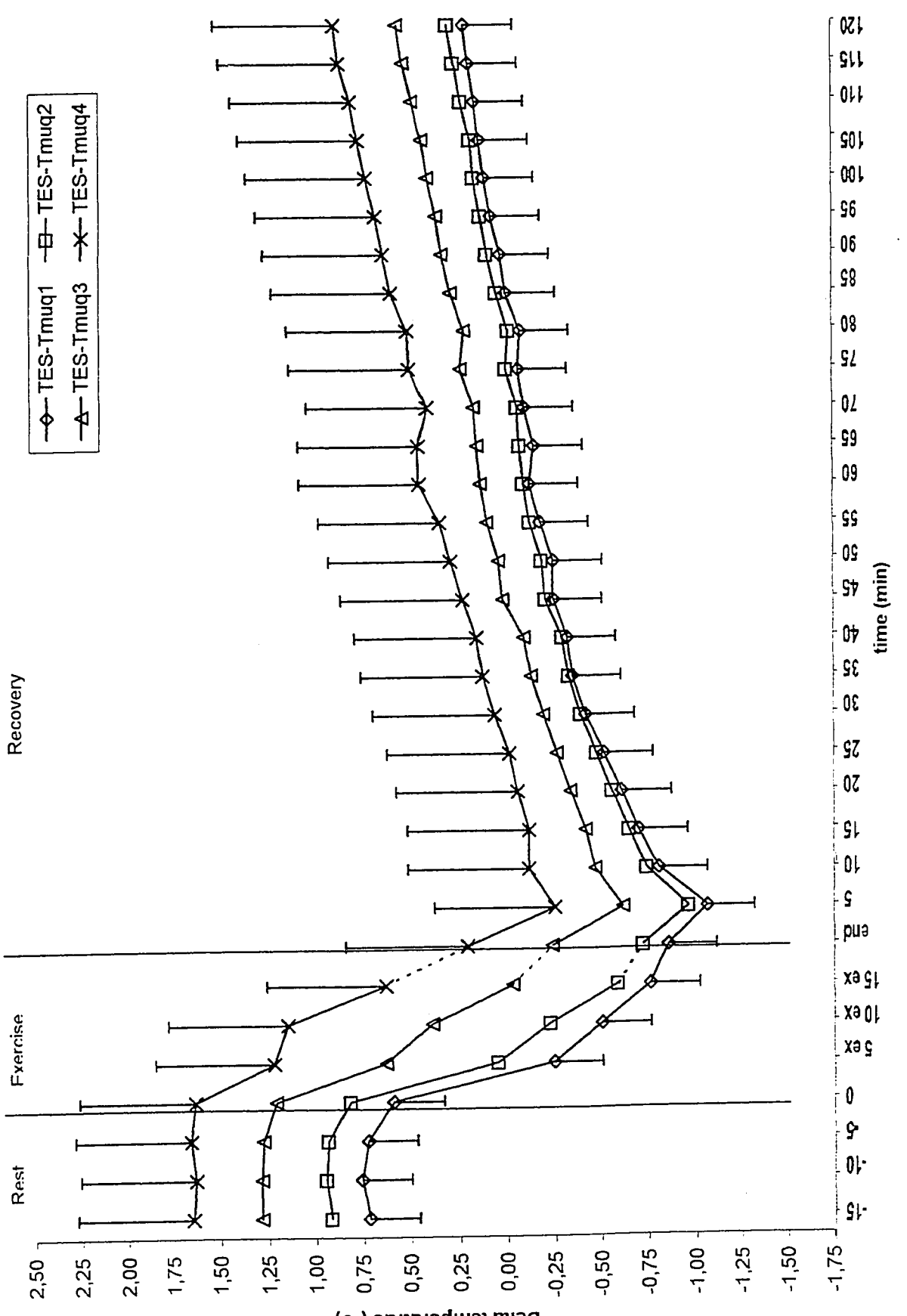


Figure 9: Mean (±SD) Temperature Gradients between Esophageal Temperature and Four Intra Muscular Temperature Sites Before, During and After Exercise

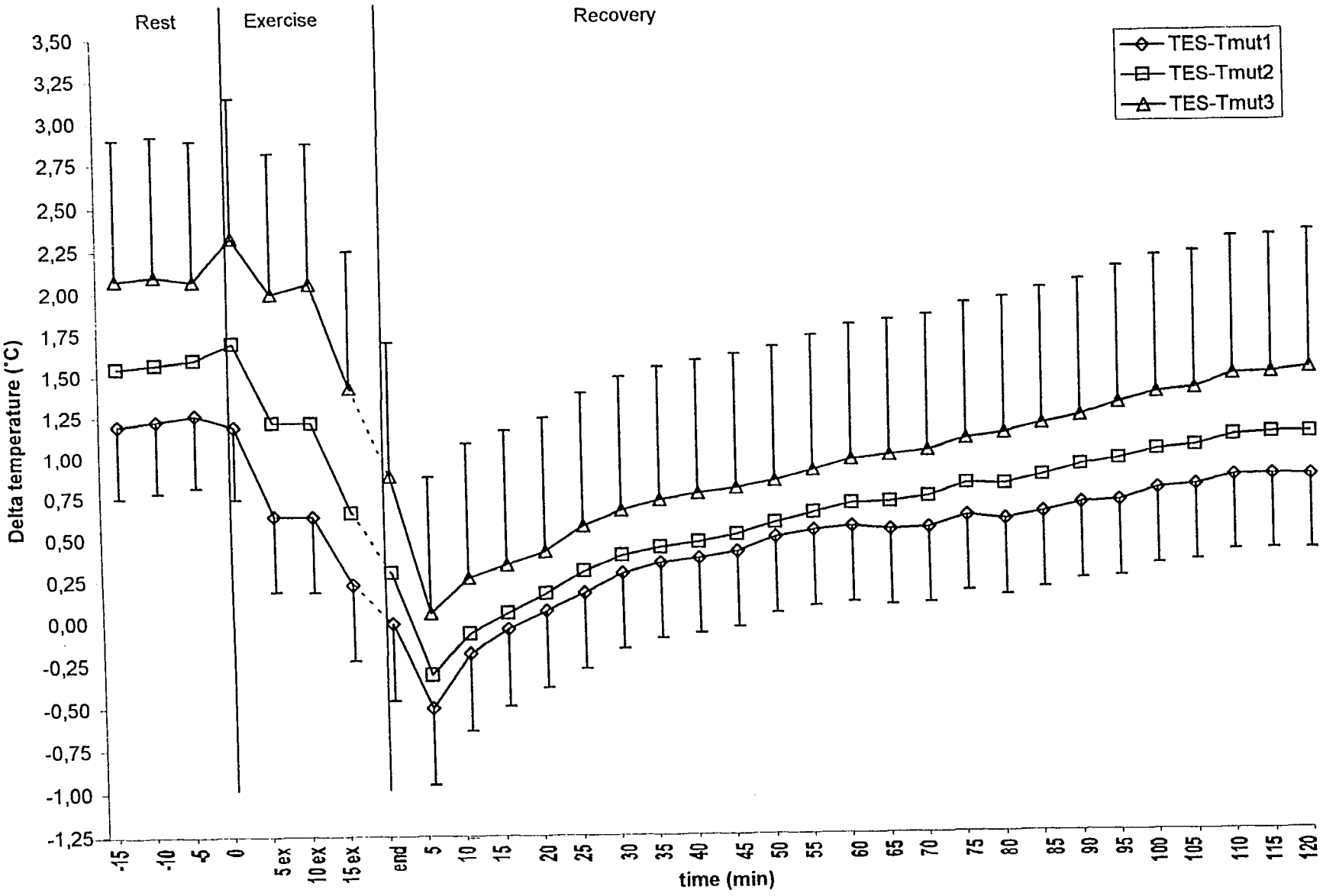


Figure 10: Mean (±SD) Temperature Gradients between Esophageal Temperature and Three Intra Muscular Temperatures Sites Before, During and After Exercise

40-minute post-exercise mark (see Figure 3). At this time, mean skin temperature remained elevated 0.58°C above pre-exercise values (n.s.).

b. Heat loss response. Post-exercise energy expenditure and oxygen consumption rapidly returned to resting values 10 minutes into recovery (see Figures 11 and 13). Cumulative heat loss for 65 minutes of resting recovery was 54.1±20.0 kJ. At 65 minutes into recovery, the rate of energy expenditure and rate heat loss were 7.46±1.68 kJ/min and 6.13±1.15 kJ/min.

c. Heart rate and blood flow response. Post-exercise initial (5 minutes post) recovery heart rate was 102±13 beats/min. During the remainder of the recovery, heart rate slowly returned to pre-exercise resting values but remained significantly ($p<0.05$) elevated for 40 minutes following exercise (see Figure 8). Following exercise, anterior forearm skin blood flow and intra-muscular blood flow did not present any statistical differences compared to their pre-exercise resting values but anterior thigh skin blood flow did remain significantly ($p<0.05$) elevated for 10 minutes above pre-exercise values. This followed by a gradual return to pre-exercise values. Throughout recovery both cutaneous blood flow site remained 100% above resting values and intra-muscular blood flow about 35 %.

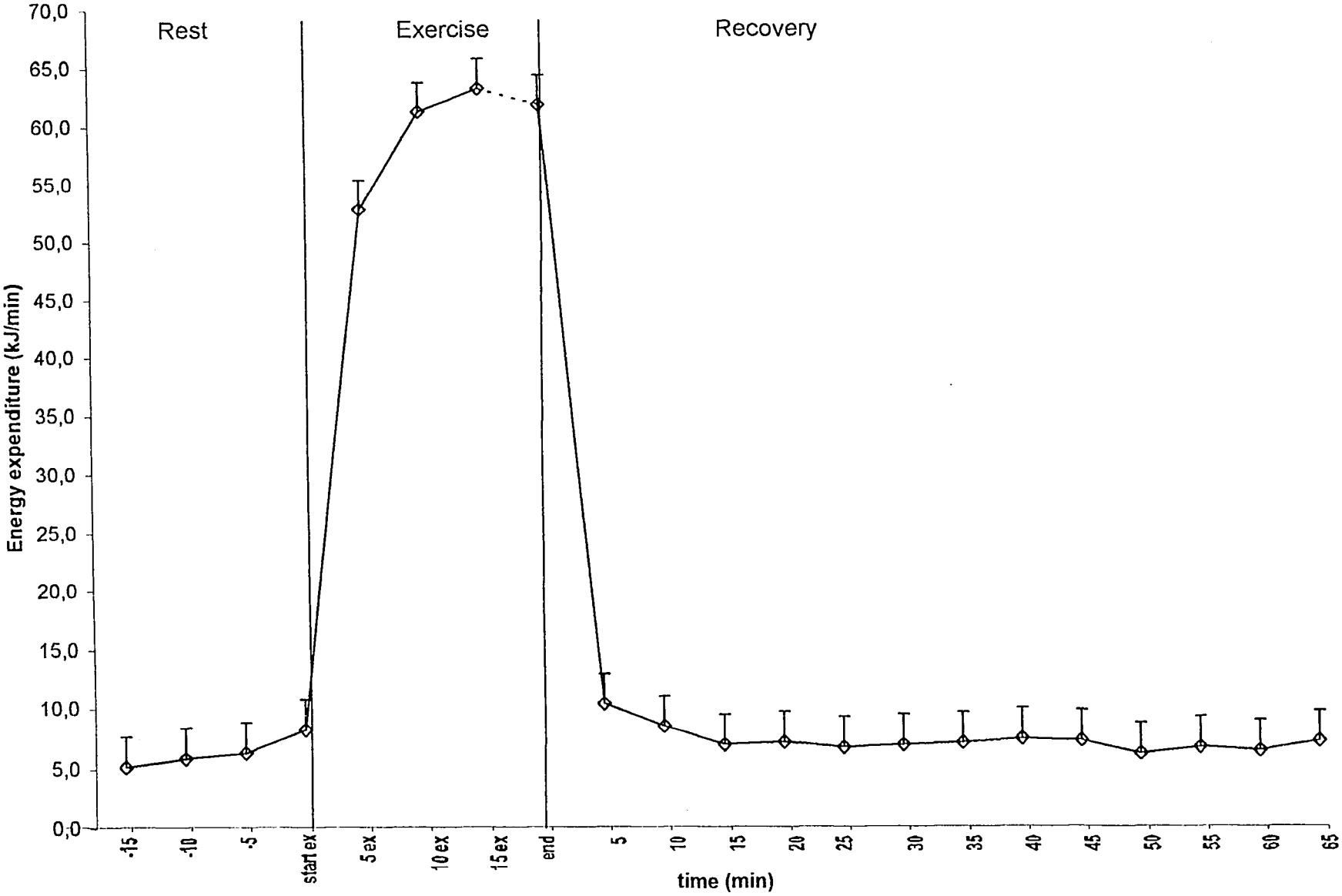


Figure 11 : Mean (±SD) Energy Expenditure Before, During and 65 minutes After Exercise

