Differentiating thermal from non-thermal eccrine sweating during exercise and heat stress

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ABSTRACT

This project investigated the non-thermal factors which influence the control of eccrine sweating during exercise, with particular emphasis upon mechanoreceptor feedback and feedforward regulation. The aim of this project was to attempt to differentiate between these two neural pathways using three experimental treatments (active exercise; passive exercise and passive heating), with core temperature clamped among treatments and two pedal frequencies used for both the active and passive exercise conditions. It was hypothesised that during active (dynamic) exercise, sweat rates (msw) and sweat expulsion frequencies (fsw) would exceed those of the passive exercise and passive heating trials. It was expected that, when the pedal force was doubled during the active exercise trials, both the msw and fsw would exceed those values observed at the lower pedal force.

Ten male subjects participated in five experimental trials: (a) two active (dynamic) exercise trials, in which the subjects voluntarily cycled at two different pedal frequencies; (b) two passive exercise trials, in which subjects were driven at the same two pedal frequencies, but did not actively recruit muscles to either track or resist the pedal motion; and (c) a seated resting trial, with subjects passively heated to track core temperature (Tc) changes in the other conditions. The combination of a water-perfusion garment and a climate chamber was used to increase and clamp Tc at similar rates across the five trials. During these trials, the following variables were measured: core temperatures at the oesophagus (Tao), auditory canal (Tao), and the rectum (Tre); skin temperatures at eight sites; msw were measured simultaneously at six locations; fsw were identified using sweat data from the forehead and forearm sites; cardiac frequency (fco); thermal sensation; and ratings of perceived exertion. Of particular interest for this project were the variables of msw and fsw, and how they were affected by differences in pedal frequency (active versus passive exercise) and passive heating.

The primary observation for these trials was that, when comparing the active and passive trials at the same pedal frequency, msw and fsw were very similar for each of the pedal frequencies, in the period from 15 to 25 minutes. However, the initial comparisons between msw and fsw of the active and passive trials were significantly different. When comparing trials at different pedal frequencies, but within the active exercise mode, a consistent trend in the msw and fsw was observed, with both being at 80 rev.min⁻¹, relative to 40 rev.min⁻¹ trials, though this was not statistically significant. For the same comparison in the passive exercise mode, the principal difference was the thermal load which was imposed on the subjects, with the data from the seated resting trials being greater than both the passive and active exercise trials.

These observations may be interpreted in the following manner. First, the role of joint and muscle mechanoreceptors feedback may have been an influencing factor in the similarities of msw and fsw in the period from 15-25 minutes. Second, in the active exercise trials, the initiation of sweating seemed to be more related to central feedforward command, a non-thermal influence, while the passive and seated resting trials, were related more to feedback control, created from the differences in thermal gradient of Tc and Tsk. Third, it would seem that thermal and non-thermal influences both play a role in the control of sweating, but their relative contribution may be modified by internal temperature and skin temperature changes.
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CHAPTER ONE: INTRODUCTION

1.1 INTRODUCTION

The regulation of body temperature in man is achieved by the integrative function of the hypothalamus. It processes temperature information from different sites in the body, and generates the appropriate signals to regulate the different effector mechanisms necessary for heat production or heat conservation (Pierau and Nakashima, 1986). Brück and Hinckel (1990) characterise the regulation of body core temperature ($T_c$) as a function of multiple inputs via a negative feedback system. The activation of the effector system depends on both the magnitude of temperature deviation and the rate of temperature change (Libert et al., 1978). The hypothalamus generates an error signal, by comparison of warm and cold inputs, which results in appropriate adjustment of the effector systems, including thermogenesis, control of cutaneous blood vessels (vasomotion), sweating (evaporative heat dissipation) and behavioural adjustments.

The current project investigated sudomotor regulation during heat stress, with an emphasis on its non-thermal regulation.

While most people are familiar with heat-induced sweating, there are three sudomotor anomalies with which we are less familiar: hyperhidrosis, anhidrosis and non-thermal sweating. Hyperhidrosis describes conditions in which large amounts of sweat are produced in an uncontrolled manner, anhidrosis is an absence of sweat production in the presence of adequate thermal stimuli (Quinton, 1983), and non-thermal sweating relates to sweat secretion elicited by mechanisms often unrelated to thermoregulation (e.g. central and generalised neural feedforward). This project addresses this last anomaly, specifically in its impact upon eccrine sweating.
At the beginning of exercise, eccrine sweating may precede a change in $T_c$, suggesting such a sweat response is influenced by non-thermal inputs preceding the change in body temperature (Stolwijk and Nadel, 1973; Nielsen, 1938). Non-thermal inputs may relate to rapid changes in arterial blood pressure, stimulation of joint and muscle mechanoreceptors, mass sympathetic outflow in parallel with motor commands from the motor cortex, or activation of chemosensitive nerve endings in the exercising muscle (Gisolfi and Wenger, 1984; Ogawa and Sugenoia, 1993; Yamazaki et al., 1993).

Central regulation of sweat glands is a function of efferent discharges carried via the sympathetic tracts. Sudomotor signals descend from the hypothalamus, through the brain stem and spinal tracts into the sympathetic chain, where they synapse with post-ganglionic fibres (cholinergic, non-myelinated class C fibres). The temporal synchronisation of the sweat glands often occurs, since many post-ganglionic fibres may fire in response to one single pre-ganglionic impulse (Bini et al., 1980), and glands normally secreted in a cyclic pattern, reflecting the pattern of sympathetic discharge. Sweat glands thus respond primarily to sympathetic impulses, derived from thermal, mechanical, respiratory and arterial stimuli (baroreceptors). Circulating epinephrine can also facilitate sweating via direct action on the $\alpha$ and $\beta$-adrenergic receptors of sweat glands (Sawka and Wenger, 1988).

Dynamic exercise imposes a thermal stress on the body. As $T_c$ increases, cutaneous vasodilation increases thermal conductance between body core and skin allowing central heat to reach the skin surface. Eccrine sweating then provides a means to transfer this heat to the environment (evaporative cooling). Numerous investigators have evaluated the contribution of the main thermal afferents, $T_c$ and skin temperature ($T_{sk}$), in the regulation of thermal sweating (see Patterson, 1995 for review). However, very little work has been undertaken to identify the
non-thermal factors which contribute to sweat secretion during combined exercise and heat stress.

The appearance of sweat within seconds of the onset of heavy exercise is frequently observed, and is too rapid for the circulation of warmed blood from the exercising muscles to either reach the brain as a bolus, or to elevate T_c. Indeed, T_c often declines with exercise onset. Similar reasoning may preclude any contribution by blood borne substances as an influence of non-thermal sweating. Furthermore, Beaumont and Bullard (1963) found that sweating was initiated at the start of exercise, even when venous occlusion prevented the warmed venous blood from leaving the exercising limbs. They discounted the participation of local temperature sensors because the response of sweat glands occurred within two seconds of the onset of the work in a warm environment, and they doubted whether heat sufficient to stimulate any temperature sensors existing in or near the muscles could be liberated within the first 2 seconds of muscular activity. More recently, the current laboratory has shown that such transient sweating responses are not driven by intramuscular thermoreceptors (Russell, 1999). Botherel et al. (1991) found a sudden decrease in sweat rate at the completion of exercise, which occurred before body temperature decreased. These observations indicate that neural factors, perhaps of mechanical origin (Botherel et al., 1991), may be interacting with sudomotor control to elicit these paradoxical sweat responses.

Saltin and Hermansen (1966) and Nielsen (1969) suggested that the liberation of chemical factors, associated with aerobic metabolism, may play a possible non-thermal role. Gisolfi and Wenger (1984) have suggested that non-thermal sweating represents a sudden increase in sympathetic nervous system activation at the beginning of exercise, similar to that seen during rapid increases in cardiac output and cardiac frequency. Regulation of this immediate cardiac
function has been shown to be neurally-based, coming from both neural feedforward and mechanoreceptor feedback. For example, Goodwin et al. (1972a) demonstrated the mechanoreceptor role when they were able to suppress cardiovascular and respiratory responses to isometric exercise, using a vibratory stimulus to block muscle afferents. Ogawa (1975) utilised a similar muscle vibration procedure to reduce sweat rate during exercise, indicating a mechanoreceptor role or interaction in sudomotor drive. Furthermore, Ohnishi (1991) reported that when the muscle exertion at a fixed workload was altered by means of partial curarization, or the tonic vibration reflex, sweat rate changed in parallel with the manipulation. However, Robinson (1962) has previously disputed the role of joint and muscle mechanoreceptor reflexes in non-thermal sweating. During passive exercise of four limbs, Robinson (1962) found that sweat rate was not out of proportion to metabolic and thermal changes. The limitation of Robinson's study was determining sweat rate ($m_{sw}$) from body weight loss, which was not sensitive enough to determine the controlling influence involved in the rapid changes in $m_{sw}$, which occur at the beginning of exercise. While these data are not necessarily without question, a re-investigation of this possible mechanoreceptor role would appear warranted.

Several authors have reported the involvement of non-thermal inputs in the regulation of sweating (Ogawa and Asayama, 1987; Yamazaki et al., 1994; Ogawa and Sugeno, 1993; Wenger, 1986), though few have been successful in identifying and quantifying these stimuli. While we are aware of significant non-thermal components being involved in the regulation of sweating, no clear picture emerges from the literature. It would seem that both central feedforward and mechanoreceptors feedback do play a role in this regulation, though the extent to which each of these inputs contributes to sweating during exercise is unknown. It was the purpose this project to attempt to identify and differentiate between some of the non-thermal
influences on sweating.

1.2 AIMS AND HYPOTHESES

This project was designed to further our understanding of the mechanisms that regulate sweating. The purpose of the present study was to help clarify our understanding of the non-thermal efferent signals, which influence eccrine sweating during exercise, with particular emphasis upon mechanoreceptor feedback from the exercising limbs, and mass sympathetic activation during heavy exercise. Five trials were performed: two involving dynamic exercise, cycling at two pedal frequencies; two passive exercise trials, at the same pedal frequencies; and a seated resting trial. The first mechanism was evaluated using a passive exercise model, where exercise patterns, identical to those achieved during dynamic exercise, were replicated within a hot environment, with subjects wearing a water-perfusion garment to replicate the thermal load. Secondly, the role of feedforward regulation, was assessed from a comparison of sweat gland function during two dynamic exercise trials, both of which involved completion of the same amount of total work, but achieved via two different means. Since an electronically-braked cycle ergometer controls total work performed, subjects were asked to cycle at a fixed work rate, but at two different pedal frequencies, one of which required twice the force generation to complete the task.

It was hypothesised that:

(i) During dynamic exercise, sweat rates and sweat expulsion frequencies will exceed those observed during both rest and passive exercise.
(ii) When pedal force is doubled during dynamic exercise, both sweat rate and expulsion frequency will exceed values observed during dynamic exercise at a lower pedal force.

(iii) Neither sweat rate nor sweat expulsion frequency will differ between the passive exercise and resting conditions.

These hypotheses were tested using indices of centrally-mediated sweat gland activation (the frequency of sweat expulsion: \( f_{sw} \)) and the local glandular response to this neural activation (sweat rate: \( m_{sw} \)), across the five experimental states, during which \( T_c \) was regulated to increase at a constant rate. Sweat expulsion frequency provided an index of sympathetic activity (Ogawa and Sugenoya, 1993), while \( m_{sw} \) was a manifestation of this activity at the skin surface. Furthermore, known thermal determinants of sweat rate (\( T_c \) and \( T_a \)) were controlled across trials, minimising their affect upon sweat regulation while allowing the non-thermal influences to be evaluated.
CHAPTER TWO: LITERATURE REVIEW

2.1 INTRODUCTION

The control of body temperature depends on the coordinated activities of many organs and tissues of the body. In mammalian thermoregulation the hypothalamic regions in the brain are essential for normal regulation of body temperature, and the hypothalamus is itself temperature sensitive (Bligh, 1973; Cabanac, 1969; Hardy, 1973). In human control systems, the sensing of the controlled quantity, and the process relating the afferent input from sensors to the efferent output to effectors can be considered as two distinct functions within the system.

For physiologists interested in control mechanisms of thermoregulation, it is recognised that the regulation of body temperature is not really an isolated system, but under certain experimental conditions, many of the external influences can be reduced or eliminated, and thermoregulation can be studied almost on its own. Eccrine sweating is considered to increase linearly with increased thermal inputs, such as $T_c$ and $T_{sk}$ (Gisolfi and Wenger, 1984; Wyndham, 1965b; Botherel et al., 1992a; Nielsen, 1990). It has been demonstrated that eccrine sweating may even commence prior to a change in body heat storage, which has been shown to have a long time constant (Yamazaki et al., 1994b; Robinson, 1962; Wenger, 1986; Ogawa and Asayama, 1987). The regulation of sweating during exercise depends on both thermal and non-thermal factors, and this project deals mainly with the latter category. The frequency of afferent impulses from the stretch receptors or mechanoreceptors of joints and muscles, could be proportional to muscular activity, and may act upon the thermoregulatory centres and facilitate efferent flow to the sweat glands.
(Asmussen and Nielsen, 1947). Another possible explanation for the very rapid onset may be related to, mechanical events creating a rapidly accommodating influence on the thermoregulatory centre, helping to lower the threshold and thus facilitate sweating (Bligh, 1973). Muscular exercise increases metabolic rate above resting levels, thus producing a considerable amount of heat which must be dissipated. This project focuses on the stimulus for thermal sweating from eccrine sweat glands, before there is sufficient time to develop enough metabolic heat to change $T_c$, and thus investigates sweating, which is of a non-thermal origin.

2.2 CENTRAL INTEGRATIVE FUNCTION OF THERMOREGULATION

Our understanding of the central nervous system connections underlying temperature regulation mechanisms is still incomplete. Benzinger (1969) reported that the pre-optic area is mainly concerned with temperature reception whilst the thermo-integrative functions are located in the anterior and posterior hypothalamus. In 1935, when Ranson and Ingram ablated areas of the brain of rhesus monkeys, they found that lesions in the rostral anterior hypothalamus prevented thermoregulatory responses to heat stress, while lesions in the posterior hypothalamus prevented responses to cold stress. Lesions in the rostral hypothalamus and in the pre-optic area prevented the animals from responding to heat stress, but did not prevent responses to cold stress. Evidence that the anterior hypothalamus was involved only in the responses to heat stress was also reported by Frazier et al. (1936) and Clark et al. (1939). The evidence derived from electrophysiological studies demonstrated thermosensitive responsiveness of the skin projected to the pre-optic region (Wit and Wang, 1968), the anterior (Hellon, 1970), and the posterior hypothalamus (Nutik, 1973).
The pre-optic anterior hypothalamus (POAH) has an important role in the centrally-mediated thermoregulation. Through its network of neural and humoral inputs, the POAH is capable of producing selectively the effector responses, which are most appropriate to changes in the thermal status of the internal and external environments. The POAH is involved in cardiovascular control as well as, thermoregulation. It receives afferent input from local and peripheral thermal stimuli, and non-thermal information which has been integrated from the limbic system and associated cortices (Hori, 1991).

2.3 AFFERENT INFORMATION

Peripheral warm and cold-receptor information converges during its ascent over multisynaptic somatosensory pathways, through anterolateral and reticular formation networks (Hensel, 1981). Most studies have identified three major types of POAH neurons: warm-sensitive, cold sensitive, and temperature insensitive neurons. Increases in hypothalamic temperature produce increased firing rates in warm-sensitive neurons and decreased firing rates in cold-sensitive neurons. Neurons classified as warm-sensitive or cold sensitive receive skin or spinal thermoreceptive input, while most of the temperature-insensitive neurons do not receive this afferent information (Boulant, 1974).

2.3.1 Central thermoreceptors

The main function of central thermoreceptors is to contribute to temperature signals from different parts of the body, which together with the cutaneous thermoreceptors form the afferent input to the POAH. Warm sensitive neurons sense both hypothalamic temperature and endogenous substance, and compare this local information with thermal and non-thermal synaptic afferents arriving over ascending pathways (Boulant, 1996). The medial dendritic tree of these neurons could receive synaptic information from preventricular pathways, or
even chemosensitive information from the third ventricle, and the lateral dendritic tree would be capable of integrating information from ascending and descending fibres in the medial forebrain bundle (Boulant, 1996). This supports the notion that thermosensitive neurons have a high degree of convergence from local and remote sites thus forming neural networks within the brain and spinal cord, to synaptically influence thermal and non-thermal homeostatic functions controlled in the hypothalamus (Hori, 1991).

Electrophysiological studies of Boulant (1973, 1974) suggest that a neuron's thermosensitivity is altered by its spontaneous firing rate. There are three different groups of warm-sensitive neurons: low-firing, medium-firing and high-firing neurons. The lowest firing warm-sensitive neurons are responsible for controlling heat loss responses (for example, sweating, panting) and have low firing rates because they are inhibited by ascending peripheral cold receptive pathways. These neurons have low firing rates because they do not receive much excitatory input from ascending warm-receptive pathways. The second group of medium-firing warm-sensitive neurons, have firing rates between 5 and 15 imp.s\(^{-1}\). These neurons are responsible for controlling heat retention responses (for example, skin blood flow, thermoregulatory behaviour), which receive a moderate amount of excitatory input from ascending pathways, and are most active in the thermoneutral range. The highest-firing warm-sensitive neurons which have spontaneous firing rates of 15-60 imp s\(^{-1}\), which receive a great amount of excitatory afferent input, and have an enhanced sensitivity to endogenous excitatory substances (Boulant, 1996).

