THE EFFECTS OF WHOLE BODY IMMERSION IN COLD WATER UPON SUBSEQUENT TERRESTRIAL AEROBIC PERFORMANCE: A STUDY IN HYPOTHERMIA

BY

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ABSTRACT

This study examined the extent to which physiological and psychological concomitants of aerobic terrestrial performance were affected by body cooling of varying degrees induced by cold water immersion (CWI).

Thirteen male and 13 female subjects underwent three randomly assigned 30min treadmill runs: a control run without prior manipulation of the subjects' thermal status and the same exercise after "central" (core temperature 1°C below pre-immersion) and "peripheral" cooling (skin heat loss 100kcal.m^{-2}.h^{-1}). During treadmill runs core temperature was measured, together with chest, leg, arm and hand temperatures, from which mean skin temperature (T_{sk}) and mean body temperature (T_b) were calculated. Heart rate, oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), minute ventilation ($\dot{V}_E$ (BTPS)), breathing frequency (f), cadence and ratings of perceived exertion (RPE) and thermal sensation (PTS) were also measured.

Both central and peripheral cooling resulted in significantly reduced T_{re} (males: control 37.9±0.3°C; central cooling: 36.8±0.5°C; peripheral cooling: 37.5±0.4°C; females: control: 37.9±0.4°C; central cooling: 37.2±0.5; $p<0.05$) during subsequent treadmill running, except following peripheral cooling for females (37.9±0.3°C). For males and females T_{sk} was lower following peripheral cooling than control values and lowest after central cooling (males: control: 30.0±1.3°C; central cooling:

36.8±0.5°C; peripheral cooling: 37.5±0.4°C; females: control: 30.5±1.2°C; central cooling: 25.9±1.8°C; peripheral cooling: 26.9±1.9°C; $p<0.05$). Female subjects experienced significantly higher T_{re} than males following central and peripheral cooling and a lower T_{sk} following central cooling.

Females experienced less of an increase in heart rate than males during exercise following central and peripheral cooling (control: 157.7±23.7b.min^{-1}; central cooling: 143.5±20.5b.min^{-1}; peripheral cooling 151.7±16.7b.min^{-1}; $p<0.05$). Male responses were the same following central cooling but higher for peripheral cooling than control values (control: 139.1±7.3b.min^{-1}; central cooling 134.7±17.5b.min^{-1}; peripheral cooling: 145.0±16.4b.min^{-1}; $p<0.05$). These data indicate a depression in cardiovascular function for females following peripheral cooling that was not apparent for males. The $\dot{V}O_2$ was not different between tests for males; only peripheral cooling resulted in a raised $\dot{V}O_2$ of 28.6±3.3ml.kg^{-1}.min^{-1} ($p<0.05$) for females compared to 27.6±2.6ml.kg^{-1}.min^{-1} (control). A biphasic response was evident for $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ (BTPS).

For both sexes overall RPE was lower for peripheral cooling (males: 9.4±1.9; females: 8.7±1.3; $p<0.05$) than for control and central cooling. Central RPE was only changed for females following peripheral cooling. Changes in cadence and step length together with the effect of low skin and leg temperatures resulted in higher local RPE for females after central cooling (9.6±1.2; $p<0.05$) than control (9.4±1.9) and peripheral cooling

(8.9±1.2). Males and females rated the same ambient temperature during the same exercise lower after peripheral cooling (males: 4.6±1.5; females: 5.3±1.3) than control values and lower still after central cooling (males: 3.8±1.8; females: 2.7±1.5). In this study T_{sk} was the primary determinant of PTS after pre-cooling.

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CHAPTER 1

INTRODUCTION

Hypothermia affects the performance of all organ systems (Lin, 1988). Even comparatively small depressions of body temperature can produce significant impairments of bodily function, both physical and mental (Coleshaw *et al.*, 1983; Nadel, 1984; Shephard, 1985; Askew, 1989; Sjödin *et al.*, 1996).

Water has a volumetric specific heat around 4000 times that of air and a thermal conductivity about 25 times greater (Nadel, 1984). These properties of the aqueous environment result in a substantial thermal drain if the water temperature is low and the body's insulation is inadequate. Thus hypothermia invariably follows prolonged immersion in water (Lin, 1988). According to Keatinge (1969), the performance of an unclothed individual of average body build will be significantly impaired from hypothermia after approximately 20 to 30min in water at 5°C and after 1.5 to 2h in water at 15°C. Obviously, thermal and physiological responses to cold stress vary considerably among individuals (McArdle *et al.*, 1984a) with numerous factors cited as influencing the heat flux during exposure to a cold water environment. Pugh and Edholm (1955), for example, reported that an experienced channel swimmer was able to withstand 16°C water for 12 to 15h without undergoing a decrease in core temperature. A different unacclimatised subject, in contrast, experienced a drop in core temperature from 37°C to 34°C following 50min of swimming in 16°C water, and was unable to stand or walk

afterwards.

Hypothermia can be defined simply as a subnormal body temperature (Chambers Twentieth Century Dictionary, 1979), although varied clinical definitions exist (see p15 for discussion). Body cooling to the extent that the individuals concerned can be described as being hypothermic is not an infrequent observation in many commercial and sporting activities in water. Before the use of wetsuits was implemented amongst Korean women divers, they experienced repeated hypothermia diving in 17 to 26°C water in summer and 7 to 10°C water in winter (Park *et al.*, 1983). Keatinge *et al.* (1980) reported that the core temperatures of North Sea divers were sometimes as low as 34.7 to 35°C at the end of dives, even when a warm water flooded diving suit was used. Similar levels of core temperature have been observed during recreational and competitive swims in cold water (Sloan and Keatinge, 1973; Bergh, 1980; Sutherland, 1992; Chatard and Millet, 1996). It has also been reported that many triathletes, particularly those who are slower swimmers, may experience the effects of hypothermia during the swimming phase of an event (Yach, 1990; Finlay *et al.*, 1995; Chatard and Millet, 1996). The International Triathlon Union rules (January 1996) state that for a swim length of 1500m or less a wetsuit is forbidden at or above a water temperature of 20°C, but is only mandatory below 14°C, with a maximum stay in the water of 1h and 10min (Chatard and Millet, 1996). Yach has commented on the increased risk to the athletes of sea swims at temperatures below 20°C, but the exact consequences of even mild hypothermia for subsequent aerobic

performance remain to be clarified.

A vast literature exists with regard to the effects of exposure to cold upon performance in a cold environment. Relatively few researchers, however, appear to have considered the consequences of exposure to cold water, with concomitant cardiorespiratory adjustments, for performance in a warmer environment on land immediately following exposure. Although, in general, somewhat equivocal findings have been reported, largely as a result of variations in methodology, several researchers have observed an increase in the total energy cost of exercising in a cold environment, both in water and on land (Patton and Vogel, 1984; Toner *et al.*, 1984; Timmons *et al.*, 1985; Shephard, 1985; Pendergast, 1988; Doubt, 1991; Sjödin *et al.*, 1996). Timmons *et al.* (1985) suggested that increases in $\dot{V}O_2$ in cold temperatures were related to exercise intensity: the higher the exercise intensity, the less marked were the effects of the cold environment.

Pendergast (1988) has discussed the effects of body cooling on oxygen delivery to active muscle whilst in a cold water environment. This author reported that the energy cost of submaximal exercise was increased 10 to 40% and oxygen delivery was reduced the same amount with a 0.5 to 1.5°C decrease in core temperature. Maximal aerobic capacity was 10 to 30% lower for a decrease in core temperature of 0.5 to 2.0°C. While it has been suggested that, post-immersion, removal of hydrostatic assistance to venous return may compromise cardiovascular

function (Golden *et al.*, 1991; Finlay *et al.*, 1995), the primary factors influencing subsequent dry performance are likely to be those relating to body cooling rather than immersion *per se*. Furthermore, it is necessary to identify differences, where they may exist, between the effects of cooling such that peripheral tissues, or the body shell alone is cooled and cooling to an extent that central or core temperatures are reduced. In the latter case, for example, central cooling will produce, amongst other results, attenuated reflex cardiovascular responses. In the face of depressed myocardial function, subsequent aerobic performance may be impaired due to the loss of vasoconstriction without adequate correction for reduced central blood volume and cardiac output (Maclean and Emslie-Smith, 1977). Cold-induced changes to peripheral mechanisms may, however, occur independently of any central cardiorespiratory alterations (Doubt, pers. comm.). Decreases in tissue temperature result in a slowing of chemical reactions (Shephard, 1985; Swenson *et al.*, 1996). The direct effect of cold on the muscles themselves will have important implications for subsequent performance where muscle temperatures are lower than optimal for activity of oxidative enzymes, for excitability of nerves and muscle, and for nerve conduction (Patton and Vogel, 1984; Shephard, 1985; Swenson *et al.*, 1996).

During exercise under normal environmental conditions, an appropriate redistribution of blood to meet the metabolic requirements of the working muscles is achieved by vasodilation in those tissues where the local metabolism is high, together

with concomitant constriction of the vasculature in inactive tissues. Cold water immersion (CWI), however, could be expected to complicate this integrated circulatory response, particularly during the early stages of subsequent aerobic performance on land. The local metabolically mediated exercise hyperemia may be blunted by cold induced vasoconstriction (Pendergast, 1988; Ferretti *et al.*, 1995).

If cognisance is taken of the above differentiation of factors influencing dry performance following CWI, the following immediate questions are raised. Given that performance is likely to be impaired by cold-induced alterations in both peripheral and central mechanisms, what is the relative importance of each when considered in terms of the economy of ensuing dry submaximal exercise? If, at this point, efficiency is defined simply as work done divided by energy expended (Cavanagh and Kram, 1985), then, at least initially, a decrease in efficiency would be predicted for dry submaximal exercise following CWI. An assumption to consider here is that the mechanical work required to complete a given task would remain unaltered following cold exposure. If, however, cold-induced changes in such factors as would influence the mechanics of locomotion are considered (increased musculo-tendinous viscosity, for example, or reduced muscle action potential firing rate), the possibility exists that the mechanical work required to perform a given submaximal task may be increased following CWI.

Much previous research into human responses to cold stress has

been criticised as being naive and severely limited in application due to lack of controls on the subjects and because the responses are dramatically influenced by the nature and duration of the stress (Graham, 1988a). Since many of the variables measured are determined by a combination of processes, some of which counteract others, quite different results are possible in different subjects , or under somewhat different conditions (Lanphier and Camporesi, 1984; Doubt, 1991; Sjödin *et al.*, 1996). Thus the almost exclusive use of young, adult, Caucasian male subjects evident in many studies of environmental stress is an unfortunate practice (Wagner and Horvath, 1985a; Graham, 1988a). Although some researchers (Fox, 1974; Wagner and Horvath, 1985a and 1985b; Graham, 1988a and 1988b; Horvath, pers. comm.) have commented on the paucity of information comparing male and female responses, the bulk of the evidence to date would suggest that thermal sensitivity to cold water in particular is different between the sexes (McArdle *et al.*, 1984a; Wagner and Horvath, 1985a; Graham, 1988b; McArdle *et al.*, 1992).

Evidence supporting the hypothesis that there are sex-specific physiological responses to body cooling lies in the observation that, during CWI at rest, women of the same skinfold thickness cool more rapidly than men; this potentially greater stimulus does not, however, result in a greater metabolic response by the women (McArdle *et al.*, 1984a; Graham, 1988b; Pendergast, 1988). There are, however, contradictory opinions in this regard. Doubt (pers. comm.) has commented that, in general, women tend to lose heat at a slower rate than males but at identical core

temperatures thermogenic responses may not be different. McArdle and co-workers observed, however, that differences in the magnitude of the thermogenic response between the sexes did became significant when core temperature dropped by more than 1.0°C. These authors suggested that similar decreases in core temperature for men and women may not necessarily reflect an equal stimulus for thermogenesis.

Clearly differences in body composition may partially account for the difference in temperature regulation during cold stress between men and women. Traditionally, the greater body fat levels generally possessed by women, as well as the differences in subcutaneous fat distribution between the sexes, have been proposed as factors which favour the female during cold stress. McArdle *et al.* (1984a) have pointed out, however, that in fact, morphological and dimensional differences between the sexes, such as a relatively small lean body mass and large surface area-to-mass ratio, may pose a liability to the female in maintaining body temperature during cold stress. The contention that morphological differences alone may not account entirely for differences between the sexes in thermoregulatory responses to cold stress is apparently supported by reports that lean women with twice the percentage of fat have similar core temperatures as lean men at all water temperatures (McArdle *et al.*, 1984a). Even where differences in lean body mass were accounted for, the thermogenic response of men to cold stress was still significantly higher (McArdle *et al.*, 1984a).

In addition, a greater vasoconstriction of the cutaneous circulation as well as the arterial flow to the deeper limb structures (and accompanying reduced limb temperatures) of women compared with men in response to cooling (McArdle *et al.*, 1984b) may further influence physiological and psychophysical responses to subsequent aerobic performance in a warmer environment. Even without prior manipulation of the status of the human organism due to cold stress, the effect of exercise on the core temperature to skin blood flow relationship is complex (Gisolfi, 1983). Johnson and Park (1981) have shown, for example, that an increase in the core temperature threshold for vasodilation may result from upright exercise. Further complication of this relationship due to an intense cold-induced vasoconstriction could be expected to alter the nature of responses to submaximal exercise, an effect which may be different in women compared to men.

Given that differences in thermoregulation between men and women at rest in cold water may exist, the consequences for physiological and psychophysiological parameters during subsequent aerobic performance may be different in women compared to men. Central to the present research, therefore, was an examination of sex differences in the response to body cooling. In noting the lack of studies comparing cold stress responses in men and women, Graham (1988) has also commented that, in those studies where comparisons are made, the menstrual status of the women is rarely reported, despite suggestions in the past that this may influence thermal and metabolic responses (Wagner and

Horvath, 1985a; Graham, 1988). Thus, in addition to control for morphological and dimensional differences between subjects, cognisance must be taken of such factors as menstrual status if valid conclusions are to be drawn.

Any examination of human responses to cold stress would be incomplete without an appreciation of the complex interaction between physiological variables on the one hand, and the perception of sensations on the other, since it is quite clear that human performance is not governed by muscle metabolism alone, but also by perception and cognition as well (Morgan, 1981). Such is the inherent variability of mankind, that the quantification and understanding of subjective symptoms and how these relate to objective findings is extremely intricate, dependent as the relationship is upon so many interacting elements.

According to Macphearson (1972), possibly the most important measure of the thermal strain taken by an individual exists at an affective level - the level at which the individual becomes aware of discomfort. This level differs from person to person and for the same person from time to time depending upon numerous physical and psychological factors. In reviewing the concept of thermal neutrality, Astrand and Rodahl (1986) explain that, generally, a state of thermal comfort is characterised by core temperatures of between 36.6 to 37.1°C and skin temperatures between 32 and 35.5°C. It has been reported, however, that thermal comfort in the cold is largely dependent on skin

temperature and mean body temperature, rather than on core temperature (Wagner and Horvath, 1985a). More specifically, sensations of thermal comfort or discomfort appear to be the result of the interaction between signals evoking temperature sensation, input signals for temperature regulation and sensations arising from thermoregulatory activities associated with skin blood flow, sweating and shivering (Hardy *et al.*, 1971).

The effect of CWI upon the perception of thermal sensation during submaximal exercise on land immediately afterwards is thus of great interest, since many of the factors mentioned by Hardy and co-workers would presumably be altered compared to during exercise without prior cold exposure. If skin temperature is seen as being a particularly important influence in the experience of thermal sensations, then a change in the skin-core mean body temperature relationship in this regard would be predicted following CWI: ratings of perceived thermal sensation may be lower at a given core temperature. Were the perceptions of thermal sensations to be altered, the risk of thermal injury during exercise in the heat following CWI may be greater.

Many of the physiological consequences of CWI are likely to have direct implications for other perceptual responses, notably the subjective assessment of effort expenditure. Morgan (1981) emphasises that psychophysiological judgements of effort-cost or perceived exertion involve the interactive configuration of numerous input parameters, and as such represent

a *Gestalt*, rather than isolated factors. Changes in any one or a combination of those factors, however, are likely to influence subjective reactions to physical work. Physiological and neuromuscular variables proposed as contributors to effort perception include heart rate, respiratory rate, minute ventilation, oxygen uptake, lactate accumulation, catecholamine production, and core and skin temperatures, all of which may be altered during endurance exercise following CWI.

The complexity of effort perception renders an examination of any one of the physiological components upon which it is based extremely difficult, and only serves to further emphasise the need for an integrated psychophysiological approach such as was attempted in the present study. The effect of ambient or body temperatures upon the rating of perceived exertion (RPE) during exercise, for example, cannot be separated from associated changes in other parameters such as minute ventilation or blood lactate levels. Carton and Rhodes (1985) have also stipulated the need to discriminate between perceived effort and perceived discomfort where skin or core temperatures have been implicated as perceptual cues affecting RPE.

Horstman (1977) found RPE to be significantly lower when exercising at 5°C, compared to the same test performed at 25°C. This reduction in RPE paralleled an increase in minute ventilation and $\dot{V}O_2$, and decreases in the respiratory quotient and venous lactate concentration at 5°C. Immersion in cold water to the extent that the subjects are hypothermic may,

however, have different effects on RPE during aerobic performance on land. A less economical performance following CWI may increase RPE at any given workload compared to exercise without prior cold exposure, although some evidence exists to suggest that RPE may not be significantly influenced by reductions in body temperatures (Horvath, 1981).

The implications of cold-induced variations in physiological responses for differentiated ratings of perceived exertion must also be considered. It has been suggested that differentiated reports of effort sense more closely reflect discrete physiological symptoms than do generalised or overall ratings (Pandolf *et al.*, 1975; Pandolf, 1978; Carton and Rhodes, 1985). Nevertheless, overall RPE may be dominated by any one parameter that becomes accentuated (Pandolf, 1978). In a review of the related literature, Carton and Rhodes (1985) concluded that local factors, such as muscular strain, exert a greater influence upon effort perception at exercise intensities which do not greatly stress central factors, while central factors exert greater influence at higher levels of work. Cognisance must, however, be taken of the fact that different methodological approaches have resulted in noticeably different results from studies that have examined differentiated RPE during exercise. Horstman *et al.* (1979) demonstrated, for example, that physical strain in the working muscles may be perceptually prominent at higher workloads if mechanical efficiency is poor. It is clear, therefore, that, within the broad context of CWI effects on subsequent aerobic performance, several fundamental questions within the area of

perceived exertion exist. Elucidation of the primary factors involved in the formation of effort sense following body cooling in this way necessitates the adoption of a holistic conceptual framework from which to begin.

STATEMENT OF THE PROBLEM.

This study sought to achieve an integrated, holistic approach to the broad question of CWI effects on subsequent aerobic performance on land. Within the context of the conceptual questions raised in the introduction, and in the light of the somewhat controversial findings reported in a relatively small number of related studies, the present investigation sought to determine the extent to which aerobic performance on land was affected by body cooling of varying degrees induced by CWI and the relative importance of peripheral *versus* central cooling in this regard. Physiological responses were examined at a pre-determined percentage of $\dot{V}O_2$ max, both with and without prior manipulation of the thermal status of the individual.

Furthermore, the research set out to establish whether, with body cooling, sex-based differences in thermoregulation did exist and, if so, the extent to which such differences might influence physiological and psychological responses to ensuing dry submaximal exercise. In the light of reports of sex differences in thermal sensitivity and the traditional explanations for this based on body compositional differences, particular focus was given to the question whether, when subjects were normalised for

morphological and dimensional differences, cooling of the core *versus* the insulative shell would affect submaximal performance differently for males compared to females.

An additional aim of the present study was the evaluation of the participants' subjective reactions to submaximal exercise following prior cold water exposure. In other words, would body cooling as a consequence of CWI exert a significant influence upon the formation of effort sense and the perception of thermal sensations? Also, what is the relative importance for both of these subjective reactions of cooling the shell only compared to central cooling? As discussed earlier, overall RPE may be dominated by either local or central cues, depending on a variety of factors. Of importance in this regard was whether body cooling of different degrees would alter the relationship of local and central cues in the expression of overall RPE at a given exercise intensity. In the case of perceived thermal sensation, it has been reported that vasoconstriction due to cold exposure is rapidly associated with sensations of cold and discomfort. In general terms, CWI prior to dry submaximal exercise may alter thermal sensations compared to control conditions. More specifically, this study sought to establish whether any modification occurred in the interaction of thermoregulation-linked signals depending on the extent of peripheral and central cooling.

Central to this research, therefore, was a clear understanding of the concept of cooling as it applied to the body core and

peripheral tissues outside the core. Although the insulative shell of the body was originally considered to comprise subcutaneous fat alone, today it is accepted that the mass of both subcutaneous fat and muscle tissue has a major influence on body heat loss. A shift to a smaller core and a larger shell, as would be expected following CWI (Kollias *et al.*, 1974; Pendergast, 1988; Rennie, 1988), would significantly modify patterns of heat flux. Thus in the present research, a predetermined loss in body heat **without** a change in core temperature (as discussed in detail in Chapter 3) was referred to as cooling of the peripheral shell. Cooling of the core was accepted as a decline in rectal temperature of 1°C. For the purposes of this study the term hypothermia was applied to such a reduction in core temperature. Hypothermia can be defined simply as a subnormal body temperature (Chambers Twentieth Century Dictionary, 1979). More clinical definitions abound. Those that have provided temperatures at which hypothermia is assumed include: a body core temperature of less than 36.6°C (Webster's Medical Dictionary, 1986), a core temperature of less than 35°C (Askew, 1989; Fritz and Perrin, 1989; Finlay *et al.*, 1995), or a continuum ranging from moderate hypothermia, where the core temperature is 35°C or less, to deep hypothermia, where the core temperature is less than 30°C (Muza *et al.*, 1988; Sutherland, 1992). It is known, however that even comparatively small depressions of body temperature can significantly impair bodily function (Coleshaw *et al.*, 1983; Nadel, 1984; Shephard, 1985; Askew, 1989). For the purposes of the present research, therefore, it was considered that the term hypothermia could

justifiably be applied to a reduction in core temperature of 1°C.

Ethical Considerations

In the light of the physiological adjustments likely to occur with body cooling, particularly in water, thorough consideration was given to the safety of the voluntary participants in this experiment. Keatinge (1969) has pointed out that, from a safety point of view, the most important effects of cold are on the heart. Of primary concern in the context of exercising subjects immediately following central cooling was thus the increased risk of cardiac arrythmias. According to Maclean and Emslie-Smith (1977), the incidence of atrial dysrythmias, especially atrial fibrillation, increases as core temperature falls, but only becomes common at body temperatures below 29.5°C. These authors comment that such atrial dysrythmias are usually benign and transient. The gravest hazard of hypothermia is considered to be ventricular fibrillation. Keatinge (1969) states that as a result of the effect of hypothermia on the oxyhaemoglobin dissociation curve, the supply of oxygen by the coronary arteries is insufficient to meet the needs of the myocardium, a situation that favours fibrillation. Several researchers have, however, stated that the likelihood of cardiac arrythmias is very rare at core temperatures above 33°C (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Hong, 1984). Indeed, Giesbrecht *et al.* (1987), in a study of the effects of exercise upon subjects cooled to a core temperature of as low as 33°C, stated that core temperatures as low as 32°C did not in themselves present any particular danger to otherwise healthy young individuals, even in the presence of

moderate exercise, provided that core temperature was monitored. Nevertheless, cognisance must be taken of the fact that where central cooling is severe enough to markedly reduce the supply of oxygen to the myocardium, exercise would pose an additional risk to the individual. Tipton (pers. comm.) has also commented on the importance for heavy exercise of attenuated reflex cardiovascular responses as a result of central cooling. Maclean and Emslie-Smith (1977) report, however, that most circulatory reflexes associated with the adjustment of arterial blood pressure are only seriously depressed below 28°C.

With this background in mind, it was imperative that the design of the present research protocol reduced to a minimum the potential risks to the subjects. Guyton (1986) has listed the average normal range in core temperature (measured rectally) as lying between 37.3°C and 37.6°C. A reduction in core temperature of 1°C, the criterion for central cooling in the present study, would result in a core temperature of 36.3°C at the lower end of Guyton's range. Such core temperatures would be well above the 35.5°C suggested by Shephard (pers. comm.) as a limit to reduced central temperatures in an experiment of this nature and the 34.5°C suggested by Keatinge (pers. comm.). The criteria for the immediate termination of an immersion during body cooling phases comprised: a rectal temperature below 35°C; the appearance of any undesirable cardiac rhythms; severe muscular discomfort; mental disorientation and/or subject request.

Furthermore, on the advice of established researchers in the field of thermal physiology, the workload performed by the subjects was set at a submaximal level. Several researchers expressed confidence that heavy exercise could safely be tackled following a reduction in core temperature to as low as 35°C (Brotherhood; Hayward; Horvath; Yach, pers. comm.). However the reservation expressed by some scientists regarding the increased risk of cardiac arrythmias with heavy exercise (Doubt; Shephard; Tipton, pers. comm.) led to the decision to limit the exercise intensity to 50% $\dot{V}O_2$ max. As a further safety precaution, however, subjects' ECGs were monitored throughout all testing sessions involving water immersion and/or exercise performance. A certified paramedic was present at all times.

RESEARCH HYPOTHESES.

The hypotheses generated in the present study were such as to enable a broad examination of the effects of CWI upon subsequent aerobic performance on land, in order to examine the question of peripheral *versus* central cooling and the implications of sex-based differences in thermal responses.

Hypothesis 1:

Neither peripheral nor central cooling induced by head-out whole body immersion in cold water has an affect upon physiological and psychophysical responses to ensuing dry submaximal exercise.

Stated statistically, the null hypothesis was:

$$H_o : \mu_a = \mu_b = \mu_c$$

$$H_a : \mu_a \neq \mu_b \neq \mu_c$$

Where: μ_a represents the mean physiological and psychophysical responses to submaximal exercise without prior CWI.

μ_b represents the mean physiological and psychophysical responses to submaximal exercise following peripheral cooling as a result of CWI.

μ_c represents the mean physiological and psychophysical responses to submaximal exercise following central and peripheral cooling as a result of CWI.

Hypothesis 2:

There are no sex-based differences in physiological or psychophysical responses to submaximal exercise on land following head-out whole body immersion in cold water.

Stated statistically, the null hypothesis was:

$$H_o : \mu_m = \mu_f$$

$$H_a : \mu_m \neq \mu_f$$

Where: μ_m represents the mean physiological and psychophysiological responses of the male subjects to submaximal exercise, both with and without prior CWI.

μ_f represents the mean physiological and psychophysiological responses of the female subjects to submaximal exercise, both with and without prior CWI.

DELIMITATIONS.

The sample comprised 13 males and 13 females between the ages of 18 and 25yr, none of whom was accustomed to CWI on a regular basis. In order to account for some of the factors said to influence responses to cold stress, initial subject selection was based upon morphological criteria. On the basis of the results of a $\dot{V}O_2$ max test, subject selection was further restricted to control for the possible effects of cardiorespiratory capacity upon CWI responses. All subjects underwent habituation to the methodological procedures prior to any data collection.

On separate occasions the participants then completed three 30min treadmill runs intended to replicate 50% $\dot{V}O_2$ max. The order in which these were performed was randomly assigned to each individual. Control data were collected for one of the sessions performed without prior cold exposure. At the remaining two data collection sessions, the same task was repeated, once following peripheral cooling by CWI and once when the subjects had

undergone peripheral and central cooling by CWI.

With the exception of rectal and skin temperatures, which were measured telemetrically, physiological parameters were measured during dry exercise by means of an on-line data acquisition system. Ratings of perceived exertion and of thermal sensation were also recorded.

Cold exposure took place in a water tank designed for this purpose, with the body immersed to the level of the neck. The temperature of the water was stable at 20°C. During the cold water exposure physiological and psychophysical variables were measured telemetrically or manually. The cold water immersions were terminated after a pre-determined fall in body heat stores without any change in core temperature, or when the rectal temperature fell by 1°C.

Statistical treatment of the data involved the use of single variable statistics to describe each set of data. Multi-variate statistics in the form of two-factor analyses of variance with repeated measures were used to test for significant differences between sets of data. The 0.05 level of significance was applied to all statistical procedures.

LIMITATIONS.

While every effort was made to ensure rigorous control of the research in general, and in particular those factors considered to influence CWI responses, the following limitations must be

noted:

1. In order to assess the relative importance of peripheral *versus* central cooling and to control for inter-individual differences in the rate of cooling, the method used in the present study required that immersion in the water tank be regulated around the time necessary for a pre-determined amount of heat to be lost (peripheral cooling), or for core temperature to drop by 1°C (peripheral and central cooling). The use of indirect calorimetry, the method employed in this study, to evaluate heat loss, has been criticised on the basis of difficulties inherent in the measurement of mean body temperature in water (Hong, 1984; Shiraki *et al.*, 1986). Nevertheless, it is acknowledged that in the absence of facilities for direct calorimetry, values obtained by estimation provide acceptable approximations and as such this method is commonly used over varying time periods during studies of CWI (Rennie *et al.*, 1962; Veicsteinas *et al.* 1982; Park *et al.*, 1983; Shiraki *et al.*, 1986; Choi *et al.*, 1988). Thus in the present study, an approximation on the basis of indirect calorimetry, calculated for each subject in the same way, was deemed acceptable.

2. Central to this research was a comparison of male and female psychophysiological responses to body cooling. Every attempt was made to match male and female subjects on the basis of morphological criteria. Part of this involved a determination of their body composition, particularly in the form of absolute and relative fat and muscle tissue masses, as calculated by the

method of Drinkwater and Ross (1980). The assessment of body composition in this manner is, however, based upon several assumptions, the most pertinent of which involve the conversion of skinfold measures into an estimation of total body fat. Martin *et al.* (1984) have remarked upon the fact that many of these assumptions may not be entirely valid. Short of cadaver dissection, any assessment of body composition remains an estimation. While it was considered useful to include an approximation of fat and muscle tissue masses, the suggestion of several authors (Ross and Marfell-Jones, 1982; Martin *et al.*, 1984; Jackson and Pollock, 1985) that skinfold measures be used directly as a structural measure was acknowledged and a total of eight skinfolds were summed.

Furthermore, it proved impossible with the use of a combination of morphological criteria in the present study to exactly match male and female subjects according to those factors said to influence thermogenic responses. Previous studies relating to this topic have been criticised for not using matched subjects (Taylor *et al.*, 1986). Of the 47 individuals who volunteered to participate in the present research, the 26 most suitable on the basis of morphological and cardiorespiratory requirements were selected as subjects. That they were not matched on every one of the chosen criteria may be regarded as a factor limiting the interpretation of sex effects upon responses to cooling. Nevertheless the scope of this attempt to establish subject uniformity for both sexes apparently extended beyond that of any reported study in this field and provided comprehensive

information concerning the physical characteristics of the subjects used.

3. Of importance with regard to the thermal responses to cold stress was the menstrual status of the female subjects, since it has been suggested that fertility can influence a woman's body temperatures (Eston, 1984; Graham, 1988). Female subjects were selected from a pool of solicited volunteers on the basis of the regularity of their cycles, together with morphological and cardiorespiratory criteria. While it was hoped to recruit females who were not utilizing oral contraceptives, a small number of the women who took part as subjects did so (3 of 13). Testing times for the female subjects were chosen so as to ensure that data collection sessions fell at the same point in the ovulatory cycle for each individual (between days 5 and 12 after the onset of menses). It has been suggested that counting days in order to establish menstrual cycles, as was the case in this study, may pose a limitation, since some subjects may ovulate late or not at all (Doolittle and Engebretsen, 1972). In the present study, however, females used charts to record the date of menstruation for the three months prior to data collection. This established cycle regularity for all subjects and by restricting the testing times to within a fixed number of days from the onset of menses, an observable phenomenon, the problems outlined by Doolittle and Engebretsen were avoided.

CHAPTER 2

REVIEW OF LITERATURE

TEMPERATURE REGULATION

In an unprotected state man can tolerate only relatively small variations in deep body temperature, a fact which has led Shephard (1985) to describe humans as being "metabolic hostages of the homeothermic condition". The temperature of the deep tissues of the body - the core - varies within about 1°C in the course of 24 hours (Sloan, 1979), sometimes even in the face of large alterations in environmental temperature (Hong, 1984). The average normal core temperature (measured rectally) is generally considered to lie between 37.3°C and 37.6°C (Guyton, 1986). Nonetheless, even under resting conditions, temperature gradients exist between central regions of the body, rectal temperature being slightly higher than other regions of the core (Minard and Copman, 1963). The temperature of the skin, by comparison, is lower than that of the core and varies considerably with different degrees of physical activity and in different environmental conditions. According to Hardy *et al.* (1971), temperature differences between the various skin areas over the body vary depending on environmental temperature. At 30°C, for example, the warmest areas are usually the trunk and head; the coolest are the arms and legs, with the hands and feet somewhat warmer owing to rapid blood flow. In the cold, larger temperature differences begin to appear; at 18-20°C the feet may be as much as 6-12°C colder than the forehead (Hardy *et al.*,

1971).

If man is to remain in thermal equilibrium the body must gain as much heat as it loses, a basic principle of thermodynamics that can be described by the equation:

$$M \pm R \pm C \pm K - E = 0$$

where: M = the internal heat production from metabolism, influenced by any work being performed; R = the rate of loss or gain of heat by radiation; C = the rate of loss or gain of heat by convection; K = the rate of heat loss or gain by conduction and E represents total evaporative heat loss, caused by the evaporation of moisture from the skin and the respiratory tract (Maclean and Emslie-Smith, 1977; Hong, 1984). In most conditions conductive heat exchange is negligible, but increases in importance during water immersion, due to the fact that the thermoconductivity of water is approximately 25 times greater than that of air (Ästrand and Rodahl, 1986; Nadel, 1984). Nadel has pointed out, however, that although a constant but small transfer of heat does occur via conduction, the primary avenue of heat transfer from the body surfaces to the water is convection. Radiation transfer is negligible because of the long-wave infrared radiation of the body surface and because water absorbs so effectively (Hong, 1984). Thus heat flux can be described simply as:

$$HF = h_c \ (T_{sk} - T_w)$$

where: HF = the rate of energy transfer; h_c = the convective transfer coefficient and $T_{sk} - T_w$ = the skin to water temperature gradient (Nadel, 1984). Rapp (1971) determined the conductive heat transfer coefficient to be about $11W.m^{-2}.°C^{-1}$, regardless of the degree of water stirring. The convective heat transfer coefficient, on the other hand, is influenced by the velocity of the water. In the study described by Rapp the convective heat transfer coefficient of still water was $94W.m^{-2}.°C^{-1}$, compared to $400W.m^{-2}.°C^{-1}$ at a swimming speed of $0.5m.s^{-1}$. Since in the above equation heat flux is affected by both the value of h_c and the temperature gradient from skin to water, the greater the resistance to heat flow from the body core to the skin, the less heat flux can be expected. Indeed, it has been pointed out that heat loss in water is limited largely by the body (core-to-skin) tissue insulation and not by the skin-to-water heat transfer coefficient, an observation based on the fact that although the convective heat transfer coefficient is significantly different between air and water, heat loss to water has been estimated to be only about two to five times that in air at the same temperature (Merrill-Smith and Hanna, 1975; Hong, 1984). Bullard and Rapp (1970) and Nadel (1984) describe the resistance to heat flow from core to skin as being composed of a variable resistance, offered by the adjustable conductance of heat in the circulation of blood from core to skin, and a fixed resistance, which is a function of the thickness of the layer of subcutaneous fat and non-perfused muscle.

Subcutaneous fat is one of two layers that separate the muscle

and central body from the environment. The other, the skin, consists of a surface epithelium, the epidermis, and a connective tissue layer, the dermis, with few fat cells and with a large vascular bed (Veicsteinas *et al.*, 1982). Guyton (1986) points out that in the most exposed areas of the body - the hands, feet and ears - the venous plexus that lies immediately beneath the skin is supplied directly from the small arteries via arteriovenous anastomoses, thus allowing blood flow to bypass the capillary bed. The vascular mass flow of heat to the skin is a product of the cutaneous blood flow and the change in blood temperature (Hertzman, 1963). Skin blood flow can vary tremendously, from barely above zero to as much as 30% of the total cardiac output, or 100 times the minimum (Maclean and Emslie-Smith, 1977; Guyton, 1986). In absolute terms Hong (1984) suggests that skin blood flow at rest ranges from about $150ml.min^{-1}$ in a cool environment to about $2000ml.min^{-1}$ for conditions of heat stress.

The mass flow of heat from deep tissues to the surface is reduced by both constriction of peripheral vascular beds and by the reduction of blood velocity as a result of a cold-induced increase in viscosity (Maclean and Emslie-Smith, 1977). The effectiveness of vasoconstriction in reducing heat loss is further enhanced by counter-current exchange of heat between arteries and veins in the distal half of the limbs (Hart, 1963; Keatinge, 1969). Rather than returning in skin veins, blood from these areas returns in deep veins alongside the artery, thus minimising heat transfer to the skin. Ultimately the body's

surface temperature falls toward the ambient temperature. Under conditions of maximal vasoconstriction the variable resistance discussed by Bullard and Rapp and by Nadel is maximised. Hong (1984) stated that, in water, the lowest end of the zone of vasomotor regulation at which maximal peripheral vasoconstriction develops is as high as 29°-33°C, depending on subcutaneous fat thickness. According to Bullard and Rapp (1970), maximum insulation values, as affected by changes in blood flow, are established at water temperatures below 30°C, whereas Rennie *et al.* (1962) established that cutaneous vasoconstriction became maximal in water temperatures between 31°C and 33°C. The latter authors stated that an analogous increase in body insulation occured as air temperature was reduced to 28°C - only comparatively slight changes in insulation accompanied any further decline in air temperature.

A large part of the thermal control of blood flow is brought about by sympathetic nervous system control (Keatinge, 1969; Guyton, 1986), although Johnson and Hales (1984) point out that the thermoregulatory system is unique amongst the major physiological systems in that it has, as effectors, no anatomically specialised structures apart from the sweat glands. The thermoregulatory blood flow through the extremities is controlled by noradrenergic sympathetic fibres, but that of the trunk and proximal parts of the limbs is governed by cholinergic fibres (Maclean and Emslie-Smith, 1977). Keatinge (1969) has pointed out that although noradrenaline-containing fibres do not normally penetrate the smooth muscle of small arteries, the

transmission of action potentials from one smooth muscle cell to another and the diffusion of noradrenaline combine to stimulate smooth muscle cells distant from the nerve fibre. Much of the action of the cholinergic mechanism governing the release of vasoconstrictor tone in response to warmth is exerted indirectly. The release of acetylcholine stimulates the sweat glands, which release kallikrein and this in turn catalyses the production of the vasoactive peptide bradykinin (Keatinge, 1969; Maclean and Emslie-Smith, 1977). The degree of vasomotor control of vessels in different bodily areas varies, with the greatest control exerted in the fingers and toes (Burton, 1963). The vessels of the head, however, are uniquely unable to vasoconstrict in response to cold because of the absence of sphincteric control in scalp vessels (Hong, 1984). Thus in response to cold there can be practically no change in the effective tissue insulation of the skin of the head, although heat does produce sweating and vasodilatation (Maclean and Emslie-Smith, 1977).

Keatinge (1969) emphasises, however, that not all thermal changes in skin blood flow are brought about by nerves. Factors such as vasodilatation, reflexly initiated by skin receptors sensitive to warmth (Maclean and Emslie-Smith, 1977), hormonal influences (Benzinger *et al.*, 1963) or the direct effect of heat or cold upon the blood vessels (Benzinger *et al.*, 1963; Hertzman, 1963; Maclean and Emslie-Smith, 1977), contribute to man's overall thermoregulatory response. Indeed Hertzman has commented that, of local skin temperature and the liberation of bradykinin, the former may be a more powerful influence with regard to the total

cutaneous perfusion.

Although the relative importance of peripheral and central components in the regulation of temperature is still debated, the role of the hypothalamus as a coordinating centre for the various processes involved in the maintenance of body temperature has long been acknowledged. The hypothalamic control areas lie deep in the cranium, far removed from external influences. The blood supply of the brain is also relatively well protected from external influences since both the internal carotid and vertebral arteries take a deep course and have little cooling influence from venous blood returning from body surfaces (Randall, 1963). Indeed, in normal circumstances, the flow of arterial blood serves to cool the anterior hypothalamus (Randall, 1963). Many scientists explain temperature regulation in terms of the concepts of systems control, where the hypothalamus acts as a central "thermostat" that makes thermoregulatory adjustments to deviations from a temperature norm or set-point (Benzinger *et al.*, 1963; Hayward *et al.*, 1977; Maclean and Emslie-Smith, 1977; Guyton, 1986). Gisolfi (1983) has commented that the very term "temperature regulation" implies the existence of a fixed temperature that various control systems attempt to maintain. The statements of Benzinger and co-workers in this regard, often cited by thermal physiologists in explaining the process of temperature regulation, deserve particular mention. Their conclusions concentrated almost exclusively upon the hypothalamus as a central sensory organ that ultimately controls all thermoregulatory mechanisms, either automatic or behavioral

(Maclean and Emslie-Smith, 1977). This view has been subject to some criticism. Benzinger's concepts were rejected by Fox (1974) on the basis of their "uncompromising simplicity". Burton (1963) questioned the proposal that, at least in response to heat, the skin does not serve as a sensory component in the control of sweating and vascular reactions except to elicit conditioned reflex activity. This author preferred instead a double control of the hypothalamic centres, from peripheral afferents as well as from brain temperature. Similarly, Hertzman (1963) commented that experiments where precipitous inhibition of sweating occurred as a result of cooling a limited area of skin without a reduction in deep body temperature were not readily explained in terms of exclusive hypothalamic control. He concluded that peripheral influences in response to heat cannot be considered minor; they may dominate a particular situation. Further evidence of this was presented by Ivanov (1984), who reported that increases in mean body temperature of as little as 0.05-0.08°C resulted in specific thermoregulatory processes and the loss of additional heat, without preliminary temperature changes in the hypothalamus or spinal cord.