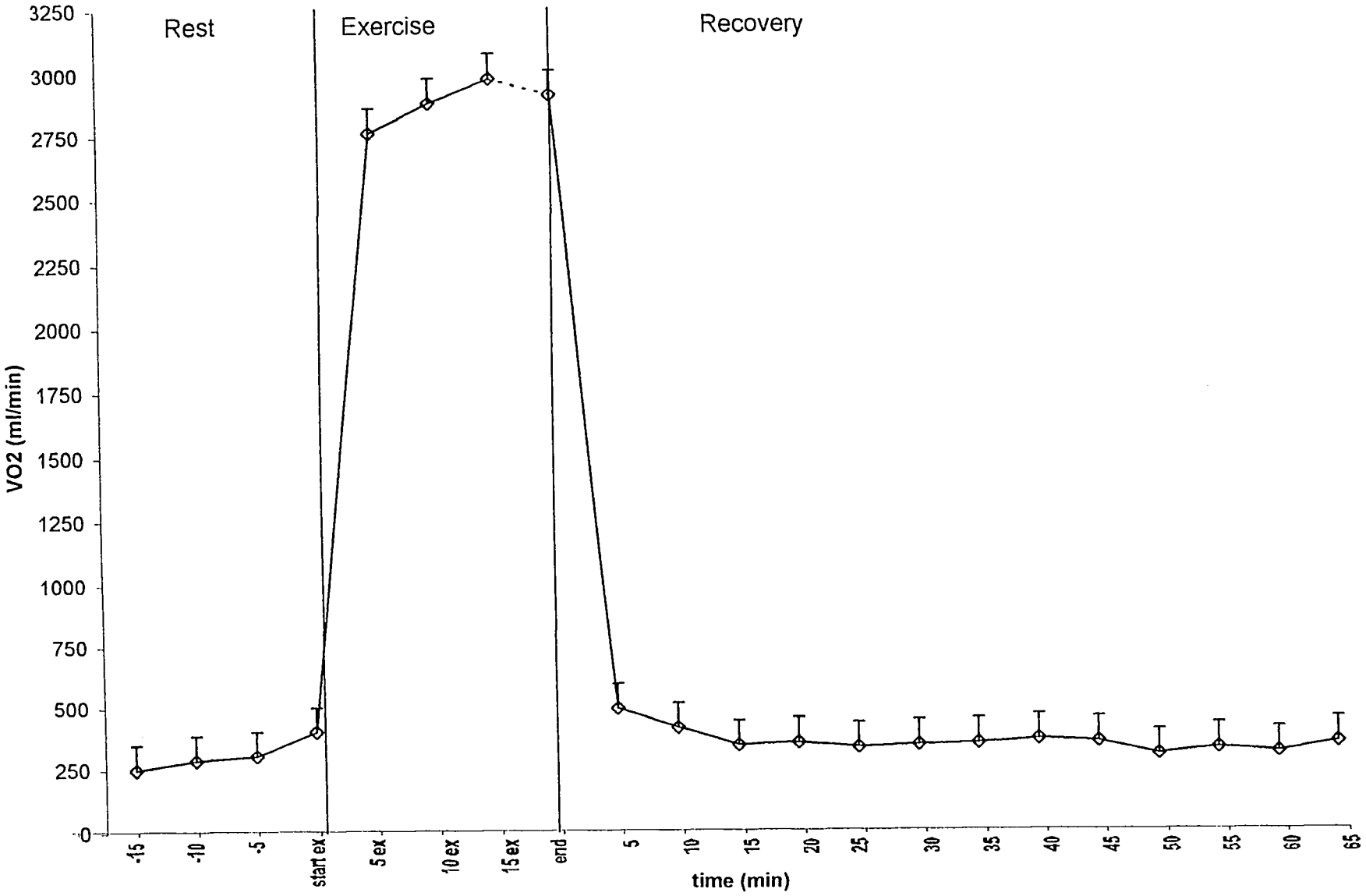


Figure 12 : Mean (±SD) Oxygen Consumption Before, During and 65 minutes After Exercise

CHAPTER V

DISCUSSION AND CONCLUSIONS

The purpose of this study was to expand the understanding of relative changes in body temperatures during and following dynamic exercise. More specifically, our aim was to investigate how exercise-induced increases in active (quadriceps) muscle temperature impact on changes in core (as measured by esophageal temperature) muscle tissue temperatures in participants during and following a cycle ergometry exercise at 70% VO_2 max. Unlike previous studies, we examined tissue temperature transients based on a standard heat produced by exercise above metabolic rate. Furthermore, this is the first study to present continuous esophageal and intra-muscular temperature measurements, for both active and inactive muscles for 120 minutes post-exercise using a newly developed technique for implantation of muscle temperature sensors. Previously published studies in humans mostly limited themselves to 15 and 60 minutes post-exercise with any number of combinations between esophageal and rectal temperature as an index of core and either continuous or non-continuous intra-muscular temperature measurements (Kenny et al., 1998a; Saltin et al., 1966, 1968). Also, previous studies did not allow for real-time precision guidance of thermal probe to a specific internal location. Our technique allows for a greater reproducibility of accurate temperature measurement within and between participants.

1. Post-Exercise Esophageal Temperature and Muscle Temperature Responses

In this study, we were able to increase esophageal temperature with exercise to a level comparable to that presented in previous studies (Kenny et al., 1999a, 1997a, 1996c; Thoden et al.,1994). However, unlike these studies, we were not able to demonstrate a sustained and elevated esophageal temperature plateau following exercise as described by Thoden et al. (1994) and Kenny et al. (1999a). Our results demonstrate a significant elevation of esophageal temperature for only10 minutes following exercise (see Figure 1). Using cycle ergometry exercise, Saltin et al. (1970, 1972) did show a non-significant post-exercise esophageal temperature elevation in different ambient conditions and in different exercise intensities, but these graphical representations were only for 30 and 15 minutes respectively.

Our results as well as those presented by Aikas et al. (1962) do not support the post-exercise esophageal temperature plateau results demonstrated predominantly during treadmill running. Following their exercise protocol, Aikas et al. (1962) showed a rapid re-establishment of esophageal temperature within 15 minutes. Although the present study was not able to reproduce the post-exercise sustained esophageal temperature elevation, it is still possible to infer the effects that elevated deep muscle temperature can have on post-exercise esophageal temperature and heat dissipation.

We have demonstrated that, statistically, esophageal temperature and deep active muscle temperature (T_{muq1}) were no longer different from each other 25 minutes following exercise and that esophageal temperature had statistically returned to baseline 10 minutes prior (see Figure 4). In light of these results, we must reject our primary hypothesis that the return of esophageal temperature to pre-exercise values corresponds to the elimination of a

negative temperature gradient between esophageal temperature and deep active muscle temperature T_{muql}. Although we did graphically demonstrate a difference between esophageal temperature and deep active muscle temperature (T_{muql}) for 80 minutes post-exercise, this difference is non-significant.

It is of interest that one of our participants (#5) did exhibit a sustained post-exercise esophageal plateau of about 0.2 for the entire 120 minutes of recovery. This participant is also the one with the largest gradient between pre-exercise and end of exercise esophageal temperatures (1.3°C). Further, three separate pilot studies consisting of 20 minutes of pre-exercise rest, 15 minutes of cycle ergometry and 30 minutes of post-exercise rest were conducted (on different and non consecutive days) on the same participant but at three different rates of heat gain (40 kJ/min, 50 kJ/min and 60 kJ/min). The results from these pilot studies show that, at the two higher rates of heat gain, there was a sustained post-exercise esophageal temperature of 0.1°C and 0.4°C for the 30 minutes post-exercise. During the higher rate of heat gain exercise, esophageal temperature differential between pre-exercise and end exercise was ~1.9°C. These results seem to suggest the possibility that such a post-exercise esophageal temperature elevation may be identifiable using cycle ergometry with a larger experimental population or with a greater heat produced by exercise above metabolic rate or rate of heat gain during cycle ergometry exercise.

2. Post-Exercise Re-Establishment of Esophageal Temperature

While the rapid re-establishment of esophageal temperature following our exercise may be partially explained by a maintenance of the hypothalamic "set point," it is likely that a more definitive answer lies in one or a combination of the explanations that follow.

Two distinct differences exist between our study and the studies showing prolonged and sustained post-exercise esophageal temperature elevation: The exercise modality used to induce the target heat produced by exercise above metabolic rate and the ambient conditions in the laboratory. No prior studies have attempted to demonstrate a post-exercise esophageal temperature plateau following cycle ergometry exercise. Previous post-exercise esophageal plateaus of at least 60 minutes following exercise have been demonstrated using a treadmill and in ambient conditions of 29°C and 50% relative humidity. Although post-treadmill exercise esophageal plateaus have been demonstrated in ambient temperatures below 29°C (i.e., 20 and 24°C, Kenny et al., 1997a) they were reported no longer then 20 minutes following exercise.

Two differences between treadmill running and cycling are the total muscle mass involved and the distribution of the working muscle mass during the exercise. Treadmill running involves greater recruitment of postural, upper extremity and lower extremity muscle masses as compared to cycle ergometry where the majority of the muscle mass recruited is isolated to the lower extremities. The greater involvement of muscle mass not directly involved in overcoming the resistance renders treadmill running less efficient than cycle ergometry. Therefore, treadmill running will produce a greater heat production by exercise above metabolic rate than cycle ergometry for a given exercise, intensity and time. Our results

and those of similar studies (Aikas et al., 1962; Saltin 1970, 1972) compared to treadmill intervention (Kenny et al., 1997a, 1999a; Thoden et al., 1994) studies seem to demonstrate that there could be a difference in thermal response pertaining to esophageal temperature depending on the exercise modality.

Further evidence that responses are different between treadmill running and cycle ergometry can be found in their respective post-exercise blood pressure responses. Comparing data from two separate studies (Brown et al., 1993; Kaufman et al., 1987) at sub-maximal intensities of 50% VO_2 max and 67% max heart rate showed that treadmill running would create a more pronounced post-exercise hypotension than cycle ergometry. If there is a strong drive to maintain post-exercise blood pressure over the re-establishment of core temperature, treadmill running would induce a greater state of post-exercise hyperthermia than would cycle ergometry at a given load.

Kenny et al. (1998a) provide further evidence that post-exercise esophageal temperature elevation may be dependent on exercise modality. In this study, with 15 minutes of isolated single leg knee extensions, they were able to increase esophageal temperature by 0.31°C but were not able to produce a post-exercise sustained elevation of esophageal temperature for more than 15 minutes.

A second possibility for the rapid return of post-exercise esophageal temperature to pre-exercise values is the ambient conditions. In higher temperature ambient conditions, it is more difficult to lose heat because of the reduced gradient between skin temperature and the environment. Also, lower humidity levels permit evaporative heat loss to be more effective during exercise. Therefore a 1000 kJ of heat produced by exercise above metabolic rate would be more rapidly dissipated in 25°C and 22% relative humidity than in 29°C and 50% relative

humidity. Further, higher ambient temperatures have been shown to have an impact on blood pressure. Higher ambient conditions cause a marked increase in cutaneous vasodilation and therefore a more pronounced post-exercise hypotension (Brown et al., 1993). Kenny et al. (1997a) did study the impact of ambient temperatures on post-exercise esophageal temperature elevation with treadmill exercise. They did show varied post-exercise esophageal temperature elevation in all conditions but for only 20 minutes post-exercise. Further, all exercise trials were conducted in 50% relative humidity.

In terms of exercise intensity and exercise duration this study is very similar to that of previous studies showing post-exercise sustained esophageal temperature elevations. Kenny et al. (1996a,1996b and 1999) used 70% of VO_2max and Thoden et al. (1994) and Kenny et al. (1997a) 75% VO_2max all with treadmill running. As for exercise duration, we chose 1000 KJ as a target for heat produced during exercise above metabolic rate because it represented a total exercise time similar to the exercise times used in previous studies showing post-exercise sustained esophageal temperature elevations. Kenny et al. (1996a,1996b, 1998a and 1999) exercised for 15 minutes, Thoden et al. (1994) and Kenny et al. (1997a) chose 18 minutes of exercise and Kenny et al.(1999) also exercised for 30 and 45 minutes. In our study the average exercise time was 22 ± 4 minutes. Kenny et al.(1997a) and Kenny et al. (1999) were designed to examine if exercise intensity and exercise duration had an impact on the reproducibility of a post-exercise prolonged and sustained esophageal temperature with treadmill running. The results show that it is possible to have a post-exercise prolonged and sustained esophageal temperature with intensities as low as 45% VO_2max and that any exercise duration between 15 and 45 minutes will also produce a post-exercise prolonged and sustained esophageal temperature.

In summary, the rapid return of esophageal temperature following cycle ergometry exercise and the maintenance of a post-exercise esophageal plateau following treadmill running, as demonstrated by Kenny et al. (1999a) and Thoden et al. (1994), strongly suggests that the extent of post-exercise hyperthermia is dependent on the exercise modalities and the ambient conditions.

3. Active and Inactive Intra-muscular Tissue Profiles

In this study, temperature gradients were measured with four points in the vastus medialis and three points in the triceps brachii, prior, during and following exercise. Temperatures across the radial distance of the thigh and the upper arm are shown in Figures 13 and 14.

It is interesting to note that the rate of increase in T_{muq1}, T_{muq2} and T_{muq3} and esophageal temperature decreased slightly when the cumulative heat produced by exercise above metabolic rate reached the 400 kJ mark while the rate of increase in temperature in T_{muq4} and all three triceps temperatures did not change very much. The rate of increase in T_{muq1}, T_{muq2}, T_{muq3} and esophageal temperature were 0.0043°C/kJ, 0.0043°C/kJ,0.0038°C/kJ and 0.0018°C/kJ, respectively, prior to 400kJ while they were 0.0014°C/kJ, 0.0015°C/kJ,0.0019°C/kJ and 0.0006°C/kJ following 400kJ. Further research would be needed to verify whether there is a specific range of heat produced by exercise above metabolic rate which may influence or trigger thermoregulator mechanisms.