2.3.2 Peripheral thermoreceptors

Peripheral temperature not only affects the firing rates of integrative hypothalamic
neurons, but it also changes their local thermosensitivities in the same way that peripheral temperature changes the sensitivity of hypothalamically-evoked thermoregulatory responses. The afferent inputs primarily affect high-firing warm-sensitive neurons and cold-sensitive neurons. Peripheral temperature has a greater effect on heat production responses compared to heat loss responses. Peripheral warming increases heat loss responses, but it decreases the hypothalamic thermosensitivity controlling these responses. Therefore, POAH warm-sensitive neurons could be excited by pathways activated by increasing peripheral temperature or inhibited by pathways activated by decreasing peripheral temperature. Differing amounts of endogenous or afferent inputs produce different ranges of warm-sensitive neurons, each having different ranges of thermosensitivity and different thermoregulatory roles (Boulant, 1996).

Many temperature-sensitive and insensitive POAH neurons respond to changes in osmolality, glucose, and endocrines which provide feedback signals to various hypothalamic regulatory systems (Boulant and Silva, 1987). Thermosensitive POAH neurons also respond to non-thermal parameters which affect body temperature regulation such as, substances linked with neurotransmission and neuromodulation, as well as endogenous substances (e.g. testosterone, oestrogen).

2.3.3 Non-thermal afferents

Kinaesthesia is a term which describes activity in mechanoreceptors, from centrally generated motor commands, and from interactions between the afferent and efferent signals in relation to static position or dynamic body movement force generation (Gandevia, 1996). The principal receptors subserving the senses of movement and position are intramuscular
receptors, probably the primary and secondary endings of the muscle spindles (McCloskey, 1978). McCloskey (1978) describes the classes of afferent fibres which are involved in kinaesthetic sensibility are those from the skin, the muscles and tendons, and from the joint capsule and ligaments. Cutaneous receptors appear to support or facilitate the specific kinaesthetic signals from intramuscular and possibly joint receptors. In addition, they may provide specific perceived signals of joint position and movement, particularly in distal joints (McCloskey, 1978). Fibres from intramuscular receptors can run in joint nerves and fibres from joint receptors can run in muscle nerves. Small diameter afferents conducting in the group III and IV range nerve fibres of skin, joint and muscles communicate mechanical, chemical and thermal nociceptive stimuli (Gandevia, 1996). This mixed composition of both joint and muscle nerves makes studies aimed at defining the central projections of joint and muscle receptors difficult to interpret because it cannot follow that one or another type of receptor is completely excluded.

2.4 THERMAL EFFERENT INFORMATION: SUDOMOTOR CONTROL

The thermoregulatory effector functions are those concerned with the control of heat production and heat loss, it is the latter which this section will focus upon. The autonomic control of non-evaporative heat loss by radiation, convection and conduction consists of adrenergic sympathetic control of peripheral vasomotor tone (Bligh, 1973). Thermoregulatory sweating is elicited from efferent impulses emanating from the POAH, which acts as an internal thermal central controller, and afferent nerve impulses, arriving from peripheral and deep thermoreceptors which stimulate the same centres via long reflex mechanisms (Randall, 1963).
2.4.1 Sweat glands

There are two classifications of sweat glands: eccrine and apocrine glands. The distinction between eccrine and apocrine glands is in their morphology, distribution, and embryologic development. The apocrine gland relies on catecholamines to initiate sweat secretion, as they have no neural innervation, while the eccrine gland is cholinergically activated (Robertshaw, 1975). It is the eccrine sweat gland that is the focus of this project.

Human eccrine sweat glands are single units that extend downward from the epidermis about two to five millimetres, consisting of two parts: a secretory coil and a duct (Quinton, 1983). These glands are densely distributed over nearly the whole body surface comprising some 3-4 * 10⁶ (Kuno, 1956). Their main role is in the dissipation of heat via evaporation. Evaporation of 1 g of sweat dissipates 2.43 kJ of heat (Gisolfi and Wenger, 1984). They have two major functions: first, secretion of electrolytes and metabolites by the secretory coil, in response to locally released acetylcholine; and secondly the reabsorption of sodium in excess of water from its secretory duct, thereby producing hypotonic sweat on the skin surface (Sato, 1977). This reabsorptive function plays a vital role in maintaining homeostasis which helps to offset or reduce the effect of hypovolemia.

Apocrine glands in man occur mainly in the axilla, the mons pubis, the external meatus, the areola and the circumanal area (Sato, 1977). The hair follicles give rise to the apocrine glands while the eccrine glands develop directly from the epidermis (Kuno, 1956). From an evolutionary perspective, these glands have diverse functions such as in the production of sexual odours, tactile and frictional sensibility (Robertshaw, 1971). Apocrine glands, in which at least some portion of the secretory process involves the rupture of the cell
membrane and the discharge of cytoplasmic material into the glandular lumen, predominantly serve a non-thermal role (Sato, 1977).

It is well established that the nerve structure of the eccrine gland is composed of nonmyelinated class C fibres issuing from the paravertebral ganglia (Houdas and Ring, 1982). The fibres are distributed to the myoepithelial and secretory cells. Yet Uno and Montagna (1975) demonstrated catecholamine-containing nerves, as well as the terminal structure of the adrenergic nerve, exist around the eccrine sweat gland suggesting a dual innervation. Sato (1977) concludes the debate that thermal sweating, as well as, nervously excited sweating is completely inhibited by atropine, supporting the theory that only cholinergic sudomotor fibres, and thus the cholinergic mechanism, are involved in the eccrine sweat gland.

Thermal sweating can begin within seconds to minutes of beginning exercise. For a given sudomotor signal to the eccrine gland, local skin temperature and skin wettedness influence the amount of sweat secreted. Early research by Benzinger et al. (1963) suggested that sweating is elicited primarily by core warming, and that skin temperature above 33°C has little effect or no effect on the central control of sweating, though body core temperatures below 33°C acted to elevate the Tc for sweating. Libert et al. (1984) confirms that sweat onset was neither linked to a definite peripheral temperature nor a core temperature, but a combination of the two. Nadel et al. (1971) suggested that the sweating response of any skin area to a given combination of Tc and Tsk increases with local Tsk. The influence Tsk has on sweating is the effect it has directly on the sweat glands. The peripheral effect is thought to multiply the central command and is important between 28 and 36°C Tsk, which is in contrast
to Benzinger's research. Patterson (1995) established that thermal sensitivity of the skin is not uniform, which implies a hierarchical integration of afferent input from the periphery, or possibly reflects differences in thermoreceptor density and differences in the POAH response to these signals.

2.4.2 Non-thermal sweating

It is proposed that non-thermal mechanisms exist that affect sweat regulation during exercise, and these mechanisms are thought possibly to result in the differences in sweating responses seen during exercise and recovery at rest (Gisolfi and Robinson, 1970). The main mechanisms are thought to be: centripetal input from the mechanoreceptors and joint receptors to sweating centres; and irradiation of descending central motor command to central sudomotor mechanisms at the beginning of work (Yamazaki et al., 1993). While this project cannot shed light onto either of these mechanisms, it is focused upon the impact of such non-thermal information on the sympathetic control of sweating.

Research by Beaumont and Bullard (1963) indicated that sweating occurred after the initiation of muscular activity which did not seem dependent on thermal stimuli, but on work rate. These authors later work (1966) showed that the rapid responses were not effected by circulatory occlusion which prevented warm blood from the active muscles reaching centrally located thermoreceptors. Gisolfi and Robinson (1970) suggested that the abrupt changes of sweat rate at the start and end of work must be related to the activation and cessation of neuromuscular reflexes. Gisolfi and Wenger (1984) suggest that non-thermal sweating at the beginning of exercise represents a sudden increase in sympathetic nervous activity and vasomotor responses, which may be related to the rapid increases in cardiac
output and heart rate. These authors make further suggestions for this occurrence, which include increase in arterial pressure; stimulation of joint and muscle mechanoreceptors; radiations from the motor cortex, and much of the change in the environment of chemosensitive endings in exercising muscle may be involved as factors responsible for the non-thermal sweating. Botherel et al., (1991b) found a sudden decrease in sweating at cessation of exercise before any body temperature decrease, further supporting the theory that mechanical factors may interact in the control of sweating. Botherel et al. (1991b) suggested that the sweating response was partly controlled by non-thermal factors, such as body fluid adjustments and muscle contraction components.

Sweat expulsions are always preceded by sympathetic bursts. Sugenoya et al. (1990) confirmed a quantitative neuro-effector relationship for the sudomotor system by the close correlation between the amplitude of the sudomotor burst and the magnitude of the corresponding sweat response. Of the three characteristics representing the magnitude of the sudomotor effector response, the rate of increase of the sweat expulsion was highly correlated with the amplitude of the burst. This is understandable if it is assumed that this characteristic represents the rate of precursor sweat formation at the secretory coil of the sweat gland, when the duct has been filled. Thus, the rate of increase of the sweat expulsion is available as an index of the intensity of the sudomotor drive.

Ogawa et al. (1993) suggests that the conduction velocity of sudomotor fibres is 1.0 to 1.4 m.s.\(^{-1}\) whereas that of vasomotor is 0.7 m.s.\(^{-1}\). In general, skin sympathetic nerve activity consists of multi unit bursts containing sudomotor, vasomotor, and pilomotor impulses. The interval from the sudomotor burst to the onset of the corresponding sweat
expulsions is 2.7 and 2.4 seconds. The greater the amplitude of the skin sympathetic nerve activity the shorter the time interval, possibly indicating a central controller influence. The latency period of 2.7 seconds included the following intra-glandular and extra-glandular processes: conduction of impulses along the sudomotor fibre from the site of recording to the nerve terminals, neuro-glandular transmission, formation of precursor sweat within secretory cell, duct filling, evaporation of sweat from the skin surface and the time lag in humidity sensing.

Saito et al. (1990) performed experiments recording the traffic of sympathetic nerves leading to the foot and skeletal muscle during isometric handgrip task whilst determining $\dot{m}_{sw}$ and skin blood flow. The authors showed an abrupt increase in sympathetic nerve activity with the commencement of the handgrip task (contraction) and thereafter was constant at 35% higher than the control value. Skeletal muscle activity showed a time dependent increase during the course of the handgrip task. There was an immediate increase in $\dot{m}_{sw}$ after the commencement of muscle contraction, and a high rate remained during the handgrip task which corresponded with the change in sympathetic nerve activity. The results may suggest that the increase in sympathetic nerve activity during static contraction is composed mainly of sudomotor nerves, which the authors suggest respond to input from the peripheral mechanoreceptors and central command, but is influenced little by the afferent input from the muscle chemoreceptors. At the cessation of the handgrip task, sympathetic nerve activity, $\dot{m}_{sw}$, and skin blood flow returned quickly to control levels.

Yamazaki et al., (1994a) reported changes in $\dot{m}_{sw}$ occurred before a change in body temperature, which they eluded was influenced by changes in non-thermal inputs preceding
the change in body temperature, and that these inputs become relatively smaller with increasing heat storage. This may suggest that central command contributes to the regulation of sweating, as it is a primary mechanism that stimulates sympathetic outflow.

2.5 SUMMARY

It is common knowledge that, as body temperature increases with exercise, the need to dissipate heat increases. Thermoregulatory sweating is elicited from efferent impulses emanating from the POAH, which acts as the central controller for afferent impulses arriving from deep and peripheral thermoreceptors, as well as, non-thermal afferents. This project's central focus is to provide information to help improve our understanding of the factors which influence sweating. However, eccrine sweating commences prior to a change in body temperature (Stolwijk and Nadel, 1973), suggesting that, at the beginning of exercise, eccrine sweat control is influenced by non-thermal factors. Many studies have tried to isolate these non-thermal factors and have given reasons suggesting possible mechanisms responsible, though the answer still remains ambiguous. This project attempts to determine the non-thermal mechanisms involved in the regulation of eccrine sweating during heat stress and exercise, using five different experimental conditions. These trials consisted of two active and passive exercise trials, at two different pedal frequencies, of 40 and 80 rev.min⁻¹, and a seated resting trial. Known thermal determinants of $m_{sw} (T_c$ and $T_{sk}$) were controlled to minimise their affects and replicate equivalent thermal strain across the experimental states to try and isolate non-thermal influences involved in the regulation of $m_{sw}$. The role of mechanoreceptors was evaluated comparing the differences in sudomotor function between the three different experimental trials. The role of feedforward regulation was evaluated from a comparison of $f_{sw}$ and $m_{sw}$ during dynamic exercise where pedal frequency was controlled.
CHAPTER THREE: METHODS

3.1 SUBJECTS

Ten healthy males volunteered as subjects. Their physical characteristics are given in Table 3.1. Each received a subject information package, provided informed consent, completed an activity questionnaire, and satisfactorily completed the Physical Activity Readiness Questionnaire (PAR-Q, 1992) before participating. All methods were approved by the University of Wollongong's Human Experimentation Ethics Committee.

3.2 EXPERIMENTAL PROCEDURES

3.2.1 General Overview

Subjects participated in five different experimental trials. The active exercise trials were completed first, so that the rates of change in $T_e$ could be determined for the passive exercise and seated resting trials, which were applied in a balanced order, and spaced not less than one week apart. These trials were designed to elicit approximately equivalent thermal strain, from various combinations of metabolic and externally-derived heat sources. One trial involved seated upright rest on a cycle ergometer, two trials involved passive exercise, where the subject's legs tracked pedal movement (without physical effort), at each of 40 and 80 rev.min$^{-1}$, and two trials involved active exercise (200 watts) at two different pedal frequencies (40 and 80 rev.min$^{-1}$). Since these last two trials were performed on an electronically-braked cycle ergometer (Excalibur Sport, Lode, Netherlands), the maintenance of the 200 watts work rate forced the subjects to exert twice the effort during the trial conducted at 40 rev.min$^{-1}$ due to the lower number of revolutions, when compared with that
Table 3.1: Physical characteristics of subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (yrs)</th>
<th>Height (cm)</th>
<th>Mass (kg)</th>
<th>$A_D$ (m²)</th>
<th>$A_D$: Mass (m².kg⁻¹)</th>
<th>$\sum 8skf$ (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20</td>
<td>198.5</td>
<td>80.4</td>
<td>2.14</td>
<td>0.27</td>
<td>65.0</td>
</tr>
<tr>
<td>2</td>
<td>22</td>
<td>188.5</td>
<td>73.6</td>
<td>1.99</td>
<td>0.27</td>
<td>90.0</td>
</tr>
<tr>
<td>3</td>
<td>30</td>
<td>189.4</td>
<td>90.9</td>
<td>2.18</td>
<td>0.24</td>
<td>98.0</td>
</tr>
<tr>
<td>4</td>
<td>31</td>
<td>170.0</td>
<td>71.7</td>
<td>1.82</td>
<td>0.25</td>
<td>50.3</td>
</tr>
<tr>
<td>5</td>
<td>24</td>
<td>177.0</td>
<td>83.0</td>
<td>2.00</td>
<td>0.24</td>
<td>207.1</td>
</tr>
<tr>
<td>6</td>
<td>27</td>
<td>184.0</td>
<td>82.4</td>
<td>2.05</td>
<td>0.25</td>
<td>132.5</td>
</tr>
<tr>
<td>7</td>
<td>22</td>
<td>186.0</td>
<td>72.5</td>
<td>1.96</td>
<td>0.27</td>
<td>63.0</td>
</tr>
<tr>
<td>8</td>
<td>21</td>
<td>180.9</td>
<td>79.6</td>
<td>1.99</td>
<td>0.25</td>
<td>72.0</td>
</tr>
<tr>
<td>9</td>
<td>20</td>
<td>180.6</td>
<td>63.6</td>
<td>1.81</td>
<td>0.29</td>
<td>81.8</td>
</tr>
<tr>
<td>10</td>
<td>26</td>
<td>183.8</td>
<td>70.6</td>
<td>1.92</td>
<td>0.27</td>
<td>108.5</td>
</tr>
</tbody>
</table>

**Mean**
- Age: 24.3 yrs
- Height: 183.9 cm
- Mass: 76.8 kg
- $A_D$: 1.99 m²
- $A_D$: Mass: 0.26 m².kg⁻¹
- $\sum 8skf$: 93.8 mm

**Max**
- Age: 31 yrs
- Height: 198.5 cm
- Mass: 90.9 kg
- $A_D$: 2.18 m²
- $A_D$: Mass: 0.29 m².kg⁻¹
- $\sum 8skf$: 207.1 mm

**Min**
- Age: 20 yrs
- Height: 170.0 cm
- Mass: 63.6 kg
- $A_D$: 1.81 m²
- $A_D$: Mass: 0.24 m².kg⁻¹
- $\sum 8skf$: 50.3 mm

**SD**
- Age: 4.0 yrs
- Height: 7.7 cm
- Mass: 7.8 kg
- $A_D$: 0.12 m²
- $A_D$: Mass: 0.15 m².kg⁻¹
- $\sum 8skf$: 47.8 mm

**Abbreviations:**
- $A_D$ = body surface area (after Dubois 1927); $\sum 8skf$ = sum of skinfolds;
- SD = standard deviation.
of the trial conducted at 80 \text{rev.min}^{-1}. Relative humidity (RH) was kept constant across all conditions (60.2\% \pm 1.2\%). The active exercise trials were performed in a temperate environment (ambient temperatures (T_a) controlled at 26.3°C (standard deviation (sd) \pm 1.0), while the passive exercise and resting trials were performed in a hot environment (T_a 39.8°C \pm 0.86), and 40.3°C \pm 1.1, respectively). The passive exercise and seated resting trials were performed in a different T_a due to the reduced metabolic load associated with these experimental trials, and to help to increase and elicit equivalent thermal strain, as that of the active exercise trials, for all experimental trials. The rate of change of T_c in the active 40 and 80 \text{rev.min}^{-1} exercise trials was used to determine thermal strain for each subject, prior to the commencement of the passive 40 and 80 \text{rev.min}^{-1} exercise and seated resting trials. Ambient and black globe temperatures were monitored continuously (Grant Instruments Ltd., 1200 series, U.K) at 0.2 Hz. Globe temperature was always within 1.0°C of T_a. Relative humidity and T_a were also monitored at the air outlet of the chamber every 30 seconds. This information was used by computer software to control climate chamber conditions. Wind velocity (Hand anemometer, OSK 756, Ogawa Seiki Co., Ltd, Japan) was observed to be negligible (less than 0.03 \text{m.min}^{-1}).