The regulating system of homeothermic man can be said to involve the complex integration of multiple input signals from a large number of widely distributed sensing elements and a multiple, distributed set of effector mechanisms (Burton, 1963). In addition to temperature receptors in the hypothalamus, sensing elements described by Burton include receptors in the skin and in a few deep tissues of the body. The latter, which detect

cold rather than warmth, are found mainly in the spinal cord, in the abdominal viscera and in or around the great veins and are therefore exposed to the core temperature rather than surface temperature (Guyton, 1986). Despite the available evidence of such deep body thermosensitivity, however, electrophysiological characterisation of receptors is poor, and is compounded by the fact that enteroceptors may respond to a variety of stimuli, of which temperature is one (Necker, 1984). Carotid body chemoreceptors and baroreceptors, for example, have been shown to be highly sensitive to warming. Although skin cold receptors are more prolific than those of warmth, both are unevenly distributed, with higher density in the skin of the extremities and the face, for example (Burton, 1963). The firing rate of such receptors depends on the rate of change of temperature, as well as on level of temperature; patterns of heat energy are converted into afferent impulses to be relayed to the hypothalamus for coordination. According to Schäfer *et al.* (1984), thermoreceptors in the skin respond to the physical stimulus heat energy in the innocuous intensity range; in other words, they exhibit activity between the extremes of heat pain and cold pain. Furthermore, maximal sensitivity to either constant temperatures or rapid temperature changes does not necessarily lie in the same temperature range. With regard to maximal static activity, cold fibre maxima are distributed in the range from -5°C to 40°C, warm fibre maxima occur in the range from 29°C to 38°C. Maximal dynamic responses, on the other hand, are distributed over a smaller temperature range of 20°C to 30°C in cold and 29°C to 32°C in warm fibres (Schäfer *et al.*,

1984). Schäfer and co-workers point out that the sensation of warm and cold in accordance with the differential sensitivity of thermoreceptors to thermal transients has enabled a division into the qualities of warmth and cold. The skin temperature at which discrimination between these qualities can be made was established by Benzinger *et al.* (1963) to be 33°C. These authors stated that, in response to cold exposure, skin thermoreception begins to stimulate metabolic activity at this threshold skin temperature, although maximal thermal stimulation took place at a skin temperature of 20°C. Benzinger *et al.* described experiments where, in response to a steady stimulus of cold at the skin, the rate of oxygen consumption attained a maximum at 20°C; at lower skin temperatures the response in oxygen consumption decreased. Increasing cold sensation at temperatures below 20°C has been attributed to the activity of low temperature cold receptors (Schäfer *et al.*, 1984).

The modulating effect during cold exposure of skin temperature upon the threshold hypothalamic temperature at which metabolic heat production is increased above the resting set point has been well established (Hayward *et al.*, 1977; Guyton, 1986). Under normal conditions the set-point of the temperature control mechanism is carefully regulated at about 37°C ± 1°C (Benzinger *et al.*, 1963; Guyton, 1986). At temperatures above or below this level responses of heat loss or heat production are initiated so that the body temperature reapproaches the original set-point. At high skin temperatures, however, sweating, as a means of promoting heat loss, begins at a much lower hypothalamic

temperature than when skin temperatures are low. Similarly, when the skin becomes cold it drives the hypothalamic thermostat to the shivering threshold even when the hypothalamic temperature itself is still quite hot (Guyton, 1986). In those situations where messages of cold from the skin contradict central warm reception, during exertion in cold ambient conditions, for example, the cold reception at the skin may interfere with thermostatic control and prevent or delay the immediate adjustment of internal temperatures through sweating (Benzinger *et al.*, 1963).

TEMPERATURE REGULATION DURING EXERCISE

Factors Influencing Temperature Regulation During Exercise

Exercise in ambient conditions other than those of thermal neutrality represents a situation where man must simultaneously effect a number of homeostatic balances. Gisolfi (1983) points out that temperature regulation results from the integration of several hierarchically arranged systems; systems which, in attempting to maintain the internal environment, must compete for limited resources (McFarland, 1984). Consequently, a given situation is characterised by an inevitable trade-off and compromise between different competing homeostatic systems (McFarland, 1984), and the final thermoregulatory outcome is determined by the relative strengths of the various homeostatic drives (Johnson and Hales, 1984). Nadel (1983b) describes the regulation of body temperature during exercise as being the outcome of a system involving several orders of complexity, of which the interaction that the temperature regulatory system has

with other regulatory systems is but one. According to this author, temperature regulation during exercise is, at its simplest, a comparison of changes in internal temperature with idealised information within the thermoregulatory centre. Some authors have described this process in terms of a proportional controller, where each regulatory response is proportional to the difference, or "load error" between a fixed internal temperature and a threshold or set-point temperature for that response (Fox, 1974; Gisolfi, 1983; Nadel, 1983b). It was originally postulated by M. Nielsen (1938) that exercise resulted in a higher body temperature set-point which accounted for increases in internal temperature, thus focusing upon the interactions between various temperature-sensitive neuronal inputs (Harrison *et al.*, 1978). The concept of a set-point shift during exercise was much debated in subsequent research, with differing conclusions. Some have reported an increase in the set-point (Robinson, 1963; B. Nielsen, 1969), others a decrease (Baum *et al.*, 1976; Tam *et al.*, 1978) and yet others no change (Cabanac *et al.*, 1971; Nadel 1983b and 1985). Nadel (1983b and 1985) has argued that the depiction of temperature regulation during exercise in terms of a sustained error signal between the actual core temperature and a regulated temperature renders the concept of a change in the set-point redundant; an elevated internal temperature is a consequence of an imbalance between the rates of heat production and heat dissipation (Gisolfi, 1983; Nadel, 1983b and 1985). Furthermore, a substantial body of evidence exists to suggest that non-thermal factors may be of considerable importance to thermoregulatory responses. Such evidence,

according to Harrison *et al.* (1978), should allow for the resolution of the apparent conflict between those who consider body heat content to be the regulated variable, and those who favour body temperature as the regulated variable. Nonetheless, Fox (1974) commented that as the "exercise factor" in temperature regulation was due to multiple factors exerting a greater or lesser influence in different situations, a satisfactory explanation for temperature control during exercise was extremely difficult to achieve.

Thus it is established that the relationships between internal temperature and the efferent thermoregulatory responses are not fixed, but are subject to a great number of influences (Nadel, 1985). During exercise the body is subjected to varying metabolic and thermal loads, depending on a number of different factors. The heat generated by exercising muscles is considerable; since mechanical efficiency is approximately 20-25%, 75-80% of the calories produced in the muscle are converted to heat (Wasserman *et al.*, 1967; Åstrand and Rodahl, 1986; Shepard, 1985; Guyton, 1986). According to Guyton, the heat produced during muscle contraction is a consequence of both intracellular chemical reactions and the creation of heat in overcoming viscous resistance to the movement of muscles and joints and to the friction of the blood flowing through the blood vessels. At rest the temperature of the muscles is relatively constant and depending on environmental conditions varies between approximately 33°C and 36.5°C (Saltin *et al.*, 1968; Nadel, 1983a). However during exercise at intensities greater than 25%

$\dot{V}O_2$ max and in ambient temperatures above 20°C (Nobel, 1986), a shift in the core-shell temperature gradient occurs, such that heat is lost from the muscles to the core, primarily via convection in the blood. Thus Saltin *et al.* (1968) stated that muscle temperature during exercise is the net result of a regional heat balance, caused by locally generated metabolic heat, heat carried to and away by the blood, heat lost to superficial tissues by conduction and local heat storage. Once at the skin, heat is dissipated from the body by convection, conduction, radiation and the evaporation of sweat. Muscular work may increase the heat production from 10 to 20 times the heat production at rest (Åstrand and Rodahl, 1986). Initially it was established that the elevation in internal temperature during exercise was a linear function of the absolute exercise intensity and independent of ambient temperature from 5°C to 30°C (Nielsen, 1938). Subsequently an even closer relationship was found between core temperature and relative exercise intensity (Saltin and Hermansen, 1966; Grucza and Hanninen, 1990; Torii *et al.*, 1996). According to Åstrand and Rodahl, a relative exercise intensity of 50% $\dot{V}O_2$ max will elicit a deep body temperature of about 38°C. Skin temperature during exercise, on the other hand, is determined solely by the environmental temperature (Fox, 1974; Torii *et al.*, 1996). This fact is of importance in explaining the independence of a rise in core temperature over a wide ambient temperature range. Kondo *et al.* (1996) have demonstrated, however, that for exercise performed under constant ambient conditions (25°C; 50% relative humidity; 0.3-0.4m.s^{-1} wind velocity), skin temperature is greater the

higher the exercise intensity.

Alterations in heat dissipation produced by the direct and indirect effects of skin temperature superimposed on a constant central drive for sweating and cutaneous vasodilation imply a more or less exact balance between the capacity of heat dissipating mechanisms and the changing ease of thermal exchange with the environment at different ambient temperatures (Fox, 1974; Gisolfi, 1983). Thus Gisolfi explains that, in declining ambient temperatures, evaporative heat loss is inhibited directly by the effect of skin temperature on sweating rate and indirectly by reducing heat transfer to the skin. Fortney and Vroman (1985) have pointed out that, since heat production is proportional to the absolute, rather than the relative exercise intensity, differences in core temperatures between individuals who are working at similar absolute exercise intensities must be due to differences in the efficiency of their heat loss systems.

Effector Mechanisms in the Control of Temperature During Exercise

Vasomotor changes

Of the effector responses available to the body in attempting to dissipate heat (primarily sweating and vasomotor changes), the marked elevation in blood flow to muscle and skin during dynamic exercise is the most important mechanism for dissipating excess heat produced (Roberts and Wenger, 1979; Nadel, 1983a). Dilation of superficial veins allows for a lengthening of the residence time of blood in vessels near the surface of the body, thereby increasing the time interval for heat transfer from warm blood

to the cooler skin (Roberts and Wenger, 1979; Fortney *et al.*, 1983; Fortney and Vroman, 1985). The rate of muscle blood flow is primarily related to the intensity of exercise, whereas the overall skin blood flow response is proportional to a combination of core and skin temperatures, with core temperature being the more influential variable (Fortney and Vroman, 1985; Ferretti *et al.*, 1989). At rest, skin blood flow is proportional to core temperature beyond a certain core temperature vasodilatory threshold. Increasing the average skin temperature causes a lowering of the internal temperature threshold for vasodilation without significantly affecting the slope of the skin blood flow to core temperature relation (Fortney and Vroman, 1985; Ferretti *et al.*, 1989). During exercise in a cool environment, when the demand for skin blood flow is relatively low, the threshold for vasodilation is raised. When skin blood flow is inhibited in a cool environment, therefore, the required heat loss from the body is largely achieved by convection and radiation; the required heat transport from the core to the periphery depends on a steep temperature gradient and more heat transported per unit volume of blood rather than a large skin blood flow (Fox, 1974). At higher skin temperatures, more heat is lost by sweating and the increased skin blood flow compensates for the decreased heat transported per unit volume of blood passing from the core to the periphery (Fox, 1974). Johnston *et al.* (1996) have reported recently that, although alcohol lowers the vasoconstrictive threshold in humans, the core cooling rate is not affected during mild cold exposure.

At the onset of exercise, there is a transient vaso- and venoconstriction which is part of the autonomic adjustment to the exercise (Johnson *et al.*, 1974; Roberts and Wenger, 1979; Fortney *et al.*, 1983; Fortney and Vroman, 1985; Nadel, 1985). Indicative of such a decrease in venous compliance was the large and rapid decrease in forearm venous volume observed by Fortney and co-workers during the first two minutes of exercise. An increase in venomotor tone in this manner may increase the total blood volume available to the central circulation by approximately 25% (Tripathi *et al.*, 1984). After the first few minutes of exercise, however, and provided cardiac output is not compromised, skin blood flow is reported to rise and to continue rising in proportion to the rise in internal temperature (Johnson *et al.*, 1974; Roberts and Wenger, 1979; Fortney and Vroman, 1985) until core temperature reaches about 38°C (Kenny, 1988). Before this temperature is reached, the rise in skin blood flow reflects the dominance of thermal vasodilatory reflexes over competing vasoconstrictor influences (Johnson *et al.*, 1974; Fortney and Vroman, 1985) such that much of the heat produced in the active muscles is delivered to the skin. Several researchers have established that the capacitance of the cutaneous veins also increases with increasing body temperature (Roberts and Wenger, 1979; Tripathi *et al.*, 1984). Tripathi *et al.* found a significant increase in forearm venous capacitance when the ambient temperature was increased from 28 to 37°C, although they noted that other investigators have reported that increasing the ambient temperature above thermoneutrality did not have an augmenting effect upon cutaneous venous capacitance as these

veins were already maximally dilated. When examining skin blood flow *per se*, Johnson and fellow researchers observed that the effect of upright exercise was to markedly reduce the slope of the skin blood flow to core temperature relation. These authors concluded that during upright exercise, particularly in the heat, the skin is relatively vasoconstricted. Furthermore, Fortney *et al.* (1983) noted a change in the consequences of exercise for forearm venous volume, which they accept as an index of venous compliance, depending on the relative intensity of the exercise. During moderate exercise (45% $\dot{V}O_2$ max) increases in forearm venous volume occurred along with increases in forearm blood volume. Heavy exercise (65-70% $\dot{V}O_2$ max), however, had the effect of decreasing forearm venous volume (and presumably, therefore, venous compliance) even though peripheral blood volume increased with rising core temperatures. In explaining what would apparently seem a contradiction in terms, Fortney *et al.* stated the following:

> "It may seem paradoxical that peripheral pooling increases over the course of exercise in the face of falling forearm venous volume. However, forearm venous volume is always measured with the same congesting pressure, and thus is an indication of the level of venous tone rather than a direct index of the amount of blood pooled in the veins; the amount of blood pooled in the veins, on the other hand, depends not only on the state of venous tone but on the pressure of the blood within the veins, which will determine whether the veins are distended or slack. As peripheral blood flow increases as a function of the body temperatures over the course of exercise, we would expect pressure in the veins below the heart to increase accordingly."
>
> (Fortney *et al.*, 1983; p.888).

The consequences of increased peripheral pooling with greater peripheral blood flow in response to rising core temperatures are of importance for the continued efficiency of temperature regulation during exercise. During dynamic exercise the circulatory system must meet the combined demands of the increased metabolic requirements of the exercising muscles and skin blood flow requirements for the transfer of heat. Nadel (1985) points out that the muscular activity of exercise thus applies a load to the shared effector loop of two regulatory systems. A thermal load that is proportional to the exercise intensity is imposed on that system involved in the regulation of body temperature and a volume load (a reduction in central blood volume) that is also related to exercise intensity is applied to the blood pressure control loop. Normally a compromise, as outlined above, is struck between the two and the steady state temperature that is attained during light to moderate exercise is primarily a function of the sensitivity and threshold characteristics of the heat dissipating mechanisms (Nadel 1983b and 1985). During heavy exercise in the heat, however, when peripheral pooling is extensive, cardiac filling pressure is progressively reduced. A reduced cardiac filling pressure unloads cardiopulmonary and arterial baroreceptors, such that a cutaneous vasoconstrictor drive is superimposed on the thermoregulatory vasodilator drive (Roberts and Wenger, 1979; Nadel *et al.*, 1979; Nadel, 1985; Fortney and Vroman, 1985; Tripathi *et al.*, 1990). This, together with an elevation in heart rate in response to stimulation of the blood pressure regulating system (Nadel *et al.*, 1979), insures an adequate

central circulating blood volume, but has the disadvantage of decreasing core-to-skin heat transfer.

The implications of blood pooling for the effectiveness of temperature regulation are further highlighted by reports of differing skin blood flow responses depending upon posture. Johnson *et al.* (1974) demonstrated that, when compared to supine rest, both upright posture and exercise consistently caused reduced skin blood flow at a given core temperature. Both Nadel (1985) and Fortney and Vroman (1985) have made the comment that the upright posture must place a strain on the human cardiovascular system since 70% of the total blood volume is located in vessels below heart level. Johnson and co-workers stated that, separately, upright posture and exercise caused significant alterations of the slope of the skin blood flow-core temperature relationship, with the greatest effect evident when the two stresses were combined. In a similar manner, the positive pressure breathing during exercise has been seen to impair thermoregulatory responses, also due to a reduction in skin blood flow and a higher vasodilatory threshold in the face of low pressure baroreceptor stimulation (Fortney and Vroman, 1985).

Clearly then, variables not measured by skin temperature or deep body temperature can play a significant role in the control of skin blood flow and therefore in the modification of thermoregulation (Johnson *et al.*, 1974; Harrison *et al.*, 1978; Nadel, 1985; Fortney and Vroman, 1985). Indeed any phenomenon

that displaces the internal temperature threshold or modifies the gain of the efferent thermoregulatory pathways will result in a different degree of heat storage in the presence of a given rate of heat production (Nadel, 1985).

A reduction in absolute blood volume during exercise is also known to attenuate the skin blood flow-core temperature slope at higher body temperatures (Nadel *et al.*, 1979; Fortney *et al.*, 1983; Fortney and Vroman, 1985). The reduction in blood volume occurs as a result of the fluid losses associated with sweating and because of the net movement of fluid into the interstitial spaces surrounding the exercising muscles (Fortney and Vroman, 1985). According to Nadel *et al.* (1979), 20min of heavy exercise may result in as much as 17% of the plasma volume, amounting to about 500ml in normal individuals, being lost to the extravascular space. It has been reported that individuals rendered hypovolaemic by progressive dehydration experience an elevation in the vasodilatory threshold with a concomitant increase in heat storage (Harrison *et al.*, 1978; Nadel, 1985; Fortney and Vroman, 1985; Noakes *et al.*, 1991). Nadel (1985) and Tripathi *et al.* (1990) have noted that cutaneous venous compliance is relatively lower in the hypovolaemic than in the normovolaemic state during moderate exercise in the heat, an observation which has been explained in terms of the increased sensitivity of low-pressure baroreceptors below a critically low cardiac filling pressure. Harrison *et al.* (1978) have demonstrated, however, that the impairment of temperature regulation by dehydration may be related, not only to a reduction

in total blood volume, but to elevated levels of plasma sodium ions and osmolarity. They concluded that either of the above may account for the adverse effects of dehydration on thermoregulation by their inhibiting effect on sweating.

Sweating

It is generally accepted that the sweating rate at a given internal temperature is affected by both hypertonicity and hypovolaemia: the exact mechanisms by which each condition mediates sweating are less well understood. In a similar vein to the remarks of Harrison *et al.*, Nadel (1985) stated that hypertonicity resulted in an increase in the internal temperature threshold for sweat onset. Reports of the underlying mechanisms behind this have included the detection by central osmoreceptors of the blood osmotic concentration and the effects of specific ions, in particular sodium ions, on the sensitivity of the sweat glands to a given neural input or on the secretion-absorption process in the sweat glands (Fox, 1974; Fortney and Vroman, 1985). In hypovolaemic subjects a decrease in the sensitivity of the sweating response, per unit of internal temperature increase, has been noted (Nadel, 1983b), with the result that core temperatures are significantly higher in such individuals (Fox, 1974; Åstrand and Rodahl, 1986). Fox (1974) reported the results of a study which indicated that core temperature rose by 0.6°C for each 1% of body-weight loss. Noakes *et al.* (1991) however, established that metabolic rate, not the level of dehydration, is the principal determinant of post-race rectal temperatures in marathon runners and is significantly related to

sweat rates. That the level of dehydration does indeed influence the rectal temperature during exercise was not questionned by these authors, it is rather that the exercising metabolic rate is a more important determinant of the response.

Within seconds of starting or stopping exercise there is an increase and a decrease respectively in the sweat rate (Fox, 1974; Åstrand and Rodahl, 1986; Gisolfi, 1983; Ayling, 1986). The speed of this response has led to the suggestion that this provides further evidence for the importance of non-thermal stimuli in the process of temperature regulation (Fox, 1974; Gislofi, 1983). Ayling (1986) has commented, however, that the speed of the initial sweating response to exercise would indicate that it is not a thermoregulatory one; it is once a steady-state sweating response is developed that it can be considered thermoregulatory. Grucza and Hanninen (1990) reported that, for cycle exercise at 22°C, the time course for the onset of sweating was reduced with increasing work intensity. It is likely that the rise in sweating with the onset of exercise reflects a general activation of the sympathetic nervous system, since it has been demonstrated that sweat glands are sensitive to epinephrine (Gisolfi, 1983). Verde *et al.* (1983) have commented that sweating responses appear to differ for a peripheral stimulus compared to a central stimulus. According to Åstrand and Rodahl (1986), skin temperature and hypothalamic temperature can independently modify the sweating response to exercise. Local skin temperature, however, can modify the effect on the output from a central controller. Nevertheless, apparently

contradictory statements do exist with regard to those factors primarily responsible for driving sweat rate. According to Nadel (1985), the sweating mechanism is primarily responsive to body temperatures. Several researchers have commented that above a sweating threshold, a linear relationship exists between core temperature and sweat rate (Saltin *et al.*, 1968; Roberts and Wenger, 1979; Fortney and Vroman, 1985). Within this context, the effect of increasing skin temperature is a parallel shift in the sweat rate to core temperature relationship (Fortney and Vroman, 1985). Skin cooling can have the opposite effect (Åstrand and Rodahl, 1986). Thus Saltin *et al.* (1968) stated that the correlation between internal body temperature and skin sweating was markedly improved if skin temperature was also taken into account. Yamazaki *et al.* (1993) observed that sweating rate during exercise was greater than during recovery at the same body temperature. This was attributed to greater central sudomotor activity during exercise and the difference in the rate of change of mean skin temperature between exercise and recovery. Fox (1974) discussed research which found that sweat rate changed during positive and negative workloads without any significant alteration in skin or deep body temperatures. It was reported that the sweat rate varied in proportion to the total body heat production. Åstrand and Rodahl (1986), however, in discussing factors which influence sweat rate, differentiate between exercise performed in constant ambient conditions, and exercise performed at a constant intensity. These authors state that in the former case, skin sweating is a linear function of heat production but unrelated to skin temperature, whereas in the

latter case the sweat rate increases with the ambient temperature (and therefore skin temperature) and is unrelated to deep body temperature. Other researchers have stated that sweat rate is greatly dependent on the absolute workload performed during exercise (Kenny, 1988; Greenhaff, 1989). This is not quite consistent with previously discussed viewpoints, since core temperature, for example, which could be proposed as a factor influencing sweat rate at any given exercise intensity, is known to be directly related to the relative rather than the absolute intensity. Greenhaff (1989) did acknowledge that when subjects exercised at a set absolute intensity, their $\dot{V}O_2$ max values significantly influenced the degree of sweat loss, although the $\dot{V}O_2$ max could only account for 45% of the variation in sweat loss during exercise.

In addition to evaporative heat lost via the process of sweating, respiratory evaporation or insensible perspiration contributes to total heat transfer. According to Fortney and Vroman (1985), although respiratory water loss can increase up to 10-fold during exercise in proportion to the metabolic rate, the total contribution of this to the overall heat balance during severe exercise or exercise in a warm environment is almost negligible. The transfer of heat and water from upper airway mucosal surfaces to inspired air via turbulent convection and evaporation is influenced by minute ventilation and the ambient air temperature and water content (Jaeger *et al.*, 1980). These authors point out, however, that net respiratory heat exchange with the environment may be considerably less than the transient decrease

responses of cold stress are merely the inverse of those of heat stress and that cold ceases to be a stress when exercise is performed. In fact, as several authors have pointed out, increased bodily movement during exercise in either air or water, has the effect of increasing the convective and evaporative heat transfer coefficients, thus increasing the rate of heat loss from skin to environment (Keatinge, 1969; Hong and Nadel, 1979; Shephard, 1985; Graham, 1988b). According to Toner *et al.* (1984), the primary physiological factors influencing heat loss during exercise in cold water are the insulation offered by adipose tissue, the body surface area-to-mass ratio, the vasomotor adjustments and the intensity of exercise. In situations on land or in water where the intensity of exercise is insufficient to maintain the body temperature, a general pre-shivering increase of muscle tension and shivering constitute defence mechanisms against continued heat loss. The degree of protection offered by shivering, however, is limited (Shephard, 1985) and it may accelerate rather than retard loss of central body heat due to greater convective and, in water, conductive heat transfer (Hanna and Hong, 1972; Hong, 1984; Wagner and Horvath, 1985a). Wagner and Horvath (1985a) have cited research which established that overt shivering was only 6% efficient in protecting the body against heat loss during 10°C exposures and 11% during -3°C exposures. While acknowledging past research on this effect, Wilkerson *et al.* (1972), however, found that in temperatures ranging from 8°C to 28°C shivering increased heat loss only slightly, while allowing for a relatively large increase in heat production.

Shivering, defined as the synchronous contraction of small groups of motor units against their immediate antagonists (Maclean and Emslie-Smith, 1977; Shephard, 1985), is reported to increase the body's metabolism by between two and five times (Hemingway and Stuart, 1963; Keatinge, 1969; Maclean and Emslie-Smith, 1977; Horvath, 1981; Shephard, 1985). Heat produced by mechanisms that are included in the term non-shivering thermogenesis and heat produced by shivering are said to be additive in their contribution to the total production of body heat (Maclean and Emslie-Smith, 1977; Paolone and Paolone, 1995). Maclean and Emslie-Smith state that acute exposure to cold causes an increase in muscular tone that starts distally and moves proximally, where shivering starts. It is worth noting, however, that there is no general rule governing which muscles shiver first or most, with the result that there is considerable inter-individual variation (Thompson, 1977; Horvath, 1981). An increase in the amount of shivering is reportedly due to an increase in both the number of muscles used and the frequency of each muscle's contractions (Wilkerson *et al.*, 1972; Thompson, 1977). In addition to the limitations to shivering outlined above in terms of the possibility of increased heat loss, increased shivering has the disadvantage of making any co-ordinated motor task difficult to perform and hastening the depletion of energy reserves (Shephard, 1985), with obvious implications for the performance of activity following a bout of severe or prolonged shivering. The onset of fatigue is likely to occur earlier, and with the depletion of stored glycogen, there is an associated water loss (Shephard, 1985) that may compromise thermoregulation

during subsequent exercise in the heat. Tipton *et al.* (1997) have cautioned against the general assumption that exercise can be used as a model for shivering with regard to substrate utilisation. In this context their research demonstrated that the two are not analogous processes. Hepatic glucose output and the rate of glucose utilisation were greater during exercise than during shivering at the same $\dot{V}O_2$. Plasma free fatty acid and beta-hydroxybutyrate levels were lower.

Åstrand and Rodahl (1986) have stated that when the shivering mechanism is switched on, the increase in metabolic rate is superimposed on the energy demand of exercise. It has been noted by other researchers, however, that the amounts of heat produced by shivering and by exercise are not additive (Maclean and Emslie-Smith, 1977), with evidence for this said to lie in the increased heat loss that may occur with shivering, and the fact that shivering is inhibited by exercise (Maclean and Emslie-Smith, 1977; Hong and Nadel, 1979; Horvath, 1981; Shephard, 1985). Indeed the cut-off point of the shivering response is reported to be an exercise intensity that will elicit an oxygen uptake of approximately $1.5 l.min^{-1}$ (Patton and Vogel, 1984). Hong and Nadel found that in a 10°C environment, once the shivering thresholds were surpassed, the increase in metabolic rate attributable to shivering was inversely related both to skin temperature during rest, prior to any changes in core temperature, and to core temperature during exercise, when skin temperature was constant. These authors concluded that the graded inhibition of shivering evident during exercise was of

central origin.

The mechanism whereby shivering is controlled has been the subject of some controversy. In 1960 Keatinge cited research which had found that shivering could occur alternatively as a result of a fall in deep body temperature, without cutaneous stimulation, or by cutaneous stimulation when deep temperatures were unchanged or even raised. On the basis of such evidence, Keatinge commented that the effect of deep body temperature was of little importance in the metabolic response to cold. In contrast, Craig (1971) stated that shivering requires two conditions, a low central temperature and a peripheral stimulus. Benzinger *et al.* (1963) stated that the control of shivering metabolism originated from cold reception at the skin, with deep sensors responsible for warmth reception and protection against overheating. In a subsequent modification of his position, Keatinge (1969) later suggested that shivering was the consequence of a reflex initiated by the cutaneous cold receptors, mediated by the hypothalamus and facilitated by a fall in hypothalamic temperature. According to Guyton (1986), hypothalamic stimulation of shivering occurs when that area in the posterior hypothalamus termed the primary motor centre for shivering is excited by cold signals from the skin and spinal cord. In stating that this centre becomes activated when the body temperature falls even a fraction of a degree below a critical temperature, Guyton does not, however, differentiate between skin and core temperatures. Park *et al.* (1983) state that shivering is not induced by cutaneous cold receptor impulses

unless the hypothalamic temperature is below a critical temperature. Below this temperature, skin temperature plays an important modifying role, such that shivering may occur at higher hypothalamic temperatures the lower the skin temperature. Whilst recognising that cold reception within the body core was the more important determinant of the shivering response, Hong and Nadel (1979) nevertheless observed that shivering metabolism was proportional to decreased core temperature when skin temperature was constant and to skin temperature when internal temperature was constant, below the respective shivering thresholds. They stated that throughout the period of skin cooling, the core temperature, as measured in the esophagus, was unchanged, suggesting that central cooling was not a pre-requisite for the initiation of the shivering mechanism. These authors support a multiplicative model for the control of shivering, which acknowledges the role of signals from both the skin and core, but emphasises the relative importance of the internal temperature. A further point of note is the comment by Hong and Nadel that, in addition to differences in technique between studies, some of the confusion attendant upon this issue may be a function of the way in which temperature changes were achieved. During water immersions, the skin rapidly assumes a more or less uniform temperature that is close to that of the water. In air, however, regional skin temperature may be quite disparate in the cold and may not produce the sensory information that would be predicted from temperature and surface area alone (Hong and Nadel, 1979).

SYMPTOMS OF COLD EXPOSURE.

There are three sequences in body cooling which have an effect on the cardiovascular, respiratory, cerebral and renal systems These are classified by Maclean and Emslie-Smith (1977) as the "safe", "transitional" and "danger" zones of hypothermia. According to these authors, the normal physiological mechanisms for heat conservation and increased heat production still operate in the "safe" zone, whereas in the "danger" zone they have largely ceased. The "transitional" zone between the two extends from about 33°C to about 30°C. Obviously the abnormal physiology associated with each of these stages is a vast topic for consideration, much of which would not be relevant to the present study. Comprehensive reviews of the physiological disturbances produced in man over an extensive range of lowered core temperatures have been provided by several authors, most notably Keatinge (1969) in a volume entitled *Survival in Cold Water*, and Maclean and Emslie-Smith (1977), in their monograph entitled *Accidental Hypothermia*. The present review therefore, was focused primarily upon the effects of body cooling as would apply to this research; in other words, within the ranges termed "safe", as outlined above.

In discussing responses to cold stress one must recognise the difficulty of drawing generalised conclusions, when many of the variables measured depend upon a combination of processes, some of which counteract others. Muza *et al.* (1988), for example, have pointed out that cardiovascular responses in particular vary depending on the nature of the cold stress (air *versus* water,

whole body *versus* local exposure, ambient temperature and duration of exposure) and the metabolic and neurophysiological status of the organism being stressed. The rate of body cooling, initial body temperatures, age, sex and prior experience of cold, are some additional factors which should be considered. Thus discussing, as some authors do, "responses to cold stress" without acknowledging the above can be misleading.

Effects of Cold Upon Cardiovascular Responses

Keatinge (1969) has commented that the most important effects of cold are on the heart. The consequences of cold for the cardiovascular system have been the subject of much research. Studies examining the specific responses of the heart rate have produced variable results, many of which can be attributed to contradictory reports of the degree of deep body cooling required to produce a depressive effect upon the circulatory system. An increase in heart rate, for example, is said to occur during mild hypothermia, the definition of which was given as a core temperature of less than 37°C but more than 35°C (Fritz and Perrin, 1989). Maclean and Emslie-Smith (1977) on the other hand state that ventricular rate gradually falls as core temperature falls into the "danger" zone, which, by their definition, is characterised by a core temperature of approximately 30°C and below. According to these authors, an initial increase in heart rate is partly due to the demands of shivering muscle, and partly to increased sympathoadrenal stimulation. In contrast, bradycardia has been observed during brief cold water immersions (Keatinge, 1969), an observation

attributed to baroreceptor stimulation as a result of a rise in arterial pressure. Keatinge did not, however, comment on the thermogenic state of the subjects in question, nor was the term "brief" elaborated upon. Other researchers have reported no significant difference in the resting heart rate of subjects exposed to 5°C and 20°C environments (Mitchell *et al.*, 1990), despite higher mean arterial blood pressure responses in the 5°C condition. Core temperatures, however, were not altered from one condition to the next.

In addition to whole body cold exposure, local stimulation of restricted parts of the body is known to activate the autonomic nervous system, with resultant modification of cardiovascular responses (LeBlanc *et al.*, 1976; LeBlanc *et al.*, 1978; LeBlanc, 1988; Quirion *et al.*, 1990; Sendowski *et al.*, 1997). Exposure of the hands results in an increase in the heart rate, together with increased plasma levels of norepinephrine, reduced blood flow in the extremities and elevation of both systolic and diastolic pressures (LeBlanc *et al.*, 1978; LeBlanc, 1988; Sendowski *et al.*, 1997). Sendowski and co-workers observed that general and local physiological responses recorded during an upper limb cold water test differed according to the area immersed (index finger, hand or forearm and hand). Index finger cooling led to earlier and faster cold-induced vasodilatation without significant cardiovascular changes, whereas hand or forearm immersion led to a delayed and slower cold-induced vasodilatation with a bradycardia at the end of the test. Exposure of the face to cold produces a reflex slowing of the

heart rate, attributed to activation of the vagus nerve induced by cold stimulation of the facial trigeminal nerve (Angell-James and de Burgh Daly, 1972; Finley *et al.*, 1979; LeBlanc, 1988; Quirion *et al.*, 1990). The depressor effect of facial cooling upon heart rate is evident during exercise, but is progressively lessened at intensities greater than approximately 65% $\dot{V}O_2$ max (Quirion *et al.*, 1990). In contrast, Gordon *et al.* (1990) found that heart rates during submaximal exercise were not significantly modified by neck cooling, although rises in rectal temperature and sweat rates were reduced.

Over an extended period of cold exposure, heart rate slows in proportion to the fall in core temperature (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Sarnaik and Vohra, 1986). Unlike bradycardia under normal physiological control, the progressive bradycardia of hypothermia is characterised by a relatively greater prolongation of systole than diastole (Maclean and Emslie-Smith, 1977). In examining the mechanism by which temperature influences heart rate, it is difficult to separate the local effects of cooling of the heart itself and the central or brain mediated effects of temperature on sinus rate (Rubin, 1987). It is known that cooling of the atria causes a decrease in the rate of spontaneous discharges of the sinus node, whereas the node speeds up if the temperature is raised (Rubin, 1987).

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According to Rubin (1987), heart rate changes during hypothermia are the reciprocal of stroke volume changes and are further adjusted depending on how cardiac output responds to alterations of skin blood flow and body metabolism which accompany changes of core temperature. It is well established that during the initial stages of hypothermia, mean arterial pressure tends to rise (Maclean and Emslie-Smith, 1977; Brown and Oldridge, 1985; Wagner and Horvath, 1985b; Muza *et al.*, 1988), although Wagner and Horvath reported that this response was influenced by age, with younger subjects showing minimal increases in mean systolic and diastolic pressures compared to older subjects. Resting mean arterial pressure was observed to be higher during exposure to a 5°C environment than to a 20°C environment (Mitchell *et al.*, 1990). Thompson (1977), however, differentiated between initial responses to a cool environment prior to the onset of shivering and those in significantly lower temperatures when cutaneous vasoconstriction is accompanied by a substantial metabolic response. According to this author, the former condition is characterised by the maintenance of constant arterial blood pressure in the face of a fall in cardiac output. This he

ascribes to the influence of a nervous reflex involving the carotid sinus and vagus nerves which slow the heart and thus regulate blood pressure despite the constriction of cutaneous blood vessels. Once the shivering response begins, there is an increase in cardiac output, met almost entirely by an increased stroke volume, and a rise in blood pressure (Thompson, 1977). Somewhat contradictory findings are therefore evident if one considers the time-frame involved in each report. A rise in cardiac output as a consequence of a greater stroke volume has been measured in response to initial cold stress (Keatinge, 1969; Brown and Oldridge, 1985; Muza *et al.*, 1988), with no conditions noted with regard to shivering. Static cardiac outputs have also been reported over two hour exposures to decreasing ambient temperatures (Wagner and Horvath, 1985a). Over a range of temperatures from 28°C to 10°C cardiac output increased by about 10% only during exposure to 10°C. Interestingly, however, all cold exposures resulted in increased stroke volume and decreased heart rate in men but not in women, suggesting once again the variability in changes in cardiovascular functions in the cold depending upon a number of modifying factors not always accounted for in the literature.

As core temperature drops steadily during cold exposure myocardial function is progressively depressed. This is reflected in a decline in cardiac output (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Sarnaik and Vohra, 1986; Muza *et al.*, 1988) which, during moderate hypothermia at least, is due to a fall in heart rate rather than in stroke volume (Keatinge, 1969).

The effect of this upon arterial blood pressure is not clear: reports of a reduction in arterial blood pressure (Keatinge, 1969; Muza *et al.*, 1988) are somewhat contradicted by the statement that in the absence of underlying heart disease, hypotension is uncommon, even during deep hypothermia where the core temperature is less than 30°C (Maclean and Emslie-Smith, 1977). In this context it has been stated that the circulatory reflexes associated with the adjustment of arterial blood pressure are only seriously depressed below 28°C (Maclean and Emslie-Smith, 1977), although more recently it has been reported that baroreceptor reflexes may be impaired by moderate hypothermia (Golden *et al.*, 1991). Moderate hypothermia, as defined by Fritz and Perrin (1989), would constitute a core temperature of between 32°C and 35°C.

Indeed the implications of dulled baroreceptor reflexes have been stressed in a recent challenge to the traditional explanation given for the collapse of victims of accidental cold water immersion during rescue or shortly afterwards (Golden *et al.*, 1991). Traditionally the cause of collapse in such situations was attributed to the direct effects of hypothermia as a result of the phenomenon known as *"after-drop"*. The term after-drop was used to describe a further decline in core temperature, typically of 1-2°C (Golden *et al.*, 1991), after removal from cold exposure, with the mechanism behind this response assumed to be the development of peripheral vasodilation from rewarming, causing cold acidotic blood to enter the heart and peripheral circulation (Hong, 1984; Sarnaik and Vohra, 1986; Golden *et al.*, 1991).

According to Hong (1984), ventricular fibrillation may occur in conjunction with after-drop because of a continued decline in core temperature, due to a temperature gradient across the myocardium, or because of hypovolaemic shock secondary to blood loss to the periphery.

Although the phenomenon of the after-drop is not in question, Golden and colleagues have challenged the assumption that this is responsible for all occasions of collapse, sometimes fatal, upon removal from cold water. As an alternative, they suggest that the primary mechanism may be the collapse of arterial pressure brought about by a combination of factors, as outlined below:

> "The circulatory collapse can come about as a result of a number of factors: primary among these are the loss of hydrostatic assistance to venous return and reimposition of the effects of gravity; hypovolaemia; increased blood viscosity; diminished work capacity of the hypothermic heart and reduced time for coronary filling; dulled baroreceptor reflexes; unmeetable demands to perfuse skeletal muscle; psychological stress and pre-existing coronary disease."
>
> (Golden *et al.*, 1991, p144)

Initially, several of the above factors could be said to be of relevance to the conditions imposed during data collection in the present study. A number of these factors did not, however, directly apply: subjects were all in sound health with no histories of heart disease, for example, and were not under psychological stress during data collection, having been thoroughly habituated prior to testing. Of paramount importance

was the fact that the present research design ensured that core temperature at no time fell to levels that would constitute a risk to the subjects. The increased blood viscosity known to occur with body cooling (Keatinge, 1969; Choi *et al.*, 1988; Golden *et al.*, 1991; Poulos and Mollitt, 1991) has been reported to occur during induced hypothermia with body core temperatures of less than 34°C (Poulos and Mollitt, 1991). The contribution of diuresis to a condition of hypovolaemia, as outlined by Golden *et al.*, was minimal in the present study. A cold-induced diuresis has been attributed to a volume regulating response secondary to the increased filling of deep capacitance vessels by blood displaced by cutaneous vasoconstriction (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Golden *et al.*, 1991). This is exacerbated during immersion when a cephalad shift in blood volume occurs (Löllgen *et al.*, 1980; Lanphier and Camporesi, 1984; Greenleaf, 1984; Lin, 1988). Without the development of hypovolaemia, the effects of the reimposition of the gravity are likely to be less dramatic than those outlined by Golden *et al.* (1991). Although the loss of hydrostatic assistance to circulatory function upon termination of immersion was unavoidable in the upright position, the enhanced "muscle pump" action elicited by immediate light exercise would facilitate venous return, as would the initial vasoconstrictory response evident at the onset of exercise (Johnson *et al.*, 1974; Fortney *et al.*, 1983). Golden and co-workers stated that the benefits of the "muscle pump" action are outweighed by the deleterious effect of vasodilation within the muscle bed upon the workload of the heart (a combination of the diminished work capacity of

the hypothermic heart, together with dulled baroreceptor reflexes, may lead to a fall in arterial pressure). However, the level of core temperature at which this scenario of events was postulated (in the vicinity of 33°C) is considerably lower than the maximum of a 1°C drop in pre-immersion core temperatures in the present study would attain. On the basis of the preceding review, it can be assumed that neither cardiac output nor baroreceptor reflexes would be significantly compromised at such core temperatures.

Ethical Considerations

In addition to the ethical considerations discussed above, the relevance of the after-drop itself to the safety of the present subjects required evaluation. The flushing of the heart with cooler blood from the peripheries has been implicated in the occurrence of cardiac arrhythmias (Hong, 1984; Shiraki, pers. comm.). With this in mind, the design of the research protocol required that core temperatures did not nearly approximate values that have been associated with an increased risk of cardiac arrhythmias. It was suggested by Keatinge (pers. comm.) that there was likely to be little risk to the subjects in the present research provided core temperature was not allowed to fall below 34.5°C, allowing for after-drop.

