Thermal Homeostasis Following Dynamic Exercise

by

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Abstract

Recent published work showed post-exercise (PostEx) esophageal temperature (Tes) recovered rapidly to a persistent plateau that was significantly elevated (0.5°C or more) above pre-exercise (PreEx) values. Non-acral skin temperatures, except over exercised muscle, returned rapidly to PreEx levels. Rectal temperatures (Tre) fell gradually during recovery reaching a plateau late in recovery (45 min) equal in magnitude of difference from Tes to PreEx values. Surface temperatures over the quadriceps remained high, indicating that heat was trapped in muscle. A similarity between the exercise Tes at which skin surface dilation occurred (Tdil) and the PostEx Tes was identified. These observations contradict the widely accepted "load-error" principle of thermoregulation which predicts that displacement of core temperature (Tco) from a hypothalamic set point (SPhy) will induce defense reflexes until the displacement is reversed. These data lead to the hypothesis that there was some residual influence related to exercise that retained the modulation of thermal reflex thresholds during recovery. Testing of the hypothesis was conducted with experiments to establish if: 1) the PostEx Tes was related to PreEx temperature; 2) exogenous thermal loading would produce the same post treatment elevation; 3) PostEx Tes elevation followed by exogenous thermal loading would result in an increase in the Tes elevation and 4) a 5 min exercise generating Tes below Tdil would result in a PostEx elevation of Tes. It was demonstrated that repeated running-recovery cycles produced patterns of rise and then fall of Tes to an elevated PostEx plateau that was equal to Tdil . This was similar to previous results except that the second exercise was begun at an elevated Tes and produced further elevation of Tdil with a comparable effect on PostEx Tes. Similarly, the third exercise further increased Tes following which it recovered to an even higher plateau equal in magnitude to Tdil. We observed that exogenous heat loading, by immersion of subjects in a bath of water at 44°C to produce a rate of increase and peak elevation of Tes equal to exercise, did not result in a post-treatment elevation in Tes. Similarly, the PostEx Tes elevated plateau, equal to Tdil, remained unchanged following water immersion at 44°C despite a larger total heat gain during the immersion. These observations eliminate whole body heat content changes as the primary cause of the Tes elevation and support the hypothesis that the homeothermic defense mechanisms become inoperative during recovery at a temperature above resting values as defined by Tdil. The physiological importance of Tdil in defining upper limits of resting temperature cannot be determined at this point. However, the physiological relationship of Tdil with PostEx Tes suggests that neuro-muscular activity significantly influences thermolytic controls which persist in recovery. That Tdil may represent the upper limit of a range of "normal temperatures" is supported by data from a 5 min exercise performed to a Tes elevation below Tdil. Within minutes of exercise termination Tes achieved a stable elevated PostEx Tes (0.3°C or greater) which was maintained with no change over 65 min of recovery. The data suggest the possibility of: 1) a metabolically induced change in SPhy thermosensitivity, 2) a decreased sensitivity to an increase LE, or 3) a range of temperature regulation defined by an upper threshold control for thermolytic temperature defense reflexes.

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List of Abbreviations

CNS - central nervous system
CO - cardiac output
Ex - exercise
HR - heart rate
LDF - laser Doppler flow
LE - load error
POA - pre-optic and anterior hypothalamus
PostEx - post-exercise
PreEx - pre-exercise
SBF - skin blood flow
SE - standard error
SPhy - hypothalamic set-point
Tam - ambient temperature
Tba - back temperature
Tca - calf temperature
Tch - chest temperature
Tco - core temperature
Tdil - esophageal temperature equivalent at the time of surface dilation
Tes - esophageal temperature
Tfh - forehead temperature
Tfa - forearm temperature
Tfi - finger temperature
Tmu - muscle temperature
Tre - rectal temperature
Tsk - skin temperature
Tw - water temperature
VO2 - oxygen consumption
VO2max - maximal oxygen consumption

Chapter 1

1.0.0 Introduction

Changes in reflex activity associated with temperature regulation during thermal and/or exercise stress have traditionally been accepted as based on a hypothalamic set-point temperature (SP_{hy}) which is maintained by thermostatic influence on circulatory, metabolic, neuromuscular and sudomotor functions. Increases or decreases in core temperature (Tco) produce a load error (LE) that, when compared to SP_{hy}, invokes those responses aimed at defending against a further rise or fall of core temperature through the action of a number of autonomic effector responses (e.g. sweating, shivering and vasomotor adjustments). Most theoretical models of human thermoregulation have been developed on the SP_{hy} concept (Gagge et al. 1971; Cabanac and Massonet 1977; Stolwijk and Hardy 1977; McIntyre 1980; Bligh 1985). However, there is still much discussion as to which modalities are being controlled. Bligh (1978) suggests that core temperature or a combination of temperatures at different anatomical sites is the controlled variable, while others hold that body heat content is being controlled (Houdas et al. 1978) or that rate of heat outflow is adjusted to balance metabolic heat production (Webb et al. 1978). It is possible that the difficulties in resolving these different views lie not in the variable being regulated but rather in the SP_{hy} theory itself.

For example, it has been well documented that non-thermal stimuli can have a significant impact on the thresholds for sweating, shivering and vasomotor control and that the thresholds are very different for each of these thermal responses (Kenney and Johnson 1992; Johnson 1992). Mekjavic et al. (1991) identified separate temperature thresholds for sweating and shivering and concluded that there was a null zone of

temperature regulation. The null zone concept accepts that the thresholds of activity for sweating and shivering are significantly different and that, unlike the SP_{hy} concept, within this zone there is no reflex activity and that thermolytic mechanisms maintain a passive defence towards small fluctuations in core temperature. Previous research had suggested the existence of a null zone (Jessen and Ludwig 1971; Mekjavic and Bligh 1989) but their results were not sufficiently conclusive to support either the SP_{hy} or null zone concepts.

A second example of results that cannot be explained by the SP_{hy} theory can be seen in data from our laboratory. The SP_{hy} theory holds that during exercise there is a concurrent increase in the rate of heat loss to attenuate the rate of increase of core temperature (Sawka and Wenger 1988). Reducing the metabolic rate at the cessation of exercise should allow core temperature to return to pre-exercise (PreEx) levels based on the large difference that exists between core temperature and normal SP_{hy}. We observed an elevation of core temperature during an investigation aimed at developing a protocol to distinguish between different types of insulative clothing's. These data were atypical in that esophageal temperature (Tes) did not return to pre-exercise levels during the period of recovery. This led to the hypothesis that thermal reflex activation was attenuated or non-existent during this period (Appendix A). In order to verify these observations, a series of preliminary investigations were performed with different types of insulative clothing, exercise intensities and ambient temperatures (3.1.0-3.2.0).

The objective of this preliminary study was to investigate the pattern of post-exercise recovery of core temperature and surface temperatures (Tsk) following dynamic exercise under different conditions of ambient temperature, exercise intensity and insulative quality. The following experiments were conducted to investigate these objectives:

1) dynamic exercise performed at low (20°C) and high (40°C) ambient conditions,
2) dynamic exercise performed at low (20°C) ambient temperatures with moderate insulation [coveralls (OV)] and heavy insulation [chemical warfare protective garment (CW)],

3) dynamic exercise performed at high (40°C) ambient temperatures with moderate insulation (OV) and heavy insulation (CW),
4) dynamic exercise performed at normal room temperature (24°C) while wearing three different levels of insulation, no insulation [shorts only (SH)], moderate insulation (OV) and heavy insulation (CW).

It was concluded from these preliminary experiments that the post-exercise esophageal temperature reached a plateau at an elevated level for a prolonged period independent of the exercise intensities, ambient temperatures and surface insulation which were used. These preliminary observations were used to develop the rationale and objectives for the series of investigations that constitute this thesis.

The first principal experiment was conducted to test the hypothesis that the homeothermic defence mechanism that incorporates reflexive control of skin vasculature and which is changed following exercise above resting values (Johnson et al. 1974; Johnson and Park 1981), remains modified in the post-exercise state with the result that core temperature becomes stable at an elevated level for extended periods.

The objective of this experiment was to:

1) examine the relationship between Tsk and Tes during dynamic exercise and in recovery at thermoneutral conditions (ambient temperature 29°C).

The post-exercise esophageal temperature was significantly ($p>0.05$) elevated from pre-exercise over the full recovery period. The esophageal temperature in recovery was equal to the esophageal temperature at the time of vasodilation of surface vasculature (Tdil) during exercise, suggesting that thermal reflexive vasodilation of surface vasculature was significantly reduced before pre-exercise resting core temperature could be re-established. The passive attitude of thermal control systems towards elevated core temperature during recovery may strengthen the idea of a change in SP_{hy} activity or

constitute a demonstration of a range of normal temperatures within which thermoregulatory mechanisms are relatively insensitive to core temperature fluctuations.

Based on these conclusions, experiments were conducted to investigate the stability of the post-exercise elevation of esophageal temperature in relation to changes in vascular conductance promoted by the generation of core temperature changes through endogenous (continuous and repeated) and exogenous (water immersion) heat load treatments (6.0.0-6.3.0). The experiments were designed to test the following hypotheses:

1) that an increase in the PreEx Tco will result in a higher PostEx Tes,
2) that an increase in the PreEx Tco will result in an elevated exercise Tdil,
3) that there is a direct relationship between the exercise Tdil and the PostEx Tes,
4) that an elevation of Tco above Tdil by an exogenous heat load (water immersion) will not result in a PostEx Tes plateau ,
5) that an increase in Tco following exercise recovery by an exogenous heat load (water immersion) will not result in an increase of Tes above PostEx value and,
6) than an increase in Tco to a value below Tdil will result in a sustained PostEx Tes elevation.

The objectives of these experiments were to:

1) investigate the Tes response following repeated exercise,
2) investigate the Tes response following hot water immersion (water temperature 44°C),
3) investigate the Tes response following a combination of exercise and hot water immersion (water temperature 44°C),
4) investigate the Tes response following an exercise duration less than the time required to reach Tdil (5 min exercise).

Chapter 2

2.0.0 Review of Literature

2.1.0 General characteristics of thermal control

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 (SP_{hy}). As with the control of many other physiological parameters such as blood composition and blood pressure, thermal homeostasis is the result of a complex organization of physiological and behavioral reflexes through which Tco is regulated within appropriate limits and most descriptive models are based on this control mechanism (Stolwijk and Hardy 1966; McIntyre 1980; Nadel 1983; Johnson and Ruhling 1985; Sawka and Wenger 1988). A change in Tco from SP_{hy} 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 SP_{hy}. Deviations from the acceptable range give rise to LE signals. A LE signal is proportional to the difference between the SP_{hy} and the weighted input from both core and surface thermosensitive receptors (Johnson and Ruhling 1985) and results in the activation of effector responses that oppose a further change.

There are three factors that determine how closely Tco will be maintained at or near the SP_{hy}. The first factor is gain or sensitivity of an effector organ and the second factor is the time lag associated with thermolytic threshold response. Gain is defined as the degree to which the output of an effector is altered for a given change in the controlled variable. Generally, if the gain is low changes in the LE signal will have a relatively small

effect on the output of the effector and the controlled variable and larger LE signals will be needed to change the output of the effector by a given amount (Sawka and Wenger 1988). Thus, the effect of a low gain is that a continuing influence of perturbing factors can force the controlled variable further from the SP_{hy} than would be the case if the gain was large.

Time lags may have several origins: the time needed for sensory information to reach the integrator; the time needed for action potentials from the integrator to travel to the effector and the delay in the response of the effector itself which is essentially immediate (Sawka and Wenger 1988). The primary impact of differing time lags among receptor and communication sites is upon the way in which the results of thermal and non-thermal stimuli are interpreted and therefore in the time required to reach threshold.

Human temperature regulation is characterized by effector responses of shivering and non-shivering thermogenesis, sweating, peripheral vasomotor adjustments and behavior. Although the mechanisms involved in homeothermy tend to stabilize internal Tco, they cannot completely prevent temperature variation in response to internal and environmental thermal stress. Thus, "normal" Tco implies specific conditions under which the measurement is made. Deviations of $\pm$ 0.5°C from mean temperatures can be expected in healthy individuals, even under carefully controlled conditions (Brengelman 1973). Furthermore, individual temperature may differ with time even though other conditions are constant. A diurnal variation (Sagot et al. 1987; Sawka and Wenger 1988; Zahorska-Markiewicz et al. 1989; Alfoldi et al. 1990; Cisse et al. 1991; Fedorova et al. 1992; Deswasmes et al. 1994) and longer-term fluctuations such as that associated with ovulation (Sawka and Wenger 1988; Frascarolo et al. 1992, may be as high as 1.0°C or more. The existence of fluctuations brings into question the generally accepted concepts of temperature regulation that contend that central control mechanism and SP_{hy} can maintain Tco within narrow limits. It also suggests that there may be non-thermal

influences such as metabolic, neuroendocrine or humoral which either directly or indirectly influence Tco regulation.

The intent of this review is to summarize the current understanding of thermal regulatory control mechanisms, models, and measurement techniques in order to provide an understanding of S_{Phy}.

2.2.0 Hypothalamic thermosensitivity

Hammel et al. (1960) established a functional relationship between changes of Tco and the subsequent regulation by special hypothalamic neurons in long term implantation studies of unanesthetized female Mongrel dogs that showed that those neurons were indeed temperature sensitive. The findings by Hammel were supported by Nakayama et al. (1961), Fusco et al. (1961) and Adams (1963) who also determined, by experimental introduction of specialized needle-shaped thermodes that produced highly localized changes in brain temperature, that the thermosensitive neurons in the brain were located in the hypothalamus. Adams (1964) 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. However, it is also well established that hypothalamic function is not limited to temperature regulation and that there are multisensor neurons sensitive to different local stimuli (i.e. temperature, osmolality, metabolic substances, hormones, cytokines) which are present and integrated in the hypothalamus (Hori et al. 1988). The hypothalamus is also the target of information transmitted from peripheral sensory systems and from

superior parts of the central nervous system (CNS) which tends to modify hypothalamic activity, therefore altering its response (Heller 1983; Pierau and Schmid 1990).

Since the identification of warm-sensitive and cold-sensitive neurons by brain slice studies, specialized thermosensitive neurons were subsequently found in slice preparations of avian POA (Nakashima et al., 1987) and mammalian extra-POA tissues such as the ventromedial hypothalamic nucleus (Hori et al., 1988), septum (Nakashima et al, 1989), organum vasculosum, lamina terminalis (Matsuda et al., 1990), red nucleus (Asami et al., 1988) and dorsal motor nucleus of the vagus in rats (Hori, 1990).

Advances in experimental techniques have only recently provided more information on the role of thermosensitive neurons in thermoregulation. Thermally induced changes in thermosensitive neurons by localized warming and cooling of the POA, medulla oblongata and spinal cord produced heat-defence responses and cold-defence responses. It was also found that the responsiveness of thermosensitive neurons in the POA and extra-POA is not limited to localized modification of temperature but is contributed as well as by peripheral changes, thus suggesting a complex array of integrated input that influences POA activity . These studies establish that a thermally induced change is coordinated by inputs derived from both extra-POA and POA thermosensitive neurons where the extra-POA neurons have direct and modifying influences that are received directly by or are indirectly targeted to the hypothalamus (Hori and Harada 1976; Boulant 1980; Nakayama 1985; Simon et al. 1986).

2.2.1 Modulators of POA thermosensitivity

Hori (1984) found that use of the neurotoxin, Capsaicin, influences thermoregulation by initially stimulating and subsequently desensitizing warm-sensitive neurons. Rat studies show that treatment with this neurotoxin specifically alters the thermolytic defence mechanism in responding to a rise in Tco, thus suggesting an

independent control of both heat and cold response. These results emphasize that changes in thermolytic response can be significantly altered by the influence of neurochemicals. Other examples of central modulators which are known to influence the activity of POA neuronal activity include lipopolysaccharide, interleukin-1, α-interferon, tumor necrosis factor, noradrenaline, dopamine and thyroid releasing hormone (Zeisberger, 1990). These modulators produce hyperthermia by inducing a decrease in the heat-defence responses and an increase in the cold-defence responses. Numerous studies have shown that they inhibit the activity of POA warm-sensitive neurons and excite a similar population of cold-sensitive neurons, and the extent to which POA neuronal activity is influenced is dependent on the specific modulator (Hori et al. 1973; Hori et al. 1982; Nakashima et al. 1985; Hori et al. 1988; Nakashima et al. 1988; Nakashima et al. 1989). In contrast, substances such as serotonin, angiotensin II and III, arginine vasopressin, and capsaicin, which evoke hypothermia by increased heat-defence and decreased cold-defence of thermoregulation, have the opposite effect on a majority of POA thermosensitive neurons (Hori et al. 1973; Hori 1984; Kiyohara et al. 1984).

Experiments involving neurally mediated changes in POA activity have demonstrated a significant influence on whole body temperature regulation. Hori et al. (1982) showed that electrical stimulation of the ventral subiculum of the hippocampus in anesthetized rats was correlated with the inhibition of a majority of warm and cold POA thermosensitive neurons. Osaka et al. (1984) found that POA thermosensitive neurons, and therefore thermoregulatory effector responses were significantly influenced following a reversible ablation of the prefrontal cortex induced by cortical spreading depression.

Single neuron studies showed that multiple integrators such as the warm signal network and the cold signal network with primarily separate and independent channels, from thermosenstive neurons to effectors, are collectively grouped into the two sets of a thermoregulatory information processing system in the CNS. These findings showed that

there are two sets of neural networks for the processing of thermal signals relating to thermoregulation, i.e. one which increases neural activity in response to a rise in Tsk and Tco, referred to as the warm signal network, and the other called the cold signal network which shows the opposite response to negative temperature changes. It has been suggested that neurons belonging to the warm signal network (i.e. warm-sensitive neurons) activate heat-defence responses of thermoregulation and that neurons in the cold signal network (i.e. cold-sensitive neurons) facilitate cold-defence responses of thermoregulation (Heller 1983).

2.2.2 Non-thermal influences on POA thermosensitivity

Cabanac (1975) concluded that it is probably more accurate to consider the control system for the regulation of Tco as consisting of a number of networks operating independently of one another. Bligh (1985) concluded that much of the difficulty in representing thermal homeostatic regulation and therefore the influence which it has on maintaining Tco is for the most part overshadowed by the rather numerous and inter-related vegetative processes which overlap with thermal control in the reflex pathways that are involved. This overlap makes it difficult to determine what principles actually underlie the stabilization of Tco relative to $SP_{hy.}$ Horowitz (1990) and Zeisberger (1990) suggest that the mechanism of regulation of thermal effector response depends on non-homeostatic influences as well as direct hypothalamic stimulation. Zeisberger (1990) offers the view that no system is independent but that each is interconnected and works in concert with the others.

Preoptic hypothalamic cooling in birds and posterior hypothalamic temperature changes in goats resulted in non homeostatic responses (Pierau and Schmid 1990). Activity of POA thermosensitive neurons have been shown to be influenced by both cardiopulmonary and baroreflex receptor inputs (Hilton and Spyer 1971; Spyer 1972;

Faires et al. 1984). In rat studies, Faires et al. (1984) demonstrated that stimulation of the POA produces a moderate hypotension and bradycardia while Hilton and Spyer (1971) showed that bilateral lesions in the POA attenuate the baroreceptor reflex. More recently, Koga et al. (1987), investigated the responsiveness of POA neurons to changes in hypothalamic temperature and arterial blood pressure in anaesthetized rats. They found that the thermosensitive neurons in the POA were at least involved in some phases of cardiovascular regulation. The implication of their results is significant in the interpretation of Tco regulation during exercise. Specifically, it was found that the activity of both warm-sensitive and cold-sensitive neurons was significantly altered. It was concluded that hypotensive or hypertensive changes can significantly alter the patterns of temperature control and therefore change thermal regulatory control thresholds.

Nakashima et al. (1985) demonstrated that local osmotic change can have a significant impact on SP_{hy} control of Tco. They observed that neuronal activity of both warm-sensitive and cold-sensitive cell types were equally depressed by hyperosmotic stimuli. Similarly, Baker and Doris (1982) concluded that, since warm-sensitive neurons and cold-sensitive neurons in the POA facilitate heat-defence responses and cold-defence responses respectively, the decreased activity of both types of thermosensitive neurons in hyperosmotic conditions may partially explain the suppressed evaporative and non-evaporative heat loss and the reduced metabolic heat production observed in dehydrated rats.

The extent to which non-thermal stimuli can influence changes in POA activity, and therefore in Tco regulation, is demonstrated by the responsiveness of the POA thermosensitive neurons to emotional and behavioral stimuli. Briese and Cabanac (1991) found that the handling and/or disturbance of the rats can result in a shift of SP_{hy} similar to fever. Exposure of laboratory animals to any of a variety of stressors has been shown to activate the hypothalamo-pituitary-adrenocortical axis and affect thermoregulatory

homeostasis to result in an elevated Tco (Briese et al. 1970; Blasig et al. 1978; Vidal et al. 1983; York et al. 1982; Singer et al. 1986; Briese and Cabanac 1990). This phenomenon has received much attention because it occurs each time a rat is approached or manipulated, thus contaminating all experimental work on non-anesthetized rats (Briese et al. 1970; Blasig et al. 1978; York and Regan 1982; Vidal et al. 1983; Singer et al. 1986).

Numerous studies suggest that the POA responsiveness to nonthermoregulatory reflexes together with their abundant neural connections to divergent areas of the brain may play a principal role in the coordination of different homeostatic functions and increase the ability to defend against severe thermal disturbances (Hori et al. 1982; Hori et al. 1984; Kiyohara et al. 1984; Shibata et al. 1988).

2.3.0 Spinal cord thermosensitivity

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. There is now ample evidence that cooling and warming the spinal cord via extradural thermodes in the vertebral canal are followed by appropriate thermoregulatory responses; cooling results in shivering, reduced skin blood flow, and pilomotoric 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 in detail by Simon (1974) and other reviews which include this topic (Bligh 1973; Cabanac 1975).

Rautenberg et al., (1963) first identified temperature sensitive spinal neurons in anesthetized dogs. Simon and Iriki (1971) detected increased activity in the anterolateral spinal columns when warm and cold stimuli were applied to the thoracic and lumbar spinal cord segments. Simon (1974) demonstrated that even in the absence of CNS input and integration of afferent input, spinal transections at high cervical levels did not fully inhibit thermoregulatory responses to thermal stimulation of the spinal cord. They concluded that thermoregulatory regulation is not exclusively controlled by POA thermosensitive neurons but is also influenced and regulated at the spinal level. Further evidence can be found in the work of Simon et al. (1966) who observed that spinal cooling in both acute or chronically spinalized dogs triggered the shivering response. Similar responses were observed in rabbits (Kosaka and Simon 1968), cats (Herdman 1978), and pigeons (Gorke and Pierau 1979).

Recent studies demonstrate the importance of the interactive effect of spinal and POA thermosensitive influence on the control of Tco. Herdman (1978) and Gorke and Pierau (1979) observed that the threshold response following spinal cooling or warming was at a lower spinal temperature in acute preparations as compared to a similar treatment on POA thermosensitive neurons suggesting the thermoregulatory response is most likely a combined response of many temperature sensitive sites. They concluded that the spinal cord activity significantly influences POA activity. Guieu and Hardy (1970) had previously suggested that such an influence can modify threshold controls of body temperature regulation.

It has been shown that thermal stimulation of the spinal cord following peripheral denervation by cutting the dorsal roots could activate with heat gain (shivering) and heat loss (vasodilation, panting) in dogs (Meurer et al. 1967; Jessen and Simon-Oppermann 1976) and in pigeons (Necker and Rautenberg 1975). Specifically, Necker and Rautenberg (1975) found that body temperature is regulated at a slightly lower level in a cold environment. Clamping the spinal cord to normal values resulted in a poikilothermic

state in a cold environment. This clearly demonstrates the ability of the spinal cord to function as a temperature-sensitive area in the control of body temperature.

The question arises whether natural fluctuations of spinal temperature can be correlated with changes in thermoregulatory effector activity. This problem has been studied only in pigeons (Graf and Necker 1979) in which variations of up to 1.0°C often occur. During continuous recordings at an ambient temperature of 20°C a mean deviation of ± 0.5°C was found during the active period, defined as daylight conditions. When correlating with the electromyogram which was used to indicate shivering, no significant correlation with the spinal temperature was found at constant ambient temperatures of 10 and 20°C. While there was no clear correlation at constant ambient temperature, there was a correlation during slow ambient cooling during which shivering was correlated positively with the spinal temperature. Graf and Necker concluded that spinal cord involvement in thermoregulatory effector activity, although not statistically significant, may be important in maintaining relative constancy in internal temperature. They suggested that it was most probably the effect of interaction of temperature-sensitive sites, which were not controlled for, which may have altered spinal cord influence on temperature control.

2.4.0 Lower brain stem thermosensitivity

In contrast to the many studies involving the spinal cord and POA, only a few investigations have examined the thermosensitivity of the lower brain stem. Inoue and Murakami (1976) found neurons in the reticular formation of the medulla of rabbit neurons that responded to local thermal stimulation. Both warm-sensitive and cold-sensitive neurons were found, but the cold-sensitive type as more numerous, which was in contrast to all other areas of the CNS where warm-sensitive ones are found to dominate

(Hensel 1976). The caudal region of the medial mid-brain of cats also was found to have a significant concentration of thermosensitive neurons (Cronin and Baker 1976). Chai and Lin (1972) investigated the effects of heating and cooling the spinal cord and medulla oblongata on thermoregulation in monkeys. Heating of the medulla oblongata by a thermode in the fourth ventricle to 42°C resulted in vasodilation and an increase in respiratory frequency. Changes in the spinal cord produced comparable results. Similarly, cooling of the medulla to 32°C produced a general vasoconstriction and decreased respiratory rate but spinal cooling was followed by shivering of the limbs.

Cooling of the rabbit mid brain has been shown to be followed by an increased shivering response and oxygen consumption (Hardy 1969). Similarly, Simon-Oppermann and Martin (1979) observed constriction of surface flow and a decreased body temperature following cooling of the caudal mid-brain and rostral medulla in ducks. Heating of the rabbit mid-brain was found to affect vasomotor control by causing increased surface flow in the rabbit (Hardy 1969), while panting was induced following localized heating of the mid-brain in chickens (Richards and Avery 1978).

Cronin and Baker (1977) found that the thermosensitivity of the mid brain was significantly lower than those of the spinal and hypothalamic thermosensitive neurons. They found that the thermal sensitivity of warm-sensitive neurons in the lower brain stem yielded a mean sensitivity of about 1.5 impulses/sec·°C as compared to findings of Simon and Iriki (1971) who found that the mean sensitivity of the spinal cord and hypothalamus was about 5 impulses/sec·°C. These observations were supported by the findings of Cabanac and Hardy (1969) who found that the effects of rabbit mid-brain cooling and heating were small compared with the response following hypothalamic stimulation. In comparison, inducing equal temperature changes in the hypothalamus or spinal cord have been observed to produce approximately equal effects (Jessen and Ludwig 1971; Jessen and Mayer 1971; Jessen and Simon 1971).

2.5.0 Intra-abdominal thermosensitivity

Rawson and Quick (1970) found that intra-abdominal heating in sheep resulted in significant thermoregulatory adjustments. Ruminal cooling to 16°C was followed by an immediate fall in respiratory frequency and subsequent shivering. 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. Cranston et al. (1977, 1978) concluded from a study of rabbits and goats that there are no temperature sensors in the right heart, lung, posterior vena cava, posterior vein, liver or hepatic vein. However, 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, spinal cord and lower brain thermal activity, the role of such stimulation and influence in overall temperature regulation is yet to be determined, although Simon and Simon-Oppermann (1979) concluded that intra-abdominal cold receptors have a significantly greater effect on temperature regulation than has been shown for brain or spinal cord stimulation in ducks.

2.6.0 Interaction between different thermosensitive areas

The relative significance of abdominal tissues, spinal cord, and lower brain stem thermosensitivity in core temperature regulation is not fully understood. These data do demonstrate that the interaction of the different thermosensitive areas in conjunction with the POA thermosensitive neurons has an integral function in maintaining some 'normal' level of regulation by which Tco is maintained within a narrow range. Much of the difficulty in studying the effects of each of the individual sites one at a time is that thermal stimulation of one site brings about a stimulus on other areas which may be opposite in nature. For example, if an animal is caused to pant by heating his spinal cord, other internal temperatures fall as a result of the increased heat loss (Bligh 1973). The other temperature-sensitive sites initiate a heat conserving response, thus offsetting the original deviation. It has been concluded that the thermosensitivity of neurons which are involved in temperature regulation seems to be cooperative in the sense that appropriate responses occur (Bligh 1973; Cabanac 1975).

There is ample evidence of appropriate thermoregulatory responses to thermal stimulation of the CNS. That there is generally a deviation of Tco during central stimulation may result from the stimulation of a selected area, a situation which does not occur under natural conditions. Since the response must be based on a change in the activity of central neurons it is not surprising that, in areas where thermal stimulation elicited thermoregulatory responses, temperature-sensitive neurons were found. Neuronal activity was found with threshold characteristics and thresholds which were often comparable to normal Tco. Finally, the responses shown in selective thermal stimulation's indicate that, at corresponding natural variations under certain conditions, a contribution of the thermosensitivity of the CNS to the overall thermoregulatory activity should predominate over all other thermosensitive regions. But this contribution of natural variations has not yet been demonstrated convincingly (Hori et al. 1988).

The difficulty in transposing the physiological importance of the interactive effects of the different thermosensitive regions is in part due to two factors: 1) The demonstration of the existence of temperature-sensitive neurons does not prove that the neurons are actually involved in temperature regulation, since neurons which clearly do not belong to the thermoregulatory system have also been shown to be temperature sensitive. 2) There is accumulating evidence that non homeostatic reactions occur in response to thermal stimulation of the CNS which in turn influences SP_{hy} activity, altering threshold control of thermoregulatory reflexes to changes in Tco (Simon-Oppermann and Jessen 1977; Simon-Oppermann et al.1979, Zeisberger 1990).

2.7.0 Thermal information processing

The details of how temperature information is sensed by the body and transferred, integrated and processed by the controller and transformed into effector signals is incomplete. In particular, the mechanism of establishing the so-called SP_{hy} and the way in which it changes with factors such as time or exercise are not fully understood. 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. Most models also agree that the primary control center for thermoregulation is the POA.

The three essential elements common to all models are:

(i) receptors that sense the existing central (controlled) temperature;

(ii) vasomotor, sudomotor and metabolic effector mechanisms that are capable of altering the central temperature; and

(iii) integrative structures that presumably compare sensed temperature to "normal" temperature (SP_{hy}), determine whether existing temperature is too high or too low, and activate the appropriate motor response.

2.7.1 Set-point concept

McIntyre (1980) developed a simplified system of temperature regulation that is representative of most models that are based on SP_{hy} theory. The model shows that if the brain temperature rises above the SP_{hy} the anterior hypothalamus causes vasodilation and sweating, whereas a reduction in skin temperature produces vasoconstriction and also contributes to shivering. Inhibitory pathways between the anterior and posterior hypothalamus provide 'clamping' and prevent the system from working against itself and experiencing unnecessary oscillation.

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 required SP_{hy}. This concept has since been described as the Load Error (LE) concept that is representative of the temperature difference between internal temperature change and SP_{hy} (Sawka and Wenger 1988; Stolwijk and Hardy 1977). Deviations from SP_{hy} 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 SP_{hy}) then the effectors which result in increased heat loss will be activated. Conversely, for small negative values of the LE (i.e. weighted signal is slightly less than SP_{hy}), heat will be conserved, while large negative values of the LE will stimulate the generation of body heat

by shivering. However, although the LE concept attempts to represent fluctuations in Tco regulation it does assume that Tco is representative of one single temperature and that the magnitude and rate of change in Tsk for different regions are similar. It has been demonstrated that SP_{hy} activity is interactively influenced by other CNS and extra-CNS thermosensitive regions (Bligh 1978, Heller et al. 1983, Hori et al. 1984). Similarly, different regions of the body have been shown to normally have significantly different temperatures, thus making it difficult to assess which Tco temperature is actually influencing SP_{hy} control (Saltin and Hermansen 1966; Livingstone et al. 1983). Finally, incorporating mean Tsk in the concept assumes that all surface areas change in a similar way to either ambient temperature or Tco changes. However, it would be more accurate if different regions have a variable input on SP_{hy} (Ducharme et al. 1991; Ducharme and Tikuisis 1992; Webb et al. 1992) where Tsk changes are primarily dependent on convective heat exchange (Ducharme et al. 1991). Therefore, the use of an integrated thermal response based on selected measurement sites to describe LE may underestimate true changes in SP_{hy} regulation during dynamic changes in Tco. Accepting the idea of variable Tsk and Tco inputs may affect how threshold changes are interpreted under different conditions.