In an attempt to induce equivalent elevations in T_c across all trials, subjects wore a water-perfusion garment in the passive and resting conditions. During these trials, this garment was perfused with warm-to-hot water to regulate the T_c rise to match that obtained in the two active exercise trials. During each trial, subjects wore shorts and sandals. Measurements during all trials included: sweat rate, frequency of sweat expulsion, change in mass, body core and skin temperatures, cardiac frequency, thermal sensation, and ratings of perceived exertion.
3.2.1.1 Active exercise condition

Subjects completed two active exercise trials at 200 watts on an electronically-braked cycle ergometer (Excalibur Sport, Lode, Netherlands) at two different pedal frequencies (40 and 80 rev.min\(^{-1}\)). Prior to each trial, subjects started cycling until continuous sweating (above baseline) was observed (sweating was primed). These sweat rates were the plotted against time. The median sweat rate for the first 2 minutes was recorded as a representation of baseline $m_{sw}$. The point where sweating became elevated above the median value for at least 5 minutes, without returning to baseline sweating was considered to be continuous sweating. The subjects then rested until sweating ceased. This exercise was undertaken to prime the eccrine glands and to attempt to eliminate any delays in sweating due to duct filling. Trials then consisted of 30 minutes active work, and a 10-minute resting recovery period.

3.2.1.2 Passive exercise conditions

Subjects completed two passive exercise trials at (40 and 80 rev.min\(^{-1}\)) on a cycle ergometer (Monark, Sweden). Passive exercise was performed by linking two cycle ergometers at the cranks. The subject sat on one ergometer, and his feet were secured to the foot plates with toe straps. An experimenter sat on the second, driving ergometer, and pedalled at the predetermined frequency, to produce passive leg motion in the subject. The ergometers were secured to the floor to minimise movement. The water perfusion suits connections were strengthened and the use of elastic straps to minimise garment movement. This proved effective, and helped to counteract the problem of varied suit fit among subjects.

For these passive trials, subjects were instrumented in the climate chamber which was
equilibrated to a temperate state (T_a 26.3°C (±1.0), RH 60.2% (±1.2)). The subjects donned the suit with the aid of two experimenters, who further secured the suit with the elastic straps (Figure 3.1). The subjects then sat on the ergometer while the sweat capsules were secured. Once the subject was fully prepared, he started cycling until continuous supra-baseline sweating was seen from at least one hygrometer channel. The subjects then rested until sweating ceased. This was to prime the sweat glands, so that this component was eliminated as a non-thermal component of thermal eccrine sweating. Once sweating had ceased, the chamber was heated to its predetermined state (T_a 39.8°C (±0.8), RH 60.2% (±0.4)) and water of 4°C (±2.5) was passed through the suit (2.05 l·min\(^{-1}\) (±0.3)) to stop the subject from being influenced by the changing T_a, which took approximately 20 minutes to stabilise. Sweating was monitored to check that it did not change prior to commencement of passive exercise. Commencement of data collection began when the hot water bath was turned on (T_{water} 58°C) and the subject commenced the passive task (Figure 3.1). Passive exercise trials lasted 40 minutes, to allow for the time delay between the application of external heat, and the rise in T_c. A 10-minute resting recovery period was again used.

3.2.1.3 Seated resting condition

Subjects completed one resting trial in a hot, moist climate (T_a 40.3°C (±0.3), RH 60.2% (±0.2)). The subjects were instrumented on the ergometer as in the other trials. Trials were completed at the end of 50 minutes, similar to the passive conditions.

3.2.2 Controlling skin and core temperatures

The perfusion suit was only used to regulate T_c, it did that by increasing T_{sk}, therefore T_{sk} was not constant across all experimental conditions. The suit comprised of two sections, a
Figure 3.1 (Top) An experimenter securing the water-perfusion garment to the subject prior to data collection. (Bottom) A view of the passive exercise trial, conducted in the climate chamber.
jacket and pants which consists of 140 metres of Tygon (B 44-3 beverage tubing) tubing. The jacket consisted of 60 one-meter tubes covering the torso and 15 one-meter tubes for each arm, with the pants having 25 one-metre tubes for each leg. Each tube had a set length of one-meter, with groups of tubes connected in parallel to ensure uniformity of flow across the suit. This was confirmed using coloured dye and high speed video (Cotter, 1998). The suit's design produced a diamond pattern, as the tubes were connected alternatively to the adjacent tubes every four centimetres. When the suit was on the subject, a maximum distance of two centimetres was maintained between adjacent tubes, resulting in diamonds of eight centimetres in length and two centimetres in width. To secure the garment to the subject, and to maximise skin contact, elastic straps (with velcro) were wrapped around the limbs and torso.

Water, at different temperatures, was pumped through the suit (2.05 l·min⁻¹ (± 0.3)) from the bottom of the torso, arms and legs, exiting at the top of the pants and jacket. The two sections are supplied by a common tube which received water from any of the three 38-litre water baths (Grant Instruments Ltd, type VFP x 2, type ZD x 1, U.K.). Each bath used a refrigeration unit along with the standard heater and pump unit (Grant Refrigeration Systems, type CK2 x 2, U.K.) to maintain temperature. For any trial, three different bath temperatures were used: one set at 4°C (± 2.5) to minimise increases in T_c and T_a, while the experimenters prepared the subject; the two other bath temperatures were 47°C and 58°C. These were determined from a number of passive pilot exercise trials. The higher temperature was used to increase T_c at the same rate as observed in both the active exercise conditions. The lower temperature was used to prevent T_c overshoot and to maintain T_c after the initial heating period.
3.2.3 Subject preparation and experimental standardisation

Subjects reported to the laboratory one hour prior to data collection. On arrival at the laboratory, subjects were instructed to void and were measured for mass. A rectal thermistor was then inserted by the subject 12 cm beyond the anal sphincter. An oesophageal thermistor was inserted under topical anaesthesia (Xylocaine, Astra Pharmaceutical, Australia) by an experimenter, and cardiac frequency monitor fitted. This allowed sufficient time for these measurements to become stable while subject preparation continued. Prior to subject arriving a 5 minute baseline of the system's noise was recorded. This was to determine the amount of inherent noise of the sweat capsule system, and to further help in determining $f_{sw}$.

To ensure that testing was performed with minimal influence of extraneous factors, subjects were asked to avoid abnormally strenuous exercise for 24 hours prior to a trial; avoid exercise on the day of testing; avoid alcohol consumption on the previous evening; avoid caffeine within four hours of the experiment; have a low fat diet for breakfast on the morning of testing; avoid high salt intake for dinner the previous evening; and subjects uniformly hydrated themselves by drinking one litre of water the evening prior to testing. Hydration state was not measured. Testing for each subject was performed at the same time of day to cater for circadian shifts in $T_c$.

3.2.4 Criteria for stopping test protocol

The test protocol was terminated if any of the following events occurred: $T_c$ reached or exceeded 39°C; cardiac frequency reached 95% of age-predicted heart rate reserve; if the subject displayed signs of thermal distress or an abnormal response to exercise; or if the
subject desired to stop. Over the 50 trials, only 4 were terminated prematurely, three of these four because of subject's own volition, and one because of syncope.

3.3 MEASUREMENTS AND APPARATUS

3.3.1 Body temperatures

Deep body (core: $T_c$) and cutaneous temperatures were measured continuously throughout each trial. Oesophageal and rectal temperatures were used to measure changes in $T_c$. The use of two different sites to monitor $T_c$ was deemed to provide a more valid indication of $T_c$ than a single measurement, since deep body thermoreceptors are located throughout the body (Jessen, 1990).

3.3.1.1 Oesophageal temperature

An oesophageal temperature probe (YSI mini-thermistor, type 401) was inserted transnassally prior to each experimental condition. The insertion length ($L$) was determined from the equation;

$$L = 0.479 \times \text{(sitting height(cm))} - 4.44 \text{ cm} \quad \text{(Mekjavic and Rempel, 1990).}$$

Accordingly, oesophageal temperature ($T_{es}$) was recorded at a depth of 41.1 cm ($\pm 2.0$) from the nose. The naso-pharyngeal mucosa were treated with a topical anaesthetic (Xylocaine, Astra Pharmaceutical, Australia) prior to probe insertion. The probe was then inserted by a trained experimenter. Once the probe entered the pharynx, subjects drank water through a straw to aid swallowing. The mass of water was noted for the determination of body mass changes during the trial. Data were recorded using a data logger (Grant Instruments Ltd., 1200 Series Squirrel, Cambridge) at 0.2 Hz and downloaded to a computer for storage at the
end of each trial.

3.3.1.2 Rectal temperature

Rectal temperature ($T_{re}$) was measured using a thermistor (Yellow Springs Instruments Co., Inc., YSI probe no, 401, Ohio, U.S.A.), inserted 12 cm beyond the anal sphincter. Data were recorded using a data logger (Grant Instruments Ltd., 1200 Series Squirrel, Cambridge) at 0.2 Hz and downloaded to a computer for storage at the end of each trial.

3.3.1.3 Mean core temperature

Mean core temperature ($T_c$) was taken as the arithmetic average of the two core temperature indices: $T_c = (T_{es}/aural + T_{re})/2$. In seven instances, where $T_{es}$ was unable to be recorded, $T_{aural}$ was used instead with $T_{re}$ always being recorded. In the case where one measurement was unable to be recorded, $T_c$ was simply that of the site recorded.

3.3.1.4 Skin temperatures

Skin temperatures were monitored at 0.2 Hz from thermistors (Grant Instruments Ltd., Cambridge) located at eight sites. These sites included: forehead, right scapular, left upper chest, right arm, left forearm, left hand, right anterior thigh, and left leg (International Standards Organisation (ISO); 1992). These measurements were used in the following equation to determine mean skin temperature ($T_{sk}$):

$$T_{sk} = ((T_{forehead} + T_{right arm} + T_{left forearm}) \times 0.07) + ((T_{right scapular} + T_{left upper chest}) \times 0.175) + (T_{hand} \times 0.05) + (T_{right anterior thigh} \times 0.19) + (T_{left leg} \times 0.2) \text{ (ISO, 1992).}$$
3.3.1.5 Mean body temperature

Mean body temperature ($T_b$) was calculated using the mean of the two measures $T_c$ and $T_{sk}$. The following equation was used to determine $T_b$:

$$T_b = (T_c * 0.8) + (T_{sk} * 0.2) \text{ (Vallerand et al., 1992).}$$

Where:

- $T_b$ = mean body temperature (°C)
- $T_c$ = mean core temperature (°C; mean of oesophageal, and rectal temperatures)
- $T_{sk}$ = mean skin temperature (°C)

3.3.1.6 Thermistor calibration

All thermistors and temperature probes were calibrated in a 38-litre stirred water bath (Grant Instruments Ltd., Cambridge) together with a NATA (National Association of Testing Authorities) calibrated reference mercury thermometer (Dobbie Instruments, Dobros total immersion, Australia) prior to testing. Thermistors were grouped together and positioned in open water near the thermometer bulb. The water bath was set at 10°C and thermistor output collected. Data were logged every 15-20 min (to allow adequate stabilisation of the water bath temperature) in the temperature range of 10 to 45°C for the thermistors. The reading time for the Dobros thermometer corresponded with data logging onset every 5-min. Linear analysis was then performed on these data and a calibration equation established for each thermistor. Using the coefficients of these linear equations, thermistor output was converted to the corrected temperatures for subsequent analysis.
3.3.2 Sudomotor function

Sudomotor function was evaluated using two measures: body mass changes ($M_{sw}$); local site sweat rates ($m_{sw}$). Fluid loss was determined from differences between pre-and post-trial body mass using high resolution platform scales (AND, Model No. fw-150k, California), uncorrected for respiratory and metabolic losses. Sweat rate ($m_{sw}$) was monitored at 0.2 Hz from six locations simultaneously. Two, four-channel capacitance hygrometry systems were used (Figure 3.2: Clinical Engineering Solutions, Australia: Turner and Gass, 1993).

Air was pumped to each sweat capsule through a sealed flask containing a hygroscopic solution (lithium chloride). This salt solution was used due to its stability and low RH (11-12%) over the temperature range from 25°C to 50°C (Winston and Bates, 1960). Air collected over this salt solution provided a fixed and reproducible RH, which was then passed, to each of the six sweat capsules (3.15 cm$^2$ (4) and 2.19 cm$^2$ (2)). Air flow for each capsule was regulated using a separate rotameter (Platon, Duff and Mactintosh, Sydney). Air was dispersed onto the skin below each capsule, at a rate of 0.6-1.2 l·min$^{-1}$, and then passed through small chambers (one per capsule) containing humidity and temperature sensors. Sweat rate was determined from changes in RH and temperature of this air. Analog output from the sweat monitors was sampled at 0.2 Hz using a IBM compatible computer and a 16 channel, 12-bit analog: digital interface (DAS1602, MetraByte Corp, USA).

Sweat capsule sites were determined and positioned to maintain consistency across trials. Capsule sites were as follows: mid-forehead; right upper-arm (0.5 of the distance from acromioclavicular protuberance to the anticubital fossa, 2 cm laterally displaced); left lower
Figure 3.2 Schematic diagram of the capacitance hygrometry sweat monitoring system used to collect sweat rate and frequency data (from: Cotter, 1998).
arm (0.44 of the distance from the olecranon to the styloid process of the ulna); chest; intersecting point of mid-sternum and mid-clavicle; thigh; (0.5 of the distance from the inguinal crease to the superior tip of the patella); and scapula.

3.3.2.1 Calculation of sweat rate

The calculation of \( m_{sw} \), using ventilated capsules, required calculation of the increase in mass of water contained within each stream of air, after the air had passed across 3.15 cm\(^2\) or 2.19 cm\(^2\) of skin, for each location (Graichen et al., 1982). The following equation was used to quantify the water added to the air stream within each capsule by evaporation from the skin. This is taken as the \( m_{sw} \) (Taylor et al., 1996):

\[
fh_{sw} = \frac{(((RH*PH_{2O}*V)/(100*T_{cap}*3.464))-((RH_{exp}*PH_{2O}*V)/(100*T_{a}*3.464)))/A}
\]

Where:

- \( m_{sw} = \) mass flow of water off the skin in g.cm\(^{-2}\).min\(^{-1}\)
- \( RH = \) relative humidity obtained from the capacitance hygrometer once air has left the skin (%)
- \( RH_{exp} = \) relative humidity of water vapour entering the capsule at the target experimental temperature (%)
- \( V = \) airflow through the capsule measured from the rotameter (l.min\(^{-1}\))
- \( PH_{2O} = \) partial pressure of water vapour that would be obtained for air entering the capsule if it were 100% saturated (mmHg)
- \( T_{cap} = \) temperature of air leaving the sweat capsule (°K)
- \( T_{a} = \) ambient temperature (°K)
- 3.464 = (water vapour gas constant)
- \( A = 3.15 \) cm\(^2\) or 2.19 cm\(^2\)

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3.3.2.2. Calibration of sweat monitor

Humidity sensors were calibrated before testing at each of the ambient temperatures used during data collection. Air was passed over two saturated solutions which were used as RH standards for calibration (lithium chloride (RH =12.5%), and sodium chloride (RH =75%)) at a known temperature. Each solution was applied for 60 minutes prior to calibration to allow hygrometer and system stability. Essential to calibration was that the water mass prior to the capsule (measured at the saturated solutions) was identical to post-capsular air (measured at the hygrometer). It is extremely important that the flasks, capsules and lines to and from the capsules are carefully checked so no air escapes from the system. Consequently, any change in RH between these two values is due to a change in temperature, and is able to be calculated, since the temperature of air prior to the sweat capsule (T1), and the temperature of air after the sweat capsule (T2), are both measured concurrently. Calibration involved recording the voltage output for each of the hygrometers, at each of the conditions. A mathematical relation was then determined between the RH output voltage and the known RH voltage.

3.3.2.3. Determination of the sweat sensitivity

The sweat sensitivity was determined for each subject, at each of the six sites individually (forehead, upper-arm, forearm, scapular, chest and thigh). Sweat threshold determination was as follows:

(i) Data were graphed and magnified to display the region in which sweat threshold appeared to occur.