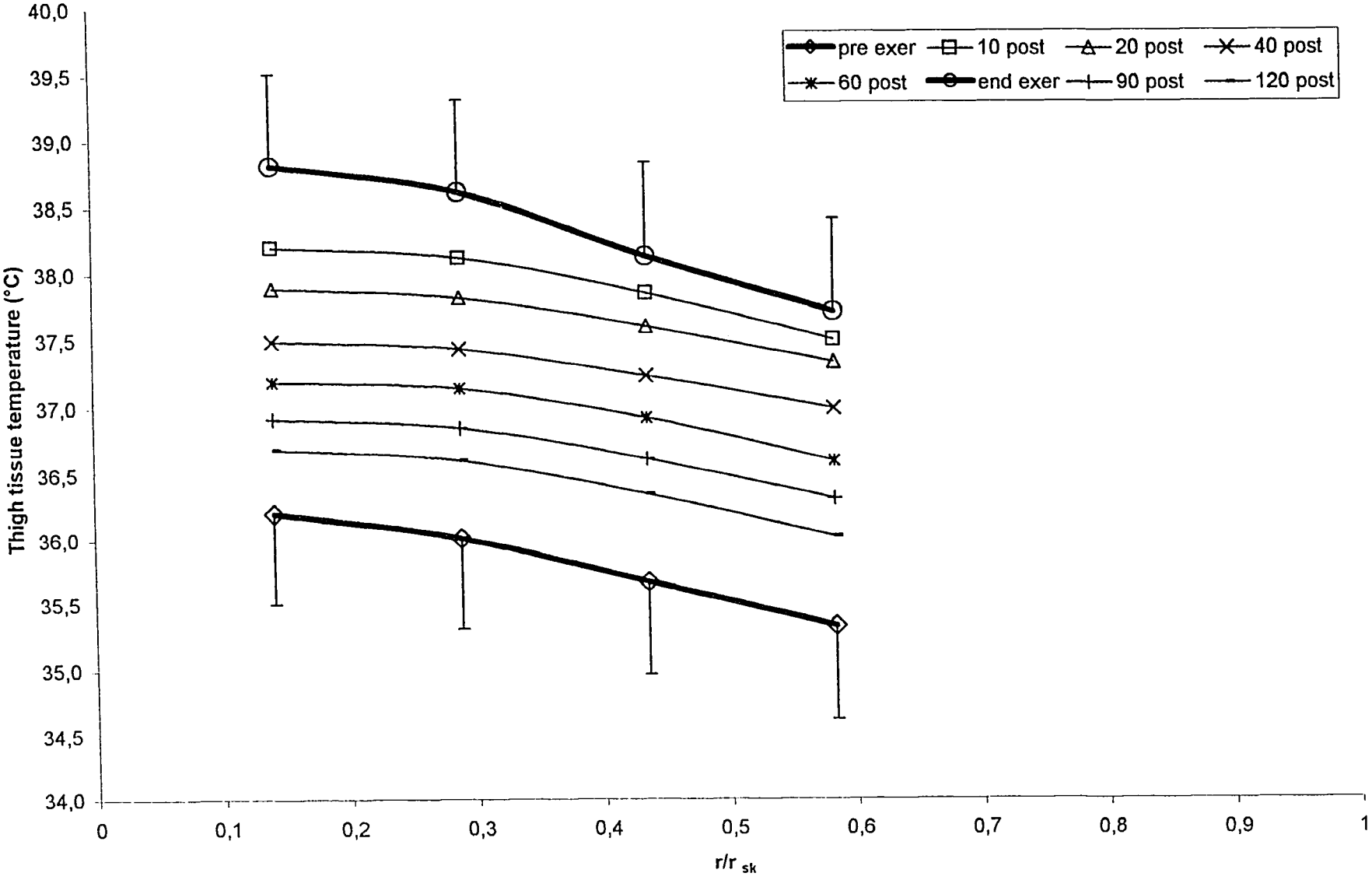


Figure 13: Mean (±SD) Active Muscle Temperature Tissue Profiles Thigh Before, During and After Exercise, Expressed in Fraction of Radius of the Limb(r/r sk).

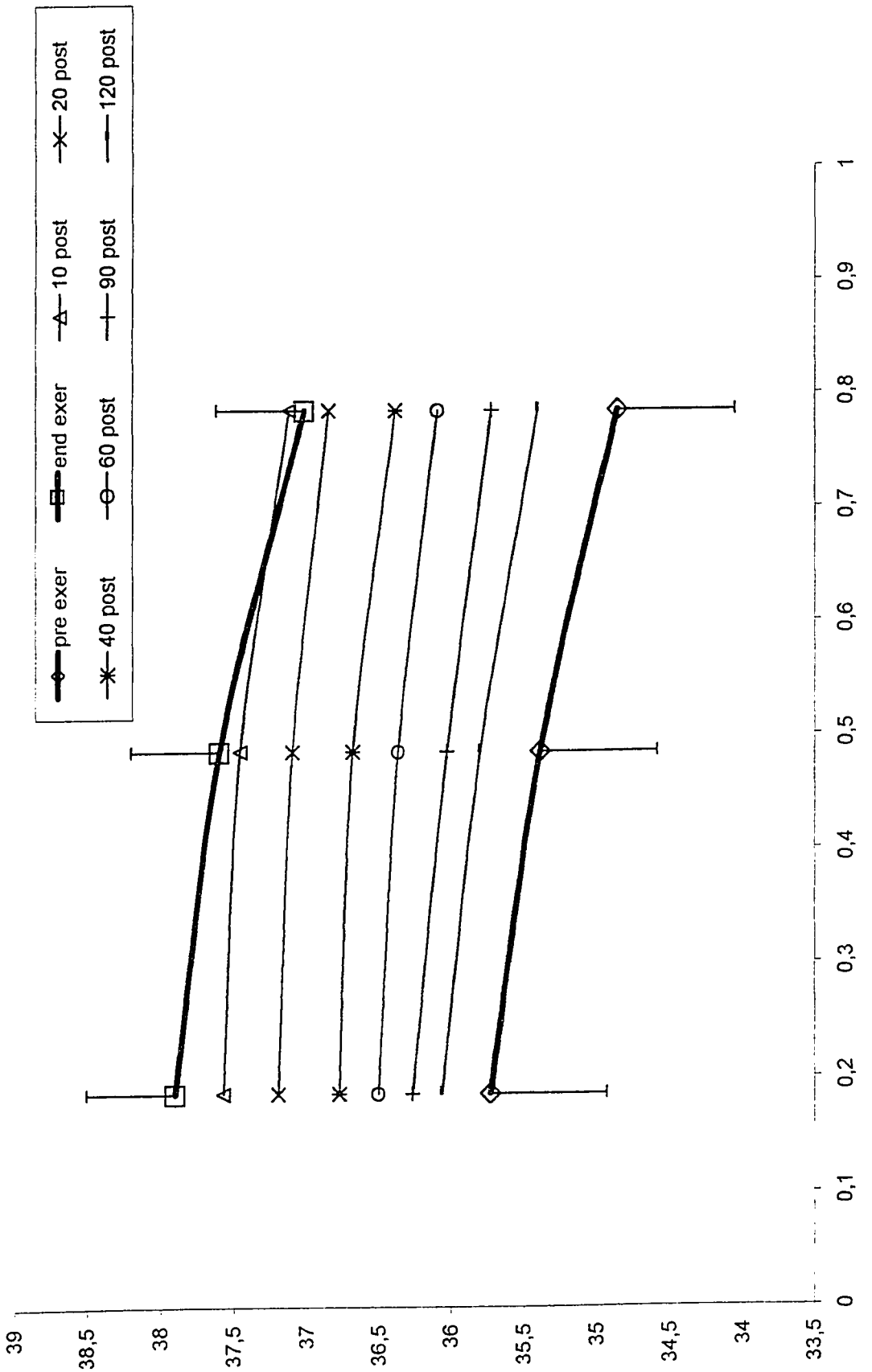


Figure 14: Mean (±SD) Passive Muscle Temperature Tissue Profiles Before, During and After Exercise, Expressed in Fraction of Radius of the Limb (r/r sk).

The demonstration of continuous inactive intra-muscular temperature measurements during and 120 minutes following exercise is unique to the literature. Previous studies including measurement of inactive muscle temperature show that the inactive tissue temperature remains constant or decreases during and following exercise. They also contend that circulation in resting muscle was significantly reduced. In contrast, Kenny et al. (1998a) showed that inactive muscle temperatures increase gradually throughout exercise and continues to rise for 10 minutes following exercise independent of a rapid decrease in esophageal temperature and active muscle temperature following exercise.

Our results from inactive muscle temperature response support those of Kenny et al. (1998a). Inactive muscle temperature was characterized by an initial drop at onset of exercise and a steady increase throughout exercise. Following exercise, inactive muscle temperatures remained constant or increased (depending on depth) for 5 minutes into the recovery period despite a decrease in esophageal and active muscle temperatures. This was followed by a gradual decrease throughout the post-exercise recovery period. Furthermore, a 300% increase in triceps muscle blood flow during exercise and a sustained increase (170% above pre-exercise values) 5 minutes following exercise would indicate continued muscle perfusion within the inactive muscle. Inactive tissue temperature remained significantly ($p<0.05$) elevated from pre-exercise resting values for a minimum of 30 minutes following exercise.

The reestablishment of a positive gradient between the body core and inactive musculature was observed after 15 minutes of recovery which would allow to better maintain heat transfer between these regions and allow better heat dissipation from the core. Also, gradients between the deep active(T_{muq1}) and superficial active (T_{muq4}) as well as between deep active (T_{muq1}) and deep inactive (T_{mut1}) muscle tissue showed a positive gradient for the

entire trial with the T_{muq1} - T_{muq4} gradient being greater. But the gradient between deep active (T_{muq1}) and deep inactive (T_{mut1}) muscle tissue after 5 minutes of exercise and until ~80 minutes into recovery becomes greater than the T_{muq1} - T_{muq4} gradient. The gradient between deep active and deep inactive would also allow for a better heat transfer from previously active to inactive for heat dissipation. Throughout recovery, blood perfusion to the inactive muscle tissue remained elevated by ~100% till 30 minutes into recovery. These results support the importance of vascular heat transfer in the cooling of active tissue and body core. Also, based on these results it is likely that inactive muscle temperature response indicates that it acts as a heat sink to dissipate residual heat from central circulation following short term dynamic exercise. It is of note, that the increase in intra-muscular blood flow during exercise maybe in part do to triceps muscle contraction for postural adjustment during the cycle ergometry exercise. Although, prior to exercise participants where instructed to use there arms as little as possible (only for balance and basic support) no specific measures (such as E.M.G.) were taken to confirm degree of activity or inactivity of triceps muscle but it is believed that arm use was kept to a minimum. Following exercise, participants arms were repositioned in arm harnesses to ensure resting state. It is thought that metabolism maybe play a role in the increased intra-muscular blood flow following exercise but energy expenditure measures returned to pre-exercise values 5 minutes following exercise. Future protocols should include measures such as E.M.G. to measure the degree of use of the passive muscle.

4. Heat Gain and Heat Dissipation

Firstly, cycle ergometry was selected as a natural progression to unilateral and bilateral leg extensions (Kenny et al., 1998a, 1998b) to create higher heat production by exercise above metabolic rate with the involvement of a greater muscle mass and because of the nature of the implant treadmill running may impose a greater risk of trauma to the active muscles. Secondly, the high mechanical efficiency (17.22±1.27 %) serves as a good counter-comparison to the low efficiency (8.13±1.54 %) of leg extensions (Kenny et al., 1998a). In Kenny et al. (1998a), the average heat produced by exercise above metabolic rate during the 15 minutes exercise was 292.3 kJ and the average elevation for esophageal temperature, 0.31°C.

In this study, we tried to better control the total heat produced by exercise above metabolic rate imposed to every participant. Our target was selected to be 1000 kJ. This target was selected following pilot studies that showed that the average time to attain 1000 kJ was between 18 and 25 minutes, which corresponded to the duration of exercise used to elicit post-exercise esophageal temperature elevation following treadmill exercise. It is noteworthy that the 1000 kJ cycle ergometry exercise was also sufficient to elevate esophageal temperature level similar to that of previous treadmill studies such as the 1999a study by Kenny et al. (37.97°C) and the 1994 study by Thoden et al. (38.22°C).

The average heat produced by exercise above metabolic rate during exercise was 930.85 kJ±78.81 kJ and it produced a 0.95°C elevation in esophageal temperature. Energy expenditure rose continuously from 8.28±3.18 kJ/min at onset of exercise to 61.9±11.68 kJ/min after an average of 21.88±4.27 minutes of exercise. Ten minutes following exercise,

the rate of energy expenditure retuned to 8.56±3.08 kJ/min. During the exercise period, cumulative dry heat loss (Hlex, as measured via heat flux transducers), accounted for a loss of 92.0±22.9 kJ. This value for heat loss from the skin surface represents 9.8% of total heat produced by exercise above metabolic rate. Taking these values into account, the net heat accumulation following exercise, without including evaporative heat loss, was 839.25 ± 79.02 kJ. This remaining 90.2% of the heat produced was either absorbed by tissue or dissipated via evaporation. The increase in active and inactive muscle temperatures as well as esophageal temperature indicates that a significant portion had not yet been dissipated at the end of exercise.

As exercise ended, energy expenditure dropped quickly to 10.43±3.57 kJ/min and then 7.02±2.14 kJ/min at 5 and 15 minutes, following exercise. As heat production decreased to pre-exercise values dry heat loss was reduced from 12.06±1.75 kJ/min ($p<0.05$) at end exercise to 8.10±1.19 kJ/min ($p<0.05$) 5 minutes following exercise and 7.28±1.06 kJ/min 15 minutes after exercise.