Stitt (1979) described the LE concept relative to an elevation of Tco in exercise. As figure 1 demonstrates, Tco rises during exercise because of the increase in metabolic heat production. SP_{hy} (Tset) remains unchanged, and heat-dissipating responses are elicited as Tco rises. Tco continues to rise until the error signal (e) is high enough to elicit heat loss responses at a rate sufficient to match heat production, and produce a new thermal balance.

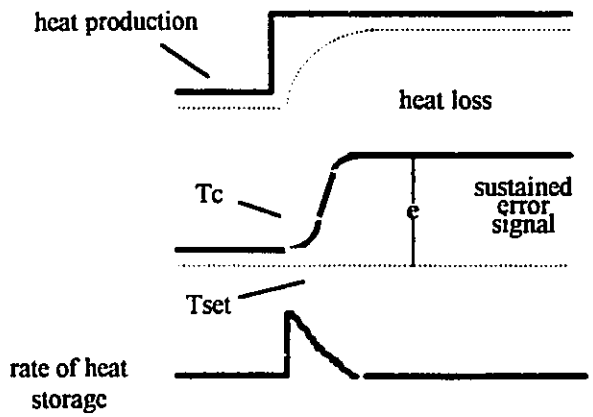

Fig. 1 Elevation of core temperature in exercise (Stitt 1979).

Also important in the control of thermal response are changes in Tsk. For example, shivering in man may occur immediately upon exposure too cold. Not only does this happen before the cooling had time to decrease Tco, but Tco may actually show a transient rise (Mekjavic and Bligh 1987). These findings are consistent with the observation that local changes in hypothalamic temperature alone cannot account for the total response of Tco regulation (Hellstrom and Hammel 1967).

Hellstrom and Hammel (1967) investigated Tco changes in unanesthetized dogs fitted with chronically implanted thermodes at various combinations of air and hypothalamic temperature. Hypothalamic temperature was clamped at desired levels by means of seven chronically implanted thermodes arranged in an array bracketing the temperature-sensitive regions in the anterior hypothalamus. It was observed that for the same hypothalamic temperature, higher evaporative rates of heat loss occur in a warmer environment owing to the influence of the higher Tsk. The authors concluded from the slopes of the curves that the higher the Tsk, the lower the hypothalamic temperature at which the first elevation in evaporative response was observed. Their experiments also showed that different rectal temperature resulted in different relationships between hypothalamic temperature and thermoregulatory response at the same environmental

temperature. Dogs with lower rectal temperatures had higher metabolic heat production than those with a lower rectal and hypothalamic temperature. It was suggested that the influence on the SP_{hy} was not limited to Tsk changes but was in part due to individual or combined inputs from other thermosenstive regions such as the spinal cord and other deep body sites.

Nadel (1983) concluded that there are multiple pathways involved in temperature regulation. 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.

To suggest that the mechanism of human thermoregulation is as simple as implied by modeling would underestimate the true complexity of the system. An appropriate conclusion based on the available data is that the description of mechanisms of thermal control is still incomplete. However, the models that have been presented do represent a generalized understanding of temperature regulation as it exists today.

2.7.2 Null zone concept

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. In contrast, observations that there was a single temperature for the activation of heat conserving and heat releasing responses were taken to indicate that Tco is regulated to a single temperature, as described by SP_{hy} theory (Cabanac and Massonet 1977).

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 esophagus 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 Tes and Tre respectively. Previous research had suggested the existence of such a null zone (Jessen and Ludwig 1971; Mekjavic and Bligh 1989) but their results of different thresholds for sweating and shivering were not statistically significant to support a null zone range.

Jessen and Ludwig (1971) investigated spinal cord and hypothalamic thermosensitive regions as core sensors of core temperature in conscious dogs. Temperatures of the spinal cord and hypothalamus were altered simultaneously and were correlated with heat production (shivering) and respiratory evaporative heat loss (panting). A 1°C difference in hypothalamic temperature was recorded between shivering and sweating. They concluded that in the sequence of effector systems involved in temperature regulation previously mentioned, shivering and panting represent opposite effectors, between which finer adjustments to thermal loads are performed by variations in cutaneous blood flow. The study is limited by the fact that only three dogs were studied over repeated trials.

Although Benzinger et al. (1961) used the term set-point to describe their experimental result, of the influence of mean skin temperature on the threshold of core temperature for evaporative heat loss by sweating and heat production in shivering, their results indicated a null zone between the core temperature thresholds for sweating and shivering of 0.5°C when Tsk is between 28 and 32°C. As Tsk was raised from 28 to

32°C, the threshold core temperature for both shivering and sweating were progressively depressed. Thus while the range of Tco null zone was essentially uninfluenced by the limited variation in Tsk, the threshold Tco's were influenced by Tsk.

Cabanac and Massonet (1977) found that during water immersion experiments the Tco thresholds for sweating and shivering were so close to justify the term set-point or null-point. When the heat content of the human body was being slowly raised by immersion to the neck in water at 38°C the threshold Tco for sweating was 37.38°C and when the water temperature was abruptly lowered to 28°C and Tco started to fall, sweating finally ceased at 37.32°C and shivering commenced at 37.40°C. It was concluded that the Tco thresholds for sweating and shivering were virtually identical. Mekjavic and Bligh (1989) following a comparable protocol obtained conflicting results to those of Cabanac and Massonet (1977). Mekjavic and Bligh (1989) immersed the subjects in a water bath maintained at 40°C until Tco reached 38.5°C or 20 min had elapsed. Upon completion of the warm water immersion, subjects were transferred into a bath in which the water temperature was maintained at 28°C where they remained until sweating was completely abolished and shivering began. In a second trial core temperature was elevated by moderate exercise on a bicycle ergometer in air at room temperature of 20-22°C. The subjects were requested to exercise on a Monark bicycle ergometer at a work rate equivalent to 50% VO_{2max} for 20 min.

These protocols were performed to assess whether the elevation of Tco with moderate exercise followed by immersion in 28°C water would produce thermal threshold Tco's as those reported by Cabanac and Massonet (1977). It was found that exogenous heating involved a considerable shift in Tsk and again upon transfer to water at 28°C while the exercise protocol resulted in a much smaller changes in Tsk, which may have had a significant impact on the measurements of thermal thresholds. The onset of sweating at a lower esophageal temperature during immersion (36.9°C) than during exercise (37.4°C) was attributed to the different Tsk. Esophageal temperature fell most rapidly and thus

sweating was extinguished at a lower Tes following 40°C immersion than following exercise. Although they failed to determine a core threshold for shivering thermogenesis, they concluded that Tes fell sufficiently below the temperature at which sweating ceased, indicating a thermoregulatory null-zone between the extinction Tes for sweating and the threshold Tco for shivering.

Unlike the work of Mekjavic et al. (1991), the original studies identified the threshold for sweating and shivering thermogenesis under separate experimental conditions and determined the two thresholds at different Tsk. They did not account for variability that exists from day to day despite standardized conditions which may make it difficult to identify differences that depend on Tco measures (Livingstone et al. 1992) and for the role that Tsk plays in establishing thresholds (Sawka and Wenger 1988).

If we consider the protocol used by Mekjavic et al. (1991), these effects are eliminated. However, their protocol required an elevation of Tco by exercise in order to measure changes in sweating threshold while literally clamping Tsk at or around 28°C by continual immersion in a water bath. This method assumed two things, although neither were mentioned in their reports. 1) exercise does not influence the threshold responses of either sweating or shivering thermogenesis; 2) the threshold activity of sudomotor and shivering responses are similar for both conditions of endogenous (exercise) and exogenous (water immersion) heating and cooling. Thoden et al. (1994a) demonstrated a PostEx disturbance of thermal homeostasis following acute dynamic exercise. It was observed that the regulation of Tco was passive [skin blood flow (SBF) was no higher than resting levels and sweating had disappeared over the period of recovery] suggesting that thermolytic thresholds had been influenced by the previous exercise. Secondly, Johnson et al. 1974 and Johnson and Park 1981 showed an elevation of the internal temperature threshold for cutaneous vasodilation by dynamic exercise. Thus, the findings of Thoden et al. (1994a) may support an hypothesis that a recovery from exercise may show the same magnitude of changes to thermal reflex thresholds as had been shown from

rest to exercise. Therefore, although the results of Mekjavik et al. (1991) are indeed significant, they must be interpreted with caution.

2.7.3 Core shell concept

Another approach to thermal modeling called the core-shell concept of temperature regulation involves compartmental heat exchange. Previous models of temperature control rely on the assumption that changes in Tco are regulated either to a single temperature or temperatures or to an upper and lower limit of control. These models assume a consistency which is maintained at steady-state rest and that under exogenous or endogenous conditions of thermal stress, Tco fluctuations from normal are controlled by the appropriate effector responses which attempt to maintain Tco at new elevated steady state or attenuate its rise. Neither the SP_{hy} nor null zone concepts consider variable inputs and that transitional changes in temperature from one region to another may not elicit consistent responses.

Aschoff and Wever (1958) formalized and introduced the core-shell concept. They suggested that body temperature regulation is primarily controlled by changes in regional heat distribution. 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. Mekjavic and Bligh (1987) showed that the thermolytic mechanisms can 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 affecting POA activity. Therefore the response to varying conditions of cold or heat stress will depend on the rate of heat flow determined by the blood supply and tissue density (Ducharme et al. 1991). Webb and co-workers (1991) speculate that the 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. Such a receptor has not been identified nor as there been any evidence to suggest its existence. Nevertheless, the notion that subcutaneous temperature receptors exists is not new. Bazett et al. (1950) first suggested the possible role of subcutaneous temperature receptors in detecting heat flow through the skin, for which indirect evidence may be found in multiple Tsk and Tco measurements.

Saltin and Hermansen (1966) compared temperature changes during dynamic exercise in three major sites of measurement: esophageal, rectal and muscle temperature. The principal observation was that there was a very different Tco recorded for each of the individual sites, thus bringing into question which measure of temperature change is being controlled by SP_{hy}. It is well known that the temperature within a given body region depends upon the metabolic rate of the surrounding tissues, the source and magnitude of blood flow, and the temperature gradients between body regions (Sawka and Wenger 1988). Therefore, considerable temperature gradients exist between and within different orifices, body cavities and blood vessels (Horvath 1950; Eichna et al. 1951; Saltin and Hermansen 1966; Shiraki et al. 1986) and it is these temperature gradients which are said to drive SP_{hy} control of Tco.

For resting humans, about 70% of the metabolic heat is produced by internal organs and viscera within the body core (Aschoff and Wever 1958; Stolwijk and Hardy 1966). During muscular exercise, however, 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 Tco. During exercise the temperature within the active skeletal

muscle rapidly exceeds Tco (Aikas et al. 1962; Saltin and Hermansen 1966; Saltin et al. 1970), while the temperature within the inactive skeletal muscles and other body regions rises more gradually and Tco is elevated regardless of the site of its measurement. 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 and 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; Giesbrecht and Bristow 1992; McArdle et al. 1992; Webb 1992). The phenomenon of an after-drop has been documented in conditions of hypothermia (Tikuisis et al. 1991; Giesbrecht and Bristow 1992; Bristow et al. 1993). Their 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. The after-rise response in Tco has been observed as a rise of body Tco following the removal of passive heating of the periphery. Similar to the after-drop response, the after-rise observed in Tco was shown to be a function of flow redistribution and therefore a convective mechanism of heat exchange. Ducharme et al., (1991) concluded that the convective effect and therefore the heat flow between compartments is an important parameter which must be considered in the overall control of Tco, especially under dynamic conditions.

2.8.0 Thermal heat balance

While the essence of thermoregulatory theory is a maintenance of Tco within a narrow range oriented around the SP_{hy}, all models including the null zone and heat exchange concepts describe how the body can maintain an internal temperature near 37°C

in terms of heat generation and heat exchange processes. In practice, what is achieved is not a steady state (constant temperature) but a dynamic equilibrium. As external conditions continually change, the body responds to 'regulate' Tco. 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. Heat would then be stored and body temperature would rise at about $1°C \cdot hr^{-1}$ for a resting person. However, in most cases there is an effective temperature gradient between the core and skin and there is a net transfer from the cells of 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.

2.8.1 Physical Mechanisms of Heat Transfer

The human body is constantly exchanging heat energy 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 rays. 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. room temperature=21-22°C), 60% of the heat loss occurs via radiation. This is possible because Tsk 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 more dense, 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 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 air movement, the convection is termed forced. Heat loss by convection 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.

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 negligible except in unusual circumstances.

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 metabolic rate is increased and under extreme environmental conditions (Taylor 1986; Sawka and Wenger 1988). The body loses 0.58 kcal of heat for each gram of water that evaporates when the environmental temperature is greater than that of the skin while the body gains heat by radiation and conduction. If the body cannot lose heat by evaporation under these circumstances, then body temperature would rise if it were not for its potential to sweat and access evaporative heat loss. 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 vapor.

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 $ml \cdot day^{-1}$. This amounts to a continual heat loss of about 12 to 18 $kcal \cdot hr^{-1}$.

Insensible water loss cannot be controlled and it occurs regardless of body temperature. However, evaporative sweat loss can be controlled by regulating the rate of

sweating. Except for insensible water loss (i.e. evaporative heat loss through ventilation and sweating under the arms and in the genital region), sweat rates are essentially zero when the Tsk is low. In hot weather, an unacclimatized individual (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 acclimated 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).

Sweat occurs both from apocrine and eccrine glands. The apocrine sweat glands exist primarily under the arms and in the genital region. Eccrine sweat glands are tubular structures consisting of a deep, coiled portion that secretes sweat and a duct portion that passes outward through the dermis of the skin. They cover most of the body and secrete a clear sweat that accounts for most of the evaporative heat loss in the body. In addition to water, sodium chloride, urea, lactic acid, and potassium ions are also lost in sweat. Efferent nerves involved in the sweating reflex originate in the hypothalamus and descend through the brain stem and spinal tract, crossing at various segmental levels and ending in the lateral horn where they synapse on peripheral neurons. Non-myelinated class C, sympathetic cholinergic fibers, are involved in sweating and were first described by Dale and Feldberg in the 1930's. Both α and β-adrenergic receptors are present in human sweat glands. However; there is considerable controversy over the interaction between the cholinergic and adrenergic controls (Sato 1977). Kuno (1965) reported that adrenergic input potentiates cholinergic actions, while Terada (1966) reported inhibition of sweating by adrenergic fibers. Both investigators agree that during exercise, adrenergic input increases total sweat output. It is well documented that neural, hormonal and mechanical factors all interact to regulate the volume and composition of sweat (Taylor 1986). The principal efferent signal which stimulates sweat production is sympathetic cholinergic output from the anterior hypothalamus. This output is largely initiated in response to afferent input and is proportional to the deep body temperature (Tam et al. 1978). Above a threshold Tco, a linear relationship exists between sweat output and the

rise in Tco (Tam et al. 1978; Astrand and Rodhal 1986; Sawka and Wenger 1988). In addition, increasing ambient temperature and mean Tsk, increases the sweat rate to Tco relationship (Sawka and Wenger 1988; Bothorel et al. 1991).

There are two principal non-behavioral 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.

2.8.2 Control of skin 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 innervated solely by noradrenergic sympathetic vasoconstrictor nerves (Fox and Edholm 1963; Rowell 1977; Johnson 1986). All thermoregulatory and nonthermoregulatory reflexes in acral skin regions are therefore mediated by alterations in active vasoconstrictor tone. The efferent neural 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 (Fox and Edholm 1963; Rowell 1977; 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 (Fox and Hilton 1958; Love and Shanks 1962; Fox and Edholm 1963; Johnson 1986). The complexity of this dual innervation has made understanding the neural control of non-acral cutaneous blood vessels more difficult.

Skin circulation at a given location is affected by temperature in two ways: 1) temperatures in the core and of the skin elsewhere on the body, affect SBF by sympathetic nervous system reflexes and; 2) Tsk affects the local vascular smooth muscle directly. During cold exposure, and with certain nonthermoregulatory reflexes such as regulation of arterial blood pressure, and control of the distribution of blood flow during exercise, SBF is reduced through the action of vasoconstrictor fibers since their action is blocked by bretylium (Blair et al. 1960; Kellogg et al. 1989). Bretylium is a positively charged, anti-adrenergic agent that is taken up by adrenergic neurons. Once within the nerve terminal the drug blocks the release of norepinephrine without interfering with axon transmission (Haeusler et al. 1979). Cutaneous vasoconstrictor nerves release norepinephrine through α_1- and perhaps α_2- receptors (Fox and Edholm 1963; Rowell

1977; Johnson 1986; Johnson et al. 1986). In the hands, feet, lips, ears, and nose, these fibers are the predominant vasomotor innervation, 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 fibers and by a unique vasodilator system, which is responsible for 95 to 100 % of the total increase in SBF during heat stress (Kenney and 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.

Skin temperature also affects SBF through direct action on the blood vessels themselves (Brown and Page 1952; Brown et al. 1953). Local temperature acts on skin blood vessels in at least two ways. Local cooling potentiates, and heating weakens, the contractile response of vascular smooth muscle to norepinephrine and other constrictor agonists, apparently by changing the affinity of α_2-adrenergic receptors (Johnson et al. 1986). Specifically, cooling has been demonstrated to influence the receptor-mediated smooth muscle response to vasoactive substances in blood vessels (Arneklo-Noblin et al. 1987). Decreasing the temperature below 37°C enhances the contraction induced by noradrenaline, acetylcholine, adenosine triphosphate and 5-hydroxytryptamine (Vanhoutte and Sheperd 1970a) and increases β-adrenergically mediated relaxation (Vanhoutte and Sheperd 1970b) in the dog saphenous vein. Cooling has also been shown to enhance the contractile response to 5-HT in the rat jugular vein (Arneklo-Nobin et al. 1987) as well as catecholamine-induced contractions in human skin vessels (Winkelmann et al. 1977). In the canine saphenous vein, cooling augments α_2-adrenergic contractile responses (Flavahan et al. 1985; Flavahan and Vanhoutte 1986). Ekenvall et al. (1988) showed in humans that cold-induced finger vasoconstriction was abolished by an α_2-antagonist but was unaffected by an α_1-antagonist. In human saphenous veins, Harker et al. (1990)

showed that cooling augmented reactivity to norepinephrine through increased responsiveness of α_2-adrenoceptors. It has been proposed that the differential effects of cooling serve to transfer blood from superficial to deep blood vessels to prevent heat loss (Vanhoutte 1980).

In contrast to the effects of cooling, it has been demonstrated that warming augmented α_1- but reduced α_2-adrenergic vasoconstriction in humans (Freedman et al. 1992). Similar results also have been obtained in studies using the canine saphenous veins (Cooke et al. 1984). In human forearm, local heating of the skin causes vasodilation, and local cooling causes vasoconstriction, even in the absence of nervous signals (Wenger et al. 1986). Through this direct vasodilator effect, local heating increases SBF so much that most of the heat delivered to the skin is carried away by the blood and little is conducted to the deeper tissues.

2.8.3 Control of skin blood flow at rest

In a resting human, the response in skin blood flow (SBF) to heat stress is characterized by an internal temperature threshold, beyond which the rise in SBF is rapid (Barcroft and Edholm 1943; Allwood and Burry 1954; Edholm et al. 1957). Since very steep slopes can only be sustained over a small rise in internal temperature, the primary advantage of a steep response is to reach maximal values of SBF with relatively small increases in internal temperature. Although internal temperature is the primary thermoregulatory drive in the control of SBF, Tsk can also reflexively affect SBF. However, this reflex effect is relatively small under conditions of supine rest (Wyss et al. 1974; Proppe et al. 1976) Further, increased Tsk initiates a reflexive control of SBF if the initial temperature is below 33°C, which is most likely due to the fact that vasoconstrictor tone is high only when Tsk is low (Sawka and Wenger 1988). Therefore, the response of

SBF to an elevation in Tsk depends on the level of core temperature and initial Tsk (Johnson and Park 1982; Johnson et al. 1984).

2.8.4 Control of skin blood flow during exercise

Dynamic exercise creates a primary drive for redistribution of blood flow away from metabolically inactive tissues, including skin, to active muscle (Kenney and Johnson 1992). 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 (Blair et al. 1960; Bevegard and Shepherd 1966; Zelis et al. 1969; Johnson and Park 1982; Hirata et al. 1983; Johnson 1990) which is directly related to the level of exercise (Taylor et al. 1988) and a comparable intensity-dependent vasoconstriction in the renal and splanchnic regional circulations (Rowell 1974). Measurement of SBF by Laser Doppler flowmetry has confirmed the initial constrictor response of the cutaneous vasculature with the onset of exercise (Taylor et al. 1989; Taylor et al. 1990). However, the origin of the cutaneous vasoconstrictor response during intense dynamic exercise in humans is not fully understood. Two neural control mechanisms are usually described in the autonomic adjustments to exercise. One has its origin in the CNS and has been called "central command" (Goodwin et al. 1972), whereas the other has its origin in skeletal muscle (Coote et al. 1971; McCloskey and Mitchell 1972) and is due to activation of mechanoreceptors and metaboreceptors reflecting the amount of work being performed (Mitchell et al. 1985). Friedman et al. (1991) demonstrated that factors related to the

activity of the exercising muscle and its metabolism rather than intended effort determine the cutaneous vasoconstrictor response to the initiation of intense dynamic exercise in humans.

As exercise is extended beyond 5-10 min, the rising Tco initiates a net cutaneous vasodilation (Johnson et al. 1974; Hirata et al. 1983). The mechanism by which reflexes involving active vasocontrictor activity cause cutaneous vasoconstriction at the onset of exercise appears to differ markedly from that by which exercise reaches the threshold for vasodilation, where only the involvement of the active vasodilator system is involved. Kellogg et al. (1991a) resolved this question through selective blockade of the adrenergic vasoconstrictor system with the iontophoretic application of bretylium to a small area of skin (see p. 31). The internal temperature at which exercise-induced vasodilation began was identical between sites treated with bretylium and untreated sites both in condition of rest and exercise (Kellogg et al. 1991b). However, the thresholds differed significantly between rest and exercise, in keeping with earlier observations (Johnson et al. 1974; Johnson and Park 1981; Kellogg et al. 1991b). These data demonstrated that the thermoregulatory threshold for cutaneous vasodilation is increased by exercise through a delay in the activation of the active vasodilator system. There is no apparent involvement of the vasoconstrictor system. The rate of SBF is primarily determined by the Tco. Above an internal Tco threshold for vasodilation, the increase in SBF is proportional to the increase in Tco. Increasing the average Tsk causes a reduction in the Tco threshold for vasodilation without affecting the slope of the SBF to Tco relation (Wenger et al. 1975; Sawka and Wenger 1988).

The fact that there are independent thresholds for resting and exercise conditions makes it difficult to compare and interpret the relationship of temperature regulation during transitions from rest to exercise and vice versa. Similarly, 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 (Zelis et

al. 1969; Tam et al. 1978; Hirata et al. 1984, 1984; Smolander et al. 1987; Smolander et al. 1991), exercise posture (Roberts and Wenger 1980; Johnson and Park 1981), duration of the exercise period (Smolander et al. 1987), exercise training (Roberts et al. 1977); heat acclimation (Roberts et al. 1977), circadian rhythms (Alfoldi et al. 1990; Tobler et al. 1993), dehydration (Thornton and Proppe 1988) and ambient temperature (Wenger et al. 1975; Johnson and Park 1982; Smolander et al. 1987). With so many factors known to influence threshold control of surface vascular controls, there is a real difficulty associated with interpreting such changes. This difficulty is complicated further by the fact that vascular controls of various origins (i.e. skin and muscle) are very different and that information from these separate areas can have a variable effect on the hypothalamic control. Hypothalamic control of vascular flow can also be modified, making the interpretation of true thermolytic response difficult to distinguish from non-thermal influences alone (Henriksen 1991).

2.8.5 Thermoregulatory controls of exercise recovery

In a series of experiments performed in our laboratory, Tco response to human exercise at different intensities and ambient temperatures while wearing differing types of insulation demonstrated a recovery response which would not have been expected according to currently accepted thermoregulatory theory. This response was demonstrated as a prolonged (65 min or longer) elevation of esophageal (Tes) (Thoden et al. 1994a). It was expected that the exercise-induced elevation of Tes following cessation of exercise would be gradually eliminated within a short period. Several studies have reported a post-exercise temperature profile (Johnson and Park 1982; Savard et al. 1988; Cable and Green 1990; Kranning and Gonzalez 1991; Kaciuba-Uscilko et al. 1992). However, none have shown a similar prolonged elevation in Tco following exercise; instead they have shown a gradual decay in Tco over time. Johnson and Park (1982) did

show a similar trend in Tes response during intermittent exercise, but their experimental protocol maintained Tsk at a constant level for the experiment.

There are those who have argued that an elevation in Tco during exercise is an indication of a change in SP_{hy} (Nielsen 1938; Jackson and Hammel 1963; Nielsen 1966; Saltin and Hermansen 1966; Tam et al. 1978). Their explanation was that the hypothalamic thermostat is reset during high metabolic rates. Principal arguments against an adjustment of the SP_{hy} re-adjustment are that, unlike fever, the high metabolic rate of exercise is required to both elevate and maintain Tco, and that the concurrent increase in the rate of heat loss to attenuate the rate of increase of Tco indicates that the SP_{hy} is lower than the level attained or maintained during the exercise (Sawka and Wenger 1988). Thus, reducing the metabolic rate by the cessation of exercise should allow Tco to return to pre-exercise levels if thermal reflex defenses remain activated.

Despite the fact that there is, at most moderate levels of work, a cardiovascular reserve with which to offset increases in Tco, accessing this potential does not seem to be a process that is adopted by the system. For example, the ability of the system to increase heat loss by increasing blood flow to the skin would seem to be a solution to limit any further rise or to even reduce the heat load retained during continued muscular work. Skin blood flow for an average man performing a work load equivalent to 50% VO_{2max} is approximately 2.6 $L \cdot min^{-1}$ (Rowell 1974; Taylor et al. 1984). Rowell (1974) and Taylor et al. (1984) noted that under full vasodilation, blood flow to the surface can be as high as 8 $L \cdot min^{-1}$. The cardiac output 50% VO_{2max} represents about 50% of maximum capacity (Sawka and Wenger 1988) and muscle blood flow at this intensity has been shown to be adequate to maintain tissue oxygenation (Savard et al. 1988), and prevent PtO_2 and PvO_2 from reaching critical levels (Astrand and Rodhal 1986). In most people, the 5.4 $L \cdot min^{-1}$ difference between SBF at 50% VO_{2max} and maximum rates is well within the capability of the remaining 50% of cardiac output. Nevertheless, despite a potential for greater SBF

the evidence is that heat loss responses are only recruited to a level which will balance heat production at an elevated Tco.

The fact that temperature regulation is mediated primarily to achieve an elevated steady state temperature would suggest that the system has either been compromised or is operating at a different criterion threshold than that associated with resting temperatures. If there is more than adequate ability to both dissipate heat and supply muscle blood flow, it could be postulated that the effector mechanisms are being controlled in a way which is consistent with a change in sensitivity or SP_{hy}. This rationale can also be extended to periods of recovery from muscular work where it was observed that Tes remains elevated for a prolonged period (Thoden et al. 1994a), making it appear comparable to a fever response. In a febrile state the system has the potential to get rid of heat but a pyrogenic-induced alteration in SP_{hy} maintains the heat load until such time as the influence on the pre-optic and anterior hypothalamic temperature sensitive neurons is removed (Riedel 1990).

The possibility that exercise causes a change in sensitivity to LE which persists into recovery is possible. Sensible heat loss mechanisms are adequate to deal with the increased heat gain at work loads below 140 $kcal \cdot hr^{-1}$. Beyond rates of heat gain of 200 $kcal \cdot hr^{-1}$ the insensible mechanism becomes the primary and most important pathway for heat loss.

It has been demonstrated under a number of different conditions, such as varying levels of work intensity (Smolander et al. 1987; Smolander et al. 1991), ambient temperature (Kenney and Johnson 1992) and skin temperature (Nadel 1977; Taylor et al. 1984; Kenney and Johnson 1992), that there are changes in the threshold temperatures for sweating (Sawka and Wenger 1988; Johnson 1992) and active vasodilation (Kellog et al. 1991; Kenney and Johnson 1992). Prostaglandin mediated effects on SP_{hy} activity during fever demonstrate similar results for thermal reflex thresholds (Riedel 1990). There are a number of other conditions which demonstrate threshold changes. These include phase of

menstrual cycle, circadian rhythms and heat acclimation (Sawka and Wenger 1988). Whether or not this same effect is demonstrated during muscular work has not been documented.

There have been a few studies which demonstrate a post-exercise (PostEx) elevation of Tco. These studies involve very intense exercise over a prolonged period. These studies may provide some insight into the possible mechanisms of a PostEx disturbance in thermal homeostasis. Haight and Keatinge (1973) and Johnson and Ruhling (1985) demonstrated that after intense prolonged work periods Tco remains elevated for several hours. This phenomenon has been ascribed to metabolic factors, plasma osmolality changes and pyrogenic factors.

Metabolic factors related to exercise-induced changes in mitochondrial function where fatty acids (or their acyl-CoA derivatives) may act as uncouplers of mitochondrial respiration and phosphorylation or lead to an increased Na^+/K^+ pump activity stimulated by the increased permeability of these ions due to the action of circulating noradrenaline (Johnson and Ruhling 1985). With respect to plasma osmolality changes, Fortney and Vroman (1985) demonstrated that exercise-induced plasma hyperosmolality resulted in a 0.6°C increase in Tes, a 0.5°C increase in the threshold for forearm vasodilation, and 0.6° C increase in the threshold for sweating during subsequent bouts of exercise when compared with controls. Cannon and Kluger (1983) demonstrated that circulating monocytes during exercise may be pyrogenic and modify the SP_{hy} through prostaglandin-dependent processes. Johnson and Ruhling (1985) suggested that PostEx hyperthermia may be the result of SP_{hy} modifications superimposed on the normal Tco elevation resulting from LE. However they cite previous work by Johnson et al. (1984) as discrediting the possibility of pyrogenic involvement by demonstrating that PostEx hyperthermia did not appear to have any components affected by antipyrogen (aspirin) treatment as long as 60 min PostEx.

However, while it seems possible to rule out an aspirin-sensitive prostaglandin mediated effect on SP_{hy}, it is possible that the effect of recruiting musculature and the resultant metabolic by-products and heat load may serve to modulate thermal control thresholds. Indeed the possibility exists that the thermal stress associated with an increased metabolic rate can have a significant influence on SP_{hy} control to result in an elevated Tco. This hypothesis is not without support. Exposure of laboratory animals to any kind of stress activates the hypothalamo-pituitary-adrenocortical axis, as demonstrated by the increased Tco following enhanced release of corticotropin-releasing hormone (CRH) from the paraventricular nucleus of the hypothalamus (Briese et al. 1970; Blasig et al. 1978; Vidal et al. 1983; York and Regan 1982; Singer et al. 1986; Cabanac and Briese 1991).

It is generally accepted that a change in any part of the thermoregulatory system is therefore accompanied by changes in other vegetative functions and vice versa (Cabanac 1975; Bligh 1985; Horrowitz 1990; Zeisberger 1990). Such a network of influences gives rise to the possibility that a thermal stress placed on the system, either by ambient temperature changes or increased metabolic rates, can cause a complex series of physiological responses that act indirectly or directly on the hypothalamus and produce changes in the thresholds of sudomotor, shivering and vasomotor controls.

There are a number of studies which demonstrate this interaction. For example, Matsumura et al. (1987) observed that changes in PCO_2 directly affected pre-optic thermosensitive neurons and produced changes in pre-optic temperature. These findings were supported by Tamaki et al. (1989) who found that changes in PCO_2 and blood acidosis significantly alter the pattern of regulation of Tco.