(ii) A region where $m_{sw}$ was first elevated for more than 5-min without returning to baseline, was chosen. This region allowed selection of pre and post-threshold sweat data, which were
then isolated and curve fitted, using first-order linear regressions.

(iii) Grid lines were then used to determine the point in time at which the sweat response deviated from the pre-threshold regression line. This point was termed the threshold for that site. The process was repeated at all six sites. These values were then averaged to obtain a mean sweat threshold time.

(iv) In situations in which sweat thresholds were difficult to distinguish, data on either side of the assumed threshold were fitted with first-order linear regressions. The point where these regression lines intercepted was taken as the sweat threshold.

(v) The $T_c$ at that mean sweat threshold time was used to derive the $T_c$ at which sweat threshold occurred.

Sweat rate sensitivity was determined from 5 minute averaged data of $m_{sw}$ against $T_{es}$. Once any obvious plateau was deleted, a first-order linear function was fit to the remaining data to obtain the slope ($mg.cm^{-2}.min^{-1}.°C^{-1}$).

3.3.2.4 Sweat expulsion frequency

Sweat expulsion frequency provides an index of the rate neural impulses are passed along the sympathetic fibres innervating the sweat glands (Yamazaki, et al., 1994). The $f_{sw}$ is determined by counting synchronous sweat expulsions which appears across all measurement sites, over fixed periods, within each trial.

Sweat expulsions were identified using sweating data from two test areas. Since it was not possible to obtain adequate sweat expulsion resolution from the existing electronic arrangement of the hygrometers, two channels of the sweat collection system were modified.
Taking into consideration that sweat expulsion is typically represented by a wave which increases rapidly and declines exponentially (Yamazaki, et al., 1994) both channels were AC-coupled (100uf capacitor). This was to increase the gain of the system, which filtered out any DC current to increase individual sweat expulsions resolution. By increasing the gain of the system, the signal to noise ratio was therefore also increased. A low pass filter (100K resistor) with a time constant set to 3 Hz, better enabled the resolution of individual sweat expulsions, to be determined by filtering any unnecessary inherent low frequency noise within the system. Using a low pass filter dramatically improved the signal resolution. The upper limit of the sweat expulsion frequency did not exceed 2 Hz.

The measurement of $f_{sw}$ required an hygrometer to be placed as close to its sweat capsule as possible (5.5 cm), to minimise the dampening effects associated with gas mixing within the tubing between the capsule and its hygrometer. A high airflow was also important when subjects were exercising (1.2 l·min$^{-1}$), to insure that water did not accumulate under the capsule, so that individual sweat expulsions did not merge and make it difficult to count individual sweat expulsions, producing a progressive saturation of the system. The hygrometer output for the $m_{sw}$ measurement was sampled at 2 Hz and digitally converted by IBM PC computer. Another modification was to use smaller capsules (2.19 cm$^2$) on these channels to help increase the flow to volume of surface area under the capsule and further decrease the possibility of progressive saturation.

3.3.2.5 Determination of sweat expulsion frequency

To determine sweat expulsion frequency:

(i) Prior to subject arriving, a 5 minute baseline of the system's noise was recorded. This
recording was taken after 40 minutes to allow sufficient time for the hygrometers to thermally equilibrate to the RH within the experimental environment. This was to determine the amount of inherent noise of the sweat capsule system, and to further help in determining $f_{sw}$ and minimise error.

(ii) Sweat rate data were plotted for the whole of the experimental condition (Figure 3.3a).

(iii) A four minute period of $m_{sw}$ at the commencement of data collection was plotted (Figure 3.3b).

(iv) The same four minute period of $f_{sw}$ determined from raw relative humidity data were plotted (Figure 3.3c).

(v) The same four minute period of $f_{sw}$ determined from the AC coupled channel was plotted (Figure 3.3d).

(vi) The mean (±95% confidence line) was determined for the noise taken prior to data collection of each trial. By determining the level of inherent noise within the system, we could differentiate between noise and individual sweat expulsions which therefore improved the accuracy with which the expulsions were counted.

(vii) The inherent noise of the system was then overlayed over each of the two minute periods of $f_{sw}$ which was plotted for each trial, this was to help minimise the error in its determination (Figure 3.4). By overlaying the noise on the signal it made it much easier to accurately determine the number of sweat expulsions per minute.

(viii) When counting individual sweat expulsions, it is important to note the synchrony of the signal across the sweat capsule sites. An example of the typical response of a subject during a trial (Figure 3.5). Figure 3.5 demonstrates that the amplitude changes throughout the trial, commencing with large distinctive expulsions. As body temperature increases and exercise continues, the expulsions become smaller and more frequent as more eccrine glands are
Figure 3.3 (A) A typical sweat response plotted for the whole experimental period (26.3°C ±0.3°C); (B) a four minute period of $r_{sw}$ at the commencement of data collection; (C) the same four minute period of $f_{sw}$ determined from raw relative humidity data; (D) the same four minute period of $r_{sw}$ determined from an AC coupled channel.
Figure 3.4 A two-minute period of $f_{sw}$ which has been overlayed over the inherent noise of the system. This helps minimise the error in determination of $f_{sw}$. 

Electronic noise (Hz)
Sweat rate (mg cm$^{-2}$ min$^{-1}$)
Figure 3.5 Typical sweat expulsion responses (forearm and forehead) of a subject recorded at six 2-minute intervals during a trial. Note the change in frequency and amplitude as the trial progresses.
recruited, it was this merging of moisture from many sweat glands that makes it difficult to count. Sweat expulsion frequency is typically represented as a peak which increases rapidly and declines exponentially (Yamazaki, et al., 1994), it is also advisable to use the original \( m_{sw} \) trace and utilise this to determine decreases and increases in \( f_{sw} \) from changes in \( m_{sw} \). It would be important to check that any rapid changes in \( f_{sw} \) and \( m_{sw} \) may be due to leaks or breakdowns within the system, and are not physiologically based.

(ix) Sweat expulsion frequency was determined at 6 different time intervals (2, 5, 10, 15, 20, and 25 minutes).

### 3.3.3 Cardiac frequency

Cardiac frequency \( (f_c) \) was monitored continuously from ventricular depolarisation at 0.2 Hz (Polar Electro SportTester, Model PE4000, Finland) and subsequently downloaded to computer for storage. Cardiac frequency, determined using this system, has been validated in our laboratory against a five-lead electrocardiogram (Figure 3.6: Osborne, 1994).

### 3.3.4 Psychophysical variables

Recordings of psychophysical variables were quantified prior to each experimental condition, and every five minutes throughout the experimental protocol. The thermal sensation scale was a modified version of that produced by Gagge et al. (1967), where the end points were extended to enable better resolution of the thermal sensation. The question was asked: "How does the temperature of your body feel?" and subjects responded on a scale of 1-13: 1 = unbearably cold ; 13 = unbearable hot.

Ratings of perceived exertion (RPE) or effort sensation, were obtained for the whole
Figure 3.6 A comparison of cardiac frequency obtained using a Sport Tester with that derived using a five lead ECG during seated rest, cycle exercise at 150 W, and seated recovery (Osborne, 1994).
body, and fractionated for the chest, and legs. Data were collected five minutes following the commencement of exercise, and at 5-minute intervals thereafter. The question was asked: "How hard do you feel you are exercising your whole body/chest/legs?". Subjects gave an answer for each region on a scale of 6-20 (Borg, 1962; 6=very very light; 20=very very hard). Differential RPE scores were used since subjects perceive exertion according to the manner in which they experience the stress, as demonstrated by Pandolf (1979). Thus, the legs may be perceived as the site for greatest physiological strain in a subject not accustomed to leg exercise. Also the fractionated RPE provided a means to evaluate effort sense during cycling at different pedal frequencies.

3.4 DESIGN AND ANALYSIS

This experiment was based upon a single, factorial design, (heat stress), with subjects fully crossed for all five levels of this factor (rest, two active exercise conditions and two passive exercise conditions). Testing sequence was ordered with the active exercise trials being performed first, then the passive exercise and seated resting trials were randomly ordered to minimise order effects. Data are presented as means with standard errors of the means. Data were analysed using repeated-measures analysis of variance. Tukey’s HSD post hoc procedure was used to evaluate sources of significant differences. Alpha was set at the 0.05 level for all analyses.
4.1 BODY TEMPERATURE CHANGES

4.1.1 Skin temperatures

The water-perfusion garment was used to increase and regulate $T_c$ at similar rates across the two experimental conditions for the five trials. The garment controlled $T_{sk}$ adequately, though differences did occur with variations in garment fit for each subject. The differing environmental conditions and combined effect of the water-perfusion garment temperatures resulted in a significantly elevated $T_{sk}$ in the passive (40 and 80 rev.min$^{-1}$) and resting trials, relative to the active trials ($p<0.05$) (Figure 4.1). Mean skin temperature averaged 33.03°C ($\pm$0.33) ($\bar{x} \pm$ SEM; this value is the standard error of the means) and 32.85°C ($\pm$0.32) for the active 40 and 80 rev.min$^{-1}$ exercise trials, while the passive 40 and 80 rev.min$^{-1}$ exercise and seated resting trials averaged 36.52°C ($\pm$0.33), 36.69°C ($\pm$0.32), and 36.65°C ($\pm$0.41), respectively. Differences in $T_{sk}$ and $T_a$ between the two experimental conditions may have influenced sympathetic activity associated with the regulation of sweating and resultant sweat output.

Mean skin temperature did not significantly differ between the active (40 and 80 rev.min$^{-1}$) exercise trials ($p>0.05$). Analysis revealed there were no significant differences between the passive (40 and 80 rev.min$^{-1}$) and seated resting trials, either ($p>0.05$) (Figure 4.1). These data from the five trials confirm that $T_{sk}$ and therefore thermal load, differed considerably between the two different environmental conditions. An increased $T_{sk}$ from the
Figure 4.1 (A) Changes in core temperatures during experimental trials, where $T_c$ was held constant with the use of a water-perfusion garment, in the passive exercise and seated resting trials. (B) Skin temperature changes during the five experimental trials. (C) Resultant changes in mean body temperature. Data are means and standard errors of the means. Significant differences are indicated by an asterisk ($p<0.05$).
water-perfusion garment temperatures would have increased local vasodilation to peripheral tissues (Rowell, 1983). This would also have been exacerbated by the higher environmental temperature of these trials. It was the aim of this study to evaluate sudomotor function from differences in $f_{sw}$, which was primarily driven by $T_{sk}$ in the passive exercise and seated resting trials. Technical difficulties associated with the measurement of $T_{sk}$ hampered the investigation into the mechanisms that regulate sweating and differentiate non-thermal influences in sudomotor function. It became evident that complete resolution of these difficulties was beyond the scope of this project, though it was still possible to address the primary aim of the thesis.

Skin temperature changed rapidly in the initial 10 minutes of the passive exercise and resting trials in comparison to the active exercise trials. The passive exercise trials at 40 and 80 rev.min\(^{-1}\), as well as, the resting trial, had slope values of 0.30 (±0.02), 0.29 (±0.04), 0.34°C.min\(^{-1}\) (±0.04), compared to that of the active 40 and 80 rev.min\(^{-1}\) exercise trials which had values of 0.17 (±0.01) and 0.17°C.min\(^{-1}\) (±0.02), respectively. Differences in the rate of change of $T_{sk}$ between the two environmental conditions were derived from a direct local heating (using an external source) to heat the skin, and reflex control of the vasoactive state of the skin (metabolic heat production). These methods used may have caused differences in peripheral circulation and sympathetic activity between the two experimental environments, further affecting the regulation of sweating.

The rates of changes in $T_{sk}$ of the active exercise trials for the period immediately following (15-25 minutes) were very similar to those of the passive and seated resting trials. The averaged $T_{sk}$ slope values for the active 40 and 80 rev.min\(^{-1}\) exercise trials were 0.04
(-0.01) and 0.02°C.min⁻¹ (+0.01), respectively. The rates of changes in $T_{sk}$ for the passive 40 and 80 rev.min⁻¹ exercise changes were similar for 15-25 minute time period (0.02°C.min⁻¹ ± 0.01) whilst the seated resting trials were slightly different (0.03°C.min⁻¹ ± 0.01). These data support the attainment of a uniform and consistent $T_{sk}$ change between trials, allowing comparisons to be made during the 15-25 minute period of data collection. The slope change of the active 40 rev.min⁻¹ exercise and seated resting trials differed slightly from the other trials, which may have been related to differences in individual responses to the experimental methodology.

4.1.2 Body Core Temperatures

The mean core temperatures did not significantly differ during the experimental data collection for the five trials (p>0.05). The $T_c$ in the passive exercise and seat resting trials were slightly higher than that of the active exercise trials in the first 2 minutes of data collection, though analysis revealed no significant difference (p>0.05). This difference may be accounted for by the higher $T_a$ and the effects of the water-perfusion garment's hot water temperatures.

For the 25 minutes of data analysis, $T_c$ averaged 37.49°C (+0.13) and 37.49°C (+0.10) in the active 40 and 80 rev.min⁻¹ exercise trials, respectively. The passive (40 and 80 rev.min⁻¹) exercise and seated resting trials averaged 37.23°C (+0.03), 37.41°C (+0.07), and 37.26°C (+0.03), respectively (Figure 4.1). Except for the initial few minutes of the passive exercise and seated resting trials, the $T_c$ in the active exercise trials were slightly higher across all of the experimental conditions, but showed no large variations.
There was a constant temperature offset between the three core temperature indices (Figure 4.2) with $T_{au}$ being $0.12^\circ C$ (±$0.07$) higher than $T_{re}$ and $T_{re}$ being $0.30^\circ C$ (±$0.05$) higher than $T_{es}$. Of the three measurements of $T_c$, $T_{es}$ showed the least variance across the five experimental trials. The measurements of $T_{re}$ and $T_{au}$ showed greater variance, which may indicate that these measurements were biased by experimental factors. The rectal temperatures may have been influenced by the lower body exercise of the experimental trials. Aural thermometry may have been biased by the ambient conditions, as well as that of the higher $T_{sk}$ due to the temperature of the water-perfusion garment during that of the passive exercise and seated resting trials. Across the five experimental trials, the passive 40 rev.min$^{-1}$ exercise trial had the lowest $T_c$.

Prior to entering the environmental chamber to the end of data collection, $T_c$ did not significantly differ, across the five experimental conditions ($p>$0.05). The clamping of core temperature change across the five experimental trials was therefore deemed to be successful. The total change in $T_c$ for active 40 rev.min$^{-1}$ exercise trial was $1.19^\circ C$ (±$0.06$) and for the passive 40 rev.min$^{-1}$ exercise trial it was $1.13^\circ C$ (±$0.04$). In the active 80 and passive 80 rev.min$^{-1}$ exercise trials similar changes occurred with total temperature changes averaging, $1.18^\circ C$ (±$0.09$) and $1.21^\circ C$ (±$0.05$), respectively. The resting trial compared similarly with the active and passive exercise trials with an averaged $T_c$ change of $1.17^\circ C$ (±$0.10$), suggesting that the combination of ambient conditions and perfusion garment's water temperatures acted to clamp $T_c$. The passive exercise (40 and 80 rev.min$^{-1}$) and resting trials average rate of change was $0.04^\circ C$.min$^{-1}$, this being the same for the active exercise (40 and 80 rev.min$^{-1}$) trials which averaged $0.04^\circ C$.min$^{-1}$, as well. The total $T_c$ change and the rate of temperature change was similar across the five trials, though this may be misleading because
Figure 4.2 Auditory canal, oesophageal, rectal, and mean core temperatures from 25 minutes of data collection. Active 40 and 80 rev.min$^{-1}$ exercise conditions were at 26.3 (±0.32) and 26.8°C (±0.08) with a relative humidity of 60.2 % (±1.2). Passive 40 and 80 rev.min$^{-1}$ exercise conditions were at 40.5 (±0.35) and 39.8°C (±0.27) with a relative humidity of 60.2 % (±0.2). The resting condition was similar at 40.3°C (±0.35). Data are means and standard error of the means.
initially in the active exercise trials $T_c$ was increasing, while $T_c$ in the passive exercise and seated resting trials was declining. These initial differences in $T_c$ may have influenced efferent sympathetic activity associated with the control of peripheral circulation and the regulation of sweating.

During the active exercise trials, $T_c$ tended to increase rapidly for the first 10 mins, after which there was a gradual but steady elevation of $T_c$ with time. In the passive and seated resting trials, $T_c$ initially declined at the beginning, caused by a combination of experimental factors; the high temperature of the water flowing through the water-perfusion garment, elevating $T_{sk}$ artificially; and the hotter conditions of the environment within which the data was collected. In the passive and resting trials, $T_c$ increased consistently with the combination of the suit's water temperatures and the passive movement after the initial decline, until the cessation of the trials.

4.1.3 Mean body temperature

The Core temperature measurement used for the calculation of $T_b$ was $T_{es}$. This was used instead of $T_c$ as $T_{es}$ had less bias from the experimental methodology such as ambient conditions, lower body exercise and water-perfusion garment temperatures elevating $T_{sk}$ in the passive exercise and seated resting trials. Elevation of $T_b$ in the passive 40 and 80 rev.min$^{-1}$ exercise trials, as well as that of the seated resting trials was expected because of the higher $T_{sk}$. The passive 40 and 80 rev.min$^{-1}$ exercise trials averaged $37.0^\circ C (\pm 0.11)$, $37.1^\circ C (\pm 0.11)$ whilst the seated resting trials averaged $37.0^\circ C (\pm 0.13)$, respectively. This was in comparison to the active 40 and 80 rev.min$^{-1}$ exercise trials which averaged $36.4^\circ C (\pm 0.16)$ and $36.4^\circ C (\pm 0.11)$. This difference in $T_b$ may be accounted for by the increased $T_{sk} (3.8^\circ C)$
of the passive exercise and seated resting trials in comparison to the active exercise trials, illustrated in Figure 4.1. Analysis revealed no significant differences between the experimental conditions ($p>0.05$).