Without an accurate measure for heat loss via evaporation, it is difficult to estimate when the remaining 839.25 ± 79.02 kJ of heat accumulated during exercise was fully dissipated. However we do know that at 65 minutes and 120 minutes following exercise, cumulative dry heat loss could account for 54.12±20.05 kJ and 59.39±39.48 kJ of the heat dissipation, respectively. Therefore, 120 minutes following exercise, there was still 779.86±82.32 kJ of heat unaccounted for and dry heat loss could only account for 16.37±4.29% of heat dissipated from the initial heat produced by exercise above metabolic rate.

In contrast, Kenny et al. (1998a) had a maximal rate of heat production of 33.72 kJ/min after 13 to 15 minutes of knee extension. Also, during the exercise, dry heat loss only accounted for 7 kJ of heat dissipation and net heat accumulation of 12 kJ during the 60 minutes of post-exercise recovery. In that study, esophageal temperature returned to baseline values 15 minutes following exercise.

Based on the changes in esophageal, rectal and active and passive muscle temperatures, it is likely that a large portion of the remaining heat was absorbed by other tissues. Esophageal temperature returned to pre-exercise values 15 minutes following exercise but rectal temperature responded in a very different manner. The slower responding rectal measure increased gradually during exercise and peaked at 20 minutes post-exercise. However the total increase in the rectal measure was only of 0.5°C and no significant differences were observed. Only 5 of our 8 participant chose to have the rectal measure administered and this may account for our lack of significant differences. Given these results and the still elevated muscle temperatures it would seem that the majority of the heat would be in muscle tissue.

5. Summary and Conclusions

In summary, the purpose of this study was to investigate changes in esophageal temperature elevation, multi-level active and inactive intra-muscular temperature during and following cycle ergometry exercise to a specific heat produced by exercise above metabolic rate. It was hypothesised (based on the theory that post-exercise hypotension creates pooling of warm blood in the previously active muscle and that the residual heat accumulated in this

muscle tissue is gradually transferred via convection to the core) that esophageal temperature would remain elevated until the gradient between core temperature and deep active intra-muscular temperature had dissipated.

The results showed that, in these conditions, the duration of the post-exercise elevation in esophageal temperature following cycle ergometry was 10 minutes, that the return of esophageal temperature to pre-exercise values following exercise did not correspond to crossover point between esophageal temperature and deep quadriceps muscle temperature, and that the exercise modality used to induce heat produced by exercise above metabolic rate and the ambient conditions may have had a profound impact on the reproducibility of a prolonged and sustained post-exercise temperature elevation.

In addition, it was observed that triceps muscle temperatures (inactive muscle tissue) parallel esophageal temperature during this type of exercise and that, following exercise, as esophageal and active muscle temperatures drop, inactive muscle tissue (T_{mut2} and T_{mut3}) continue to rise for 5 minutes suggesting that inactive musculature acted as a heat sink to help with heat dissipation during the post-exercise recovery period.

It is clear following this study that the sustained and prolonged post-exercise esophageal temperature found in Thoden et al. and Kenny et al. studies is not automatic in all conditions. It would seem that there are still many factors and conditions surrounding the reproducibility of this esophageal temperature elevation. Although this study was able to give certain answers, questions still remain. Where has all the heat gone? Is humidity a key factor in esophageal temperature sustained elevation? Does the amount of heat produced by exercise above metabolic rate affect post-exercise esophageal temperature sustained elevation? In order to answer certain questions raised by this study future studies should include full

calorimetric analysis to include evaporative heat loss and a better control of the passive musculature during the exercise period. Future studies should also compare treadmill running with cycle ergometry in different ambient conditions and following different accumulation of heat produced by exercise above metabolic rate i.e. 500 kJ, 1500kJ and 2000kJ and at different rates of heat production such as 40 kJ/min, 50 kJ/min and 60 kJ/min.

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APPENDIX A

Participant Activity Rediness Questionnaire (Par-Q)

1) Your age _____ _____
2) Are you taking medication (other than heart and blood pressure) on a regular basis? _____
3) When was the last time you exercised on a regular basis? _____
4) Do you currently have any physical limitations or an old injury that might be aggrevated by certain movements?

Physical Activity Readiness Questionnaire - PAR-Q (revised 1994)

PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
☐	☐	1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?
☐	☐	2. Do you feel pain in your chest when you do physical activity?
☐	☐	3. In the past month, have you had chest pain when you were not doing physical activity?
☐	☐	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
☐	☐	5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?
☐	☐	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
☐	☐	7. Do you know of any other reason why you should not do physical activity?

If you answered

YES to one or more questions

Talk your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want - as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active - begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal - this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively.

DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever - wait until you feel better; or
- if you are or may be pregnant - talk to your doctor before you start becoming more active.

Please note: If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

There is no change to my medical status: _____ _____ _____ _____
year initials year initials

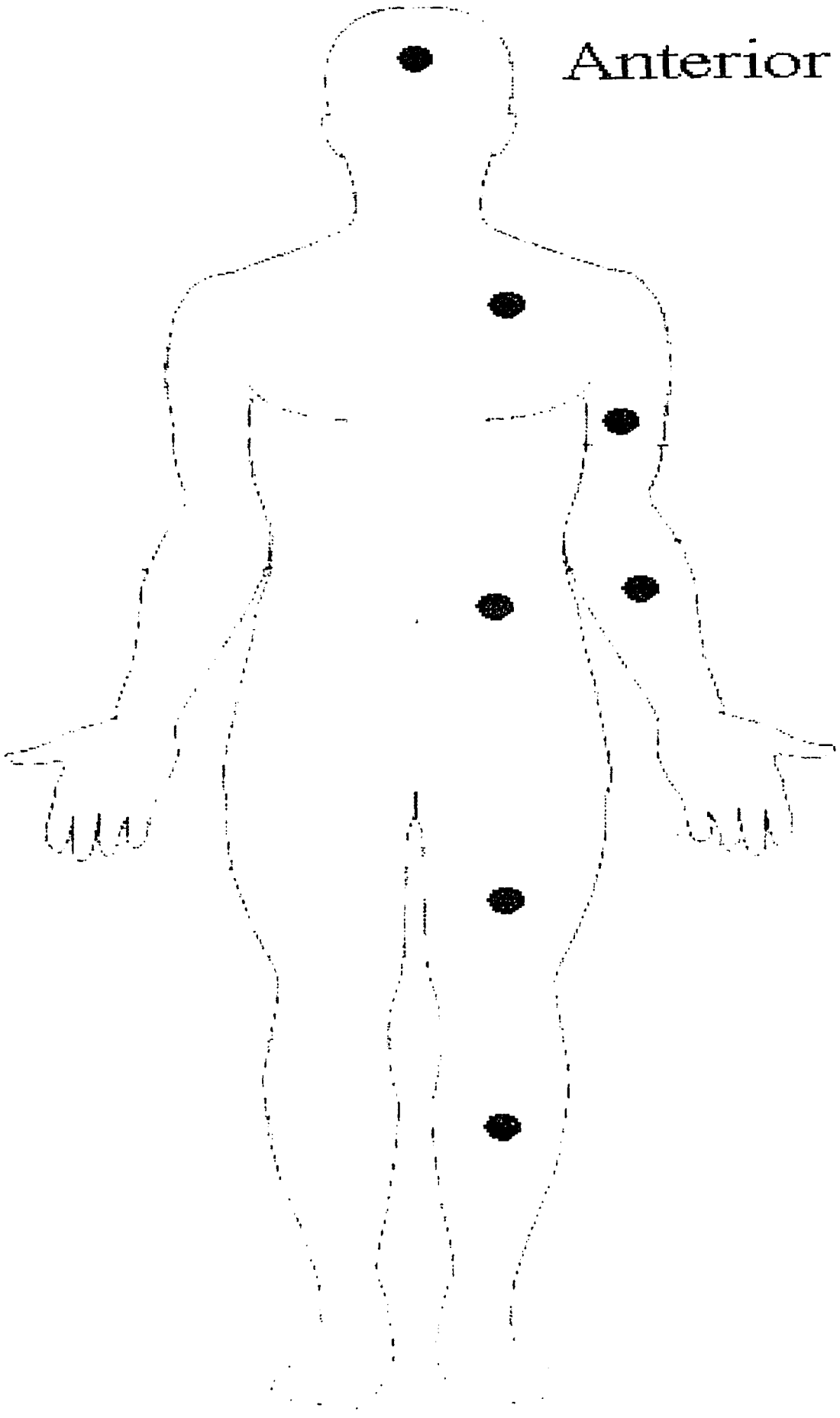
I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction.

NAME (Please Print) _____ SIGNATURE _____

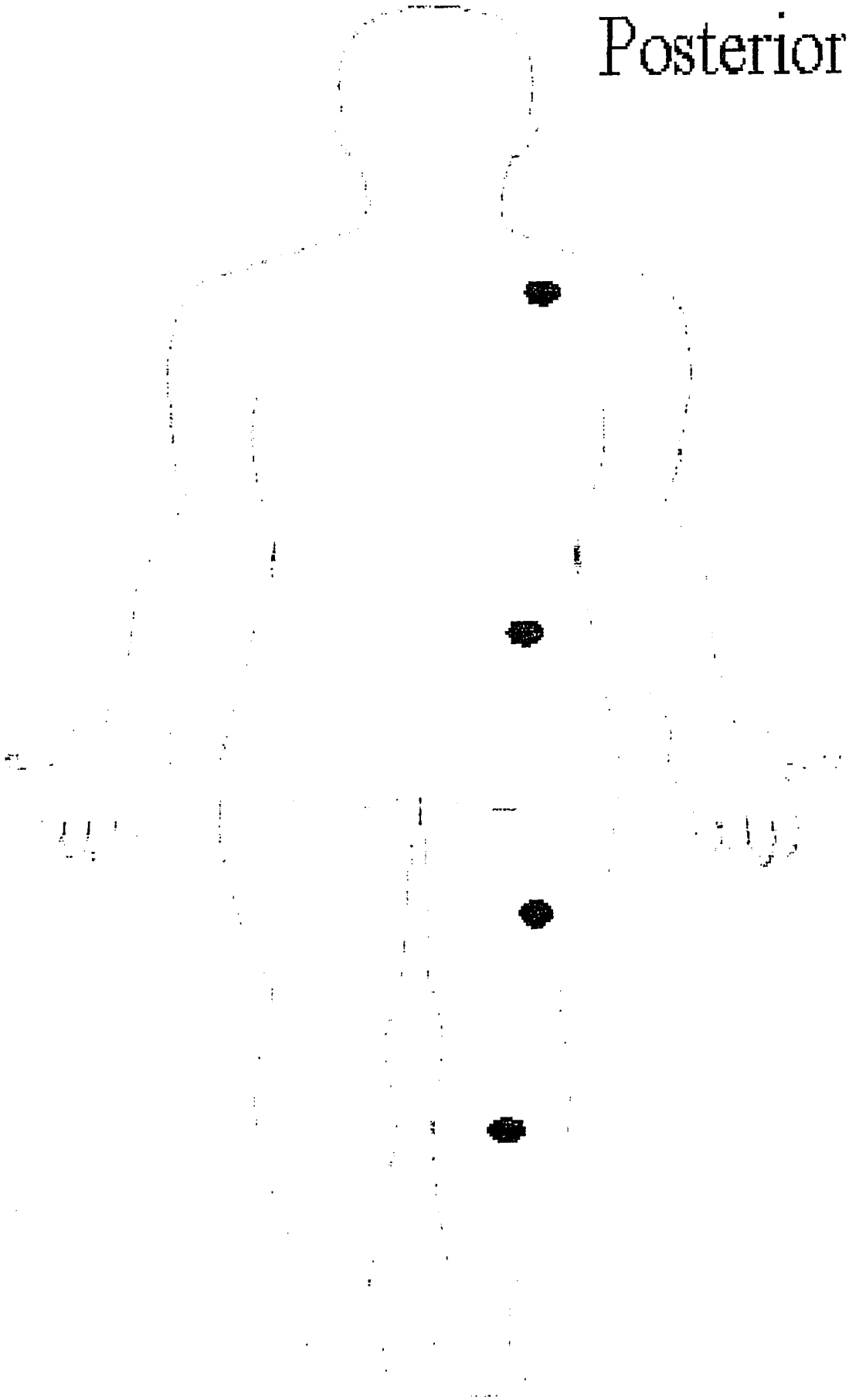
DATE _____ WITNESS _____

APPENDIX B

Temperature Sensor and Heat Flux Transducer Placement


Anterior

Posterior



APPENDIX C

Time Line of Protocol

Typical Time Line for Muscle trials 2000

-180: take **pre-dry weight** adjust bike and table with participant and practice Cardiac output

-120: appointment at O.G.H. (Check and finish set up of equipment, check Nico for 20 sec default, check probe and channels, set up book and check for every bodies role etc..)