Koga et al. (1987) demonstrated that the pre-optic thermosensitive neurons are affected by changes in arterial blood pressure as a consequence of thermal stress and exercise. In fact dynamic exercise can precipitate other changes in non-thermal regulatory responses which act indirectly to modify thermolytic control mechanisms. Travis and

Johnson (1993) noted that dynamic exercise caused increase in plasma osmolality, a decrease in plasma volume and a resultant increase in Tco. They state that these changes can cause the release of vasopressin into the systemic circulation, where it may have profound cardiovascular effects. This peptide is a potent vasoconstrictor that can cause marked increase in vascular resistance and decreases in blood flow to regional vascular beds such as skin.

A phenomenon called the PostEx hypotensive effect has been observed following dynamic exercise by Wilcox et al. 1982; Kaufman et al. 1987 and Brown et al. 1993 who concluded that it is related to cutaneous cooling in recovery. They suggest that the inverse relationship between Tsk and blood pressure underlies a probable peripheral (i.e. lower limb) and/or visceral organ pooling of blood, thus trapping deep body heat and reducing stroke volume by small reductions in venous return. These responses demonstrated a physiological reduction in the capacity for heat loss despite an elevation of Tco.

The effects of the preceding factors underscore the complex interaction of both thermal and non-thermal influences on temperature regulation. Moreover, as discussed earlier in the chapter, they are known to modulate threshold activity of SP_{hy} control of thermolytic and thermogenic responses to changes in Tco, and must therefore be considered as potential sources to explain the PostEx hyperthermia.

2.9.0 Measurements of core temperatures

It is important to use the appropriate measures that best represent normal changes and to understand the mechanisms of thermal control. There are numerous methods available to measure Tco. For example, the rectum, axilla, sublingual sulcus, ear canal or tympanic membrane, and esophagus (behind the left atrium and greater vessels) are all

sites which 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 per unit of organ mass determines thermal inertia or 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 in the esophagus when muscle and skin change temperature. Conversely, tissues of the lower abdomen and sublingual sulcus 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 (Cranston et al. 1954; Gerbrandy et al. 1954; Cooper and Kenyon 1957; Mairiaux et al. 1983; Saltin and 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 which is able to monitor acute thermal transient changes.

Although mixed venous blood might provide the best measure of some "average" Tco, it cannot be assumed that the inputs from individual organs to central thermosensors will result in the same average. There is still considerable debate among thermal physiologists concerning the best measurement site and the correct interpretation of the different indices of Tco (Brengelmann, 1987). The idea that a single temperature measurement can provide an index of the average internal temperature may not be reasonable. Early researchers agreed that the practice of representing average internal temperature as a single measurement was not valid (Bazett et al. 1950; Horvath et al. 1950). Yet despite these conclusion it is common practice to express changes in Tco

based on a single measured value and with measurements of body temperature based on a weighted sum of mean skin and core temperatures.

The idea that a single temperature measurement can provide a close approximation of the Tco input to the thermoregulatory controller presents theoretical and practical problems. First, it is well documented that the thermoregulatory controller receives temperature input from many possible sources (animal studies). Second, although SP_{hy} is believed to be a major input into the thermoregulatory controller, neither brain temperature nor hypothalamic temperature has been well quantified in humans (Whitby and Dunkin, 1971).

2.9.1 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 (Sawka and Wenger 1988; Mekjavic and Rempel 1990). At the level of the left atrium, the heart and esophagus are in contact and are fairly isothermal (Brengelmann, 1987). Most thermal physiologists agree that Tes is the best non-invasive index of Tco for humans, based on the findings that Tes responds rapidly to changes in blood temperature and closely parallels changes in SP_{hy} (Molnar and Read 1974;. Shiraki et al. 1986). Shiraki et al. (1986) found that Tes 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 Tes and the temperature of active skeletal muscle during exercise (Aikas et al., 1962; Saltin and Hermansen 1966; Saltin et al. 1970; Saltin et al. 1972). The results demonstrated that during exercise Tes and active muscle temperature achieved steady-state values almost simultaneously after 15-20 min. The rapid response time for Tes is due to the low heat capacity of the

esophagus and its proximity to the heart, allowing it to be rapidly warmed and cooled by post-respiratory mixed venous blood (Sawka and Wenger 1988).

2.9.2 Rectal temperature

Temperature measurements are uniform within the rectum up to 100-270 mm from the anal sphincter (Nielsen and Nielsen 1962). Rectal temperature (Tre) 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 (Saltin and Hermansen 1966; Astrand and Rodhal 1986). Rectal temperature is generally higher than values measured in arterial blood (Aulick et al. 1981; Sawka and Wenger 1988) and other Tco sites. Steady state value of Tre is generally ~0.2°C higher than simultaneously measured Tes (Saltin and Hermansen 1966; Saltin et al. 1970) and they are independent of ambient temperature. Steady-state Tre, however; provides a good index of body heat storage (Saltin and Hermansen 1966). The main problem with Tre is that it is slow to respond to changes in blood (Saltin and Hermansen 1966). During exercise it takes approximately 25-40 minutes to achieve steady-state Tre values (Aikas et al. 1962; Nielsen and Nielsen 1962; Saltin et al. 1970; Greenleaf and Castle 1972; Saltin et al. 1972). The reason for the slow response of Tre 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 Tre a poor index for estimating the input to the thermoregulatory controller (Saltin et al. 1970). Yet despite the limitations, steady-state Tre is widely employed by thermal physiologists because of the ease and comfort in its use and the majority of thermoregulatory theory and modeling as well as the presumed acute responses are based on data from this source.

Chapter 3

3.0.0 Basic experimental methods

All subjects were volunteer students from the University of Ottawa and they responded negatively to all questions on the ParQ (Appendix I) questionnaire. Each subject was thoroughly acquainted with the experiment during an information session and gave informed consent as prescribed by the Faculty of Health Sciences Research Ethics Committee. VO_{2max} was predicted by linear extrapolation of heart rate (HR) during maximal treadmill running and used as a basis for establishing relatively equivalent individual subject exercise intensities, as recommended by Astrand (1960) for equating thermal stress.

Experiments were conducted in the morning following a 24-h period without heavy or prolonged physical activity, the last 12 h of which included a pasta-based meal, abstinence from stimulants and alcohol, 8 h of sleep and a minimum of 0.25 l of water during each waking hour. Breakfast consisted only of fruit juice and care was taken to avoid major thermal stimuli or substantial increases of metabolic rate between rising and starting the experiment (Appendix B). Esophageal temperature (Tes) and rectal temperature (Tre) were monitored as indices of internal temperature and recorded via catheters. The Tes catheter containing a thermocouple (Baxter, model 400) was placed via the nares and swallowed to a depth equal to one-quarter of body height at the level of the left atrium and greater vessels (Sawka and Wenger, 1988; Mekjavic and Rempel 1990). The Tre catheter (Yellow Springs Instruments model 401) was inserted by the subject to a depth of 110-120 mm into the anus. Tsk was monitored with thermistors (Yellow Springs Instruments model 44004) taped to the following sites using medical adhesive: the center of the forehead (Tfh), the left pectoralis chest (Tch), left anterior

forearm midway between the wrist and elbow (Tfa), left anterior middle finger tip (Tfi), left anterior thigh midway between hip and knee (Tth), the left posterior calf (Tca) midway between the ankle and knee and the left upper back (Tba) (Note: not all sites were used for each of the experiments. Those sites used in the respective investigations will be indicated accordingly). Bilateral measurements were usually taken for verification or as backup data. Catheters and thermistors were calibrated regularly against a water bath and mercury thermometer with a 0.01°C scale. Temperatures were collected and digitized (Hewlett Packard data acquisition module, model 3497A) at 5-s intervals, stored (Hewlett Packard, model PC-312, 9000) and statistically analyzed without smoothing. Heart rate (HR) was recorded by means of a wireless transmission device (Polar Electro PE3000). Oxygen consumption (VO_2) was determined by open-circuit analysis using an automated gas collection system (Quinton Qplex, model 1).

All experimental protocols followed a common experimental design format. Subjects entered a thermal control room for a 2 hr of sitting habituation to the experimental temperature. The equilibration period was followed by a treatment period of either exercise or water immersion treatments. Treatment was begun after the resting Tes did not vary by more than ± 0.05°C over 10 min. Prior to all exercise periods, the subject stood, mounted the treadmill, inserted the two-way breathing valve used for VO_2 collection (Collins, Hans-Rudolph, model 2700) and remained relatively still until postural effects on Tes had ceased. No water was permitted after mouthpiece insertion and the subject expelled saliva into the valve in order to minimize the effect of swallowing on the Tes thermistor. The treadmill (Quinton, model 18-54) running exercise intensity was chosen based on the subject's VO_{2max} (Appendix G). Following the treatment period the subjects stood leaning against a support in the temperature control chamber over a prescribed period of time. Treatment and recovery procedures were repeated in the case of intermittent exercise and endogenous and exogenous heating. Temperature and heart rate data were collected for a minimum of 10 min pre-exercise, and for the entire

treatment and recovery periods. Oxygen consumption was collected 10 min pre-treatment, during treatment and 10 min into recovery. Oxygen consumption was not collected during exogenous heating periods. Deviations from these methods which are specific to a particular experiment are described in subsequent sections.

Statistical analysis was performed between pre-treatment and post-treatment data using a repeated measures analysis of variance using the Dunn's procedure for planned comparisons (Kirk 1968). Data analysis was performed using the general linear model procedure. In order to deal efficiently with the correlation of repeated measures, the general linear model process uses the multivariate method of specifying the model even if a univariate analysis is required (SAS Institute Inc. 1985, Freund et al. 1986). Variable factorial designs for repeated measures analysis of variance were performed on the preliminary data (Section 3.1.0 and 3.2.0). Rate dependent comparisons of single temperature responses within conditions were compared using a paired t-test procedure (Kirk 1968). Pearson correlation analysis was performed between pre-selected variables (surface vascular dilation temperatures and post-exercise esophageal temperature) in intermittent exercise.

3.1.0 Phase 1 - Condition of sensible and insensible heat exchange

3.1.1 Introduction

During a series of investigations aimed at developing a protocol to distinguish between light, moderate and heavy insulation, a trend to sustained elevated Tes was observed following repeated 15 min bouts of intermittent exercise (Appendix A). The duration of the elevations following the first and second bouts of exercise were unclear because of the rather short recovery periods (10 min) and Tes tracings that were

interrupted due to the effects of swallowing (Appendix A). However, Tes following the third and final exercise bout was unquestionably elevated compared to PreEx values and remained elevated for the full 30 min of recovery. This observation was found regardless of the type of clothing worn and was atypical of the response that was expected based on the SP_{hy} theory. It was expected that the exercise-induced elevation of Tes following cessation of exercise would be gradually eliminated within a short period.

Further experiments were subsequently designed in conjunction with Defence Research Establishment of Ottawa (DREO) to generate a protocol that could discriminate between insulation type using a shorter experimental protocol than had been previously employed in evaluations of clothing. Intermittent exercise protocols which had been previously used were time-consuming and stressful without any significant differences being demonstrated.

Following a series of pilot studies, a single bout exercise period using a work intensity equivalent of 75% VO_{2max} lasting no longer than 20 min was found to consistently produce measurable differences between clothing types. In addition, the experimental technique of encouraging the subject to expel saliva into the mouthpiece and avoid swallowing was developed to improve the quality of Tes tracings.

While the principal purpose of this experiment was to investigate changes in Tco and Tsk response during exercise in order to distinguish clothing type, the data were also used to investigate the Tes response following dynamic exercise.

3.1.2 Specific methods

Six male volunteer subjects participated in the experiment. Their average age, height and weight were 22±2 yr, 178±2 cm and 78±3 Kg respectively. Measurements of internal temperatures were monitored by Tes and Tre and Tsk measurements included Tfh,

Tch and Tfi. The subjects entered the thermal chamber for a 2 hr acclimation period at 24°C, 50% humidity wearing only shorts and running shoes. At 15 min PreEx, the subject stood and was fitted with the one of the following insulation types:

1) no insulation (SH)

2) moderate insulation - single knit coverall garment (OV) and,

3) heavy insulation - chemical warfare protective garment (CW).

The clothing change was completed after the first 5 min of standing. The subject mounted the treadmill and inserted the two-way breathing valve for VO_2 gas collection. Treadmill running exercise was chosen to stimulate about 75% of the subject's VO_{2max}. Following cessation of exercise the subjects stood leaning against an elevated support for 30 min of recovery .

3.1.3 Results

PreEx esophageal (Tes) and rectal (Tre) temperatures for all conditions were similar and stable prior to exercise for all insulation levels. PreEx Tes was less than Tre by 0.3°C to 0.4°C for SH, OV and CW conditions respectively (table 1). Surface temperatures, finger (Tfi), forehead (Tfh) and chest (Tch) were stable and at a plateau (table 2). At the onset of exercise Tes fell below PreEx values. After approximately 2-2.5 min it began to rise at a rate equal to 0.16°C·min^{-1}, 0.17°C·min^{-1} and 0.18°C·min^{-1} for each of SH, OV and CW conditions respectively. At 9-10 min into exercise all conditions showed a downward inflection in Tes. This inflection represented a significantly different ($p>0.05$) rate of rise of Tes equal to 0.03°C·min^{-1} (-78%), 0.05°C·min^{-1} (-71%), and 0.07° C·min^{-1} (-64%) for SH, OV and CW respectively (fig. 2). In contrast, Tre did not show any inflections and was much slower in response across all conditions. Tsk showed a small drop at the onset of exercise. Tfi SH fell the most (2.1°C), while Tfh and Tch

showed a much smaller decrease in the order of 0.1°C and 0.5°C respectively. The fall in Tsk was reduced with increase in insulation and no fall was recorded in the CW condition for Tfh and Tch. However, there was a delay in Tsk rise demonstrated by both the non-acral and acral sites. At approximately 7 min into exercise there was a sharp upward inflection in Tsk. The change in Tsk preceded the inflection in Tes which occurred approximately 2 min later. All temperatures continued to rise throughout the remainder of exercise.

Table 1. Mean core temperatures for different insulation type.

	SH Tes (°C)	**SH Tre** (°C)	**OV Tes** (°C)	**OV Tre** (°C)	**CW Tes** (°C)	**CW Tre** (°C)
PreEx 10 min	36.60	37.02	36.59	37.02	36.61	36.95
SE	.08	.08	.07	.08	.09	.10
Start Ex	36.64	37.02	36.67	37.06	36.72	36.99
SE	.08	.08	.09	.08	.13	.12
End Ex	37.73†‡	37.95‡	38.18‡	38.02‡	38.42‡	38.08‡
SE	.12	.09	.16	.14	.15	.08
PostEx 10 min	37.18†‡	38.02‡	37.46‡	38.11‡	37.70‡	38.26‡
SE	.14	.10	.13	.16	.10	.10
PostEx 20 min	37.19†‡	37.99‡	37.37‡	38.03‡	37.57‡	38.21‡
SE	.14	.09	.12	.17	.09	.11
PostEx 30 min	37.19†‡	37.92‡	37.36‡	37.98‡	37.55‡	38.17‡
SE	.13	.10	.11	.17	.07	.12

(† - indicates significant difference from CW; ‡ -indicates significant difference from PreEx, $p>0.05$)

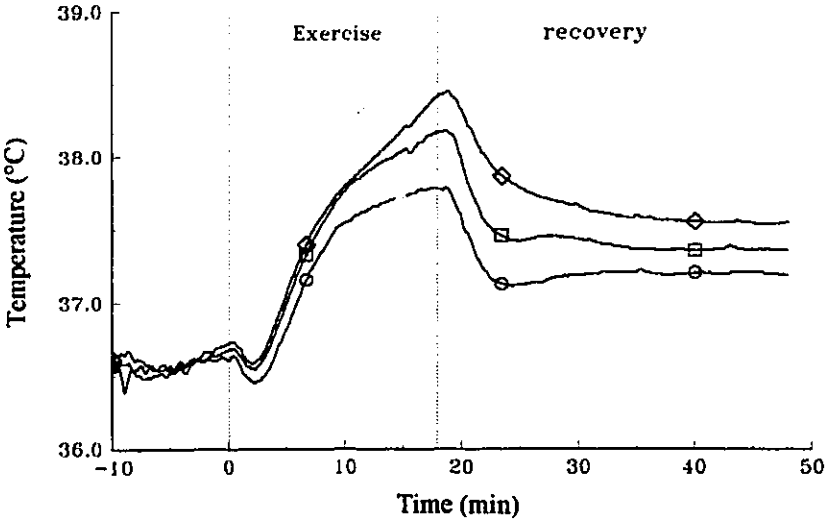

Fig. 2. Mean esophageal temperatures from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery for different insulation types. Esophageal temperature for very light insulation (O), moderate insulation (□), and heavy insulation (◊). (Ambient temperature, 24°C; relative humidity, 50%; n=6).

Following the cessation of exercise there was a significant fall in Tes in the first few minutes. This was not paralleled by a similar change in Tre which remained stable or began a slow rate of fall. Non-acral surface measurements returned gradually to PreEx within the first 10-15 min with SH showing the quickest fall to temperatures that were even lower than PreEx values. Acral temperature (Tfi) remained elevated despite the changes observed at the Tfh and Tch non-acral sites. The changes in Tsk were qualitatively similar for all insulation conditions, except that OV and SH showed a slower but complete return to PreEx by the end of the recovery period (table 2). Both HR and VO_2 showed the same pattern of abrupt and then more gradual increase until about 5-6 min of exercise, after which VO_2 became relatively stable while HR continued to increase gradually until exercise termination. Recovery of VO_2 was rapid, reaching essentially PreEx values in 10-12 min for all conditions. HR remained slightly elevated above PreEx values with an increased magnitude across insulative conditions (table 3).

Table 2. Mean surface temperatures for different insulation type.

	SH			OV			CW		
	Tfh (°C)	**Tch** (°C)	**Tfi** (°C)	**Tfh** (°C)	**Tch** (°C)	**Tfi** (°C)	**Tfh** (°C)	**Tch** (°C)	**Tfi** (°C)
PreEx 10 min	33.43	32.74	23.94	33.67	33.16	27.46	33.84	32.99	27.10
SE	.35	.30	1.10	.48	.38	1.26	.26	.43	1.58
Start Ex	33.43	32.76	23.90	33.52	33.31	25.00	34.64	33.49	25.50
SE	.35	.30	1.04	.46	.40	1.12	.27	.27	1.40
End Ex	34.18	34.16‡	31.09‡	35.21‡	35.07‡	31.51‡	35.87‡	35.27‡	31.81‡
SE	.32	.30	.59	.53	.32	.95	.35	.41	1.22
PostEx 10 min	32.82†	33.42†	32.73‡	34.03	34.74	33.31‡	35.68	35.81‡	33.98‡
SE	.40	.43	.40	.56	.28	.38	.21	.50	.30
PostEx 20 min	32.30†	32.52†	32.95‡	33.43	33.62	33.40‡	35.28	35.25‡	34.10‡
SE	.73	.50	.39	.62	.40	.37	.13	.52	.39
PostEx 30 min	32.26†	31.91†	32.89‡	32.90†	32.83	33.44‡	34.89	34.63‡	33.94‡
SE	.65	.57	.36	.53	.46	.36	.26	.53	.28

(† - indicates significant difference from CW; ‡ -indicates significant difference from PreEx, $p>0.05$)

Table 3. Mean oxygen consumption and heart rate values for different insulation type.

	SH		OV		CW	
	VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	**HR** ($b \cdot min^{-1}$)	**VO_2** ($ml \cdot kg^{-1} \cdot min^{-1}$)	**HR** ($b \cdot min^{-1}$)	**VO_2** ($ml \cdot kg^{-1} \cdot min^{-1}$)	**HR** ($b \cdot min^{-1}$)
PreEx 10'	3.19	70	4.21	95	4.96	104
End Ex	37.05‡	171‡	37.45‡	179‡	38.29‡	186‡
PostEx 5'	5.52	103‡	5.89	114	5.90	117
10'	3.92	96	4.24	107	4.84	110
20'	na	95	na	104	na	107
30'	na	93	na	104	na	104

(na = not available, data was collected until 10 min into recovery only; ‡ -indicates significant difference from PreEx, $p>0.05$)

3.1.4 Discussion

Dynamic exercise is routinely used to distinguish different clothing insulation qualities (Goldman 1974; Epstein et al. 1990; Nielsen and Endrusick 1990). The effect of the different insulation's is to reduce the potential for heat loss and therefore cause Tco, usually represented by Tre, to rise more rapidly. In this experiment Tes showed a significant difference in the rate of rise of Tes following the inflection point while no significant differences were demonstrated in Tre between conditions. This observation demonstrates that the proper location of Tco measurement is very important when monitoring changes associated with dynamic responses to heat gain. It is well documented that Tes is a sensitive site for transient changes in heat gain or loss (Brengelmann 1987) and therefore is a better indicator of thermal stress during short dynamic exercise (Molnar and Read 1974; Shiraki et al. 1986). Tre does not respond in a similar way primarily due to the fact that it does not receive blood directly from areas of high metabolic rate or heat dissipation and because the measurement site has a high tissue density which contributes to a high thermal inertia. This therefore reduces the effectiveness of Tre as a measure to differentiate acute transient changes in heat gain or loss (Saltin and Hermansen 1966; Aulick et al. 1981; Sawka and Wenger 1988).

The very different response in Tes with different insulation types shows that the rate of heat gain was 2.4°C·hr^{-1} greater during the full insulation (CW) conditions than very light insulation (SH). Despite even the heavy insulation, there was a 61% drop in the rate of rise of Tes following the time when Tsk began to increase, thus demonstrating the remarkable capacity for heat loss even under the severely restricted conditions experienced while wearing heavy clothing. Yet despite the measurable reduction in heat gain during exercise, the PostEx Tes for all conditions was significantly elevated, suggesting a reduced capacity for heat loss during the 30 min recovery period.

3.1.5 Conclusion

Investigation of different insulation types during exercise at 24°C, 50% humidity provided a qualitative understanding of the chronology of changes in Tsk and Tes during and following muscular work. In addition the data demonstrated a general consistency of Tsk and Tco in their response to very different insulation types.

The abrupt rise in Tsk was found to be chronologically related to Tes inflection and thus indicated the influence of Tsk on thermal reflex response (Nadel et al. 1979; Johnson and Park 1982; Sawka and Wenger 1988; Kellogg et al. 1991b; Kruk et al. 1991; Johnson 1992). The elevated PostEx Tes in the non-insulated conditions was an interesting observation in light of the potential for heat loss demonstrated during exercise, the relatively cool ambient temperature and available surface area for evaporative heat loss. Although these data did not provide direct evidence, they served to develop the primary hypothesis of this thesis that the PostEx Tes elevation was influenced by an exercise- induced change in the threshold of the thermoregulatory reflexes required to deal with PostEx hyperthermia.

3.2.0 Phase 2 - Conditions of sensible and insensible and ambient temperature

3.2.1 Introduction

The principal objective of this investigation was to investigate blood pressure changes during different conditions of ambient temperature and insulation type. However, the design of the protocol was such that further investigation of the chronology of the time-related response of Tsk and Tco under two extreme conditions of ambient temperature was possible. The conditions were: low (20°C) and high (40°C) ambient

temperatures, while wearing no insulation (SH - shorts only) and full insulation (CW - chemical warfare protective garment) during the performance of a low intensity work load.

3.2.2 Specific methods

Five male volunteer subjects participated in the experiment. Their average age, height and weight were 21±1 yr, 176±2 cm and 79±3 Kg respectively. Internal temperatures were monitored by Tes and Tre, and Tsk measurements (including Tfh, Tch, and Tfi). The subjects were required to complete four trials which were randomly assigned. The conditions were:

1) 20°C, 50% humidity with no insulation [shorts only (SH)],
2) 20°C, 50% humidity with full insulation [chemical warfare protective garment (CW)],
3) 40°C, 50% humidity with no insulation [shorts only (SH)]; and
4) 40°C, 50% humidity with full insulation [chemical warfare protective garment (CW)].

The subjects entered the thermal chamber for a 2 hr acclimation period wearing only shorts and running shoes. At 10 min PreEx, the subject stood and was prepared for the exercise period and if required the proper insulation was fitted. Treadmill running exercise was chosen to stimulate about 40% of the subject's VO_{2max} (6.5 METS), an intensity which was found to be a comfortable walk that could easily be continued for 18 min without producing significant physical stress or fatigue. Recovery was limited to 20 min in order to minimize the thermal stress associated with wearing the heavy insulation at 40°C and because previous experiments demonstrated that Tes achieved a stable elevation within 5 minutes of recovery.

3.2.3 Results

At the onset of exercise, Tes showed a slight fall in the order of 0.1-0.2°C for the SH conditions (fig. 3). There was a delay in the beginning of rise in Tes at Tam 40°C for each condition of clothing. This was immediately followed by an inflection of Tes at a rate of elevation equal to $0.08°C \cdot min^{-1}$ and $0.06°C \cdot min^{-1}$ for the Tam 20°C and 40°C for SH conditions, respectively. In contrast, the rates for the CW conditions were $0.09°C \cdot min^{-1}$ and $0.06°C \cdot min^{-1}$ for Tam 20 and 40°C. During this period Tre rose at a lower rate than Tes but showed a trend to maintaining the same rate for the full period of exercise.

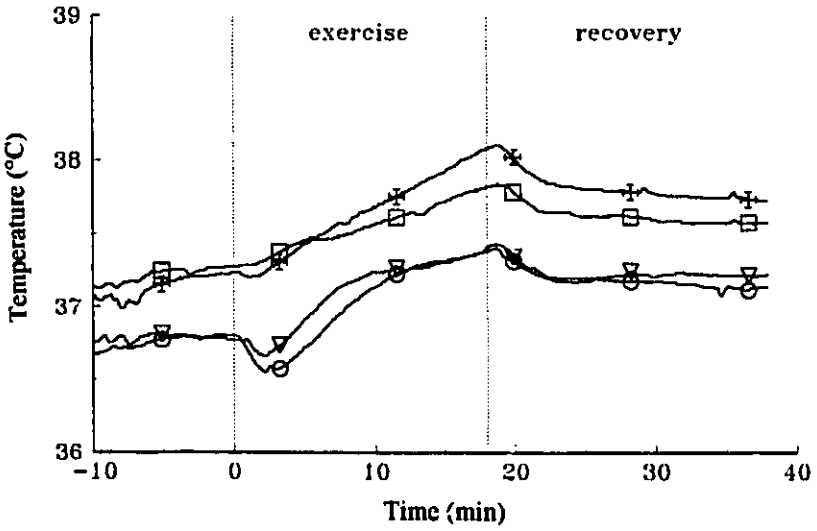

Fig. 3. Mean esophageal temperatures from rest through the start and end of exercise at 6-7 METS and during standing, leaning recovery for different insulation types and ambient temperatures. Esophageal temperature for no insulation at 20°C (O), no insulation at 40°C (□), heavy insulation at 20°C (▽) and heavy insulation at 40°C (✱) (n=5).

Surface temperatures showed a fall at the onset of exercise at Tam 20°C with no such change being observed at Tam 40°C for either condition of clothing. However, the inflection in surface temperatures preceded the inflection in Tes for all conditions. For the

SH conditions the inflection of Tes resulted in a reduction of the rate of Tes rise to 0.03°C·min^{-1} and 0.03°C·min^{-1} for Tam 20 and 40°C. The CW Tes inflection resulted in a reduction of the rate of Tes rise to 0.02°C·min^{-1} and 0.05°C·min^{-1} for Tam 20 and 40°C. Both Tco and Tsk continued to rise until cessation of exercise. All Tsk temperatures with the exception of the acral Tfi showed a falling trend to PreEx within minutes of end of exercise. The Tsk for Tam 20°C of CW and SH showed the largest temperature increase during exercise and the largest drop following cessation of exercise. The Tsk at Tam 40°C demonstrated the smallest absolute change and either increased slightly or remained fairly stable during exercise.

Following cessation of exercise during all conditions Tes fell precipitously reaching a plateau within the first 5 minutes of recovery (table 4). Although the plateaus for the Tam 40°C were significantly higher than those for Tam 20°C, there were no significant differences between clothing conditions. Once achieved, the elevated Tes remained stable for the full recovery. In contrast to Tes, Tre did not show a precipitous fall but remained elevated for the full period of recovery (fig. 3). Tsk showed a gradual PostEx drop to values lower than PreEx values (table 5).

Table 4. Mean core temperatures for different insulation type and ambient temperature (Tam 20 and 40°C, 6.5 METS)

	SH Tam 20 °C		SH Tam 40 °C		CW Tam 20 °C		CW Tam 40 °C	
	Tes (°C)	Tre (°C)	Tes (°C)	Tre (°C)	Tes (°C)	Tre (°C)	Tes (°C)	Tre (°C)
PreEx 10 min	36.80	37.10	37.27	37.35	36.79	37.12	37.23	37.45
SE	.07	.08	.06	.07	.06	.08	.07	.08
Start Ex	36.81	37.07	37.30	37.49	36.79	37.07	37.27	37.48
SE	.07	.08	.07	.08	.07	.09	.07	.09
End Ex	37.38†‡	37.49‡	37.81‡	37.96‡	37.31†‡	37.53‡	38.08‡	38.10‡
SE	.11	.13	.11	.14	.12	.13	.10	.11
PostEx 10 min	37.17	37.53†‡	37.64	38.04‡	37.19	37.51†‡	37.84‡	38.23‡
SE	.11	.13	.10	.13	.12	.14	.11	.12
PostEx 20 min	37.13	37.50†	37.62	38.03‡	37.18	37.51†‡	37.79‡	38.27‡
SE	.11	.12	.11	.12	.12	.14	.11	.13

(† - indicates significant difference from CW 40°C; ‡ -indicates significant difference from PreEx, $p>0.05$)

Table 5. Mean surface temperatures for different insulation type and ambient temperature (Tam 20 and 40°C, 6.5 METS)

	SH Tam 20 °C			SH Tam 40 °C			CW Tam 20 °C			CW Tam 40 °C		
	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfh (°C)	Tch (°C)	Tfi (°C)
PreEx 10 min	30.71†	32.82†	21.89	35.59	36.74	35.98	32.91†	33.29†	25.08	35.88	36.55	36.21
SE	.37	.29	1.30	.26	.22	.76	.32	.27	1.00	.22	.21	.71
Start Ex	30.60	32.74	21.72	35.55	36.81	36.02	32.80	33.20	25.09	35.88	36.67	36.10
SE	.39	.28	1.29	.25	.22	.89	.33	.29	.91	.21	.19	.74
End Ex	31.86†	33.91†	26.93‡	35.91	36.82	36.14	33.84	35.58	27.68‡	36.05	36.82	36.36
SE	.34	.38	.69	.30	.27	.45	.34	.30	.89	.23	.19	.81
PostEx 10 min	30.76	33.20†	29.22‡	35.71	36.44	36.03	33.24	35.56	30.41‡	35.91	36.91	36.21
SE	.42	.51	.59	.29	.28	.43	.37	.38	.76	.18	.29	.61
PostEx 20 min	30.35	33.30†	29.52‡	35.71	36.26	36.06	32.57	35.06†	30.94‡	35.85	36.74	36.14
SE	.49	.60	.45	.24	.25	.43	.36	.43	.82	.23	.30	.62

(† - indicates significant difference from CW 40°C; ‡ -indicates significant difference from PreEx, $p>0.05$)

3.2.4 Discussion

The results show that for this work intensity the activation of thermal mechanisms significantly reduced the rate of heat gain for both SH and CW conditions with the reduction following Tes inflection equal to 0.03°C·min^{-1} for both conditions at 20°C. These results are interesting when it is considered that full insulation eliminated air exchange and that only a very small fraction of the total surface area was exposed for exchange with the ambient air. Thus it is evident by these data that the system had the ability to adjust or maintain the rate of heat gain without an additional elevation from normal. However, this ability was challenged when the same work load was performed at Tam 40°C.

PostEx Tes did not fall significantly during any condition. Of interest was the observation that the heaviest insulation (CW) did not show any difference in the recovery Tes compared to the lightest insulation (SH) at Tam 20°C. Since ambient temperature was below the "comfort" zone of temperature and provided a gradient for heat loss, it would be expected that the Tam 20°C SH would show a very different response from CW. Tre in recovery for all conditions was stable and elevated in recovery. Rectal temperature for both SH and CW at Tam 20°C were similar. It was expected that the low Tam 20°C and the reduced exercise intensity would provide the appropriate conditions for the thermoregulatory system to compensate and rapidly return Tco to PreEx resting temperature. Yet these conditions did not produce the expected reduction in elevation of Tes relative to PreEx values.