Indeed, the problems encountered in the present research with what could be termed a "safe" decrease in core temperature, most noticeably from the point of view of an increased risk of cardiac arrhythmias, served to highlight an interesting ethical dilemma

with regard to the formulation of a safe research protocol. It is a basic ethical requisite of experimentation involving the use of human subjects that a comparison of all foreseeable risks and reasonably probable benefits to the subject must indicate that the latter clearly outweigh the former (Capron, 1978 and 1989; Beauchamp, 1989). In respecting the well-being of human subjects, the researcher is obliged to conduct only experiments that have been designed to maximise possible benefits and minimise possible harms. Capron (1989) points out that almost any intervention risks generating some harm compared to the absence of the experiment. Nevertheless, a person's willingness to be a subject is not an adequate sanction for performing an experiment if the investigator could reasonably have reduced the experiment's risks. During the development of the present thesis, several highly esteemed thermal physiologists were contacted for their opinions regarding the acceptability of the proposed methodology. This, in its initial form, involved the performance of both maximal and submaximal exercise following a reduction in core temperature with CWI to about 35°C. The range in responses, while not quite contradictory, certainly posed some difficulty with regard to suggested criteria acceptable for experiments of this nature.

The diversity in the opinions forwarded is reflective on one level of the variability in methodological approach to the broad area of cold stress, such that different combinations of modifying factors, as outlined earlier, may have led to different conclusions being drawn. The attendant confusion is exacerbated

by the general lack of uniformity evident in defining various stages of hypothermia and the physiological and psychophysical significance attached to each. For example, deep body temperatures of 35°C and as low as 33°C have been termed "mildly hypothermic" (Hayward, pers. comm.; Giesbrecht *et al.*, 1987), whereas a core temperature of 34°C was considered by Ferrara *et al.* (1990) to be "severely hypothermic". A central tension at the outset of research design involving human experimentation is the obligation on the part of the investigator to use methods which permit the strongest possible conclusions to be drawn from the data, while at the same time exposing people to the minimum risk. Given this scenario, dissimilarities in ones' philosophical standpoint relative to the available ethical choices in any situation may account for some of the disparity with regard to what is and what is not considered an acceptable experimental intervention. Thus Veatch (1989) remarks that different ethical positions will lead to different conclusions about what is morally appropriate in certain circumstances.

Cardiac Irregularities During Cold Exposure

The gravest hazard of hypothermia, as reviewed in the first chapter, is considered to be ventricular fibrillation (Keatinge, 1969; Maclean and Emslie-Smith, 1977), although there is no general agreement about the exact mechanism responsible for its occurrence. The electrical threshold, the conduction velocity and the refractory period of the myocardium do not appear to be causally related to the onset of ventricular fibrillation (Maclean and Emslie-Smith, 1977), suggesting that it is not

brought about by the direct effects of cold on the cardiac muscle itself (Keatinge, 1969). Keatinge has suggested that it results from an insufficiency of the supply of metabolic energy to the cardiac muscle. This is due primarily to a reduction in the supply of oxygen to the myocardium as a consequence of hindered oxygen dissociation from haemoglobin with cooling (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Sarnaik and Vohra, 1986; Coetzee and Swanepoel, 1990). Whatever the precise cause, ventricular fibrillation is considered a major risk during deep hypothermia, when core temperatures drop to 30°C and below (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Hong, 1984; Sarnaik and Vohra, 1986); above such temperatures the likelihood of its occurrence is remote (Keatinge, 1969; Maclean and Emslie-Smith, 1977).

The possibility of cardiac irregularities other than ventricular fibrillation does exist with marked reductions in core temperature. A core temperature of 32°C to 33°C and below is commonly quoted as that point at which cardiac arrhythmias may increasingly be encountered (Keatinge, 1969; Maclean and Emslie-Smith 1977; Hong, 1984), although this does not preclude the incidence of cardiac irregularities at higher core temperatures. Keatinge (pers. comm.) has observed atrial fibrillation at a core temperature of 34.8°C. Unlike ventricular fibrillation, atrial fibrillation interferes only slightly with the function of the heart. Atrial dysrhythmias are especially common at body temperatures below 29.5°C, but are usually benign and transient (Maclean and Emslie-Smith, 1977). Sudden exposure to cold,

especially in water, is also known to increase the risk of cardiac arrhythmias (Keatinge, 1966; Hong, 1984; Fritz and Perrin, 1989). The initial reflex cardiorespiratory responses, particularly to CWI, have been assigned the collective term "cold shock" (Tipton and Golden, 1987). With respect to the circulatory system, sudden CWI is associated with intense vasoconstriction, with concomitant increases in venous and arterial pressures, and increases in cardiac output and heart rate (Maclean and Emslie-Smith, 1977; Tipton and Golden , 1987). Such responses pose a particular threat to those with underlying cardiovascular disease. Coupled with this is an intense gasp reflex lasting approximately one to two minutes (Keatinge, 1969; Cooper *et al.*, 1976; Maclean and Emslie-Smith, 1977; Tipton and Golden, 1987; Fritz and Perrin, 1989), and a transient, marked increase in metabolic rate for approximately the first five minutes, a period which Hayward *et al.* (1977) termed the phase of "dynamic sensitivity". Hong (1984) has remarked that although a sense of difficulty in breathing is often encountered along with the gasp reflex, as much as 50-60l.min^{-1} of air may be moved in and out of the lungs. The rapidity of the onset of the hyperventilation produced by cooling the skin led Keatinge (1969) to propose that it was a reflex initiated from cold receptors in the skin. Whether this alone accounts for the response is still a matter of some debate, however, since receptors sensitive to an increase in muscle tension with cold immersion have also been implicated (Cooper *et al.*, 1976; Tipton and Golden, 1987). Furthermore, Cooper and co-workers have suggested that higher centres may influence the magnitude of the response. They

commented that high anxiety levels may exaggerate the ventilatory response to skin cooling. If the hyperventilation induced by sudden CWI is severe enough, a reduction in arterial PCO_2 may result in respiratory alkalosis that, in some cases, may lead to tetanic spasms and possible loss of consciousness (Cooper *et al.*, 1976; Maclean and Emslie-Smith, 1977; Hong, 1984).

Effects of Cold Upon Respiratory Responses

The effects of cold stress upon respiratory responses has received relatively limited study (Maclean and Emslie-Smith, 1977; Muza *et al.*, 1988). Following the initial hyperventilatory response to sudden cold exposure, minute ventilation generally increases progressively in response to the increasing metabolic demands of the cold stress (Muza *et al.*, 1988). The raised minute ventilation is a function of increases in both tidal volume and breathing frequency, with the former playing a relatively greater role (Cooper *et al.*, 1976; Muza *et al.*, 1988). In a study of pulmonary function following exercise at cold ambient temperatures (-11°C) Chapman *et al.* (1990) found that changes in lung volumes under cold ambient conditions were similar to those seen following warm exercise. According to Muza and colleagues, once the initial ventilatory response to cold exposure is over, temperature receptors in the skin and hypothalamus play only an indirect role in the control of ventilation. Rather, these inputs stimulate metabolic and circulatory adjustments in an attempt to maintain thermoregulatory homeostasis; the ventilatory response is then governed by metabolic requirements. As core temperature falls

to about 30°C and below with prolonged cold exposure, respiratory depression occurs (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Fritz and Perrin, 1989), the extent of which is proportional to the fall in body temperature and the decreased rate of metabolism (Maclean and Emslie-Smith, 1977).

Respiratory depression during deep hypothermia, with core temperatures below 30°C, may result in significant disturbances of the body's acid-base equilibrium. According to Sarnaik and Vohra (1986), each 1°C decrease from a reference temperature of 37°C will increase pH by 0.015. Acidosis may result from carbon dioxide retention, but at other times shivering and an increased production of lactic acid, major vessel occlusion and compromised hepatic metabolism of organic acids may each cause metabolic acidosis during hypothermia (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Sarnaik and Vohra, 1986).

Effects of Cold Upon Metabolism

Initial exposure to cold, particularly in water, causes a striking increase in the metabolic rate, which is primarily determined by an increase in muscle tension caused by cold shock (Maclean and Emslie-Smith, 1977; Tipton and Golden, 1987). Cold stress is known to be a powerful stimulus for increased sympathetic nervous activity, reflected in increases in the levels of circulating catecholamines (Johnson *et al.*, 1977; Mitchell *et al.*, 1990). According to Johnson *et al.* (1977), the responses of both the sympathetic nervous system and metabolic rate are more sensitive to alterations in skin temperature that

arise from exposure to sudden cold or warm environments than the slower changes in core temperature. Thus Tipton and Golden (1987) differentiate between "cold shock" and "thermoregulatory" $\dot{V}O_2$ responses, with the latter determined by inputs from the whole body. Following the initial transitory responses, Johnson and co-workers found a close correlation between plasma norepinephrine concentrations and metabolic rate. Although they caution that this does not necessarily imply a causal relationship, they suggest that norepinephrine may facilitate shivering thermogenesis. The $\dot{V}O_2$ during cold stress is primarily determined by the absolute change, as well as the rate of change, of skin and core temperatures (Johnson *et al.*, 1977; McArdle *et al.*, 1984; Tipton and Golden, 1987). Other than the immediate transient increase in $\dot{V}O_2$ with cold exposure, the maximum sustained $\dot{V}O_2$ during cold stress, both in air and water, would appear to be in the region of two to three times the resting non-shivering rate (Merrill-Smith and Hanna, 1975). These authors, however, noted greater variability in the metabolic response to cold air than to cold water.

Below a core temperature of approximately 33°C the shivering response gives way to persistent muscle rigidity. Once shivering is prevented, tissue oxygen consumption and metabolic rate are progressively reduced as the body temperature falls, such that at a core temperature of 28°C, metabolic rate is reduced by 50% (Maclean and Emslie-Smith, 1977; Hayward *et al.*, 1984; Sarnaik and Vohra, 1986; Willford *et al.*, 1990).

One of the consequences of elevated catecholamine secretion during cold stress is an increase in plasma free fatty acid levels (Thompson, 1977; Maclean and Emslie-Smith, 1977; Sloan, 1979; Timmons *et al.*, 1985; Doubt and Hsieh, 1991; Tipton *et al.*, 1997). According to Maclean and Emslie-Smith, this instantaneously activated mobilisation of triglyceride from adipose tissue provides fuel for shivering thermogenesis induced by cold, but normally decreases after about eight hours of cold exposure, unless hypothermia supervenes. During acute cold exposure, concentrations of blood glucose usually increase (Keatinge, 1969; Thompson, 1977; Maclean and Emslie-Smith, 1977). According to Thompson, the rate at which glucose is oxidised in the body also increases, more or less in proportion to the increase in total body metabolism. Maclean and Emslie-Smith, however, comment that, following an increase in the rate of breakdown of hepatic glycogen during the initial stages of exposure a decrease in peripheral utilisation of glucose as lower temperatures are reached, results in a condition of hyperglycaemia.

Effects of Cold Upon Mental Function

It has long been known that cerebration may be impaired during cold exposure, depending on the extent of body cooling. Indeed, Keatinge (1969) has commented that it is surprising that the cerebral activity responsible for consciousness is not more readily disrupted by hypothermia than it is. Loss of consciousness is most often observed when the core temperature is 32°C or less (Hong, 1984; Fritz and Perrin, 1989), but

occasional reports exist of conscious individuals being able to respond to questions when core temperature was as low as 27°C to 28°C (Keatinge, 1969; Maclean and Emslie-Smith, 1977; Coleshaw *et al.*, 1983). The extent of body cooling required to elicit noticeable changes in mental function is not entirely agreed upon amongst researchers in this area. Such differences of opinion are, however, made more understandable when one appreciates the lack of uniformity evident with regard to the definition of various stages of body cooling and in methodological approach to this area of study, as discussed earlier. According to Maclean and Emslie-Smith, an individual with a core temperature above 34°C usually presents as alert, well orientated and cooperative, and it is only at temperatures below this that drowsiness and difficulty in understanding increase and responses become slower. In contrast, others have stated that comparatively small depressions of body temperature might produce significant impairments of mental function (Coleshaw *et al.*, 1983; Askew, 1989; Bridgman, 1990; Giesbrecht *et al.*, 1993). Askew (1989) stated that as the body cools below the normal core temperature of approximately 37°C neural co-ordination deteriorates and confusion and lethargy increase. Coleshaw *et al.* (1983) similarly cited research which found impairments of cognitive processing in subjects who experienced mean decreases in rectal temperature of 0.7°C and 1.1°C. In such circumstances the distracting effects of low cutaneous temperature may account for alterations in mental function (Coleshaw *et al.*, 1983; Bridgman, 1990), as may the effects of cold upon the neuromuscular aspects of speech (required for responses to some of the tasks)

(Giesbrecht *et al.*, 1993). Coleshaw and colleagues have demonstrated, however, that the relative importance of peripheral and central cooling in this context is as yet unclear. When the skin temperatures of their subjects were raised during rewarming so that they felt comfortable, but their core temperatures remained low, certain aspects of cognitive function were found to be significantly impaired. In their study memory registration was impaired as core temperature fell from 36.7°C, but memory recall was not affected even at body temperatures of 34.6°C. These authors suggested that such mental impairment as was evident was caused directly by the low temperature of the brain. Amnesia for events that occur while the body temperature is low is a commonly reported finding, usually at a core temperature below 34°C (Keatinge, 1969; Hong, 1984; Fritz and Perrin, 1989).

Effects of Cold Upon Local Tissue Function

The direct effects of cold upon peripheral and central tissues can cause similar temporary interference in movement ability, a factor which is likely to be of direct consequence for subsequent exercise performance. Exercise performance may be detrimentally affected when muscle temperatures are lower than optimal for activity of oxidative enzymes, for excitability of nerves and muscle and for nerve conduction (Patton and Vogel, 1984; Shiojiri *et al.*, 1997). Keatinge (1969) noted that steady voluntary contraction of the forearm muscles could be maintained for longer after local cooling of the forearm in 18°C water, an observation he attributed to a cold-induced slowing of the rate of relaxation

of actomyosin. Maximum tension generated during sustained voluntary contraction of cooled forearm muscles is, however, significantly decreased at muscle temperatures below 27°C (Clarke *et al.*, 1958). A reduction in grip strength with cooling (Cooper *et al.*, 1976; Horvath, 1981; Choi *et al.*, 1988) was ascribed by Cooper *et al.* to a reduction in ulnar nerve conduction velocity and delayed response to direct muscle stimulation. These authors commented that muscle cooling was thus likely to "reduce the effectiveness of muscular effort". Giesbrecht and co-workers (1995) observed significant decrements in the performance of fine and gross motor tests following isolated arm cooling to a biceps temperature of 18.1°C. This, they concluded, was due almost entirely to the local effects of arm tissue cooling. According to McCormick and Sanders (1983), the performance of manual tasks is directly related to both the temperature of the hand and the rate at which it is cooled. Furthermore, the hand temperature at which performance impairment occurs may differ from one task to another. Cooling of the leg muscles to 28.4°C in a separate study decreased peak power output during cycle exercise by 32% (Davies and Young, 1983).

Sargeant (1983), reporting on the effect of muscle temperature on short term dynamic exercise lasting 20s or less, found decreases in force and power and suggested that the magnitude of the temperature effects were velocity dependent. According to Keatinge (1969), "moderate" cooling of muscle slows the rate at which acetylcholine is broken down after being released by the motor nerve terminal and the rate at which the motor endplate of

the muscle can repolarise after depolarisation, a situation which favours cramp and muscle stiffness. Shephard (pers. comm.) has cautioned that a loss of flexibility with cooling might increase the risk of muscle tears during subsequent activity. At core temperatures of 35°C and below, voluntary movements are considerably slowed, and muscular incoordination is clearly evident (Maclean and Emslie-Smith, 1977; Fritz and Perrin, 1989); Maclean and Emslie-Smith state that the gait becomes ataxic between 32°C and 34°C. In reviewing the related research, Horvath (1981) noted that cooling of peripheral and motor sensory nerves appeared to lead eventually to a dysfunction equivalent to peripheral paralysis at local temperatures of 8°C to 10°C. Others have reported that, during severe hypothermia, when core temperatures drop to about 27°C, complete sensory and motor paralysis occurs (Keatinge, 1969; Maclean and Emslie-Smith, 1977).

EFFECTS OF COLD UPON EXERCISE PERFORMANCE

Within the broad scope of thermoregulation, the effects of cold upon exercise performance have received relatively little attention. Graham (1988) has rightly pointed out that many reviews in the area of thermal physiology concentrate almost exclusively on heat stress (for example: Rowell, 1974; Gisolfi, 1983; Nadel, 1983b and 1985; Fortney and Vroman, 1985; Kenny and Hodgson, 1987). The relatively limited data which exist pertaining to cold exposure and performance, the inadequate regulation in some cases of factors known to modify thermogenic responses and the somewhat equivocal findings that have been

reported suggest that many unsolved problems remain. On the basis of the above, a call has been made by eminent researchers for increased participation in the general area of physiological adjustments to cold stress (Horvath, 1981; Graham, 1988; Pendergast, 1988; Doubt, 1991; Sjödin *et al.*, 1996). The present review is intended as an adjunct to those symptoms of cold exposure already discussed in the previous section.

According to Horvath (1981), the evaluation of cold effects upon exercise performance has involved three major stress conditions: 1. long-term exposure to a cold environment, 2. acute exposure to ambient cold, and 3. reduction of body temperature to near-critical hypothermic levels with activity measured at room temperatures. Commenting on the latter practice, Horvath stated, however, that the responses of subjects under such conditions, reflective as they are of marked alterations in core temperature, are of importance in rather stringent conditions and do not represent effects observed in acute and short-term cold exposure. Added to the above in terms of the variability of methodological approach is the fact that both air and water exposures have been used to induce cold stress; as Graham (1988) and Doubt (1991) have pointed out, responses to cold air and water are not merely quantitatively different. Physiological adjustments that occur with cooling are dependent on body (core and skin) temperatures. Since these in turn are influenced by several factors, not least amongst which are the nature and duration of the cold exposure, comparison between studies is difficult (Rowell, 1974; Pendergast, 1988; Doubt, 1991; Sjödin

et al., 1996).

Within the framework of the relatively limited number of studies that exist, contradictory data, depending on those factors outlined above together with the intensity of the exercise performed, are offered to describe the effects of cold upon submaximal exercise. On the basis of a comparison of exercise performed during cold air exposures at 20°C and -20°C, Patton and Vogel (1984) concluded that, although submaximal endurance time was markedly lower in the latter condition, aerobic capacity was not significantly altered by cold exposure. The mean intensity of the exercise performed, however, was approximately 78% $\dot{V}O_2$ max. In another study endurance time at 80% of peak $\dot{V}O_2$ was increased by 12% (Olschewski and Brück, 1988). Several additional studies have indicated that the intensity of the exercise performed is of importance in determining cooling effects upon oxygen cost. In general it would appear that, for exercise performed during cold exposure, the greatest calorigenic effect of a reduction in body temperature is observed at light to moderate intensities; the $\dot{V}O_2$ gradient between normal exercise and that after cooling is reduced or not apparent at higher intensities (Strõmme *et al.*, 1963; Pugh, 1967; McArdle *et al.*, 1976; Timmons *et al.*, 1985; Pendergast, 1988; Doubt and Hsieh, 1991). However, the intensity at which the difference in oxygen cost is no longer evident has yet to be agreed upon. Pendergast (1988) proposed that this gradient in $\dot{V}O_2$ was not affected by workloads up to approximately 80% $\dot{V}O_2$ max. This would explain the insignificant effect of cooling upon aerobic

capacity found by Patton and Vogel at 78% $\dot{V}O_2$ max. These authors pointed out that such intensities were well above the approximate 1.5l.min^{-1} cut-off level of the shivering response during exercise. A critical point, where no differences in the $\dot{V}O_2$ required to perform the same task in 5°C and 26°C to 28°C environments, was identified by Strõmme *et al.* (1963) as around 1.2 to 1.4l.min^{-1}. Clearly the energy expenditure at a given intensity is influenced by the amount of associated shivering; when heat input equals or exceeds heat loss the incidental influence of cold-induced shivering can be counteracted (Horvath, 1981). It is worth noting, however, that Pugh (1967) reported an increase in the metabolic cost of exercise at 5°C that was evident at exercise levels which required up to 2.1l.min^{-1} in a comfortable environment. Furthermore, in contrast to Strõmme and colleagues, Pugh found no difference in the oxygen requirements for exercise when subjects worked in dry clothing with no air movement. The differences that he recorded were for subjects in wet clothing exposed to wind. The point has already been made, however, that considerable variability in the outcome of one study to the next can be expected, depending on the uniformity, or lack thereof, of a host of modifying factors. An increase in $\dot{V}O_2$ is a result of a decrease in both core and skin temperatures, as well as the physical and physiological characteristics of the subjects (Pendergast, 1988). Horvath (1981) reported that in a different type of experiment, where exercise was performed after, rather than during cold exposure, increases in the metabolic cost of exercise were found at levels that, without cooling, had required an oxygen uptake of

$2.0 l.min^{-1}$.

According to Pendergast (1988), the absolute $\dot{V}O_2$ gradient between cold and warm exercise (specifically in water) increases with the degree of body cooling. This author stated that for a 0.5°C to 1.5°C decrease in core temperature, the energy cost of exercise is increased by 10 to 40%. For the same drop in core temperature, the capability to supply oxygen to meet the increased demand is also reduced by 10 to 40%. Pendergast proposed as further evidence for the effects of cold the appearance of lactic acid in the blood at lower intensities, the faster rate of lactic acid accumulation and the fact that peak lactic acid is lower exercising in the cold than under normal conditions. At this point it should be noted that a portion of the increase in lactate with cold has been ascribed to factors other than increased anaerobic production (Doubt and Hsieh, 1991), with changes in diffusion through tissue proposed as a possible explanation. The extent to which oxygen availability is affected as the body cools is the subject of some debate, and, as with much discussion in the area of cold stress, is complicated by the diversity of protocols employed. Circulatory responses to exercise in the cold, an obvious influence on oxygen transport, differ depending on the nature and the extent of the cold stress. Commenting on the difficulty evident in interpreting those data that exist, Pendergast (1988) pointed out that cardiac output, for example, is influenced by the cardiac preload and afterload, venous return, heart rate and metabolic drive, all variables that may be influenced in turn by factors

inherent in the methodology adopted. Most of the reports dealing with the effects of cold stress upon cardiac output during exercise pertain to exercise performed during CWI, the details of which will be discussed later in this section. In relation to cold air exposure, Hanna *et al.* (1975) stated that during submaximal exercise at 5.5°C compared to 22.5°C, stroke volume and cardiac output were higher at a given $\dot{V}O_2$. Horvath (1981), however, has suggested that their data do not actually demonstrate a significant increase in cardiac output.

Heart rate responses to exercise with body cooling are equally controversial. A reduction in heart rate during exercise in a cold water environment has been reported (McArdle *et al.*, 1976; Maw *et al.*, 1993). McArdle *et al.* proposed that the bradycardia they observed with exercise in 18°C water compared to 33°C water was a temperature effect rather than a water effect, since heart rates at a given $\dot{V}O_2$ were similar during work in air and 33°C water. Horvath (1981) cites a study where a 10 min shower prior to submaximal exercise produced significantly lower heart rates. Precooling by CWI (to the extent that core temperature was reduced by 0.7°C) produced lower heart rates for the first 10min of a 30min running test in an experiment by Booth *et al.* (1997). Others, however, have found exercising heart rates to be unaltered during both cold air and cold water exposures (Patton and Vogel, 1984; Mitchell *et al.*, 1990; Doubt and Hsieh, 1991; Sjödin *et al.*, 1996) and during exercise in various ambient conditions preceded by cold stress (Davies *et al.*, 1975; Bergh, 1980; Hessemer *et al.*, 1984; Giesbrecht *et al.*, 1987; Kruk

et al., 1990).

Timmons *et al.* (1985) have commented that, whereas at lower intensities cold and exercise appear to be additive rather than synergistic in increasing total energy utilisation, at high intensities, exercise substitutes for the thermogenic stimulation of cold rather than adding to this effect. Nevertheless, evidence exists to suggest that even at high intensities cold exerts a significant affect on performance. Patton and Vogel (1984) observed that although aerobic capacity was not altered by cold exposure, submaximal endurance performance (at approximately 78% $\dot{V}O_2$ max) was. Speculating on the mechanisms responsible for the decrement seen in exercise performance, these authors proposed the direct effect of cold on the muscle itself as a possible factor. Horvath (1981) has pointed out, however, that confusion persists as to the exact effects of cooling on muscle strength and/or endurance. Reports do exist of improved endurance following precooling manoeuvres (Hessemer *et al.*, 1984; Lee and Haymes, 1995; Booth *et al.*, 1997). Once again, the tremendous scope of this area of research must be stressed. The effect of muscle temperature upon performance, for example, is dependent upon the intensity and duration of the exercise and the extent of the deviation of muscle temperatures from the norm. During exercise of a short duration in a temperate environment, increased muscle temperatures have been reported to improve performance and decreased temperatures to impair it (Bergh and Ekblom, 1979). On the other hand, observations have been made of a faster rate of fatigue and shorter endurance times with

increased muscle temperature (Horvath, 1981; Kozlowski *et al.*, 1985; Sargeant, 1983). Thus the relative importance of muscle temperature, viscosity changes in connective tissue, circulatory changes, local metabolic activity and even psychological factors remains to be elucidated. As reviewed earlier, cooling of the muscle is known to reduce maximum tension (Clarke *et al.*, 1958; Cooper *et al.*, 1976, Horvath, 1981; Choi *et al.*, 1988; Doubt, 1991) and power output (Davies and Young, 1983; Sargeant, 1983) via a variety of proposed mechanisms. According to Swenson *et al.* (1996), nerve conduction is continually slowed down by decreasing temperature, with conductivity failure evident in peripheral nerve fibres when the temperature is less than 27°C. Cooper and colleagues concluded that cold stress would reduce the "effectiveness" of muscular effort, whilst others have stated more specifically that exercise in the cold is likely to result in impaired mechanical efficiency compared to exercise under normal conditions (McArdle *et al.*, 1976; Pendergast, 1988; Askew, 1989). Doubt and Hsieh (1991) suggested as a contributory factor in reported decreases in mechanical efficiency an increase in the EMG activity of cooled muscles even in the absence of shivering. Their observation was that "increases in EMG activity in muscles of an active limb might increase the resistance to limb movement, thereby raising the oxygen cost for a fixed external workload and decreasing mechanical efficiency". It has been speculated that higher rates of lactate production and shorter endurance times noted in exercise in the cold may be a function of a greater recruitment of fast twitch muscle fibres in significantly cooled muscle (Doubt, 1991).

Effects of Precooling Upon Exercise Performance

The contention that surrounds studies investigating the implications of performance during cold stress extends to those which have examined performance in various environments following cold exposure. Horvath (1981) remarked that contradictory data, implying both detrimental and beneficial effects of prior cold exposure upon performance, have been published, and suggested that part of the problem may lie in the criteria employed to determine improved or worsened performance. The issue is complicated by varied results according to whether the whole body or local sites only are cooled and the extent of the cold stress imposed on the subject. Cold showers prior to a fixed submaximal exercise task, for example, resulted in significantly reduced heart rates, although the $\dot{V}O_2$ was higher following cold exposure (Horvath, 1981). The so-called beneficial effect was only evident for the first 10 to 20min. The use of a water-cooled calorimeter suit at 10 and 15°C by Sjödin and colleagues (1996) to reduce skin temperatures without a reduction in core temperature, much along the lines of the peripheral cooling condition in the present study, similarly produced a higher energy expenditure at the lower temperature, but had no effect upon heart rate or respiratory quotient. These authors nevertheless suggested that some metabolic regulator may be triggered peripherally, possibly in skin and/or muscle. Booth *et al.* (1997) concluded that water immersion whole body precooling increased endurance in hot, humid conditions with an enhanced rate of heat storage and decreased thermoregulatory strain. In their experiment T_{re} and T_{sk} decreased by 0.7°C and by

5.9°C respectively. The $\dot{V}O_2$ was not changed despite their subjects running further in the prescribed 30min. Heart rates at 5 and 10min into exercise were lower following cooling. Similar findings were reported by Lee and Haymes (1995), whose subjects T_{re} was dropped by 0.37°C by cold air exposure. Exercise duration (82% $\dot{V}O_2$ max to exhaustion) was longer, $\dot{V}O_2$ was lower initially and heart rate and blood lactate were unchanged. Hessemer *et al.* (1984), on the other hand, reported that precooling increased $\dot{V}O_2$ by 9.6% over 60min of cycle ergometer exercise, where subjects exercised "as hard as possible". Once again, however, heart rate and blood lactate were unchanged. Cooling of the lower extremities in water at 15°C between repeated heavy bouts of leg ergometry was reported to increase the number of exercise bouts that could be tolerated compared to without cooling (Horvath, 1981).

Rowell (1974) has commented on the differences in the circulatory responses of normothermic *versus* hyperthermic subjects exposed to cold showers. In contrast to the rapid increases in cardiac output and mean arterial pressure observed in normothermic subjects, a rapid decrease in skin temperature of 2°C produced fast decreases in heart rate and mean arterial pressure and sudden increments in central blood volume and stroke volume in hyperthermic subjects. On the basis of the attenuation of some cardiovascular responses, Rowell advocated the use of cooling devices for subjects performing in heat stress. It is worth noting, however, that some have reported no beneficial effects of cooling previously heated exercising subjects (Sherman and

Deutsch, 1983; Bassett *et al.*, 1987), whilst others have observed differing responses between progressive prolonged exercise and progressive maximal exercise (Quirion *et al.*, 1990).

Some experimental studies exist to suggest that performance is significantly impaired by cold exposure prior to performance in a temperate environment. Impaired oxygen delivery with body cooling was implicated in a study which observed a decrease in peak $\dot{V}O_2$ during maximal effort in the order of 5% to 6% for every 1°C drop in core temperature following a swim in 19°C water (Bergh, 1980). Compared to under normothermic conditions, the VO_2 max was reduced when core temperature dropped below 37.5°C. The duration of the exercise was also reduced following cooling. Kruk *et al.* (1990) reported that whole body precooling significantly raised $\dot{V}O_2$ for submaximal exercise (50% $\dot{V}O_2$ max) at an ambient temperature of 5°C. Similarly Davies *et al.* (1975) reported an increase in the energy cost of submaximal activity at room temperature following a drop in core temperature to 35°C.

The implications of body cooling of varying degrees followed by performance in a warmer environment have direct application to the triathlon, which includes a swimming, a cycling and a running event. Fast growing in popularity since its advent in Hawaii in 1978 (Kohrt *et al.*, 1987), the triathlon and those who participate in the sport have received relatively limited attention (O,Toole *et al.*, 1987; Kreider *et al.*, 1988). With specific reference to thermal responses to triathlon performance, Kreider and co-workers (1988) reported that during a simulated

short triathlon (i.e., 0.8km swim, 40km cycle and 10km run) the swim in 23°C water elicited a 0.72°C increase in core temperature. These authors went on to discuss the importance of a cumulative rise in core temperature over the three events which was observed to begin during the swimming session. Once again, however, cognisance must be taken of the possible variation in responses over longer swimming distances, in colder water. Concern has been expressed that the increased number of casual participants together with greater interest shown in sea swims at temperatures below 20°C may see more hypothermia-related incidents (Yach, 1990; Chatard and Millet, 1996). In a study of a 3.2km swim in 16°C water, Yach reported that 24.7% of participants failed to complete the swim and had to be removed from the water. Although body temperatures were not recorded, both unsuccessful and successful swimmers reported a high incidence of muscle cramps, disorientation and headaches and many experienced difficulty walking and talking. While the consequences of such effects for subsequent cycling and running have yet to be clarified, several researchers have stressed the importance of economy as a determinant of successful competition in triathlons (Dengel *et al.*, 1986; Kreider *et al.*, 1988). The possibility of impaired economy due to body cooling may thus be of some consequence for this specific sport.

Effects of Cold Upon $\dot{V}O_2$ Max: Peripheral versus Central Limitations

There is some evidence to suggest that maximal aerobic capacity may be compromised with reductions in body temperature. This

has implications for submaximal exercise, since in the face of a reduction in $\dot{V}O_2$ max, together with the possibility of an increase in the absolute energy cost of a given task, the relative metabolic load when accompanied or preceded by cold stress would be higher. According to the work of Bergh (1980), as mentioned earlier, maximum $\dot{V}O_2$ is significantly reduced with cold stress, the extent of the reduction dependent upon the magnitude to which core temperature is lowered. In summarising some of the studies that have investigated the question of maximal exercise in the cold, Horvath (1981) reported in contrast that no differences in aerobic power have been found during acute exposure to temperatures ranging from 35°C to -5°C. Extending the temperature at which this has been studied to apparently the lowest yet reported, Patton and Vogel (1984) similarly found that $\dot{V}O_2$ max was not altered after 2 to 2.5h of exposure to -20°C. It is significant to note, however, that prior to the test, subjects in this study wore cold-weather clothing and experienced no reduction in core temperature. In conjunction with the findings of Bergh, this suggests that, provided adequate protection is allowed to prevent a reduction in body temperature, the performance of a maximal test in a cold air environment is not of itself sufficient to alter $\dot{V}O_2$ max. Indeed Askew (1989) has commented that an adequately clothed person is essentially unexposed to cold because of the microclimate beneath the clothing, although there is a small energy increment for replacing insensible heat loss. The importance of core temperature was implied by Rodahl *et al.* (1962) who observed that, in subjects subsisting on a low protein diet, $\dot{V}O_2$ max was

reduced after 9 days of exposure to 8°C compared to 22°C, as were blood lactate levels. Maximum heart rates were not different between conditions. Subjects on a normal diet, however, demonstrated no change in $\dot{V}O_2$ max, maximum heart rate or blood lactate between conditions. Although the importance of nutrition with regard to cold stress is acknowledged (Askew, 1989), Horvath (1981) has pointed out that the exact role played by the diets of these subjects in influencing their responses awaits further investigation.

The ability to sustain exercise and eventually to attain $\dot{V}O_2$ max under any conditions is dependent on the uptake, delivery and utilisation of oxygen (Rowell, 1974; Shephard, 1984; Pendergast, 1988). The cause of the limit in $\dot{V}O_2$ has thus been sought in limitations of pulmonary gas exchange, maximal blood flow and metabolic capacity of working muscle (Rowell, 1974). Continuance of the debate within the context of the effects of body cooling upon $\dot{V}O_2$ max is of considerable interest, but complex, due to the plethora of equivocal findings.

It would appear that, in normal subjects not exposed to cold stress, pulmonary factors impose no limitations on oxygen transport (Rowell, 1974; Cerretelli, 1984; Shephard, 1984). Whether this is so when exposed to cold stress deserves further study. Pendergast (1988) has suggested that, even where ventilation appears normal, cold-induced changes in arterial and venous oxygen content may pose a problem. It has been suggested that the leftward shift of the oxyhemoglobin curve during

hypothermia may be detrimental to oxygen delivery (Coetzee and Swanepoel, 1990). The extent of the cooling required to produce this effect, however, is not clear. Furthermore, it has been speculated in the past that, in the face of observed reductions in cardiac output during cold stress, the $\dot{V}O_2$ during hypothermic conditions may be met by an increased arterio-venous oxygen difference (McArdle *et al.*, 1976; Pendergast, 1988), although Kaijser (1970) reported a narrowing of the maximum arterio-venous oxygen difference after muscle cooling. Certainly during CWI the possibility of a ventilatory limitation to $\dot{V}O_2$ max is plausible. In addition to the effects of raised central blood volume upon static and dynamic lung volumes, there is an increased load on the respiratory muscles, such that resting respiratory work during head-out immersion may be elevated by about 60% over that at rest in air (Lanphier and Camporesi, 1984; Lin, 1988). A lower pulmonary ventilation has been reported (Magel and Faulkner, 1967; Dressendorfer *et al.*, 1976; Coetsee and Thiart, 1978; Holmér, 1978), although contrasting findings do exist where graded leg exercise on land and underwater at a depth of 1.22m yielded similar minute volumes (Moore *et al.*, 1970). It has been proposed, however, that the opposing effects of hydrostatic pressure and increased intrathoracic blood volume upon gas exchange may result in sufficient oxygenation of arterial blood during immersion. Furthermore, the interaction of a lower minute ventilation and a higher oxygen extraction may result in $\dot{V}O_2$ values during immersion that are equivalent to those obtained on land (Magel and Faulkner, 1967; Dressendorfer *et al.*, 1976; Holmér, 1978; Lin, 1988).

The consequences of body cooling for tissue metabolism, a peripheral limitation to $\dot{V}O_2$ max, are unclear. Several authors have stated that, under normal thermal conditions, the ability of tissue enzyme systems to utilise oxygen is not likely to limit maximal aerobic capacity (Rowell, 1974; Shephard, 1984; Aunola *et al.*, 1988). One researcher who has argued strongly for a tissue enzymic limitation to $\dot{V}O_2$ max observed that cooling of active muscles leads to a reduction in the maximal arterio-venous oxygen difference, which he attributed to a reduction in the activity of peripheral enzyme systems (Kaijser, 1970). In addition, it is well known that chemical reactions slow as a consequence of reduced temperature (Shephard, 1985; Pendergast, 1988; Askew, 1989). According to Swenson and colleagues (1996), metabolic enzymic activity is decreased by approximately 50% when the temperature is lowered by 10°C. Although Pendergast has remarked that, even during cooling experiments where the core temperature may fall to 36°C, this is unlikely to interfere with metabolism to any significant degree, it is clear that more work is required to clarify the importance of reduced temperatures for the oxidative capacity of exercising muscles. In refuting Kaijser's claims, Shephard (1984) stated that such peripheral limitations as may exist probably relate more to difficulty in perfusing the working muscle than to enzymic limitations. In examining the kinetics of $\dot{V}O_2$ during maximal exercise at different muscle temperatures Ferretti *et al.* (1995) observed that, after cooling the thigh muscles by CWI, $\dot{V}O_2$ max and maximal power output were lower and blood lactate accumulation was greater. This was attributed to an impaired muscle blood flow

and decreased muscle oxygen unloading as well as a temperature-dependent decrease of the velocity constants of oxidative reactions. The same factors were cited by Shiojiri *et al.* (1997) in explaining a significantly depressed increase in $\dot{V}O_2$ at the start of moderate exercise following CWI.

According to Shephard (1984), the primary limitation to maximal aerobic capacity under normal conditions is central circulatory failure. The impairment in maximal aerobic capacity accompanying body cooling was similarly attributed to central factors by Horvath (1981), who, commenting on the research of Bergh (1980), proposed that the reduction in maximal heart rate evident with cooling led to impaired oxygen delivery. Although it was acknowledged by Pendergast (1988) that maximal heart rate drops 10 to 30b.min^{-1} when the core temperature is 0.5°C to 2.0°C lower than normal, he stated, however, that since neither heart rate nor cardiac output appeared to plateau as $\dot{V}O_2$ increased during cold stress, neither of these parameters appeared to limit $\dot{V}O_2$ at maximum. He proposed that muscle blood flow was the primary factor limiting maximal aerobic power. Indeed the importance of muscle blood flow as a determinant of $\dot{V}O_2$ max has been stressed by several scientists (Cerretelli, 1984; Cerretelli *et al.*, 1986; Aunola *et al.*, 1988; Pendergast, 1988), on the basis of observations of a plateau in muscle blood flow at approximately 60% to 80% $\dot{V}O_2$ max. According to Pendergast, the local metabolically mediated hyperaemia evident with exercise may be blunted during cold stress by a persistent vasoconstriction mediated by the sympathetic nervous system. Speculating on the

outcome of this for oxygen transport, this author proposed that the effect of body cooling could be to reduce muscle blood flow and result in a plateau of blood flow at levels lower than previously measured. This has been supported by more recent research (Ferretti *et al.*, 1995; Shiojiri *et al.*, 1997).

Exercise Responses During CWI

A review of the effects of cold upon performance would be incomplete without consideration of exercise responses during CWI as opposed to cold air exposures. It is known that immersion in water produces prominent changes in cardiorespiratory functions. According to Greenleaf (1984), overall immersion responses in the upright position are the result of two major alterations in this condition: firstly, the increased mechanical pressure on connective tissue, which is proportional to depth, and secondly, the compression force on the abdomen, which forces the diaphragm upward and compresses cardiorespiratory structures in the thorax.

Combination of these factors results in cephalad redistribution of the circulating blood, to the extent that central blood volume increases by approximately 700ml (Burki, 1976; Löllgen *et al.*, 1980; Lanphier and Camporesi, 1984; Greenleaf, 1984; Lin, 1988; Pendergast, 1988). The increase in heart volume has been estimated at approximately 100ml (Lange *et al.*, 1974), which may in turn exert additional pressure on respiratory structures (Greenleaf, 1984). Central vascular engorgement therefore has direct implications for both the cardiovascular and respiratory

systems.

Reports of the precise nature of the modifications evident during immersion, however, frequently appear to be in conflict. For example, cardiac output has been described to decrease (Nadel, 1984), to remain unchanged (McArdle *et al.*, 1976) or to increase (Denison *et al.*, 1972; Löllgen *et al.*, 1980; Lin, 1984 and 1988; Pendergast, 1988). Conflicting results also exist with regard to the effects of immersion upon heart rate (Denison *et al.*, 1972; McArdle *et al.*, 1976; Dressendorfer *et al.*, 1976; Coetsee and Thiart, 1978; Lin, 1984 and 1988; Greenleaf, 1984), pulmonary ventilation (Magel and Faulkner, 1967; Cooper *et al.*, 1976; McArdle *et al.*, 1976; Dressendorfer *et al.*, 1976; Coetsee and Thiart, 1978; Greenleaf, 1984), pulmonary compliance (Burki, 1976; Löllgen *et al.*, 1980; Buono, 1983) and diffusing capacity (Löllgen *et al.*, 1980; Lundgren and Pasche, 1984).