-80: return to main campus (lab 309)

-80 to -50: rest after return from hospital (go wash room)

-50 to 00: instrumentation of participant- heart rate monitor, positioning of heat flow sensors , skin temperature thermocouples and thermistors finger probe, set up sweat capsule, blood flow cutaneaous and muscle, manual blood pressure cuff, finapress, esophageal probe.
******make sure all these items are ready and plugged for acquisition.*** *****

00 to 40: rest on chair for baseline data ***prepare Med graphic for acquisition***

5: start gaz collection, **don't forget nose plug**
10: manual blood pressure,
15: **Cardiac Output,** Start Finapress
20: manual blood pressure
29: prepare for bike,

0 - 25 : exercise session (2. KP, 2 minutes, at 10.5 to 12 Kcal/min)

0 : Start of exercise 70revs/min.
15:
25: **stop exercise**

0 - 120 : post exercise resting 120 minutes or wait until Tmus Tes is back to near baseline

0 : start finapress
5:
10:
15:
20:**cardiac output** manual blood pressure
25:
30: Stop finapress
35:
40:**cardiac output** and manual blood pressure
45:
50:
55:
60:**cardiac output** and manual blood pressure
65: **stop Vo2 collection**
70:
120: end session

END: download information (data acquisition, Finapress, HR monitor, and Med graphics) and back up information, remove instrumentation from participant, *start clean up make sure everything is nice and clean.*
analyse data

APPENDIX D

D.1 Certification of Institutional Human Research Ethics Committee

D.2 Certification of Ottawa Hospital Research Ethics Board

Université d'Ottawa • University of Ottawa

Faculté des sciences de la santé
Cabinet de la doyenne

Faculty of Health Sciences
Office of the Dean

November 2, 1999

Professor Glen Kenny
Student Paul Denis
School of Human Kinetics
125 University (232)
INTRA

Subject: **Your project entitled:** ***"The effect of endogenous and exogenous heating on muscle temperature transients in humans"***

Dear Professor and Student,

It is my pleasure to inform you that the Faculty of Health Sciences, Human Research Ethics Committee, after study of the documentation provided, concluded that your project met the appropriate standards of ethical acceptability and falls within CATEGORY 1A.

I hereby attach a copy of the certificate of clearance granted by the University Human Research Ethics Committee.

This certificate is valid for a period of one year from the time of issuance. I would also like to remind you that, in accordance with the policies of the UHREC, it is your responsibility to notify the Committee of any major changes in this project.

On behalf of the Committee, I wish you success in your project.

Sincerely,

J. Roger Proulx, Ph.D.
Chair, Human Research Ethics Committee

451, ch. Smyth
Ottawa (Ontario) K1H 8M5 Canada

451 Smyth Rd.
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Université d'Ottawa · University of Ottawa

Faculté des sciences de la santé
Cabinet de la doyenne

Faculty of Health Sciences
Office of the Dean

CERTIFICATION OF INSTITUTIONAL HUMAN RESEARCH ETHICS COMMITTEE
FACULTY OF HEALTH SCIENCES

This is to certify that the Institutional Human Research Ethics Review Committee of the Faculty of Health Sciences has examined the research proposal **from Professor Glen Kenny and Student Paul Denis from the School of Human Kinetics** for the project ***"The effect of endogenous and exogenous heating on muscle temperature transients in humans"*** and concludes that, in all respects, the proposed research protocol meets the appropriate standards of ethical acceptability, at a Category 1A level.

MEMBERS OF THE COMMITTEE

Name (Optional)	Position held	Department of discipline
Victor Boucher	Professor	Audiology and Speech-Pathology Program
François Tremblay	Professor	Physiotherapy Program
Claire-Jehanne Dubouloz	Professor	Occupational Therapy Program
Jocelyne Tourigny	Professor	School of Nursing
Rock Paquin	Member-at-Large	
Pierre Boudreau	Professor	Faculty of Education
J. Roger Proulx	Chair	Human Research Ethics Committee School of Human Kinetics
Nicole Denis	Student	School of Nursing

SIGNATURE

02/11/1999

Date

Committee Chairperson - J. Roger Proulx, Ph.D.

451, ch. Smyth
Ottawa (Ontario) K1H 8M5 Canada

451 Smyth Rd.
Ottawa, Ontario K1H 8M5 Canada

The Ottawa Hospital | L'Hôpital d'Ottawa

Research Ethics Board
Conseil d'éthique en recherches
761-4146 ~ 761-4902 ~ 761-5072
Fax No. ~ 761-4920

February 15, 2000

Mr. PaulM. Denis
School of Human Kinetics
Faculty of Health Sciences
University of Ottawa
125 University Private
Ottawa, ON K1N 6N5

Dear Mr. Denis:

Re: Protocol # - 1999275-01H **Muscle Tissue Temperature Transients and Post Exercise Core Temperature Elevation**

Protocol approval valid until - **February 14, 2001**

You have met the requirements of the Board and the above listed project has been granted approval by the Ottawa Hospital Research Ethics Board (OHREB). No addenda may be made in the protocol or the consent form without the OHREB review and approval.

The validation date should be indicated on the bottom of all consent forms and information sheets (see copy attached). Approximately one month prior to that time, a single renewal form should be sent to Research Services.

Medical Research Council guidelines require a greater involvement of the Research Ethics Board in studies over the course of their execution. You must maintain as part of your records copies of the signed consent form. As well, you must inform the Board of adverse events encountered during the study, here or elsewhere, or of significant new information which becomes available after the Board review, either of which may impinge on the ethics of continuing the study. The OHREB will review the new information to determine if the protocol should be modified, discontinued, or should continue as originally approved.

Yours sincerely,

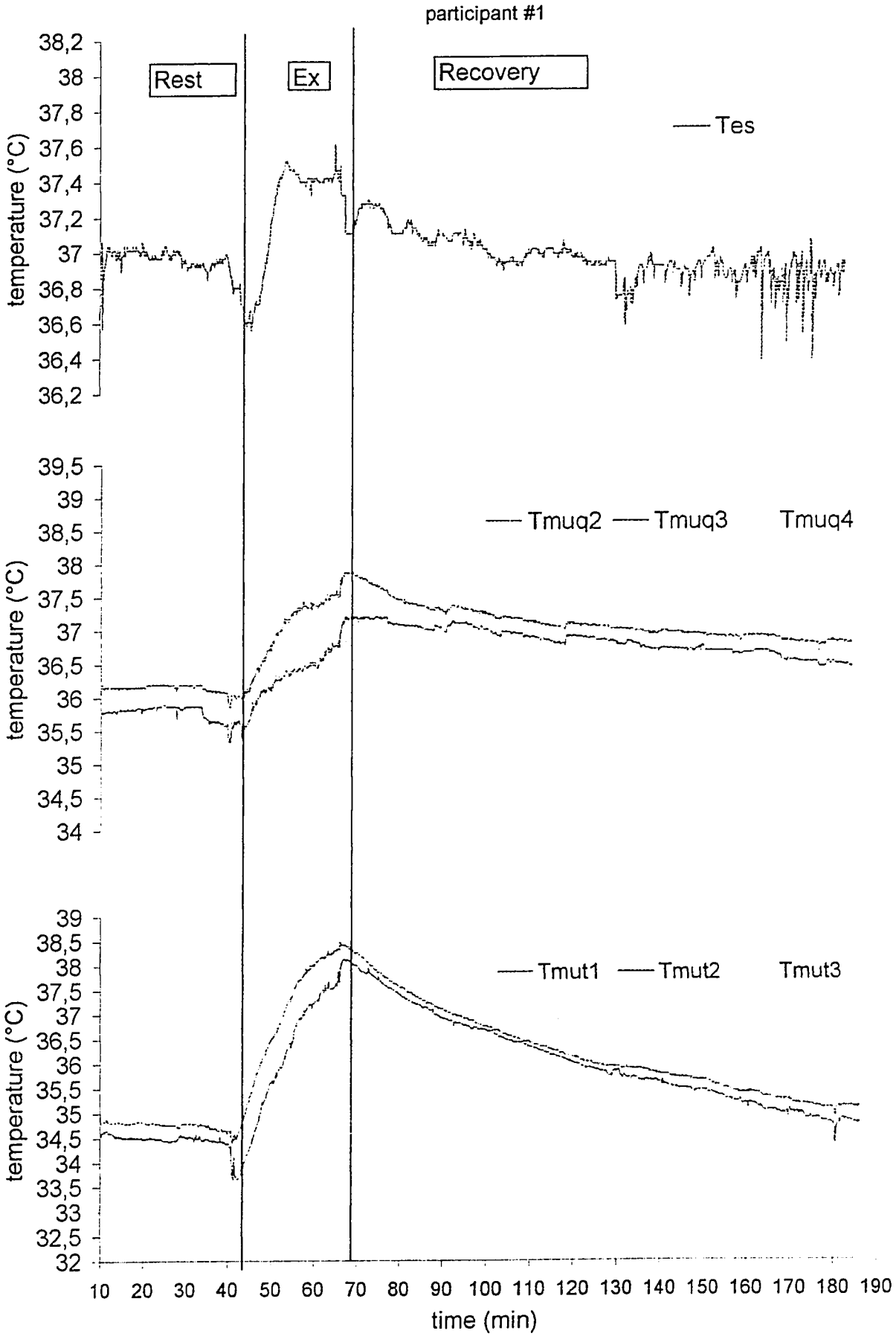
Raphael Saginur, M.D.
Chairman
Ottawa Hospital Research Ethics Board