During exercise the change in the rate of heat loss was the result of hypothalamic-mediated effector responses i.e. sudomotor activity and vascular conductance changes (Sawka and Wenger 1988). This is evidence to demonstrate that in the face of a sharply increasing Tco, the hypothalamus is able to respond most effectively by recruiting the appropriate thermolytic reflex to reduce and almost eliminate any further rise in Tes. As

figure 3 indicates, a significant attenuation of the rate of Tes increase during exercise was observed even under heavy insulation conditions at Tam 20°C (i.e. chemical warfare garments which severely impair any surface exposure to the environment).

The exercise response at 20°C clearly demonstrated that in the face of a large increase in Tco the regulatory system had the capacity to achieve a new thermal balance despite considerable restrictions placed on it. In light of this potential, it would be expected that with a reduced metabolic rate where the competing influences of both thermal and non-thermal reflexes were eliminated, hypothalamic regulation should have been able to deal more effectively with an elevated Tco. There was evidence of an active regulatory response occurring during the exercise period which was also demonstrated for the recovery period. Even though Tsk fell to PreEx (table 5), Tes remained elevated and stable. Thus, whatever factor or influence was responsible for maintaining the aggressive thermolytic response during exercise, it seemed to be lacking in recovery.

A comparison of Tes between Tam 20°C (SH) and Tam 40°C (SH) showed that the PreEx temperatures were significantly different ($p>0.05$) demonstrating that the latter was significantly more stressful at rest. Yet during exercise the rise in Tes prior to inflection was 0.06°C·min^{-1} (3.6°C·hr^{-1}) for Tam 40°C, which was less than the 0.08° C·min^{-1} (4.8°C·hr^{-1}) for Tam 20°C. The inflection of Tes also occurred 4.5 min sooner at Tam 40°C. However, both showed a similar rate of heat gain following the inflection equal to 0.03°C·min^{-1} (1.8°C·hr^{-1}). The different responses between Tam conditions may be explained by significantly different Tsk and Tco (Benzinger et al. 1961, Nadel et al. 1971, Wenger et al. 1975). SP_{hy} regulation of Tco is controlled by a weighted input signal of Tsk and Tco. An elevated Tsk lowers the threshold temperature of thermolytic regulation for a given Tco response (Benzinger et al. 1961, Nadel et al. 1971; Wenger et al. 1975). Despite the obvious stress imposed by the very hot ambient conditions, PostEx Tes difference from PreEx for Tam 20°C and Tam 40°C respectively was similar (0.5°C).

3.2.5 Conclusion

A PostEx Tes elevated plateau was demonstrated during two very different ambient temperatures. The fact that Tes remained elevated following dynamic exercise in a cool environment at a similar magnitude of difference from PreEx as was produced by a hot environment, suggests that ambient temperature conditions were not the cause of the PostEx disturbance in thermal homeostasis. A comparison of PostEx Tes response following exercise at an intensity of approximately 12 METS (3.1.0) with the exercise performed in this study at a much lower intensity, 6.5 METS, demonstrated that a PostEx Tes elevation occurred regardless of the exercise intensity. However, any conclusion on the effects of exercise intensity on the magnitude of the PostEx Tes can not be made based on these data. The experiments were performed on a separate subject population and during different seasonal periods. Therefore, we can only conclude that the existence of a PostEx Tes elevation is not principally related to ambient temperature or exercise intensity but rather is due to the metabolic heat load generated during exercise.

Chapter 4

4.0.0 Disturbance of thermal homeostasis during post-exercise hyperthermia

4.0.1 Introduction

Exercise and hyperthermia represent significant and conflicting challenges to circulatory function. When experienced together there is a compromise which includes a non-thermally induced vasoconstriction in inactive tissue in order to maintain or increase arterial blood pressure in the face of massive vasodilation in active muscle; and a simultaneous attenuation of active vasodilation with the effects of relaxing homeothermic defence and allowing modest elevation of Tco (Johnson 1992; Kenney and Johnson 1992). The parity and divergence of interactions between thermal and non-thermal homeostatic reflexes was evident in our data of Tsk and Tco.

Johnson and Park (1982) and Kellogg et al. (1991c) demonstrated that the relationship of SBF and core temperature was significantly different under conditions of rest and exercise. Their results showed that, although rising Tco was accompanied by significant cutaneous vasodilation in both cases, the elevation in SBF was delayed until a higher Tco was reached during exercise. The effect of this upward shift in the Tco threshold for vasodilation was that SBF is lower at any given Tco during exercise than at rest. Therefore, it is possible that the PostEx plateau of Tes following an exercise-induced elevation in Tco may be explained by a change in the thermolytic reflexive controls brought about by an exercise related effect of thermal and non-thermal factors .

The following experiment was conducted to test the hypothesis that the homeothermic defence mechanism which incorporates reflexive control of skin vasculature

becomes inoperative during recovery at a temperature above resting values, with the result that Tco becomes stable for extended periods at an elevated level.

4.0.2 Specific methods

Five male volunteer subjects participated in the experiment. Their average age, height and weight were 23.8±2.0 yr, 183.6±5.78 cm and 78.5±2.7 Kg respectively. Internal temperatures was monitored by Tes and Tre. Skin temperature measurements included Tfh, Tch, Tfa, Tth and Tfi.

The subjects entered the thermal chamber for a 2 hr acclimation period wearing only shorts and running shoes at 29°C 50% humidity. At 10 min PreEx, the subject stood and was prepared for an 18 min exercise period. Treadmill running exercise was chosen to stimulate about 75% of the subjects VO_{2max} (12 METS). Recovery data was collected for a 65 min period.

The hypothesis was investigated by monitoring the chronology of changes in the Tfa - Tfi gradients as a qualitative indication of changes in skin blood flow (SBF) and in Tsk, Tes and Tre prior to, during and following exercise. The early use of skin thermometry as a simple, qualitative tool for identifying reflex control of SBF is well documented (Cooper et al. 1949; Felder et al. 1954; Greenfield 1963), and has been verified by quantitative measurements (Hirata et al. 1983; Johnson 1992). Skin temperatures have been recently validated as a measure of centrally mediated peripheral vasoconstriction when Tfi is used in conjunction with Tfa to calculate surface temperature gradients under steady state resting conditions (Rubenstein and Sessler 1990). They established a correlation of $r=0.98$ between changes in skin surface temperature gradients from the forearm to middle finger and changes in finger blood flow measured by venous occlusion plethysmography and concluded that such gradients are accurate measures of

thermoregulatory activity. They further stated that there is no lag time in temperature changes with an increase in vascular conductance induced by vasodilation of surface vasculature and that the gradient is therefore decreased almost immediately. This method has not been utilized during exercise, however, pilot data demonstrates that the application of the Tfa-Tfi gradient when compared to Laser-Doppler Flowmetry can be used to identify changes in vascular conductance (5.0.0). At the onset of exercise, there is a large increase in Tsk gradient as a result of the exercise induced vasoconstriction which results in a more precipitous fall of Tfi as compared to Tfa. As Tco increases, a threshold temperature is reached which initiates a general vasodilation of surface vasculature that is demonstrated as a rapid narrowing of the Tsk gradient. Therefore, an abrupt fall in the Tsk gradient (Tfa - Tfi) was used to identify the vasodilation temperature (Tdil) as the simultaneously occurring Tes.

4.0.3 Results

Figure 4 shows the time-course of VO_2, Tes, Tre and Tsk and HR during PreEx resting, exercising and PostEx recovery periods. Both HR and VO_2 showed the same pattern of abrupt and then more gradual increase until about 6-7 min of exercise, after which VO_2 became relatively stable while HR continued to increase gradually until exercise termination. Recovery of VO_2 was rapid, reaching essentially PreEx values in 10-15 min. Initial HR recovery to about 100 BPM or less than 50% of the exercise adaptation occurred in 5-6 min and the remaining recovery was much slower.

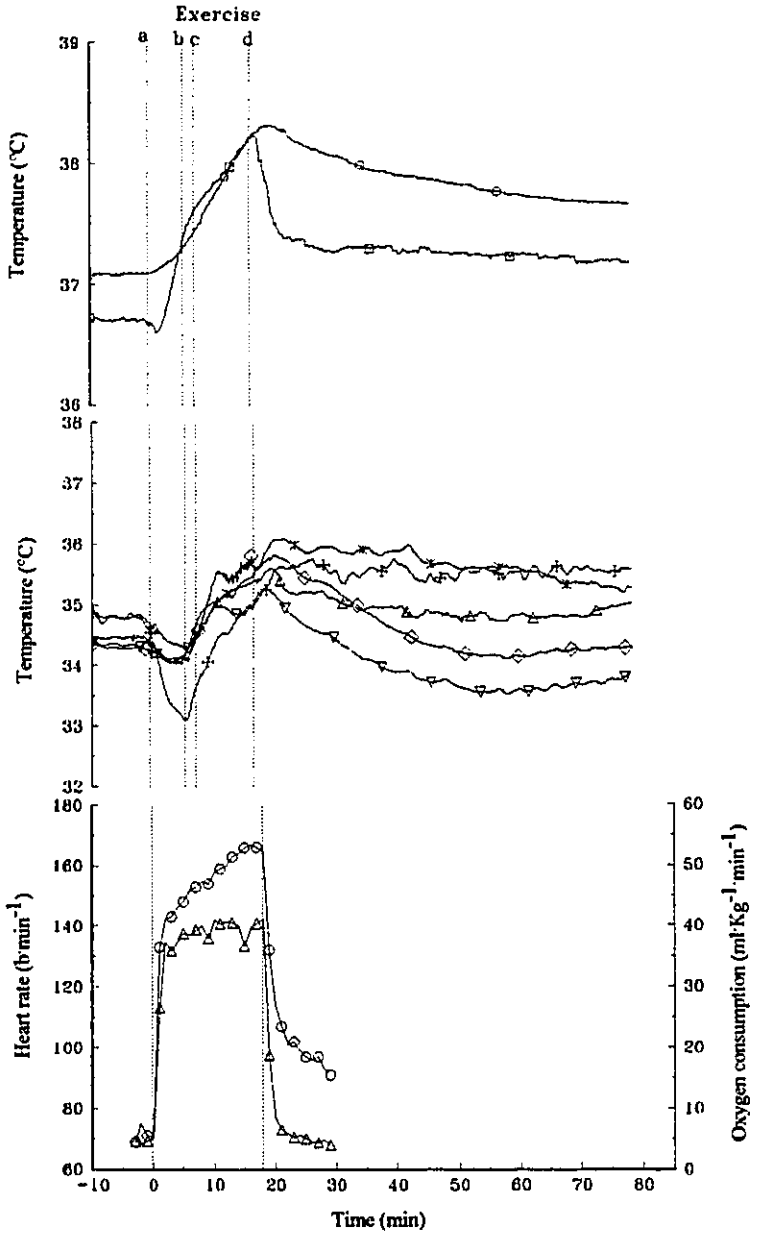

Fig. 4. Mean temperatures at sites from rest through the start at time a and end at d of exercise at 10-13 METS and during standing, leaning recovery. Top panel; esophageal (□,Tes) and rectal (O, Tre). Middle panel; temperatures at surface (head, ∇; chest, ◊; finger, +; forearm, Δ; thigh, ✱). Bottom panel; mean oxygen uptake (Δ), heart rate (O). Skin vasodilation occurred at b and was followed by a reduced rate of increase of Tes beginning at c. Tes reduction during recovery had become essentially plateaued at 0.64°C above pre-exercise values at c after 8-10 min of recovery. (Ambient temperature, 29°C; relative humidity, 50%; n=5).

Tsk fell at the start of the exercise but began to rise abruptly 6 -7 min after beginning activity. At the end of exercise, Tsk had increased by 0.7-1.7°C and continued to rise for at least 2-3 min after cessation of exercise before beginning to recover. By 20-30 min PostEx, Tfh, Tch and Tfa returned to a stable temperature at or just above PreEx values while Tfi remained stable at its immediate PostEx value for the entire recovery period (table 6). A continuous but slowly falling Tth was still above 50% of the exercise-induced elevation by the end of the recorded period. Tre rose continuously throughout the exercise, peaked at about 5 min PostEx and then fell gradually at a continuous rate but was still at 50% of the exercise-induced elevation by 65 min PostEx. Tes fell for the first 2-3 min of exercise and then rose rapidly until there was a sudden reduction in the rate of rise from 0.17 to 0.07°C·min^{-1}, which represents a 56% fall in the rate of heat gain. This change in slope occurred about 2 min after the observation of Tdil. The increase of Tes continued until about 1-2 min PostEx and was followed by a rapid fall at a rate that declined from 0.23°C·min^{-1} in the first minute to 0.17°C·min^{-1} in the 5th min before reaching a plateau (essentially zero rate of change) after 10 min of recovery. This plateau at 37.3°C was essentially maintained for the remaining 55 min of the recovery period and was significantly elevated (0.6°C) from the PreEx value of 36.7°C but not significantly different from Tdil 37.4°C (table 7).

Table 6. Mean skin temperatures at thermoneutral conditions.

	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfa (°C)	Tth (°C)
PreEx 10 min	34.20	34.26	34.20	34.26	34.32
SE	0.15	0.26	0.34	0.24	0.08
Start Ex	34.18	34.33	34.18	34.22	34.35
SE	0.16	0.25	0.38	0.25	0.07
End Ex	35.02‡	35.63‡	35.02	35.63‡	35.48‡
SE	0.22	0.35	0.31	0.25	0.11
PostEx 10 min	34.73	35.76‡	34.73	35.40‡	35.89‡
SE	0.30	0.26	0.26	0.35	0.28
20 min	34.34	34.84	34.09	34.84	35.93‡
SE	0.29	0.31	0.24	0.38	0.43
30 min	33.72	34.33	33.72	34.33	35.67‡
SE	0.27	0.38	0.12	0.35	0.48
40 min	33.54	34.15	33.54	34.15	35.60‡
SE	0.31	0.39	0.10	0.34	0.45
50 min	33.56	34.19	33.56	34.19	35.47‡
SE	0.38	0.36	0.16	0.27	0.42
60 min	33.67	34.24	33.67	34.24	35.26
SE	0.27	0.36	0.15	0.16	0.32
65 min	33.89	34.28	33.89	34.28	35.25
SE	0.22	0.37	0.23	0.16	0.23

(‡-indicates significant difference from PreEx, $p>0.05$)

Table 7. Mean core temperatures at thermoneutral conditions.

	Tes (°C)	Tre (°C)
PreEx 10 min	36.67	37.09
SE	0.15	0.25
Start Ex	36.68	37.08
SE	0.16	0.21
End Ex	38.22‡	38.23‡
SE	0.24	0.15
PostEx 10 min	37.31‡	38.13‡
SE	0.24	0.20
20 min	37.30‡	37.95‡
SE	0.19	0.19
30 min	37.29‡	37.86‡
SE	0.17	0.18
40 min	37.23‡	37.77‡
SE	0.16	0.17
50 min	37.21‡	37.70‡
SE	0.16	0.17
60 min	37.21‡	37.66‡
SE	0.15	0.18
65 min	37.19‡	37.64‡
SE	0.15	0.18

(‡-indicates significant difference from PreEx, $p>0.05$)

4.0.4 Discussion

The results of this experiment confirmed our previous observation of a PostEx elevation of Tes which was still present after 65 min (table 6). All non-acral temperatures except Tth fell to a plateau just above PreEx about 20-30 min after Tes established its plateau at 0.6°C elevation above PreEx values (table 7). Tre showed a gradual fall [$0.012°C \cdot min^{-1}$ ($0.72°C \cdot hr^{-1}$)] during the initial 30 min of recovery with a reduction in the rate of fall [$0.004°C \cdot min^{-1}$ ($0.24°C \cdot hr^{-1}$)] in the last 10 min of recovery but remained significantly elevated over the full recovery period. The persistent elevation of Tth was a

reasonable observation, being in a location over the quadriceps which had been used in running. This conclusion is supported by the demonstration by Webb et al. (1992) of the parallels and gradients which exist between Tco and Tsk. Thus, the high Tth suggests that a good deal of the metabolic heat remained in leg musculature. Whatever heat reached the central blood volume must have been dissipated rapidly at the surface to result in the high rate of decline of Tes and at most skin sites during early recovery.

The Tes at which the exercise reflexive control of skin vasculature (i.e. active vasodilation) occurred was equal to 37.4°C and was very similar to the PostEx Tes 37.3°C. The esophageal temperature at the time of surface vasculature dilation (Tdil) was used to represent the threshold of homeothermic defence mechanism (i.e. vasodilation) in response to increased Tco. Threshold control of vasodilation is significantly different from rest and exercise (Johnson and Park 1982; Kellogg et al. 1991c) and influenced by increasing hyperthermia (Johnson and Park 1982). Although Tdil was not measured in recovery, the sustained elevation of Tes suggests that homeothermic defence mechanisms may not be the same as in PreEx. This conclusion is supported by the fall in Tsk, absence of any visible sweating response, and a return of VO_2 to PreEx values within the first 10-20 min of recovery despite a significantly elevated Tco maintained for 65 min.

It was observed in preliminary experiments that the abrupt rise in Tsk was found to be chronologically related to Tes inflection. Measurements of Tfa-Tfi gradients reinforced this observation demonstrating a delayed response between an increase in SBF and Tes response. In addition, this study demonstrated that the Tes equivalent for active vasodilation (Tdil) was similar to the PostEx Tes. Table 8 presents Tdil and PostEx Tes from preliminary experiments (3.1.0 and 3.2.0). A similar relationship was demonstrated between Tdil and the PostEx Tes for each of the conditions.

Table 8 - Mean Tdil and PostEx Tes values.

	Phase 1	**Phase 2**	**Thermoneutral**
Dilation threshold (Tdil)	37.3°C	37.1°C	37.4°C
PostEx recovery Tes	37.2°C	37.1°C	37.3°C

Although phase 1 (Tam 24°C,12 METS, 75% VO_{2max}) represented a more stressful condition of ambient temperature and exercise intensity than phase 2 (Tam 20°C, 6.5 METS, 40% VO_{2max}) the Tdil to PostEx Tes relationship was not changed. The data suggest that exercise intensity and ambient temperature did not have a measurable effect on this relationship. The consistency of the relationship would suggest that the thermal reflex of vasodilation became inoperative once Tes had recovered to Tdil. Mekjavic et al. (1991) identified separate thresholds for sweating and shivering and referred to the range of temperature between the thresholds as the "null zone". Tdil might be considered to represent the upper limit of a "normal" range of temperature control. Thus, the observation in this study of a persistent elevation of PostEx Tes at the same value as the exercise Tdil in the face of a declining non-acral Tsk suggestive of falling SBF, and a visibly disappearing sweat response may well indicate that Tco had fallen below the upper limit of active Tco regulation. This interpretation is supported by the lack of any visible thermoregulatory response as represented by a return of Tes and Tre to PreEx values despite the available potential for heat loss and the unused but easily accessed SBF available with which to do so. However, no explanation can be offered for these data that Tdil during exercise was at the same value as indicated by the plateau Tes during recovery at a time when it was apparent that vasodilation was disappearing.

It is equally possible that some exercise related response which has thermal effects, such as metabolic rate (Kaciuba-Uscilko et al. 1992), plasma osmolarity (Horowitz 1990), central modulators (Zeisberger 1990) or surface temperatures (Bothorel et al. 1991) may

have altered SP_{hy}. To do so it must be shown that heat dissipation responses are inactive at the new SP_{hy}. These data show that the reduction of Tes toward PreEx values was almost curtailed before complete recovery and thus provide indirect evidence of inactive thermal reflexes. However, even though VO_2 had been reduced to resting levels by 10-15 min, the residual heat in the exercised muscle [i.e. elevated Tth and muscle temperature (Appendix C)] and pelvic tissue (i.e. elevated Tre) represented a metabolic heat source and prevents any conclusion that a fever-like elevation of Tco had occurred.

4.0.5 Conclusion

It was quite clear from these data that PostEx Tes reached and maintained a plateau at elevated levels for up to 65 min. This plateau was equal in magnitude to Tdil during exercise, suggesting that thermal reflexive vasodilation was significantly reduced before PreEx resting Tco could be reestablished. The passive attitude of thermal control systems towards elevated Tco during recovery considerably strengthens the idea of a range of normal temperatures, within which thermoregulatory mechanisms are relatively insensitive to Tco fluctuations

Chapter 5

5.0.0 Physiological interpretation and technique for thermal threshold identification during endogenous heating.

5.0.1 Introduction

The change in the level at which Tes was maintained during recovery following an exercise-induced rise in Tco suggests that the effects of exercise may have persisted in the form of changes in one or more of the thresholds at which the regulatory reflexes respond. As previously stated, our findings of a relationship between active vasodilation during exercise and the PostEx Tes temperature provides a rationale for understanding and describing the changes in thermal homeostasis following dynamic exercise. Tsk measurements are commonly used to assess changes in thermal conductance at the skin surface (Kellogg et al. 1991a; Kenney and Johnson 1992; Briese and Cabanac 1992; Johnson 1992). Although the literature indicates that the changes in Tsk are qualitatively representative of changes in vascular conductance (Kellogg et al. 1991a; Kenney and Johnson 1992), there are limited data that describe the time-lag between vessel dilation and temperature change. Thus, conclusions based on changes of SBF which has been identified only by changes in Tsk are limited because the Tco at which active vasodilation occurs may be incorrectly measured.

The following series of experiments were designed to investigate the hypothesis that changes in Tsk during exercise are directly linked chronologically to changes in SBF. Tes was assumed to be representative of hypothalamic temperature changes. Tes at the level of the left atrium and greater vessels is at a point where all mixing of venous blood and any cooling effect of pulmonary circulation has occurred, and is therefore

representative of the combined effects of peripheral and central temperature changes on central blood temperature. In addition, there is little opportunity for arterialized blood leaving the heart to become cooled before reaching the hypothalamus. Therefore, blood temperature measured by Tes may be assumed to be representative of hypothalamic temperature (Cooper and Kenyon 1957; Shiraki et al. 1986) and is commonly used both as indicator of the temperature at which vascular changes occur and as an indicator of the effectiveness in dissipating body heat (Webb et al. 1978, Webb 1992).

This series of experiments was conducted to test the hypothesis that Tes at the time of skin vasodilation during exercise measured by Laser-Doppler flowmetry (LDF) is comparable to measurements by temperature gradients between forearm and finger (Tfa-Tfi gradient) using surface thermistors. The study had three objectives:

1) to establish the chronology of events in SBF and temperatures during PreEx, exercise and PostEx periods,
2) to compare measurements of Tdil using direct (LDF) and indirect (Tfa-Tfi gradient) methods, and
3) to investigate the relationship of Tdil and PostEx using LDF and Tfa-Tfi gradient methods.

5.0.2 Specific methods

Nine male volunteer subjects participated in the experiment. Their average age, height and weight were 23±1 yr, 177±2 cm and 76±2 Kg respectively. Measurements of internal temperature were monitored by Tes and Tre and Tsk measurements included Tfh, Tch, Tfi, Tfa, Tth, Tca and Tba. The subjects entered the thermal chamber for a 2 hr acclimation period at 29°C, 50% humidity wearing only shorts and running shoes. At 10 min PreEx, the subject stood, mounted the treadmill and inserted the two-way breathing

valve for VO_2 gas collection. Treadmill running exercise was chosen to stimulate about 75% of the subject's VO_{2max} for 15 min. Following cessation of exercise the subjects stood for 30 min until end of recovery.

The hypothesis was investigated by monitoring the chronology of changes in SBF and in Tfa-Tfi gradients, Tsk, Tes and Tre prior to, during and following exercise. Laser-Doppler blood flow (LDF) from the skin was measured on the mid-frontal forearm (blood perfusion monitor, TSI, St. Paul MN). LDF provided a linear index of SBF from approximately 1 mm^2 of skin area and was based on the frequency of shift of coherent laser light induced by moving erythrocytes within the cutaneous vessels (Taylor 1989; Johnson 1990; Kellogg et al. 1991a).

5.0.3 Results

PreEx Tco remained stable and consistent at 36.7°C for Tes and 37.0°C for Tre for the 10 min period prior to exercise (fig. 5). At the onset of exercise Tes fell 0.2°C in the first 2 min and was accompanied by a fall in Tsk of 0.3°C (Tfa) to 0.8°C (Tth). Acral sites (Tfi) showed the largest drop of 1.1°C at approximately 5.4 min into exercise (fig. 6). Tre did not show any indication of a fall for the first minute of exercise and thereafter rose slowly in contrast to Tes which demonstrated a sharp rise following the initial drop (fig. 5). The initial rate of rise of Tes was 0.17°C·min^{-1} (10.4°C·hr^{-1}). An increase in Tsk was followed 2.5 min later by a significant drop in the rate of rise of Tes. The effect of heat loss at the skin resulted in the subsequent 70% reduction in the rate of rise of Tes to 0.05°C·min^{-1} (3.2°C·hr^{-1}) and this was maintained for the remainder of the exercise period. Tre rose continuously during the exercise at a rate of 0.6°C·min^{-1} (3.7°C·hr^{-1}).

End exercise Tes was 37.9°C and was maintained for a period of 55 secs after which it fell precipitously at a peak rate of -0.22°C·min^{-1} (-13.3°C·hr^{-1}). In contrast, Tre

was maintained at 37.8°C for 25 min of recovery, and reached a final temperature of 37.7°C at the end of 30 min recovery (table 9). Tsk reached peak temperatures approximately 4.5 - 5.5 min PostEx which was nearly simultaneous with the time that Tes began to reach a plateau at 6.4 min PostEx. Tsk reached PreEx temperatures within 15-20 min of cessation of exercise (table 10). Tes remained elevated 37.2°C from 6.4 min into recovery until the end of the recovery period. PostEx measurements of VO_2 reached PreEx values 5-7 min following exercise and did not change significantly.

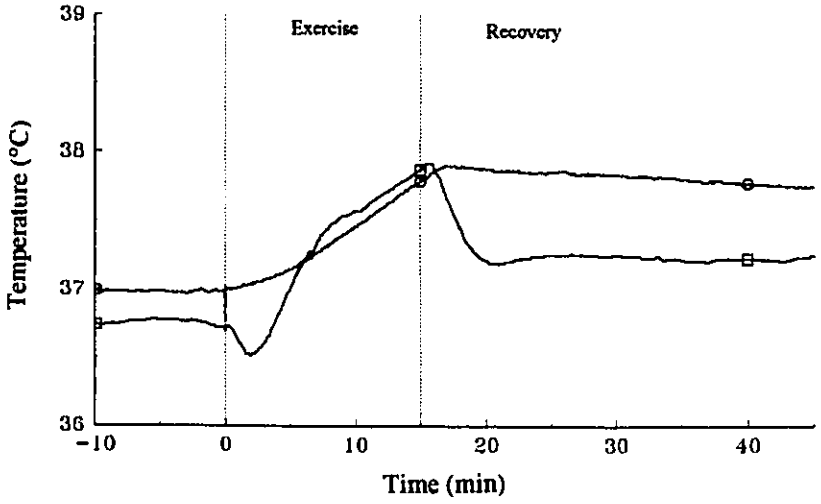

Fig. 5. Mean exercise core temperature response from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery. Rectal temperature (O) and esophageal temperature (□). (Ambient temperature, 29°C; relative humidity, 50%; n=9).

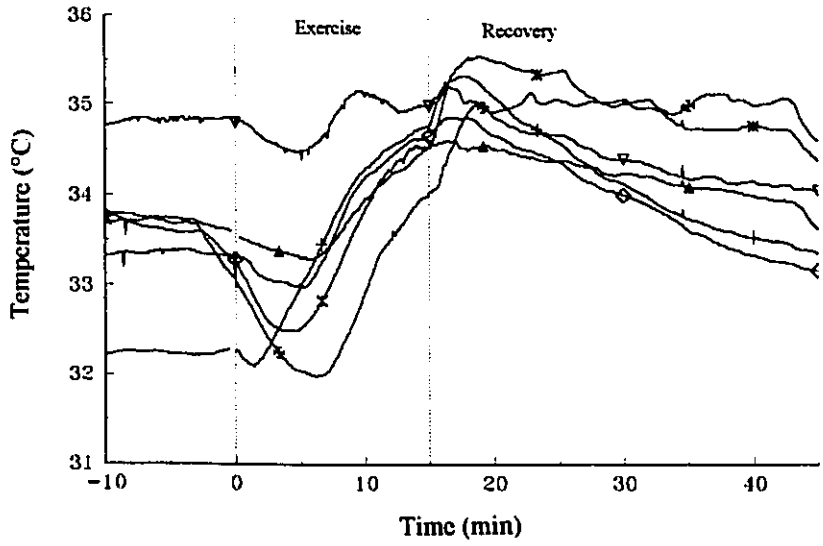

Fig. 6. Mean skin temperatures at sites from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery (head, ∇; chest, ◊; finger, ⊛; forearm, Δ; thigh, ✱; calf, +). (Ambient temperature, 29°C; relative humidity, 50%; n=9).

Table 9. Mean core temperatures.

	Tes (°C)	Tre (°C)
PreEx 10 min	36.74	36.98
SE	.05	.06
Start Ex	36.73	37.00
SE	.05	.06
End Ex	37.87‡	37.78‡
SE	.04	.07
PostEx 10 min	37.24‡	37.84‡
SE	.06	.06
20 min	37.21‡	37.79‡
SE	.06	.06
30 min	37.23‡	37.73‡
SE	.06	.06

(‡ -indicates significant difference from PreEx, $p>0.05$)

Table 10. Mean surface temperatures.

	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfo (°C)	Tth (°C)	Tca (°C)	Tba (°C)
PreEx 10 min	34.74	33.32	33.84	33.78	33.74	32.22	34.74
SE	.11	.22	.34	.19	.25	.15	.14
Start Ex	34.82	33.30	32.95	33.51	33.22	32.27	34.72
SE	.13	.23	.31	.19	.19	.25	.16
End Ex	34.99	34.65‡	34.02‡	34.54‡	34.52	34.77‡	36.42‡
SE	.17	.21	.38	.18	.18	.26	.22
PostEx 10 min	34.66	34.36‡	35.05‡	34.38‡	35.38‡	34.58‡	35.89‡
SE	.22	.31	.31	.19	.21	.20	.26
20 min	34.18	33.59	34.99‡	34.08	34.73	33.72‡	35.32
SE	.24	.38	.16	.26	.26	.23	.30
30 min	34.04	33.15	34.60‡	33.63	34.36	33.34‡	34.88
SE	.24	.37	.14	.19	.19	.21	.26

(‡ -indicates significant difference from PreEx, $p>0.05$)

SBF (table 11) at the onset of exercise increased immediately from a resting value of about 4.5 $ml \cdot kg^{-1} \cdot 100gm^{-1}$tissue to an intermediate plateau equal to approximately 14-15 $ml \cdot min^{-1} \cdot 100gm^{-1}$tissue. The plateau was maintained for 6 min followed by a second inflection upward which was consistent with literature descriptions of a Tco induced reflex active vasodilation of surface vasculature (Kellogg et al. 1991a; Johnson 1992). SBF increased from 14-15 $ml \cdot min^{-1} \cdot 100gm^{-1}$tissue to a final plateau equal to 23-24 $ml \cdot min^{-1} \cdot 100gm^{-1}$tissue. On cessation of exercise, SBF fell precipitously to 12 $ml \cdot min^{-1} \cdot 100gm^{-1}$ tissue within 30 secs and gradually reached PreEx values within 10 min of recovery (fig. 7).

Table 11. Mean forearm Laser-Doppler flow values.