In general, the pattern of responses in submaximal $\dot{V}O_2$ during CWI appear to follow those observed during cold air exposures, with the elevation in the energy cost of light to moderate exercise becoming less apparent at heavier intensities (Moore *et al.*, 1970; McArdle *et al.*, 1976; Pendergast, 1988). McArdle *et al.* (1976) noted that the increase in $\dot{V}O_2$ which occurred during submaximal exercise in 18°C water appeared greater than could be accounted for by shivering thermogenesis alone, and discussed the possibility of impaired mechanical efficiency as a result of the thermal stress. Lower submaximal heart rates have been found during exercise in cool water compared to warm water

(Dressendorfer *et al.*, 1976; McArdle *et al.*, 1976; Pendergast, 1988), or the same exercise in air (Moore *et al.*, 1970). The consequences of a reduction in heart rate for the functional capacity of the cardiovascular system is a matter of some debate. Dressendorfer *et al.* (1976) discussed changes in cardiac output observed with decreases in water temperature in terms of decreases in heart rate in conjunction with a maintained stroke volume. McArdle and co-workers, on the other hand, found the cardiac output-oxygen consumption relationship to be similar during exercise in air and water at 18°C, 25°C and 33°C. The prominent adjustment to the decrease in heart rate in cooler water was an increase in stroke volume, which was most prominent in 18°C water. Lin (1988) and others (McArdle *et al.*, 1976) have suggested that altered pre-load is responsible for the increased stroke volume during immersion. Pendergast (1988) has commented, however, that due to vasoconstriction during CWI, an increase in cardiac after-load, as evidenced by an increase in diastolic pressure, could offset, at least in part, the effects of increased cardiac pre-load, such that decreases in heart rate may not be entirely compensated for by increases in stroke volume. Conclusions based upon available data are thus difficult to draw.

Although it has been stated that the immersion effect on the circulation diminishes as the water temperature decreases (Lundgren and Pasche, 1984), the bulk of the evidence suggests that modifications in cardiorespiratory functions during immersion are likely to be most pronounced in upright (or

sitting) subjects exercising maximally in cold water (Denison *et al.*, 1972; Greenleaf, 1984). The consequences of immersion for maximal oxygen consumption, however, are somewhat contradictory. In general, the $\dot{V}O_2$ max obtained during swimming tends to be lower than that obtained on a treadmill or cycle ergometer (Åstrand and Rodahl, 1986; Coetsee and Thiart, 1978), although highly trained elite swimmers may attain similar values swimming or running (Magel and Faulkner, 1967; Holmér, 1978). The importance of cold stress as a determinant of the $\dot{V}O_2$ max was illustrated by Nadel (1984), who reported that at temperatures of 18°C and 26°C subjects were only able to reach 85% and 92% of their maximal aerobic power in 33°C water. Dressendorfer and colleagues, on the other hand, noted that cycling exercise during head-out immersion yielded $\dot{V}O_2$ max values that were unchanged in 25°C, 30°C and 35°C water, although this was accompanied by significantly lower heart rates in 25°C and 30°C water. Compared to the same exercise performed on land, the $\dot{V}O_2$ max values measured by these authors during head-out immersion were no different, on the basis of which Dressendorfer *et al.* concluded that the reduction in $\dot{V}O_2$ max typically reported for swimming in comparison to running or cycling should be related to inherent differences in the activities studied, rather than the effects of water immersion *per se*. In contrast, several others have reported a decrease in $\dot{V}O_2$ max during ergometry in cold water (Moore *et al.*, 1970; Pendergast, 1988; Manley, 1989), although, as pointed out by Manley (1988), dissimilarities in the experimental designs used, and at times a lack of uniformity between the land and water testing methods, tend to limit

interpretation of the obtained results. Furthermore, Pendergast (1988) has stressed the importance of a reduction in core temperature for the outcome of $\dot{V}O_2$ max tests performed in water and on land, stating that the absolute reduction in $\dot{V}O_2$ max during CWI would appear to be 10% to 30% for a reduction in core temperature of 0.5°C to 2.0°C.

MALE AND FEMALE RESPONSES TO COLD STRESS

Thermoregulation, like all other physiological systems, does not function in isolation; its actions are intricately inter-related with the actions of other regulatory systems (Johnson and Hales, 1984). McFarland (1984) has remarked that various homeostatic systems within the body are designed to counteract or forestall disturbances in the physiological state of the organism, at minimum cost. The extent of the load imposed by cold stress and the physiological costs to be paid are, however, modified by numerous factors. Throughout this review the reader has been reminded of the multifaceted nature of cold stress as an area of experimental investigation, both from a conceptual and a methodological point of view, a fact which often renders comparison between studies difficult. Factors which have been implicated as playing a role, either directly or indirectly, in thermogenic responses to cold stress include sex (McArdle *et al.*, 1984a and 1984b; Wagner and Horvath, 1985a and 1985b; Graham, 1988b), age (Keatinge, 1969; Horvath, 1981; Wagner and Horvath, 1985a and 1985b; Anderson *et al.*, 1996), body composition and peripheral insulation (Keatinge, 1960; Buskirk *et al.*, 1963; Hong, 1984; Eichner, 1991; Tikuisis *et al.*, 1991; Savourey

et al., 1996), initial thermal state of the body (Keatinge, 1960; Buskirk *et al.*, 1963; Cooper *et al.*, 1976; Kruk *et al.*, 1990; Torii *et al.*, 1996), food consumed and overall nutritional state (Rodahl *et al.*, 1962; Macdonald and Bennet, 1984; Askew, 1989), training state (Buskirk *et al.*, 1963; Horvath, 1981; Wagner and Horvath, 1985b; Eichner, 1991), ethnic background (Buskirk *et al.*, 1963; Hanna and Hong, 1972; Kollias *et al.*, 1974), level of awareness and psychological appraisal of the environmental conditions (Buskirk *et al.*, 1963; Budd *et al.*, 1993), ingestion of alcohol (Eichner, 1991; Kinzinger *et al.*, 1991; MacDonell and Wrenn, 1991; Johnston *et al.*, 1996), previous levels and extent of cold exposure (Wilkerson *et al.*, 1972; Sasaki and Tsuzuki, 1984; Tikuisis *et al.*, 1991; Budd *et al.*, 1993; Torii *et al.*, 1996) and disease (Buskirk *et al.*, 1963; Herr and White, 1991; MacDonell and Wrenn, 1991; Dawson *et al.*, 1994).

In the face of such a list it is clear that the examination of responses to cold stress separated from all such contaminating factors is unattainable. Furthermore, Buskirk *et al.* (1963) have highlighted difficulties that exist with regard to the isolation of factors said to modify thermal responses. As an example they suggest that changes occurring in response to physical conditioning may be as much due to changes in muscle and fat masses and therefore peripheral insulation, as to other physiological training effects. Similarly they propose that age, sex and ethnic differences in metabolic response may be explained partially by variations in insulation, or variations in the metabolically active mass of the body.

As in the case of many fields of study where numerous factors are said to modify psychophysiological responses, the situation has been exacerbated by the tendency of many investigators to isolate particular elements considered to be of importance, often without an apparent awareness of possible additional influential elements. While a thorough examination of individual variables is in itself necessary, one cannot ignore the entire network of causation if meaningful statements about thermal responses to cold stress are to be made. This necessitates the rigorous control and standardisation or, at the very least, detailed consideration of such factors as those listed above. Review of the literature over several decades would suggest an improvement in this regard. An analysis of 74 articles, all experimental investigations of human thermogenic responses to cold stress, revealed subject descriptions in articles published 16 to 20 years ago that gave as little information as: "five normal healthy male subjects" or "four physically well-trained male subjects, aged 24-39 years". More recently, subject characteristics have tended to include a report of $\dot{V}O_2$ max, percent fat, sum of skinfolds and body surface area, in addition to age, height and weight.

Common to the majority of the literature, however, is a tendency to focus virtually exclusively on the responses of the young, adult Caucasian male or, even amongst relatively recently published researchers, to pool data for males and females. Of the 74 articles considered for analysis, for example, only 18 (24.3%) included female responses in any way. Seven of those

studies (38.8%) pooled male and female data without any apparent consideration of possible sex effects upon the thermogenic responses of their subjects. Sjödin and colleagues, for example, in 1996 lamented the difficulties of interpreting and comparing data due to such factors as "differences in the subjects studied", yet, by pooling the data of 11 men and 2 women subjects, went on to ignore the most fundamental of differences between subjects. This despite general recognition that greater numbers of men and women are being exposed both at work and recreationally to circumstances where cold temperatures may limit performance and calls by the likes of Graham (1988b) for closer matching of the physical characteristics of male and female subjects in order to facilitate comparison between the sexes. The importance of this has been demonstrated in heat stress studies, where apparent superior male tolerance has been questioned on the basis of disparate anthropometric dimensions and heat acclimation rather than sex *per se* (Taylor *et al.*, 1986).

The data that do exist comparing male and female responses to cold stress indicate that thermal sensitivity is different; the understanding of such differences is, however, limited and explanations have tended to focus upon morphological factors. Furthermore, there appear to be no studies which have considered the implications of cold stress for subsequent dry aerobic activity in terms of sex differences in thermogenic responses.

Several authors have reported that, during CWI at rest, women

cool more rapidly than men and yet do not show a greater metabolic response (Kollias *et al.*, 1974; McArdle *et al.*, 1984a; Graham, 1988b; Pendergast, 1988). Contradictory opinions such as those expressed by Doubt (pers. comm.) that women tend to lose heat at a slower rate than men and that at identical core temperatures thermogenic responses may not be different, may in part be due to the diversity of protocols used to examine such responses and a failure to match correctly male and female subjects. Research has demonstrated that, where females possess larger skinfolds and percentages of body fat than their male counterparts, reductions in core temperature may be similar over a range of water temperatures; where these factors are more closely matched, the picture is quite different, with women unable as effectively to maintain rectal temperatures (McArdle *et al.*, 1984a and 1992). McArdle and co-workers (1984a) suggested that, for resting subjects, CWI produced significantly larger metabolic increases for males only once core temperature dropped by more than 1°C. Quiescent older women have been shown to be able to maintain their rectal temperature during cold air exposures (10 to 28°C) in contrast to men of the same age or younger men and women, but at greater metabolic cost (Wagner and Horvath, 1985a). This study highlighted the importance of the amount and distribution of body fat, but also postulated that the rapidity of the metabolic increase in older women was likely due to a greater availability of energy substrates circulating in their blood since, during the first hour of exposure, they had higher blood glucose levels and free fatty acid levels double those of the other subject groups.

The complex nature of the relationship between body structure and response to cold stress is further evident in studies of exercising men and women. Having suggested that differences in lean body weight and in the surface area-to-mass ratio between the sexes may account for some of the observed differences in response to cold stress at rest, McArdle and colleagues (1984b) observed that submaximal exercise had the effect of preventing or retarding a fall in rectal temperature in both male and female subjects during cold stress. These authors proposed that part of the reason may lie in the extra protection from heat loss due to increased blood flow in the exercising limbs afforded to the female by relatively greater limb fat for the same trunk fat thickness compared to males. It has long been known that tissue insulation above that offered by skin and adipose tissue is attributable mostly to the insulation of the nonperfused muscle shell. This effect is reduced during exercise in cold water in proportion to the intensity of exercise, and total insulation approaches very closely the level of skin and adipose tissue alone in severe exercise (Park *et al.*, 1983). Thus, despite a larger surface area-to-mass ratio and a smaller active muscle mass, the reduced conductance afforded by higher subcutaneous adiposity at specific sites is likely to act in the females' favour during exercise. Kollias *et al.* (1974) commented that the heat conductance of a 3cm layer of subcutaneous fat is equivalent to that of 6cm of bloodless muscle. Furthermore, in comfortable to cold environments women experience greater vasoconstriction of the cutaneous circulation and arterial flow to the deeper limb structures (McArdle *et al.*, 1984b; Wagner and Horvath, 1985b).

The resultant decrease in limb temperatures would further favour thermal balance for the female exercising in the cold.

In reviewing the limited number of studies in this area, Graham (1988b) has pointed out, however, that although sex differences in deep body cooling apparently disappear when subjects are forced to a given level of metabolism, data collected in uncontrolled situations where metabolic rates can be individually selected resemble those reported for subjects at rest. Shiraki *et al.* (1986), for example, reported that female breath-hold divers tolerated a 1.4°C greater drop in rectal temperature over the same time period as male divers. Although estimated heat loss was similar for males and females, this was compensated for in males by a relatively high heat production, hence a comparatively smaller degree of deep body cooling.

Clearly therefore, sexual dimorphism in body structure is of significance for insulation, ease of heat exchange and in heat production capacity. The relative importance of such aspects in determining an individual's response to cold stress, be it during CWI, air or cold pressor tests, at rest or whilst exercising, either at a fixed metabolic rate or in situations where metabolic rates can be individually selected, is as yet poorly understood. They are likely to be of greater relevance where differences in subjects are extreme or in subjects at rest (Graham, 1988b).

A growing number of scientists support the contention that morphology alone cannot account for the differences that exist

between males and females in their responses to cold stress (LeBlanc *et al.*, 1978; McArdle *et al.*, 1984a and 1984b; Wagner and Horvath, 1985a; Graham, 1988a and 1988b; McArdle *et al.*, 1992). While the evidence in this regard is by no means complete, sufficient exists to support the contention that there are fundamental sex differences in the sensitivity and capacity of the thermogenic response to cold stress, with females showing reduced sensitivity compared to males (McArdle *et al.*, 1984a; Graham, 1988b).

According to Graham, the likelihood of such differences is strengthened by similar reports in responses to cold pressor tests, since dimorphism during localized application of cold to the face or to a hand or foot is likely to be of lesser significance than during whole body cooling. LeBlanc *et al.* (1978) observed a depression in female cardiovascular functions (heart rate and blood pressure) as a result of cold hand and cold face tests, data which are apparently supported by unpublished findings cited by Graham (1988b). Others, however, have failed to confirm that women have a less responsive blood pressure response to cold pressor tests (Mannino and Washburn, 1987), or whole body cooling in air (Wagner and Horvath, 1985b).

LeBlanc and colleagues postulated that differences in sympathetic or cholinergic responses or some hormonal reactions related to sex may underlie the depressed female cardiovascular functions found in their study. They questioned whether reduced sympathetic activity may be due to lower catecholamine response

to the stressor in women. Indications of low sympathetic adrenal-medullary reactivity in females compared to males have been reported for subjects undergoing intelligence tests and during film-induced sexual arousal (Frankenhaeuser, 1975). Frankenhaeuser has commented that the fact that females do not mobilize epinephrine with the same readiness as males, may mean that they live more "economically".

It is clear, therefore, that the topic of sex differences in thermoregulation leaves more questions unanswered than answered. This is particularly true in relation to the underlying mechanisms for such differences; Graham (1988b) has speculated that reported sex differences in the preoptic area of the human hypothalamus may have functional significance in this regard, since it is known that the central control of thermoregulation is based here, but this is an area which warrants further investigation. Although, as Doubt (1991) has pointed out, there is no single response that covers every type of cold exposure, exercise protocol, or individual, there is a real need for more thorough consideration of subject characteristics and other factors which may influence thermoregulation in order that more meaningful comparisons between studies can be made.

PSYCHOPHYSICAL RATINGS

Ratings of Perceived Exertion (RPE)

Many neuromuscular and physiological parameters have been proposed as contributing to effort perception during exercise. It is evident that the perception of exertion is complex in

nature, and rather than being the outcome of a single primary cue, represents the synthesis of a variety of factors during different types of physical work (Pandolf, 1978). Gamberale (1985) points out that exertional ratings represent a subjective, distal reaction; the extent to which this distal reaction reflects the reaction within the individual organism is mediated by various influences depending on the situation. As an estimation of effort expenditure, the 15-point RPE scale (Borg, 1970) is known to be influenced by factors such as age, sex, physical training, personality structure, pain tolerance, mode of exercise and intensity and duration of the activity (Sidney and Shephard, 1977; Pandolf, 1978; Morgan, 1981; Robertson, 1982; Carton and Rhodes, 1985; Thomas *et al.*, 1995).

Morgan (1981), in describing effort sense as being based upon physiological cost, cognition (thinking) and perception (feeling), has cautioned against simplistic models which focus upon a single variable (eg. heart rate) or system (eg. respiratory) in attempting to understand its formation. It is generally recognised today that RPE represents a *Gestalt*; nevertheless, a large portion of the extant literature has been directed at assessing the primary factors driving perceived exertion. Since Borg's original concept focusing on the association of perceptual responses and heart rate as an index of exercise strain, more recent research has centred upon a two-factor model, as proposed by Ekblom and Goldbarg (1971), in an attempt to assess the relative contributions of local *versus* central factors. To facilitate this further, a differentiated

rating system was introduced by Pandolf and colleagues (1975): local ratings incorporate feelings of strain in the working muscles and joints, central values rate sensations involving the cardiopulmonary systems and an overall rating involves integrating signals from both of these with whatever weightings are deemed appropriate. It has been suggested that differentiated reports of effort sense may more closely reflect discrete physiological symptoms than a generalised version representing both physiological and non-physiological factors (Pandolf *et al.*, 1975; Pandolf, 1978; Carton and Rhodes, 1985).

The degree to which any one physiological variable drives the perception of effort is a topic of considerable discussion. Many scientists adhere to the theory that a critical work threshold must be reached before central rather than local cues dominate the perception of effort, with a relative workload of 50% VO_2 max cited as the point at which this occurs (Pandolf, 1978; Carton and Rhodes, 1985; Rejeski, 1985; DeMello *et al.*, 1987). In other words, during dynamic exercise, the relative contributions of local and central cues to effort sense is mediated by both the intensity and duration of the exercise. According to Carton and Rhodes (1985), once the critical work threshold is reached, the intensity of the central stimulus increases proportionately as a function of the absolute rather than relative increases in ventilation. The prominence of pulmonary ventilation and respiratory resistance as cues for RPE has been stressed in the past (Pandolf, 1978; Mihevic, 1981; Robertson, 1982; Carton and Rhodes, 1985). In examining possible "sensing" mechanisms

through which the relative workload might be assessed, DeMello and colleagues proposed that the lactate threshold is an important physiological anchor point for the perception of effort. They reported that RPE at the lactate threshold was similar for trained and untrained men and women, even though such thresholds occurred at substantially different percentages of $\dot{V}O_2$ max and absolute levels of $\dot{V}_E$, $\dot{V}O_2$ and heart rate. Further support for this contention is offered by Hetzler *et al.* (1991) and Seip *et al.* (1991), all of whom reported closer agreement between RPE and blood lactate levels than heart rate, $\dot{V}O_2$, $\%\dot{V}O_2$ max, $\dot{V}_E$, and the ventilatory equivalent for oxygen.

Given the diversity of variables which influence RPE, it is clear that the interplay between local and central factors will vary depending on how those variables combine. It has been pointed out that any physiological variable linearly related to exercise intensity will correlate quite highly with RPE (Hetzler *et al.*, 1991); correlational evidence, however, does not necessarily imply causality (Pandolf, 1982). For example, Pandolf has cautioned researchers that many of the hypothesised factors signalling local effort (mechanoreceptor and Golgi tendon organ activity, and sensations from muscle, skin, joints and ligaments) would be nearly impossible to quantify truly. Similarly, others have advised prudence in the interpretation of data where the perception of fatigue, rather than the perception of effort *per se*, may complicate the picture (Pandolf *et al.*, 1972; Carton and Rhodes, 1985). With specific reference to studies which have implicated skin or core temperature as perceptual cues affecting

RPE, Carton and Rhodes have cautioned that this may represent a failure to discriminate between perceived effort and perceived discomfort. Others, however, have claimed that subjects were able to distinguish accurately between thermal and comfort sensations and effort perception (Pandolf *et al.*, 1972; Toner *et al.*, 1986).

The challenge in investigating the effects of cold upon RPE lies in understanding the degree to which the constructs of exertion and fatigue or discomfort can be totally separated. Similarly, there is a need to identify the extent to which thermal variables themselves may mediate effort perception, and the implications of changes in thermal variables for other factors already known to influence RPE.

Studies implicating thermal responses as cues for the setting of RPE have tended to focus upon the effects of heat stress - those relating the effects of cold and RPE are relatively few and their conclusions somewhat varied. Carton and Rhodes (1985) have commented that the postulated relationships between physiological variables and RPE can obviously be disturbed when exercise is performed under irregular conditions, such as under heat or cold stress, while Pandolf (1982) has stated that when a particular physiological cue is markedly altered over others during exercise, the resultant sensation can easily dominate RPE. Indications from studies in hot conditions, however, are that heat load does not have an impact upon perceived exertion comparable to the physiological strain it produces (Pandolf

et al., 1972; Pandolf, 1978; Gamberale, 1985). The importance of esophageal, rectal and muscle temperatures as specific mediators of effort perception during heat stress has been queried (Morgan, 1981; Pandolf, 1982), although Pandolf suggested that mean skin temperature may play a significant role, particularly in local ratings.

Much further research is required with regard to rated perceptual exertion during and after cold stress. Patton and Vogel (1984) describe studies where subjects have found exercise intensities more tiring in a wet-cold (5°C) situation or have become exhausted more rapidly at 0°C than at warmer temperatures. Horstman (1977), on the other hand, found RPE to be significantly lower during exercise at 5°C than at 25°C. This reduction in RPE paralleled an increase in $\dot{V}_E$ and $\dot{V}O_2$, an observation which contradicts past studies linking an increase in $\dot{V}_E$ in particular to a rise in RPE. At the same time, the respiratory quotient and venous lactate concentrations decreased at 5°C. Maw *et al.* (1993) also observed a significant decrease in overall RPE during exercise in cool (8°C) ambient conditions compared to neutral (24°C) or hot (40°C) conditions. This was accompanied by a reduction in heart rate. Similarly, significant reductions in RPE were observed in subjects exercising at high intensities in 20°C water compared to 26°C water (Toner *et al.*, 1983 and 1986). At low intensities RPE was unchanged, despite $\dot{V}O_2$ being significantly elevated for the cold water condition. While these authors state that the change in $\dot{V}O_2$ associated with exercise in cold water does not add to the overall perception of

exertion, they conclude that RPE is related to cardiopulmonary variables rather than to thermal measures.

This suggestion appears to be supported by the work of Gordon and colleagues (1990), who examined the implications of a neck cooling device for performance during strenuous exercise. Heart rate and RPE were unaltered by neck cooling, despite a reduction in rectal temperature when the device was worn. These scientists point out too that rectal temperature may have even underestimated the effectiveness of the device in reducing temperatures at other sites in the body. Thus the suggestion that core temperature plays little role as a central mediator of percept formation, as is suggested by some studies involving heat stress, appears to be upheld. Another study examining the effect of cold upon submaximal exercise reported that RPE was not significantly affected, despite an increase in $\dot{V}O_2$ and decreases in skin and core temperatures (Horvath, 1981). Once again, heart rate was unchanged.

The exact mechanisms by which RPE may be lowered during certain types of exercise under cold stress remain to be elucidated. Calls have been made repeatedly for the systematic evaluation of local and central factors in order to better understand the overall perception of effort during exercise. While this has been addressed by many scientists, it clearly holds true for exercise during and after cold stress.

PERCEIVED THERMAL SENSATION

It was only in the Second Century A.D. that Galen (160 A.D.) suggested that in addition to the sense of touch, as described by Aristotle (350 B.C.) in his doctrine of the five senses, there should be added a sense of heat and cold (Gagge *et al.*, 1967). Research this century has described and continues to investigate the empirical relationship between temperature, ambient and perceived, and the complex interaction of factors which make up Man's experience of it.

Initially the sensation of thermal comfort was defined as "that state of mind which expresses satisfaction with the thermal environment" (American Society of Heating, Refrigeration and Air-Conditioning Engineers (ASHRAE), 1966, as cited by Gagge *et al.*, 1967). This was operationalized in studies which used temperature perception judgements based on verbal scales with categories ranging from cold through to hot. It became usual to define thermal comfort as the perception of an environment judged as neither cool nor warm (sensory thermoneutrality), as well as slightly cool or slightly warm (Grivel and Candas, 1991). In other words, scales used to assess thermal comfort in this way are based on verbal anchors which make reference to temperature sensation. Expanding on this concept, Gagge and colleagues devised separate scales for the assessment of thermal comfort and of thermal sensation, commenting that this made possible the evaluation of combined sensations such as "Pleasantly Warm" or "Uncomfortably Cool". Their scale of thermal sensation followed the ASHRAE scale, except that their point of no thermal sensation

was termed "Neutral", thus replacing the ASHRAE use of the word "Comfortable":

COLD-COOL-SLIGHTLY COOL-NEUTRAL-SLIGHTLY WARM-WARM-HOT.

Clearly the concept of human comfort is complex, involving far more than the sensation of temperature alone. One's sense of comfort and discomfort applies to and is influenced by the entire environment; it differs from person to person and for the same person from time to time depending on a multitude of physical and psychological factors. Comfort is usually associated with conditions that are pleasant and compatible with health and happiness, whereas discomfort is related to pain which is usually interpreted as unpleasant (Hardy *et al.*, 1971). In the assessment of environmental stress at a psychophysical level therefore, a subtle distinction can be made between scales which are perceptual in nature (such as temperature sensations) and those which are affective in nature (such as comfort or pleasantness) (Grivel and Candas, 1991). Macphearson (1972) has stated that the affective level - the point at which Man becomes aware of discomfort and resents the environment - may be the most important measure of physiological strain and hence of environmental stress for an individual.

Given the diversity of factors which influence thermal neutrality or thermal comfort at any one time, it is not surprising that scientists have listed very different temperatures as those which fall within these zones. According to Gagge *et al.* (1967), for example, thermal neutrality for American subjects lies between

28-30°C for steady exposure to a given environment. This is somewhat higher than the 24°C cited by Åstrand and Rodahl (1986) as the preferred indoor temperature in the United States, and considerably higher than 18°C, the preferred indoor temperature in Britain. Grivel and Candas (1991) reported that for European subjects the preferred temperature was 26.6°C, whereas Macphearson reported that the figure for Australians was 22.9°C. As this author correctly points out, however, a considerable portion of the population would disagree with a mean rating; in his example some subjects said they were too warm at 18°C, while others protested they were still cool at 27°C.

Similar discrepancies are evident with regard to core and skin temperatures classified by subjects as comfortable or neutral. Values for core temperature range between 36.2°C to 37.7°C (Gagge *et al.*, 1967; Åstrand and Rodahl, 1986; Grivel and Candas, 1991) and values for skin temperature between 31.9°C to 36.2°C (Benzinger *et al.*, 1963; Åstrand and Rodahl, 1986; LeBlanc *et al.*, 1976; Grivel and Candas, 1991). It is interesting to note that while some authors contend that comfort temperature may be subject to some degree of accustomization (Åstrand and Rodahl, 1986), others hold that the preferred temperature - the level of thermal neutrality - stays the same (Macphearson, 1972), even though the degree of physiological strain induced by a given environmental stressor may be altered. Thus cold acclimatization is associated with reduced subjective feelings of cold and in any given environment the core temperature is allowed to drop to a lower level (Macphearson, 1972; Shephard,

1985; Jansky *et al.*, 1996). Park *et al.* (1983) discovered that, during CWI, habitual divers usually complained of internal chilling, not external chilling, whereas nondivers complained of external chilling. These authors suggested that this may imply that cold receptor activity was reduced in divers. It has been observed that the rate of cutaneous cold receptor discharge (both static and dynamic) is lower in long-term acclimatised animals than in warm-acclimatised animals (Park *et al.*, 1983).

Thermoreceptors act as transducers converting patterns of heat energy into afferent impulses. They respond most actively in the innocuous intensity range; in other words, they exhibit activity between the extremes heat pain and cold pain (Schäfer *et al.*, 1984). At very cold temperatures, cold nociceptors alone are stimulated; as the temperature rises to 10 to 15°C, pain impulses cease, but cold receptors begin to be stimulated. Above about 30°C warmth receptors become stimulated while cold receptors fade out at about 43°C. At around 45°C, pain fibres begin to be stimulated by heat. Paradoxical discharges can be elicited by temperatures at either end of these ranges, such that cold temperatures may produce burning hot sensations and hot temperatures feelings of extreme cold (Fox, 1974; Schäfer *et al.*, 1984; Guyton, 1986) Schäfer and colleagues have pointed out that maximal sensitivity to constant temperatures and to rapid changes in temperature may not lie in the same temperature range. On the basis of their studies involving mammalian and reptilian responses, static activity maxima were distributed in the range from -5°C to 40°C for cold fibres and 29°C to 38°C for warm

fibres. Maximal dynamic responses on the other hand, ranged from 20°C to 30°C and from 29°C to 32°C for cold and heat receptors respectively. Although it is recognised that some free nerve endings can act as cold receptors, a definitive cold receptor has been identified, this being a small myelinated nerve ending, the tips of which protrude into the bottom surfaces of basal epidermal cells (Guyton, 1986). As yet, however, distinctive warmth nerve endings have not been identified histologically; they are presumed to be free nerve endings distributed deeper than cold receptors (Guyton, 1986).

According to Fox (1974), exactly how the peripheral receptors act as thermal transducers is a matter of speculation, although the stimulus is apparently temperature itself, rather than the thermal gradient across the receptor terminals. Kenshalo and Nafe (1963) described three factors which materially affect the size of the thermal sensory threshold: the temperature of the skin stimulated, the rate at which the skin temperature is changed and the area stimulated.

Kenshalo and Nafe observed that, when skin temperature is low, the cool threshold is unaffected; however, the warm threshold rises markedly with decreasing skin temperature. The opposite occurs with rising skin temperatures: the warm threshold is unaffected but the cool threshold increases. This factor is likely to be of significance where subjects are exposed to cold and then asked to rate their perceptions of thermal sensations during subsequent exercise in a warmer environment, as was the

case in the present study. According to Gagge *et al.* (1967), the development of temperature sense is very rapid when subjects move from warm to cool or cool to warm environments, and during the first five minutes appears to be constant even though the skin temperature is falling or rising.

With regard to the rate of change of skin temperature, Kenshalo and Nafe stated that, within the range of skin temperatures in which complete thermal adaptation is possible, about 32°C to 37°C, thermal sensations depend upon the rate of change of the temperature produced by the stimulus. Outside of this range, persisting warm or cool sensations occur, even though the tissue temperature is stable. Contradictory evidence does exist, however, for subjects whose skin temperature varied between 35°C to 36°C. In spite of seemingly suprathreshold rates of skin temperature change, Hendler *et al.* (1963) failed to elicit changes in thermal sensation appropriate with changes in skin temperature. In other words, warmth sensations did not immediately accompany rapid rises in skin temperature, nor did the sensation immediately cease when the skin temperature began to fall.

The third factor suggested by Kenshalo and Nafe as contributing to the size of the thermal sensory threshold is the area of skin that is stimulated. A tenfold reduction in the warm threshold has been documented with an increase in the area of thermal stimulation from $1cm^2$ to $4cm^2$ (Kenshalo and Nafe, 1963). Indeed rapid changes in temperature of as little as 0.01°C can be

detected if this is applied to the entire surface of the body simultaneously, since thermal signals from the entire area summate (Guyton, 1986). Interestingly, Tipton and Golden (1987) observed that, for initial responses to cold immersion, subjects whose torso was protected but whose limbs were exposed to 10°C water reported considerably greater thermal discomfort than when the limbs were protected and the torso exposed. This lead these authors to suggest that some difference may exist between the torso and limbs with regard to cold nociception. The importance of specific sites as drivers of PTS has been examined by Boutcher and co-workers (1995). In a comparison of exercise in hot, cool and thermoneutral environments, they concluded that forehead skin temperature, in contrast to other skin sites, contributed significantly to thermal sensation during exercise in cool conditions, but not in hot and neutral conditions.

The specific importance of each of these factors lies in the assumption that skin temperature is the primary variable leading the perception of thermal sensation or comfort, a position subscribed to by several researchers (Burton and Bazett, 1936; Gagge *et al.*, 1967 and 1971; Hayward *et al.*, 1977; Wagner and Horvath, 1985a). Hayward *et al.*, for example, found that subjects whose skin temperature had dropped to 12.8°C during CWI experienced no conscious sensation of being cold once the skin temperature had risen to the preimmersion level of 33°C to 34°C, despite core temperature being as low as 33.5°C. In differentiating between thermal sensation and thermal comfort, Hardy and co-workers (1971) found cold sensation to be more

responsive to lowering skin temperature than discomfort: a small decrease in skin temperature resulted in a rapid rise in sensory judgement of cold, in contrast to a small increase in discomfort sensation. Once again, however, not all scientists concur. Whilst recognising that skin temperature does play a role, Nielsen and Endrusick (1990) stated that 80-90% of whole body thermal sensation could be accounted for by core temperature alone. Although these authors employed a work/rest protocol, Hardy *et al.* (1971) by contrast, considered the sensation of temperature during exercise over the range 25-75% maximal work capacity to be dominated by sensor mechanisms in the skin and independent of core temperature. They considered the sensation of discomfort to be principally governed by thermoregulatory effector mechanisms such as sweating and cardiovascular responses. Toner *et al.* (1983), on the other hand, found thermal sensation correlated only slightly with skin and core temperatures during low and high intensity exercise in cold water.

As with any psychophysical relationships, dependent as they are upon so many interacting variables, what holds true under one set of conditions may not under others. Hardy and colleagues (1971) caution that although mean skin temperature and sensations of comfort or discomfort appear closely correlated during static conditions, such a correlation is limited and not applicable to dynamic conditions. During studies of the effects of transient ambient conditions, they observed a hysteresis effect between the sensor elements and the sources of temperature change. On

moving from cold to "neutral" conditions, subjects' sensory changes appeared anticipatory in the sense that they preceded skin and body temperature changes. Furthermore, cognisance must be taken of the fact that other factors such as deep body temperatures of the spinal cord, muscle, hypothalamus, viscera and so on, may contribute to thermal and comfort sensations (Gagge *et al.*, 1967; Hardy *et al.*, 1971). It is impossible to separate entirely such factors as core, skin and mean body temperatures in the evaluation of thermal perception, particularly where complicated by the interactions of other variables which influence such sensations (McCormick and Sanders, 1983). The relative contributions of each, however, over a range of conditions and for varying subjects does remain to be further clarified.

CHAPTER 3

EXPERIMENTAL METHODS AND PROCEDURES

PILOT TESTING

Phase I: Reliability of the Computer Assisted Data Acquisition System for Expired Air Analysis.

The metabolic response of the subjects to both maximal and submaximal exercise was assessed by means of an on-line, computer-aided expired air analysis system, the principles of which have been reported by Goslin *et al.* (1985).

A test-retest protocol was employed to determine the reliability of data acquisition during steady- and un-steady state exercise. Three male subjects reported to the laboratory on four occasions, each separated by at least 48 hours. Maximal exercise responses were obtained twice on separate occasions using the protocol outlined on pages 132-136. Two sets of steady-state responses were recorded every 30s over a 10min run at 8km.h^{-1}.

Results

For each of the ventilatory, respiratory and cardiovascular variables assessed during this phase the coefficient of variation was caculated on the basis of the difference between the test and retest results of steady state exercise. These values were as follows: heart rate (b.min^{-1}) = 5.2%; VO_2 (ml.kg^{-1}.min^{-1}) = 4.8%;

VO_2 ($l.min^{-1}$) = 5.3%; V_E (STPD) ($l.min^{-1}$) = 5.7%; f ($br.min^{-1}$) = 7%. Maximal values, as assessed by Student's related t-test, were not significantly different between testing sessions.

This phase of pilot testing thus ensured procedural consistency and established the reliability of data collection via the described method.

Phase II: Determination of Body Heat Loss Criterion for Peripheral Cooling.

Peripheral cooling was defined as a predetermined loss of body heat **without** a change in core temperature. It was therefore necessary as part of the pilot work for the experiment to establish a fixed heat loss that could be tolerated by all subjects without a drop in core temperature.

Three males and three females were recruited as participants in this preliminary phase, each of whom conformed to the subject requirements laid down on pages 126-128. Subject preparation prior to data collection was as prescribed on pages 131-132; heart rate was the only parameter not measured before or during the immersion. Using the techniques outlined on page 136-138, resting $\dot{V}O_2$ was measured every minute while the subject sat immersed up to the level of the jugular notch. Core temperature and skin temperatures at four sites were recorded every minute for the duration of the immersion. The subject remained in the tank until such time as the core temperature had dropped by 1°C compared to the pre-immersion value. Total skin heat loss was

calculated by the methods described on page 143-145 for pre-immersion, the minute immediately preceding a drop in core temperature and for the final minute of the immersion, at which time core temperature was 1°C lower than before immersion.

Results

The results of this phase indicated that, as expected, interindividual variability existed with respect to the rate at which heat was lost ($\bar{x}$ water temperature 20.7 ± 1.6°C). McArdle *et al.* (1984a) similarly reported that, at a water temperature of 20°C, the core temperature of male subjects remained at pre-immersion levels for the first 10 to 20min of immersion. The time required for a drop in core temperature to be evident was, on average, 13.5 ± 10.9min, at which time the total skin heat loss (H_{sk}) for these subjects ($\bar{x}$ stature 176.05 ± 10.16cm; $\bar{x}$ mass 69 ± 10kg; $\bar{x}$ %fat 11.75 ± 4.2%; $\bar{x}$ body surface area (BSA) 1.84 ± 0.19m²) ranged from 101.5kcal.m^{-2}.h^{-1} to 206.96kcal.m^{-2}.h^{-1} ($\bar{x}$ 144.9 ± 35.7kcal.m^{-2}.h^{-1}). On the basis of these results, together with the knowledge that maximal peripheral vasoconstriction develops at water temperatures as high as 29°C to 33°C (Rennie *et al.*, 1962; Bullard and Rapp, 1970; Hong, 1984), the fixed amount of heat loss that was to be termed peripheral cooling was set at 100kcal.m^{-2}.h^{-1}, since this would have accommodated all of the subjects studied. While it must be remembered that many variables combine to affect the rate and amount of heat lost during cold exposure, the values obtained in this experiment seemed to compare well to those of Webb (1978), who reported a loss of 210kcal with a drop in core

temperature of 0.3°C to 1.1°C. Similarly the subjects of Craig and Dvorak (1975) experienced a total skin heat loss of 275kcal after 1h in 24°C water.

Phase III: Effects of Body Cooling of Varying Degrees Upon Subsequent Dry Exercise Performance.

The purpose of this phase of the pilot study was to confirm that the decision made with regard to the amount of heat that was to be lost during peripheral cooling sessions would indeed create significantly different thermal conditions for the subjects from one session to the next. It was also deemed necessary to establish that the prescribed exercise intensity (50% $\dot{V}O_2$ max) would represent steady state exercise for the subjects.

Table I: Summary of physical and physiological characteristics of six pilot study subjects (phase III).

	$\bar{x}$	s.d.
Age (yr)	21.6	0.6
Mass (kg)	68.7	2.3
Stature (mm)	1727.0	38.0
Sum of Skinfolds (mm)	73.1	19.4
% fat	10.6	2.1
% muscle	43.4	2.3
BSA:mass ratio	37.8	0.6
$\dot{V}O_2$ max ($ml.kg^{-1}.min^{-1}$)	54.8	4.1
Ventilatory threshold $\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)	41.0	3.1
Ventilatory threshold % $\dot{V}O_2$ max	74.9	3.4

Three males and three females, each of whom conformed to the subject requirements laid down on page 126-128 (see Table I), took part in phase III of the pilot study. This involved the completion of each of the sessions, exactly as would occur during final data collection, specific details of which appear in this chapter. In other words, each of the subjects completed a $\dot{V}O_2$ max test and three submaximal treadmill runs: one with no prior thermal manipulation (control), one following a core temperature drop of 1°C (central) and one following peripheral cooling (peripheral) along the lines described above.

Results

Statistical analyses by means of two factor ANOVA with repeated measures on both factors revealed significant differences between the conditions and over time for all temperature-related data. Heart rate, cadence and the subjects' perception of thermal sensation were also affected by the experimental treatment. All other variables, except breathing frequency, showed significant changes over time which appeared to be due to changes occurring during the cold treatments, rather than during the control condition.

That subjects were able to maintain steady state exercise at the prescribed intensity during the control condition was confirmed. A one-way ANOVA with repeated measures revealed no significant change in $\dot{V}O_2$ over the 30min exercise period. In addition, for each of the subjects the ventilatory threshold which occurred during the graded $\dot{V}O_2$ max test was determined as a break in

linearity of expired ventilation ($\dot{V}_E$ (STPD)) and of expired CO_2 ($\dot{V}CO_2$) when plotted as a function of $\dot{V}O_2$ (Powers *et al.*, 1983; Powers *et al.*, 1984; Prud'homme *et al.*, 1984; Aunola *et al.*, 1988).

The mean ventilatory threshold occurred at 74.9 ± 3.4% $\dot{V}O_2$ max, with a range from 69.3 to 80.5% $\dot{V}O_2$ max. These values concur with a range reported in the literature of between 61 to 86% $\dot{V}O_2$ max for similar moderately to well trained individuals (Powers *et al.*, 1983; Hagberg, 1984; Prud'Homme *et al.*, 1984; Williams *et al.*, 1991). Over the exercise period the six pilot subjects averaged a $\dot{V}O_2$ of 29.1ml.kg^{-1}.min^{-1}, or 53% $\dot{V}O_2$ max, clearly well below the ventilatory threshold. Given the information gleaned from phase III of the pilot study, together with prior reservations expressed with regard to heavy exercise following body cooling, 50% $\dot{V}O_2$ max was concluded to be a suitable and attainable intensity of steady state exercise.

SUBJECT SELECTION

Initially 47 healthy Caucasian students volunteered to participate in this study following standard informed consent procedures (see Appendix A). The age of the subjects was limited to between 18 and 25yr. None of the individuals was accustomed to cold exposure, either in water or air, on a regular basis. Initial subject selection was based upon morphological criteria and was then further restricted on the basis of $\dot{V}O_2$ max test results to minimise differences in cardiorespiratory capacity between subjects. The final sample comprised 13 males and 13

females.