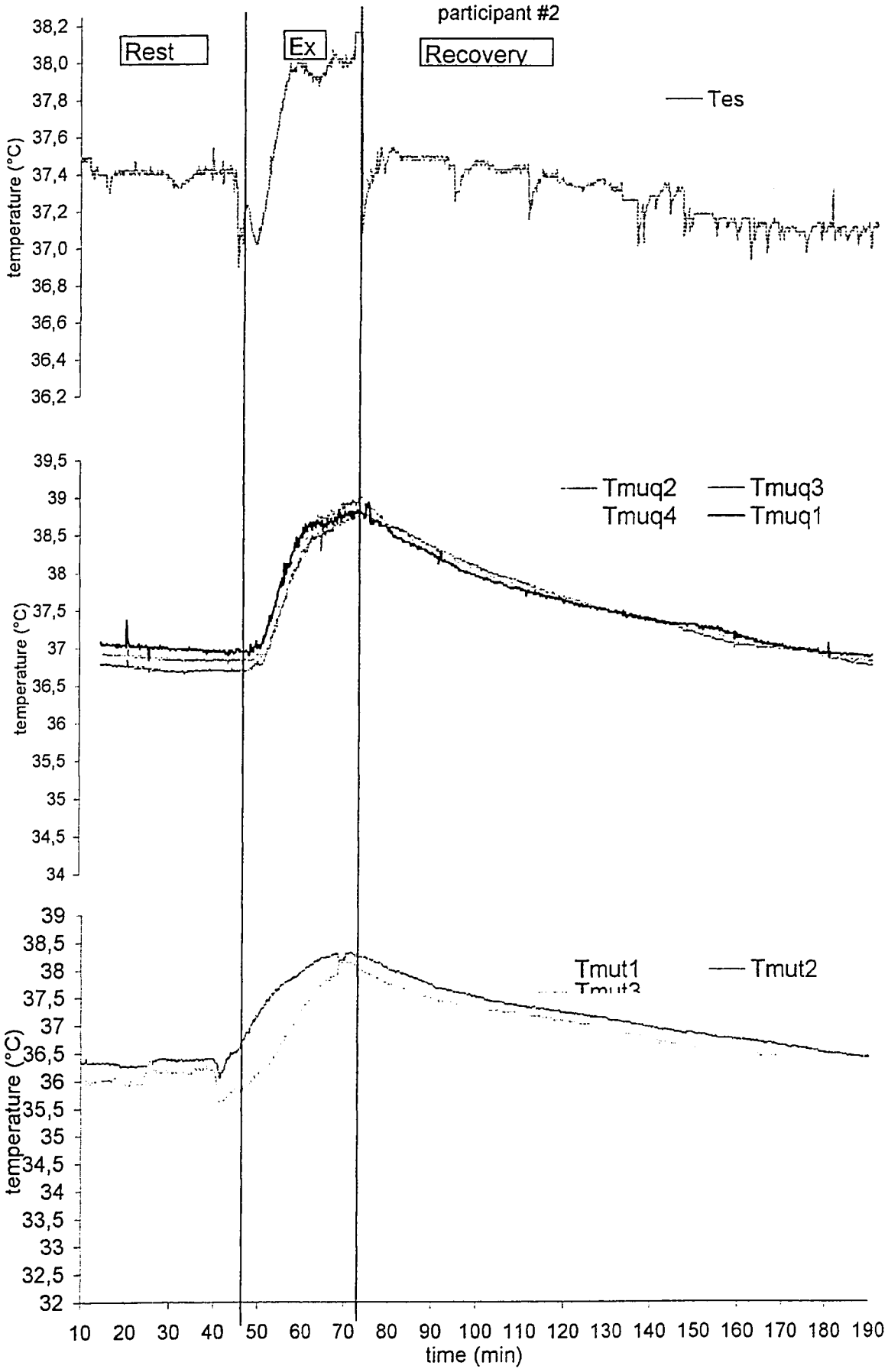
APPENDIX E

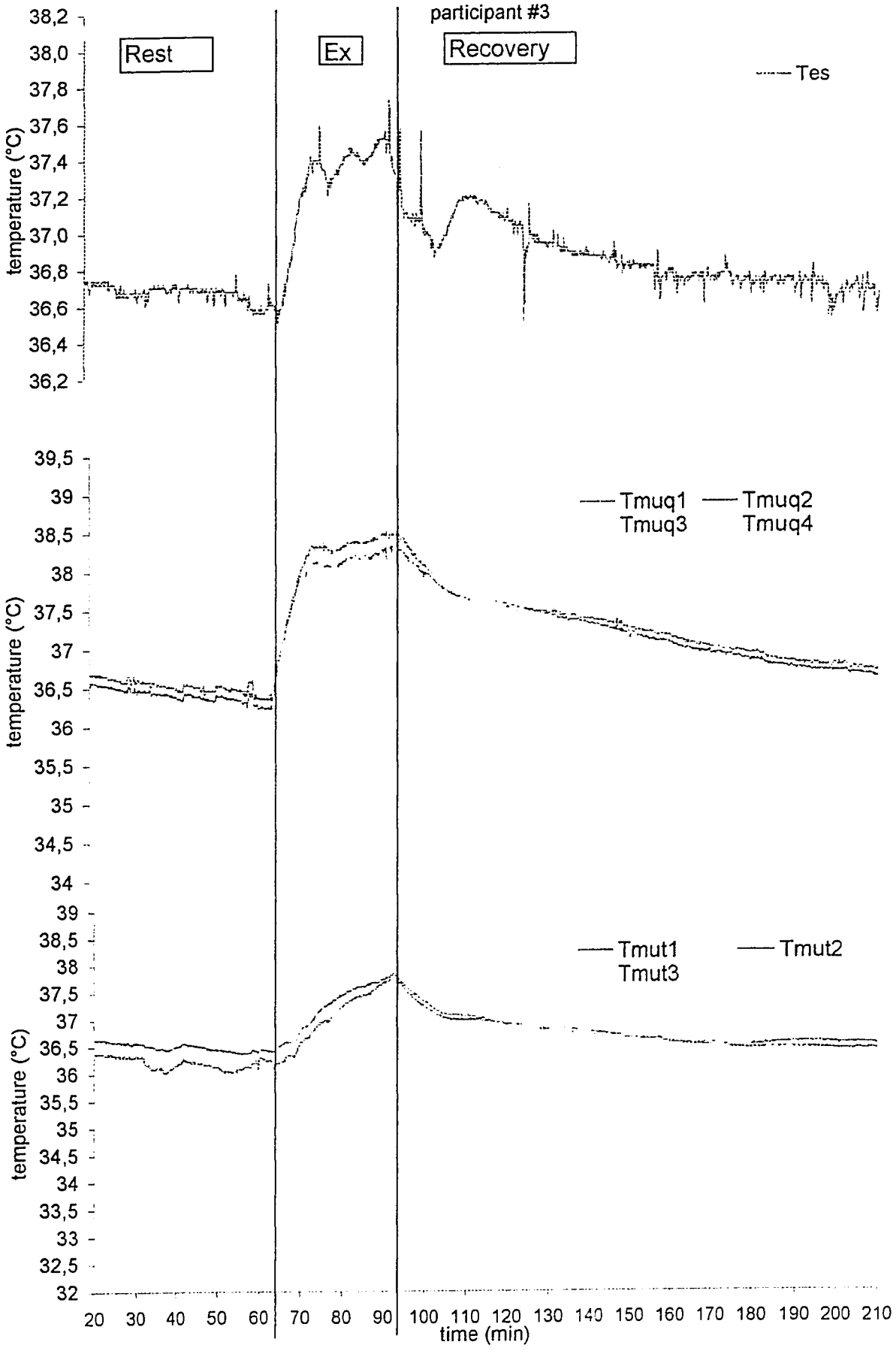
E.1 Individual Esophageal and Muscle Temperatures (n=8)

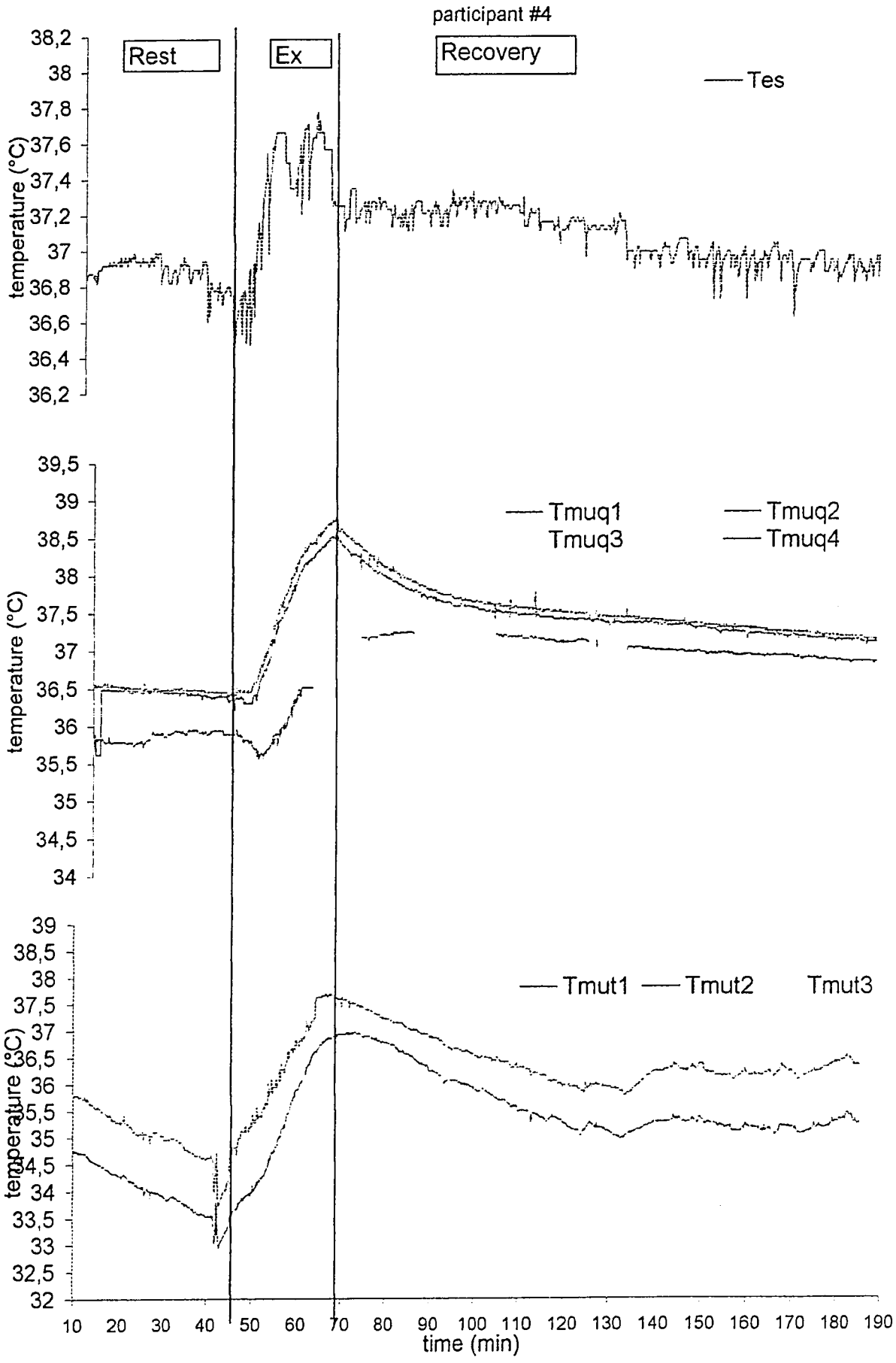
E.2 Complete Results Summary Table

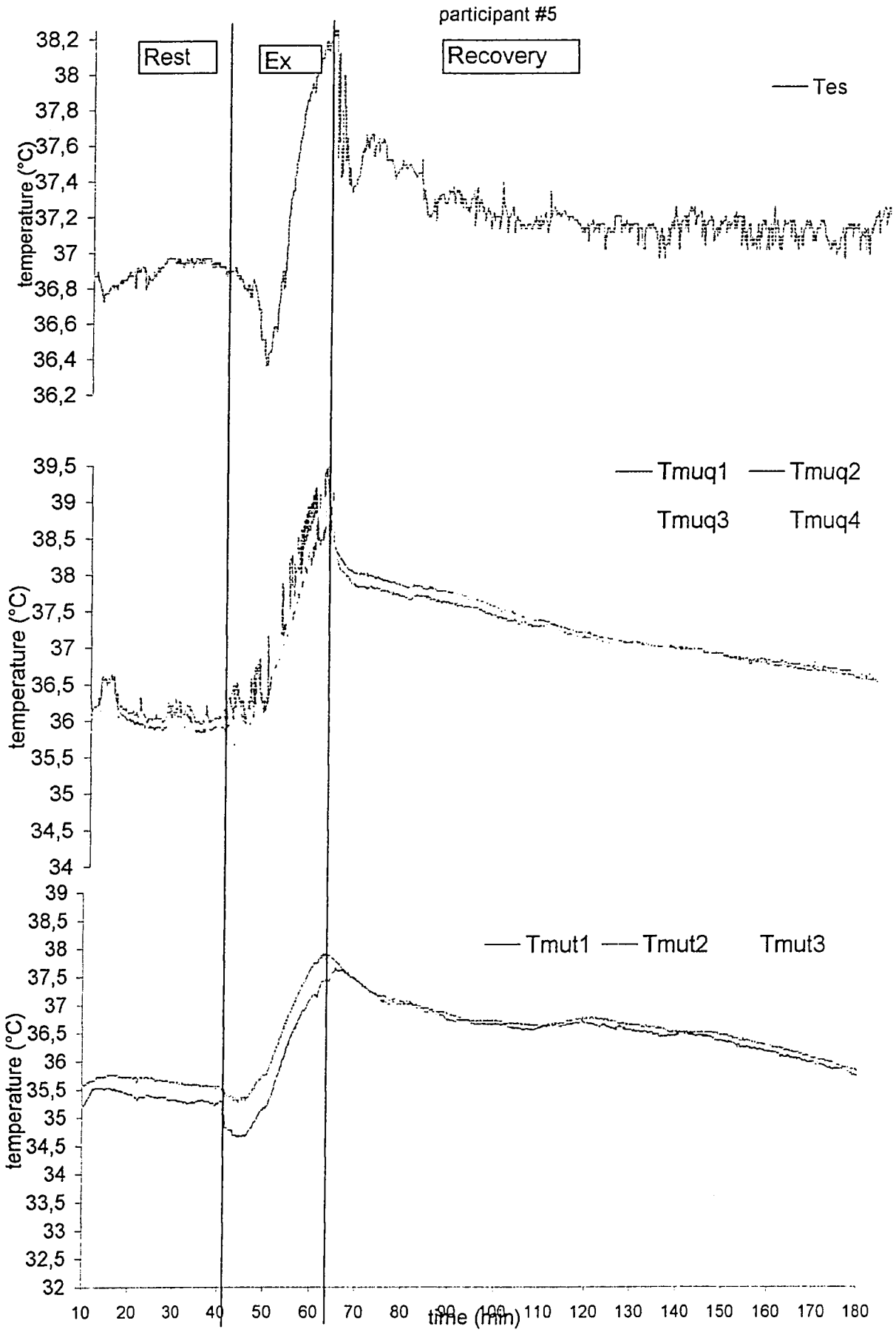
E.3 Blood Pressure Data

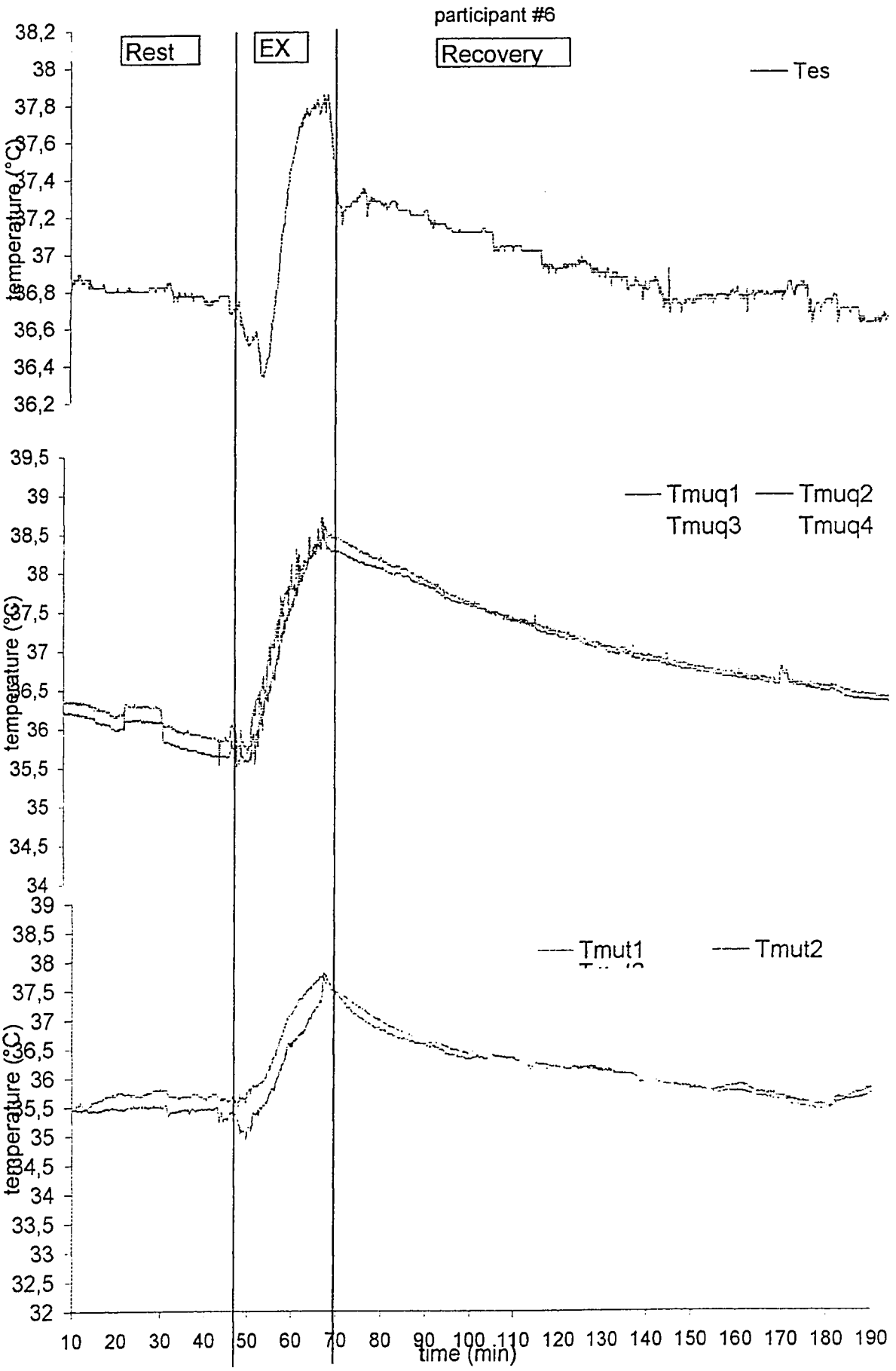

participant #1
Rest
Ex
Recovery
Tes
Tmuq2
Tmuq3
Tmuq4
Tmut1
Tmut2
Tmut3
temperature (°C)
temperature (°C)
temperature (°C)
38,2
38
37,8
37,6
37,4
37,2
37
36,8
36,6
36,4
36,2
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190
time (min)