	LDF ($ml \cdot 100gm^{-1} \cdot min^{-1}$)
PreEx 10 min	4.5
Start Ex	9.0
Plateau A	13.0-14.0
Plateau B	23.0-24.0
End Ex	24.6
PostEx 10 min	6.5
20 min	5.7
30 min	5.5

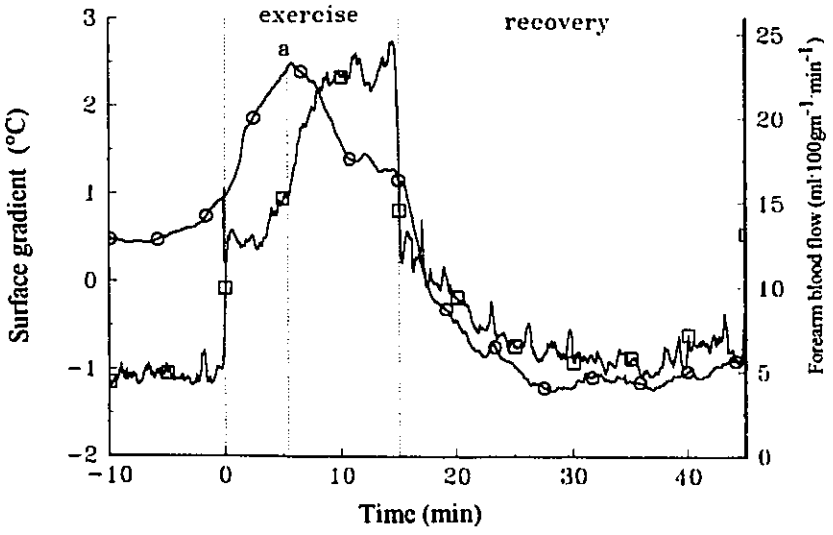

Fig. 7. Identification of mean skin vasodilation (Tdil) during exercise at time a measured by mean temperature gradient between the forearm and middle finger (Tfa-Tfi, O), as described by Rubenstein and Sessler (1990) and Laser-Doppler flowmetry (LDF, □) (Ambient temperature, 29°C; relative humidity , 50%; n=9).

Figure 7 demonstrates the chronology of LDF and Tfa-Tfi gradient methods in the determination of Tdil. Graphical representation shows that the Tfa-Tfi gradient rose quickly at the onset of exercise reaching a peak and holding for a few seconds after which this difference was quickly reduced. Based on the results obtained for the determination

of the Tes equivalent at the time of active vasodilation, Tsk inflection, LDF and Tfa-Tfi gradient methods gave essentially identical information. Table 12 lists the calculations of Tdil for each of the methods. The measurement of Tdil by Tsk method was 37.18°C, by Tfa-Tfi gradient 37.22°C and by LDF 37.16°C. The range of time difference was only 0.4 min (24 secs) with the Tfa-Tfi gradient method showing the longest delay relative to LDF. This time-delay was not long enough to produce a significant difference in Tes. As in previous experiments, the PostEx Tes temperature was similar to the measured value of Tdil equal to 37.22°C (LDF) and 37.16°C for PostEx Tes and Tdil.

Table 12. Tdil values determined by LDF (A), Tfa-Tfi gradient (B) and Tsk (C) methods.

	A	**B**	**C**		
	SBF	**Tfa-Tfi**	**Tsk**	**Diff b-a**	**Diff c-a**
Tdil (°C)	37.16	37.22	37.18	0.06	0.02
Time (min)	5.8	6.2	5.9	0.4	0.1

Data from this investigation were compared with previous experiments (table 13) performed at nearly the same exercise intensity under thermoneutral conditions. These data represented different populations of subjects and different periods during the calendar year but were statistically identical and therefore demonstrate that the responses were independent of population and chronological effects.

Table 13. Mean pre-exercise, exercise and recovery Tes values of experiments performed at thermoneutral conditions and 12 METS (Tdil measurement by Tfa-Tfi method).

	PreEx Tes (°C)	Ex Tdil (°C)	PostEx Tes (°C)	Time Tdil (°C)
Expt #1 (n=5)	36.89	37.25	37.33	5.8
Expt #2 (n=9)	36.68	37.17	37.16	6.3
Expt #3 (n=5)	36.67	37.39	37.30	6.3
Expt #4 (n=9)	36.74	37.22	37.24	6.2
Avg	**36.74**	**37.26**	**37.26**	**6.1**

The range of variation between Tdil and PreEx Tes was 0.5, 0.5, 0.4 and 0.7°C for expt #1 to expt #4 respectively. The time-period for the active vasodilation of forearm SBF provides another important observation. It was found that the time ranged from 5.8 - 6.3 secs with an average time of 6.1 secs. These data demonstrate that there was reasonable consistency in the measurements and that all techniques resulted in essentially equivalent measurements (table 14).

Table 14. Mean forearm skin blood flow , surface gradient and Tsk measurements of Tdil from experiments performed at thermoneutral conditions and 12 METS.

	SBF Tdil (°C)	Tfo-Tfi Tdil (°C)	Tsk Tdil (°C)
Expt #1 (n=6)	na	37.25	37.27
Expt #2 (n=9)	na	37.17	37.10
Expt #3 (n=5)	na	37.39	37.40
Expt #4 (n=9)	37.16	37.22	37.18
Avg		**37.26**	**37.24**

(note: na= not available. Skin blood flow by Laser-Doppler flowmetry was not calculated for these experiments)

5.0.4 Discussion

Table 13 presents the PreEx, Tdil and PostEx Tes values. The similarity of Tdil and [illegible]s value reinforces the previous conclusions that Tdil may represent the upper [illegible] a tor[illegible]erature range within which only passive temperature regulation is operating (i.e. no active dilation of surface vessels). This would imply that the exercise condition and the events associated with endogenous heat generation were the cause of the elevated PostEx Tes. If the generation of a significant heat load by exercise produced a persisting elevation of Tes it would be logical to conclude 1) that there had been a disturbance in thermal homeostasis, and 2) that a different heat load (i.e. exogenous) would have produced very similar effects. However, if the changes in the thermal integrity of the system were related to non-thermal reflexes associated with exercise such as volume/baroreceptor mediated effects (Johnson and Park 1981; Kenney and Johnson 1992; Johnson 1992), hormonal changes (Travis and Johnson 1993), disturbance of the state of hydration (Fortney and Vroman 1985) or some other central nervous system affect associated with neuromuscular recruitment (Horowitz 1988), it is plausible to suggest that homeostasis would not be affected in a similar way with exogenous heating.

5.0.5 Conclusion

The data presented in this investigation support the hypothesis that there was a relationship between Tdil and PostEx Tes. The consistent demonstration of this relationship under similar conditions for different experiments suggests that it has a physiological basis. The methods presented for measuring Tdil show that all techniques generate similar data. Thus, the relationship was used in the design of subsequent experiments to investigate changes in thermal homeostasis for different treatment conditions.

Chapter 6

6.0.0 Intermittent exercise

6.0.1 Introduction

The existence of an elevated Tes plateau, in the face of declining non-acral Tsk (suggesting falling SBF), and a visibly disappearing sweat response may well challenge SP_{hy} theory. The passive attitude of thermal control systems toward elevated Tco during recovery is consistent with the existence of a range of "normal" temperature regulation as opposed to a single SI^2 temperature (Thoden et al. 1994a). Clearly, there was no demonstration of an enhanced thermoregulatory drive to eliminate the residual heat remaining from the exercise period despite the absence of the antagonist influences of exercise on blood flow control. Conversely, there is a possibility that these data might be seen as a demonstration of an elevation of SP_{hy}. For this to occur, according to Sawka and Wenger (1988), it must be shown that heat dissipation responses are inactive at the new SP. The observation of a PostEx Tes plateau equal in magnitude to Tdil during exercise, suggests that thermal reflexive vasodilation was reduced before PreEx resting Tco could be re-established. In view of the elevated PostEx Tes it is equally possible that metabolic by-products of exercise may have caused an elevation of SP_{hy} similar to a fever response or that the responsiveness of SP_{hy} to an elevated Tes (i.e. a large positive load-error) is influenced.

In order to investigate the controls of a PostEx Tes elevation further experiments were required to examine the existence and stability of the relationship between Tdil and PostEx Tes during repeated endogenous work periods. An intermittent exercise protocol was chosen as a way to increase resting Tco such that an investigation of changes in

resting Tco could be undertaken to evaluate the relationship to Tdil and PostEx Tes. Based on the current findings from a continuous exercise protocol, the following experiment was designed to investigate the following hypotheses:

1) that an increase in the PreEx Tco will result in an a higher PostEx Tes plateau,
2) that an increase in the PreEx Tco will result in an elevated exercise Tdil and,
3) that there is a direct relationship between the exercise Tdil and the PostEx Tes.

6.0.2 Specific methods

Nine subjects were required to perform 3 cycles of 15 min exercise and 30 min recovery. Their average age, height and weight were 24±1 yr, 178±2 cm and 80±3 Kg respectively. Measurements of internal temperature were monitored by Tes and Tre and Tsk measurements included Tfh, Tch, Tfi, Tfa, Tth and Tca. Measurements of SBF were collected 10 min PreEx until completion of the experiment. The subjects entered the thermal chamber for a 2 hr acclimation period at 29°C, 50% humidity wearing shorts only and running shoes. At 10 min PreEx, the subject stood, mounted the treadmill and inserted the two-way breathing valve for VO_2 gas collection. Treadmill running exercise was chosen to stimulate about 75% of the subjects VO_{2max}. Following cessation of exercise the subjects stood for 30 min until end of recovery. This exercise-rest cycle was repeated for an additional two periods.

6.0.3 Results

PreEx Tco remained stable and consistent at 36.7°C for Tes and 37.0°C for Tre for the 10 min period prior to exercise (fig. 8). At the onset of exercise, Tsk fell quickly and Tfi showed the greatest effect of the exercise-induced vasoconstriction. While a drop

in all non-acral sites was measured, the magnitude of the fall was not as great as for acral changes. Tca remained low during PreEx and early exercise, suggesting that the calf may have been poorly perfused relative to other regions during PreEx. The response profile was identical to previous experiments (fig. 9).

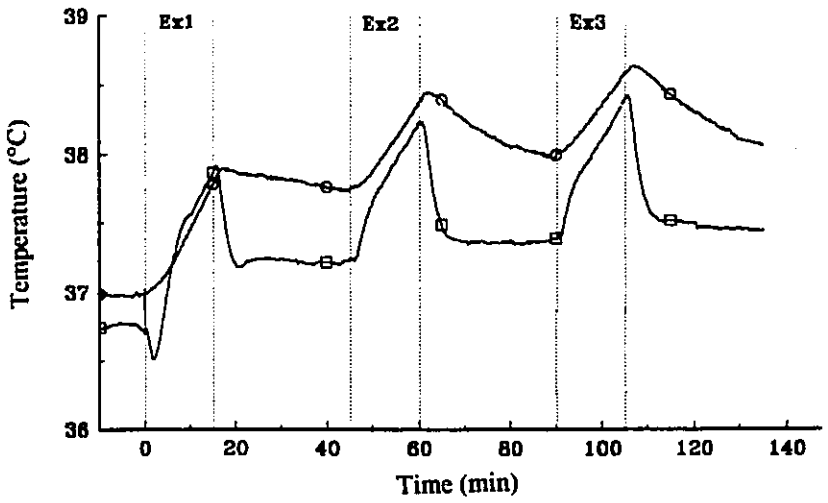

Fig. 8. Intermittent exercise mean core temperature response from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery. Rectal temperature (O) and esophageal temperature (□). (Ambient temperature, 29°C; relative humidity, 50%; n=9).

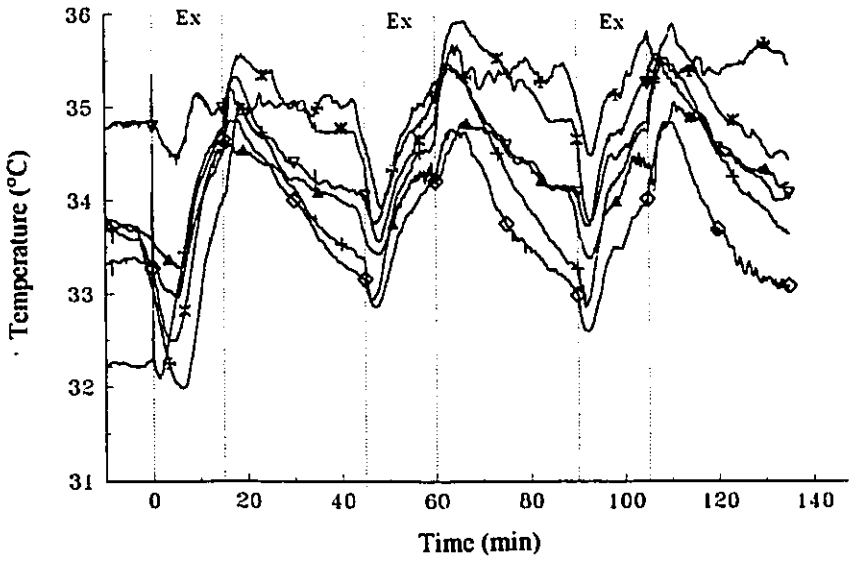

Fig. 9. Mean temperatures at sites of intermittent exercise from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery. Temperatures at surface (head, ∇; chest, ◊; finger, ✠; forearm, Δ; thigh, ✱; calf, +). (Ambient temperature, 29°C; relative humidity, 50%; n=9).

Tes fell 0.2°C in the first 2.1 min of exercise followed by a rise of 0.17°C·min^{-1} (10.2°C·hr^{-1}). Tre did not show a similar drop and began to rise immediately at the onset of exercise at a rate of 0.06°C·min^{-1} (3.8°C·hr^{-1}) which was essentially maintained for the exercise period. At 7.9 min into exercise Tes showed a significant fall in the rate of temperature increase which was preceded by a rise of Tsk with a lag time of 1.6 min between Tsk and Tes inflections. Heat loss from the skin resulted in a reduction in the rate of rise of Tes following inflection equivalent to a 69% drop in the rate of heat gain (fig. 8).

At the end of exercise Tes fell precipitously with a peak rate of fall equal to -0.22°C·min^{-1} (-13.2°C·hr^{-1}). Approximately 6.0 min into recovery Tes reached a minimum temperature after which it increased slightly to an elevated sustained plateau at 37.2°C (a 0.5°C elevation from PreEx) and was maintained for the full recovery period (table 15). Tre showed a persistent elevation in recovery with only a gradual fall of

0.003°C·min^{-1} over the latter part of the recovery period. Tsk returned to PreEx values within 15-20 min of recovery with the exception of Tfi and Tth (table 16).

Table 15. Mean core temperatures - intermittent exercise.

	Exercise 1		Exercise 2		Exercise 3	
	Tre (°C)	**Tes** (°C)	**Tre** (°C)	**Tes** (°C)	**Tre** (°C)	**Tes** (°C)
PreEx 10'	36.99	36.74				
SE	.05	.06				
Start	37.00	36.73	37.73	37.23	37.99	37.38
SE	.05	.06	.06	.06	.07	.07
End Ex	37.78†‡	37.87†‡	38.37†‡	38.22†‡	38.59†‡	38.40†‡
SE	.04	.07	.05	.09	.03	.11
PostEx 10'	37.84†‡	37.24†‡	38.25†‡	37.36†	38.43†‡	37.51†
SE	.06	.06	.07	.07	.08	.09
20'	37.79†‡	37.21†‡	38.05†	37.36†	38.20†	37.46†
SE	.06	.06	.07	.06	.08	.06
30'	37.73†‡	37.23†‡	37.99†	37.38†	38.05†	37.44†
SE	.06	.06	.07	.07	.10	.08

(† - indicates significant difference from PreEx1, ‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

Table 16. Mean surface temperatures - exercise period 1.

	Tfh (°C)	**Tch** (°C)	**Tfi** (°C)	**Tfa** (°C)	**Tth** (°C)	**Tca** (°C)
PreEx 10'	34.76	33.32	33.83	33.78	33.73	32.22
SE	.11	.22	.34	.19	.25	.15
Start	34.82	33.30	33.95	33.51	33.22	32.27
SE	.13	.23	.31	.19	.25	.16
End Ex	34.99‡	34.65‡	34.02	34.54‡	34.52‡	34.77‡
SE	.17	.21	.38	.18	.26	.22
PostEx 10'	34.66	34.36	35.05‡	34.38‡	35.38‡	34.58‡
SE	.22	.31	.19	.21	.20	.26
20'	34.18	33.59	34.99	34.09	34.73‡	33.73‡
SE	.24	.38	.16	.26	.23	.30
30'	34.04	33.15	34.60	33.63	34.36‡	33.34‡
SE	.24	.37	.14	.19	.21	.26

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

Similar changes in Tco and Tsk chronology were observed during the second (Ex2) and third (Ex3) exercise periods. In contrast to Ex1, Tes did not fall at the onset of Ex2 but showed a delay of 1.3 min before a measurable increase was noted. At this point Tes increased at a rate of 0.14°C·min^{-1} (8.4°C·hr^{-1}) which appeared lower than but not significantly different from Ex1. The magnitude of the fall of Tsk at the onset of Ex2 was less than Ex1 with a significantly shorter period of time before the inflection upward of Tsk [2.8 min as compared to 6.3 min for Ex1 (fig. 9)]. The inflection upward in Tsk was followed a short time later by the inflection in Tes with a delay of 2.5 min comparable to Ex1. The effect of increased surface flow was to reduce the rate of rise of Tes to 0.05°C·min^{-1} (3.0°C·hr^{-1}). Tre began to rise immediately to reach a constant rate equal to 0.05°C·min^{-1} (3.0°C·hr^{-1}).

Following the second exercise Tes fell precipitously, reaching a peak rate of fall equal to -0.24°C·min^{-1} (-14.4°C·hr^{-1}), and a minimum temperature of 37.3°C by 7.5 min into recovery. This was immediately followed by the demonstration of a significant elevated plateau equal to 37.4°C (table 15). Tre showed a gradual fall during recovery at a rate of -0.01°C·min^{-1} (-0.9°C·hr^{-1}) which was significantly greater than following Ex1. Tsk returned to PreEx within 20 min of recovery (table 17).

Table 17. Mean surface temperatures - exercise period 2.

	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfa (°C)	Tth (°C)	Tca (°C)
Start	34.04	33.15	34.60	33.63	34.36	33.34
SE	.17	.23	.24			
End Ex	35.12‡	34.21‡	35.27‡	34.23	34.86	34.67‡
SE	.17	.30	.34	.29	.16	.24
PostEx 10'	34.92‡	34.34‡	35.24‡	34.78‡	35.64	34.85‡
SE	.23	.38	.17	.22	.19	.18
20'	34.41	33.49	35.47‡	34.38	35.20	33.89
SE	.24	.46	.12	.27	.20	.24
30'	34.08	32.98	35.03	33.72	34.64	33.26
SE	.23	.40	.10	.28	.23	.21

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

At the onset of Ex3, Tsk fell to a minimum temperature in 2.5 min (comparable to the 2.8 min of Ex2 but significantly lower than the 6.3 min during Ex1). Tes remained stable during the first 1.3 min of exercise after which it increased at a rate of 0.14°C·min^{-1} (8.4°C·hr^{-1}) while Tre began to rise immediately at the onset of exercise at a rate of 0.05°C·min^{-1} (3.0°C·hr^{-1}). Tsk inflection upward preceded the drop in the rate of rise of Tes equal to 0.05°C·min^{-1} (3.0°C·hr^{-1}). Both Tsk and Tco continued to rise throughout exercise (fig. 8 and 9). Following cessation of exercise Tes fell precipitously, reaching a peak rate of -0.25°C·min^{-1} (15°C·hr^{-1}) and a minimum temperature of 37.4°C after 8.8 min before achieving a stable elevated plateau of 37.5°C which was maintained for the full recovery period. Skin temperatures demonstrated a gradual return to PreEx over the full period of recovery (table 18).

Table 18. Mean surface temperatures - exercise period 3.

	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfa (°C)	Tth (°C)	Tca (°C)
Start	34.08	32.98	35.03	33.72	34.64	33.26
SE	.23	.40	.10	.28	.23	.21
End Ex	35.28‡	34.03‡	35.81‡	34.36	34.87	34.75‡
SE	.19	.36	.23	.36	.24	.24
PostEx 10'	34.98	34.19‡	35.45	34.93‡	35.48	35.08‡
SE	.21	.36	.14	.31	.26	.22
20'	34.40	33.32	35.50	34.37	34.81	34.09
SE	.26	.43	.14	.32	.31	.27
30'	34.08	33.10	35.46	34.19	34.44	33.65
SE	.34	.65	.18	.51	.51	.36

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

Both HR and VO_2 showed the same pattern of abrupt and then more gradual increase for each of the exercise periods. Recovery VO_2 was rapid, reaching essentially PreEx values in 10 min. Initial HR recovery to about 100 beats·min^{-1} or less than 50% of the exercise adaptation occurred in 10-12 min and the remaining recovery was much slower (table 19). Minor variations in HR and VO_2 during subsequent recovery periods was statistically insignificant (table 19).

Table 19. Mean oxygen consumption and heart rate values - intermittent exercise

	Exercise 1		Exercise 2		Exercise 3	
	VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR ($b \cdot min^{-1}$)	VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR ($b \cdot min^{-1}$)	VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR ($b \cdot min^{-1}$)
PreEx 10'	3.19	70†	4.21	95	4.96	104
End Ex	37.05‡	171‡	37.45‡	179‡	38.29‡	186‡
PostEx 5'	5.52	103‡	5.89	114	5.90	117
10'	3.92	96	4.24	107	4.84	110
20'	na	95	na	104	na	107
30'	na	93	na	104	na	104

(na = not available, data was collected until 10 min into recovery only;† - indicates significant difference from exercise period 3; ‡-indicates significant difference from PreEx, $p>0.05$)

All PostEx temperatures were significantly ($p>0.05$) elevated above the initial PreEx Tes by 0.5°C, 0.7°C and 0.8°C for PostEx1, PostEx2, and PostEx3 respectively. Significant difference was found between PostEx1 and PostEx3 with no significant difference between PostEx1 and PostEx2 and PostEx2 and PostEx3. PostEx Tre was significantly ($p>0.05$) elevated above the initial PreEx for all PostEx periods (0.7°C, 1.0°C and 1.1°C for PostEx1, PostEx2 and PostEx3 respectively). Tre for PostEx1 was significantly different from PostEx2 and PostEx3 with no significant difference between PostEx2 and PostEx3.

Absolute temperature changes of PostEx Tes relative to PreEx Tes for Ex1 was equal to 0.5°C. The difference was less for both PostEx2 relative to the PreEx2 value of 0.2°C and for PostEx3 relative to the PreEx3 value of 0.1°C. These data indicate that the occurrence of an elevated Tes was not equally matched to the heat gained with repeated exercise. Measurements of Tes at exercise termination showed a 1.2°C, 1.0°C and 1.0°C elevation for Ex1, Ex2 and Ex3. When the PostEx Tes was represented as a relative difference from end of exercise Tes and PreEx Tes, it was found that the PostEx1 plateau

37.2°C represented a 48% elevation from PreEx1. The PostEx2 elevation was 20% relative to PreEx2 and the PostEx3 elevation was only 10% relative to PreEx3.

Estimates of body heat storage [calculated by integrating the area under the Tes curve - $des \cdot dt^{-1}$ (appendix H)] showed a significantly ($p>0.05$) greater heat storage during PostEx1 (1379°C·min^{-1}) as compared to PostEx2 (508°C·min^{-1}) and PostEx3 (415°C·min^{-1}) with no significant difference between PostEx2 and PostEx3. These data demonstrate that the magnitude of the PostEx Tes elevated plateau was not related to the absolute heat gain from exercise.

Measurements of Tdil for each exercise period were calculated using all three previously described methods of identification [Tsk inflection, Tfa-Tfi gradient and LDF methods (table 20 and fig. 10)]. Pearson correlation's showed values of r = 0.8 and r = 0.7 between Tsk and LDF and Tfa-Tfi gradient and LDF. Correlation's of 0.8 and 0.8 were calculated for Ex2 and 0.8 and 0.8 for Ex3. These data support the previous conclusions that Tsk and Tfa-Tfi gradient methods had provided good representations of SBF changes (fig. 10).

Table 20. Mean Tdil measurement determined by Tsk, Tfa-Tfi and LDF methods - intermittent exercise.

	Tsk inflection (°C)	**Tfa-Tfi gradient** (°C)	**LDF** (°C)	**Average** (°C)
Ex 1	37.16†	37.22†	37.18†	37.19
Ex 2	37.43	37.53	37.39	37.45
Ex 3	37.62	37.65	37.51	37.59

(† - denotes a significant difference from Ex 3)

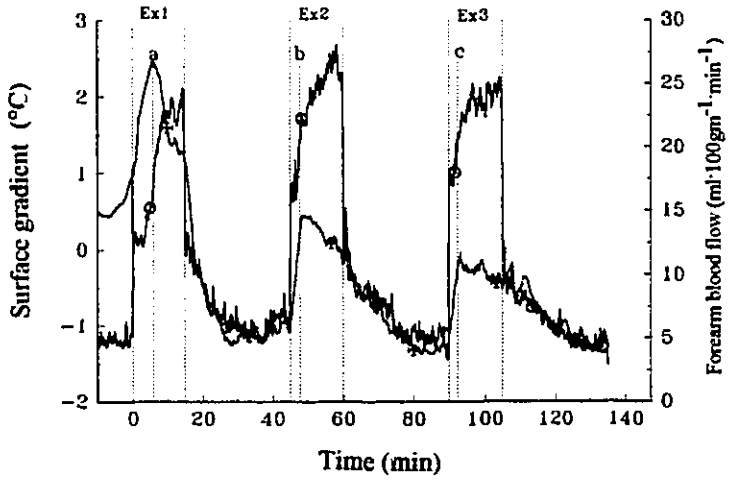

Fig. 10. Identification of mean skin vasodilation (Tdil) during intermittent exercise at time a (exercise 1), b (exercise 2) and c (exercise 3) measured by temperature gradient between the forearm and middle finger (Tfa-Tfi, ⊛), as described by Rubenstein and Sessler (1990) and Laser-Doppler flowmetry (LDF, O) (Ambient temperature, 29°C; relative humidity , 50%; n=9).

The magnitude of increase in Tdil between exercise periods was comparable to the increase in PostEx Tes. Tdil measurements were 37.2°C, 37.4°C and 37.6°C with a PostEx Tes of 37.2°C, 37.4°C and 37.5°C for Ex1, Ex2 and Ex3 respectively (table 20). Pearson correlation's between Tdil and PostEx Tes showed a high correlation of r=0.8, 0.8 and 0.9 for Ex1, Ex2 and Ex3 respectively. These data (table 21) show comparable trends to those of other preliminary experiments using intermittent exercise (Appendix D).

Table 21. Mean Tdil and PostEx Tes - intermittent exercise with 20 and 30 min recovery

	Surface Dilation (Tdil)		**PostEx Tes**	
	(20 min)	**(30 min)**	**(20 min)**	**(30 min)**
	(°C)	(°C)	(°C)	(°C)
Ex/Rec 1	37.41	37.16	37.34	37.22
Ex/Rec 2	37.64	37.46	37.48	37.38
Ex/Rec 3	37.75	37.63	37.65	37.46

6.0.4 Discussion

The primary conclusion to be drawn from these data is that there was a strong relationship between Tdil and PostEx Tes such that change in Tdil with repeated exercise was paralleled by a similar changes in PostEx Tes. A PostEx Tes elevated plateau was observed despite a return of SBF, Tsk, and VO_2 to PreEx values within 10-12 min of each recovery period. The change may suggest that the threshold for active vasodilation to Tco relationship increased as a function of repeated exercise. This suggests the possibility of: 1) a metabolically induced change in SP_{hy} thermosensitivity, 2) a decreased sensitivity to an increased LE, 3) a heat-load mediated alteration of SP_{hy} control of Tco, or 4) a range of temperature regulation defined by an upper threshold control for thermolytic temperature defence reflexes. It is only possible to speculate as to the origin of this effect and of the relationship between Tdil and PostEx.

Figure 11 is a schematic representation of the effects of changes in Tco on Tdil. Line A represents the type of change in Tdil associated with increases in Tco as demonstrated by this experiment. Line B represents the changes expected with SP_{hy} control whereby an increase in Tco would not cause an increase in Tdil but result instead in a larger LE signal. The observations from this study suggest that the changes in Tdil which paralleled the PostEx Tes are representative of changes precipitated by either thermal [large heat load gains (Johnson and Park 1982; Kaciuba-Uscilko et al. 1992)] or non-thermal [volume/baroreceptor (Johnson 1992; Kenney and Johnson 1992), hormonal (Stebbins and Symons 1993; Travis and Johnson 1993) or osmolarity (Fortney and Vroman 1985)] where these effects can be seen as the difference between line B and line A. However, whether the physiological control mechanism demonstrated by the PostEx maintenance of Tes involves SP_{hy} control (Sawka and Wenger 1988) or a range of

"normal" temperature controls (Mekjavic et al. 1991) cannot be determined from these data.

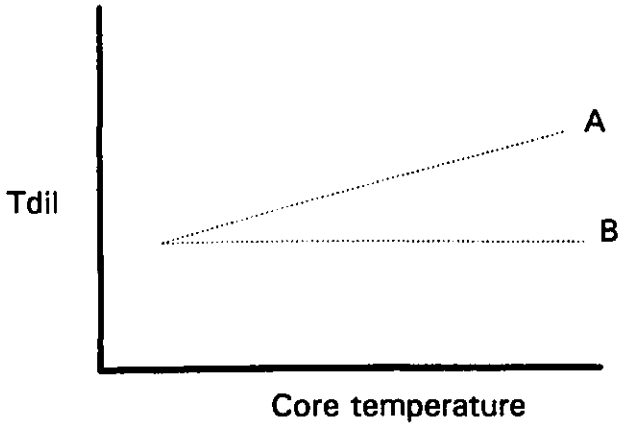

Fig. 11 Schematic diagram of Tdil relationship with Tco.

Finally, the observation of changes in the response of PostEx Tes with repeated work bouts showed that Tdil in Ex1 was 0.5°C above PreEx. However, the elevation of Tco before Tdil occurred was only 0.2°C for Ex2 and 0.1°C for Ex3. The ability of the system to invoke heat loss mechanisms more quickly may be a physiological adaptation whose result it is to prevent the accumulation of heat. If Tdil was triggered by solely absolute temperature changes, Tco may have reached dangerous levels. Thus it is possible that there is an important physiological benefit to maintaining an elevated Tco as a mechanism with which to facilitate sensitivity and/or magnitude of heat dissipation reflexes.

6.0.5 Conclusion

Intermittent exercise demonstrated a disturbance of PostEx thermal homeostasis which was similar to continuous exercise. This was identified by a maintenance of the Tes elevation at progressively higher values with each exercise and similar elevations in Tdil.

In addition, these data show that changes in Tco did not affect the Tdil to PostEx Tes relationship.

6.1.0 Exogenous heat load - hot water treatment

6.1.1 Introduction

It was demonstrated that repeated running-recovery cycles produced patterns of rise and then fall of Tes to an elevated PostEx plateau that was equal to Tdil. This was similar to previous results except that the second exercise was begun at an elevated Tes and produced further elevation of Tdil with a comparable effect on PostEx Tes. However, it was still not possible to describe the mechanism which produced the change in PostEx thermal stimulus threshold. It is possible that the significant elevation of whole body heat content may have been the primary cause of the PostEx Tes elevation and which therefore eliminated the metabolic origin which has only been demonstrated following prolonged strenuous exercise (Haight and Keatinge 1973; Johnson and Ruhling 1985). Therefore the following investigation was designed to examine the effect of avoiding muscular activity by generating a heat load through water immersion (Tw 44°C). The experiment was conducted to test the hypothesis that an elevation of Tco above Tdil by an exogenous heat load (water immersion) will not result in a PostEx Tes plateau.

6.1.2 Specific methods

Nine male subjects were required to sit in a water bath at 44°C for a period of 15 min. Their average age, height and weight were 24±1 yr, 178±2 cm and 80±3 Kg respectively. Measurements of internal temperature were monitored by Tes and Tre, and Tsk measurements (including Tfh, Tch, Tfi, Tfa, Tth and Tca). Measurements of SBF were made 20 min pre-treatment until completion of the experiment for one subject only. The subjects entered the thermal chamber for a 2 hr acclimation period at 29°C, 50%

humidity wearing only shorts and running shoes. At 2 min before treatment time subjects were taken into an adjacent room where they entered the water tank. The temperature of the water was maintained at 44°C. The choice of the water temperature was based on data collected from pilot work (Appendix F) which was conducted to determine a protocol which would produce a rate of heat gain and elevation in Tes comparable to exercise. The subjects remained in the water tank for 15 min after which they were removed and taken into the thermal chamber (Tam 29°C) for a 60 min recovery period.

6.1.3 Result

Core temperatures remained stable and consistent at 36.7°C for Tes and 37.0°C for Tre for the 10 min period prior to exercise (table 22). The subjects required 2 min to walk the short distance from the thermal chamber to the water tank. The effect of this move was shown as a fall in Tes equal to 0.14°C. Upon immersion there was an immediate rise in Tes equal to 0.18°C·min^{-1} (10.8°C·hr^{-1}). Tre did not show a similar fall but remained stable and began to rise at constant rate 0.05°C·min^{-1} (3.0°C·hr^{-1}) 2.2 min following immersion (4.2 min following removal from thermal chamber).