In order to control for morphological factors that are known to influence responses to thermal stress, particularly where male *versus* female responses are concerned, volunteers had to satisfy several requirements in order to qualify as subjects for this research. All subjects conformed to a narrow range of body surface area-to-mass ratios. The body surface area (BSA) was calculated from stature (m) and mass (kg) according to DuBois and DuBois (1916):

$$BSA\ (m^2) = \frac{(71.84 \times M^{0.425} \times S^{0.725})}{10\ 000}$$

Where M = mass (kg); S = stature (cm)

In addition, body composition was determined using the method of Drinkwater and Ross (1980), the calculations for which appear in Appendix B. This four-compartmental analysis of body composition provided absolute and relative values for the fat and muscle tissue of the whole body. Once again, subjects were selected for their similarities in both of these tissue estimations. At the same time, however, cognisance was taken of reservations expressed with regard to the conversion from skinfold measures to percent fat (Ross and Marfell-Jones, 1982; Martin *et al.*, 1984; Jackson and Pollock, 1985). While it was deemed useful to retain some estimation of overall muscle and fat tissue, skinfold thicknesses were used directly to monitor the structural status of the subjects. The six skinfolds measured for body composition analysis (triceps, subscapular, suprailiac,

abdominal, front thigh and medial calf), together with the biceps and forearm skinfolds, were used to screen subjects in terms of fat distribution.

Following informed consent, all female volunteers were required to complete a questionnaire concerning gynaecologic history. It was intended that only those who had menstrual cycles falling within normal limits, who reported no unusual pre-menstrual discomfort or dysmenorrhoea and who were not currently taking oral contraceptives or other estrogen supplements would be considered as subjects for the research. Since basal body temperature rises at ovulation and remains elevated in the luteal phase (Eston, 1984; Pivarnik *et al.*, 1992), experimental data collection sessions for the female subjects all fell between days 5 and 12 after the onset of menses. Ovulation was defined as occurring 13 to 14 days after the onset of menses (Eston, 1984; Guyton, 1986; DiBrezzo *et al.*, 1991). Charts dating the onset of menses were maintained for all female subjects for the three months preceding data collection to ensure cycle regularity and identify the correct testing times for each individual (Lamont, 1986; Dombovy *et al.*, 1987; DiBrezzo *et al.*, 1991; Quadagno *et al.*, 1991).

ASSISTANT

The same assistant aided during pilot testing and each of the testing sessions for all subjects. She was experienced in scientific research and familiar with the exact protocols and equipment being used. The author is a certified paramedic

trained in life support and cardiopulmonary resuscitation.

MEASUREMENT OF ENVIRONMENTAL CONDITIONS

In both the laboratory and the underwater testing environments dry bulb temperature, relative humidity and barometric pressure were recorded for each testing session. Testing occurred in March through to May, during which period the water temperature in the tank was maintained at 20°C.

A review of the literature revealed that the opinions of researchers differ with regard to water temperatures that can be described as cold. Keatinge (1969), for example, described 17°C water as lukewarm, whereas a temperature of 17.5°C was considered by Choi *et al.* (1988) to be cold. Similarly, Hanna and Hong (1972) described water of 23°C as being "mild" when several other scientists have classified water temperatures above that as "cold" (Merrill Smith and Hanna, 1975; McArdle *et al.*, 1984a). For the purposes of the present study, and in keeping with many studies relating to cold water exposure (McArdle *et al.*, 1984a and 1984b; Toner *et al.*, 1984 and 1986), a water temperature of 20°C was accepted as meeting the criterion for CWI.

EXPERIMENTAL PROTOCOL

The research protocol can be divided broadly into four phases:

i) the administration of the informed consent information sheet, collection of anthropometric and biographic data and verbal familiarisation of the subjects with the

research procedure,

ii) habituation of the subjects to treadmill running and to the equipment used for data collection,

iii) a progressively incremented treadmill test of $\dot{V}O_2$ max,

iv) a series of submaximal treadmill runs at 50% $\dot{V}O_2$ max both with and without prior manipulation of the thermal status of the individual.

HABITUATION

The need to habituate subjects naive to treadmill locomotion prior to data collection has been stressed, particularly where measurements are to be made of gait patterns (Wall and Charteris, 1980 and 1981). Where gait parameters are not to be included for measurement, as in the case of a $\dot{V}O_2$ max test, for example, a brief period immediately prior to the test has been considered sufficient to accommodate subjects to the treadmill (O'Toole *et al.*, 1987). Although gait kinematics were not a primary feature of the present research, basic locomotor data were obtained as a means of assessing the work done on the treadmill. For subjects naive to treadmill locomotion, a programme of discontinuous practice sessions, for a total of approximately one hour, has been suggested by Wall and Charteris. The present subjects, however, were Human Movement Studies students who were largely familiar with performance on a motorised treadmill. In order to ensure that all subjects were accommodated to treadmill

locomotion, however, each performed a series of discontinuous practice sessions over a total of 30min. The treadmill speeds used for habituation sessions were a walk (at 5km.h^{-1}) and two runs (at 9km.h^{-1} and 13km.h^{-1}). A Quinton motor-driven treadmill (Model 643) was used for all testing.

Whilst on the treadmill during the habituation sessions subjects were required to accommodate to the equipment used to monitor respiratory responses. Thus a two-way pulmonary valve was used to breathe via a gas analysis system. Subjects were also asked to rate perceived exertion and thermal sensations during exercise, having been instructed in the correct use of the relevant scales.

SUBJECT PREPARATION PRIOR TO TESTING

For the submaximal exercise sessions each subject reported to the laboratory between 7h30 and 9h00 after abstaining from any pharmacological agent, tobacco or beverage other than water for 10h. The timing of food and fluid ingestion prior to testing followed that suggested by Schwellnus *et al.* (1990) in a study of the specific effects of diet upon body temperature. At least 2h before testing commenced, the subject consumed a standard meal comprising four slices of toast with 10ml margarine and unlimited amounts of water. The subject was then acclimated for 60min before testing in the environmentally stable laboratory. Thus all subjects completed the submaximal exercise sessions before 12h00, reducing circadian variations in physiological and psychophysical variables (Faria and Drummond, 1982; Shephard,

1984; Reilly *et al.*, 1984) as much as possible. During the time that the subjects stayed in the laboratory no additional food was consumed. Water was consumed *ad libitum* until 15min before exercise, at which time all subjects were required to drink 200ml of tap water. Approximately 15min before the completion of the 60min period the subject changed into a swim suit and ECG electrodes, temperature probes and the breathing apparatus were applied for a 10min pre-immersion, or in the case of the control condition, pre-exercise recording of baseline physiological measures.

Subjects were not required to undergo a period of supervised rest prior to the administration of the $\dot{V}O_2$ max test. They were, however, requested to abstain from any food, beverages, other than water, and tobacco for at least 2h before the test, and to refrain from any energetic physical activity for the 24h preceding the test. Consecutive experiments on any subject were performed after intervals of 48h. Such intervals provided for sufficient rest between tests yet enabled testing of female subjects to be completed within the stipulated 5 to 12days after the onset of menses.

ASSESSMENT OF MAXIMAL OXYGEN CONSUMPTION

The protocol used involved a direct, continuous, progressively incremented treadmill running test (Goslin *et al.*, 1985). With the treadmill set at 0% gradient, the subjects began 1min of warm-up at a speed of 5km.h^{-1}. Once the test was commenced at a speed of 8km.h^{-1}, the speed of the treadmill was increased by

1km.h^{-1} every minute until a speed of 17km.h^{-1} was reached. This speed was then maintained for the remainder of the test, while the angle of the treadmill was increased by 1% every minute until the subject could no longer continue. The end-point of exercise was subjectively determined by the subject, although all subjects were verbally encouraged to achieve their highest level in the test. Experienced assistants were on hand throughout to ensure the safety of the participant.

Physiological data recorded via an on-line computer-aided system included measures of oxygen consumption ($\dot{V}O_2$), carbon dioxide production ($\dot{V}CO_2$), respiratory exchange ratio (R value), minute ventilation ($\dot{V}_E$ (STPD)) and breathing frequency (f). In order to measure these the subject breathed via a Hans-Rudolf (Model 2700) two-way pulmonary valve. Expired air was directed through Collins ridged tubing (3cm diameter) to a Vista metabolic measurement system (Model 17520), where the temperature of the air was recorded. From this the expired air was sampled for analysis. Expired air analysis was performed by a previously calibrated Ametek (3-SAI) oxygen analyzer and a Gould Capnograph (mark III) carbon dioxide analyzer.

Throughout the study, during both dry and wet conditions, heart rate was monitored by means of Hewlett Packard ECG equipment. This consisted of a monitor (Model 78660 A/B), a telemetry transmitter (Model 78100 A), a telemetry receiver (Model 7810 A), and Narco-Scientific self-adhering surface electrodes. The frequency range of the telemetry transmitter was 450-470MHz.

Electrodes were attached to the subject's chest in a modified lead-II ECG configuration. The sites of attachment comprised the right and left sterno-clavicular joints and the V5 position (mid-axillary line). If necessary, chest hair was shaved at the attachment sites and the skin prepared by rubbing with an abrasive material before being cleaned with ethanol. The electrodes were prepared with Hewlett Packard Redux creme. The mean heart rate value displayed on the ECG monitor at the required time intervals was recorded for data purposes.

Every 30s, as physiological data were recorded during the VO_2 max test, subjective ratings of perceived exertion (RPE) were obtained from the subject. During an earlier habituation session subjects received standard instructions, adapted from Pandolf (1982) and Mihevic (1983), on how to rate effort perception (see Appendix C) and were familiarised with the use of the scale during exercise on the treadmill. The scale used was that of Borg (1970):

RPE SCALE

6	
7	VERY, VERY LIGHT
8	
9	VERY LIGHT
10	
11	FAIRLY LIGHT
12	
13	SOMEWHAT HARD
14	
15	HARD
16	
17	VERY HARD
18	
19	VERY, VERY HARD
20	

For the purposes of the $\dot{V}O_2$ max test, subjects rated overall RPE

only, these values being used as one of the criteria to ascertain whether the individual attained a satisfactory test result. As verbal communication by the participants was impossible for the duration of the test, subjects pointed to the ratings of their choice. These were verbally confirmed by the researcher before being recorded.

The primary criterion for the attainment of $\dot{V}O_2$ max was a plateauing of VO_2 despite an increase in exercise intensity (Hammond and Froelicher, 1984; Shephard, 1984; McConnell, 1988). The plateau criterion was that $\dot{V}O_2$ rose by less than $0.15 l.min^{-1}$ with further increases in exercise intensity (Shephard, 1984). Shephard has pointed out, however, that there is some disagreement as to how many subjects can reach a plateau and that, even with repeated testing, a plateau may not be demonstrated. In the event that a plateau did not occur in the present study, $\dot{V}O_2$ max was said to have been attained if all of the following physiological indicators were evident:

i) Subjective fatigue, exhaustion and an inability to continue (Shephard, 1984; McConnell, 1988),

ii) an R value of 1.0 or more (McConnell, 1988),

iii) heart rate within 10 beats of age-predicted maximum (Hammond and Froelicher, 1984; McConnell, 1988)

iv) an RPE of 19 or 20 (Hammond and Froelicher, 1984;

McConnell, 1988).

Where none of the above criteria was met in the absence of a plateau in $\dot{V}O_2$, the test was to be repeated. This possibility, however, never occurred (see Results).

DATA COLLECTION DURING CWI

In this study CWI was used to produce decreases of body temperature of varying degrees prior to ensuing dry submaximal exercise. Following approximately 45min of supervised rest, as discussed earlier, the subject changed into a swim suit and baseline physiological measures were recorded for 10min while the subject sat quiescent in a comfortable air environment in the vicinity of the water tank which had the inner (waterline) dimensions of 1.3 x 2.3 x 2.5m and a capacity of approximately 7500l. The subject then entered the tank of stirred water. During immersion a chair constructed of plastic tubing (see Figure 1) was used that allowed free circulation of water around the body. The subject sat still with the water level with the jugular notch.

Physiological and psychophysical data were collected every minute for the duration of each immersion. Respiratory data were captured by means of an oxylog (P. K. Morgan) secured to the outside wall of the water tank. The subject breathed through a face mask fitted with a turbine flow meter on the inspiratory side, with a hose connected to an analyzer on the expiratory side. The oxylog measured the volume of inspired air, the partial

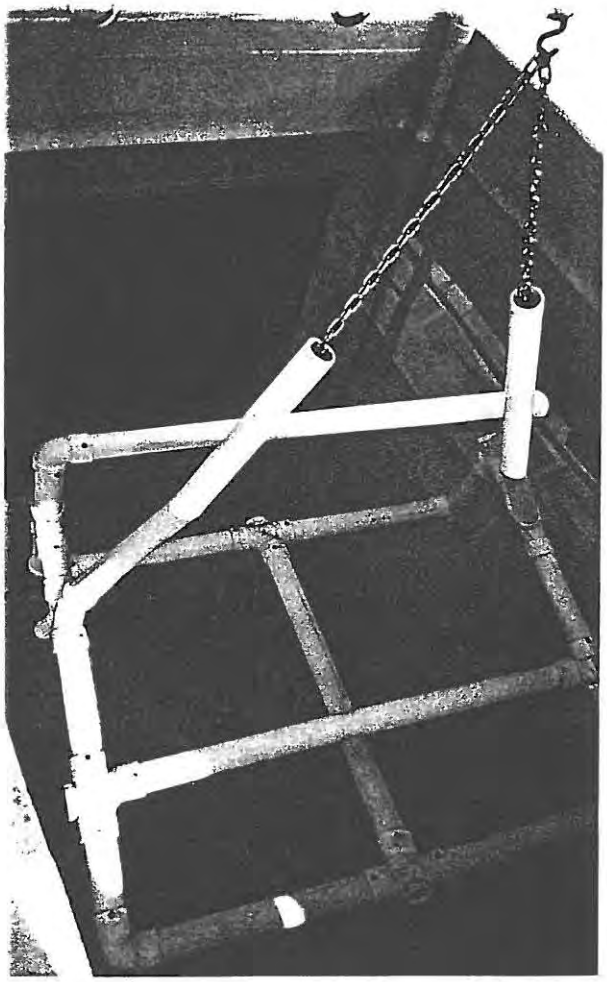


Figure 1: Water tank and seating arrangement used to collect data during CWI.

pressure of oxygen with two polarographic oxygen sensors for inspired and expired air, and air temperature (Louhevaara *et al.*, 1985). From a digital display $\dot{V}O_2$ and minute ventilation volume were recorded. Using the oxylog, calculated values for $\dot{V}O_2$ within a range of 0.25 to 3.01.min^{-1} are correct for a respiratory exchange ratio of 1.0 and a relative humidity of 50% (Louhevaara *et al.*, 1985). Previous research would suggest that the respiratory exchange ratio is likely to be very close to 1.0 during CWI (Tipton, pers. comm.; Webster, 1989). In a critique of the oxylog in comparison to the Douglas bag technique, Louhevaara and his colleagues have reported that, although the oxylog may over- or underpredict $\dot{V}O_2$ due to fluctuations in relative humidity around 50%, it is nevertheless very well suited to field measurements.

Heart rate was recorded with the same Hewlett Packard ECG equipment used for dry testing, with slight modification to some of the components. The electrodes, attached once again in a modified lead-II configuration, were covered with a layer of adhesive water-proof plaster and then with another layer of highly adhesive medical plaster. The electrode wires were lengthened to approximately 3m and connected to a telemetry transmitter placed inside a sealed bag taped to the stairs outside the water tank. The ECG equipment was set up on a trolley which was wheeled to the laboratory for treadmill testing immediately the subject left the tank.

Psychophysical assessment whilst at rest in the water tank

involved the use of a rating scale of perceived thermal sensations (PTS) according to Hardy *et al.* (1971):

PTS

1. COLD
2. COOL
3. SLIGHTLY COOL
4. NEUTRAL
5. SLIGHTLY WARM
6. WARM
7. HOT

Subjects were instructed in the correct use of the scale during an habituation session (see Appendix C for instructions). Whilst breathing through the mask during each 60s data collection bout, subjects were asked to indicate their subjective evaluation of temperature sensation. The values were confirmed verbally by the tester before being recorded.

Core and skin temperatures were also recorded during each 60s bout. Core temperature was obtained by rectal thermometry: a flexible thermistor (Yellow Springs Instrument Co. Model 401) was inserted approximately 10cm past the anal sphincter. Temperatures were recorded from an analogue display accurate to 0.1°C (Yellow Springs Instrument Co. Model 461 UC) All thermistors used in this study were matched for response time and temperature reading in various thermal baths between 20°C and 50°C. Although use of the rectal temperature as an index for core temperature has been debated (Hardy, 1961; Benzinger and

Taylor, 1963; Murray-Smith *et al.*, 1984), it provides an easily accessible measure of core temperature and continues to be frequently used, particularly in studies of CWI where standard equations for heat loss calculations are used (Rennie *et al.*, 1962; Veicsteinas *et al.*, 1982; Toner *et al.*, 1984; Wagner and Horvath, 1985a; Choi *et al.*, 1988; Tikuisis *et al.*, 1988; Doubt and Hsieh, 1991), thus allowing for meaningful cross-comparison between studies. Indeed, according to Hayward *et al.* (1977), rectal thermometry remains the most common method of obtaining core temperature. Jaeger *et al.* (1980) found esophageal temperature to be 0.6°C lower at rest, but the two measures became statistically indistinguishable after 5min of submaximal exercise. Similarly, Kenney (1988) stated that rectal temperature tracked core temperature well, despite a slight lag behind esophageal temperature. Saltin *et al.* (1968) reported differences of 0.14°C between esophageal and rectal temperatures and of 0.16°C between tympanic and rectal temperatures during submaximal exercise and concluded that rectal temperature did provide a satisfactory index of core temperature. They noted that the disparity between rectal and central arterial blood temperature was in the region of 0.15°C.

Skin temperature was obtained at four sites, according to the method employed by Choi *et al.* (1988). Disc thermistors specialised for surface temperature measurements (Yellow Springs Instrument Co. Model 409(A)) were attached as follows:

i) the chest, T_c, positioned on the left sternal border at

the 4th intercostal space,

ii) the leg, T_l, positioned over the gastrocnemius muscle,

iii) the arm, T_a, positioned over the triceps muscle,

iv) on the dorsum of the hand, T_h.

The mean temperature of the skin (T_{sk}) and the mean body temperature (T_b) were calculated as follows:

$$T_{sk}\ (°C) = 0.50\ T_c + 0.36\ T_l + 0.09\ T_a + 0.05\ T_h$$

where the fractions of the body's total surface area represented by each specific area are those designated by Choi *et al.* (1988).

$$T_b\ (°C) = 0.67\ T_{re} + 0.33\ T_{sk}$$

where T_{re} = rectal temperature (Kollias *et al.*, 1974).

Through the years several different equations for the evaluation of skin temperature, with a varying number of sites, have been suggested, most of them based upon an area-weighted mean value. Olesen (1984), in an examination of the number of sites necessary

to accurately estimate mean skin temperature, reported that, in warm conditions, 2 to 4 sites were adequate; in cold conditions 8 to 12 sites may be required. This author concluded, however, that the number of sites to be used has often to be decided as a compromise between what is ideal and what is technically possible. Such a compromise was evident in the present study. Since, immediately following immersion, the subjects ran on the treadmill, during which time body temperatures were recorded, thermistor placement had to be such as to allow for unrestricted movement. Furthermore, immersion in cold water is considered by many scientists to provide a very uniform skin temperature, clamped to the temperature of the surrounding water (Rennie *et al.*, 1962; Hanna and Hong, 1972; Kollias *et al.*, 1974; Park *et al.*, 1983; Pendergast, 1988), although evidence to suggest that regional differences in the skin to water temperature gradient does exist (Hayward *et al.*, 1973). Rennie (1988) has commented that the small temperature gradient from skin to water justifies the assumption that skin temperature equals water temperature, with only a small margin of error. The measurement of skin temperature at four sites was therefore considered adequate for the purposes of the present study. Nevertheless, it is pertinent to point out that in order for heat to be transferred from the body to the water, as occurs even once skin temperatures approximate the water temperature, a temperature gradient must exist (Bullard and Rapp, 1970; Tipton, pers. comm.) Cognisance was taken of research that has reported a mean temperature difference between the water and the skin of 2.8°C in 10°C water (Hayward *et al.*, 1977), and of between 1.5°C to 3°C

in 27°C and 10°C water (Cooper *et al.*, 1976). At a water temperature of 22°C, Bullard and Rapp (1970) found a skin to water temperature differential of only 0.45°C, whereas Witherspoon *et al.* (1971) reported a 1°C to 2°C difference under similar conditions. Over a water temperature range of 28°C to 33°C, Hanna and Hong (1972) reported skin to water temperature differences of less than 0.2°C.

Heat Loss Calculations

It should be pointed out that without facilities for direct calorimetry, the determination of body heat loss during water immersion is not entirely accurate (Merrill-Smith and Hanna, 1975; Hong, 1984; Shiraki *et al.*, 1986). This is mainly because of problems associated with the calculation of the T_b of subjects in water. Comparisons of ΔT_b values obtained from direct calorimetry and those computed from ΔT_{re} and ΔT_{sk} during cooling in water have revealed that the latter may either overestimate or underestimate T_b, depending on the degree of cooling (Hong, 1984). Based upon the observations of Nadel *et al.* (1974) that, after 7 to 10min of immersion in still water, resting heat loss as measured by indirect calorimetry was only 22% of that measured with heat flow discs, Merrill-Smith and Hanna (1975) suggested that a longer period of immersion was required to achieve steady heat flow from cooling peripheral tissues. Nevertheless, it is acknowledged that in the absence of facilities for direct calorimetry, values obtained by estimation provide acceptable approximations (Hong, 1984; Shiraki *et al.*, 1986), and as such this method is commonly used over

varying time periods during studies of CWI (Rennie *et al.*, 1962; Veicsteinas *et al.*, 1982; Park *et al.*, 1983; Shiraki *et al.*, 1986; Choi *et al.*, 1988). In the present study, where body heat loss was used to regulate the immersion time around the amount of time necessary to cool peripheral tissues without a drop in core temperature, an approximation, calculated for each subject in the same way, was deemed acceptable.

Based upon a review of the literature, the change in body heat stores (*S*) was calculated as follows:

$$S\ (W.m^{-2}) = \frac{BM \times 0.83 \times \Delta T_b}{BSA}$$

where BM is body mass (kg), 0.83 is the specific heat of the body ($kcal.°C^{-1}.kg^{-1}$) and BSA is body surface area (m^2) (Wilkerson *et al.*, 1972; Kollias *et al.*, 1974; Choi *et al.*, 1988).

Metabolic heat production (*M*) was calculated as follows, according to the method employed by Choi *et al.* (1988):

$$M\ (kcal.m^{-2}.h^{-1}) = 4.83\ (kcal.l^{-1}) \times \dot{V}O2\ (l.m^{-2}.h^{-1})$$

where oxygen consumption is converted to standard temperature and pressure, dry (STPD). Skin heat loss was assumed to equal metabolic heat production (*M*) minus respiratory heat loss (0.08*M*) (Merrill-Smith and Hanna, 1975; Veicsteinas *et al.*, 1982; Park *et al.*, 1983; Choi *et al.*, 1988). Where the T_b changed during

water immersion the change in body heat store was added to $0.92M$ in estimating overall skin heat loss (H_{sk}) (Rennie *et al.*, 1962; Merrill-Smith and Hanna, 1975; Veicsteinas *et al.*, 1982; Park *et al.*, 1983; Choi *et al.*, 1988).

SUBMAXIMAL TREADMILL TESTING

The aim of this research was the assessment of physiological and psychophysical responses to dry exercise immediately following various degrees of body cooling. The exercise that was performed was a 30min submaximal treadmill run at a speed that would elicit an energy cost that was 50% of each individual's previously determined $\dot{V}O_2$ max. A series of three such runs, preceded by differing degrees of body cooling, was completed by each subject. These were performed in random order. The pre-exercise immersion profiles are described below.

During each of the treadmill sessions, physiological and psychophysical data were collected every 3min using technology identical to that used for $\dot{V}O_2$ max testing. In addition, core and skin temperatures were recorded, as were ratings of PTS. Furthermore, a more detailed evaluation of effort sense was obtained. Using the 15-point Borg scale discussed earlier, subjects were asked to indicate a local rating pertaining to feelings or sensations of strain in the exercising muscles and joints, a central rating that related to the cardiopulmonary system, and an overall, general rating of exertion. For the overall rating, subjects were told to integrate their local and central feelings with whatever weightings they deemed appropriate

(Pandolf, 1982). Body mass was measured before each run.

Control Condition

This required no prior immersion. Fifteen minutes before the end of the 60min period of supervised rest, the subject changed into a swim suit and running shoes and baseline data were collected for 10min prior to exercising on the treadmill.

CWI: Peripheral Cooling

After 60min of supervised rest and the collection of 10min pre-immersion baseline data wearing only a swim suit, the subject entered the water tank at 20°C. Physiological and psychophysical data were collected during the immersion. Subjects left the tank when a skin heat loss of 100kcal.m^{-2}.h^{-1} (as calculated manually) was attained without an apparent drop in core temperature. The duration of the immersion was recorded and the subject moved immediately to the laboratory for the completion of the treadmill run.

CWI: Central Cooling

After 60min of supervised rest and the collection of 10min of pre-immersion baseline data wearing only a swim suit, the subject entered the water tank at 20°C. Physiological and psychophysical data were collected during the immersion. The subject left the tank when core temperature had dropped 1°C below the pre-immersion baseline value. This was accepted as constituting both peripheral and central cooling, and subjects in this state were referred to as hypothermic. The duration of

the immersion was recorded and the subject moved immediately to the laboratory for the completion of the treadmill run.

The Mechanics of Locomotion

Earlier in this thesis it was proposed that even where economy may be adversely affected by prior CWI, cold-induced changes in the mechanics of locomotion may increase the amount of work done at a given treadmill speed, such that efficiency, defined simply as work done divided by energy expended (Cavanagh and Kram, 1985), would not be significantly altered. Economy has been defined as the submaximal oxygen consumption required to perform a given task (Wilcox and Bulbulian, 1983; Goslin, 1985; Williams and Cavanagh, 1987; Williams *et al.*, 1991; Svedenhag and Sjödin, 1994). Goslin (1985) has pointed out, however, that quantifying the amount of work done during treadmill locomotion is technically complex and expensive. In the calculation of net efficiency where the denominator is defined as the "energy expended above that at rest", the power output of an individual on the treadmill is reliant primarily upon the velocity of locomotion. Since, in the present study, the velocity of locomotion from one experimental session to another was standardised at a level that would elicit an energy expenditure 50% of the individual's $\dot{V}O_2$ max without prior CWI, the subtleties of the above postulate would not be evident. The numerator, calculated in this way, would not change from one condition to another. Were submaximal $\dot{V}O_2$ to increase, efficiency would automatically be reduced. While it was beyond the scope of this study to include a detailed study of locomotory mechanics

as they related to the efficiency of performance, it was decided to include basic assessment of the mechanics of locomotion in an attempt to determine whether this was in fact affected by CWI. This provided an indirect assessment of the amount of mechanical work done at a given speed on the treadmill. Cadence was counted visually using a hand-held counter over 10s intervals during each data collection period for the duration of the treadmill exercise. From these data the subject's stride lengths were calculated: treadmill speed was converted from $km.h^{-1}$ to $m.min^{-1}$ to derive the distance covered by each subject in one minute. Cadence ($steps.min^{-1}$) was divided into this to produce mean step length (m) and multiplied by 2 to provide mean stride length.

STATISTICAL ANALYSES

Statistical treatment of the data involved the use of single variable statistics to describe each set of data. Student's related t-tests were used to test for significant differences between the environmental conditions measured at each data collection session and the independent t-tests for differences in morphological criteria and immersion times in the water tank between males and females.

Multifactor, repeated measures analysis of variance (ANOVA) was used to determine if the factors "sex" (male and female) or "exposure" (control, peripheral cooling and central cooling) had significant effects on collected and derived variables. Where the ANOVAs revealed significant effects, Tukey's critical

difference was calculated and used to locate significant differences between means.

The 0.05 level of significance was selected for all statistical procedures in order to reduce the possibility of committing a Type I error, without unduly increasing the probability of a Type II error.

CHAPTER 4

RESULTS AND DISCUSSION

SUBJECT CHARACTERISTICS

Subject participation in this research was dependent upon a strict range of morphological and physiological criteria. Of the 47 individuals who initially volunteered as subjects, 15 were rejected on the grounds of complete morphological incompatibility. In order to match the sexes as closely as possible following the administration of the $\dot{V}O_2$ max test, 26 of the remaining volunteers, divided equally between males and females, were chosen as subjects.

The physical characteristics of the subjects appear in Table II. Significant differences between the sexes existed only for body surface area and for the triceps, forearm, thigh and calf skinfolds, with females significantly higher in each of the latter (Figure 2). Body surface area was not, however, a selection criterion; there was no significant difference in the body surface area-to-weight ratio.

The morphological and $\dot{V}O_2$ max data collected on the male subjects compare favourably with those of other South African and Canadian students of the same age (Ross and Marfell-Jones, 1982; du Toit et al., 1985; Rorke, 1988; Manley, 1989; Manley et al., 1990; Manley and Hayter, 1992). The $\dot{V}O_2$ max value of the present female subjects reflected that of those who have been described

Table II: Summary of physical and physiological characteristics of 13 male and 13 female subjects (mean ± standard deviation).

	Male	Female
Age (yr)	21.1 ± 1.2	20.9 ± 1.9
Stature (mm)	1754.0 ± 42.0	1727.0 ± 34.0
Mass (kg)	72.2 ± 4.7	69.1 ± 3.2
% fat	10.3 ± 2.5	11.9 ± 1.6
% muscle	44.4 ± 2.8	43.1 ± 2.6
Sum of SF (mm)	69.5 ± 20.6	81.9 ± 12.5
Triceps SF (mm)	9.5 ± 3.5	12.6 ± 3.2
Biceps SF (mm)	4.8 ± 1.7	5.9 ± 1.6
Forearm SF (mm)	5.1 ± 1.0	6.3 ± 1.6
Subscapular SF (mm)	9.6 ± 4.1	9.6 ± 2.1
Abdominal SF (mm)	10.8 ± 5.5	11.0 ± 3.3
Suprailliac SF (mm)	7.2 ± 2.5	8.3 ± 2.3
Thigh SF (mm)	13.7 ± 4.0	16.9 ± 2.8
Calf SF (mm)	8.8 ± 2.9	11.1 ± 2.3
BSA (m^2)	1.9 ± 0.1	1.8 ± 0.1
BSA:Mass ratio	38.0 ± 1.2	38.4 ± 0.8
$\dot{V}O_2max$ ($ml.kg^{-1}.min^{-1}$)	55.7 ± 5.7	53.2 ± 6.1

Where SF = Skinfold fat measurement

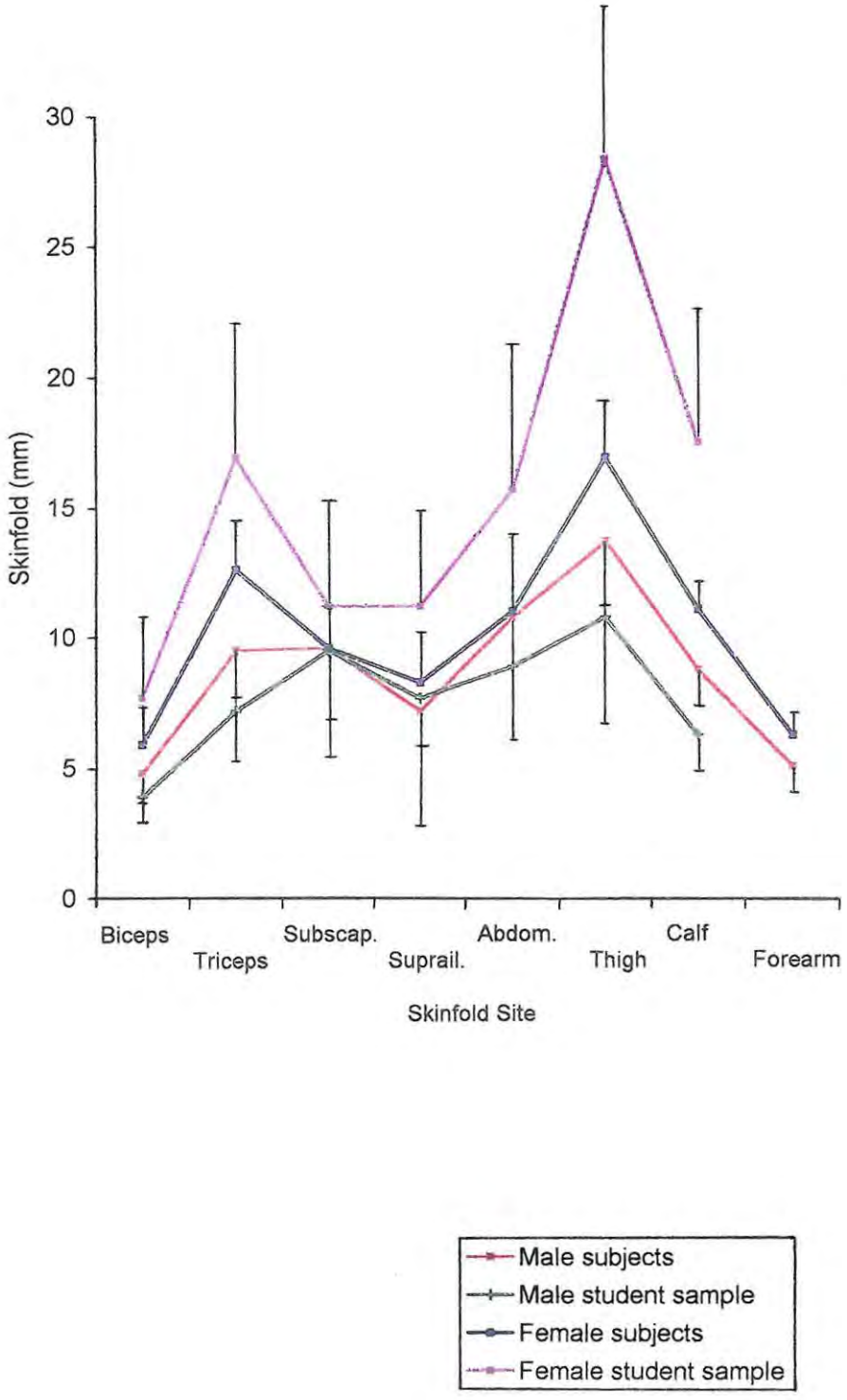


Figure 2: Skinfold profiles of male and female subjects compared to those of South African student populations of the same age.

elsewhere (LeBrun *et al.*, 1995) as "trained" female athletes. In other words, equal to or greater than 50ml.kg^{-1}.min^{-1}. Figure 2 illustrates the skinfold profile of male and female subjects in this study compared to a South African student population of the same age (n = 20 for each sex) (Manley, unpublished data).

A questionnaire concerning the gynecological history of the female volunteers in this research revealed that three of the 13 final subjects (23%) were currently taking oral contraceptives. None of the women was taking estrogen supplements at the time of testing and all reported menstrual cycles which fell within normal limits.

ENVIRONMENTAL CONDITIONS

The mean environmental conditions that the subjects were exposed to during each of the separate testing sessions are presented in Table III. There were no significant differences in any of these variables from one condition to the next. As was to be expected, a significant difference did exist between the central and peripheral conditions for immersion time. Other than water temperatures, the given values reflect ambient conditions in the Work Physiology Laboratory at the Department of Human Movement Studies, Rhodes University, where each of the treadmill testing sessions occurred. In order to ensure cold water conditions in the water tank within the department, the data collection period extended from May to July (ie. through mid-winter).

Table III: Environmental conditions experienced by subjects during data collection (mean ± standard deviation).

	Control	Central	Peripheral
Temperature (°C)	15.7 ± 1.2	15.1 ± 1.0	15.2 ± 1.4
Barometric Pressure (mmHg)	718.2 ± 3.4	718.2 ± 3.4	720.8 ± 4.8
Relative humidity (%)	70.9 ± 5.4	70.0 ± 6.4	70.2 ± 5.2
Water temperature (°C)	-	19.6 ± 1.1	20.2 ± 0.6
Immersion time (min)	-	28.3 ± 9.5	6.7 ± 2.2

Where: Control = conditions during submaximal exercise without prior CWI.
Central = conditions during submaximal exercise following central cooling by CWI.
Peripheral = conditions during submaximal exercise following peripheral cooling by CWI.

RESULTS

Overall Cooling Effects

The overall effects of central and peripheral cooling upon performance were examined separately for males and females.

Males

Temperature data

Compared to the control run, males exhibited significantly lower rectal temperatures (T_{re}) for both cooling conditions (Figure 3(a)), with lower values for central than peripheral cooling (Table V), despite there having been no difference in immediate pre-run values between the latter (Table IV). Indeed, when

Table IV: Pre-run body heat store changes (S), metabolic heat production (M), skin heat loss (H_{sk}) and thermal variables for male and female subjects without prior CWI (control) and during central and peripheral cooling in cold water (mean ± standard deviation).

	Male	Female	Sig.
Control			
S ($W.m^{-2}$)	5.2 ± 2.9	1.0 ± 2.0	*
M ($kcal.m^{-2}.h^{-1}$)	59.9 ± 12.7	49.0 ± 9.2	*
H_{sk} ($kcal.m^{-2}.h^{-1}$)	59.2 ± 12.8	49.9 ± 8.4	*
T_b (°C)	35.1 ± 0.4	35.1 ± 0.3	-
T_{re} (°C)	37.5 ± 0.2	37.6 ± 0.2	-
T_{sk} (°C)	30.2 ± 1.2	30.0 ± 0.8	-
PTS	2.7 ± 1.2	2.9 ± 0.8	-
Central cooling			
S ($W.m^{-2}$)	131.6 ± 20.7	106.8 ± 23.9	*
M ($kcal.m^{-2}.h^{-1}$)	172.4 ± 22.4	109.7 ± 75.4	*
H_{sk} ($kcal.m^{-2}.h^{-1}$)	271.7 ± 29.8	190.3 ± 75.5	*
T_b (°C)	31.1 ± 0.5	31.8 ± 0.6	-
T_{re} (°C)	36.6 ± 0.3	36.8 ± 0.2	-
T_{sk} (°C)	19.7 ± 1.4	21.4 ± 1.6	*
PTS	1.0 ± 0.8	1.0 ± 0.9	-
Peripheral cooling			
S ($W.m^{-2}$)	76.9 ± 24.2	75.2 ± 18.3	-
M ($kcal.m^{-2}.h^{-1}$)	79.9 ± 23.9	56.9 ± 19.9	*
H_{sk} ($kcal.m^{-2}.h^{-1}$)	139.7 ± 35.1	111.7 ± 26.9	*
T_b (°C)	32.4 ± 0.7	32.8 ± 0.7	-
T_{re} (°C)	37.5 ± 0.2	37.6 ± 0.1	-
T_{sk} (°C)	22.3 ± 2.3	23.3 ± 2.0	-
PTS	1.1 ± 1.0	1.0 ± 0.9	-

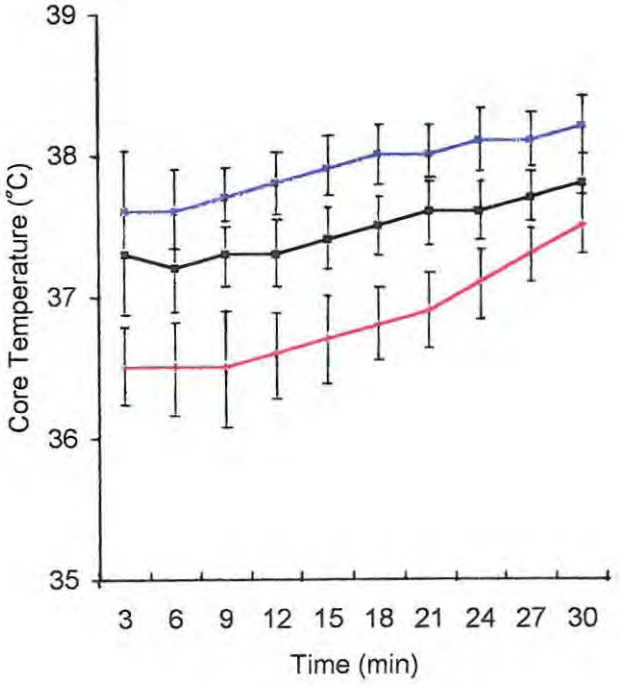
Where: * = Significant difference ($p<0.05$) between males and females.
n = 13 for males and females

Note: Values reflect variables measured during the last minute of baseline data collection (see Methods)

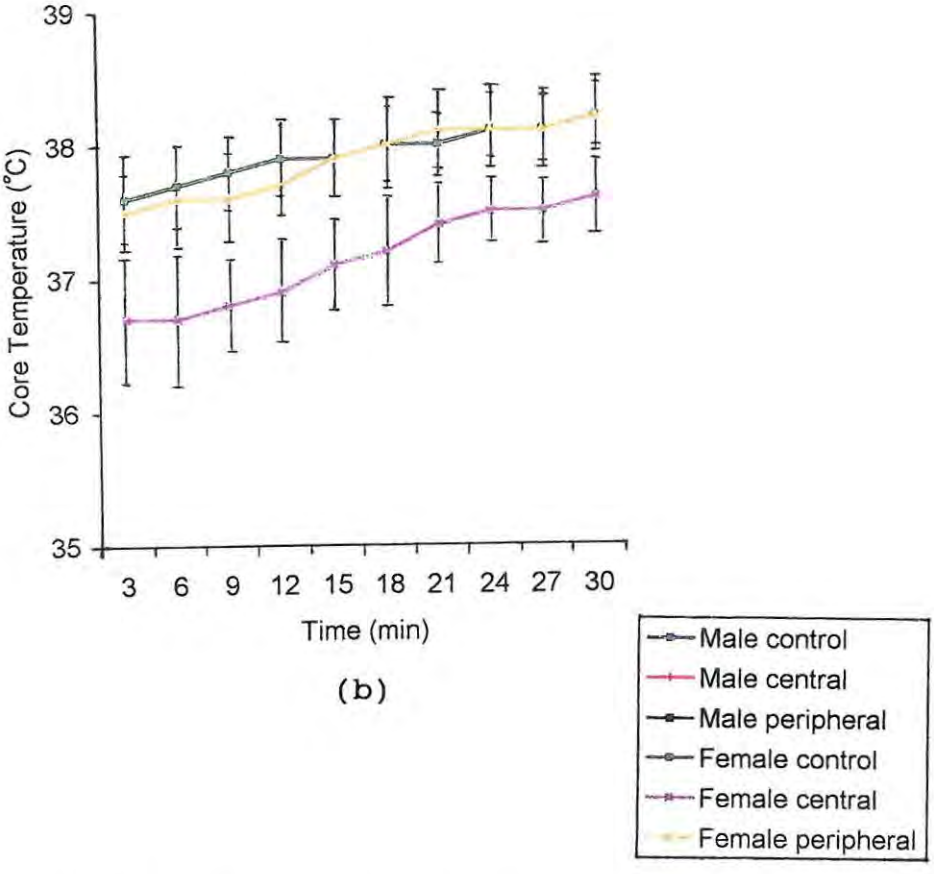
Table V: Physiological and psychophysical responses of male subjects to submaximal exercise without prior CWI (control) and following central and peripheral cooling during CWI (n = 13).