### 4.1.4 Normalised temperatures

For the five experimental trials, $T_{es}$, $T_{sk}$ and $T_b$ were normalised to a common value. This was taken as the temperature observed at 2 minutes during the active 40 rev.min$^{-1}$ exercise trial. This normalisation minimised the confounding affect of using the water-perfusion garment to control $T_c$ in the passive exercise and seated resting conditions (Figure 4.3). The averaged total change in $T_{es}$ across all five trials was consistently higher in the active exercise trials than the passive exercise and seated resting trials, though there was no significant differences between the 40 and 80 rev.min$^{-1}$ for either the passive or active trials ($p>0.05$). The active 40 and 80 rev.min$^{-1}$ exercise trials averaged $0.60^\circ C \pm 0.10$ and $0.50^\circ C \pm 0.08$, whilst the passive 40 and 80 rev.min$^{-1}$ exercise trials and seated resting trials, averaged $0.33^\circ C \pm 0.05$, $0.37^\circ C \pm 0.06$ and $0.29^\circ C \pm 0.04$, respectively.

As expected the averaged total change in $T_{sk}$ was consistently greater in the passive exercise and seated resting trials than that of the active exercise conditions. The passive 40 and 80 rev.min$^{-1}$ exercise trials and seated resting trials averaged, a total change of $2.99^\circ C \pm 0.47$, $2.66^\circ C \pm 0.43$ and $3.64^\circ C \pm 0.57$ in comparison to the active 40 and 80 rev.min$^{-1}$ exercise trials which averaged $2.23^\circ C \pm 0.39$ and $2.02^\circ C \pm 0.36$, respectively. These data, provide evidence that the results differed for the active, passive and seated resting trials between the two experimental protocols.
Figure 4.3 (A) Changes in normalised mean core temperatures during experimental trials, where $T_c$ was held constant with the use of a water-perfusion garment in the passive exercise and resting trials. (B) Changes in normalised mean skin temperature. (C) Resultant changes in normalised mean body temperature. Data are means and standard errors of the means.
The active 40 and 80 rev.min\(^{-1}\) exercise trials averaged change in normalised $T_b$ were $0.92°C (±0.38)$ and $0.80°C (±0.33)$, in comparison to the passive 40 and 80 rev.min\(^{-1}\) exercise and seated resting trials, which averaged $0.70°C (±0.26)$, $0.75°C (±0.28)$ and $0.81°C (±0.30)$, respectively. The averaged total change in normalised $T_b$ was highest in the active 40 rev.min\(^{-1}\) exercise with the active 80 rev.min exercise comparing very closely with that of the seated resting trial. The total average change in $T_b$ for the passive 40 and 80 rev.min\(^{-1}\) exercise trials were very similar as well. The averaged total rate of change for $T_b$ was the same for all five experimental trials ($0.03°C.min^{-1}$) which was very similar to the total averaged change of $T_c$ across the experimental data collection period ($0.04°C.min^{-1}$). The changes in sweat output and sweat expulsion frequency could therefore be said to be related to thermal influences.

4.2 CARDIAC FREQUENCY

4.2.1 Active exercise responses

Cardiac frequency was similar between the active 40 and 80 rev.min\(^{-1}\) exercise trials, with no significant difference, at any time period ($p>0.05$) (Figure 4.4a), averaging 139.47 ($±3.11$) b.min\(^{-1}\) and 142.56 ($±3.72$), respectively. The $f_c$ for the active 40 and 80 rev.min\(^{-1}\) exercise trials followed the same consistent trend of uniform change for the duration of the experiment, though the $f_c$ for the active 80 rev.min\(^{-1}\) exercise trial was always slightly greater than that of the active 40 rev.min\(^{-1}\) exercise trial. The change in $f_c$ from the start to the end of the data collection period averaged 19.95 b.min\(^{-1}\) for the active 40 rev.min\(^{-1}\) exercise trials compared with that of 24.65 b.min\(^{-1}\) for the active 80 rev.min\(^{-1}\) exercise trials.
Figure 4.4 Changes in cardiac frequency for the duration of the five experimental trials. Differences in thermal strain between cardiac frequency and mean body temperature. Active 40 and 80 rev.min$^{-1}$ exercise conditions were at 26.34 (±0.32) and 26.80°C (±0.08) with a relative humidity of 60.2% (±1.2). Passive 40 and 80 rev.min$^{-1}$ exercise conditions were at 40.52 (±0.35) and 39.88°C (±0.27) with a relative humidity of 60.2% (±0.2). The resting condition was similar at 40.31°C (±0.35). Data are means and standard errors of the means. Significant differences are indicated by an asterisk (p<0.05).
Cardiac frequency for the passive 40, 80 rev.min\(^{-1}\) exercise and seated resting trials averaged 94.29 (±3.25), 98.33 (±3.41), and 105.96 (±5.09) b·min\(^{-1}\), respectively, with no significant differences (p>0.05). By the end of data collection \(f_c\) had changed by an average of 20.28 b·min\(^{-1}\) for the passive 40 rev.min\(^{-1}\) exercise trials, compared with that of 20.72 b·min\(^{-1}\) for the passive 80 rev.min\(^{-1}\) exercise trials. The seated resting trials had a greater change in \(f_c\) (33.48 b·min\(^{-1}\)) for the same corresponding period. The large difference in \(f_c\) between the active exercise, passive exercise and seated resting trials, may be explained by the two different experimental conditions.

The total change in \(f_c\) from the start to the end of the experiment was similar across the five trials, with the largest change occurring in the seated resting trial (33.48 b·min\(^{-1}\)). This may indicate that the thermal load of this trial was the greatest. The seated resting trial was the only experimental trial that involved no movement. This may suggest that the movement of the legs in the active and passive exercise trials, helped to lower the thermal strain of the conditions by convective cooling. This convective cooling helped to balance positive heat storage, which helped to lower the thermal strain on the cardiovascular system. This is evident with the consistently lower \(f_c\) of the passive exercise trials. The seated resting trials was elevated by 11.67 and 7.63 b·min\(^{-1}\) in comparison to the passive 40 and 80 rev.min\(^{-1}\) exercise trials. These differences in \(f_c\) between trials may be related to shifts in blood volume associated with cutaneous vasodilation and increased intra-muscular blood flow, which was met by increased cardiac output due to stimulation of central and cutaneous thermoreceptors during the exercise trials (Rowell, 1983).
The similarities in total change of $f_c$ between the five experimental trials, and especially, the active and passive exercise trials may help to indicate that similar amounts of total work were performed over the duration of the experimental trials. Similar change in $f_c$ across the five experimental trials may suggest that the same amount of work was performed for the active and passive exercise, which may suggest that the role of mechanoreceptors across the four trials was consistent. If the same amount of work was performed during the active and passive exercise trials then differences in $f_c$ for the active exercise conditions may be attributed to the thermal load generated metabolically and not external heat sources.

The large difference in $f_c$ (45.18 b·min$^{-1}$) between the active and passive 40 rev.min$^{-1}$ exercise trials was consistent with the active and passive 80 rev.min$^{-1}$ exercise trials which had a similar mean difference (44.23 b·min$^{-1}$) verifying that these trials were truly passive. Analysis revealed that there was a significant difference between the active and passive 40 and 80 rev.min$^{-1}$ exercise trials, as well as that of the active 40 and 80 rev.min$^{-1}$ exercise and seated resting trials ($p<0.05$) (Figure 4.4a).

The comparison between $f_c$ and $T_b$ in Figure 4.4b, reinforces two distinctly different patterns between the active and passive exercise, as well as that of the active exercise and seated resting trials. This Figure (4.4b) also helps to reinforce that the thermal load between the two experimental conditions was different. The differences in $f_c$ between the two experimental conditions may reflect the differences in demand for peripheral blood flow. These differences in haemodynamics may be associated with increases in intra-muscular blood flow due to vasodilation of capillary beds in the passive exercise and seated resting trials.
4.3 PSYCHOPHYSICAL VARIABLES

4.3.1 Perceived exertion

The changes in perceived exertion (RPE) were uniform and consistent for the two environmental conditions as illustrated in Figure 4.5a. These data support that the \( f_c \) of the passive exercise trials were truly passive. The RPE for the active exercise conditions differed significantly from those of the passive exercise and resting trials \( (p<0.05) \). The active 40 and 80 rev.min\(^{-1} \) exercise trials averaged, 13.80 (±0.29), 13.85 (±0.38), while that of the passive 40, 80 rev.min\(^{-1} \) exercise and resting trials averaged, 6.98 (±0.12), 7.23 (±0.20) and 7.39 (±0.30), respectively. An unexpected result was a slight increase in RPE, which occurred from 20-25 minutes of data collection. This indicates that RPE may have been biased from an increase in heat storage and an inability to maintain a thermal balance at this point in time. The increase in RPE scores was more apparent in the active and passive 80 rev.min\(^{-1} \) exercise trials, which changed by 0.6, while that of the active and passive 40 rev.min\(^{-1} \) exercise trials changed by 0.2 for this five minute period. These data, though not significant, may suggest that these differences were related to differences in the speed of leg movement (40 and 80 rev.min\(^{-1} \)) in the active and passive exercise trials \( (p>0.05) \).

Previous research (Maw et al., 1993) indicates that RPE is most responsive to \( T_{sk} \) and \( f_c \), though there has not been a lot of investigation on the influence that \( T_b \) has on RPE scores. The high \( T_{sk} \) and higher \( T_b \) associated with lower \( f_c \) and RPE scores of the passive exercise and seated resting trials which were conducted in hot environmental conditions, differ in comparison to the active exercise trials of high RPE scores, low \( T_{sk} \) and lower \( T_b \) with higher \( f_c \) which were conducted in temperate environmental conditions (Figure 4.5b). This may
Figure 4.5 (A) Changes in perceived exertion during 25 minutes of experimental trials. (B) Changes of perceived exertion in relation to mean body temperature. Data are means and standard errors of the means. Significant differences are indicated by an asterisk ($p<0.05$).
indicate that $f_c$ has a greater influence on effort perception than previously thought.

4.3.2 Thermal sensation

The thermal sensation of the five experimental trials followed a similar trend with analysis revealing no significant differences, between trials ($p>0.05$) (Figure 4.6a). The elevation of thermal sensation in the passive exercise and seated resting trials occurred at a higher $T_b$ and lower $f_c$ than that of the active exercise trials (Figure 4.6c and 4.6d). The active 40 and 80 rev.min$^{-1}$ exercise trials averaged, 9.30 (±0.46), 9.12 (±0.43), while the passive 40, 80 rev.min$^{-1}$ exercise and resting trials averaged, 9.84 (±0.66), 9.55 (±0.61) and 10.18 (±0.67), respectively. The higher mean thermal sensation of the resting condition compared to the other four experimental trials, indicates a higher thermal strain, which supports previous data that the passive leg movement of the passive exercise trials, helped to lower the thermal strain by convective cooling.

Previous research indicates that thermal sensation depends primarily on $T_{sk}$ (Hensel, 1981). From data observed in Figure 4.6b the slightly higher thermal sensation associated with higher $T_{sk}$ of the passive exercise and seated resting conditions supports this previous finding of Hensel. Though, combining data from this study for both RPE and thermal sensation it would seem apparent that $T_b$ is the primary determinant to RPE and thermal sensation, which differs with the current consensus on this issue (Figure 4.6c).

4.3.3 Summary

Two different environmental conditions were used to create similar physiological changes across the five experimental trials. One of the major thermal determinants ($T_{sk}$) was
Figure 4.6 (A) Thermal sensation changes for the five experimental trials for 25 minutes of data collection. (B) Changes in thermal sensation in relation to changes in mean skin temperature. (C) Thermal sensation and its changes in relation to changes with mean body temperature. (D) Changes for thermal sensation with concurrent changes in cardiac frequency. Data are means and standard errors of the means.
not successfully controlled across all five trials. The unsuccessful control of $T_{sk}$ can be attributed to the higher $T_s$ of the passive and seated resting trials and the hot temperature of water used in the water-perfusion garment. This disparity of results may have influenced sympathetic activity associated with different demands for blood flow. As $T_{sk}$ was different between the two environments, therefore, $T_b$ would be as well. Core temperature and the rate of $T_c$ change was successfully controlled across the five trials. Cardiac frequency differed between the active exercise and passive exercise trials, as well as that of the seated resting trials. The large difference in $f_c$ of the passive exercise trials in comparison to the active exercise trials reinforced that the passive exercise was of a passive nature. Though, $f_c$ differed between the five trials, similar total changes from start to the end of the experiment occurred in the passive and active exercise trials, indicating that similar amounts of work had been achieved, therefore, helping to control the role of mechanoreceptors, which was therefore consistent across the trials. Perceived exertion differed between the trials due to the differences in environments and resulting differences in $T_{sk}$. There were no differences in thermal sensation between the trials, though $T_b$ would seem to be a major determinant in thermal sensation.

4.4 SWEAT RATE CHANGES DURING EXPERIMENTAL MANIPULATIONS

4.4.1 Central and peripheral local sweat rates

Raw data of a typical $m_{sw}$ response of one subject to each of the five experimental conditions is represented in Figure 4.7. The active 40 and 80 rev.min$^{-1}$ exercise trials (4.7a and 4.7c) depict similar and gradual changes in $m_{sw}$ over the 25 minutes of the experiment. The forehead sweat site was consistently the highest output of the 6 sweat sites measured.
Figure 4.7 Typical sweat response of one subject at the six local sweat collection site for 25 minutes of data collection, for the five experimental trials. (A) This is the active 40 rev.min\(^{-1}\) exercise trial, which is very uniform. (B) The passive 40 rev.min\(^{-1}\) exercise trial. (C) The active 80 rev.min\(^{-1}\) exercise trial. (D) The Passive 80 rev.min\(^{-1}\) exercise trial. (E) The seated resting trial. Data are means for the individual.
This result was consistent with the combined averaged data of all the subjects for the individual sweat sites over the 5 experimental trials (Figure 4.8). The passive 40 and 80 rev.min\(^{-1}\) exercise and seated resting trials patterns of m\(_{sw}\) were not similar in appearance to those of the active exercise trials, though the individual sweat sites displayed similar secretion output patterns, such that forehead was typically the greatest sweat output (Figure 4.7b, 4.7d and 4.7e). The forearm and scapula sites were the next highest sweat output sites, except in the passive 40 rev.min\(^{-1}\) exercise trial, where the order was reversed.

Combining the raw data for all subjects, more consistent patterns emerged (Figure 4.8). The active and passive 40 rev.min\(^{-1}\) exercise trials elicited a similar secretion pattern in m\(_{sw}\), which in sequence from highest to lowest was: forehead, forearm, scapula, thigh, chest and then the arm sweat site (Figure 4.8a and 4.8b). The active and passive 80 rev.min\(^{-1}\) exercise and seated resting trials had a similar trend except for the passive 80 rev.min\(^{-1}\) exercise trial where the chest sweat site was slightly greater than that of the thigh (Figure 4.8d).

The six sweat collection sites were divided into two groups to see if there was a difference between central and peripheral sweating. The central m\(_{sw}\) sites consisted of the forehead, scapula and chest, while the peripheral sites consisted of the forearm, thigh and arm. When dissecting the data for time periods and experimental trials it was during the initial 10 minutes of data collection in which the greatest changes and differences occurred. The central and peripheral m\(_{sw}\) sites demonstrated similar trends for m\(_{sw}\) for the five trials (Figures 4.9a and 4.10a). Analysis revealed significant differences at 2 minutes for the forehead and forearm site, between the active exercise trials, the passive exercise and seated
Figure 4.8 The mean data of the six local sweat collection sites used. (A) The sweating response of the six sites of the active 40 rev.min⁻¹ exercise trials. (B) The passive 40 rev.min⁻¹ exercise trials. (C) The active 80 rev.min⁻¹ exercise trials. (D) The passive 80 rev.min⁻¹ exercise trials. (E) The seated resting trials. Data are means and standard errors of the means.
Figure 4.9 The central local sweat sites of the forehead (a), scapula (b) and chest (c) used for data collection. Evident from averaged data for all 5 experimental trials the central $m_{sw}$ sites showed a consistently higher $m_{sw}$ than those of the peripheral $m_{sw}$ sites seen in Figure 4.10. Data are means and standard errors of the means.
Figure 4.10 The peripheral local sweat collection sites of the forearm (a), thigh (b) and arm (c) used for data collection. From combined data for all 5 experimental trials the peripheral $m_{sw}$ sites showed a consistently lower $m_{sw}$ than those of the central sweat collection sites. Data are means and standard errors of the means.
resting trials ($p<0.05$). This difference in forehead and forearm $m_{sw}$ may be attributed to changes in $T_{sk}$ and $T_a$ between the two experimental environments. The forehead $m_{sw}$ of the active 40 and 80 rev.min$^{-1}$ exercise trials averaged, 0.23 and 0.31 mg.cm$^2$.min$^{-1}$ at 2 minutes, in comparison to 1.21, 0.91 and 1.31 mg.cm$^2$.min$^{-1}$ for the passive 40 and 80 rev.min$^{-1}$ exercise, as well as that of the seated resting trials, respectively (Figure 4.9a). The forearm $m_{sw}$ at 2 minutes averaged, 0.17 and 0.23 mg.cm$^2$.min$^{-1}$ for the active 40 and 80 rev.min$^{-1}$ in comparison to 0.63, 0.76 and 0.93 mg.cm$^2$.min$^{-1}$ for the passive 40 and 80 rev.min$^{-1}$ exercise and seated resting trials, respectively.