Table 22. Mean core temperatures - water immersion.

	Tre (°C)	Tes (°C)
PreH₂O 10'	36.96	36.74
SE	.05	.06
Start	36.97	36.65
SE	.05	.06
End H₂O	37.62‡	37.88‡
SE	.08	.09
PostH₂O 10'	37.61‡	36.78
SE	.04	.05
20'	37.47‡	36.81
SE	.06	.06
30'	37.39	36.89
SE	.06	.06
40'	37.35	36.93
SE	.04	.05
50'	37.30	36.91
SE	.04	.04
60'	37.26	36.91
SE	.06	.07

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

The absolute temperature rise was 1.3°C and 0.7°C for Tes and Tre respectively which compared closely to the 1.1 °C and 0.8 °C elevation with Ex1 of intermittent exercise. The initial slopes for both Tes and Tre were similar for both conditions suggesting the water bath temperature was ideal for generating Tco changes comparable to exercise (Fig. 12).

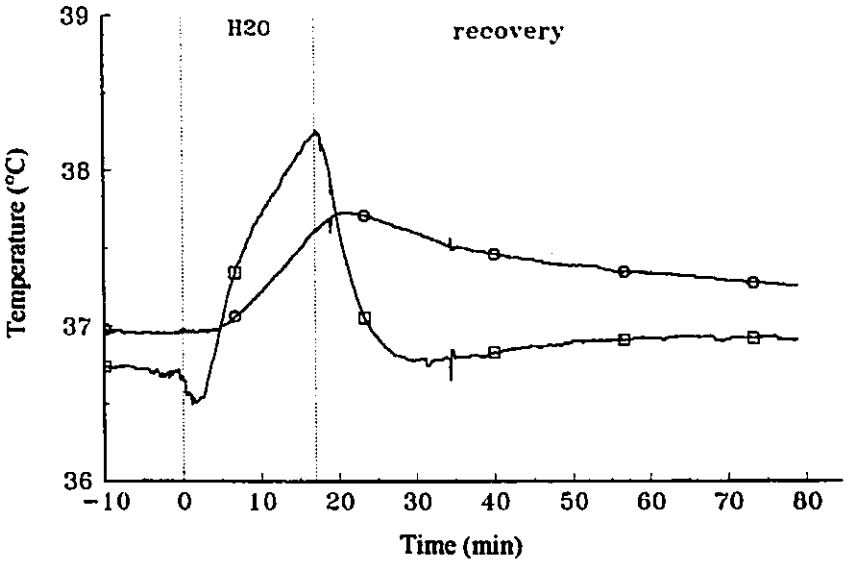

Fig. 12. Mean core temperatures at sites from rest through the start and end of water immersion (Tw 44° C) and during standing, leaning recovery. Temperature at esophageal (□) and rectal (O). (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

Changes in Tsk at the same anatomical sites used for other experiments were impossible to monitor because the majority of the probes would have been under water. Thus, only Tfa, Tfh and Tfi were used to evaluate the effects of the exogenous heat load on Tsk response. Tsk fell prior to the immersion, most likely related to the changes associated with the movement of the subject from the thermal chamber to the water tank, but increased rapidly following immersion. Tsk increased at a consistent rate over the first 8 min of immersion after which an upward inflection in Tsk was observed (fig. 13). Generally, the inflection occurred later than was observed during Ex1 of intermittent exercise, but this trend, based solely on Tfa measurement, was not easily identified in all subjects. Although graphical representation demonstrates this change (fig. 13), statistical evaluation does not show a significant fall in the rate of increase in Tfa. Thus an interpretation of changes similar to those for exercise and implicating Tdil was not possible based on these data and only the trend in Tsk chronology is reported. The change in the rate of rise of Tfa was from 0.12 °C/min^{-1} (7.3°C·hr^{-1}) to 0.05°C·min^{-1} (3.3°C·hr^{-1}). A similar inflection to exercise was observed in Tes with a drop in the rate of rise to

0.090°C·min^{-1} (5.4°C·hr^{-1}) from an initial increase of 0.18°C·min^{-1} (10.8°C·hr^{-1}). This was equivalent to a 50% drop in the rate of heat gain. No change was observed in Tre values.

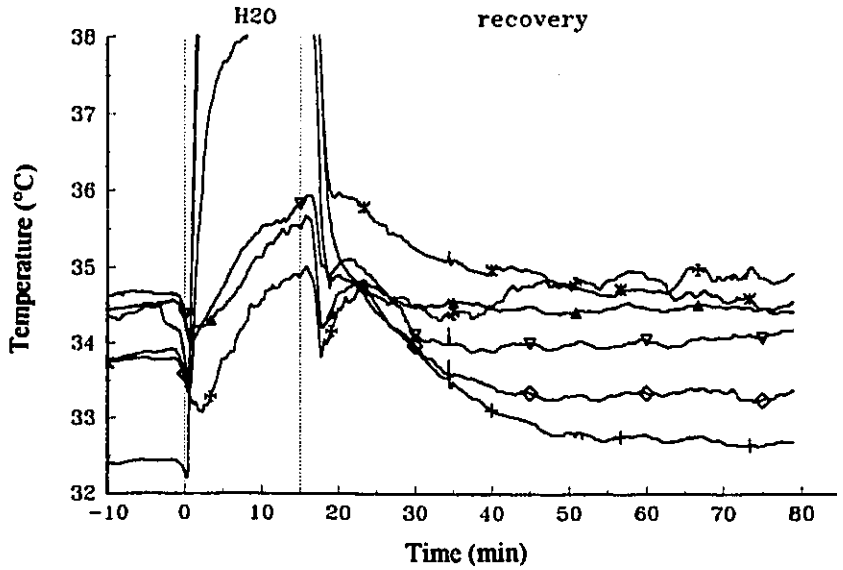

Fig. 13. Mean temperatures at sites from rest through the start and end of water immersion (Tw 44°C) and during standing, leaning recovery. Temperatures at surface (head, ∇; chest, ◊; finger, ✻; forearm, Δ; thigh, ✱; calf, +). (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

At the end of the treatment session, Tes fell immediately, while Tre remained elevated at 37.7°C for 2.0 min before beginning to fall. Tes fell at a rate equal to -0.2°C·min^{-1} (-12°C·hr^{-1}) reaching a minimum temperature of 36.8°C at 13 min PostEx which was maintained for the full period of recovery. Tre fell to a final temperature of 37.3°C equal to a 0.3°C elevation from PreEx. Tsk returned to PreEx values in the first 10 min of recovery with the exception of Tth and Tca which remained elevated for most of recovery (table 23).

Table 23. Mean surface temperatures - water immersion.

	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfa (°C)	Tth (°C)	Tca (°C)
PreH₂O 10'	34.63	33.77	34.38	34.44	33.73	32.40
SE	.06	.16	.21	.10	.16	.17
Start	34.39	33.59	33.79	34.32	33.54	32.29
SE	.05	.18	.27	.11	.13	.16
End H₂O	35.75‡	37.67‡	34.67	35.13	39.71‡	40.19‡
SE	.11	.20	.24	.24	.15	.24
PostH₂O 10'	34.27	34.05	34.57	34.49	35.31	34.01
SE	.12	.21	.25	.13	.09	.15
20'	33.89	33.44	34.40	34.47	34.90	33.17
SE	.18	.22	.34	.06	.09	.18
30'	33.94	33.25	34.74	34.37	34.77	32.77
SE	.14	.23	.24	.08	.10	.18
40'	34.02	33.33	34.92	34.45	34.69	32.76
SE	.15	.24	.23	.12	.16	.19
50'	34.03	33.36	34.80	34.45	34.61	32.69
SE	.15	.24	.18	.12	.14	.18
60'	34.15	33.36	34.89	34.52	34.40	32.68
SE	.23	.37	.24	.16	.23	.29

(‡ -indicates significant difference from each separate PreEx period. $p>0.05$)

Forearm LDF measurements from a single subject showed a slow rise over the period of treatment; however, the magnitude of increase was not comparable to exercise. LDF gradually returned to PreEx values over the period of recovery (fig. 14).

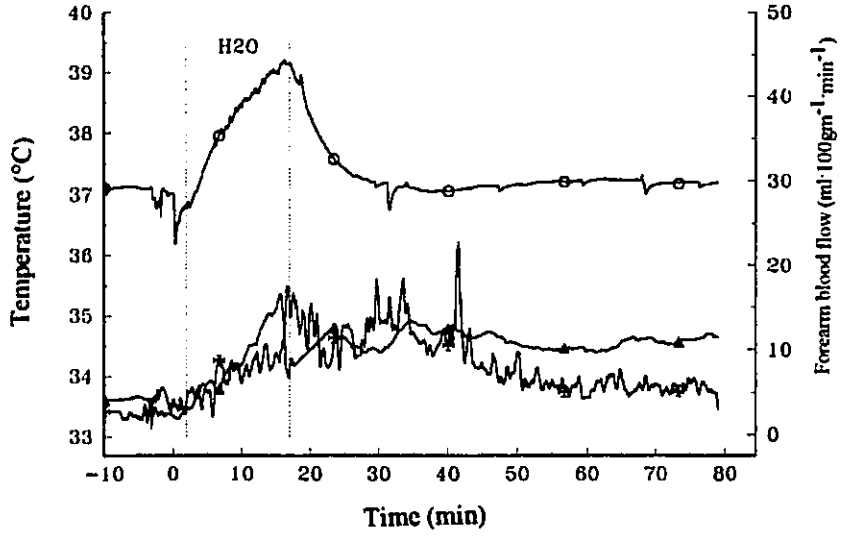

Fig. 14. Esophageal (O) and forearm temperatures (Δ) and forearm blood flow (⊛) for one subject from rest through the start and end of water immersion (Tw 44°C) and during standing leaning recovery. Forearm surface blood flow measured by Laser-Doppler flowmetry. (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

PostEx HR and VO2 were comparable to PreEx values. HR increased to a peak value during immersion at around 100-110 beats·min^{-1} returning to PreEx values within minutes of treatment termination (table 24).

Table 24. Mean oxygen consumption and heart rate values - water immersion

		VO_2 (ml·kg^{-1}·min^{-1})	HR (b·min^{-1})
PreEx	**10'**	3.10	68
End H_2O		na	98‡
PostH_2O	**5'**	4.96	74
	10'	3.92	72
	20'	na	68
	30'	na	73
	40'	na	69
	50'	na	70
	60'	na	73

(na = not available, data was collected 10 min into recovery only; ‡ -indicates significant difference from PreEx, p>0.05)

6.1.4 Discussion

The primary observation to be drawn from these data is that a PostEx Tes elevated plateau was not achieved following a significant heat gain produced by water immersion. The experiment was designed to generate a heat load comparable to exercise but different in origin from the metabolic heat load of exercise which generated a sustained PostEx elevation in Tes (Thoden et al. 1994a). There was an immediate fall in Tes with temperatures returning to PreEx values within 10 min of recovery. Although figure 12 suggests a trend for Tes to rise during the balance of recovery, its final value of 36.9°C was not statistically different from PreEx. Following the withdrawal of the subject from the water bath (Tw 44°C), Tsk fell rapidly in response to a much cooler Tam 24°C (the temperature of the laboratory prior to entrance into the thermal chamber) and resulted in shivering in some subjects which may have contributed to the slight elevation. The shivering ceased following a short period of time in the thermal chamber at Tam 29°C.

The absolute increase of Tes during treatment was 0.2°C greater than for Ex1 of intermittent exercise, and whole body heat gain during immersion was estimated at 750°C·min^{-1} as compared to 450°C·min^{-1} for Ex1 of intermittent exercise. In contrast to exercise, recovery Tes following water immersion at Tw 44°C was not significantly different from pre-treatment levels despite the differences in absolute temperature and heat gain following water treatment. During exercise heat is produced by the metabolic activity of contracting musculature whereas during water immersion heat is gained by the core through convective exchange. If Tes is representative of hypothalamic blood temperature changes it could be assumed that thermolytic controls would be triggered by an elevation of Tco independent of the origin. An increase in Tco should therefore have been regulated in a similar manner for both conditions. However, this clearly was not so in this

experiment. The difference in recovery Tes may be attributed to a difference in SP_{hy} responsiveness to an LE signal which is dependent on a weighted signal of Tco and Tsk inputs (Gisolfi and Wenger 1984) or an exercise-related effect. Therefore, it could be speculated that PostEx elevations were the product of a metabolic stimulus of thermolytic controls rather than the product of a heat gain.

6.1.5 Conclusion

It can be concluded that an increase in whole body heat content alone does not produce a disturbance of PostEx thermal homeostasis. This conclusion suggests that there may be a neuro-muscular or other metabolically related origin for the PostEx Tes elevation.

6.2.0 Endogenous and exogenous heating combined

6.2.1 Introduction

The PostEx Tes elevated plateau was maintained for a prolonged period of time (65 min or longer) and was equal in magnitude to Tdil during exercise, suggesting that thermal reflexive vasodilation was significantly reduced before PreEx resting Tco could be re-established. The passive attitude of thermal control systems towards an elevated Tco during recovery considerably strengthens the idea of a range of "normal" temperatures to which thermoregulatory mechanisms are relatively insensitive during Tco fluctuations. Water immersion at Tw 44°C did not produce a comparable Tes elevation. Therefore, if the PostEx Tdil represents an upper limit of control defined by some exercise-related effect, it should be expected that an exogenous increase of Tco above Tdil would not result in an elevation of Tes above pre-treatment levels, regardless of the pre-treatment conditions.

The following experiment was designed to test the hypothesis that a PostEx elevation of Tes following exercise will not be further increased following an exogenous heat load (water immersion).

6.2.2 Specific methods

Nine subjects performed a 15 min exercise and a 15 min recovery (Tam 29°C) followed by a 15 min period of water immersion to the neck (Tw 44°C) and 30 min recovery (Tam 29°C) . Their average age, height and weight were 24±1 yr, 178±2 cm and 80±3 Kg respectively. Measurements of internal temperature were monitored by Tes and Tre, and Tsk measurements (including Tfh, Tch, Tfi, Tfa, Tth and Tca).

Measurement of SBF was done from 20 min pre-treatment until completion of the experiment for one subject. The subjects entered the thermal chamber for a 2 hr acclimation period at 29°C, 50% humidity wearing only shorts and running shoes. At 10 min PreEx the subject stood and mounted the treadmill. Exercise intensity was equal to about 75% of VO_{2max}. Following cessation of exercise the subjects recovered for 15 min after which they where taken into an adjacent room where they entered the water tank at Tw 44°C. The subject remained in the water for 15 min after which they were removed and taken into the thermal chamber for a 60 min recovery period (Tam 29°C).

6.2.3 Results

PreEx Tco remained stable (Tes = 36.7°C and Tre = 36.9°C) for the 10 min prior to exercise (table 25). At the onset of exercise Tes fell 0.2°C in the first 2.1 min and Tsk during the first 6.3 min. Tre did not show any indication of a fall for the first minutes of exercise and thereafter rose slowly at a rate of 0.06°C·min^{-1} (3.5°C·hr^{-1}). The effect of an upward inflection in Tsk on Tes was demonstrated approximately 1-1.5 min later as a significant drop in the rate of rise of Tes from 0.16°C·min^{-1} (9.6°C·hr^{-1}) to 0.05°C·min^{-1} (3.0°C·hr^{-1}). For the remainder of the exercise, Tre and Tes continued to rise at constant rates reaching values equal to 37.5°C and 37.7°C a 0.6°C and 1.0°C elevations from PreEx (Fig. 15).

Table 25. Mean core temperatures - Ex/H2O.

		Tre (°C)	Tes (°C)
PreEx	**10'**	36.86	36.68
SE		.05	.05
Start		36.84	36.64
SE		.04	.05
End Ex		37.52‡	37.68‡
SE		.04	.05
PostEx	**5'**	37.64‡	37.06
	SE	.02	.06
	10'	37.68‡	37.13‡
	SE	.03	.05
	15'	37.68‡	37.11‡
	SE	.02	.06
End H_2O		38.22‡	38.77‡
SE		.06	.10
Post H_2O	**10'**	38.25‡	37.16‡
	SE	.06	.04
	20'	38.01‡	37.01
	SE	.04	.06
	30'	37.79‡	37.07‡
	SE	.03	.06
	40'	37.66‡	37.11‡
	SE	.02	.05
	50'	37.55‡	37.12‡
	SE	.02	.05
	60'	37.52‡	37.10‡
	SE	.04	.06

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

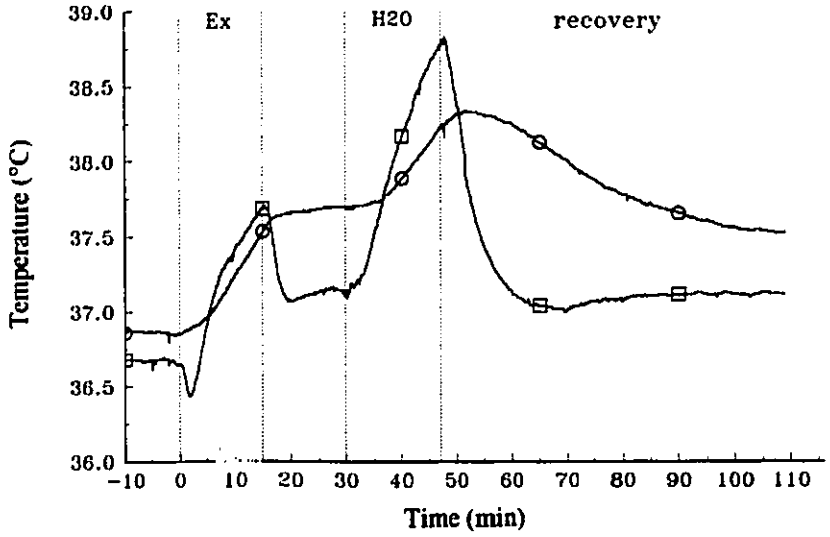

Fig. 15. Mean temperatures at sites from rest through the start and end of exercise (10-13 METS) and standing, leaning recovery followed by the start and end of water immersion (Tw 44°C) and during standing leaning recovery. Temperature at esophageal (□) and rectal (O). (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

Following cessation of exercise, Tes fell at a peak rate equal to -0.24°C·min^{-1} (-14.4°C·hr^{-1}) to a minimum of 37.1°C by 5 min into recovery where it remained elevated and stable for the remainder of recovery. Tre did not show a fall and reached 37.7°C by 10 min PostEx where it remained throughout recovery. Tsk reached peak temperatures approximately 4.0 ± 0.5 min PostEx which was nearly simultaneous with the 5 min PostEx time that Tes began to reach a plateau (table 26). Non-acral Tsk returned to PreEx values within the 15 min recovery with the exception that Tth and Tca remained elevated (fig. 16). VO_2 returned to PreEx values within minutes of exercise termination while HR remained slightly elevated over the full 15 min recovery period (table 27).

Table 26. Mean surface temperatures -Ex/H2O.

	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfa (°C)	Tth (°C)	Tca (°C)
PreEx 10'	34.87	33.43	34.22	34.11	33.90	32.57
SE	.10	.24	.14	.19	.18	.11
Start	34.98	33.60	33.94	34.10	33.73	32.69
SE	.06	.18	.14	.15	.15	.10
End Ex	35.24	34.74‡	33.37	33.84	34.85	35.13‡
SE	.13	.24	.36	.31	.10	.15
PostEx 5'	35.16	34.67‡	34.49	34.47	35.57‡	35.36‡
SE	.12	.22	.18	.17	.12	.13
10'	34.87	34.23	34.99	34.30	35.43‡	34.90‡
SE	.13	.23	.14	.11	.12	.15
15'	34.46	33.77	34.70	34.02	35.11‡	34.50‡
SE	.17	.25	.11	.10	.11	.14
EndH2O	36.28‡	38.11‡	35.33‡	35.72‡	40.50‡	41.15‡
SE	.15	.46	.17	.16	.12	.18
PostEx 10'	34.94	34.53	35.25	34.69	35.68‡	34.77‡
SE	.09	.15	.22	.14	.12	.20
20'	34.52	33.83	35.15	34.61	35.43‡	34.01
SE	.16	.18	.14	.15	.13	.29
30'	34.32	33.37	35.23	34.63	34.92	33.78
SE	.15	.27	.14	.16	.18	.23
40'	34.24	33.31	35.18	34.58	34.59	33.56
SE	.17	.30	.13	.22	.20	.22
50'	34.35	33.29	35.27	34.67	34.42	33.47
SE	.17	.29	.10	.16	.23	.21
60'	34.30	33.42	35.36	34.73	34.19	33.34
SE	.29	.37	.16	.19	.33	.26

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

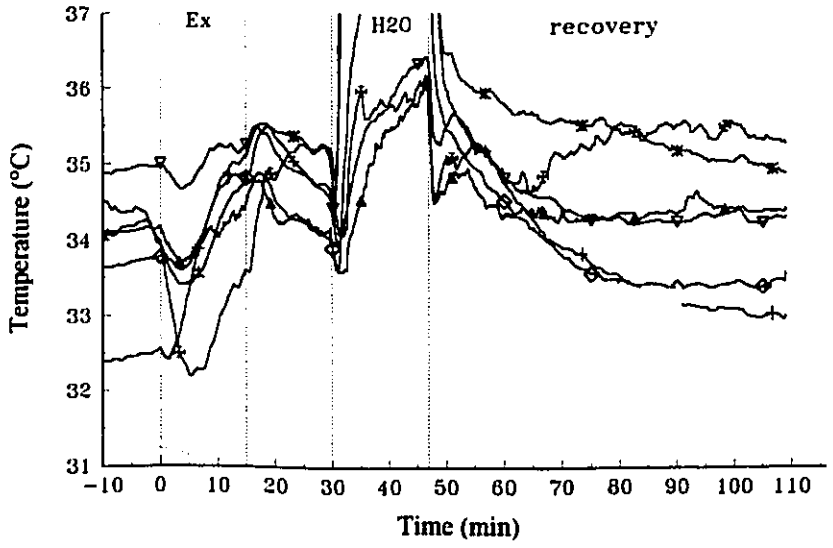

Fig. 16. Mean temperatures at sites from rest through the start and end of exercise (10-13 METS) and standing, leaning recovery followed by the start and end of water immersion (Tw 44°C) and during standing leaning recovery. Temperatures at surface (head, ∇; chest, ◊; finger, ✱; forearm, Δ; thigh, ✱; calf, +). (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

Following the exercise recovery period in the thermal chamber the subjects were submerged in a water bath of 44°C for a period of 15 min. Starting temperatures were therefore equal to those temperatures recorded at the end of recovery. Upon immersion an upward inflection of Tes occurred at a rate of 0.15°C·min^{-1} (9.0°C·hr^{-1}). Unlike during exercise there was no significant fall in Tes, however, the rate of rise was reduced after 5.7 min in the water bath. The rate for the remaining of the water immersion was equal to 0.08°C·min^{-1} (4.8°C·hr^{-1}). Tre remained at 37.7°C for the first 2.8 min after which it increased at a constant rate of 0.05°C·min^{-1} (3.1°C·hr^{-1}) (fig. 15).

Tsk, represented solely by Tfa, increased at a rate of 0.37°C·min^{-1} (average of 4 Tfa sites), which was maintained for the first 1.6 min followed by a downward inflection equal to 0.09°C·min^{-1}. Although this trend in Tsk was observed graphically no statistical significance was measured. Individual data did not show a consistent trend therefore it was not possible to apply an exercise (Tdil) interpretation to these data.

Following the treatment, Tes fell precipitously at a peak rate of -0.17 $C \cdot min^{-1}$ (-10.2°$C \cdot hr^{-1}$) reaching a minimum temperature of 37.1°C at 10.9 min that was maintained during recovery (table 25). Tre remained stable for the first 10 min of recovery at 38.3°C and fell gradually over the next 40 min reaching 37.5°C which was maintained for the remainder of recovery (fig. 15). No elevation was demonstrated in VO_2 following immersion and HR returned to pre-treatment values within minutes of treatment termination (table 27).

Table 27. Mean oxygen consumption and heart rate values - Ex/H_2O.

		Exercise				H_2O	
		VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR ($b \cdot min^{-1}$)			VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR ($b \cdot min^{-1}$)
PreEx	**10'**	4.34	64	**PreH_2O**	**10'**	4.32	64
End Ex		36.80‡	168‡	**EndH_2O**		NA	128‡
PostEx	**5'**	5.52	92	**PostH_2O**	**5'**	5.26	92
	10'	4.29	86		**10'**	4.32	86
	15'	na	88		**20'**	na	104‡
					30'	na	90
					40'	na	89
					50'	na	87
					60'	na	85

(na = not available, data was collected 10 min into recovery only; ‡ -indicates significant difference from PreEx, $p>0.05$)

Data from one subject only demonstrated that forearm LDF returned to PreEx values within 10 min (fig. 17). During water immersion LDF showed an increase and gradual return over the period of recovery but it is known that movement artifacts may cause unusual elevations in LDF measurements.

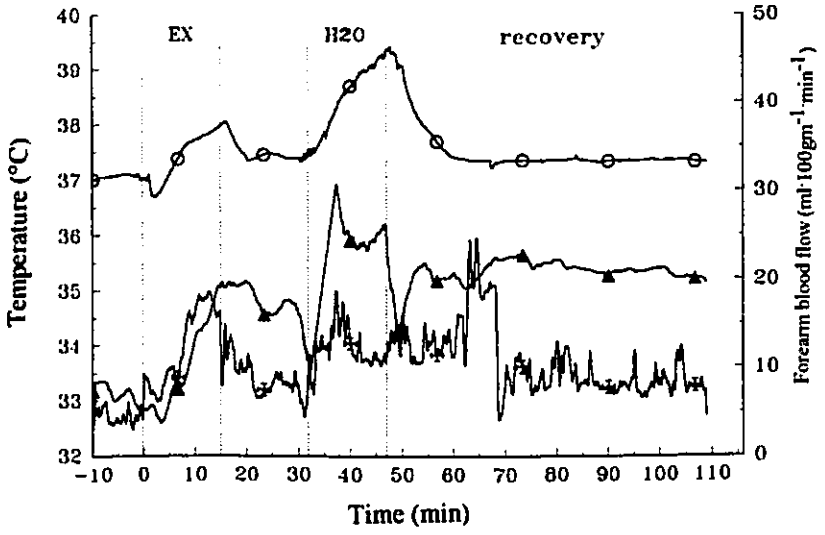

Fig. 17. Esophageal (O) and forearm temperatures (Δ) and forearm blood flow (✠) for one subject from rest through the start and end of exercise (10-13 METS) and during leaning recovery, followed by the start and end of water immersion (Tw 44°C) and during standing, leaning recovery. Forearm surface blood flow measured by Laser-Doppler flowmetry. (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

6.2.4 Discussion

The observation following the PostEx water immersion of an elevated Tes that was equal in magnitude to that following PostEx Tes supports the previous conclusion that the heat load was not the primary cause of a PostEx disturbance of thermal homeostasis. First, the magnitude of elevation of Tes was slightly greater during water immersion (1.5°C) as compared to exercise (1.0°C) and this resulted in a difference in body heat content equal to 850°C·min^{-1} and 430°C·min^{-1} for H_2O and exercise respectively. Despite the significant heat gained during water immersion (Tw 44°C), Tes was not significantly elevated from pre-treatment (or PostEx) values. Secondly, it was demonstrated that repeated running-recovery cycles (fig. 8) produced patterns of rise and then fall of Tes to an elevated PostEx plateau that was equal to the immediately previous Tdil and that the second exercise was begun at an elevated Tes and produced further elevation of PostEx

Tes equal to 0.2°C. However, an elevation of Tes following immersion did not occur (fig. 12).

Based on SP_{hy} theory, heat loss following immersion should have taken longer than following exercise due to the large differences in the whole body heat content. However, the opposite was demonstrated. The passive attitude toward the maintenance of a significant elevation of Tco following endogenous (exercise) treatment appeared very different following exogenous treatment.

6.2.4 Conclusion

The PostEx Tes elevated plateau was unchanged following a significant exogenous (water immersion) heat gain. These data support the conclusion that an increase in whole body heat content was not the primary cause of a disturbance in thermal homeostasis. The data suggest that exercise heat load and non-metabolic heat load affect the regulatory system in different ways.

6.3.0 Five-minute Exercise

6.3.1 Introduction

That a neuro-muscular or metabolic-related effect is the primary cause of a sustained PostEx Tes elevation is supported by the preceding investigations. However, the exact mechanism which produces this change cannot be determined from these data. It can be suggested that these observations are inconsistent with SP_{hy} theory and are consistent with the concept of a range of "normal" temperature for Tco regulation. There is some consistency of these observations with those of Nadel et al. (1974) and Fox and Edholm. (1963) who demonstrated the plasticity of thermal thresholds. Exercise has been demonstrated to cause a change in threshold activity which is significantly different from rest (Kellogg et al. 1991c). However, it has never been demonstrated to persist following exercise. In fact, there has been little research which has dealt with the question of thermal thresholds following exercise. The data presented in the previous sections would suggest an absence of appropriate thermolytic reflex mechanism in light of the existing elevated Tco. The consistent observation of a relation between PostEx Tes and the Tes equivalent at the time of surface vascular vasodilation in intermittent exercise suggests that there is some physiological significance to this observation. Single exogenous heat load treatments support an exercise-related effect and when applied during exercise recovery demonstrates a lack of an effect on the Tes elevation. Therefore it is plausible that exercise may have an effect on the establishment of PostEx thermal thresholds which has not been previously reported.

The following experiment was designed to investigate the effect of short duration exercise, which produces a esophageal temperature (Tes) elevation to a value below that core temperature required to initiate surface vasodilation (Tdil), on recovery Tes response

in order to test the hypothesis that this will result in a sustained post-exercise esophageal temperature plateau.

6.3.2 Specific methods

Nine subjects performed a 5 min exercise followed by a 60 min recovery. Their average age, height and weight were 24±1 yr, 178±2 cm and 80±3 Kg respectively. Measurements of internal temperature were monitored by Tes and Tre, and Tsk measurements (including Tfh, Tch, Tfi, Tfa, Tth and Tca). Measurement of SBF was done from 20 min pre-treatment until completion of the experiment for three subjects only. HR was measured continuously for the full period of the experiment. VO_2 was measured from 10 min PreEx, throughout exercise and until 15 min into recovery. The subjects entered the thermal chamber for a 2 hr acclimation period at 29°C, 50% humidity wearing only shorts and running shoes. At 10 min PreEx, the subject stood mounted on the treadmill and inserted the two-way breathing valve for VO_2 gas collection. Treadmill running exercise was chosen to stimulate about 75% of the subject's VO_{2max}. Following cessation of exercise the subject stood for 60 min until end of recovery.

6.3.3 Results

Pre-exercise core temperature remained stable and consistent at 36.8°C for Tes and 36.9°C for Tre during the 10 min period prior to exercise (table 28). At the onset of exercise Tes fell 0.1°C to 36.7°C in 2.0 min, after which it increased at a rate of 0.18° $C \cdot min^{-1}$ ($10.8°C \cdot hr^{-1}$) while Tre began to rise immediately (fig. 18). Non-acral (with the exception of Tca) and acral skin temperatures fell during exercise with no demonstration of an inflection upward consistent with an increase in surface blood flow related to surface

vasodilation (fig. 19). As a result no significant drop in the rate of rise of esophageal temperature was observed during the period of exercise.

Table 28. Mean core temperatures - 5 min exercise.