	Control	Central cooling	Peripheral cooling	Sig.
	$\bar{x}$ ± s.d.	$\bar{x}$ ± s.d.	$\bar{x}$ ± s.d.	
T_b (°C)	35.3 ± 0.6	33.7 ± 0.8	34.3 ± 0.8	✓ * #
T_{re} (°C)	37.9 ± 0.3	36.8 ± 0.5	37.5 ± 0.4	✓ * #
T_{sk} (°C)	30.0 ± 1.3	27.5 ± 1.6	27.9 ± 1.9	✓ * #
Chest temp. (°C)	31.9 ± 1.7	28.9 ± 1.5	29.2 ± 2.0	✓ * -
Leg temp. (°C)	29.9 ± 1.9	27.2 ± 2.4	27.6 ± 2.4	✓ * -
Arm temp. (°C)	26.8 ± 2.5	24.8 ± 1.4	25.4 ± 1.4	✓ * #
Hand temp. (°C)	24.9 ± 2.8	19.0 ± 1.7	21.9 ± 1.7	✓ * #
$\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)	30.8 ± 1.6	31.2 ± 3.2	31.5 ± 2.5	- - -
$\dot{V}CO_2$ ($l.min^{-1}$)	2.0 ± 0.3	1.9 ± 0.3	2.1 ± 0.3	- - #
$\dot{V}_E$ (BTPS) ($l.min^{-1}$)	64.8 ± 8.7	67.9 ± 9.5	65.5 ± 9.8	✓ - -
f ($br.min^{-1}$)	32.7 ± 3.9	33.5 ± 3.7	33.2 ± 4.2	- - -
Heart rate ($b.min^{-1}$)	139.1 ± 7.3	134.7 ±17.5	145.0 ±16.4	✓ * #
PTS	5.4 ± 1.4	3.8 ± 1.8	4.6 ± 1.5	✓ * #
RPE Overall	10.1 ± 1.9	10.0 ± 1.9	9.4 ± 1.9	- * #
RPE Central	9.2 ± 1.9	9.7 ± 1.9	9.2 ± 1.9	- - -
RPE Local	10.9 ± 2.4	10.3 ± 2.4	10.7 ± 2.4	✓ - -
Cadence ($steps.min^{-1}$)	80.4 ± 3.6	82.2 ± 4.0	81.6 ± 3.6	- - -
Stride length (m)	1.9 ± 0.5	1.9 ± 0.6	1.9 ± 0.4	- - -

Where: Sig. = Significant differences ($p<0.05$):
✓ = Control *vs* central
* = Control *vs* peripheral
# = Central *vs* peripheral
- = No difference



(a)



(b)

Figure 3: The effects of control, central cooling and peripheral cooling upon core temperature during a 30min submaximal treadmill run for male (a) and female (b) subjects.

examined in the light of Table IV, mean T_{re} over the 30min period showed little deviation from immediate pre-run values, with control and central conditions exhibiting 0.4°C and 0.2°C rises respectively and the peripheral condition none at all. Significant heating of the core over the exercise period did occur. The change in T_{re} over time, as represented by a comparison of pre-run and 30min values (control: 0.7°C; central: 0.9°C; peripheral: 0.3°C), was significant for each of the conditions. Mean skin temperatures (T_{sk}) were significantly different across the conditions, as was T_b (Figure 4). The lowest T_{sk} was produced by the central condition; peripheral T_{sk} values were lower than control figures. Of the individual sites measured, hand and arm temperatures were significantly lower for central than peripheral cooling; leg and chest temperatures were significantly different across the conditions. The increase in T_{sk} at the final data collection point compared to immediate pre-run figures was significant for the central and peripheral conditions, but not the control condition (control: 0.6°C; central: 9.6°C; peripheral: 7.7°C).

Cardiorespiratory data

According to the mean values presented in Table V, $\dot{V}O_2$ for males was unchanged, irrespective of the degree of cooling. Figure 5(a), however, indicates that, specifically following central cooling, initially elevated responses were camouflaged by the overall mean figure. The $\dot{V}O_2$ over the control run exhibited little fluctuation around the mean of 30.8 ± 1.6ml.kg^{-1}.min^{-1}. At

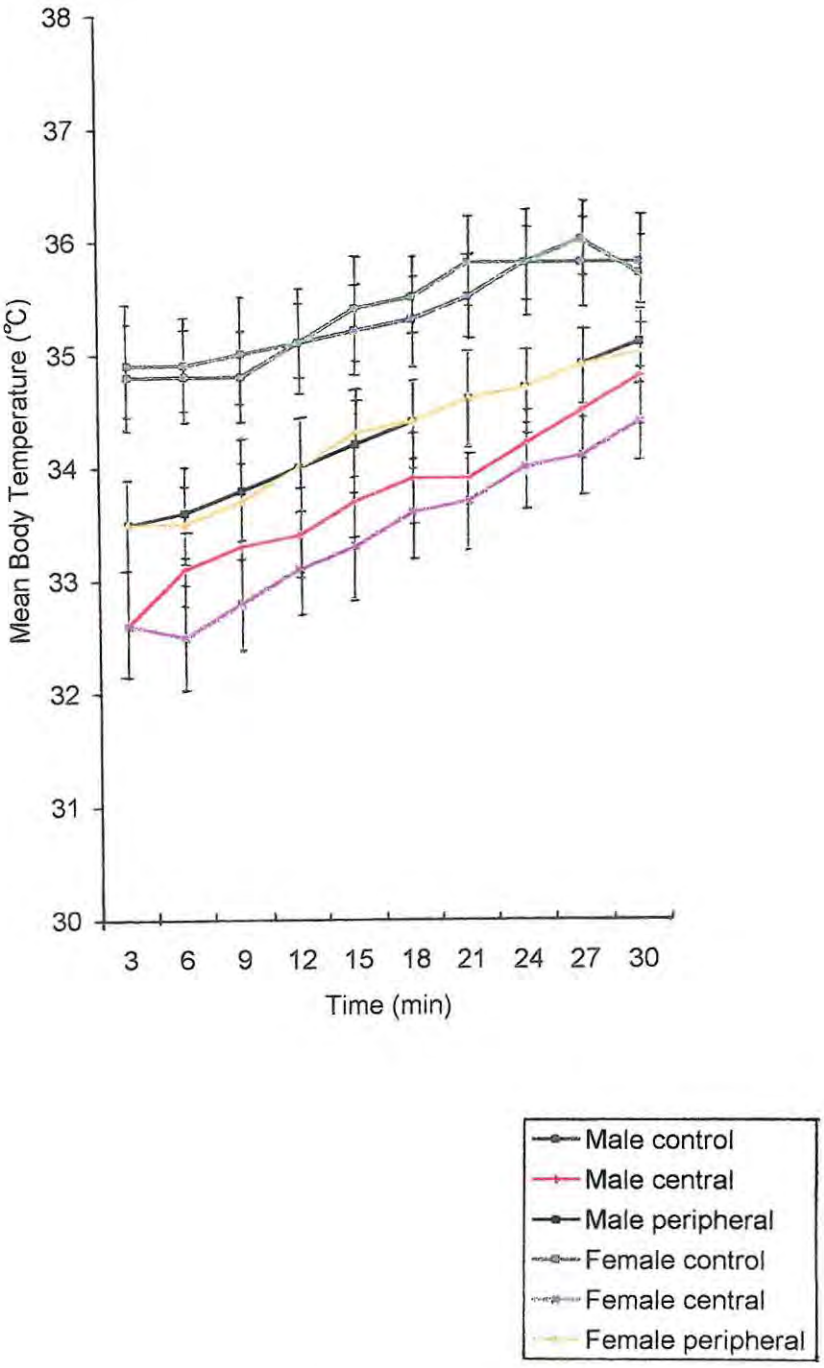
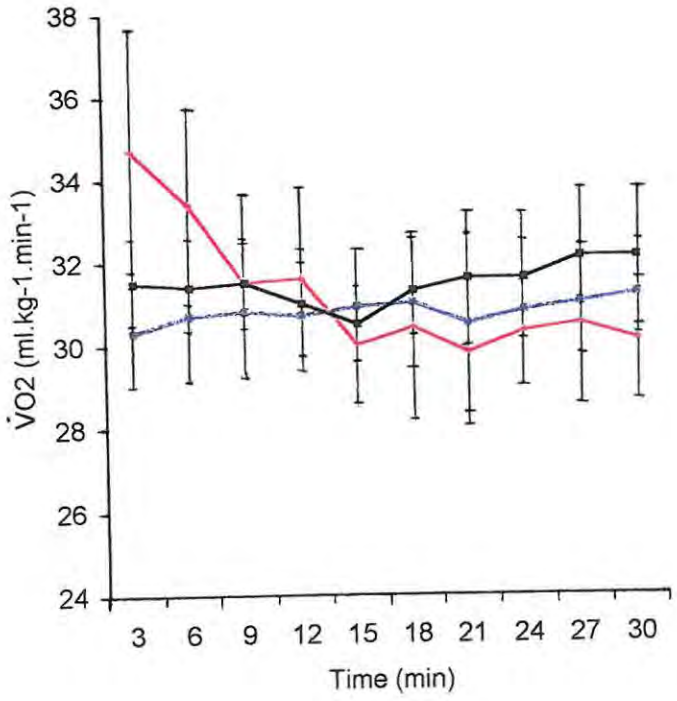
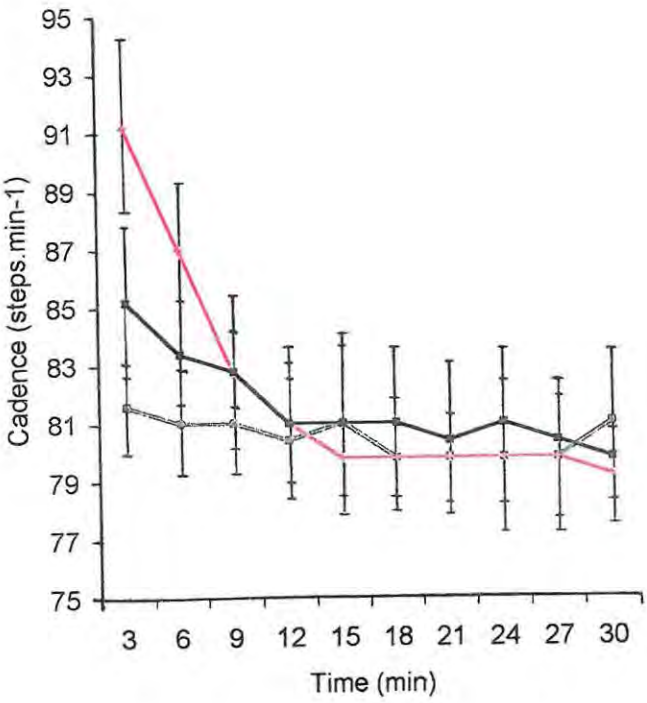


Figure 4: Male and female mean body temperature responses to a 30min submaximal treadmill run during control, peripheral cooling and central cooling conditions.



(a)



(b)

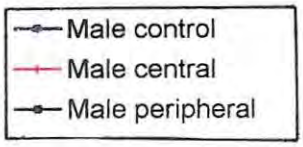


Figure 5: The effects of control, central cooling and peripheral cooling conditions upon oxygen consumption ($\dot{V}O_2$) (a) and cadence (b) during a 30min submaximal treadmill run for male subjects.

the 3min mark, the central value (34.6ml.kg^{-1}.min^{-1}) was 12.4% higher than the corresponding control value, whereas the same peripheral value was only 3.8% higher. By 30min, however, all of the $\dot{V}O_2$ values were within 4% of each other. Examination of Figure 5(a) suggests the possibility of a levelling off in the responses at 15min which would apply in particular to the central condition.

The somewhat biphasic nature of the response identified for central $\dot{V}O_2$ was also evident for the same condition for male $\dot{V}CO_2$ and $\dot{V}_E$ (BTPS). Figure 6 illustrates the same levelling off of central $\dot{V}CO_2$ at 15min with an overall mean that was significantly less than for peripheral cooling, although not different to the control mean (Table V). Minute ventilation means were significantly different only between the control and central conditions. Breathing frequency (f) did not demonstrate the same biphasic pattern in response, nor were any significant differences revealed across the conditions (Figure 7). Mean heart rates, however, were significantly altered from one condition to the next, with peripheral cooling producing the highest value and central cooling the lowest (Table V).

Psychophysical data

Peripheral and central cooling produced significant reductions in the perceptions of thermal sensation (PTS) (Figure 8(a)). All subjects rated PTS lower following central compared to peripheral cooling.

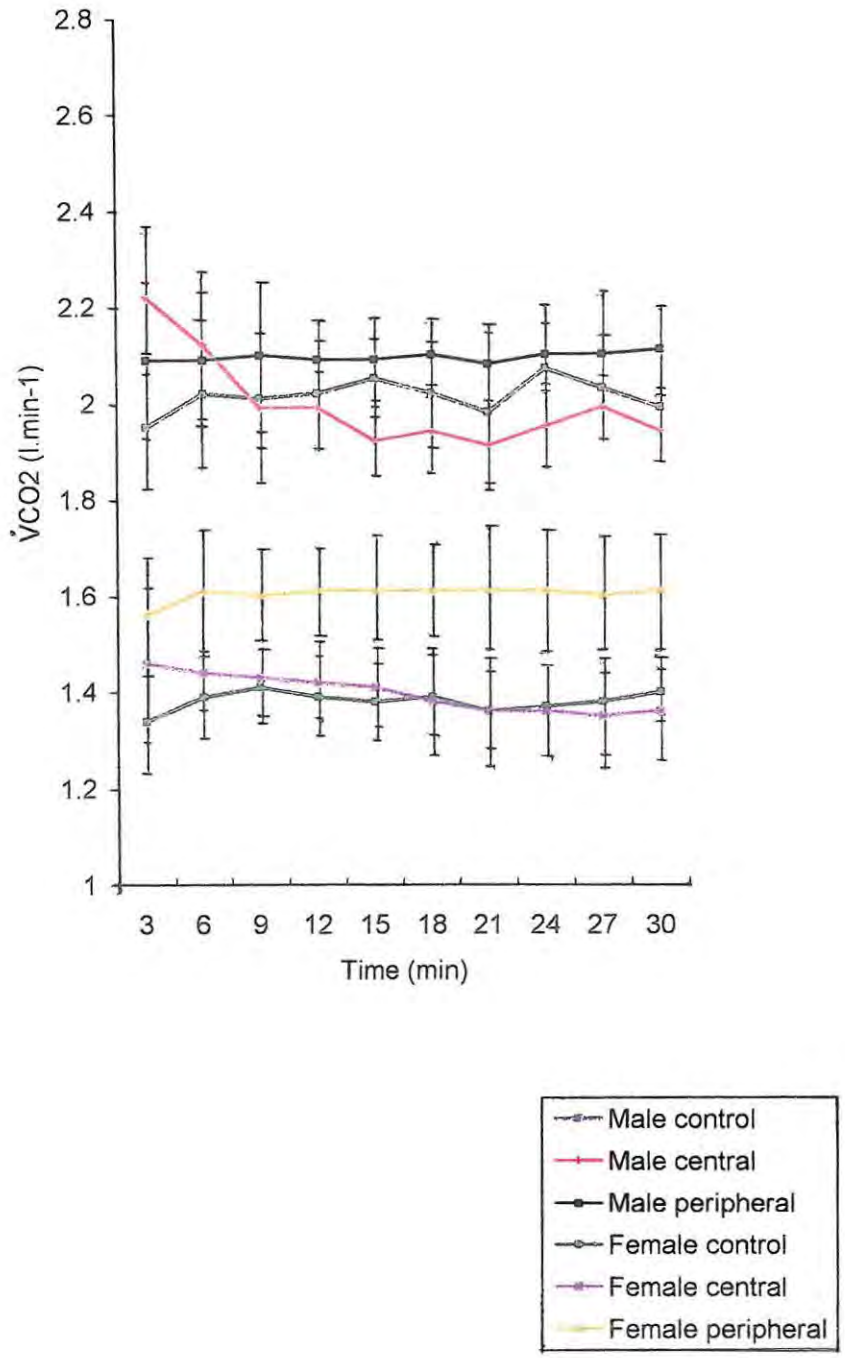


Figure 6: Male and female $\dot{V}CO_2$ responses to a 30min submaximal treadmill run during control, peripheral cooling and central cooling conditions.

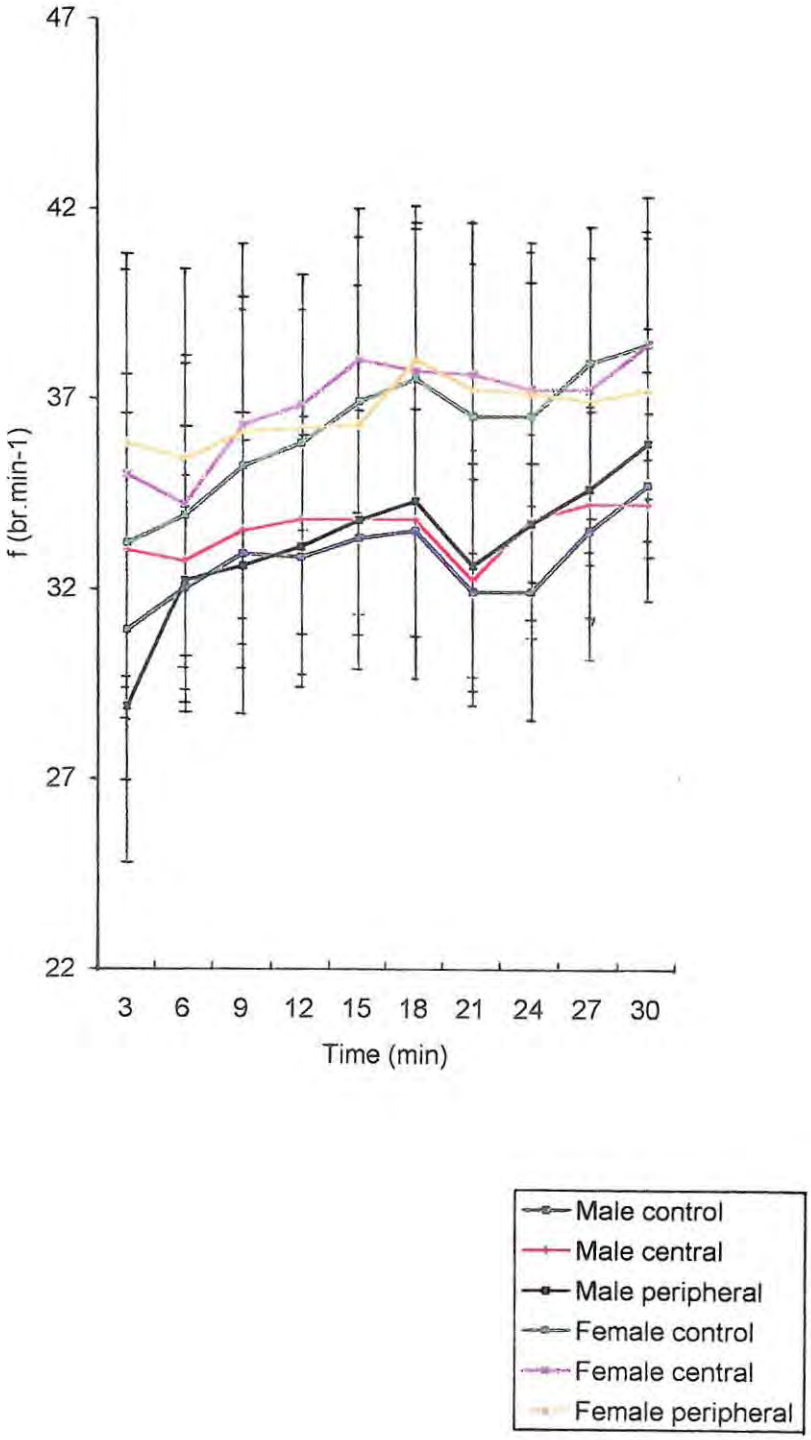
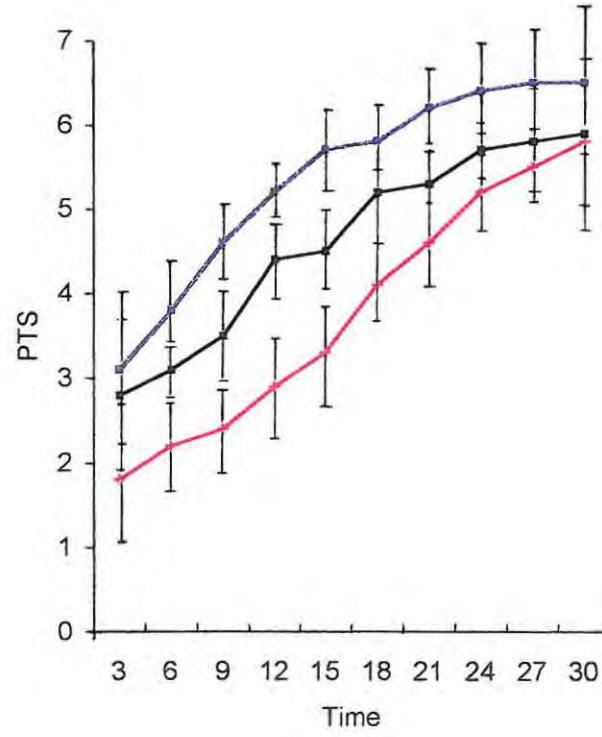
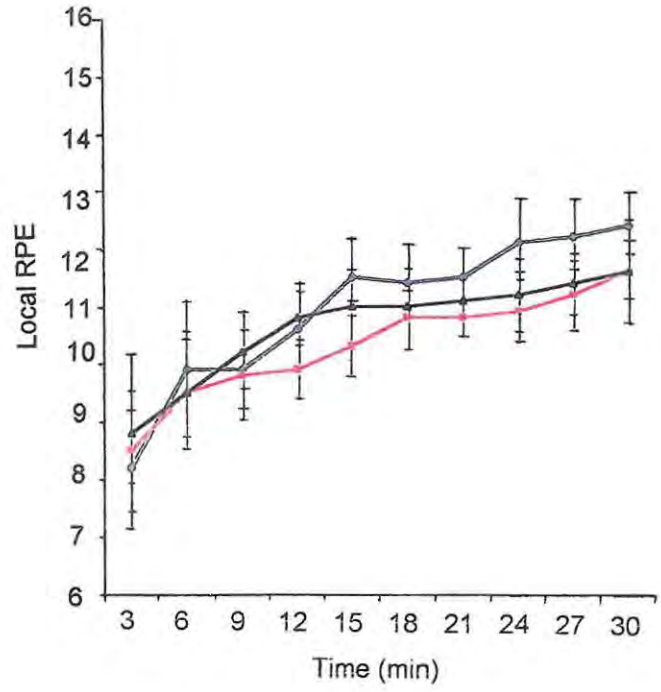


Figure 7: Male and female breathing frequency responses to a 30min submaximal treadmill run during control, peripheral cooling and central cooling conditions.



(a)



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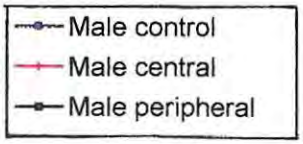


Figure 8: The effects of control, central cooling and peripheral cooling conditions upon perceived thermal sensation (PTS) (a) and local ratings of perceived exertion (RPE) (b) during a 30min submaximal treadmill run for male subjects.

Peripheral cooling resulted in a significantly lower overall RPE in comparison to control and central values, but no other differences were identified, whereas the only significant change for locally rated perceptions of exertion was a reduction for central compared to control ratings (Figure 8(b)). Central RPE, on the other hand, was not significantly altered by either degree of cooling.

No significant differences were evident in mean values for cadence largely as a function of a reduction in stepping rate in the central condition from 91.2steps.min^{-1} at 3min to 79.2steps.min^{-1} at the end collection point (Figure 5(b)). Identification of significant differences for stride length followed the same pattern as for cadence.

Females

Temperature data

The T_{re} for females was significantly lower for central than control and peripheral conditions (Figure 3(b)), but control and peripheral values were identical. Nevertheless, the significant increase in T_{re} from pre-run values to the 30min data collection point, whilst identical for the control and peripheral conditions (0.6°C), was greatest for the central condition (0.8°C). The same was true of T_{sk}, which increased by 6.3°C during the central condition (control: 1.5°C; peripheral: 5.2°C), but the mean value of which was significantly different across the conditions (Table VI). Of the individual temperature sites significant differences were demonstrated across all conditions, with the exception of

Table VI: Physiological and psychophysical responses of female subjects to submaximal exercise without prior CWI (control) and following central and peripheral cooling during CWI (n = 13).

	Control	Central cooling	Peripheral cooling	Sig.
	$\bar{x}$ ± s.d.	$\bar{x}$ ± s.d.	$\bar{x}$ ± s.d.	
T_b (°C)	35.4 ± 0.6	33.4 ± 0.9	34.3 ± 0.8	✓ * #
T_{re} (°C)	37.9 ± 0.4	37.2 ± 0.5	37.9 ± 0.3	✓ - #
T_{sk} (°C)	30.5 ± 1.2	25.9 ± 1.8	26.9 ± 1.9	✓ * #
Chest temp. (°C)	32.9 ± 1.0	27.5 ± 1.9	28.5 ± 2.6	✓ * #
Leg temp. (°C)	29.9 ± 1.9	25.4 ± 2.2	26.2 ± 1.7	✓ * #
Arm temp. (°C)	29.6 ± 2.3	24.3 ± 2.3	24.7 ± 2.4	✓ * -
Hand temp. (°C)	25.2 ± 3.5	18.5 ± 1.1	22.1 ± 2.9	✓ * #
VO_2 ($ml.kg^{-1}.min^{-1}$)	27.6 ± 2.6	28.2 ± 2.7	28.6 ± 3.3	- - -
VCO_2 ($l.min^{-1}$)	1.4 ± 0.2	1.4 ± 0.2	1.6 ± 0.4	- * #
V_E (BTPS) ($l.min^{-1}$)	45.7 ± 4.4	48.7 ± 5.9	51.5 ± 7.2	✓ * #
f ($br.min^{-1}$)	36.2 ± 4.7	36.8 ± 5.5	36.6 ± 5.1	- - -
Heart rate ($b.min^{-1}$)	157.7 ±23.7	143.5 ±20.5	151.7 ±16.7	✓ * #
PTS	5.3 ± 1.3	2.7 ± 1.5	4.8 ± 1.4	✓ * #
RPE Overall	9.2 ± 1.9	9.5 ± 1.2	8.7 ± 1.3	- * #
RPE Central	9.3 ± 1.8	9.5 ± 1.2	8.7 ± 1.3	- - #
RPE Local	9.4 ± 1.9	9.6 ± 1.2	8.9 ± 1.2	✓ - #
Cadence ($steps.min^{-1}$)	82.8 ± 4.8	85.2 ± 4.8	83.4 ± 4.8	✓ - #
Stride length (m)	1.9 ± 0.4	1.9 ± 0.3	1.9 ± 0.5	✓ - #

Where: Sig. = Significant differences ($p<0.05$):
✓ = Control *vs* central
* = Control *vs* peripheral
# = Central *vs* peripheral
- = No difference

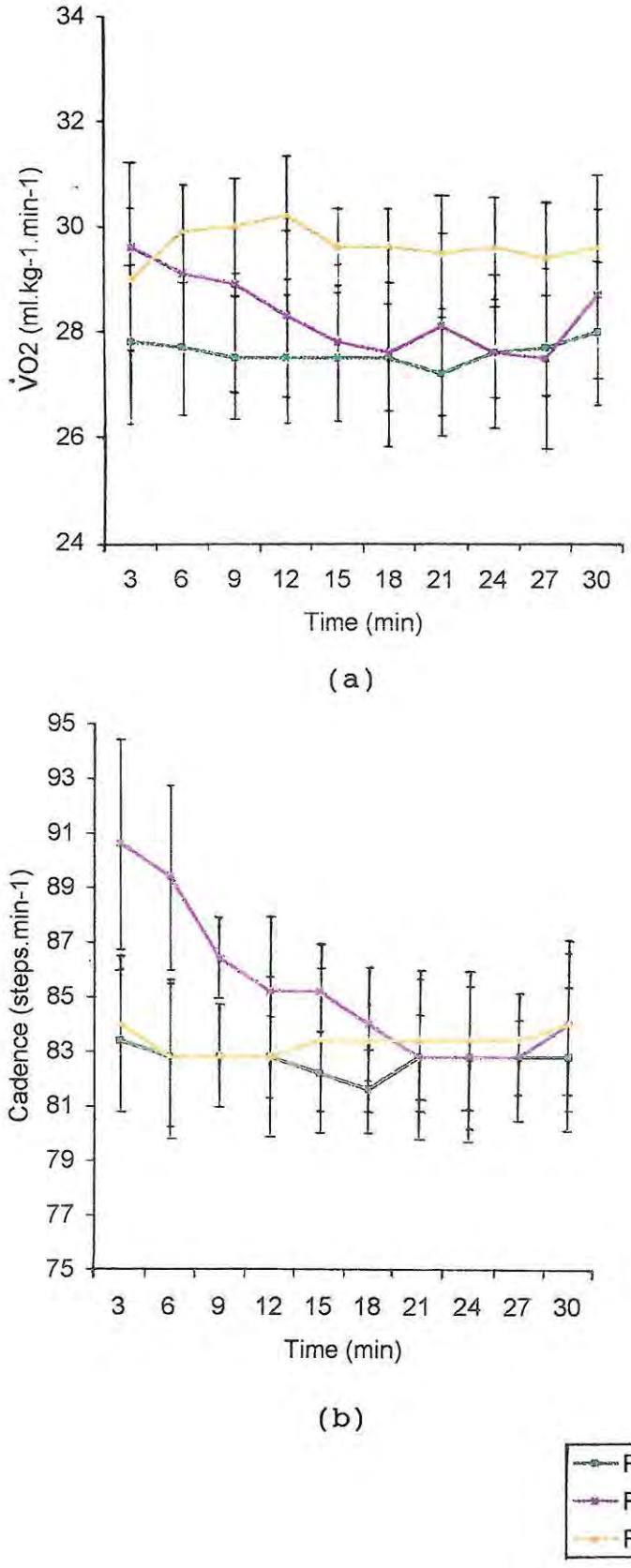


Figure 9: The effects of control, central cooling and peripheral cooling conditions upon oxygen consumption ($\dot{V}O_2$) (a) and cadence (b) during a 30min submaximal treadmill run for female subjects.

arm temperature, where central and peripheral values were the same. As was to be expected, given the above, T_b was lower for the central compared to the peripheral condition, and for both of these in comparison to the control value (Figure 4).

Cardiorespiratory data

The mean peripheral $\dot{V}O_2$ value for females was significantly higher than the control figure, but no other differences were revealed (Figure 9(a)). Mean data for control and central $\dot{V}CO_2$ were identical, whereas the peripheral mean was significantly higher than each of these. The mean $\dot{V}_E$ (BTPS) was significantly different for each of the conditions, whereas f was not at all (Table VI). Female heart rates were significantly reduced for both cooled states compared to the control run, with central values significantly lower than peripheral.

Psychophysical data

Both peripheral and central cooling produced significant reductions in the perceptions of thermal sensation for females (Figure 10(a)), this despite there being no difference in control and peripheral T_{re}. These subjects perceived the workload as less strenuous following peripheral cooling for overall, central and local RPE. In the case of overall RPE the reduction was significant when compared to both control and centrally cooled conditions; central and local RPE were significantly less than following central cooling, but not when contrasted with the control condition. The only difference between control and central conditions was identified for local ratings, which were

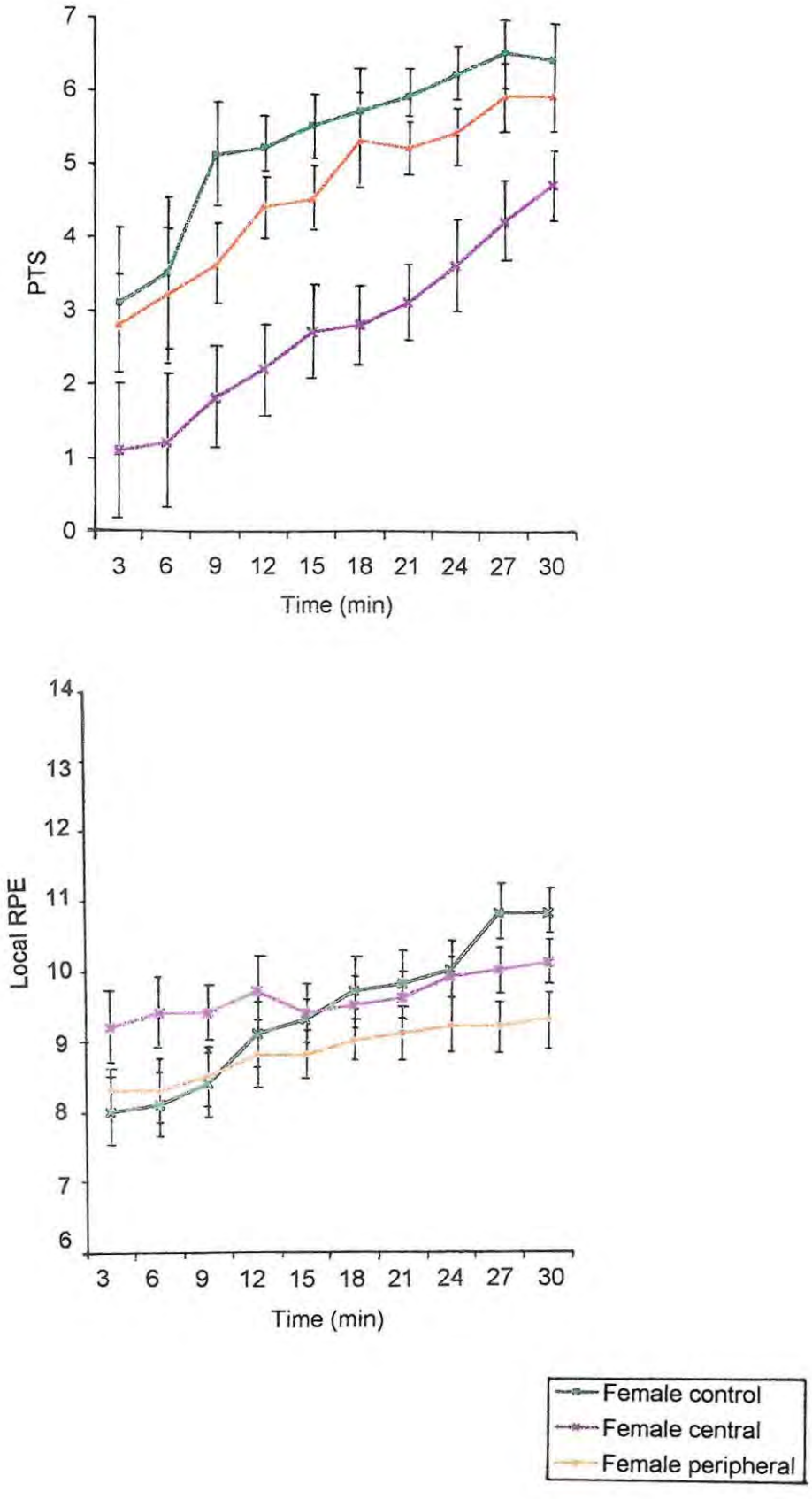


Figure 10: The effects of control, central cooling and peripheral cooling conditions upon perceived thermal sensation (PTS) (a) and local ratings of perceived exertion (RPE) (b) during a 30min submaximal treadmill run for female subjects.

significantly higher following central cooling (Figure 10(b)).

For cadence the differences between control and central conditions and central and peripheral conditions were significant (Figure 9(b)). Once again, identification of differences for stride length followed the same pattern as for cadence.

Male *versus* Female Responses

Experimental data were separated into control, central and peripheral cooling categories in order to assess the specific significance of sex for collected and derived variables in each condition.

Temperature data

With regard to skin temperatures under control conditions, significant differences between males and females were evident for three of the four sites measured. While leg temperature was significantly lower for females than males, chest and arm temperatures were significantly higher. Leg and chest temperatures were the only variables in the control condition to demonstrate significant interactions between factors; thus the temperature responses at these sites were qualitatively different for males and females at the different time intervals over 30min. Hand temperature was not significantly different between males and females. The T_{re} and T_b for the two groups were virtually identical (Table VII), in slight contrast to the findings of Grivel and Candas (1991), who found that, on average, men exhibited 0.3°C lower T_{re} than women. The T_{sk} was not different

Table VII: Physiological and psychophysical responses of male and female subjects to submaximal exercise without prior CWI (control) (mean ± standard deviation).

	Male	Female	Sig.
T_b (°C)	35.3 ± 0.6	35.4 ± 0.6	-
T_{re} (°C)	37.9 ± 0.3	37.9 ± 0.4	-
T_{sk} (°C)	30.0 ± 1.3	30.5 ± 1.2	-
Chest temperature (°C)	31.9 ± 1.7	32.9 ± 1.0	*
Leg temperature (°C)	29.9 ± 1.9	28.1 ± 1.5	*
Arm temperature (°C)	26.8 ± 2.5	29.6 ± 2.3	*
Hand temperature (°C)	24.9 ± 2.8	25.2 ± 3.5	-
$\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)	30.8 ± 1.6	27.6 ± 2.6	*
$\dot{V}CO_2$ ($l.min^{-1}$)	2.0 ± 0.3	1.4 ± 0.2	*
$\dot{V}_E$ (BTPS) ($l.min^{-1}$)	64.8 ± 8.7	45.7 ± 4.4	*
f ($br.min^{-1}$)	32.7 ± 3.9	36.2 ± 4.7	*
Heart rate ($b.min^{-1}$)	139.1 ± 7.3	157.7 ± 23.7	*
PTS	5.4 ± 1.4	5.3 ± 1.3	-
RPE Overall	10.1 ± 1.9	9.2 ± 1.9	-
RPE Central	9.2 ± 1.9	9.3 ± 1.8	-
RPE Local	10.9 ± 2.4	9.4 ± 1.9	*
Cadence ($steps.min^{-1}$)	80.4 ± 3.6	82.8 ± 4.8	-
Stride length (m)	1.9 ± 0.5	1.7 ± 0.4	-

Where: * = Significant difference ($p<0.05$)
n = 13 for males and females

Table VIII: Physiological and psychophysical responses of male and female subjects to submaximal exercise following central cooling during CWI (mean ± standard deviation).

	Male	Female	Sig.
T_b (°C)	33.7 ± 0.8	33.4 ± 0.9	-
T_{re} (°C)	36.8 ± 0.5	37.2 ± 0.5	*
T_{sk} (°C)	27.5 ± 1.6	25.9 ± 1.8	*
Chest temperature (°C)	28.9 ± 1.5	27.5 ± 1.9	*
Leg temperature (°C)	27.2 ± 2.4	25.4 ± 2.2	*
Arm temperature (°C)	24.8 ± 1.4	24.3 ± 2.3	-
Hand temperature (°C)	19.0 ± 1.7	18.5 ± 1.1	-
$\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)	31.2 ± 3.2	28.2 ± 2.7	*
$\dot{V}CO_2$ ($l.min^{-1}$)	1.9 ± 0.3	1.4 ± 0.2	*
$\dot{V}_E$ (BTPS) ($l.min^{-1}$)	67.9 ± 9.5	48.7 ± 5.9	*
f ($br.min^{-1}$)	33.5 ± 3.7	36.8 ± 5.5	*
Heart rate ($b.min^{-1}$)	134.7 ± 17.5	143.5 ± 20.5	-
PTS	3.8 ± 1.8	2.7 ± 1.5	*
RPE Overall	10.0 ± 1.9	9.5 ± 1.2	-
RPE Central	9.7 ± 1.9	9.5 ± 1.2	-
RPE Local	10.3 ± 2.4	9.6 ± 1.2	-
Cadence ($steps.min^{-1}$)	82.2 ± 4.0	85.2 ± 4.8	*
Stride length (m)	1.9 ± 0.6	1.7 ± 0.3	*

Where: * = Significant difference ($p<0.05$)
n = 13 for males and females

Table IX: Physiological and psychophysical responses of male and female subjects to submaximal exercise following peripheral cooling during CWI (mean ± standard deviation).