As evident from averaged data, the central local $m_{sw}$ sites showed a consistently higher $m_{sw}$ (forehead: 1.47 mg.cm$^2$.min$^{-1}$; scapula: 0.88 mg.cm$^2$.min$^{-1}$; chest 0.59 mg.cm$^2$.min$^{-1}$) than those of the peripheral sites (forearm 1.13 mg.cm$^2$.min$^{-1}$; thigh: 0.66 mg.cm$^2$.min$^{-1}$; arm: 0.44 mg.cm$^2$.min$^{-1}$). Also, for all sweat collection sites, across the five experiment trials, the $m_{sw}$ for the active and passive 80 rev.min$^{-1}$ exercise trials were consistently greater than that of the active and passive 40 rev.min$^{-1}$ exercise trials, except on one occasion. That occasion was the arm sweat site for the active 40 rev.min$^{-1}$ exercise trial and the active 80 rev.min$^{-1}$ exercise trial, which resulted in $m_{sw}$ of 0.42 ($\pm0.10$) and 0.37 ($\pm0.07$) mg.cm$^2$.min$^{-1}$, respectively.

Data for forehead and forearm $m_{sw}$ sites were added together and averaged then compared with data for the scapula, chest, arm, and thigh (Figures 4.11a and 4.11b). The two groups of $m_{sw}$ sites differed considerably, as the two highest $m_{sw}$ sites of forehead and forearm were combined and compared with the other sites used. The $m_{sw}$ for the combined group of scapula, chest, thigh and arm compared similarly across the five experimental trials,
Figure 4.11 Combined $\dot{m}_w$ data for forehead and forearm in comparison with data for scapula, chest, arm and thigh for the five experimental trials. Data are means and standard errors of the means.
except initially, the active exercise trials differed slightly with the passive exercise and seated resting trials. These initial differences can be attributed to the different environments and $T_{sk}$ of these trials. The active and passive 40 rev.min$^{-1}$ exercise trials averaged 0.61 (± 0.12) and 0.58 mg.cm$^2$.min$^{-1}$ (±0.06), while that of the active and passive 80 rev.min$^{-1}$ exercise trials, as well as that of the resting trial averaged, 0.66 (±0.13), 0.73 mg.cm$^2$.min$^{-1}$ (±0.09) and 0.59 mg.cm$^2$.min$^{-1}$ (±0.06), respectively. These data confirm that the 80 rev.min$^{-1}$ exercise trials produced a greater $m_{sw}$ than the 40 rev.min$^{-1}$ exercise trials for these $m_{sw}$ sites.

The $m_{sw}$ of forehead and forearm did not show the same consistency for the five trials as was evident with the group of scapula, chest, thigh and arm. The active exercise trials showed the most uniformity across the experimental trials, while the seated resting trial was consistently the greatest $m_{sw}$. The passive exercise trials showed no similar pattern for the duration of the trials. The active and passive 80 rev.min$^{-1}$ exercise trials averaged 1.26 (±0.24) and 1.23 mg.cm$^2$.min$^{-1}$ (±0.15), while that of the active and passive 40 rev.min$^{-1}$ exercise trials, as well as that of the seated resting trial averaged, 1.04 (±0.21), 1.33 mg.cm$^2$.min$^{-1}$ (±0.15), and 1.67 (±0.16) mg.cm$^2$.min$^{-1}$, respectively. The seated resting trial was consistently the largest $m_{sw}$ which supports the notion that the leg movement of the active and passive exercise trials helped to lower the thermal strain on the cardiovascular system by convective cooling.

Analysis of $m_{sw}$ revealed no significant differences between the means of each experimental trial ($p>0.05$) (Figure 4.12). Though, there were significant differences between the means of the active exercise when compared to the passive exercise and seated resting trials, at 2 minutes ($p<0.05$). Sweat rates for the active 40 and 80 rev.min$^{-1}$ exercise trials
Figure 4.12 The mean sweat rate responses for 25 minutes of data collection for the five experimental trials. Data are means and standard errors of the means. Significant differences are indicated by an asterisk ($p<0.05$).
showed similar trends though analysis revealed no significant differences, which averaged, 0.75 (±0.15) and 0.84 (±0.19) mg.cm².min⁻¹, respectively (p>0.05). The slightly higher averaged \( \dot{m}_{sw} \) in the active 80 rev.min⁻¹ exercise trials was consistent with the hypothesised results of this study, that the higher pedal frequency would produce higher sweat rates than those of the lower pedal frequency (active 40 rev.min⁻¹) exercise trials, though these differences were not significant. This may reinforce the suggestion that mechanoreceptors play a role in the regulation of sweating, and the notion of a feedforward control being involved in the initiation of \( \dot{m}_{sw} \), prior to a change in \( T_c \).

The passive 40 and 80 rev.min⁻¹ exercise trials also showed consistent changes, which was initially much greater than that of the active 40 and 80 rev.min⁻¹ exercise trials. The higher initial \( \dot{m}_{sw} \) was related to the high temperatures of the water-perfusion garment and the hotter \( T_a \) of the passive exercise and seated resting trials. The \( \dot{m}_{sw} \) of the passive 40 and 80 rev.min⁻¹ exercise trials averaged, 0.83 (±0.10) and 0.88 (±0.12) mg.cm².min⁻¹, whilst the resting trials, averaged, 0.96 (±0.10) mg.cm².min⁻¹. There were no significant differences between these trials (p>0.05) (Figure 4.12). The \( \dot{m}_{sw} \) of the passive 80 rev.min⁻¹ exercise trial was greater than that of the passive 40 rev.min⁻¹ exercise trial, though no significant differences were found.

At 2 minutes, the active (40 and 80 rev.min⁻¹) exercise trials averaged 0.15 (±0.02) and 0.17 (±0.02) mg.cm².min⁻¹, while that of the passive (40 and 80 rev.min⁻¹) exercise, as well as that of the seated resting trials, averaged 0.60 (±0.09), 0.60 (±0.12) and 0.66 (±0.09) mg.cm².min⁻¹, respectively (p<0.05). Again, these data confirm that the initial differences in \( \dot{m}_{sw} \) were related to differences in \( T_{sa} \) and \( T_a \), as \( T_c \) did not have enough time to change and
influence $m_{sw}$. Another possible explanation may relate to differences in effort between the active and passive exercise trials.

Comparison of $m_{sw}$ for the active and passive exercise trials from 15-25 minutes showed corresponding equivalent changes (Figure 4.12). The traces of $m_{sw}$ for the active 80 rev.min$^{-1}$ exercise and seating resting trials for the same period were very similar. The $m_{sw}$ traces of the active 40 rev.min$^{-1}$ exercise trial during the period of 15-25 minutes mimicked that of the passive 40 rev.min$^{-1}$ exercise trial, while the active and passive 80 rev.min$^{-1}$ exercise were similar but did not overlap each like the 40 rev.min$^{-1}$ exercise trials (Figure 4.12).

Another interesting result, was that the total change in $m_{sw}$ was higher in the active and passive 80 rev.min$^{-1}$ exercise trials (1.08 and 0.59 mg.cm$^2$.min$^{-1}$) than the active and passive 40 rev.min$^{-1}$ exercise trials (0.96 and 0.51 mg.cm$^2$.min$^{-1}$). The total change in $m_{sw}$ were very similar between the passive 80 rev.min$^{-1}$ exercise trials (0.59 mg.cm$^2$.min$^{-1}$) and that of seated resting trial (0.58 mg.cm$^2$.min$^{-1}$; $p<0.05$). These data, may indicate that the differences in pedal frequencies may have influenced $m_{sw}$, but this affect failed to reach significance.

The sensitivity of the sweating response was determined from 5 minute averaged data of $m_{sw}$ against $T_{es}$. Once any obvious plateau was deleted, a first order linear function was fit to the remaining data to obtain the slope. The active exercise trials were significantly different from those of the passive exercise and resting trials ($p<0.05$). The active 40 and 80 rev.min$^{-1}$ exercise trials averaged, 1.23 mg.min$^{-1}$.cm$^2$.°C$^{-1}$ (±0.17) and 1.22 mg.min$^{-1}$.cm$^2$.°C$^{-1}$ (± 0.20),
while the passive 40 and 80 rev.min⁻¹ exercise and resting trials averaged, 0.47 mg.min⁻¹.cm⁻²°C⁻¹ (±0.08), 0.57 mg.min⁻¹.cm⁻²°C⁻¹ (±0.11) and 0.73 mg.min⁻¹.cm⁻²°C⁻¹ (±0.13), respectively. The discrepancy of results in sweat rate sensitivity between the active exercise, passive exercise and seated resting trials may be explained by the differences in the experimental environmental conditions and the use of the water-perfusion garment to increase Tsk artificially to increase Te at similar rates in the passive and seated resting trials in comparison to those of the active exercise conditions.

4.4.2 Sweat rate changes and accompanying changes in body temperature

For the two environmental conditions, Tb was predominantly dependent on Tsk changes, as Te was constant across the trials. As the water-perfusion garment was only used in passive exercise and seated resting trials, differences in ms can be attributed to suit water temperature, as well as the Ts. The ms in comparison to Tb for the active conditions gradually increased, whereas that of the passive exercise and resting trials was more abrupt, starting at a higher ms for a similar Tb. These differences are illustrated in Figures 4.13a and 4.13b. A regression line was fitted through the normalised data of Tb and ms. The slopes of these normalised Tb and ms relationships were not significantly different between the five experimental trial groups (p>0.05). The active 40 and 80 rev.min⁻¹ exercise trials slope values were, 0.98 and 0.99 mg.min⁻¹.cm⁻²°C⁻¹, respectively. The passive 40 and 80 rev.min⁻¹ exercise and seated resting trials slope values averaged, 0.94, 0.96, and 0.98 mg.min⁻¹.cm⁻²°C⁻¹, respectively. The active 40 rev.min⁻¹ exercise trials were the same as that of the seated resting condition, with all trials slope values being similar. The active and passive 80 rev.min⁻¹ exercise trials slope values were greater than those of the active and passive 40 rev.min⁻¹ exercise trials, which supports the earlier results of ms related to pedal frequency
Figure 4.13 (A) Changes in sweat rate and body temperature for 25 minutes, for five experimental trials. The passive exercise and resting trials had a consistently higher Tsk than that of the active exercise conditions helping to account for differences in Tb. This higher Tsk was from the use of a water-perfusion garment and hot environmental conditions in these trials. (B) Changes in sweat rate and normalised Tb. Data are means and standard errors of the means.
already reported in this chapter.

4.5 CHANGES IN FREQUENCY OF SWEAT EXPULSIONS

The $f_{sw}$ in the active exercise trials changed abruptly initially, then slowed and changed more gradually. The passive exercise and seated resting trials changed consistently, though were initially higher due to the higher $m_{sw}$ at the beginning of these trials which was associated with the higher $T_b$ in comparison to the active exercise trials. These results were consistent with the sweat rate data. The $f_{sw}$ of the seated resting trials were more abrupt, though the passive exercise trials mimicked each other more closely than that of the resting trial.

Frequency of sweat expulsions did not differ significantly between the active exercise trials ($p>0.05$). The active 40 and 80 rev.min$^{-1}$ exercise trials averaged, 15.81 ($\pm$2.08) and 16.84 exp.min$^{-1}$ ($\pm$2.55), respectively. The $f_{sw}$ for the passive 40 and 80 rev.min$^{-1}$ exercise and resting trials averaged, 17.22 ($\pm$1.14), 17.33 ($\pm$1.52) and 17.23 exp.min$^{-1}$ ($\pm$1.14) which were not significantly different either ($p>0.05$). Analysis revealed significant differences between the active and passive exercise trials, as well as that of the seated resting trials at 2 minutes ($p<0.05$). The $f_{sw}$ for the active 40 and 80 rev.min$^{-1}$ exercise trials at 2 minutes averaged, 7.5 ($\pm$1.62) and 7.45 ($\pm$1.53) exp.min$^{-1}$, while the passive 40 and 80 rev.min$^{-1}$ exercise, as well as that of the resting trials averaged, 13.15 ($\pm$1.50), 12.75 ($\pm$1.42) and 13.20 exp.min$^{-1}$ ($\pm$0.80), respectively (Figure 4.14).

4.5.1 Comparison of sweat rate expulsion frequency and sweat rates.

Sweat expulsion frequency and $m_{sw}$ increased constantly during the experimental
Figure 4.14 The mean sweat expulsion frequency responses for 25 minutes of data collection for the five experimental trials. Data are means and standard errors of the means. Significant differences are indicated by an asterisk ($p<0.05$).
manipulations (Figure 4.15), a regression line was put through this data for the five trials. The slope values for the relationship between $f_{sw}$ and $m_{sw}$ for the five trials were similar and revealed no significant differences ($p > 0.05$). The active 40 and 80 rev.min$^{-1}$ exercise trials averaged 0.98 and 0.99, while that of the passive 40 and 80 rev.min$^{-1}$ exercise and seated resting trials averaged, 0.89, 0.96 and 0.97, respectively. The higher $m_{sw}$ and $f_{sw}$ in the first 5 minutes of the passive exercise and resting trials compared with those of the active exercise trials, may help to understand the controlling influences responsible for the changes in $m_{sw}$ and $f_{sw}$ (while $T_c$ was constant across experimental trials). These influences may be related to the different pedal frequencies between experimental trials and $T_s$ which differed between the two experimental environments. The average total change of $f_{sw}$ for the active 40 and 80 rev.min$^{-1}$ exercise trials was, 12.25, 15.10 exp.min$^{-1}$, while the passive 40 and 80 rev.min$^{-1}$ exercise and resting trials changed by an average of 6.05, 7.85 and 7 exp.min$^{-1}$, respectively. The disparity between these data was related to the initial differences in $f_{sw}$ and $m_{sw}$.

4.5.2 Comparison of central and peripheral frequency of sweat expulsions.

Frequency of sweat expulsion data from the forehead (representing the central input) and the forearm (representing the peripheral input) followed similar trends, though analysis revealed no significant differences across the five experimental groups ($p > 0.05$) (Figure 4.16). The forehead $f_{sw}$ averaged, 15.77 ($\pm 2.03$) and 16.57 exp.min$^{-1}$ ($\pm 2.49$) for the active 40 and 80 rev.min$^{-1}$ exercise trials, while the passive 40 and 80 rev.min$^{-1}$ exercise, as well as the resting trials averaged, 16.61 ($\pm 0.99$), 16.71 ($\pm 1.67$) and 17.02 exp.min$^{-1}$ ($\pm 1.13$), respectively (Figure 4.15). The forearm $f_{sw}$ averaged, 15.92 ($\pm 2.12$) and 17.12 exp.min$^{-1}$ ($\pm 1.39$) for the active 40 and 80 rev.min$^{-1}$ exercise trials, which was very similar to that of the forehead $f_{sw}$. The passive 40 and 80 rev.min$^{-1}$ exercise and resting trials for forearm $f_{sw}$ averaged, 17.12
Figure 4.15 Sweat rate plotted against frequency of sweat expulsions for the five experimental trials. Data are means and standard errors of the means.
Figure 4.16 Comparison of central (A) and peripheral (B) frequency of sweat expulsions for the five experimental trials. Data from the forehead frequency site (representing the central input) and the forearm (representing the peripheral input) were not significantly different across treatment for all five experimental groups ($p > 0.05$). Data are means and standard errors of the means.
(±1.39), 17.67 (±1.29) and 17.68 exp.min⁻¹ (±1.11), respectively. The active and passive 80 rev.min⁻¹ exercise trials were consistently higher than those of the active and passive 40 rev.min⁻¹ exercise trials, though there were no significant differences (p>0.05).

Also, the forearm \( f_{sw} \) for all five trials were slightly greater than that of forehead, this was unexpected result, as the forehead had the largest \( r_n_{sw} \) consistently across the trials, and has the greatest density of sweat glands.