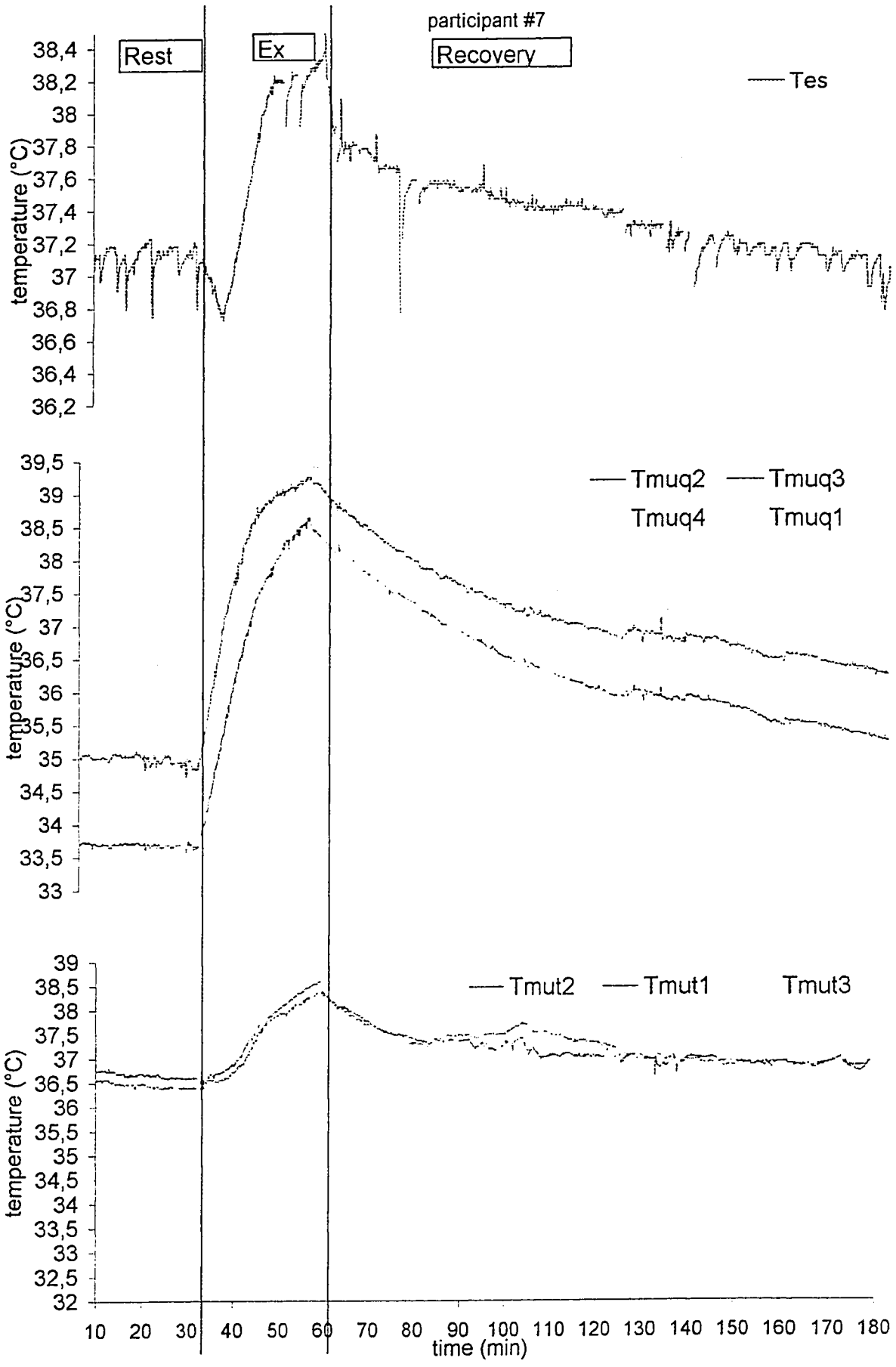

participant #2
Rest
Ex
Recovery
Tes
temperature (°C)
38,2
38,0
37,8
37,6
37,4
37,2
37,0
36,8
36,6
36,4
36,2
Tmuq2
Tmuq3
Tmuq4
Tmuq1
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
Tmut1
Tmut2
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190
time (min)

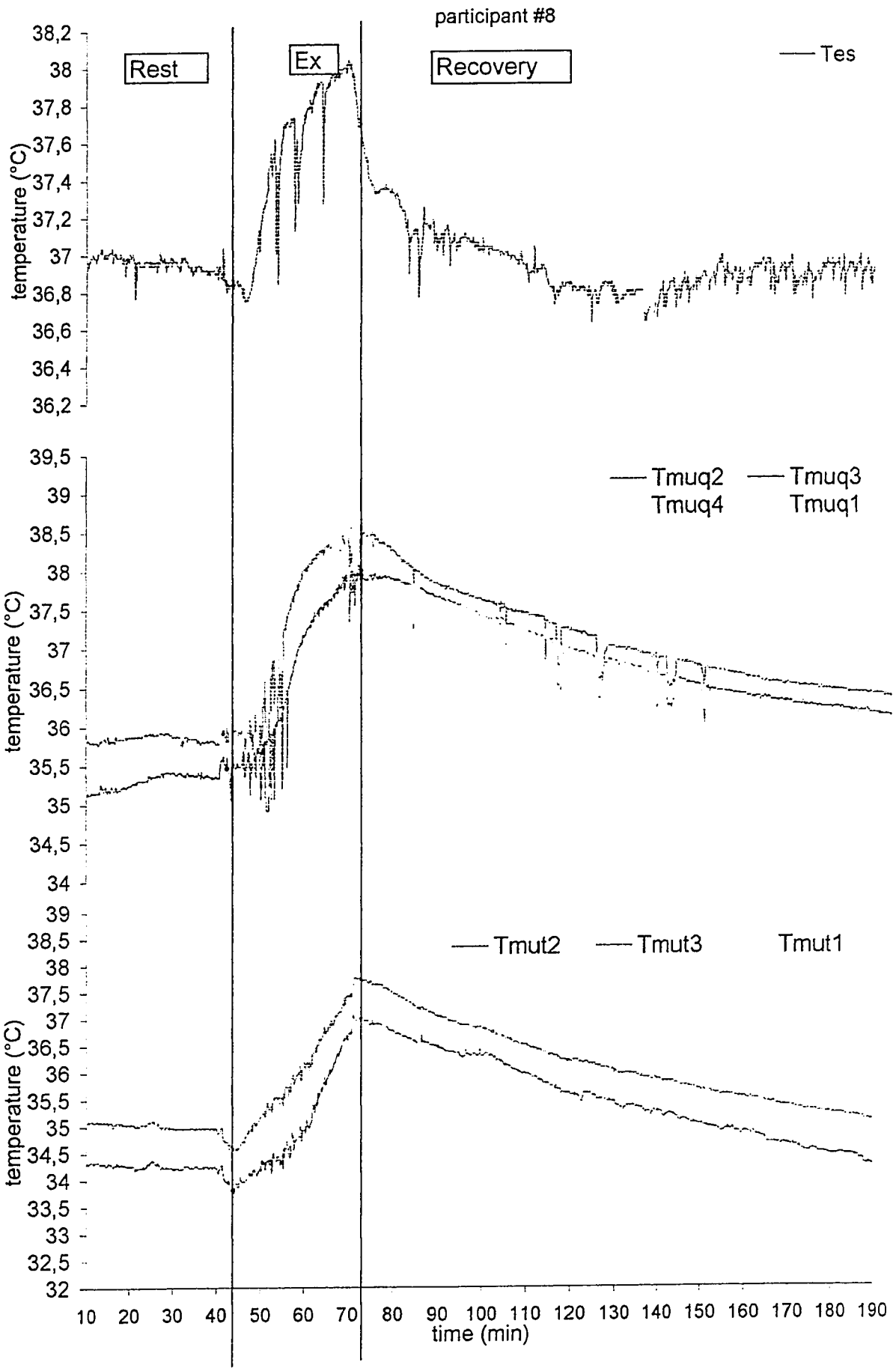

participant #3
Rest
Ex
Recovery
Tes
temperature (°C)
38,2
38,0
37,8
37,6
37,4
37,2
37,0
36,8
36,6
36,4
36,2
Tmuq1
Tmuq2
Tmuq3
Tmuq4
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
Tmut1
Tmut2
Tmut3
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190 200 210
time (min)


participant #4
Rest
Ex
Recovery
Tes
temperature (°C)
38,2
38
37,8
37,6
37,4
37,2
37
36,8
36,6
36,4
36,2
Tmuq1
Tmuq2
Tmuq3
Tmuq4
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
Tmut1
Tmut2
Tmut3
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190
time (min)


participant #5
Rest
Ex
Recovery
Tes
temperature (°C)
38,2
38
37,8
37,6
37,4
37,2
37
36,8
36,6
36,4
36,2
Tmuq1
Tmuq2
Tmuq3
Tmuq4
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
Tmut1
Tmut2
Tmut3
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
10
20
30
40
50
60
70
80
90
100
110
120
130
140
150
160
170
180
time (min)

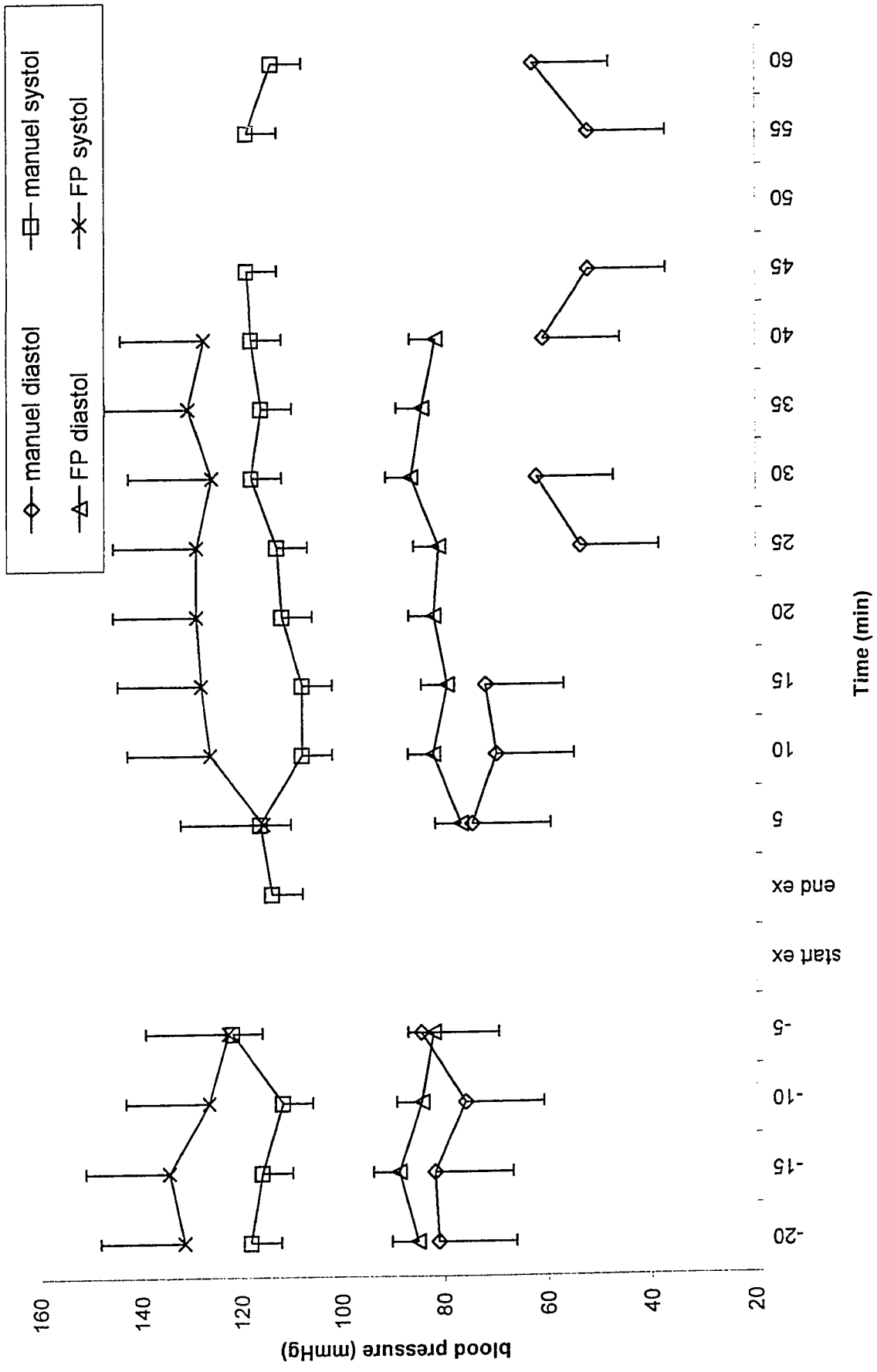

participant #6
Rest
EX
Recovery
Tes
temperature (°C)
38,2
38
37,8
37,6
37,4
37,2
37
36,8
36,6
36,4
36,2
Tmuq1
Tmuq2
Tmuq3
Tmuq4
temperature (°C)
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
Tmut1
Tmut2
temperature (°C)
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190
time (min)