	Male	Female	Sig.
T_b (°C)	34.3 ± 0.7	34.3 ± 0.8	-
T_{re} (°C)	37.5 ± 0.4	37.9 ± 0.3	*
T_{sk} (°C)	27.9 ± 1.9	26.9 ± 1.9	-
Chest temperature (°C)	29.2 ± 2.0	28.5 ± 2.6	-
Leg temperature (°C)	27.6 ± 2.4	26.2 ± 1.7	*
Arm temperature (°C)	25.4 ± 1.4	24.7 ± 2.4	-
Hand temperature (°C)	21.9 ± 1.7	22.1 ± 2.9	-
$\dot{V}O_2$ ($ml.kg^{-1}.min^{-1}$)	31.5 ± 2.5	28.6 ± 3.3	*
$\dot{V}CO_2$ ($l.min^{-1}$)	2.1 ± 0.3	1.6 ± 0.4	*
$\dot{V}_E$ (BTPS) ($l.min^{-1}$)	65.5 ± 9.8	51.5 ± 7.2	*
f ($br.min^{-1}$)	33.2 ± 4.2	36.6 ± 5.1	*
Heart rate ($b.min^{-1}$)	145.0 ±16.4	151.7 ±16.7	-
PTS	4.6 ± 1.5	4.8 ± 1.4	-
RPE Overall	9.4 ± 1.9	8.7 ± 1.3	-
RPE Central	9.2 ± 1.9	8.7 ± 1.3	-
RPE Local	10.7 ± 2.4	8.9 ± 1.2	*
Cadence ($steps.min^{-1}$)	81.6 ± 3.6	83.4 ± 4.8	-
Stride length (m)	1.9 ± 0.4	1.7 ± 0.5	-

Where: * = Significant difference ($p<0.05$)
n = 13 for males and females

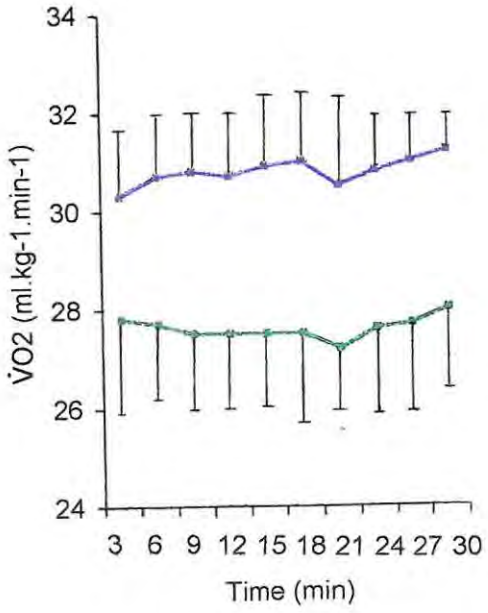
for the groups.

Performance of the same submaximal exercise following central cooling (Table VIII) produced significant differences between males and females for only two of the four skin temperature sites: the chest and leg sites. This was sufficient, however, to result in a significantly lower overall T_{sk} for females. Core temperature, on the other hand, was significantly higher for females than males. This, coupled with the T_{sk} effect, resulted in no significant difference for T_b. Of the temperature related data examined, all except chest temperature demonstrated qualitative differences for females compared to males.

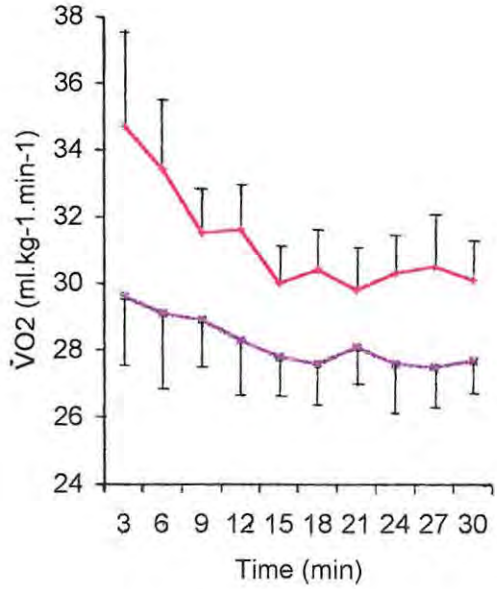
Of the skin temperature sites measured following peripheral cooling, only leg temperature was significantly different, being lower for females than males. Core temperature, on the other hand, was both quantitatively and qualitatively different, with females recording higher values than males (Table IX). Mean body temperature, however, was not significantly different (Figure 4), nor was T_{sk}.

Cardiorespiratory data

During the 30min submaximal run performed under control conditions the female subjects produced significantly lower values for $\dot{V}O_2$ (Figure 11), $\dot{V}CO_2$ and $\dot{V}_E$ (BTPS) than males, with the mean $\dot{V}O_2$ for males and females equating to 55.2% and 52% of their respective $\dot{V}O_2$ max values. Breathing frequency was significantly higher for females, as was heart rate.



(a)



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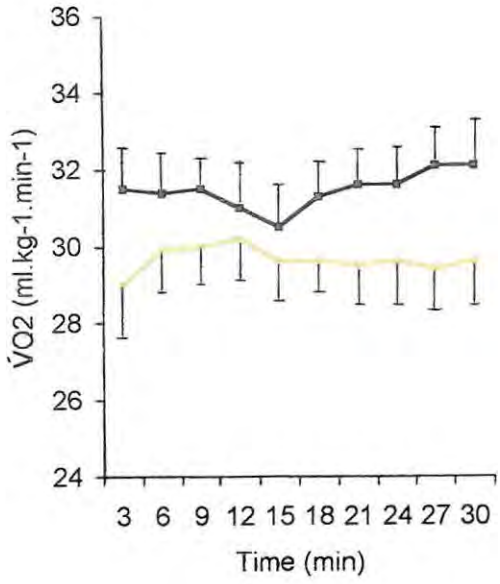

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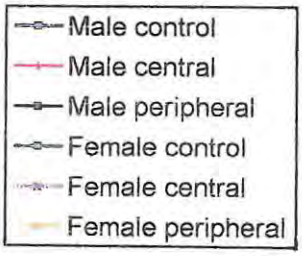


Figure 11: Comparison of male and female oxygen consumption ($\dot{V}O_2$) responses during a 30min submaximal treadmill run under control (a), central cooling (b) and peripheral cooling (c) conditions.

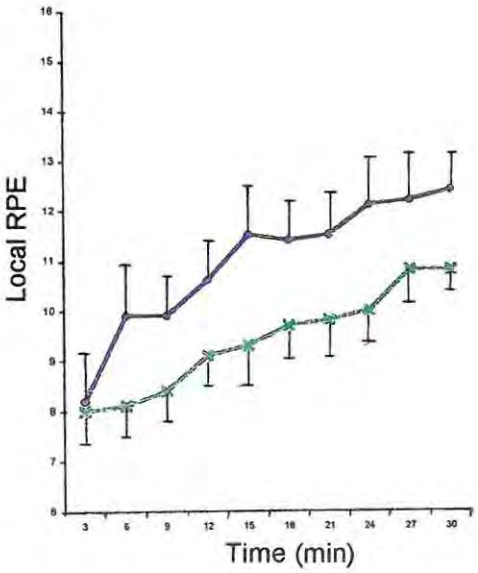
The respiratory and ventilatory data collected following central cooling demonstrated the same trends that were evident under control conditions: $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ (BTPS) were significantly lower for females than males, f was significantly higher. Indeed the difference between male and female values, when expressed as a percentage, remained within 4% of the difference calculated for control conditions for each of these parameters. The mean heart rates calculated after central cooling were not significantly different.

The significant differences evident under control conditions were again repeated for peripheral cooling: $\dot{V}O_2$, $\dot{V}CO_2$ and $\dot{V}_E$ (BTPS) were significantly lower for females than males, f was significantly higher (Table IX). Heart rate was not significantly different between males and females following peripheral cooling.

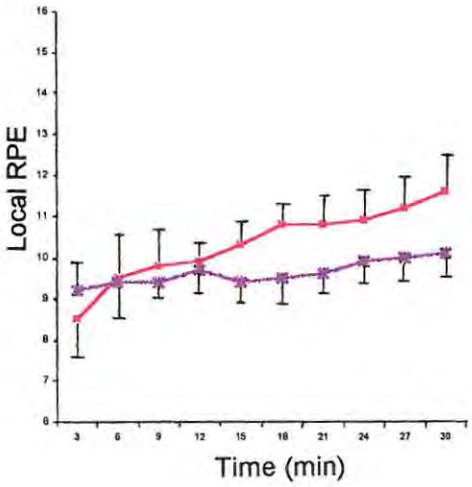
Psychophysical data

With the exception of local RPE, which was significantly higher for males than females (Figure 12(a)), ratings of overall and central perceived exertion and of thermal sensation (Figure 13(a)) were not significantly different under control conditions. Nor was cadence significantly different between males and females. This can be explained in terms of slower treadmill speeds for female subjects.

Central cooling had the effect of increasing the cadence of the females to the extent that it was significantly higher than that of the males and responses recorded over 30min demonstrated a



(a)



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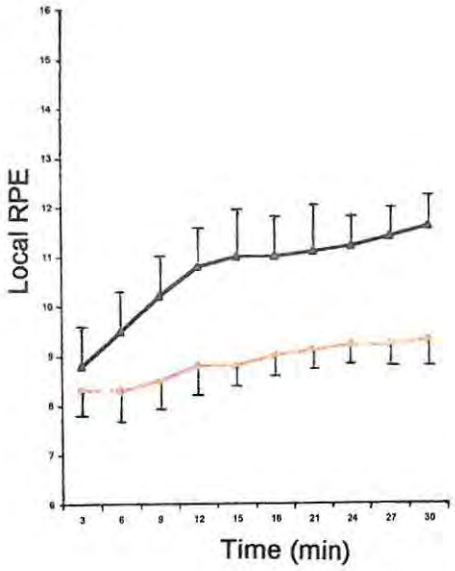

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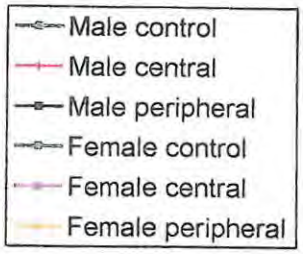

Figure 12: Comparison of male and female local perceived exertion (RPE) responses during a 30min submaximal treadmill run under control (a), central cooling (b) and peripheral cooling (c) conditions.

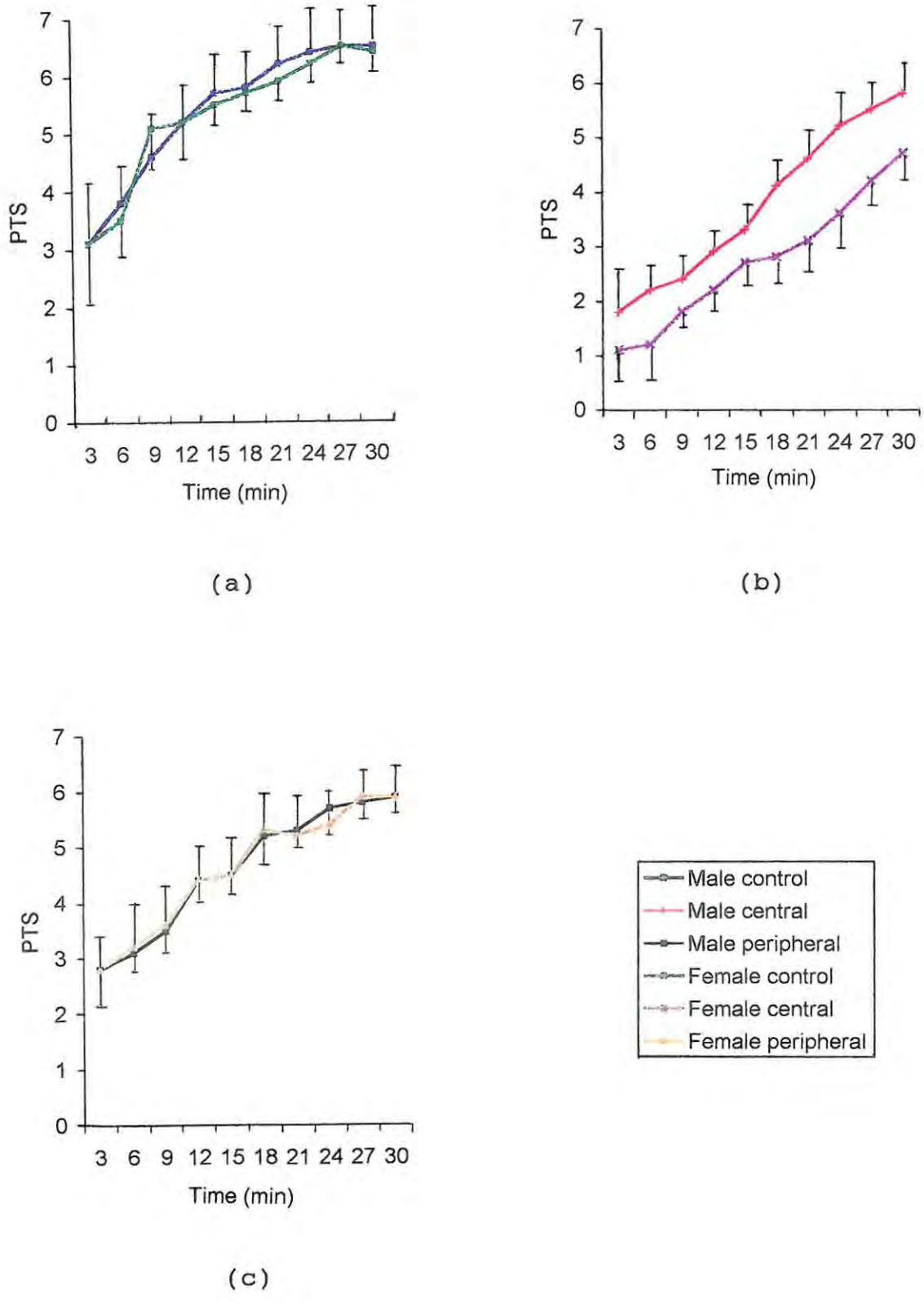

Figure 13: Comparison of male and female perceived thermal sensation (PTS) responses during a 30min submaximal treadmill run under control (a), central cooling (b) and peripheral cooling (c) conditions.

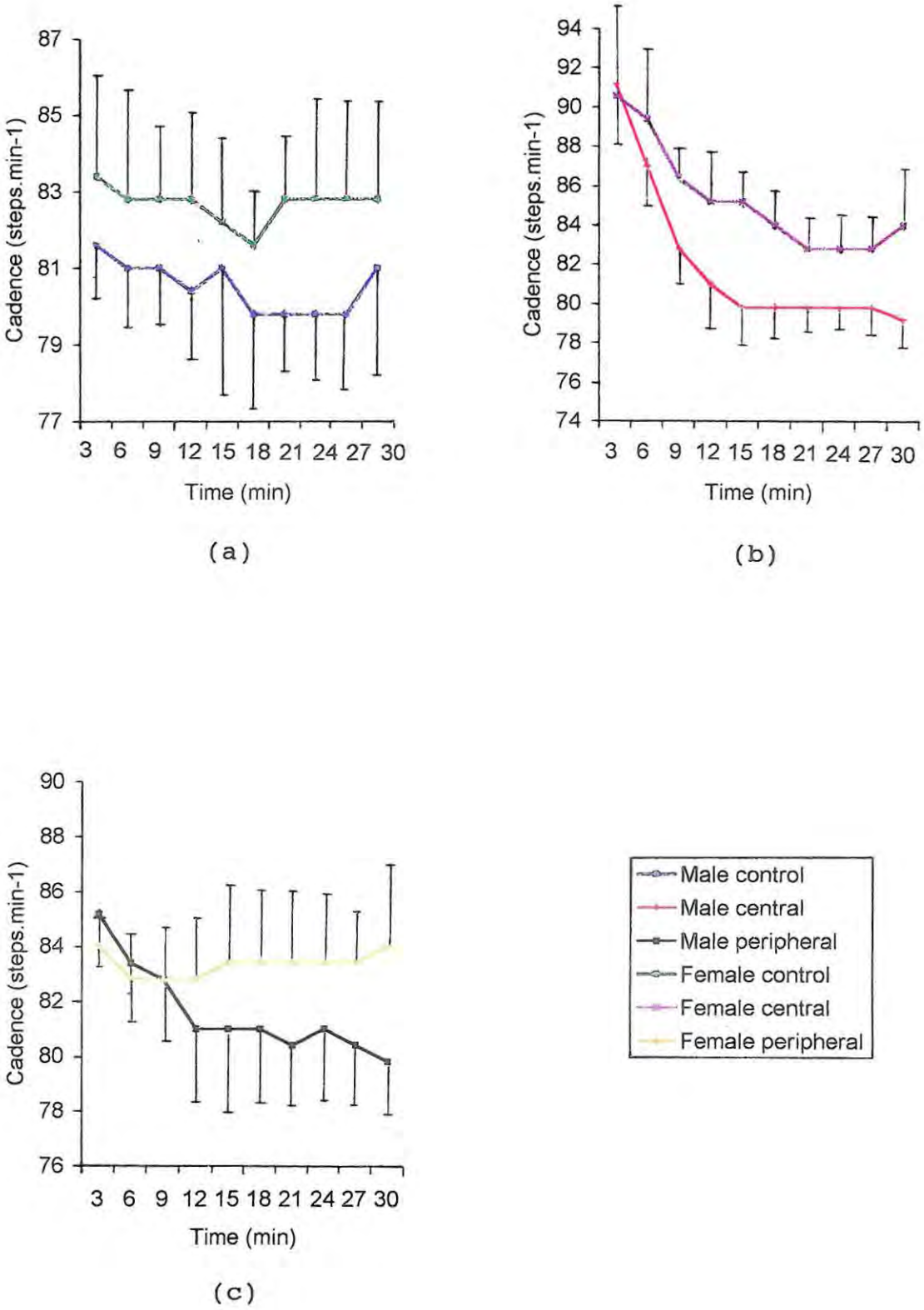

Figure 14: Comparison of male and female cadence responses during a 30min submaximal treadmill run under control (a), central cooling (b) and peripheral cooling (c) conditions.

significant interaction (Figure 14(b)).

Females rated their PTS significantly lower than males following central cooling (Figure 13(b)). While local RPE showed significant interactions, the mean values for each sex were not significantly different (Figure 12(b)), nor were those for overall and central RPE, both of which were qualitatively the same for males and females.

Following peripheral cooling only local RPE was significantly lower for females than males (Figure 12(c)), no differences existed for overall and central RPE, PTS or cadence.

DISCUSSION

Introduction

The focus of this research was an examination of the extent to which dry aerobic performance was affected by body cooling of varying degrees induced by CWI and the relative importance of peripheral *versus* central cooling in this regard. Crucial to the opening of this discussion, therefore, is a consideration of whether in fact these cooling objectives were met. For the purposes of this experiment the body was represented by a core and shell model, with the core at rectal temperature (T_{re}) and the shell comprising a variable resistance offered by the adjustable constriction of peripheral vascular beds, and a fixed resistance, which was a function of the thickness of the layer of subcutaneous body fat. In terms of this model the body core is reduced in mass during cooling and that portion at or near

surface temperature is greatly increased, although, as Bullard and Rapp (1970) point out, the increase in shell size occurs primarily in the extremities and not in the trunk or head. Numerous previous scientists have stated that maximal tissue insulation during CWI occurs at water temperatures between 29°C to 33°C (Bullard and Rapp, 1970; Merrill-Smith and Hanna, 1975; Hong, 1984; Doubt and Hsieh, 1991), clearly well above water temperatures recorded during testing conditions in this study (see Table III).

For the purposes of this research peripheral cooling was defined as a predetermined loss of body heat, without a change in core temperature; central cooling was accepted as a decline in T_{re} of 1°C. Standardisation of the degree of peripheral cooling around the predetermined total H_{sk} of 100kcal.m^{-2}.h^{-1} proved more difficult during data collection with a larger sample than pilot testing had revealed. Table IV indicates that the mean H_{sk} following peripheral cooling was 139.7 ± 35.1kcal.m^{-2}.h^{-1} for the males and 111.7 ± 26.9kcal.m^{-2}.h^{-1} for the females, both values in excess of the stipulated standard. However, examination of the T_{re} of each subject revealed that not one of the subjects had experienced a drop in core temperature at the point at which they were removed from the water tank. Indeed T_{re} was not significantly altered for males or females between the control and peripheral cooling conditions.

The control, central and peripheral cooling conditions did in fact represent significantly different thermogenic states for

these subjects in terms of each of the other thermogenic variables listed in Table IV. Despite there being no difference in core temperature between the control and peripheral cooling states, T_b was significantly altered as a consequence of a significantly lowered T_{sk}. The change in body heat stores (S), M and H_{sk} were significantly greater following peripheral cooling. This applied to both sexes, with the exception of M for the females, which was not significantly changed by this degree of cooling. Central cooling, on the other hand, produced significant reductions in T_{re}, T_{sk} and T_b, with concomitant rises in S, M and H_{sk} compared to control and peripheral conditions. Only perceived thermal sensation (PTS) was the exception to this, with central and peripheral cooling producing virtually identical values, both significantly lower than for the control condition.

Thus it was accepted that, within the limitations of the higher than desired H_{sk} values for peripheral cooling, each of the conditions did represent significantly altered thermogenic states, such that broad definitions of central and peripheral cooling could be applied.

The present study revealed a complex scenario with regard to the interplay of central and peripheral factors as regulators of thermogenesis. It served to highlight the variability in response that can be expected depending on the manipulation of a plethora of factors, the most important in this study being the degree of body cooling and the sex of the subject, and confirmed recent calls for additional research in this area (Doubt, 1991;

Sjödin *et al.*, 1996).

Temperature Responses

The T_{re} data of subjects in this study demonstrated significantly lowered values following central and peripheral cooling compared to control values, although not for females following peripheral cooling. During exercise such as this involving large muscle groups the temperature of the core changes in response to the bulk convective mixing of blood returning from the active muscles and the surrounding shell blood (Rubin, 1987). The reduced mean T_{re} following central cooling is thus consistent with a significantly reduced starting T_{re} compared to the control and peripheral conditions, together with significantly lower T_{sk} and individual temperatures at all sites measured, with the exception of arm temperature in females.

A comparison of pre-run data revealed no significant difference in the T_{re} between control and peripheral conditions for either males or females. During the peripheral condition, however, the mean T_{re} for males remained unelevated compared to the pre-run value, whereas that of the females rose in the same fashion as during the control run, suggesting that at this exercise intensity thermogenesis was sufficient to overcome the 11°C temperature gradient between the core and the skin for females, but was not for males. The corresponding core-to-skin temperature difference for males was 9.6°C. While lowered values of T_{sk} are usually indicative of an increase in insulation, Tikuisis and colleagues (1991) have pointed out that

a more critical examination of an insulative response is obtained by considering the skin to ambient temperature difference - the lower this value, the more insulative the skin is. There was a nominal but not significant tendency towards a lower T_{sk} for the peripheral cooling condition than the control condition in females, thus presenting a reduced body to ambient temperature gradient compared to males. Of the individual sites measured, leg temperature for females was, however, significantly lower for this condition compared to other conditions, with a concomitantly lower skin-to-ambient temperature gradient, as could be anticipated on the basis of a significantly thicker thigh skinfold.

The pre-run data indicated that the immersion times required to produce the necessary cooling effects under both conditions were not significantly different for males and females. This apparent contradiction to previous suggestions that women cool more rapidly than men (McArdle *et al.*, 1984a; Graham, 1988; Pendergast, 1988) may be partly explained by the fact that, in the present study, subjects were closely matched for muscle as well as fat composition (Table II). At that point where T_{re} had dropped by 1°C for both sexes as a result of CWI, T_{sk} was lower for females than males, as were S, M, and H_{sk}. In other words, assuming that lower a T_{sk}, for example, represents a more active response to cooling in females than males, it can be argued that the female subjects in this study responded more vigorously and physiologically more appropriately to cold water immersion than did the men. Although McArdle and co-workers have suggested that

larger metabolic increases for males during CWI may only be evident after a T_{re} drop of more than 1°C, data from the peripheral cooling condition in the present study would suggest otherwise, since with no change in T_{re} at all for either sex, females still recorded significantly lower M and H_{sk} values than males. These data, together with the differences in many of the physiological responses observed during subsequent aerobic performance, as discussed throughout this section, thus add strong support to the contention that sex-specific differences in the physiological response to body cooling do exist. Although it was beyond the scope of the present study, further research is required to clarify the underlying mechanisms behind such differences.

Cardiovascular Responses

The regulation of blood flow by subcutaneous and central thermal stimuli, as integrated by the hypothalamus, and the subsequent effect upon cardiovascular parameters is the subject of some debate, contributed to substantially by dissimilar subject characteristics and/or experimental methodologies. Rubin (1987) has pointed out that a lowering of deep temperature causes a decrease in the rate of spontaneous discharges of the sinus node, in other words a slowing of the heart rate. This epicardial structure is affected by the temperature of vena cava blood entering the right atrium and also by core temperature transmitted to the sinus node through the sinus node artery. Nevertheless, changes in heart rate associated with gross changes in core temperature may be adjusted depending on how cardiac

output responds to alterations of skin blood flow and body metabolism that also accompany changes of core temperature.

Thus a decrease in heart rate as a consequence of cold exposure has not been universally observed in the past. The few studies that have employed protocols similar to that of the present study, examining exercising heart rates following cooling of differing degrees, rather than during cold exposure, have produced equivocal findings. The reduction in heart rate over the 30min period observed following central cooling for males and females in this study contrasts with some previous reports of unaltered heart rates during submaximal exercise, despite reduced T_{re} (Davies *et al.*, 1975; Bergh, 1980; Giesbrecht *et al.*, 1987). Sjödin *et al.* (1996) reported unaltered heart rates in the face of lowered T_{sk} but the same T_{re} during moderate cooling. In contrast, others have described reduced heart rates during submaximal exercise following cold showers (Horvath, 1981) (a process that is likely to produce peripheral cooling without a drop in core temperature) and 10min into a 30min exercise bout following lowered T_{re} and T_{sk} (Booth *et al.*, 1997). In the present study, however, females experienced a significant reduction under the peripheral condition compared to the control condition, but males did not, exhibiting higher heart rates than for either of the other two conditions.

The significance of reduced skin and core temperatures for cardiovascular parameters can be applied to the present data, with the exception of male heart rates following peripheral

cooling. Given the T_{sk} values calculated for males and females as a consequence of CWI, significant constriction is likely to have occurred in cutaneous and muscle vascular beds due to increased sympathetic tone (see pre-run data, Table IV). The resultant rise in central blood volume and consequent increase in blood pressure would precipitate a parasympathetically mediated reduction in heart rate.

The onset of exercise under such conditions would pose contradictory demands on the active musculature: the need to preserve body heat due to low core and/or skin temperatures contrasted with the need for increased blood flow to meet metabolic demands. Pendergast (1988) and Ferretti *et al.* (1995) have suggested that cold induced vasoconstriction may persist during exercise and blunt the exercise hyperemia. Given the above scenario, the present data support such a postulate. Central cooling prior to the 30min run resulted in a mean heart rate reduction for females of 9.1% and a smaller although still significant 3.2% for males, compared to control values. Peripheral cooling in females resulted in a heart rate that was 3.8% lower than the control value. Unlike previous research where a reduction in heart rate due to 10min cold showers lasted only 10 to 20min during post-shower exercise (Horvath, 1981), the depression in heart rate for females in the latter condition persisted for the entire 30min period.

Obviously the extent to which a cold induced vasoconstriction would persist in the face of exercise performed in a warmer

environment would depend on several factors, such as the degree of prior body cooling, the intensity of exercise and the thermal load imposed on the individual. In the present study the laboratory temperature to which the subjects were exposed during exercise ranged between 15.1°C and 15.7°C, air temperatures which can be classified as cool (Dawson *et al.*, 1994) or cold (Wagner and Horvath, 1985b) rather than temperate. Clearly this temperature range would favour the prolongation of cutaneous vasoconstriction more so than would warmer air temperatures. Although it was beyond the scope of this work, a systematic examination of muscle temperatures for varying degrees of body cooling (the work of Giesbrecht *et al.* (1995), for example, is a step in the right direction) and the consequences thereof for the control of muscle blood flow would add greatly to the understanding of exercise performance following pre-cooling.

Furthermore, in the case of central cooling the direct effects of a reduced core temperature upon the heart, as outlined by Rubin (1987), must be taken into account. For the females in the present research, the effects of central and peripheral cooling were additive; in other words the heart rate was depressed more for central cooling compared to control values than it was for peripheral values (Table VI). For the males this was not the case.

These data thus indicate both quantitative and qualitative differences in male and female heart rate responses to exercise following cooling. A direct comparison of the mean normothermic

value revealed a significant sex difference; central and peripheral conditions did not. This in itself, however, supports a greater relative depression in heart rate for females compared to males, although it does mask the rise that occurred for males following peripheral cooling. While this does contradict a previous report of reduced heart rates for males exercising in the cold, but not for females (Doubt, 1991), others have suggested a generalised depression in female cardiovascular functions compared to males during brief cold hand tests and during recovery from these and cold wind tests to the face (Le Blanc *et al.*, 1978). It should also be noted that women may experience a greater vasoconstriction of the cutaneous circulation and the arterial flow to deeper limb structures in response to cooling (McArdle *et al.*, 1984b). While it is unclear how much of this can be related to subcutaneous adipose tissue differences, the present data do suggest a depressor effect following peripheral cooling for females compared to males with regard to heart rate. Pendergast (1988) has commented that the control of muscle blood flow in relation to exercise during or after cooling is dependent upon a complex interaction of muscle temperature, T_{re} and T_{sk}, the exact nature of which is as yet unclear. Given that T_{sk} was 1.6°C lower for females than males during exercise following central cooling and nominally, although not significantly, 1°C lower during the peripheral condition, it is possible that these subjects experienced a greater prolongation of vasoconstriction during exercise than the males, resulting in a greater relative depression in heart rate. That males experienced a rise in heart rate for the peripheral

condition in the presence of diminished vasoconstriction compared to their female counterparts, but a reduction during central cooling, may indicate the relative importance of central factors, such as the direct effect of cold upon the heart.

Furthermore, one cannot rule out the possible effects of factors that influence heart rate but were not measured here. Psychological measures of arousal or anxiety, for example, were not determined during testing. It is unlikely, however, that this would be responsible for the observed results, since the sample sizes were relatively large, and cooling tests were administered randomly. Tables V and VI demonstrate reasonably high standard deviations around mean heart rate responses to cooling, but this was evident for both central and peripheral conditions.

In overall terms, therefore, these data support previous suggestions of a depressor effect of prior cooling upon heart rate (greater for females than males), which in this case was largely explained as being a function of a blunted exercise hyperemia. The exception to this was males' heart rates following peripheral cooling.

Respiratory and Metabolic Responses

Some researchers have suggested that the central cardiovascular response does not constitute a direct limitation of $\dot{V}O_2$ during moderate exercise (Pendergast, 1988; Doubt, 1991) since an elevated stroke volume may counter a reduction in heart rate and

maintain cardiac output; peripheral circulatory factors, on the other hand, may do so. In the present study $\dot{V}O_2$ did not appear to be compromised by either of the pre-cooling conditions, with the exception of peripheral cooling for females. This is in contrast to several studies which have reported an increase in the energy cost of a given activity during or after cold stress (Davies *et al.*, 1975; McArdle *et al.*, 1976; Bergh, 1980; Horvath, 1981; Pendergast, 1988; Kruk *et al.*, 1990; Sjödin *et al.*, 1996), although once again the considerable variability in the outcome of one study to the next depending on the exact nature of the experiment must be stressed. Olschewski and Brück (1988), for example, observed that a prior reduction in deep body temperature in the order of 0.5 to 1°C had no significant effect upon $\dot{V}O_2$ during submaximal exercise on a cycle ergometer at 18°C, a similar ambient temperature to that of the present study.

As outlined above, the mean figures for 30min camouflaged a change in $\dot{V}O_2$ kinetics that was evident particularly for males after central cooling, such that the response when compared to the control condition was qualitatively quite different. During moderate normothermic exercise $\dot{V}O_2$ reaches a new steady state within 3min in normal subjects, with little or no sustained rise in blood lactate (Barstow, 1994). This was evident for males and females during the control run, where little fluctuation around the mean was recorded for either sex (Figure 11(a)).

In terms of the mean values recorded for 30min, the $\dot{V}O_2$ for males and females for central and peripheral cooling conditions

compared to control figures were 1.3% and 2.2% higher and 2.3% and 3.5% higher respectively. In each case the T_{re} was lower than control values by 1.1°C and 0.4°C for males and by 0.9°C and 0°C for females. These findings do not support Pendergast's (1988) statement that the energy cost of exercise is increased 10 to 40% by a 0.5° to 1.5°C decrease in T_{re}, although it must be remembered that the figures for the present study do not represent a drop in T_{re} over the 30min, but rather less of an increase compared to the control condition. Furthermore, if the differences are considered at 3min, 6min and 9min, the disparity does become somewhat less. For males, the $\dot{V}O_2$ was 12.4%, 8.1% and 2.3% higher than control conditions for central cooling at these collection points and 3.9%, 2.3% and 2.3% higher for peripheral cooling. For females these values were 6.1%, 4.9% and 4.2% and 0.4%, 4.2% and 4.9% respectively. In the case of the present study the fact that T_{re} was reduced as a result of a cooling manoeuvre which was no longer applied during the exercise bout must have implications for the length of time over which an increase in $\dot{V}O_2$ is evident, in this case 9 to 15min for the most obviously affected condition - males following central cooling. A similar pattern of response to that seen in this condition was reported by Olschewski and Brück (1988) for previously cooled males over 28min of cycle ergometer exercise at 80% peak $\dot{V}O_2$. The $\dot{V}O_2$ was reportedly 13.5% higher at the onset of exercise, but lower than control values during the latter half of the exercise period, although it was never significantly altered. This trend following central cooling was evident for females in the present study, although not to the same degree. As discussed earlier, for

this degree of cooling females appeared to respond physiologically more appropriately than did males. The T_{sk} was lower for females than males for this condition and pre-run values for *S*, *M* and H_{sk} were significantly lower, suggesting that, particularly initially, the effects of pre-cooling upon $\dot{V}O_2$ would be less dramatic for females than for males. There have, however, been reports that the effect of whole body precooling may be to reduce initial $\dot{V}O_2$ at the onset of exercise (Lee and Haymes, 1995; Shiojiri *et al.*,1997).

Whilst central circulatory parameters may not be the major limiting factor with regard to oxygen cost, there is evidence to suggest that oxygen delivery and utilisation at the active tissue may be affected as a direct effect of cooling (Ferretti *et al.*, 1995; Shiojiri *et al.*,1997). In addition to the effects of cold upon peripheral circulation, as discussed earlier, the leftward shift of the oxyhemoglobin curve during hypothermia may be detrimental to oxygen delivery (Pendergast, 1988; Coetzee and Swanepoel, 1990), although the extent to which this would be evident at the temperatures measured in the present study is not clear.

The same biphasic nature of the response for males following central cooling was illustrated for $\dot{V}CO_2$ and $\dot{V}_E$ (BTPS). Once the initial response to cold exposure is over, alterations in $\dot{V}_E$ are governed primarily by metabolic requirements. Studies which have specifically examined the effect of cold stress upon ventilatory parameters are few and differ in important ways from the present

research. Those incorporating CWI are complicated by a reduction in vital capacity evident during immersion as a result of chestward displacement of the diaphragm and pooling of blood and the opposition to inspiratory effort caused by the hydrostatic pressure (Lange *et al.*, 1974; Burki, 1976; Buono, 1983; Lin, 1988). Since cold air is dry, those which examine performance during cold air exposure must consider the possible effects of cooling and drying upon airway smooth muscle (Giesbrecht and Younes, 1995). The significantly higher $\dot{V}_E$ for males following central cooling and for females following both cooling manoeuvres appeared to be a function of increases in tidal volume rather than breathing frequency since this was not significantly altered for any condition. This would support previous observations by Cooper *et al.* (1976) and Muza *et al.* (1988).

In addition to those factors already discussed, one of the most obvious reasons for an increased energy cost as a result of cold stress is the effect of increased muscular activity due to shivering. As noted subjectively, shivering was evident for all male and female subjects following cooling, but to a greater degree following central than peripheral cooling, which is in accordance with the view that a reduction in deep body temperature facilitates the response (Keatinge, 1969; Hong and Nadel, 1979). This is likely to have been a major contributor to the elevation in $\dot{V}O_2$ evident over the initial 9 to 15min of exercise following central cooling.

Whilst the exercise intensity was well above the approximate

1.5l.min^{-1} cut-off level of the shivering response as reported by Patton and Vogel (1984), it must be remembered that the control of shivering metabolism depends upon the complex interaction of signals from the skin and core, with the relative importance of the latter stressed by some (Hong and Nadel, 1979). That peripheral cutaneous receptor stimulation may initiate shivering in the absence of a drop in core temperature was supported in the present study by the fact that shivering was noted during peripheral cooling, where T_{re} remained unchanged. Even where overt shivering is not evident, however, activation of the piloerectic muscles in the skin or an increased muscular tonus may contribute to a greater energy expenditure compared to normothermic conditions (Paolone and Paolone,1995; Sjödin *et al.*, 1996).

The critical exercise intensity at which heat generation equals or exceeds heat loss and shivering no longer affects oxygen cost is a matter of some debate and, as the present study highlights, is complicated by the variety of exercise paradigms and combinations of temperatures (human and ambient) that are possible and that change during exercise. Previous discussions of this critical level have included those of Pugh (1967), for example, who found that exercise levels that required less than 2.1l.min^{-1} under normothermic conditions were not sufficient to prevent a drop in T_{re} and an increase in $\dot{V}O_2$ in wet, cold conditions. Horvath (1981) described a protocol which produced increases in the metabolic cost of exercise at levels that, without cooling, had required an oxygen uptake of 2.0l.min^{-1}.

This level of $\dot{V}O_2$ may be of consequence for the results of the present study. During the control run, the intensity elicited a significantly different mean absolute $\dot{V}O_2$ of 2.21.min^{-1} for males, who were thus above this threshold, and 1.91.min^{-1} for females, who were below it. This may help explain the significant difference observed in the mean $\dot{V}O_2$ response for females following peripheral cooling, whereas no differences were revealed for males for any condition. It must be remembered, however, that other research has proposed lower critical levels (Strõmme *et al.*, 1963; Horvath, 1981) and has tended to concentrate exclusively on the responses of male subjects, so it is not known if the same level can be applied to females.

In statistical terms the lack of significant effect of peripheral cooling upon male $\dot{V}O_2$ responses (in contrast to that experienced by the females) may be accounted for by the higher percentage difference for females between peripheral and control data during the first third of the run, as discussed earlier, despite significantly lower H_{sk} for females at the end of immersion (Table IV). These findings extend those of Sjödin and colleagues (1996), who found that peripheral cooling produced a significant increase in energy expenditure during 90min of cycle ergometer exercise in a mixed sample of males and females. As in the present study, the increase in their subjects' T_{re} during exercise was the same with and without cooling, but T_{sk} was significantly lower (see Table VI). This lead these authors to propose that some metabolic regulator is triggered peripherally, probably in skin and/or muscle. For the females in the present

study the mean T_{sk} for the run was not significantly different from that of the males following peripheral cooling. However a comparison of the mean T_{sk} obtained for control and peripheral conditions revealed that the difference between conditions (ie. control *versus* peripheral) was greater for females than it was for males (see Figure 9). For the females the difference in mean T_{sk} between these conditions equated to 3.6°C, whereas for the males it was 2.1°C. In other words, the difference in mean T_{sk} between control and peripheral conditions was 1.5°C greater for the females than it was for the males. This may help account for the significantly higher $\dot{V}O_2$ seen in females but not in males. It does not account for the fact that no differences were revealed for either sex following central cooling, where T_{sk} was lower still for both sexes and combined with a significantly reduced T_{re}. However it should be clear from earlier discussion that the $\dot{V}O_2$ responses at the onset of the run following central cooling were very different from those suggested by the overall mean.

Thus the present data revealed that only female $\dot{V}O_2$ during the peripheral condition was altered by prior cooling, although an initially elevated response for all conditions was camouflaged by mean values for the 30min run. Explanations for the raised female $\dot{V}O_2$ following peripheral cooling may lie in a critical (absolute) intensity of exercise, where heat generation equals or exceeds heat loss, together with some form of peripheral metabolic trigger, although the latter does not explain the lack of effect following central cooling.

The Mechanics of Locomotion

According to Wasserman *et al.* (1967), the primary determinant of the amount of oxygen consumed during normothermic exercise depends on the amount of external work done by the muscle and the efficiency. There are, however, inherent difficulties in the estimate of work done, together with much confusion surrounding the qualification of the concept of "efficiency" (Cavanagh and Kram, 1985; Goslin, 1985; Williams and Cavanagh, 1987). In the calculation of net efficiency, where the denominator is defined as the "energy expended above that at rest" (Cavanagh and Kram, 1985), the power output of an individual on the treadmill is reliant primarily upon the velocity of locomotion. The numerator in the calculation of efficiency would thus remain constant, since the treadmill speed was the same for a given individual across each of the conditions. It has been argued, however, that the effort required to conduct dynamic exercise following or during cold exposure may be increased (Doubt, 1991). Doubt has proposed that, in the face of shivering and indeed even in the absence of overt shivering, increased activity of agonist and antagonist muscle groups may oppose movement and raise the oxygen cost of work. In addition, more motor units may be recruited to meet the required work output, potentially raising $\dot{V}O_2$, and thus adversely affecting economy.

Running economy, defined as the rate of submaximal oxygen consumption for a given velocity (Wicox and Bulbulian, 1983; Goslin, 1985; Williams and Cavanagh, 1987; Williams *et al.*, 1991; Svedenhag and Sjödin, 1994), has repeatedly been examined as a

determinant of running performance, with variable results depending on factors such as the homogeneity of the study sample and the performance criterion used. Studies which sought to relate running mechanics, economy and performance concluded that mechanical aspects of running style do have a significant influence on energy expenditure during endurance events (Heinert and Stull, 1986; Williams and Cavanagh, 1987). In the present study the nominal trend for the male subjects was for a progressive increase in the mean value for cadence for control through peripheral and central cooling (Table V) with concomitant decreases in stride length, although none of these alterations were significant. The females, by contrast, exhibited a significantly higher mean stepping rate and reduced stride length for central cooling compared to control and peripheral conditions. Figure 14(b), however, reveals that, following central cooling, cadence was in fact markedly elevated for males and females for the first 15min of exercise. At the first data collection point this equated to stride lengths which were 8.3% shorter for the females compared to control values and 9.5% shorter for the males. Heinert and Stull (1986) demonstrated that shortening the stride length by 8% was sufficient to significantly raise $\dot{V}O_2$ compared to freely chosen stride lengths. This is apparently supported by the initial data obtained following central cooling in the present study, although the relative contributions of such aspects of running style, and physiological, psychological or other factors have not been clearly defined. In terms of mean values over 30min, it is clear that neither cadence nor stride length had a significant effect

upon oxygen cost. For females following peripheral cooling (the only condition to exhibit a significant rise in $\dot{V}O_2$) cadence and stride length remained unchanged.