4.5.3 Changes in frequency of sweat expulsion and body temperature.

Initial differences in \( T_b \) and \( f_{sw} \) between the active and passive 40 and 80 rev.min⁻¹ exercise trials and seated resting trials, resulted from the two experimental protocols used (Figure 4.17a). Both the active exercise conditions followed similar trends, with \( f_{sw} \) and \( T_b \) increasing consistently. The passive exercise and resting conditions started at a higher \( f_{sw} \) for a higher \( T_b \) than that of the active exercise trials, but which increased gradually and consistently throughout the rest of the experimental period (Figure 4.17a). These results would suggest differences in the central control and regulation of body temperature, due to the way in which heat sources were manipulated (external and metabolic), and which is reflected in Figures 4.15 and 4.17a. When \( f_{sw} \) was compared to normalised \( T_b \) the five experimental trials were grouped closely together, though the initial differences in \( f_{sw} \) were still apparent, which may support the notion that the initial differences occurred due to the immediacy with which the temperatures of the water-perfusion garment was imposed on the subjects (4.17b). When \( f_{sw} \) was compared to normalised \( T_b \) and regressions were fitted to the five experimental trials, no significant differences were found (4.17b). The active 40 and 80 rev.min⁻¹ exercise trials slope values were 0.97 and 0.98, respectively. These slope results for
Figure 4.17 (A) Changes in frequency of sweat expulsion and mean body temperature for the five experimental trials. (B) Changes in sweat expulsion frequency and normalised mean body temperature. Data are means and standard errors of the means.
and normalised $T_b$ compared closely to those of the passive 40 and 80 rev.min$^{-1}$ exercise and seated resting trials, which averaged 0.96, 0.98 and 0.98. These data would suggest that as $T_b$ increased, $f_{sw}$ increased as an efferent feedback control to regulate temperature. The initial differences in $f_{sw}$ and $T_b$ may suggest that temperature was being regulated by two different control mechanisms, a feedforward mechanism initially, and a feedback control mechanism dominated with time.

4.6 SUMMARY

The two different environmental conditions, as well as the use of the water-perfusion garment in this present investigation, created a disparity within the results. Data from the present investigation confirm that $T_{sk}$ was not successfully controlled across trials, though $T_c$ was successfully regulated. As $T_{sk}$ was not successfully controlled between the two environments, initial differences in $f_{sw}$ and $m_{sw}$ could be explained by a variety of influences, such as: the rate of change in $T_{sk}$; the temperature gradient between $T_{sk}$ and $T_c$; differences in $T_b$ and $T_s$; differences in efferent sympathetic activity, central command and associated changes in skin blood flow.

From the $f_c$ data, it was apparent that the passive exercise trials were of a passive nature, which is supported from the thermal sensation data, which was not significantly different between trials ($p > 0.05$). Similar total changes in $f_c$ occurred from the start to end of the passive and active exercise trials, indicating similar amounts of work were achieved between these two groups. These data helped to exclude the role of mechanoreceptors in the initial differences of $f_{sw}$ and $m_{sw}$.
Sweat rates and $f_{sw}$ were significantly different between the active and passive exercise, as well as that of the seated resting trial, at 2 minutes ($p<0.05$). The hierarchal pattern of $m_{sw}$, which was produced across the six local sweat measurement sites for the five experimental trials, was consistent, suggesting that temperature was regulated similarly between the sites for the five experimental trials. The $m_{sw}$ data of the forehead and forearm sweat collection sites were very similar, confirming that the central neural controller regulated temperature consistently, centrally and peripherally. The $m_{sw}$ and $f_{sw}$ were consistently higher in the active and passive 80 rev.min$^{-1}$ exercise trials in comparison to the active and passive 40 rev.min$^{-1}$ exercise trials. This result supports the hypothesis, that when pedal force is doubled during the dynamic exercise both $m_{sw}$ and $f_{sw}$ were greater. The initial differences in $f_{sw}$ and $m_{sw}$ of the passive exercise and seated resting trials when compared to the active exercise trials, resulted from differences in $T_b$ and $T_a$. 

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CHAPTER FIVE: DISCUSSION

This project investigated the non-thermal efferents which influence and regulate eccrine sweating during exercise. While many studies have reported the involvement of non-thermal inputs in the regulation of sweating, few have been successful in identifying and quantifying these stimuli. It would seem that both central feedforward and peripheral feedback (e.g. mechanoreceptors) may play a role in this regulation. As no clear evidence has emerged from previous research, this project attempted to identify, and differentiate between, some of the non-thermal influences on eccrine sweating. The role of feedforward regulation was assessed from a comparison of sweat gland function during dynamic exercise, in which pedal frequency was manipulated. The role of possible mechanoreceptor feedback was evaluated using a passive exercise model, with the legs being driven passively at two pedal frequencies. Exercise patterns of the passive exercise trials were identical to those of the active exercise trials, except the former trials were performed within a hot environment, and the subjects wore a water-perfusion garment to replicate thermal load.

5.1 REGULATION OF BODY TEMPERATURES AND SUDOMOTOR CONTROL

It is commonly accepted that eccrine sweating increases linearly with increased thermal inputs, such as $T_c$ and $T_{sk}$ (Stolwijk and Nadel, 1973; Nielsen, 1938). The water-perfusion garment, in conjunction with hot ambient conditions, were used to change $T_c$ and replicate thermal loads across the passive exercise and seated resting trials, similar to those produced in the active exercise trials. This was successful, though, in doing so, $T_{sk}$ was not adequately controlled and differed significantly, between the two different environmental protocols used (Figure 4.1).
Having controlled $T_c$, we may conclude that other factors were involved in the initial and rapid response of sudomotor control. The $f_{sw}$ and $m_{sw}$ of the present investigation, did not statistically differ between the resting trials and passive exercise trials, suggesting that mechanical reflexes or mechanical stimulation were not critical to the initiation of non-thermal sweating. This is in contrast to that of Beaumont and Bullard (1963), who observed that the rapid response of sweating with the start of work was not affected by circulatory occlusion, which prevented warm venous blood from the working muscles from reaching centrally located thermoreceptors. The authors indicated that $m_{sw}$ occurred after the initiation of muscular activity, suggesting the sweating was influenced by movement or work rate, but not blood temperature or muscle temperature. The $f_{sw}$ and $m_{sw}$ of the active exercise trials, did differ significantly when compared to those of the passive exercise and seated resting trials, at 2 minutes. The $f_{sw}$ and $m_{sw}$ of the active exercise trials, were very similar for the first 10 minutes of the trials. It would seem that the $f_{sw}$ and $m_{sw}$ of the active exercise trials, was not initiated from a need to dissipate heat, as $T_c$ did not have sufficient time to change significantly, to warrant this effector response, but that some other mechanism was involved.

It is possible that these initial changes in $f_{sw}$ and $m_{sw}$ were part of a feedforward mechanism to regulate temperature, which involved central command turning on a number of control systems, one of these being sudomotor function. In the study by Vissering et al. (1991), static handgrip at 30% maximal voluntary contraction was performed for 2 minutes followed by post-exercise circulatory occlusion for 2 more minutes. They observed that muscle sympathetic nerve activity increased slowly, with a latency of almost a minute from the onset of muscle tension development to the onset of sympathetic activation, skin sympathetic nerve activity increased abruptly at the start of static exercise, with the first burst of activity immediately
preceding the onset of muscle tension. Interestingly, this cutaneous sympathetic activation is accompanied by increases in both electrodermal activity and vasomotor tone, with the relative targeting of efferent outflow to sweat glands and vascular smooth muscle. They concluded that central command and peripheral reflex mechanisms can be an important stimuli to sympathetic outflow. This central command mechanism may be the main influence in the initiation of $m_{sw}$ with the commencement of exercise.

Comparison of $m_{sw}$ in the passive exercise and resting trials were significantly greater in the first 2 minutes to that of the active exercise trials, giving support to movement or mechanical events playing a minimal role in sudomotor regulation, in these first 2 minutes (Figures 4.12 & 4.14). Wells and Buskirk (1971) observed an increase in local $m_{sw}$ with an increase in local muscle activity under ipsilateral leg and arm exercise. These authors explained these results by proposing that the increase in local $m_{sw}$ was due to local heating related to muscle contraction. These results would support those of Beaumont and Bullard (1963) who suggested mechanical reflexes or work rate are involved in the initiation of non-thermal sweating, which is similar to the results of this study. Neither of these past investigations discussed the possibility of a rate of change in the thermal equilibrium as a possible factor involved in the control of non-thermal sweating. Direct comparisons of the active exercise trials with those of the passive exercise and resting trials is difficult, as both the environment and $T_{sk}$ were significantly different, and any conclusions made may not be considered conclusive.

Saito et al. (1990) demonstrated an immediate increase in $m_{sw}$ after the commencement of muscle contraction, and a high rate remained during the handgrip task which corresponded with the change in sympathetic nerve activity. Their results suggest that the increase in
sympathetic nerve activity responded to input from the peripheral mechanoreceptors and central command. At the cessation of the handgrip task, sympathetic nerve activity, $m_{sw}$ and skin blood flow returned quickly to control levels. If mechanical events were to be considered an influencing role, then it would be expected that $m_{sw}$ and $f_{sw}$ of the resting trials would be less than those of the active and passive exercise trials (Figures 4.12 & 4.14).

Gisolfi and Robinson (1970) found abrupt changes in sweating at the beginning and end of work in a cool room. These observations are further supported from evidence of this present investigation, which also reports abrupt changes in $m_{sw}$ at the beginning of exercise, prior to a significant change in $T_e$ (Figures 4.12 & 4.13a). Bothorel et al. (1991b) supported the findings of Gisolfi and Robinson (1970), observing a sudden decrease in sweating at the cessation of exercise, before any change in body temperature. Taken collectively, it is evident that non-thermal factors appear to play a role in the control of sweating. Though $T_e$ was controlled across the five experimental trials, $T_e$ was decreasing for the first five minutes in the passive exercise and resting trials, which was in contrast to the active exercise trials where $T_e$ was increasing (Figure 4.1a). This helps to reinforce that $T_e$ was not a contributing influence in these initial minutes of sweating which concurs with these other studies, as it would be expected that $f_{sw}$ and $m_{sw}$ would be greater in the active exercise trials, in comparison to the passive and resting trials, and as there was insufficient time for $T_e$ to have changed significantly.

The changes in $T_{sk}$ in the initial five minutes was a key focus of the investigation, as the rates of change differed between that of the active exercise group with that of the passive exercise and resting groups (Figure 4.1b). These differences in $T_{sk}$ may have had considerable influence at the commencement of sweating, which concurs with Libert et al. (1978) who
reported that the onset of sweating was predominantly affected by the skin temperature, and the rates with which it changed. Differences in $T_{sk}$ resulted between the two experimental protocols, suggesting possible differences in central control and regulation of body temperature. Other researchers agree, indicating that the rates of change in $T_{sk}$ play an important role in the control of sweating (Banerjee et al., 1969, Libert et al., 1979, and Werner, 1983). The $T_{sk}$ differed significantly in the passive exercise and resting groups in comparison to the active exercise groups, with initial differences in $f_{sw}$ and $m_{sw}$ resulting (Figures 4.12 & 4.14). These data helped to confirm that $T_{sk}$ played a fundamental role in the initial control of sweating, especially in the passive exercise and seated resting trials.

5.2 INFLUENCE OF BODY TEMPERATURE ON SUDOMOTOR CONTROL

The differences in $T_{sk}$ greatly influenced $T_b$, even though $T_c$ was constant over the experimental period. One of the aims of the present investigation was to try and control both $T_c$ and $T_{sk}$ for the experimental period. However, this was not possible across all five trials, but was largely achievable (separately) within the active and passive exercise trials. By controlling $T_c$ and $T_{sk}$, we aimed to isolate thermal feedback and thereby permit possible mechanoreceptor inputs to be revealed and learn more in the control of eccrine sweating.

Bothorel et al. (1991a) found no local effect of $T_{sk}$ or local muscle temperature on local $m_{sw}$, suggesting that sweat rates were affected by exercise intensity, indicating a central drive as the predominant factor involved in the control of sweating. Exercise intensity and mechanical reflexes could be eliminated as an influence in the initiation of sweating in the seated resting trials, which involved no movement, and which did not differ significantly in $f_{sw}$, $m_{sw}$, or $T_{bp}$ when compared to the passive exercise groups (Figures 4.13 and 4.17). These results are in
contrast to those of Heising and Werner (1987) who reported sweating being higher on an active limb exposed to heat compared to one that was resting. The authors tried to maintain an equal heat flow transfer between the two limbs by using climatic boxes, they suggested, that a local $T_{sk}$ effect and a local effect of muscle activity, were the primary factors involved in the initiation of sweating. These factors are probably more involved in the control of sweating with active dynamic exercise, but may not play such an important role, in passive dynamic exercise or in a seated resting position, as was used in this investigation. In their study, the local $T_{sk}$ of the resting limb was not significantly different from that of the active limb, as it was in this investigation. Kondo et al. (1997) indicated that $f_c$ and $m_{sw}$ were significantly greater during active cycling than that of passive cycling, in which there were no significant differences in $T_{cr}$ $T_{sk}$ or $T_b$. These data may strengthen the argument that $T_{sk}$ played a dominant role in the initial differences in $m_{sw}$ and $f_{sw}$ of the passive exercise and seated resting trials.

Ogawa and Sugenoya (1993) suggested that the control of sweating during exercise was closely related to effort, rather than work rate, which coincides with the findings of Bothorel et al. (1991a). These findings would help explain the resulting data of the active exercise trials of the present investigation, which differs from that of the passive exercise and resting trials, as the effort of these trials, was considerably greater than that of the passive exercise and resting groups, which is statistically supported from the RPE and $f_c$ data (Figures 4.4 & 4.5).

The reason RPE in the passive exercise trials was above resting may have been due to the changes in haemodynamics involved in the passive movement. However, in the resting trials, $f_c$ was consistently higher than observed in the passive exercise conditions. One explanation for these differences may be related to convective cooling of the subjects moving legs and
associated changes in the haemodynamics involved in the passive exercise trials. This cooling occurred due to an increase in muscle blood flow, which may have helped to lower thermal strain and heat storage, and thereby reduced $f_c$. Goodwin et al. (1972a) studied the cardiovascular responses to static exercise while varying the amount of central motor command required to maintain a given level of developed force. Their study suggested there were parallel changes in $f_c$ and effort with central motor command. Ohnishi (1991) observed that when the exertion of a given work rate was increased or decreased by means of partial curarisation and tonic vibration, sweating also increased or decreased, indirectly indicating that sudomotor activity was related predominantly to central neural control. Kondo et al. (1997) suggested that central command was important for increases in $f_c$ and $m_{sw}$ during active cycling, and that central command was not involved during passive cycling, in which the activation of muscle mechanoreceptors invoked the pressor response. The differences in $m_{sw}$ and $f_{sw}$ between the active and passive exercise trials, as well as, that of the seated resting trials, may have been related solely, to differences in central command. Though the differences in this study of $m_{sw}$ and $f_{sw}$ in the passive exercise and seated trials seemed to be predominantly related to differences in $T_{sk}$.

The magnitude of change in the $T_{sk}$, and therefore $T_{b}$, confounded the resulting data of the present investigation. To help minimise the confounding effect of the large differences in $T_{sk}$, $T_{sk}$ for all trials was normalised to the same $T_{sk}$ as of that of the active 40 rev.min$^{-1}$ exercise trial, at 2 minutes of experimental data collection (Figure 4.3). When these data were normalised, it was apparent there were still considerable differences in the resulting data, with the passive exercise and seated resting trials having greater $f_{sw}$ and $m_{sw}$ at the same temperatures, as of those of the active exercise trials (Figures 4.13b and 4.17b). Though, a lot can be learned from these
data, as comparisons of $T_b$ to that of $f_{sw}$ and $m_{sw}$ (Figures 4.13 and 4.17) clearly show differences. When normalised to the same $T_b$, the differences in $f_{sw}$ and $m_{sw}$ may indicate that two different mechanisms were involved in the initial differences of $f_{sw}$ and $m_{sw}$ between the two experimental environments. These two mechanisms may be related to the elevated skin temperature of the passive exercise and resting trials, which may have facilitated sweating in two ways, by increasing afferent input to the central controller and by increasing the activity of the sweat glands, locally (Ogawa, 1987). Bullard et al. (1967) suggested that local skin temperature locally modifies the central control of sweating. Other theories suggest that a rise in local skin temperature increases the amount of neurotransmitter substance released at the neuroglandular junction (McIntyre et al., 1968), thereby increasing sweat gland sensitivity (Ogawa and Ayasama, 1986). The differences in $f_{sw}$ and $m_{sw}$ between the two experimental environments, may simply be due to the abrupt thermal load imposed upon the subjects when the hot water was initially flushed through the water-perfusion garment at the beginning of the trials, generating an immediacy to the central controller to dissipate heat.

From 15 minutes to 25 minutes, the rates of change for $T_{sk}$ across the five experimental trials were very similar, with no significant differences (Figure 4.1b). As the experimental trials progressed with time, it would seem that the sweating became more dominated by thermal inputs than non-thermal inputs, as body temperatures, $f_{sw}$ and $m_{sw}$ were very similar during this period (Figures 4.12, 4.14, & 4.17b).

It was important to control $T_{sk}$, as $T_{sk}$ affects both local control of vasomotor activity directly, and reflex control of the vasomotor tone of the skin. Rowell et al. (1969) reported evidence for distinctive changes in peripheral circulation when the body was heated using a
water-perfusion garment. The significant differences of $T_{sk}$ between the two experimental groups, would indicate marked differences in vasomotor tone between the active exercise trials, where, initially, a vasoconstrictor activity would have occurred, in comparison to an increased vasodilator activity in peripheral circulation during the passive exercise and seated resting trials. Thus, making it difficult to make direct comparisons of the resultant effector responses. Therefore, comparisons should only be made within each experimental environment such that, comparisons of the active exercise trials are made separately to those of the passive exercise and resting trials.