	Tre (°C)	Tes (°C)
PreEx 10'	36.92	36.77
SE	.07	.04
Start	36.92	36.77
SE	.06	.05
End Ex	37.08	37.05
SE	.06	.05
PostEx 10'	37.25	37.04
SE	.06	.05
20'	37.28	37.03
SE	.07	.05
30'	37.26	37.02
SE	.07	.04
40'	37.24	37.05
SE	.07	.04
50'	37.22	37.04
SE	.07	.04
60'	37.19	37.04
SE	.11	.05

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

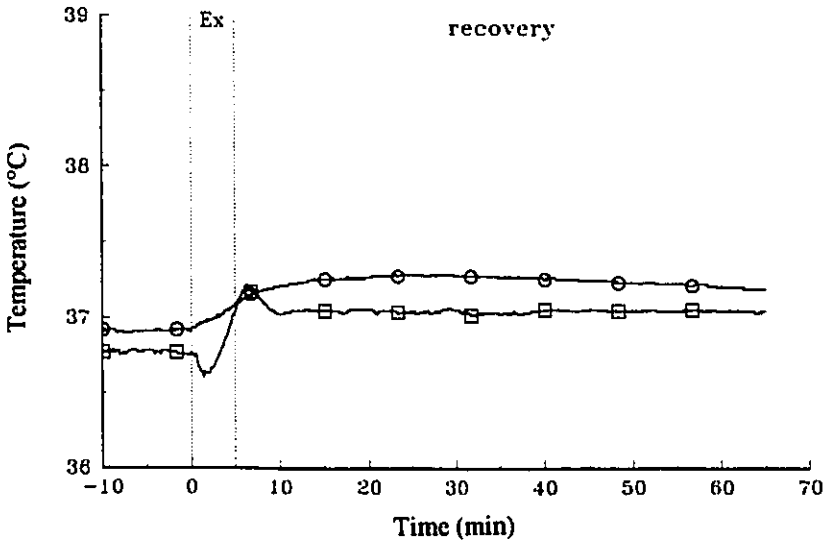

Fig. 18. Mean temperatures at sites from rest through the start and end of exercise (10-13 METS) and standing, leaning recovery. (Exercise performed for 5 min). Temperature at esophageal (□) and rectal (O). (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

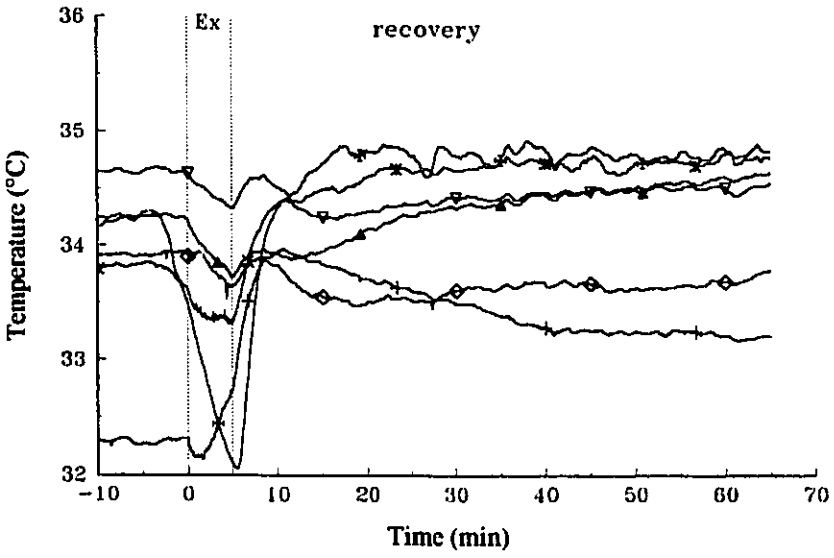

Fig. 19. Mean temperatures at sites from rest through the start and end of exercise (10-13 METS) and standing, leaning recovery. (Exercise performed for 5 min). Temperatures at surface (head, ∇; chest, ◊; finger, ✻; forearm, Δ; thigh, ✱; calf, +). (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=9).

Tes reached 37.1°C at the end of exercise and continued to rise for a period of 1.5 min to 37.2°C after which it fell to a stable plateau around 37.0°C that was 0.3°C above PreEx. End exercise Tre was equal to 37.1°C, increasing to 37.3°C by 8.6 min into recovery. Tre remained elevated for the first 30 min at 37.3°C and then fell slightly to 37.2°C where it remained for the duration of recovery. VO_2 returned to PreEx values within 5-7 min of recovery and HR was not statistically different from PreEx (table 29).

Table 29. Mean oxygen consumption and heart rate values - 5 min exercise.

		VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	HR ($b \cdot min^{-1}$)
PreEx	**10'**	3.98	67
End Ex		36.20	159
PostEx	**5'**	5.31	85
	10'	3.83	82
	20'	na	81
	30'	na	78
	40'	na	76
	50'	na	73
	60'	na	74

(na = not available, data was collected 10 min into recovery only; ‡ -indicates significant difference from PreEx, $p>0.05$)

Changes in Tsk showed that both acral and non-acral temperatures with the exception of Tca were lower than pre-exercise. Tfh, Tch and Tfa remained lower than during PreEx for the first 20 min of recovery and Tfi, Tth and Tca showed a gradual elevation in the initial minutes of recovery (table 30).

Table 30. Mean surface temperatures - 5 min exercise.

	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfa (°C)	Tth (°C)	Tca (°C)
PreEx 10'	34.64	33.90	34.16	34.23	33.79	32.29
SE	.04	.08	.19	.16	.22	.18
Start	34.63	33.91	33.48	34.21	33.60	32.28
SE	.10	.18	.26	.12	.22	.19
End Ex	34.32	33.64	32.09	33.72	33.34	32.75
SE	.12	.20	.32	.16	.28	.24
PostEx 10'	34.25	33.56	34.72	33.95	34.49	33.84
SE	.14	.24	.16	.12	.22	.16
20'	34.36	33.52	34.78	34.26	34.64	33.60
SE	.09	.23	.14	.20	.20	.14
30'	34.41	33.63	34.75	34.36	34.73	33.38
SE	.10	.22	.11	.19	.21	.13
40'	34.46	33.66	34.76	34.48	34.85	33.25
SE	.09	.22	.12	.19	.21	.16
50'	34.49	33.65	34.81	34.54	34.73	33.25
SE	.07	.23	.12	.15	.18	.19
60'	34.54	33.78	34.76	34.64	34.82	33.20
SE	.11	.05	.10	.32	.21	.31

(‡ -indicates significant difference from each separate PreEx period, $p>0.05$)

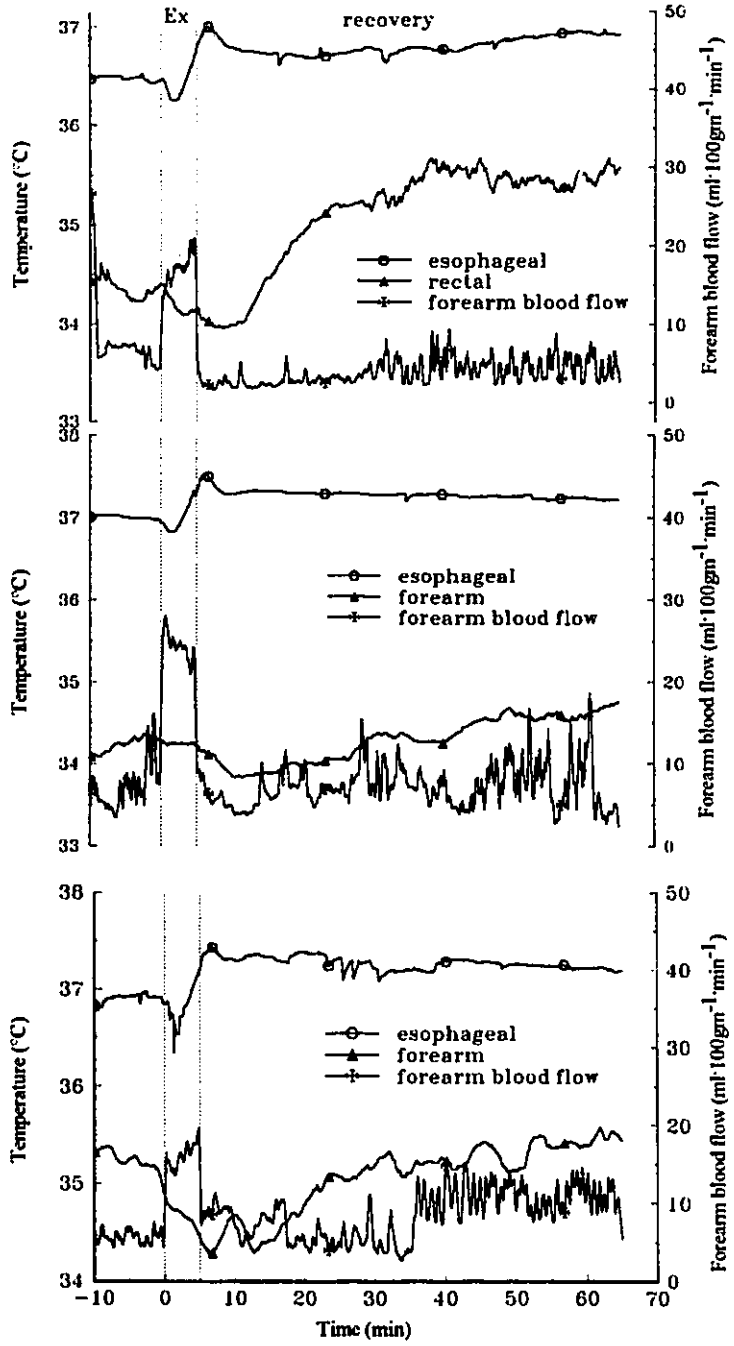

Fig. 20. Esophageal (O) and forearm temperatures (Δ) and forearm blood flow (✠) for individual subjects from rest through the start and end of exercise (10-13 METS) and during leaning recovery (Exercise performed for 5 min). Forearm surface blood flow measured by Laser-Doppler flowmetry. (Rest and recovery ambient temperature, 29°C; relative humidity, 50%).

6.3.4 Discussion

This experiment was designed to investigate the PostEx Tes response following an elevation of Tes to a level which was below previously measured values of Tdil. Tdil measurements during Ex1 of intermittent exercise and exercise of Ex/H_2O treatment demonstrated that Tdil occurred at 6.1 min and 6.3 min into exercise. Individual SBF measurements (fig. 20) did not show an upward inflection of SBF consistent with active vasodilation. Therefore, a 5 min period was considered to be short enough to avoid initiating vasodilation.

It was demonstrated that exercise performed to a Tes greater than Tdil resulted in a significantly elevated PostEx Tes. The observation that the post-exercise Tes plateau was comparable to the esophageal temperature equivalent for surface vasodilation indicated the possibility that Tco regulation is maintained by a range of "normal" temperatures with an upper limit defined by the thermolytic reflex threshold of surface vasodilation. Therefore, Tco within this range appears to be passively regulated. The performance of a 5 min exercise, which was shown not to cause active vasodilation, was expected to result in a sustained elevation. It was shown that PostEx Tes was stable and elevated 0.3°C above PreEx values for the full recovery period. In addition, the rate of exercise heat gain was estimated to be 33°C·min^{-1}, which resulted in approximately 9-10% of the total estimated exercise heat gain (470°C·min^{-1}) induced by 15 min exercise. The PostEx Tes elevation following 5 min exercise was equal to approximately 60% of the Tes elevation following 15 min exercise (based on comparisons with Ex1 of intermittent exercise).

These data suggest that the heat load developed during an exercise exposure time which was less than that required to achieve Tdil was not actively regulated and that regulation of Tco in recovery was primarily passive. While the system had the potential to deal with such a small heat gain, the results suggest that this potential was not accessed

during the recovery period. The data provides further support for the conclusion that Tco regulation is primarily passive within a range of "normal" temperatures which have been identified as an upper and lower limit of a null zone (Mekjavic et al. 1991).

6.3.5 Conclusion

Regulation of Tco following exercise was devoid of vasodilation reflex during recovery. The fact that the PostEx Tes plateau was generated without an elevation of Tes above Tdil suggests that the PostEx Tes regulation may have been defined by a threshold temperature equal in magnitude to Tdil.

Chapter 7

7.0.0 Discussion and conclusions

7.0.1 Discussion

Despite extensive reviews by Hammel 1968; Brengelmann 1973; Cabanac 1975; Nadel 1977; Bligh 1978; Bligh 1979; Bligh 1984; Fortney and Vroman 1985, there is still conflicting information on the mechanism of human temperature regulation. The "set-point" theory has generally been accepted as the working model of temperature regulation. However; the evidence to support it is not without opposition by those supporting a null-zone concept of temperature regulation (Benzinger et al. 1961; Jessen and Ludwig 1971; Bligh 1978; Mekjavic and Bligh 1989; Mekjavic et al. 1991) or the core-shell concept introduced by Aschoff and Wever (1958) and supported by Houdas et al. 1973, Webb et al. (1991), Ducharme et al. (1991), Giesbrecht and Bristow (1992) and Bristow et al. (1993).

The post-exercise core temperature elevation presented in this study, is another example of results that contradicts current set-point theory (Thoden et al. 1994a; Thoden et al. 1994b). A post-exercise hyperthermia was present in spite of a return to pre-exercise values of skin temperature, forearm surface blood flow, oxygen consumption , and the absence of a noticeable sweating response. Sustained elevations of core temperature were observed at different ambient temperatures (Kenny et al., 1993), exercise intensities (Kenny et al., 1993) and under different levels of insulation (Reardon et al., 1992). Other initial data in this study reported that the post-exercise esophageal temperature is quantitatively similar to the esophageal temperature at which skin vessels dilate during exercise, thus suggesting a link between the post-exercise esophageal

temperature elevation and thermal reflex activity (Thoden et al. 1994a). These observations led to the hypothesis that there was some residual influence related to exercise that retained the modulation of thermal reflex thresholds during recovery.

The fact that core temperature seems to be modulated sufficiently to maintain an elevated steady state temperature rather than to reestablish resting core temperature would suggest that either the system has been compromised or it has adopted a new reference point. If there is more than adequate ability to both dissipate heat and supply muscle blood flow, it is difficult to avoid postulating that the effector mechanisms are being controlled in a way which is consistent with a change in sensitivity or a change in hypothalamic set-point. The hypothalamic set-point is theoretically visualized as very stable and subject to acute adjustment only by the pyrogens accompanying fever while acclimatization (Sawka and Wenger 1988), circadian rhythms (Sagot et al. 1987; Sawka and Wenger 1988; Zahorska-Markiewicz et al. 1989; Alfoldi et al. 1990; Cisse et al. 1991; Fedorova et al. 1992; Deswasmes et al. 1994) and menstrual cycle (Sawka and Wenger 1988; Frascarolo et al. 1992) adaptations are more gradual.

Nielsen in 1938 showed that with increases in ambient temperature, reductions in heat loss by radiation and convection (dry or sensible heat loss) were exactly balanced by an increase evaporative cooling (insensible heat loss). Nielsen concluded that core temperature during exercise increased as a consequence of a change in the regulatory adjustment of body temperature and not of an insufficient ability to dissipate heat, as had been previously thought. His conclusions appear to have often been understood to mean that the body's thermostat is re-set at a higher level during exercise comparable to a fever response or equivalently that the core temperature threshold for the thermoregulatory effectors are elevated, as they are in fever. While it has been proposed that muscular activity may "reset" the temperature that the hypothalamus attempts to maintain, the majority of evidence supports that temperature elevation during exercise simply reflects an

uncompensated thermal error which is reversed when muscular demand for blood flow is terminated (Sawka and Wenger 1988).

If a positive adjustment of set-point had occurred, a similar response to fever would have been expected. A transitional period following the shift in set-point would have resulted in an increase in resting metabolic rate and skin temperature (Briese and Cabanac 1990; Riedel 1990). Data from the initial stage of this study showed that oxygen consumption had returned to pre-exercise resting levels within 5-10 min after moderate intensity running and skin temperatures reached pre-exercise values within 10-15 min. The observation of an elevated muscle temperature during exercise recovery limits any significant discussion on the possibility an elevated post-exercise set-point. In addition, the post-exercise core temperature elevation was reversed upon immersion in a water bath at 29°C (Appendix E). It is also possible that the residual heat may itself promote and maintain the elevated core temperature. If this were the case, the return of core temperature to PreEx following cool water immersion would support a core-shell model of temperature regulation. In either case, there has been little evidence, other than the work of Nielsen and Nielsen (1962) and Haight and Keatinge (1973), to suggest that exercise causes an elevation of set-point comparable to a fever response. In fact the evidence has been inconclusive on this issue and leaves room for speculation.

Jackson and Hammel (1963) demonstrated, using hypothalamic temperature displacement by direct thermode cooling and heating of the hypothalamus during treadmill exercise in dogs at different ambient temperatures, that set-point actually fell 0.8°C. Similarly, Tam et al. (1978) concluded that exercise lowers the reference core temperature of sweating in man, which would seem to support a set-point lowering theory. On the other hand, Benzinger et al. (1961) reported no change in set-point as did Cabanac and Massonet (1977). Despite the inconsistent findings, there is one common factor which must be considered in evaluating the importance of their conclusions. The studies all measured changes in thermal reflex threshold response as an indication of hypothalamic

set-point activity. If the present study was to follow the same procedure, it could be concluded that there is sufficient evidence to support an elevation of set-point based on the relationship established between the esophageal temperature at the time of surface vasodilation and post-exercise esophageal temperature.

Effects of thermal and non-thermal reflexes on the thresholds for sweating and vasomotor control are well documented from exercise research. For example, it has been demonstrated under a number of different conditions that there are changes in the threshold temperatures for sweating and vasodilation such as varying levels of work intensity (Hirata et al. 1983,1984; Smolander et al. 1987; Smolander et al. 1991), ambient temperature (Nadel et al. 1971; Wenger et al. 1975; Roberts and Wenger 1980; Taylor et al. 1984; Smolander et al. 1987), exercise posture (Johnson et al. 1974; Johnson and Park 1981; Roberts and Wenger 1980), exercise duration (Kenney et al. 1991), state of hydration (Nadel et al. 1980), fitness level (Nadel et al. 1974; Roberts et al. 1977) and acclimation (Nadel et al. 1974; Roberts et al. 1977; Hessemer et al. 1986). Johnson and Park (1981) demonstrated that thresholds for sweating and vasodilation were significantly different between rest and exercise. Therefore, it could be speculated that during exercise hypothalamic set-point is not changing but rather the core temperature threshold for thermal reflex control is changed in recovery as a function of some exercise related effect whether thermal or non-thermal in origin. A post-exercise effect on thermal threshold control has not been previously demonstrated. However; the observation of a relation between exercise dilation temperature and the post-exercise elevated esophageal temperature may be sufficient evidence to support this possibility.

Could the post-exercise elevation be an indication of a change in set-point thermosensitivity as a result of an exercise induced elevation of core temperature? The load-error theory contends that as core temperature rises during exercise, a load error signal develops which is proportional to the difference between the core temperature and hypothalamic set-point temperature. As the magnitude of the load-error increases, thermal

reflexes are triggered which attempt to maintain or lower core temperature towards set-point. Therefore it is equally plausible that some residual effect of exercise, whether neural, humoral or neuroendocrine has altered the thermosensitivity of the hypothalamus to an elevated load-error.

There is one further possibility for explaining the results of this study. During exercise, according to the load-error theory, core temperature rises a certain amount without any active defence other than the sensible heat loss component (i.e. convective and conductive heat loss). Therefore, there is a period of time during which a core temperature displacement results in no active recruitment of additional thermal responses from rest. This temperature displacement has been referred to by some as the restricted-band or null-range (Cabanac 1975; Mekjavic and Bligh 1989). These terms are used to identify the existence of a range of temperatures within which fluctuations of core temperature occur without recruitment of either sweating or shivering responses.

Bligh (1978) discussed two temperature control points. These are the hypothalamic set-point and null-point. The hypothalamic set-point is considered the hypothalamic regulatory temperature about which core temperature regulation is maintained while the null-point refers to the temperature at which thermal reflexes are initiated at a given core temperature. Under normal resting conditions, the set-point and null-point are essentially identical and undergo a similar magnitude of change in cases like fever response. Under certain conditions, as with a hibernating animal, the null-point is variable. For example, during hibernation the thresholds for heat production and heat loss move in opposite directions, most probably as a result of powerful inhibitory influences on both cold and warm temperature sensitive neurons of the hypothalamus (Bligh 1978). Yet it is suggested that the set-point remains fixed. The obvious benefit to this would be in conserving energy during the period of hibernation. The widening of the null-point results in a null-range of temperature control.

Benzinger et al's. (1961) findings support a null-range theory of temperature regulation. Their demonstration of changes in threshold response as a function of changing skin temperatures have often been referred to as a change in set-point. Their data demonstrate that there is a range for the core temperature threshold of sweating and shivering for skin temperatures between 28-32°C equal to 0.5°C. As skin temperatures increase, the range of temperature difference is depressed. On the other hand, the evidence has not been altogether supportive of null-range temperature concept. In fact Cabanac and Massonet (1977) found that whole-body heating (38°C) and subsequent cooling (28°C) by water immersion did not result in significantly different threshold temperatures for sweating and shivering. However, Mekjavic and Bligh (1989) repeated this protocol and added exercise in order to elevate core temperature by a different heat source. They found inconclusive evidence to support either a set-point or null-range concept. Part of the difficulty they experienced can be explained by the very different skin temperature stimulus and response attributed to the different protocols (i.e. whole-body heating by water immersion at 40°C; exercise at 50% VO_{2max} at an ambient temperature of 22°C; both followed by whole-body cooling at 28°C for each condition) which resulted in significantly different rates of cooling. Mekjavic et al. (1991) attempted to eliminate the effect of variable skin temperatures by having the subject exercise in a 28°C water bath and then recovering the subject in the same water temperature in order to keep skin temperature constant at a cool temperature. Their results were significant in that they showed a difference of 0.6°C between the threshold temperature for sweating and shivering.

The evidence supporting either a set-point or null-range theory of temperature regulation has not necessarily been strong for either concept. However, part of the problem is probably related to the rather complex nature of the operating system as discussed earlier. But there is also the problem of interpreting the information. Interpretation of changes in temperature response is made more difficult by the fact that

changes in either core or skin temperature result in a change of one or more of many other variables which can not be controlled. Therefore, part of the inconsistency demonstrated in theories of temperature control may be the result of not controlling or taking all possible inputs into consideration.

Based on the data of this study it is plausible that the thermoregulatory system either: 1) had become satisfied that thermal homeostasis had been reestablished at a higher temperature after exercise, thus indicating a hypothalamic set-point change, 2) other non-thermal influences had affected the operational temperatures for thermal reflexes (i.e. blood pressure, osmolarity or plasma volume changes), or 3) operated on a less well defined hypothalamic set-point than is usually described, as indicated by observations that there are separations of or a "normal" range of temperatures between thermal reflex thresholds for defending against heating and cooling . Two important observations from these data support the "range of 'normal' temperatures" hypothesis. The first observation was the rather passive nature of the thermolytic mechanism towards a significant elevation of esophageal temperature (0.5°C or greater) for a period in excess of 60 min. This effect was reversed upon immersion in a water bath at 29°C (Appendix E). There was no shivering response as core temperature fell below pre-exercise, but shivering would have been expected with a set-point elevation. Secondly, a similarity was identified between the exercise esophageal temperature at which skin surface dilation occurred and the post-exercise esophageal temperature. The dilation temperature identified during exercise marks the increase in skin blood flow (specifically the vasodilation of surface vasculature) that is induced by a rising core temperature and therefore represents a hypothalamic-driven defence against a further elevation in core temperature. Therefore it was concluded that the passive response of thermal control systems towards the elevated core temperature during recovery considerably strengthens the idea of a range of "normal" temperature, within which the thermoregulatory mechanisms are relatively insensitive to

core temperature fluctuations. If this were not the case, one should expect a gradual but continuous elimination of any residual heat source.

These observations lead to the hypothesis that there was some residual influence related to exercise, and not whole body heat content, that retained the modulation of thermal reflex thresholds during recovery. The data eliminate whole body heat content changes as the primary cause of the core temperature elevation and suggest that changes in core temperature by endogenous and exogenous heating are regulated by different thermoregulatory mechanisms. This observation is not unique to this study. Mekjavic and Bligh (1989) found a similar difference in their study which they attributed to the very different skin temperature profile resulting from the heating protocol of exercise versus water immersion. Although these differences also occurred in this study, it brings into question whether it was skin temperature changes or in fact an exercise effect that maintained the modulation. Their results show that during the exercise protocol they were unable to demonstrate a shivering response despite an extended exposure to the cool 28°C water, while this problem was not experienced following exogenous heating in 40°C water. It is therefore possible that the same exercise related effect that produced post-exercise modulations in the present study caused a change in the threshold for shivering that would have required an even lower core temperature before shivering was demonstrated.

The physiological importance of esophageal temperature at the time of surface dilation in defining upper limits of resting temperature can not be completely described at this point. However, the physiological relationship of the dilation temperature to the post-exercise esophageal temperature suggests that neuro-muscular activity significantly influences thermolytic controls which persist in recovery. That the dilation temperature may represent the upper limit of a range of "normal" temperatures is further supported by data from the 5 min exercise performed to an esophageal temperature elevation below

dilation temperature. Within minutes of exercise termination esophageal temperature achieved a stable elevation (0.3°C or greater) that was maintained with no change over 65 min of recovery.

Clearly, the consistent relationship demonstrated between the dilation temperature and the post-exercise esophageal temperature following both single or repeated exercise and the stable elevation in recovery following 5 min exercise supports the hypothesis that temperature regulation involves a range of temperatures within which core temperature is passively regulated. A range of temperature in which regulation is without thermal reflexes would imply that the thresholds for temperature defence, sweating and vasodilation at the upper end and shivering and vasoconstriction at the lower end, are significantly different from each other. Only when core temperature is elevated above or reduced below the threshold temperature are the thermal reflexes activated. Within the range of temperatures defined by the thermal thresholds, sensible heat loss (i.e. convective and conductive heat loss) would maintain core temperature regulation about a hypothalamic set-point temperature, but be very slow in responding to temperature changes.

The data from this study support a range of normal core temperature regulation, however; the data does not dispute a set-point in temperature regulation but rather its role in explaining core temperature changes during exercise and recovery. Incorporating both the set-point and null-range concepts into a model of temperature regulation model is plausible. It is well documented that the thermal threshold response is influenced by a number of factors including exercise, posture, ambient temperature and many others. In this model, the range of temperature thresholds would be variable as a result of either thermal and/or non-thermal reflexes while the hypothalamic set-point remains stable and only under conditions of fever, where there is a direct pyrogenic effect on the hypothalamus, would it be expected that set-point would change. Similarly, this model would also incorporate the load-error concept where an elevation in core temperature

results in a threshold temperature at which thermolytic mechanisms respond. The temperature elevation required to achieve this activation or thermal defence would represent the upper range of temperature elevation. Although the existing data suggest inconsistencies with current temperature regulation models it is possible that the null-range and set-point concepts are not mutually exclusive and future models should consider both concepts in their design.

7.0.2 Conclusion

Testing of the following hypotheses gave rise to the accompanying conclusions.

1) The homeothermic defence mechanism that incorporates reflexive control of skin vasculature becomes inoperative during exercise recovery at a temperature above resting values, with the result that Tco becomes elevated and stable for extended periods at an elevated level (4.0.0).

a. PostEx Tes reached and held an elevated plateau for up to 65 min following exercise.

b. The PostEx Tes plateau was equal in magnitude to Tdil during exercise.

2) Tes at the time of skin vasodilation during exercise measured by Laser-Doppler flowmetry (LDF) is comparable to measurements by temperature gradients between forearm and finger (Tfa-Tfi gradient) using surface thermistors (chapter 5.0.0).

a. LDF measurements support that there was a PostEx Tes relationship to Tdil during exercise.

b. LDF and Tfa-Tfi gradient methods result in the same value of Tes at the time of reflexive vasodilation during exercise.

3) An increase in the PreEx Tco will result in an elevated exercise Tdil (6.0.0).

a. the Tes at the time of active vasodilation increased with an elevation of PreEx Tco.

4) An increase in the PreEx Tco will result in an a higher PostEx Tes plateau (6.0.0).

a. the PostEx Tes plateau increased with an elevation of PreEx Tco.

5) There is a direct relationship between the exercise Tdil and PostEx Tes with successive exercise (6.0.0).

a. The PostEx Tes plateau was equal in magnitude to Tdil during each exercise.

6) that an elevation of Tco above Tdil by an exogenous heat load (water immersion) will not result in a PostEx Tes plateau (6.1.0).

a. PostEx Tes plateau was not significantly elevated above PreEx Tes values following an increase in Tes above Tdil.

7) A PostEx elevation of Tes following exercise will not be further increased following an exogenous heat load (6.2.0).

a. Post-treatment Tes did not demonstrate a significant elevation from pre-treatment Tes value.

8) An increase in Tco to a value below Tdil will result in a sustained PostEx Tes plateau (6.3.0).

a. PostEx Tes remained elevated above PreEx Tes value for the full period of recovery.

8.0.2 Future consideration

Observation of a PostEx thermal disturbance of thermal homeostasis generates a number of questions about the way body temperature is regulated. The present study documents changes in PostEx Tco which are inconsistent with a traditional SP_{hy} mechanism. Based on the PostEx Tes elevation which was comparable to exercise Tdil and the lack of an elevation following endogenous heating, it was suggested that Tco regulation is based on a range of "normal" temperatures. However, specific factors contributing to this disturbance could not been identified. These data serve as the basis for investigation of fundamental questions about the interactions between thermal reflexes and the non-thermal reflexes of exercise and their effects on thermoregulatory control.

Topics for subsequent investigations include:

1) assessment of resting thresholds for shivering, sweating and vasomotor control with and without exercise and with and without shivering should establish if the exercise of voluntary motor activity and of shivering have similar or different effects on thermal reflexes.

2) separation of the roles of cutaneous vasoconstrictors and active vasodilators should give the basis for isolating the effects of PostEx factors.

3) experimentally isolating the PostEx effects of baroreceptor-related and chemically related influences on thermal reflexes should provide further information for evaluating whether temperature regulation is variable or simply subject to interaction with other vegetative reflexes.

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Appendix A

Phase 0 - Preliminary intermittent exercise protocol

The following shows individual data of Tes recordings during an intermittent exercise protocol (fig. 1). Subjects were acclimated in a temperature control room for one hour. Three exercise/recovery cycles were performed - 10 min exercise followed by a 10 min recovery. The recovery was extended to 30 min following the third exercise. The experiment was conducted at 32°C 45% humidity. The data demonstrate the elevation of Tes following each exercise period. However; the effect of swallowing on the Tes recordings and the short recovery period made it difficult to interpret changes in the initial two recovery periods.

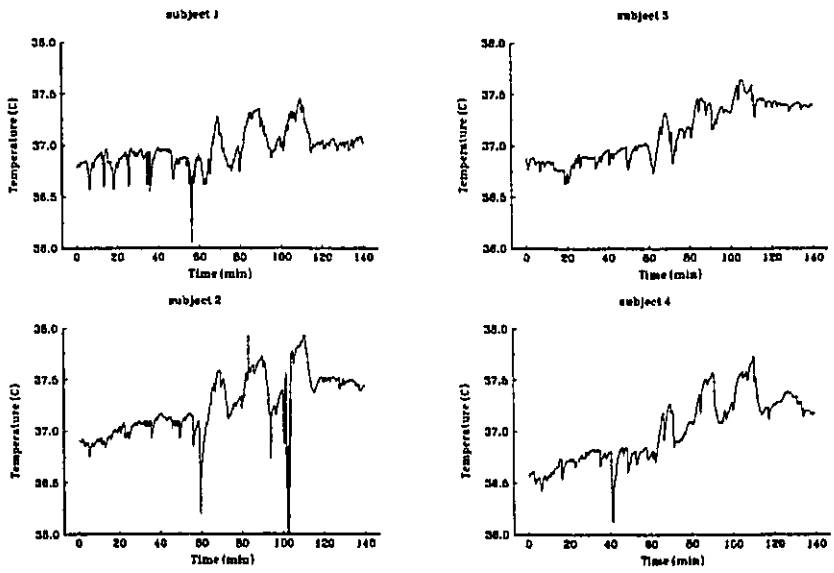

Fig. 1. Intermittent exercise esophageal temperature response from rest through the start and end of exercise (10-13 METS) and standing, leaning recovery for individual subjects. (Ambient temperature, 32° C; relative humidity, 45%).

Appendix B

Investigation of resting core temperature changes under controlled conditions

Introduction

In order to investigate Tco changes under controlled conditions subjects were asked to participate in a study where they were required to stay overnight in the laboratory (fig. 2). All subjects reported to the laboratory at 7 p.m. the evening before. Rectal probes were inserted and the subjects were placed in the thermal chamber at 29°C 50% humidity. The room was equipped with a television, desk, and bed as well as refreshments to ensure that the subject was comfortable.