participant #7
Rest
Ex
Recovery
Tes
temperature (°C)
38,4
38,2
38
37,8
37,6
37,4
37,2
37
36,8
36,6
36,4
36,2
Tmuq2
Tmuq3
Tmuq4
Tmuq1
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
Tmut2
Tmut1
Tmut3
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180
time (min)


participant #8
Rest
Ex
Recovery
Tes
temperature (°C)
38,2
38
37,8
37,6
37,4
37,2
37
36,8
36,6
36,4
36,2
Tmuq2
Tmuq3
Tmuq4
Tmuq1
39,5
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
Tmut2
Tmut3
Tmut1
39
38,5
38
37,5
37
36,5
36
35,5
35
34,5
34
33,5
33
32,5
32
10 20 30 40 50 60 70 80 90 100 110 120 130 140 150 160 170 180 190
time (min)

	resting	onset of exercise	5 min exercise	10 min exercise	15 min exercise	end of exercise	post5	post10	post15	post20	post25	post30	post35	post40
Tes	36,95	36,81	36,90	**37,60**	**37,71**	**37,89**	**37,35**	**37,38**	37,29	37,26	37,23	37,23	37,19	37,14
stdev	0,22	0,22	0,24	**0,21**	**0,34**	**0,33**	**0,21**	**0,23**	0,23	0,17	0,17	0,19	0,20	0,22
Tmuq1	36,31	36,23	**37,17**	**38,13**	**38,52**	**38,81**	**38,44**	**38,20**	**38,02**	**37,89**	**37,78**	**37,67**	**37,57**	**37,50**
stdev	0,39	0,45	**0,50**	**0,34**	**0,34**	**0,42**	**0,38**	**0,34**	**0,32**	**0,28**	**0,23**	**0,20**	**0,19**	**0,19**
Tmuq2	36,10	36,00	**36,85**	**37,83**	**38,30**	**38,61**	**38,31**	**38,12**	**37,94**	**37,82**	**37,71**	**37,63**	**37,52**	**37,44**
stdev	0,50	0,60	**0,56**	**0,46**	**0,48**	**0,60**	**0,33**	**0,30**	**0,29**	**0,26**	**0,22**	**0,17**	**0,16**	**0,16**
Tmuq3	35,75	35,60	36,27	37,21	37,74	**38,13**	**37,97**	**37,85**	**37,71**	**37,60**	**37,50**	**37,43**	**37,32**	**37,23**
stdev	0,84	0,92	0,89	0,77	0,73	**0,75**	**0,44**	**0,39**	**0,37**	**0,35**	**0,35**	**0,33**	**0,35**	**0,37**
Tmuq4	35,33	35,18	35,68	36,45	37,08	**37,71**	**37,62**	**37,50**	**37,41**	**37,33**	**37,25**	**37,17**	**37,06**	**36,99**
stdev	0,88	0,97	1,09	1,18	1,08	**0,96**	**0,70**	**0,55**	**0,47**	**0,47**	**0,46**	**0,43**	**0,43**	**0,42**
Tmut1	35,82	35,63	36,26	36,97	37,49	**37,90**	**37,86**	**37,57**	**37,34**	**37,20**	**37,07**	**36,95**	**36,85**	**36,77**
stdev	0,70	0,87	0,65	0,58	0,47	**0,44**	**0,45**	**0,44**	**0,41**	**0,37**	**0,34**	**0,37**	**0,35**	**0,34**
Tmut2	35,48	35,12	35,69	36,39	37,06	**37,60**	**37,66**	**37,46**	**37,25**	**37,10**	**36,94**	**36,84**	**36,76**	36,68
stdev	0,89	1,25	1,03	0,96	0,76	**0,62**	**0,42**	**0,39**	**0,37**	**0,37**	**0,40**	**0,49**	**0,50**	0,55
Tmut3	34,95	34,49	34,91	35,55	36,29	**37,02**	**37,30**	**37,12**	**36,96**	**36,85**	**36,67**	**36,57**	36,48	36,39
stdev	1,18	1,36	1,20	1,10	0,90	**0,79**	**0,64**	**0,53**	**0,50**	**0,49**	**0,53**	**0,58**	0,56	0,64
Tskn	32,46	32,35	32,21	32,70	33,63	**34,15**	**34,29**	33,93	33,69	33,41	33,27	33,17	33,09	33,05
stdev	0,88	0,97	0,96	1,17	1,12	**0,96**	**0,70**	0,55	0,47	0,47	0,46	0,43	0,43	0,42
Trec	37,21	37,13	37,16	37,26	37,39	37,58	37,65	37,67	37,69	37,71	37,71	37,69	37,66	37,63
stdev	0,20	0,18	0,16	0,16	0,20	0,26	0,27	0,28	0,27	0,25	0,24	0,24	0,25	0,28
Flux kJ/min	5,87	6,11	8,02	**9,57**	**11,17**	**11,80**	**8,10**	7,43	7,28	6,82	6,76	6,61	6,58	6,44
stdev	0,72	1,21	1,36	**1,26**	**1,41**	**1,70**	**1,19**	0,87	1,06	0,99	0,88	0,91	1,01	0,97
HR	65,58	**86,88**	**138,88**	**151,75**	**157,75**	**158,13**	**102,46**	**96,09**	**93,47**	**93,78**	**89,81**	**89,56**	**88,93**	**87,22**
stdev	5,79	**19,35**	**12,46**	**11,50**	**12,62**	**10,38**	**12,74**	**12,81**	**12,25**	**13,29**	**12,98**	**13,04**	**12,50**	**12,37**
E.E kJ/min	5,56	8,28	**52,88**	**61,28**	**63,26**	**61,87**	10,43	8,56	7,02	7,25	6,8	7,01	7,17	7,49
stdev	2,16	3,18	**18,73**	**11,30**	**12,04**	**11,68**	3,57	3,08	2,14	2,73	2,49	2,04	1,69	2,29
Quad BF	3,29	3,16	10,86	11,79	15,12	**17,89**	**20,21**	**16,46**	15,10	14,22	9,18	7,18	6,86	5,55
stdev	1,81	2,52	7,83	9,11	7,97	**8,71**	**10,07**	**10,55**	10,61	11,33	5,84	4,76	5,24	4,04
4Arm BF	9,37	10,89	18,20	63,06	90,03	92,11	61,25	44,23	27,43	21,63	15,82	14,96	15,55	12,29
stdev	6,70	6,84	8,10	83,56	119,01	117,02	97,94	66,04	37,22	28,75	16,57	20,42	21,69	11,99
Musc BF	10,10	11,24	25,89	25,91	25,39	19,71	14,56	14,16	10,62	10,37	8,29	2,97	4,09	2,96
stdev	0,88	0,97	38,79	46,66	45,51	0,96	0,70	0,55	0,47	0,47	0,46	0,43	0,43	0,42
BP Sys F	129,05						**115,74**	126,36	128,23	129,15	129,12	126,03		
stdev	5,25						**14,56**	16,21	10,75	11,55	11,35	15,02		
BP DiasF	85,31						**76,80**	82,14	79,44	81,99	81,00	86,46		
stdev	2,82						**4,18**	8,52	7,62	11,53	10,53			
BP Sys	117,06						116,33				113,00	118,00		118,00
stdev	4,19						8,52				4,24	0,00		6,32
BP Dias	80,97917						74,571				80	82,667		76
stdev	3,63						4,28				5,66	4,16		7,12

	post45	post50	post55	post60	post65	post70	post75	post80	post85	post90	post95	post100	post105	post110	post115	post120
Tes	37,11	37,09	37,07	37,05	37,00	36,95	36,98	36,93	36,95	36,94	36,92	36,93	36,91	36,91	36,91	36,90
stdev	0,22	0,22	0,23	0,23	0,23	0,23	0,21	0,17	0,17	0,14	0,14	0,13	0,13	0,16	0,16	0,20
Tq1	**37,39**	**37,35**	**37,26**	**37,19**	**37,16**	**37,08**	**37,06**	**37,02**	**36,97**	**36,92**	36,86	36,83	36,79	36,76	36,72	36,69
stdev	**0,29**	**0,22**	**0,28**	**0,30**	**0,27**	**0,35**	**0,28**	**0,29**	**0,29**	**0,29**	0,28	0,27	0,28	0,28	0,29	0,29
Tq2	**37,32**	**37,28**	**37,20**	**37,14**	**37,08**	**37,01**	**36,99**	**36,95**	**36,91**	**36,85**	**36,79**	**36,77**	**36,73**	**36,69**	**36,65**	36,61
stdev	**0,20**	**0,17**	**0,19**	**0,19**	**0,21**	**0,25**	**0,21**	**0,22**	**0,22**	**0,23**	**0,25**	**0,24**	**0,25**	**0,26**	**0,28**	0,29
Tq3	**37,10**	**37,05**	**36,98**	**36,92**	**36,86**	**36,78**	**36,75**	**36,72**	**36,67**	36,61	36,57	36,54	36,48	36,44	36,39	36,35
stdev	**0,41**	**0,40**	**0,42**	**0,43**	**0,45**	**0,45**	**0,44**	**0,43**	**0,42**	0,43	0,47	0,49	0,49	0,50	0,51	0,52
Tq4	**36,89**	**36,80**	**36,73**	**36,60**	**36,54**	**36,54**	**36,49**	**36,43**	36,37	36,31	36,25	36,22	36,15	36,12	36,06	36,02
stdev	**0,44**	**0,45**	**0,46**	**0,47**	**0,49**	**0,48**	**0,50**	**0,52**	0,53	0,54	0,56	0,58	0,62	0,62	0,63	0,63
Tt1	36,71	36,60	36,55	36,50	36,48	36,41	36,38	36,35	36,33	36,27	36,24	36,18	36,13	36,08	36,07	36,06
stdev	0,39	0,39	0,44	0,46	0,46	0,48	0,44	0,44	0,47	0,50	0,50	0,53	0,56	0,59	0,61	0,59
Tt2	36,61	36,52	36,44	36,37	36,32	36,23	36,19	36,14	36,11	36,03	35,98	35,94	35,89	35,83	35,81	35,80
stdev	0,66	0,67	0,68	0,73	0,70	0,71	0,65	0,63	0,64	0,66	0,69	0,74	0,72	0,75	0,78	0,74
Tt3	36,33	36,26	36,18	36,10	36,02	35,94	35,91	35,82	35,79	35,73	35,63	35,58	35,54	35,45	35,44	35,41
stdev	0,78	0,81	0,80	0,84	0,83	0,87	0,81	0,82	0,83	0,86	0,92	0,95	0,97	0,98	1,09	1,06
Tskn	33,03	33,03	33,01	33,02	33,03	33,01	33,03	33,03	33,02	33,00	32,96	32,94	32,92	32,90	32,89	32,86
stdev	0,44	0,45	0,46	0,47	0,49	0,48	0,50	0,52	0,53	0,54	0,56	0,58	0,62	0,62	0,63	0,63
Trec	37,59	37,55	37,50	37,48	37,42	37,40	37,40	37,36	37,34	37,30	37,30	37,28	37,28	37,27	37,25	37,24
stdev	0,27	0,26	0,23	0,22	0,21	0,22	0,21	0,22	0,21	0,21	0,21	0,21	0,21	0,21	0,21	0,23
Flux kJ/min	6,38	6,30	6,18	6,14	6,13	6,05	5,95	5,96	5,95	5,98	5,96	6,01	6,02	5,92	5,86	5,97
stdev	1,12	1,12	1,13	1,14	1,15	1,02	0,92	0,96	0,96	0,92	0,91	0,88	0,92	0,85	0,81	0,98
HR	84,05	83,33	85,20	83,77	83,36	78,38	78,91	78,09	76,09	76,15	76,53	74,03	76,50	73,46	71,35	73,44
stdev	14,20	16,23	11,14	11,48	9,60	9,88	10,02	11,96	7,54	6,81	8,89	7,13	9,40	6,61	5,94	8,12
E.E kJ/min	7,33	6,25	6,78	6,51	7,27											
stdev	1,99	1,64	1,8	2,07	1,98											
Quad BF	5,54	5,03	4,58	4,71	4,26	4,94	4,77	4,20	4,70	3,96	4,17	4,45	3,99	3,52	3,82	3,62
stdev	4,04	3,93	4,32	3,40	3,15	3,81	4,23	3,81	3,61	3,48	2,85	3,55	3,44	2,96	3,58	3,42
4Arm BF	11,33	10,62	10,55	10,26	10,64	10,50	9,31	9,68	10,87	11,28	10,82	11,50	10,76	12,46	11,97	10,14
stdev	10,68	10,06	11,60	7,96	7,29	7,61	6,45	6,54	9,66	11,93	9,95	11,68	6,16	12,51	10,31	9,13
Musc BF	6,53	3,34	3,02	5,60	3,86	15,17	7,35	4,79	2,31	2,43	3,84	4,35	4,86	2,99	4,19	2,98
stdev	0,44	0,45	0,46	0,47	0,49	0,48	0,50	0,52	0,53	0,54	0,56	0,58	0,62	0,62	0,63	0,63
BP Sys F																
stdev																
BP DiasF																
stdev																
BP Sys	119,00		119,00	114,00												
stdev	7,07		4,24	4,90												
BP Dias	78		78	78,5												
stdev	2,83		2,83	5,97												



Appendix E.3 : Mean (±SD) systolic and diastolique blood pressure