5.3 SWEAT RATE CHANGES

This study aimed to help further our understanding of sudomotor function, by trying to identify and differentiate between some of the non-thermal influences on eccrine sweating, prior to a change in body temperature. Therefore, the main focus of this investigation was to determine if there were any differences in $f_{sw}$ and $m_{sw}$ occurring across the trials in the first few minutes, and possibly explain the reasons for these occurrences. Comparison of the means of the five experimental trials resulted in no significant differences. Further, analysis resulted in significant differences of $m_{sw}$ between that of the active exercise groups with that of the passive exercise and seated resting trials, at 2 minutes (Figure 4.12). On the basis of these data, one may reject the hypothesis that the $m_{sw}$ of the active exercise trials would exceed that of the passive exercise and seated resting trials, adding strength to the notion that non-thermal factors, such as mechanoreceptors, effort, and exercise intensity, are not predominant factors involved in these initial differences in $m_{sw}$. If these did play a dominant role, it would be expected that $m_{sw}$ would have been greater in the active exercise trials, in comparison with the passive and seated resting trials, but as $T_{sk}$ was significantly different between the two experimental protocols used, no
definitive conclusions could be made.

These differences in \( m_{sw} \) between the two experimental protocols, would support the notion that \( T_{sk} \), and the rate at which it changes, are important in the control of sweating. The initial differences in \( m_{sw} \) may also, relate to differences in \( T_{sk} \) having a local effect on peripheral thermoreceptors, inducing a greater peripheral thermal feedback in the passive exercise and seated resting trials (Libert et al., 1983). Differences in \( m_{sw} \) of the present study, could be related to differences in the gradients between \( T_{sk} \) and \( T_c \), especially in the passive exercise and seated resting trials, as these gradients were completely different to that of the active exercise trials. Libert et al. (1983) commented that sweat regulation may be affected by differences in the gains of central and peripheral temperatures, supporting the notion that the gradients or differences between core and skin temperatures and the rate at which they change, may be contributing factors. These differences in temperature gradients would have been accentuated not only by the water-perfusion garment's high temperatures and the hotter \( T_o \) of these passive exercise and seated resting trials, but by the immediacy with which these temperatures were imposed. The differences in the afferent thermal feedback from these affects may indicate that temperature was regulated and that \( m_{sw} \) was being controlled differently, between the two experimental environmental groups.

Though, there were initial differences in \( m_{sw} \) between the active exercise trials and that of the seated resting and passive exercise trials, there were also similarities amongst the groups in the first 10 minutes. The active 40 and 80 rev.min\(^{-1}\) exercise trials mimicked each other very closely, with the passive 40 and 80 rev.min\(^{-1}\) exercise trials, doing the same (Figure 4.12). So, at least, in these first few minutes of the trials, the non-thermal and thermal influences were
consistent for each of the environmental groups. The \( m_{sw} \) of the active 40 and 80 rev.min\(^{-1}\) exercise trials changed dramatically in these first 10 minutes in comparison to that of the passive 40 and 80 rev.min\(^{-1}\) exercise trials, which commenced with a much greater initial \( m_{sw} \) (Figure 4.12).

From 10 minutes to the end of the experimental period, the initial trend of \( m_{sw} \) between the active 40 and 80 rev.min\(^{-1}\) exercise trials and that of the passive 40 and 80 rev.min\(^{-1}\) exercise trials diverged, with active and passive 40 rev.min\(^{-1}\) exercise trials and that of the active and passive 80 rev.min\(^{-1}\) exercise trials becoming comparable (Figure 4.12). The rate of change in \( T_c \) and \( T_{sk} \) were consistent between the trials during this period of time, so thermal determinants would not help explain these similarities which occurred, therefore it would seem that non-thermal inputs played a role. Pedal frequency was the common link between the active and passive exercise trials, therefore the role of mechanoreceptors may have been an influencing factor for the similarities between these trials. The active and passive 40 rev.min\(^{-1}\) exercise trials and that of the active and passive 80 rev.min\(^{-1}\) exercise trials followed similar trends from 15 minutes to the end of the experiment. The seated resting trials followed a comparable trend in \( m_{sw} \), as that of the active and passive 80 rev.min\(^{-1}\) exercise trials. As there is no clear explanation, these data may indicate that thermal and non-thermal influences may be modified by each other depending on the need to regulate temperature. These results may help to support the notion that feedforward controls were dominant initially in the active exercise trials with the rapid changes in thermal equilibrium which occurred, and that feedback controls dominated as temperature became successfully regulated.

Comparisons of central and peripheral local \( m_{sw} \) demonstrated very clear similarities
between the two groups, which would suggest that sudomotor function was controlled consistently, centrally or peripherally (Figures 4.9 & 4.10). The local $m_{sw}$ of the six sites measured, showed a consistent $m_{sw}$ pattern, across the five experimental trials. The water-perfusion garment therefore, did not seem to change the local $m_{sw}$ characteristics between the two experimental protocols. The only significant differences in local $m_{sw}$ were from the sites of forehead and forearm at 2 minutes, between the active and passive exercise trials, as well as that of the seated resting trials, again confirming that efferent output was controlled similarly centrally or peripherally. These differences can be accounted for again by the initial differences in $T_{sk}$ between the trials. The forehead and forearm local $m_{sw}$ were similar to each other, in the active and passive exercise trials, though the seated resting trial was consistently greater in these sites when compared to the other four trials (Figures 4.9 & 4.10). These differences between these two sites may be accounted for by the greater $m_{sw}$ initially in the passive exercise and seated resting trials, in comparison to those of the active exercise trials.

When the two individual $m_{sw}$ of forehead and forearm were combined, there was a strong resemblance to that of the combined average $m_{sw}$ for the six sites measured (Figures 4.11 and 4.12). From the six $m_{sw}$ sites measured for the five experimental trials, a consistent secretion pattern emerged, with the forehead having the largest $m_{sw}$ output, then the forearm, scapula, thigh, chest, and arm, respectively. These results are similar to other research from the same laboratory by Cotter et al. (1995), who reported that, during steady state sweating at rest, the largest $m_{sw}$ was on the forehead and least on arms and chest. Topographical differences can also be accounted for by differences in density of sweat glands for example the greatest density of sweat glands can be found in the forehead region (Kuno, 1956).
Though \( \bar{m}_{sw} \) was consistently greater in the active and passive 80 rev.min\(^{-1}\) exercise trials when compared to the active and passive 40 rev.min\(^{-1}\) exercise trials, no significant differences resulted. Therefore, one may reject the hypothesis that the \( \bar{m}_{sw} \) of the active 80 rev.min\(^{-1}\) exercise trials would exceed those of the active 40 rev.min\(^{-1}\) exercise trials. The hypothesis that \( \bar{m}_{sw} \) would not differ between the passive exercise and resting trials was accepted as the data did not significantly differ.

### 5.4 CHANGES IN THE FREQUENCY OF SWEAT EXPULSION

In the initial 10 minutes, there were no differences in \( f_{sw} \) between the passive exercise and seated resting trials, which helped to reinforce the suggestion that non-thermal influences such as, mechanoreceptors, exercise intensity and effort had minimal, or no involvement in the initial differences in \( f_{sw} \) between these trials and those of the active exercise trials, but that \( T_{sk} \) and environmental differences were dominant factors for these differences. This suggestion is further supported when \( T_b \) was normalised and compared to \( f_{sw} \) as the \( f_{sw} \) of the passive exercise and seated resting trials, is still significantly different from that of the active exercise trials, at 2 minutes (Figure 4.17b). These data support the notion that it was the immediacy with which the temperature was applied in the passive exercise and seated resting trials, which brought about differences in the initial temperature gradients of \( T_c \) to \( T_{sk} \), which may have created a greater central drive to regulate temperature at 2 minutes, in comparison to that of the active exercise trials. It would therefore, be reasonable to suggest that it was thermal influences that created the initial differences in \( f_{sw} \) between the passive exercise and seated resting trials, when compared to those of the active exercise trials, and not non-thermal influences. Though non-thermal factors, such as central command may have played a dominant role in the commencement of \( \bar{m}_{sw} \) in the active exercise trials, as thermal balance was changed with the commencement of
work. When normalised $T_b$ increased above $36^\circ$C the plots of $f_{sw}$ for the active and passive exercise trials, as well as that of the seated resting trials, merge together (Figure 4.17b), suggesting that as normalised $T_b$ increased thermal influences seemed to play a dominant role in control of $f_{sw}$.

The five experimental trials had similar mean rates of $T_c$ change over 25 minutes of data collection and similar changes in $f_{sw}$ and $m_{sw}$ in the time period from 15 to 25 minutes, adding further support to this notion. This may help us to understand eccrine sweating better, for if $f_{sw}$ and $m_{sw}$ do not differ in this period, movement, effort, exercise intensity would seem to play a minimal role and that the rate with which $T_b$ changes played the dominant role. Changes in $f_{sw}$ and normalised $T_b$ showed a strong positive relationship (Figure 4.17b) which is consistent with past research (Yamazaki et al., 1994b; Sugenoya and Ogawa, 1993). This adds further support to the notion that the differences in $T_{sk}$ between the two experimental protocols played a predominant role in the initial differences in eccrine sweating.

Comparison of central and peripheral $f_{sw}$ showed no significant differences (Figure 4.16). Both the forehead and forearm $f_{sw}$ showed uniformity and synchrony, which would indicate that there was no difference in the sudomotor response either centrally or peripherally to the efferent information and that temperature was being regulated similarly at these two sites. These data helped to understand that any resulting differences in $f_{sw}$ across measurement sites, would have been due to local factors such as, eccrine gland density and recruitment and not due to differences in neural control.

As with previous research, when sweating was profuse the pattern of $f_{sw}$ changed in
amplitude and frequency (Sugenoya and Ogawa, 1985; Yamazaki et al., 1992). Initially, the amplitude of $f_{sw}$ was large and infrequent, but as time increased and sweating became more profuse, the amplitude decreased and the $f_{sw}$ increased. This pattern concurs with previous literature (Sugenoya and Ogawa, 1985; Yamazaki et al., 1992). These changes in $f_{sw}$ may indicate that sweat gland response was influenced by centrally-driven sudomotor activity, with previous research suggesting that it is caused by the facilitation of transmitter release at the neuroglandular junction, or an increase in sensitivity in the glandular cell to transmitter substances or possibly both (Ogawa and Asayama, 1986). Yamazaki et al. (1993) suggested that these changes may also represent the rate at which internal temperature is changing, which would support previous suggestions. As the rate of change in $T_c$ and $T_{sk}$ starts to slow or plateau, the amplitude pattern of $f_{sw}$ becomes smaller and more frequent, for the period of 15-25 minutes, adding further support to the notion of Yamazaki et al. (1993). This change in nervous activity were similar for all trials, and may have represented changes in temperature regulation and sudomotor control. The rate of change of $T_c$ is the greatest in the first 10 minutes of this study, which coincides with the largest amplitude seen in $f_{sw}$, but these changes in amplitude may also be related to the initial differences between that of the $T_c$ and $T_{sk}$ and the rate at which they were changing and possibly represents the different control mechanisms involved in regulating temperature.

The $f_{sw}$ were significantly different between the active and passive exercise trials, as well as that of the seated resting trials, at 2 minutes. On the basis of these data, one may reject the hypothesis that the $f_{sw}$ of the active exercise trials will exceed those of the passive and resting trials. The $f_{sw}$ in the passive exercise and resting trials were consistently higher than those of the active exercise trials. Though from these data one could accept the hypothesis that there will not
be a difference in $f_{sw}$ between the passive exercise and resting trials. These data provides the basis to which one may reject the hypothesis that the $f_{sw}$ in the active 80 rev.min$^{-1}$ exercise trials would exceed those of the active 40 rev.min$^{-1}$ exercise trials.

5.5 CONCLUSION

Distinctive differences were apparent between the two experimental protocols which influenced the resultant outcomes of this investigation. The confounding effect of $T_{sk}$ in the passive exercise and seated resting trials, made direct comparisons of control mechanisms, as well as thermal and non-thermal influences, to those of the active exercise trials, difficult. The initial differences in $f_{sw}$ and $m_{sw}$, between the two environmental groups, was predominantly due to the immediacy with which $T_{sk}$ was changed and the thermal load imposed upon the subjects in the passive exercise and seated resting trials. This suggestion may have helped us to understand the controlling influences underlying the initiation of eccrine sweating in the passive exercise and seated resting trials, the suggestion also helped us to eliminate some of the possible non-thermal influences in the initiation of sweating in the active exercise trials. It would seem apparent there were two different control mechanisms involved in the initial differences in $f_{sw}$ and $m_{sw}$ between the two experimental protocols: a feedback control for the passive exercise and seated resting trials, was created by the difference in thermal gradient of $T_c$ and $T_{sk}$; and secondly a feedforward control of central command initiating eccrine sweating in the active exercise trials, prior to a change in $T_c$.

When comparisons were made between central and peripheral $f_{sw}$ and $m_{sw}$ it was clear that there was no differences in the regulation of temperature either centrally or peripherally. This would tend to indicate that if there were any differences in $m_{sw}$ between any of the sites
measured, the differences would be related to local factors.

The $m_{sw}$ data of the active exercise trials may not have provided conclusive evidence of a feedforward mechanism involvement, as there was no significant differences when comparing the data of active 40 and 80 rev.min$^{-1}$ exercise trials, though there was a strong trend with the $m_{sw}$ and $f_c$ of the active 80 rev.min$^{-1}$ exercise trials being consistently greater than that of the active 40 rev.min$^{-1}$ exercise trials. Previous research of Kondo et al. (2002) suggested that $m_{sw}$ should be significantly less in the passive exercise trials, in comparison to that of the active exercise trials, as was hypothesised in this project, due to the role of central command being involved in active exercise and not in passive exercise. This notion of Kondo et al. (2002) may help to support the suggestion that central command played a key role in the commencement of $m_{sw}$ in this project prior to a change in $T_c$.

In the period from 15-25 minutes there were no significant differences in $f_{sw}$ and $m_{sw}$ between the five trials, and $T_b$ changed at a similar rate for all trials, which would seem to indicate that $f_{sw}$ and $m_{sw}$ was strongly linked with $T_b$, which has already been confirmed. Though for this period, the $f_{sw}$ and $m_{sw}$ of the active and passive 40 rev.min$^{-1}$ exercise trials, and that of the active and passive 80 rev.min$^{-1}$ exercise trials, resembled each closely, which would tend to indicate that mechanical reflexes played a role in the control of eccrine sweating. This is contrary to the period of time from 0-10 minutes, when there were no significant differences of $f_{sw}$ and $m_{sw}$ between the passive and seated resting trials, which would help to support the notion that mechanical reflexes were not initially involved in the control of eccrine sweating. These data may help us to further our understanding of eccrine sweating, because it would seem that thermal and non-thermal influences both play a role in the control of sweating, but that they are
modified by internal temperature changes.

The initial stages of \( m_{sw} \) was the main focus of this project and it would seem apparent that non-thermal influences seemed to play a minimal role, or were masked by the effects the water-perfusion garment had on \( T_{sk} \) in the passive exercise and seated resting trials. In the active exercise trials, the initiation of \( m_{sw} \) seemed to be related to central command, a non-thermal influence. The data from this study indicated the importance of clamping body temperatures to isolate non-thermal influences on the control mechanisms involved in \( m_{sw} \).

5.6 RECOMMENDATIONS FOR FUTURE RESEARCH

This project was designed to clarify the control mechanisms, as well as the thermal and non-thermal influences, which were involved in the initial stages of eccrine sweating. The water-perfusion garment in the passive and seated resting trials was used so that \( T_e \) could be replicated similarly to that of the active exercise trials for each individual. Though in doing so, \( T_{sk} \) differed considerably between the two experimental protocols. If the water temperature of the water-perfusion garment could have been regulated via a computer to allow for more precise and graded responses in \( T_e \) and \( T_{sk} \), the confounding effect of \( T_{sk} \) of this investigation may not have occurred. As this was the first time the water-perfusion garment had been used in conjunction with exercise, it may prove worthwhile for future studies to change the design to contour the body better, and make it more sturdy, as well as increase the number of tubing for each body segment to maximise skin contact of each body region. The ability to increase \( T_e \) artificially without influencing \( T_{sk} \) may provide more insight into the thermal and non-thermal influences involved in sudomotor control. This project provided confirmation of the importance of controlling body temperatures, when trying to differentiate the underlying influences in the
It would be useful for future researchers to be able to replicate similar changes in $T_s$ and $T_c$ across passive and active exercise trials, as well as that of the resting trials, to help determine the role of non-thermal and thermal influences on $f_{sw}$ and $m_{sw}$. It would have been advantageous to record blood pressure, skin blood flow, peripheral vascular resistance, cutaneous vascular conductance and sympathetic nerve activity, to help determine the changes in vasomotor activity associated with the differences in $f_{sw}$ and $m_{sw}$. The possibility of designing a sweat capsule with a sensor built into it, to minimise time lags between capsule and sensor, would also be beneficial.

The evaluation of the role of mechanoreceptors in the control of $m_{sw}$ warrants further investigation, as does the role of feedforward mechanisms. The possibility of using different pedal frequencies of 50 and 100 rev.min\(^{-1}\) may prove worthwhile. Though it may be beneficial to take into account the possibility of different thermoregulatory responses when using the two pedal frequencies, as Kondo et al. (2002) suggested that sweating responses vary depending on the rates of differing internal temperatures changes. These authors further suggested that the degree of thermal input may regulate the way in which non-thermal inputs modulate the control of sweating from eccrine glands. This reinforces the importance of keeping the experimental treatments similar between trials, and minimising any differences of thermal determinants.
5.7 REFERENCES