Results

The changes in Tre showed that following entry into the chamber there was a gradual fall in the Tre. During the first 6 hours in the chamber Tre fell approximately 1.3° C (4 a.m.) where it was maintained for approximately 2 hours (4 am to 6 am) with minimal fluctuation (0.1-0.3°C). Tre began to rise between the hours of 6 am and 7 am which most likely reflects the period of time the subject began to awaken. The following figure shows repeated trials. Changes in Tre were consistent for all trials. These data demonstrate the importance of controlling subject activity prior to the test morning.

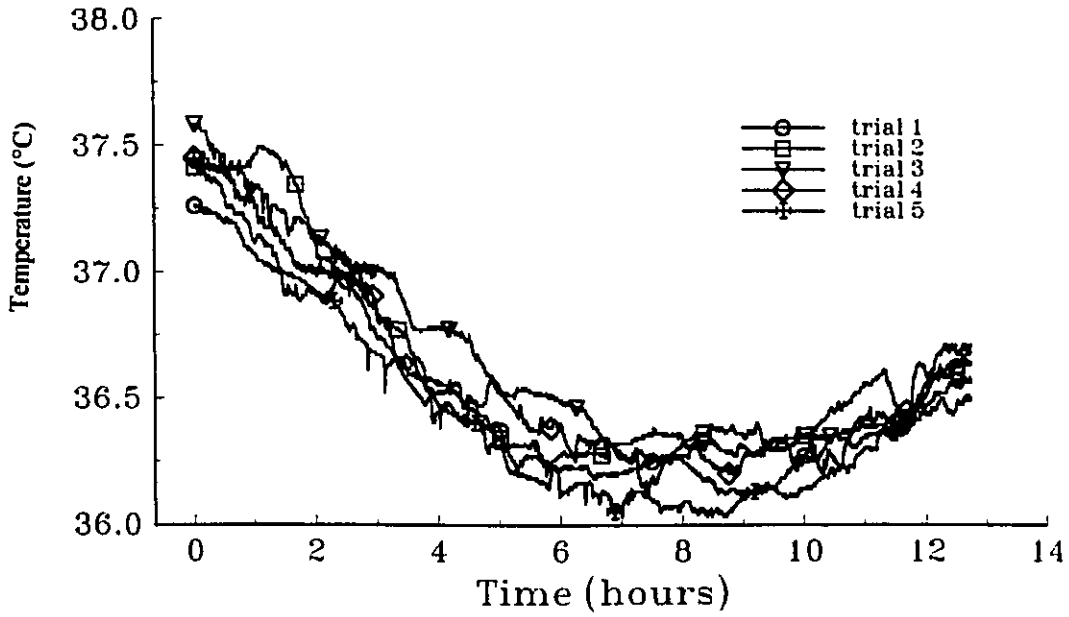

Fig. 2. Repeat rectal temperature response of one subject during an overnight control study. Subject entered the temperature control chamber at 9 pm and the test was completed at 9 am the following morning. Subjects remained inactive during the 12 hr period. (Ambient temperature, 24°C; relative humidity, 50%).

Appendix C

Investigation of muscle temperature

Introduction

Changes in Tco showed a stable elevation for a prolonged period of time. The elevation suggests a residual heat source, most likely originating from the previously active muscle. The following experiment was conducted to measure deep muscle temperature and examine muscle temperature chronology during acclimation, exercise and recovery.

Method

The subjects entered the thermal chamber at 29°C 50% humidity for a 1 hr acclimation period wearing only shorts and running shoes. At 10 min PreEx, the subject stood and was prepared for a 15 min exercise period. Treadmill running exercise was chosen to stimulate about 75% of the subjects VO_{2max} (12 METS). Recovery data was collected for a 30 min period.

Muscle temperature (Tmu) was measured at 3 depths in the thigh (upper leg) about midway between and lateral to a line joining the anterior superior iliac spine and the superior aspect of the center of the patella. This point was chosen as it represents a considerable muscle mass well lateral to the femoral vein, artery and nerve. The skin, subcutaneous tissue and muscle were anesthetized by infiltrating ~ 2 ml of 1% lidocaine without epinephrine to a depth of 45 mm with a 25-gauge needle inserted perpendicular to the skin. An 18-gauge, 45-mm-long polyethylene catheter (Cathlon, Critikon Canada,

Markham, Ontario) was then inserted in the anesthetized tract to its full length and the stylet removed. A sterile Teflon coated multisensor temperature probe (1 mm diameter, IT-17:3, Physitemp Instruments, Inc., Clifton, New Jersey, USA), was then introduced until the end of the probe reached the end of the catheter. This distance was verified by an external mark on the multisensor probe. The probe fit snugly within the polyethylene catheter and the space between the probe and catheter hub was sealed by soft, sterile bone wax. The catheter and multisensor probe assembly was then secured to the skin by a waterproof transparent dressing and waterproof tape (procedures from Giesbrecht and Bristow 1992). Each multisensor probe had thermocouples at 15 (T_{mu15}), 30 (T_{mu30}), and 45 (T_{mu45}) mm from the skin surface. Measurement was limited to one volunteer subject.

Results

At the beginning of the acclimation period Tmu was 1.5, 0.8 and 0.6 °C lower than Tes (37.0°) for T_{mu15}, T_{mu30} and T_{mu45} respectively. Tmu increased gradually to a PreEx Tmu 0.2°C greater than Tes. Tes fell gradually from the start of the acclimation period reaching a stable temperature at 45 min equal to 36.7°C. At the onset of exercise Tes fell slightly after which it showed an upward inflection followed by a downward inflection at approximately 6 min into exercise. Tmu increased immediately with deep muscle demonstrating the largest rise (fig. 3).

Tmu at cessation of exercise was equal to 39.7, 39.7 and 40.3 °C for T_{mu15}, T_{mu30} and T_{mu45} respectively. End-test Tes was equal to 38.4°C, falling quickly in the first minutes of recovery to an elevated plateau 0.5°C above PreEx value. T_{mu15} and T_{mu30} demonstrated a very similar trend, falling quickly in the initial minutes to an

elevated plateau at 15 min equal to 38.7°C. T_{mu45} fell gradually over the full period of the recovery but did not demonstrate a plateau late in recovery.

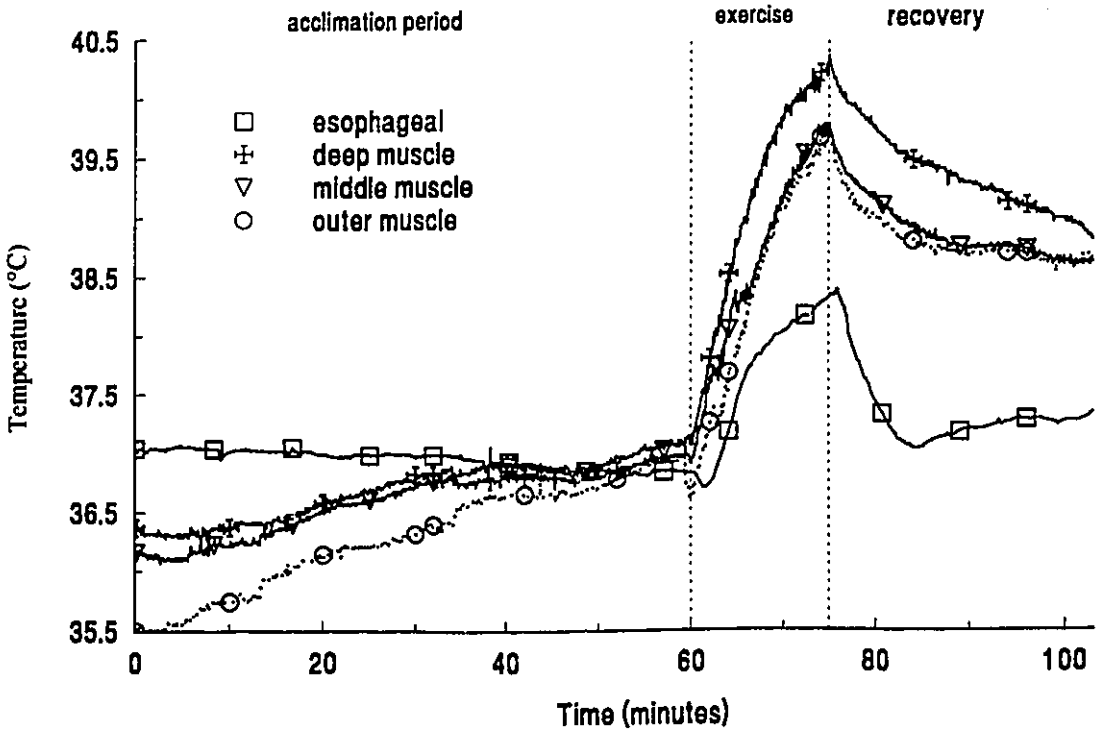

Fig. 3. Esophageal and muscle temperature response from rest through the start and end of exercise (10-13 METS) and standing, leaning recovery for one subject. Muscle temperature measured in the thigh at 3 different depths from the skin surface, deep muscle 45 mm (+), middle muscle 30 mm (∇), outer muscle 15 mm (O), and esophageal temperature (□). (Ambient temperature, 29°C; relative humidity, 50%).

Discussion

The primary observation was that the PostEx Tes and Tmu remained elevated for a prolonged period. Therefore, the elevated Tmu could be considered a heat source for maintaining Tes at an elevated plateau. However, a plateau in Tmu occurred at a similar time to Tes (except T_{mu45}) which suggests that the rate of heat loss at both sites was reduced. These observations support our observations of a reduced thermal activity in PostEx demonstrated as a return of Tsk, SBF and VO_2 back to PreEx within 10-15

minutes. If Tmu was the heat source which maintained Tes at an elevated plateau it would be expected that Tmu would gradually return to PreEx values.

Conclusion

These data suggest that the post-exercise elevation of Tes may be maintained at an elevated level as a result of a continual warming of central blood volume heat gain from residual muscle heat. However, further research on muscle temperature profile following exercise is required to conclusively determine the impact of high post-exercise muscle temperature on core temperature regulation during exercise recovery.

Appendix D

Forearm-finger gradient measurements using an intermittent exercise protocol

Introduction

Intermittent exercise was performed to evaluate Tdil measurement using Tfa-Tfi gradients (Sessler and Rubenstein 1990). The technique had been used to demonstrate a relationship between Tdil and PostEx Tes in continuous exercise (Thoden et al. 1994) but has not been previously used for intermittent exercise.

Specific methods

Six subjects performed 3 cycles of 15 min exercise and 20 min recovery. The subjects were randomly assigned to each of the conditions. Their average age, height and weight were 22±1 yr, 177±1 cm and 82±3 Kg respectively. Measurements of internal temperature were monitored by Tes and Tre, and Tsk measurements (including Tfh, Tch, Tfi, and Tfa). HR was measured continuously for the full period of the experiment. VO_2 was measured 10 min PreEx, during exercise until 15 min into recovery. The subject entered the thermal chamber for a 1 hr acclimation period at 29°C, 50% humidity wearing only shorts and running shoes. At 10 min PreEx, the subject stood mounted on the treadmill and inserted the two-way breathing valve for VO_2 gas collection. Treadmill running exercise was chosen to stimulate about 75% of the subject's VO_{2max}. Following cessation of exercise the subject stood for 20 min until end of recovery. This exercise-rest cycle was repeated for an additional two periods.

Results

PreEx Tco remained stable (36.9°C for Tes and 37.1°C for Tre) for the 10 min period prior to exercise (table 1). At the onset of exercise, Tsk fell quickly. The response profile was identical to previous experiments. Tes fell 0.2°C in the first 2.0 min of exercise followed by a rise of 0.18°C·min^{-1} (10.8°C·hr^{-1}). Tre did not show a similar drop and began to rise immediately at the onset of exercise at a rate of 0.09°C·min^{-1} (5.6°C·hr^{-1}) which was essentially maintained for the exercise period (fig. 4). At 6.5-7.5 min into exercise Tes showed a downward inflection which was preceded by an inflection of Tsk. Heat loss from the skin resulted in a reduction in the rate of rise of Tes following inflection equivalent to a 52% drop in the rate of heat gain.

Table 1. Mean core temperatures - intermittent exercise (pilot).

	Exercise 1		**Exercise 2**		**Exercise 3**	
	Tre (°C)	**Tes** (°C)	**Tre** (°C)	**Tes** (°C)	**Tre** (°C)	**Tes** (°C)
PreEx 10'	37.08	36.88				
Start	37.07	36.87	37.82	37.36	38.19	37.48
End Ex	37.88	37.95	38.55	38.45	38.82	38.73
PostEx10'	37.87	37.34	38.40	37.46	38.58	37.67
20'	37.82	37.34	38.19	37.48	38.26	37.65

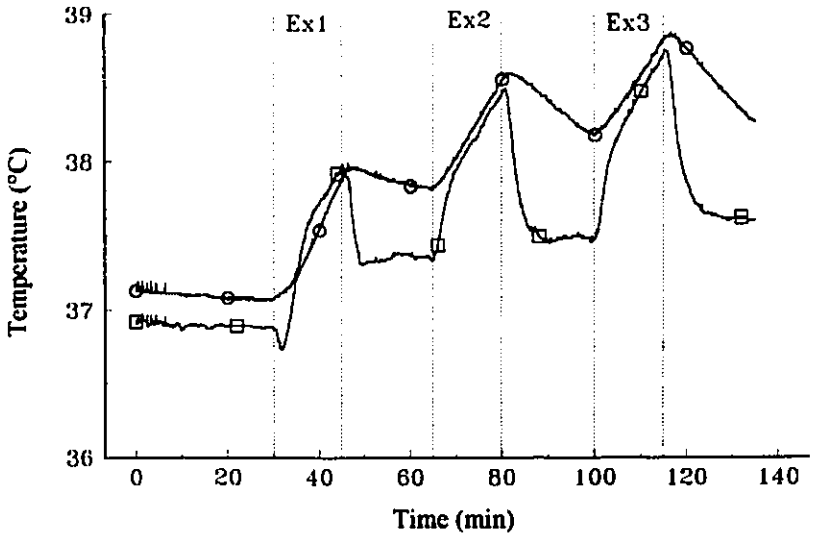

Fig. 4. Men intermittent exercise core temperature response from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery. Rectal temperature (O) and esophageal temperature (□). (Ambient temperature, 29°C; relative humidity, 50%; n=6).

At the end of exercise Tes fell precipitously. Approximately 4.0 min into recovery, Tes reached a minimum temperature after which it increased slightly to an elevated sustained plateau at 37.4°C, a 0.5°C elevation from PreEx that was maintained for the full recovery period. Tre showed a persistent elevation in recovery with only a gradual fall equal to 0.2°C·min^{-1} (1.2°C·hr^{-1}) . Tsk returned to PreEx values within 10-15 min of recovery with the exception of Tfi and Tth (table 2).

Table 2. Mean surface temperatures - exercise period 1(pilot).

	Tfh (°C)	**Tch** (°C)	**Tfi** (°C)	**Tfa** (°C)	**Tth** (°C)	**Tca** (°C)
PreEx 10'	34.68	33.73	34.06	34.22	33.53	32.11
Start	35.47	34.59	34.14	34.72	33.78	32.23
End Ex	35.47	34.59	34.14	34.72	34.63	34.66
PostEx 10'	34.87	34.23	35.45	34.55	35.08	34.42
20'	34.47	33.70	35.12	34.35	33.86	34.77

Similar changes in Tco and Tsk chronology were observed for the second (Ex2) and third (Ex3) exercise periods. In contrast to Ex1, Tes did not fall at the onset of Ex2 but showed a delay before a measurable increase was noted. At this point Tes increased at a rate of 0.15°C·min^{-1} (9.3°C·hr^{-1}) which was slightly lower than during Ex1 but not significantly different. The magnitude of the fall of Tsk at the onset of Ex2 was less than Ex1 with a significantly shorter period of time before the inflection upward of Tsk, 2.1 min as compared to 5.9 min for Ex1. The inflection upward in Tsk was followed a short time later by the inflection in Tes. The effect of increased surface flow was to reduce the rate of rise of Tes to 0.05°C·min^{-1} (3.0°C·hr^{-1}). Tre began to rise immediately reaching a constant rate equal to 0.06°C·min^{-1} (3.6°C·hr^{-1}).

Following the second end of exercise Tes fell precipitously to an elevated plateau equal to 37.5°C. Tre showed a gradual fall over the recovery which was greater than Ex1 equal to 0.3°C·min^{-1} (1.8°C·hr^{-1}) . Tsk returned to PreEx within 10 min of recovery (table 3).

Table 3. Mean surface temperatures - exercise period 2 (pilot).

	Tfh (°C)	**Tch** (°C)	**Tfi** (°C)	**Tfa** (°C)	**Tth** (°C)	**Tca** (°C)
Start	34.47	33.70	35.10	34.35	33.86	32.11
End Ex	35.85	34.06	34.99	34.96	34.63	34.66
PostEx 10'	35.18	33.55	35.38	34.75	35.08	34.42
20'	34.84	32.89	35.41	34.61	34.92	34.00

At the onset of Ex3, Tsk fell reaching a minimum temperature in 2.0 min. Tes remained stable during the first 1.3 min of exercise after which it increased at a rate of 0.17°C·min^{-1} (10.2°C·hr^{-1}) while Tre began to rise immediately at the onset of Ex at a rate of 0.05°C·min^{-1} (3.0°C·hr^{-1}). Tsk inflection upward preceded the drop in the rate of rise in Tes to 0.06°C·min^{-1} (3.6°C·hr^{-1}). Both Tsk and Tco continued to rise throughout exercise.

Following cessation of exercise Tsk returned to PreEx values within 15-20 min (table 4) and Tes fell precipitously reaching a stable elevated plateau of 37.6°C that was maintained for the full recovery period (fig. 4). All PostEx temperatures were significantly ($p>0.05$) elevated above the initial PreEx Tes by 0.46°C, 0.60°C and 0.77°C for PostEx1, PostEx2, and PostEx3 respectively. Significant difference was found between PostEx1 and PostEx3 with no significant difference between PostEx1 and PostEx2, and between PostEx2 and PostEx3 (fig. 4).

Table 4. Mean surface temperatures - exercise period 3 (pilot).

	Tfh (°C)	**Tch** (°C)	**Tfi** (°C)	**Tfa** (°C)	**Tth** (°C)	**Tca** (°C)
Start	34.84	32.89	35.41	34.61	34.92	34.00
End Ex	36.09	33.76	35.32	35.16	35.02	34.83
PostEx 10'	35.42	33.59	35.57	34.87	34.97	34.64
20'	35.08	33.08	35.67	34.69	34.66	34.11

Table 5. Mean oxygen consumption and heart rate values - intermittent exercise (pilot)

	Exercise 1		**Exercise 2**		**Exercise 3**	
	VO_2 ($ml \cdot kg^{-1} \cdot min^{-1}$)	**HR** ($b \cdot min^{-1}$)	**VO_2** ($ml \cdot kg^{-1} \cdot min^{-1}$)	**HR** ($b \cdot min^{-1}$)	**VO_2** ($ml \cdot kg^{-1} \cdot min^{-1}$)	**HR** ($b \cdot min^{-1}$)
PreEx 5'	3.73	75	3.22	95	4.87	102
End Ex	41.53	167	42.75	179	43.10	184
PostEx 5'	5.10	99	5.47	113	6.23	117
10'	4.02	93	4.82	102	4.37	107

Measurements of Tdil for each exercise period were identified using the previously described methods of identification (Tsk inflection and Tfa-Tfi methods). The magnitude of increase of Tdil between exercise periods was comparable to the increase in PostEx Tes. Tdil measurements were 37.4°C, 37.6°C and 37.7°C with a PostEx Tes of 37.3°C, 37.5°C and 37.7°C.

Discussion

The primary observation of this experiment was that the Tes was more elevated following successive exercises. In each case the PostEx Tes was found to be comparable to the exercise Tdil measured by Tfa-Tfi gradient. However, our data demonstrated that Tdil measurement using Tfa-Tfi gradient method during the third exercise period was difficult to interpret, suggesting that this method would have to be supported with another technique (i.e. Laser-Doppler flow).

Appendix E

Investigation of PostEx whole-body cooling

Introduction

The following study was conducted to evaluate the effect of water immersion at Tw=29°C on the PostEx Tes stability.

Specific methods

Four subjects performed a 15 min exercise followed by a 15 min recovery after which they were immersed in a water bath (Tw 29°C). Measurements of internal temperature were monitored by Tes and Tre, and Tsk measurements (including Tfh, Tch, Tfi, and Tfa). The subject entered the thermal chamber for a 2 hr acclimation period at 29°C, 50% humidity wearing shorts only and running shoes. At 10 min PreEx, the subject stood and mounted the treadmill. Treadmill running exercise was chosen to stimulate about 75% of the subject's VO_{2max}. Following cessation of exercise the subject's stood for 15 min after which he entered a water bath (Tw 29°C) for a minimum of 30 min.

Results

The following graph shows the time-course response of Tre and Tes (fig. 5).

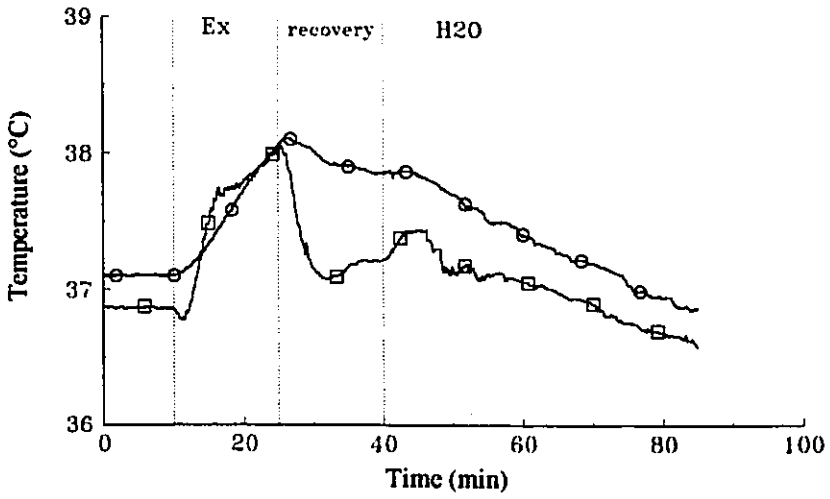

Fig. 5. Average core temperature response from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery followed by body cooling in a water bath (Tw 29°C). Rectal temperature (O) and esophageal temperature (□). (Ambient temperature, 29°C; relative humidity, 50%; n=4).

Discussion

Following immersion in the cool water bath, Tes began to drop immediately. The rate of fall for Tre and Tes were comparable. The results demonstrated that the PostEx Tes elevation was reduced. All subjects were removed from the water once pre-exercise temperatures were achieved. Interestingly, in those subjects who remained longer, Tco always dropped below Pre-Ex before shivering was noted by the subject. However, any conclusions on this observation are limited by the protocol and number of subjects used. This study demonstrated that the PostEx maintenance of Tco could be reversed.

Appendix F

Investigation of an exogenous heat load protocol

Introduction

A PostEx disturbance of thermal homeostasis was documented following both continuous and intermittent exercise. The following experiment was conducted to investigate a protocol to generate a non-metabolic heat load. The objective was to determine a water temperature that could produce a similar rate and magnitude of Tes elevation comparable to exercise. Different water temperatures (with single subjects) were tested; however only those data used to arrive at a final protocol are presented.

Specific methods

Five male subjects were required to sit in a water bath at 44^{o}C for 15 min. Measurements of internal temperature were monitored by Tes and Tre, and Tsk measurements (including Tfh, Tch, Tfi and Tfa). Measurement of SBF was done during pre-treatment until completion of the experiment. The subjects acclimated for a 2 hr at 24^{o}C, 50% humidity wearing only shorts and running shoes. At 1 min before treatment time the subject entered the water tank. The temperature of the water was maintained at 44^{o}C. The subject remained in the water tank for 15 min after which he was removed and taken into the thermal chamber for a 30 min recovery period.

Results

Data from this experiment are presented in table 6 and figures 6 and 7.

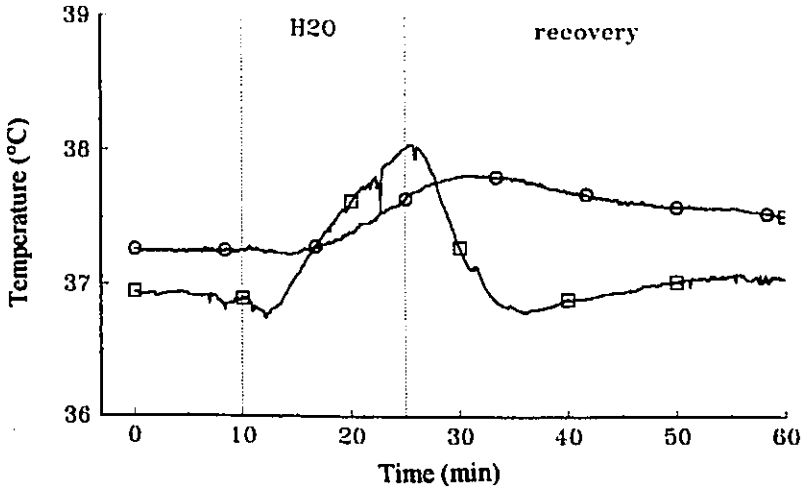

Fig. 6. Mean core temperature response from rest through the start and end of water immersion (Tw 44° C) and during standing, leaning recovery. Rectal temperature (O) and esophageal temperature (□). (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=6).

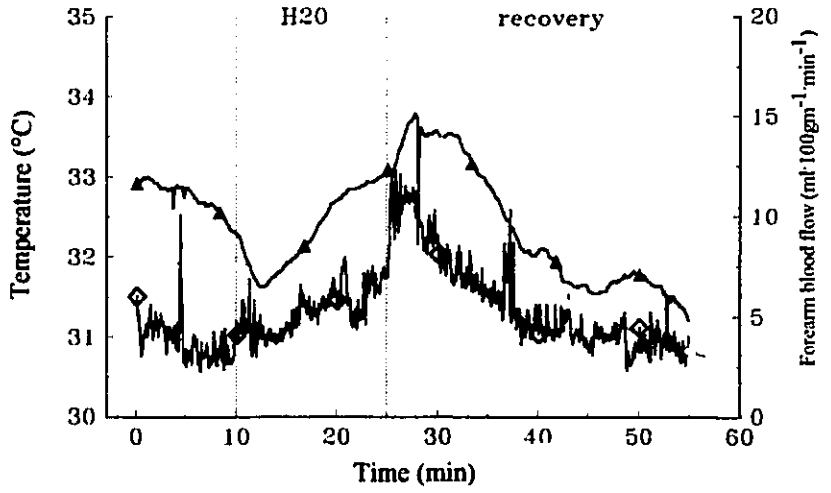

Fig. 7. Forearm temperature (Δ) and forearm blood flow (◊) from rest through the start and end of water immersion (Tw 44°C) and during standing, leaning recovery. Forearm surface blood flow measured by Laser-Doppler flowmetry. (Rest and recovery ambient temperature, 29°C; relative humidity, 50%; n=6).

Table 6. Mean core and surface temperatures and forearm surface blood flow - water immersion (pilot)

	Tre (°C)	Tes (°C)	Tfh (°C)	Tch (°C)	Tfi (°C)	Tfa (°C)	SBF (ml·min⁻¹·100gm⁻¹)
PreH2O 10'	37.26	36.94	33.88	31.97	32.09	32.91	4.84
Start	37.25	36.89	33.87	31.90	31.79	32.28	4.77
EndH2O	37.63	38.01	35.90	34.84	34.91	33.07	6.98
PostH2O 10'	37.77	36.82	33.18	32.33	32.54	32.88	6.00
20'	37.61	36.93	32.80	31.31	31.28	31.62	3.45
30'	37.56	37.04	32.86	30.88	30.57	31.41	3.70

Discussion

The data demonstrated that the post-treatment Tes was not significantly elevated above pre-treatment value. Figure 25 shows forearm surface blood flow returned to Pre-

treatment values within minutes into recovery with a similar trend in Tfa. It was found that Tw 44°C was sufficient to induce similar Tes elevation demonstrated during exercise.

Appendix G

Testing procedure for VO_{2max} determination

Testing procedure followed a progressive protocol described by Thoden et al. (1982).

1. The purpose and procedures of the test were explained to the subject.
2. Introduction to treadmill running began at 4.0 mph and gradually increased to the subject's preferred warm-up speed (usually 5.0-7.0 mph). This was continued for a minimum of 5.0 min, or until the subject was satisfied.
3. Subjects attaining a heart rate greater than 160 bpm during the warm-up were run at 7.5 mph; of 140 to 160 bpm, at 8.0 mph; of less than 140 bpm, at 8.5 mph.
4. The subject was attached to the breathing apparatus and began running at 0% grade.
5. Measurement of gas variables was made in the last 45 secs of each load.
6. The treadmill angle was increased by 2% of its length each two minutes.
7. The subject was instructed to advise if the next load could not be completed. When the signal was made, the final measurement was taken. The test was terminated at this point.
8. The subject was encouraged to "cool down" by resuming moderate exercise at 0% grade and a speed of 4.0 to 5.0 mph for several minutes.
9. The VO_{2max} was taken as the highest value attained in the test.

Appendix H

Heat Storage Calculation

Calculations of body heat storage were performed by defining the area under the esophageal temperature curve (des·dt^{-1}). Figure 8 shows the calculated regions for intermittent exercise. Heat storage was measured for each of the exercise and recovery periods. The baseline was defined by the pre-exercise esophageal temperature. Exercise heat storage was calculated from the baseline to the end of exercise and post-exercise heat storage was calculated from the end of exercise until the start of the next exercise or until termination of the experiment.

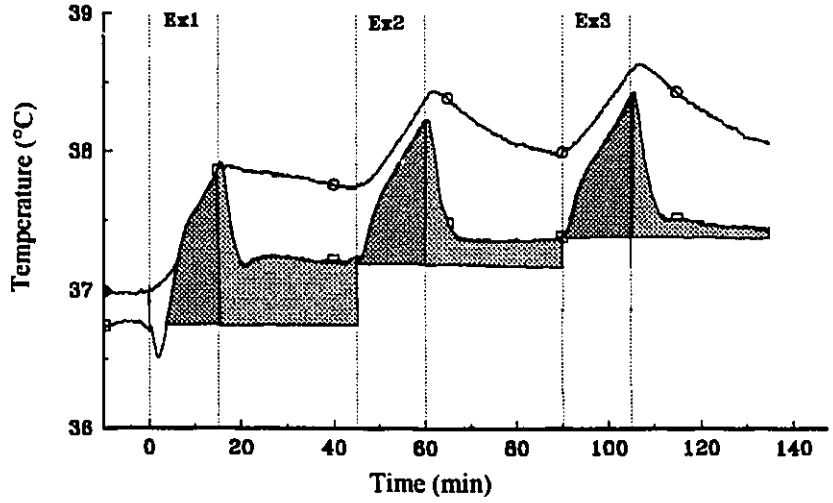

Fig. 8. Intermittent exercise mean core temperature response from rest through the start and end of exercise at 10-13 METS and during standing, leaning recovery. The shaded regions represent the individual areas used in the calculation of body heat storage. Rectal temperature (O) and esophageal temperature (□). (Ambient temperature, 29°C; relative humidity, 50%; n=9).

Appendix I

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check the ☑ YES or NO opposite the question if it applies to you.

YES	NO		
☐	☐	1.	Has your doctor ever said you have heart trouble?
☐	☐	2.	Do you frequently have pains in your heart and chest?
☐	☐	3.	Do you often feel faint or have spells of severe dizziness?
☐	☐	4.	Has a doctor ever said your blood pressure was too high?
☐	☐	5.	Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
☐	☐	6.	Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
☐	☐	7.	Are you over age 65 and not accustomed to vigorous exercise?

If You Answered

YES to one or more questions

If you have not recently done so, consult with your personal physician by telephone or in person BEFORE increasing your physical activity and/or taking a fitness test. Tell him what questions you answered YES on PAR-Q, or show him your copy.

programs

After medical evaluation, seek advice from your physician as to your suitability for:

- unrestricted physical activity, probably on a gradually increasing basis.
- restricted or supervised activity to meet your specific needs, at least on an initial basis. Check in your community for special programs or services.

NO to all questions

If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for:

- A GRADUATED EXERCISE PROGRAM - A gradual increase in proper exercise promotes good fitness development while minimizing or eliminating discomfort.
- AN EXERCISE TEST - Simple tests of fitness (such as the Canadian Home Fitness Test) or more complex types may be undertaken if you so desire.

postpone

If you have a temporary minor illness, such as a common cold.