Ratings of Perceived Exertion (RPE)

This study examined the perception of effort during running at 50% $\dot{V}O_2$ max under normothermic conditions and following peripheral and central body cooling. For the control condition male and female overall and central RPE data were not significantly different from one another but were slightly higher than values previously reported, with overall values being 10.1 and 9.2 for males and females respectively. DeMello and colleagues (1987), for example, reported undifferentiated ratings of about 8 at 50% $\dot{V}O_2$ max on the treadmill for "trained" men and women, with a $\dot{V}O_2$ max higher than those of the present subjects, and of around 10 for subjects whose $\dot{V}O_2$ max values were considerably less. Robertson (1982) presented values for 50% $\dot{V}O_2$ max which varied between 9 and 11 for cycling work, which has been found to produce higher values than treadmill running (Thomas *et al.*, 1995).

The present examination of differentiated ratings following body cooling provided further evidence of the variety of response possible depending on factors which include the type of rating and degree of body cooling involved. Other studies have found RPE to be lower with cooling (Horstman, 1977; Toner *et al.*, 1983) or unchanged despite reductions in T_{re} and T_{sk} (Horvath, 1981; Gordon *et al.*, 1990). The subjects of Patton and Vogel (1984),

on the other hand, found exercise intensities more tiring in a cold-wet situation (5°C), although RPE itself was not measured. For both males and females in the present study overall RPE was significantly lower following peripheral cooling compared to control and central conditions (Tables V and VI). In terms of mean responses, explanations for the significantly lowered overall RPE following peripheral cooling cannot lie in established relationships with physiological variables. For males this RPE result was obtained in the face of a significantly higher heart rate and unchanged $\dot{V}_E$ (BTPS), increases or decreases in which have been associated with similar changes in RPE. For females both heart rate and $\dot{V}_E$ (BTPS) were significantly higher than control and central conditions and $\dot{V}O_2$ was higher than the control figure.

This is of particular relevance to central ratings of RPE, which relate to sensations in the cardiopulmonary systems. Whilst it can be argued that this encompasses variables which were not measured in the present study, such as the haemodynamics of the heart itself over and above pulse rate, together with mean arterial pressure or blood lactate levels, the fact that central RPE recorded following core cooling was not significantly different to the control value for either males or females suggests that the possible effects of deep body cooling upon cardiovascular parameters, as discussed in the earlier section of these results, were not of consequence for central ratings of RPE and could not account for peripheral ratings being significantly lower. For the males significant differences in

physiological variables from one condition to the next were not associated with any change in central RPE.

For the present female subjects local RPE was significantly higher following core cooling than for the control and peripheral conditions. This was associated with the same result for cadence, with a concomitantly shorter stride length for central cooling. In other words, changes in the mechanics of running were reflected by sensations of greater strain in the working muscles and joints. In addition, the direct effect of cold upon many of the factors signalling local effort, although not measured here, must be acknowledged in relation to local RPE. Questions arise, however, with regard to the length of time this response occurred. Figure 10(b) reveals that at 15min the plots for the control and central cooling conditions intersected. For males the mean local exertion rating was significantly lower for core cooling compared to control figures. For these subjects no difference was observed for cadence or stride length.

The fact that males rated local exertion lower and females higher following central cooling compared to the other conditions resulted in there being no significant difference between the sexes where the females' local ratings had been consistently lower for the other two conditions. This difference in the direction of the response between the males and females following central cooling may lie in the fact that T_{sk} and leg temperature (that temperature most relevant to the limbs active in running) were 1.6°C and 1.8°C lower for females than males. This would

add support to Pandolf's (1982) contention that, where thermal conditions are irregular, T_{sk} may be of particular significance to local ratings.

These data suggest that thermal measures may have exerted more of an influence over RPE than has elsewhere been proposed (Toner *et al.*, 1983). It is clear that when a particular cue is markedly altered over others during exercise the resultant sensation can easily dominate RPE (Pandolf, 1982; Scott *et al.*, 1991). The fact that, for both males and females, the overall RPE was significantly lower after peripheral cooling than for the other two conditions once again suggests the relative importance of T_{sk}. For both sexes elevations in physiological variables normally associated with an increase in RPE could not be related to the obtained results. It is unlikely that T_b or T_{re} played a pivotal role since for the females' T_{re} there was no difference compared to the control condition, and for both sexes it was lower for central cooling, where overall RPE was significantly higher than for the peripheral condition. In general, therefore, it can be argued that established relationships between physiological parameters and RPE can no longer be confidently applied following prior body cooling. Rather, local RPE responses for females followed changes in running mechanics (which were not evident for males) and T_{sk} appeared to be an important influence on all differentiated ratings.

It has been postulated that at exercise intensities of 50% $\dot{V}O_2$ max and above central rather than local cues dominate the

perception of effort (Pandolf, 1978; Carton and Rhodes, 1985; Rejeski, 1985; DeMello *et al.*, 1987). The present data suggest that, under the conditions imposed by this protocol, this generalisation may not be applied. For the females overall ratings did follow central ratings more closely than local ratings for all conditions, whereas for the males this was only true of peripheral cooling; overall RPE for the remaining conditions fell on the mean of central and local values. It has been recognised that the interplay of these two factors does depend to a large degree on a host of other factors (Pandolf, 1978; Scott *et al.*, 1991) amongst which thermal conditions are obviously important. Clearly this is an area which warrants further investigation, both in terms of a reassessment of the critical work threshold during incremental work following and during body cooling and the extent to which differing degrees of body cooling influence this relationship.

The present results, as with those of Pandolf *et al.* (1972) and Toner *et al.* (1986), indicate that subjects were able to discriminate successfully between perceived exertion and thermal sensations, in contrast to the findings of Carton and Rhodes (1985). The present subjects were well instructed in the use of both perceived exertion and thermal sensation scales prior to testing and rated both separately and with differing trends. Whether or not perceived discomfort was incorporated into any of the ratings (Carton and Rhodes, 1985) is more difficult to assess. Seen in the context of differentiated ratings obtained following cold stress, however, such sensations must play a role

in local ratings, which by definition are a measure of localised strain in the working muscles and joints and depend upon numerous input parameters.

Ratings of Perceived Thermal Sensation (PTS)

The PTS ratings obtained during baseline data collection before exercise confirmed comments made earlier in this section with regard to the environmental temperatures recorded for this research. Quiescent in the laboratory before the control run and wearing only a swim suit, subjects rated the ambient temperature of 15.7°C at 2.7 for males and 2.9 for females, or "cool" to "slightly cool". This temperature has been described elsewhere as "cold" (Wagner and Horvath, 1985b), although it should be recognised that considerable individual variation in the subjective experience of a given temperature does exist. The preferred indoor temperature in Britain, for example, is reported to be 18°C (Åstrand and Rodahl, 1986). That the water temperatures used for thermal manipulation did constitute cold water immersion was confirmed by ratings of 1.0 from both males and females for immediate pre-run data following central cooling and 1.1 and 1.0 from males and females respectively after peripheral cooling.

The overall effect of prior thermal manipulation was that both males and females perceived the same ambient temperature during the same exercise differently depending on the degree of cooling that had occurred. For all subjects ratings were significantly different from one condition to the next, with the control run producing the highest and central cooling the lowest (Tables VI

and VII). The mean rating for both sexes fell between "slightly warm" to "warm" for the control run and between "neutral" and "slightly warm" following peripheral cooling. The females' rating of 2.7 ("slightly cool" to "cool") for central cooling was significantly lower than the 3.8 ("slightly cool" to "neutral") of the males. For each of the conditions T_{sk} was significantly different for both sexes; T_{re} and T_b were not consistently different.

The lack of a significant sex effect for PTS for the control condition (Figure 13(a)) corresponded with there being no significant difference in T_b, T_{re} and T_{sk}, despite there being differences in the individual temperatures at the chest, leg and arm sites for males compared to females (Table VII). For the peripheral condition T_{re} was significantly higher for females, but PTS was the same for both sexes (Figure 13(c)). Again, however, T_b and T_{sk} were not different (Table IX). The emerging importance of T_{sk} as a driving influence for PTS was confirmed by examination of the data for central cooling. In this case the PTS rating was significantly lower for females than males (Figure 13(b)), despite no difference in T_b and a significantly higher T_{re} (Table VIII). The T_{sk}, however, was 1.6°C lower for females than males. Thus several lines of evidence add further support to the contention that skin temperature is the primary variable leading the perception of thermal sensation (Burton and Bazett, 1936; Gagge *et al.*, 1967; Hardy *et al.*, 1971; Hayward *et al.*, 1977; Wagner and Horvath, 1985a). This is different to the findings of Toner *et al.* (1983), who found T_{sk} and T_{re} played little role

in PTS and in direct contrast to more recent work by Nielsen and Endrusick (1990), who stated that 80-90% of thermal sensation could be accounted for by core temperature alone.

In 1963 Kenshalo and Nafe suggested that the temperature of the skin stimulated was of direct consequence for the threshold at which warm or cold receptors were activated and therefore for how a given temperature was perceived. They stated that when skin temperature was low, the cool threshold was unaffected, but the warm threshold rose. In other words T_{sk} had to be higher before a sensation of warmth was experienced. The PTS data from the present research, when examined in conjunction with those of T_{sk}, indicate the opposite to be true for the conditions under which this experiment occurred. Maximal receptor sensitivity to changes in skin temperature have been reported as ranging from 20°C to 30°C and from 29°C to 32°C for cold and heat receptors respectively (Schäfer *et al.*, 1984). In the present study under control conditions a sensation of warmth as opposed to coolness or thermal neutrality was recorded for males and females (PTS ratings 5.2 for both) at a T_{sk} 29.5°C and 30.1°C (12min) respectively, both of which relate to the ranges described above. Following peripheral cooling, however, ratings of 5.2 and 5.5 ("slightly warm" to "warm") were obtained at a T_{sk} of 28.3°C and 27.2°C (18min) for males and females respectively. For males during the central cooling condition a rating of 5.2 was again recorded at a T_{sk} of 28.2°C (24min); females did not rate PTS higher than 4.7 (30min) during this condition. In other words, subjects perceived a sensation of slight warmth at a lower T_{sk}

than they had previously during normothermic exercise of the same intensity and at the same ambient temperature. The size of the area of skin stimulated has also been indicated as being of importance to the sensory threshold (Kenshalo and Nafe, 1963). While this is conventionally understood to refer to the size of the area to which the cold stressor is applied, it may be of relevance to studies such as the present. While the calculation of T_{sk} provides a figure which is then used as being representative of the entire body, it must be remembered that differences do exist for the temperatures measured at each site. Tables V and VI bear testimony to this. Thus it is feasible that, as skin temperature rises during exercise following CWI, temperatures at some sites will no longer fall within the ranges of dynamic sensitivity for cold receptors. Chest temperature for males following peripheral cooling, for example, rose to over 30°C at 30min, that is, within the range of maximal sensitivity of heat receptors to changes in temperature (Schäfer *et al.*, 1984), whereas all other temperature sites measured were below this, and therefore outside of that range. Although this was apparently not of consequence for the peripheral PTS rating, which did not differ from that of the females, where the above did not apply, it raises an interesting avenue for future researchers, particularly in light of discrepancies noted in the perception of thermal discomfort between the torso and limbs (Golden and Tipton, 1987). Extension of the temperature ranges beyond those observed in the present study, together with the thermal manipulation of individual sites following cold stress would provide useful data in this regard.

CHAPTER 5

SUMMARY, CONCLUSIONS AND RECOMMENDATIONS

A vast literature exists with regard to the effects of exposure to cold upon performance in a cold environment. Comparatively little research has considered the consequences of exposure to cold water (with concomitant cardio-respiratory adjustments), for subsequent performance on land in a warmer environment. Studies to date have reported equivocal results depending on the protocol employed and the combination of factors examined. Adopting an holistic approach, the present research examined the extent to which physiological and psychophysical concomitants of dry aerobic "control" performance were affected by body cooling of varying degrees induced by CWI and the relative importance of "peripheral" (skin heat loss 100kcal.m^{-2}.h^{-1}) *versus* "central" (core temperature 1°C below pre-immersion) cooling in this regard. Given the almost exclusive use of young adult, Caucasian male subjects in many studies of environmental stress, sex-based effects were analysed under control, central cooling and peripheral cooling conditions.

HYPOTHESES

The hypotheses tested were as follows:

1. Neither peripheral nor central cooling induced by head-out whole body immersion in cold water has an affect upon physiological and psychophysical responses to ensuing dry

submaximal exercise.

2. There are no sex-based differences in physiological or psychological responses to submaximal exercise on land following head-out whole body immersion in cold water.

SUMMARY OF PROCEDURES

Of paramount importance in the design of this investigation, which by virtue of its aims had to incorporate a significant environmental stressor to which subjects were exposed, was the physical well being of these individuals. The initial phase of the study thus involved the thorough consideration of safety issues as they related to the proposed experimental design. Several eminent researchers in the field of thermal physiology were approached for advice. Their opinions, together with a detailed examination of the literature, led to two major decisions:

1. That central cooling would constitute a drop in core temperature of no more than 1°C.
2. That the exercise intensity performed by the subjects would be limited to 50% $\dot{V}O_2$ max.

These decisions set a premium of ethical practice over investigative need in the present study.

A pilot testing protocol was designed to examine in the first instance the reliability of the available computer assisted data acquisition system for expired air analysis. This, and procedural consistency, were confirmed by data collected from

three male subjects who performed repeated submaximal and maximal treadmill exercise bouts.

Further pilot testing assessed the degree of body heat loss that could be tolerated by all subjects to meet the criterion of peripheral cooling: in other words, a predetermined loss of body heat **without** a change in core temperature. Three males and three females, each of whom conformed to general subject requirements, sat immersed in cold water until such time as core temperature was 1°C lower than the recorded pre-immersion value. Core and skin temperatures at four sites and $\dot{V}O_2$ were recorded every minute for the duration of the immersion. Based on total skin heat loss calculated for the minute immediately preceding a drop in core temperature the fixed amount of heat loss that was to be termed peripheral cooling was set at $100kcal.m^{-2}.h^{-1}$.

The final phase of the pilot study endeavoured to examine whether the criteria set to distinguish central and peripheral cooling would indeed create significantly different thermal conditions for the subjects from one session to the next. The six subjects who took part in the initial immersion experiment then participated in a $\dot{V}O_2$ max test and three submaximal treadmill conditions: a control run, a peripheral cooling condition and a central cooling condition. Statistical analyses revealed significant differences between the conditions and over time for all temperature-related data and for heart rate, cadence and perceived thermal sensation (PTS). This phase of pilot testing further established that 50% $\dot{V}O_2$ max was a suitable and

attainable intensity of steady state exercise.

Thirteen males and 13 females aged between 18 and 25yr, none of whom was accustomed to cold exposure on a regular basis, participated in the final data collection. In order to ensure the homogeneity of the sample, subject selection was based upon morphological criteria and the results of a $\dot{V}O_2$ max test. Female subjects provided a gynaecological history and experimental data collection sessions for these subjects all fell between days 5 and 12 after the onset of menses. All subjects underwent habituation to treadmill locomotion, to the equipment used to monitor respiratory responses, and to the relevant psychophysical rating scales prior to data collection.

Experimental data collection involved a series of three randomly performed treadmill runs at 50% $\dot{V}O_2$ max: a control condition without prior manipulation of the subjects' thermal status; a central cooling condition where core temperature was reduced during CWI by 1°C before the run; and a peripheral cooling condition where a predetermined heat loss ($100kcal.m^{-2}.h^{-1}$ without a reduction in core temperature) was prescribed before the run. Subjects reported to the laboratory between 7h30 and 9h00 for a 60min acclimation period, during the last 15min of which they were prepared for testing and a final 10min pre-immersion or pre-exercise recording of baseline physiological measures was made.

Immersions took place in a water tank ($\bar{x}$ 19.8°C water temperature over both immersion conditions), with the seated subject immersed

to the neck. During these conditions respiratory data were collected by means of an Oxylog; heart rate was monitored telemetrically in both wet and dry conditions via electrodes attached in a modified lead-II ECG configuration; core temperature was obtained by rectal thermometry and skin temperatures via thermistors attached at chest, leg, arm and hand sites. Ratings of perceived exertion (during treadmill runs only) and of perceived thermal sensation were obtained manually.

Once the prescribed levels of body cooling were achieved during CWI subjects began a 30min treadmill run at 50% $\dot{V}O_2$ max. During each of the treadmill sessions physiological and psychophysical data were collected every 3min using the methods described above, with the exception of respiratory data, which were collected via an on-line computer-aided system of expired air analysis. Cadence was counted visually over 10s intervals during data collection periods and then converted to minute stepping rates, and from this subjects' stride lengths were calculated.

Statistical treatment of the data involved the use of Student's related t-tests to reveal significant differences between environmental conditions relevant to each condition and independent t-tests for differences in morphological criteria between males and females. Multifactor, repeated measures analysis of variance (ANOVA) was used to determine if the factors "sex" or "exposure" had significant effects on collected and derived variables. Tukey's critical difference was used to locate significant differences between means.

SUMMARY OF RESULTS

1. Examination of the pre-run thermogenic data established that subject preparation in terms of the requirements of the working definitions of central and peripheral cooling were met, with the exception, however, of higher than desired H_{sk} values for peripheral cooling and significantly lower values for females compared to males.

2. Prior manipulation of body temperatures resulted in a significant displacement of the relative exercise intensity/ T_{re} relationship evident under normothermic conditions. This was not true, however, for females following peripheral cooling. The T_{re} was significantly lower over the 30min run for all conditions compared to the control except peripheral cooling in females. At that point where T_{re} had dropped by 1°C for both sexes as a result of CWI, T_{sk} was lower for females, as were S, M and H_{sk}. In other words, for the same drop in T_{re} the physiological response of females was more appropriate than that of the males. This, together with the differences in many of the physiological responses observed during subsequent aerobic performance, adds strong support to the contention that sex-specific differences in the physiological response to body cooling do exist.

3. Indeed, for the central cooling condition, 61% of the variables examined showed sex-based differences in response. Following peripheral cooling certain variables, such as chest and arm temperature or heart rate, showed no

differences in response where there had been differences under control conditions. Thus, despite there being fewer total differences (38%), it is strongly suggested that the use of data pooled from males and females should be discontinued and more precise matching of subjects aimed for, especially where they are to be drawn from both sexes.

4. Equivocal reports have appeared in the past describing the heart rate response to body cooling. That it is influenced by the degree of cooling and the sex of the subject was upheld by the present data. Females experienced less of an increase in heart rate during exercise under both conditions compared to the control values. Male responses were the same following central cooling, but not peripheral cooling, where mean values were higher than control and central figures. These data support the possibility of a depression in female cardiovascular functions following peripheral cooling that was not apparent for males. For females the effects of peripheral and central cooling were additive; for the males this was not the case.

5. In contrast to several studies which have reported an increase in the energy cost of exercise during or after cold stress, $\dot{V}O_2$ in the present study was not altered from one condition to the next, with the exception of peripheral cooling for females where $\dot{V}O_2$ was elevated. The use of mean figures over the duration of the 30min run did, however, camouflage qualitative differences in the response

following cooling. Initial elevations in $\dot{V}O_2$ followed by a return towards control values produced a biphasic response, which was especially evident for males following central cooling. This biphasic response was also evident for $\dot{V}CO_2$ and $\dot{V}_E$ (BTPS).

6. For female subjects cadence and stride length were significantly altered following central cooling compared to control and peripheral values. Although adverse changes in these components of running style as a result of cooling may have influenced $\dot{V}O_2$ in the initial stages of the run for both sets of subjects, they did not appear to be associated with alterations in the mean $\dot{V}O_2$ for females over the 30min run following peripheral cooling.

7. The RPE data in the present study suggest that thermal measures may exert more of an influence over the perception of effort than has otherwise been proposed. For both sexes elevations in physiological variables normally associated with an increase in RPE could not be related to the obtained results. For both sexes overall ratings of perceived exertion were significantly lowered under peripheral cooling compared to both other conditions. Central RPE was lower in the same condition (peripheral cooling) for females but unchanged for the males. Males rated local exertion lower and females higher following central cooling compared to the other conditions. This was explained in terms of changes in cadence and stride length together with the effect of

lower T_{sk} and leg temperature for females under this condition.

8. The consequences of body cooling for PTS were that both males and females perceived the same ambient temperature during the same exercise differently depending on the degree of cooling that had occurred. Ratings were significantly different for both sexes across the conditions: control PTS was highest and central cooling the lowest. In this study T_{sk} emerged as the primary variable mediating the perception of thermal sensation.

CONCLUSIONS

This study confirmed that, for both males and females, physiological and psychophysical responses during dry submaximal exercise were altered differently depending upon the degree of previous body cooling to which they were subjected. Unlike many studies in the past, strict matching of subjects in terms of morphology and cardiorespiratory capacity allowed for the meaningful comparison of data from males and females. Strong evidence emerged to support the contention that, for matched subjects, sex-based differences in responses to body cooling do exist. However the importance of defining the exact criteria to which such a statement can relate was highlighted by the degree of dissimilarity between the sexes when individual variables under different conditions were examined. In other words, generalised statements should be made with caution; in the present study the same variable often responded differently,

depending on the condition examined, for the same subjects performing the same exercise.

Specifically, the pre-run responses of females in terms of *S*, *M* and H_{sk} appeared to be less sensitive following both peripheral and central cooling; the same subjects, however, experienced an elevation in mean exercise $\dot{V}O_2$ following peripheral cooling which was not apparent for any of the other conditions (males and females) studied. This was coupled with a depression in heart rate for females following peripheral and central cooling, the effects of which appeared additive, whereas for males this was not the case. Neither cadence nor stride length had a significant effect upon oxygen cost for either sex.

The investigation of subjective reactions to exercise following different degrees of cooling represented a challenge in that many of the observed relationships between RPE and physiological variables such as heart rate or ventilation appeared to be altered. The effects of deep body cooling upon selected variables were not of specific consequence for RPE. Rather, T_{sk} emerged as the driving influence for effort perception as a particular cue which was altered sufficiently over others following both central and peripheral cooling to dominate sensations. This also applied to PTS, where T_{sk} emerged as a dominant cue rather than T_{re} or T_b.

HYPOTHESES

Based on the fact that the hypotheses generated in this study were such as to enable a broad examination of the effects of CWI upon subsequent aerobic performance on land, the bulk of the evidence in each case, as outlined above, leads to decisions in respect of the null hypotheses ($p<0.05$) as follows:

1. The null hypothesis (CWI has no physiological or psychological effect) was in this case rejected in favour of the alternative hypothesis:

   Both peripheral and central cooling induced by head-out whole body immersion in cold water significantly affect physiological and psychophysical responses to ensuing dry submaximal exercise.

2. The null hypothesis (sexes respond identically) was in this case rejected in favour of the alternative hypothesis:

   There are significant sex-based differences in physiological and psychophysical responses to submaximal exercise on land following head-out whole body immersion in cold water.

RECOMMENDATIONS

It is advisable that future studies deal closely with subject characteristics as a primary determinant of the outcome of any research on this topic. Thus, for example, the pooling of male and female data, which has occurred in recent research, must be

discouraged if meaningful statements about the effects of thermal manipulation upon performance are to be made. It is suggested also that future research look at female responses in more detail. This is particularly true of the critical exercise intensity at which heat generation equals or exceeds heat loss and shivering no longer affects oxygen cost.

Within the context of an increased awareness of the need to standardise as many as possible of those factors said to influence thermal physiology, further clarification is necessary of the range of responses possible given the variety of exercise paradigms and temperatures (human and ambient) that can be combined. While not within the scope of the present study, it would be useful to examine a range of ambient temperatures to which pre-cooled subjects were exposed during exercise, and to extend the exercise intensities investigated.

The relationship between skin and muscle temperatures and blood flow during experiments such as this requires further clarification. Data from the present study indicated that after approximately 15min of exercise the effects of cold induced vasoconstriction of blood vessels were of less consequence for physiological variables such as $\dot{V}O_2$ or $\dot{V}_E$ (BTPS). Future researchers would do well to provide information regarding the points at which the release of vasoconstriction occurs followed by the initiation of active vasodilation during exercise preceded by cold exposure.

Finally, many aspects of the subjective perception of effort following body cooling of differing degrees remain to be further elucidated. The effect of cold upon RPE has produced equivocal findings. Ratings of perceived exertion are clearly influenced by several factors, not all of which could be incorporated into the present study. Further research could extend the range of exercise intensities examined under similar conditions to those of the present study and establish that point at which RPE is altered by cold exposure for males compared to females and for peripheral compared to central cooling. Furthermore, the domination of RPE by either local or central cues above a critical work threshold (50% $\dot{V}O_2$ max) may have to be re-examined in the context of cooled subjects. This study provided some evidence that T_{sk} may be the driving influence even at 50% $\dot{V}O_2$ max; it is necessary that this be clarified using the appropriate experimental protocol.

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APPENDIX A

Pre-Test Information to Subjects

Informed Consent

RHODES UNIVERSITY

DEPARTMENT OF HUMAN MOVEMENT STUDIES

INFORMED CONSENT INFORMATION SHEET

THESIS: **The effects of whole body immersion in cold water upon subsequent terrestrial aerobic performance: a study in hypothermia.**

GENERAL: The purpose of this study is to assess the relative importance of peripheral and central body cooling by immersion in cold water for ensuing dry land performance. In doing so, the question of whether sex-based differences in thermoregulation exist following cold water exposure will be addressed. Furthermore, the study will evaluate subjective responses to exercise following prior cold water exposure. In other words, will body cooling exert a significant influence upon one's perception of the effort required to perform a given task and the perception of thermal sensations?

PROCEDURE: Please note that your participation in this study is entirely voluntary and that you may withdraw at any stage prior to or during the research. As a subject you will be required to complete the following:

1. the collection of biographic data such as age and sex (as part of this females will be asked to maintain charts indicating the time of menstruation) and the assessment of body composition. This will involve the measurement of your height, weight, skinfolds, limb girths and bone widths.

2. thorough habituation to treadmill walking and running and to the equipment used for data collection. On the treadmill you will be asked to breathe through a two-way valve. Heart rate electrodes will be attached to your chest at three sites and skin temperature probes will be applied to your chest, thigh, upper arm and hand. You will be required to wear a rectal probe for the measurement of core temperature.

3. a treadmill test of maximal aerobic capacity ($\dot{V}O_2$ max), where you will be asked to run at increasing speeds until you can no longer continue. You will be requested to abstain from food, drinks other than water, and tobacco for at least 2h before the test, and energetic physical activity for the 24h prior to the test.

4. a series of treadmill runs both with and without prior exposure to cold water. Each of the runs will

last 30min, during which time physiological data and your perceptions of effort and of thermal sensation will be recorded.

5. before each of the runs you will report to the laboratory between 7h00 and 9h00 after abstaining from any food, drinks other than water, tobacco and pharmacological agent overnight (10h). At the laboratory you will be provided with a standard meal. You will be required to remain in the laboratory for the 1h before testing starts. At the end of this period resting physiological data will be collected.

6. the following pre-exercise preparation will be required, according to the experiment underway:
   i) control - a 1h period of laboratory rest with no water immersion.
   ii) peripheral cooling - a 1h period of laboratory rest followed by immersion in cold water until body heat stores are reduced by a set amount, without a drop in core temperature.
   iii) central cooling - a 1h period of laboratory rest followed by immersion in cold water until core temperature drops by 1°C.

7. you will be required to inform the tester of any injury sustained before or during testing and of any illness or medication taken during the testing period.

8. you will be required to report for testing suitably dressed. Shorts, a tee-shirt and suitable shoes are acceptable for treadmill running and a swimming costume (two-piece for females) for water immersions.

RISKS: you are assured that every precaution will be taken to ensure your safety should you participate as a subject in this study. The research protocol has been designed to minimise all risks to the subjects and has been reviewed and accepted by the Rhodes Ethics Committee. Nevertheless, you should be aware of the following factors:

1. you will be asked to exercise maximally during the test of $\dot{V}O_2$ max. The risks you may encounter during this test are mainly those encountered during light to heavy exercise - dizziness and fatigue, for example.

2. there is the possibility of injury due to slipping on the motor-driven treadmill. Habituation will, however, ensure that you are thoroughly at ease with treadmill locomotion before testing starts.

3. due to the nature of the study a warm-up after cold

water immersion cannot be permitted prior to treadmill running. Thus there is a possibility of initial feelings of discomfort and of muscle strain. However the exercise to be performed is of a moderate intensity and should not pose undue problems.

BENEFITS: you will receive information relating to your body composition and $\dot{V}O_2$ max. The general results of the study will also be made available to you. In addition, you will be exposed to new and different equipment and methods of data collection. The findings of the study, of which you will be an integral part, will contribute to man's understanding of human performance and will be of practical importance in both sporting and ergonomic areas.

Your participation as a subject would be most appreciated.

LIZ MANLEY

SUBJECT CONSENT FORM

I,....., having been fully informed of the nature of the research entitled:

The effects of whole body immersion in cold water upon subsequent terrestrial aerobic performance: a study in hypothermia.

do hereby give my consent to act as a subject in the abovenamed research. I am fully aware of the procedures involved as well as the potential risks and benefits attendant to my participation as explained to me verbally and in writing. In agreeing to participate in this research, I waive any legal recourse against the researchers or Rhodes University from any and all claims resulting from personal injuries sustained. This waiver shall be binding on my heirs and personal representatives. I realize that it is necessary for me promptly to report to the researcher any signs or symptoms indicating any abnormality or distress.

I am aware that I may withdraw my consent and may withdraw from participation in the research at any time. I am aware that my anonymity will be protected at all times, and agree that the information collected may be used and published for statistical and scientific purposes.

I have read the information sheet accompanying this form and understand it. Any questions which may have occurred to me have been answered to my satisfaction.

SUBJECT:	.....	.....	.....
	(print name)	(sign)	(date)
RESEARCHER:	.....	.....	.....
	(print name)	(sign)	(date)
WITNESS:	.....	.....	.....
	(print name)	(sign)	(date)
SUPERVISOR:	.....	.....	.....
	(print name)	(sign)	(date)

APPENDIX B

Equations for the calculation of body composition according to Drinkwater and Ross (1980)

Procedures and Formulae for the Drinkwater Tactic (Drinkwater and Ross, 1980).

1. Once all the measurements have been taken the arm, chest, thigh and calf girths must be corrected for fat using the following formulae:

1.1 Corrected arm girth = arm girth - $\left(\pi \times \frac{\text{triceps SF}}{10} \right)$

1.2 Corrected chest girth = chest girth - $\left(\pi \times \frac{\text{subscap SF}}{10} \right)$

1.3 Corrected thigh girth = thigh girth - $\left(\pi \times \frac{\text{thigh SF}}{10} \right)$

1.4 Corrected calf girth = calf girth - $\left(\pi \times \frac{\text{calf SF}}{10} \right)$

Where SF = Skinfold (mm); girth (cm)

2. Convert all the skinfolds, diameters, corrected girths and uncorrected forearm, wrist and ankle girths to Z-scores using the formula below and the relevant phantom reference values and standard deviations (see Ross and Marfell-Jones, 1982):

$$Z = \frac{1}{PSD} \left[AV \left(\frac{170.18}{S} \right) - PV \right]$$

Where:
Z = Z-score or value
PSD = phantom standard deviation
AV = anthropometric variable
S = measured stature (cm)
PV = phantom value

3. Fat Mass

3.1 Absolute Fat Mass

$$AFM = \frac{\left(\frac{\Sigma\ SF}{6} \times 3.25\right) + 12.13}{\left(\frac{170.18}{S}\right)^3}$$

Where:
AFM = absolute fat mass (kg)
Σ SF = sum of the triceps, subscapular, supra-iliac, abdominal, thigh and calf skinfold phantom Z-scores
S = measured stature (cm)
3.25 = mean phantom standard deviation for fat component
12.13 = mean phantom value for fat component

3.2 Relative Fat Mass

$$RFM = \frac{AFM}{MBM} \times 100$$

Where:
RFM = relative fat mass (%)
AFM = absolute fat mass (kg)
MBM = measured body mass (kg)

4. Bone Mass

4.1 Absolute Bone Mass

$$ABM = \frac{\left(\frac{\Sigma\ DG}{4} \times 1.57\right) + 10.49}{\left(\frac{170.18}{S}\right)^3}$$

Where:
ABM = absolute bone mass (kg)
Σ DG = sum of the bi-epicondylar humerus diameter, bicondylar femur diameter, wrist girth and ankle girth phantom Z-scores
S = measured stature (cm)
1.57 = mean phantom standard deviation for bone component
10.49 = mean phantom value for bone component

4.2 Relative Bone Mass

$$RBM = \frac{ABM}{MBM} \times 100$$

Where: RBM = relative bone mass (%)
AFM = absolute bone mass (kg)
MBM = measured body mass (kg)

5. Muscle Mass

5.1 Absolute Muscle Mass

$$AMM = \frac{\left(\frac{\Sigma\ CG}{5} \times 2.99\right) + 25.55}{\left(\frac{170.18}{S}\right)^3}$$

Where: AMM = absolute muscle mass (kg)
Σ CG = sum of the corrected arm, chest, thigh and calf girths and the uncorrected forearm girth phantom Z-scores
S = measured stature (cm)
2.99 = mean phantom standard deviation for muscle component
25.55 = mean phantom value for muscle component

5.2 Relative Muscle Mass

$$RMM = \frac{AMM}{MBM} \times 100$$

Where: RMM = relative muscle mass (%)
AMM = absolute muscle mass (kg)
MBM = measured body mass (kg)

6. Residual Mass

6.1 Absolute Residual Mass

$$ARM = \frac{\left(\frac{\Sigma\ D}{4} \times 1.90\right) + 16.41}{\left(\frac{170.18}{S}\right)^3}$$

Where: ARM = absolute residual mass (kg)
Σ D = sum of the biacromial, transverse chest, bi-iliac and A-P chest diameter phantom Z-scores
S = measured stature (cm)
1.90 = mean phantom standard deviation for residual component
16.41 = mean phantom value for residual component

6.2 Relative Residual Mass

$$RRM = \frac{ARM}{MBM} \times 100$$

Where: RRM = relative residual mass (%)
ARM = absolute residual mass (kg)
MBM = measured body mass (kg)

7. Predicted Body Mass

$$PBM = AFM + ABM + AMM + ARM$$

Where: PBM = predicted body mass (kg)
AFM = absolute fat mass (kg)
ABM = absolute bone mass (kg)
AMM = absolute muscle mass (kg)
ARM = absolute residual mass (kg)

8. Error of Estimation

$$EE = \frac{PBM - MBM}{MBM} \times 100$$

Where: EE = error of estimation (%)
PBM = predicted body mass (kg)
MBM = measured body mass (kg)

Note: A negative sign indicates an underprediction while a positive sign indicates an overprediction.

APPENDIX C

Psychophysical Rating Scales:

RPE Instruction

RPE Scale

PTS Instruction

PTS Scale

The following instruction was read to each subject prior to the $\dot{V}O_2$ max test, which required overall RPE only:

> "We want you to estimate how hard you <u>feel</u> the work is: that is we want you to rate the degree of perceived exertion you feel. By perceived exertion we mean the total amount of exertion and physical fatigue, combining all sensations and feelings of stress, effort and fatigue. Don't concern yourself with any one factor such as leg discomfort or shortness of breath, but try to concentrate on your <u>total inner</u> feeling of exertion. Try to estimate as honestly and objectively as possible. Don't underestimate the degree of exertion you feel, but don't overestimate it either. Just try to estimate as accurately as possible. When you are asked to rate your work, you should do so by giving the numerical value on the scale in front of you which indicates your evaluation of your perceived exertion at that moment. A rating of 6 corresponds with feelings of exertion while standing quietly on the treadmill. A rating of 20 reflects maximal exertion."
>
> (Adapted from Mihevic, 1983 and Pandolf, 1982)

The following instruction was read to subjects before each data collection session that required differentiated ratings:

> "We want you to estimate how hard you feel the work is: that is we want you to rate the degree of perceived exertion you feel. By perceived exertion we mean the total amount of exertion and physical fatigue, combining all sensations and feelings of stress, effort and fatigue. You will be asked to produce a local rating pertaining to feelings or sensations of strain in the exercising muscles and joints, a central rating which involves sensations from the heart and lungs, and an overall, general rating of exertion. For the overall rating we would like you to integrate your local and central ratings with whatever weightings you deem appropriate. Try to estimate as honestly and objectively as possible. Don't underestimate the degree of exertion you feel, but don't overestimate it either. Just try to estimate as accurately as possible. When you are asked to rate your work, you should do so by giving the numerical value on the scale in front of you which indicates your evaluation of your perceived exertion at that moment. A rating of 6 corresponds with feelings of exertion while standing quietly on the treadmill. A rating of 20 reflects maximal exertion."
>
> (Adapted from Mihevic, 1983 and Pandolf, 1982)

RPE SCALE

6
7 VERY, VERY LIGHT
8
9 VERY LIGHT
10
11 FAIRLY LIGHT
12
13 SOMEWHAT HARD
14
15 HARD
16
17 VERY HARD
18
19 VERY, VERY HARD
20

The following instruction was read to each subject prior to each experimental data collection session:

"We want you to indicate how you are experiencing the temperature of your body: that is we would like you to rate your perception of thermal sensation. Try not to concern yourself with any one part of your body, but rather concentrate on your overall sensation of temperature. Try to estimate as honestly and objectively as possible. When you are asked to rate your sensation of temperature, you should do so by giving the numerical value on the scale in front of you which indicates your evaluation of your perceived thermal sensation at that moment. A rating of 1 corresponds with a sensation of cold, a rating of 7 reflects a sensation of heat.

PTS SCALE

1 COLD

2 COOL

3 SLIGHTLY COOL

4 NEUTRAL

5 SLIGHTLY WARM

6 WARM

7 HOT

APPENDIX D

Data Collection Sheets:

Anthropometric and biographic data

Female subjects: Gynaecological History

Experimental Data Collection

ANTHROPOMETRIC AND BIOGRAPHIC DATA

NAME:.....**CODE:**.....

STATURE (cm):

MASS (kg):

SKINFOLDS (mm):

Triceps:	Subscapular:	Front thigh:
Biceps:	Suprailiac:	Medial calf:
Forearm:	Abdominal:	

GIRTHS (cm):

Relaxed arm:	Thigh:
Relaxed forearm:	Calf:
Wrist:	Ankle:
Chest:	

WIDTHS (cm):

Bi-epicondylar:	Bi-iliac:
Bi-condylar:	Transverse chest:
Bi-acromial:	A-P chest:

SUITABILITY:
BSA: MASS RATIO.....
% FAT.....
% MUSCLE.....
SKINFOLD DISTRIBUTION RANGES.....
$\dot{V}O_2max$.....

FEMALES - GYNAECOLOGICAL HISTORY.....

ACCEPTABILITY.....

FEMALE SUBJECTS: GYNAECOLOGICAL HISTORY

SUBJECT CODE:.....

Please answer the following questions accurately. The information is important since the body temperature differs over the normal menstrual cycle and it is essential to control for this during data collection.

1. Do you usually experience a regular 28 day menstrual cycle?

   YES NO

2. If you answered no to the above question, what is your menstrual cycle?

   .....

3. Do you suffer from pre-menstrual discomfort?

   YES NO

4. Over the past six months have you menstruated regularly?

   YES NO

5. Do you take oral contraceptives?

   YES NO

6. Do you take any estrogen supplements?

   YES NO

EXPERIMENTAL DATA COLLECTION

EXPERIMENT: CONTROL | PERIPHERAL COOLING | CENTRAL COOLING

DATE:.....

ASSISTANT:.....

SUBJECT NAME:.....

MASS (kg):.....

BASELINE MEASURES (10min):

ENVIRONMENTAL CONDITIONS:

DRY BULB (°C):.....
WET BULB (°C):.....
WATER (°C):.....
P_B (mmHg):.....
RH (%):.....

	1	2	3	4	5	6	7	8	9	10
HR ($b.min^{-1}$)										
PTS										
CORE TEMP (°C)										
SKIN TEMP (°C)										
CHEST										
LEG										
ARM										
HAND										
$\dot{V}O2$ ($l.min^{-1}$)										
$\dot{V}e$ ($l.min^{-1}$)										

* Attach on-line print-out to this sheet.

IMMERSION DATA:

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
HR															
PTS															
CORE TEMP															
SKIN TEMP CHEST															
LEG															
ARM															
HAND															
$\dot{V}O_2$ (Ab)															
$\dot{V}e$															

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
HR															
PTS															
CORE TEMP															
SKIN TEMP CHEST															
LEG															
ARM															
HAND															
$\dot{V}O_2$ (Ab)															
$\dot{V}e$															

SUBMAXIMAL TREADMILL RUN (30MIN):

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
HR															
PTS															
RPE (L)															
RPE (C)															
RPE (O)															
CORE TEMP															
SKIN TEMP CHEST															
LEG															
ARM															
HAND															
CADENCE															

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
HR															
PTS															
RPE (L)															
RPE (C)															
RPE (O)															
CORE TEMP															
SKIN TEMP CHEST															
LEG															
ARM															
HAND															
CADENCE															

* Attach on-line print-out to this sheet.

APPENDIX E

Subject Feedback

August 1992

Dear

Included with this letter you will find information relating to your recent participation in my research. Unfortunately for ethical reasons the names and data of other subjects could not be included, but direct comparison of your results with those of other subjects may be of interest to you. Where possible I have included mean values and normative data for your information. If you have any queries at all, or wish to see the results in more detail, don't hesitate to contact me.

Finally, I must thank you most sincerely for the enthusiastic and good natured way in which you took part in this research. Obviously without you it would not have been possible and I am extremely grateful.

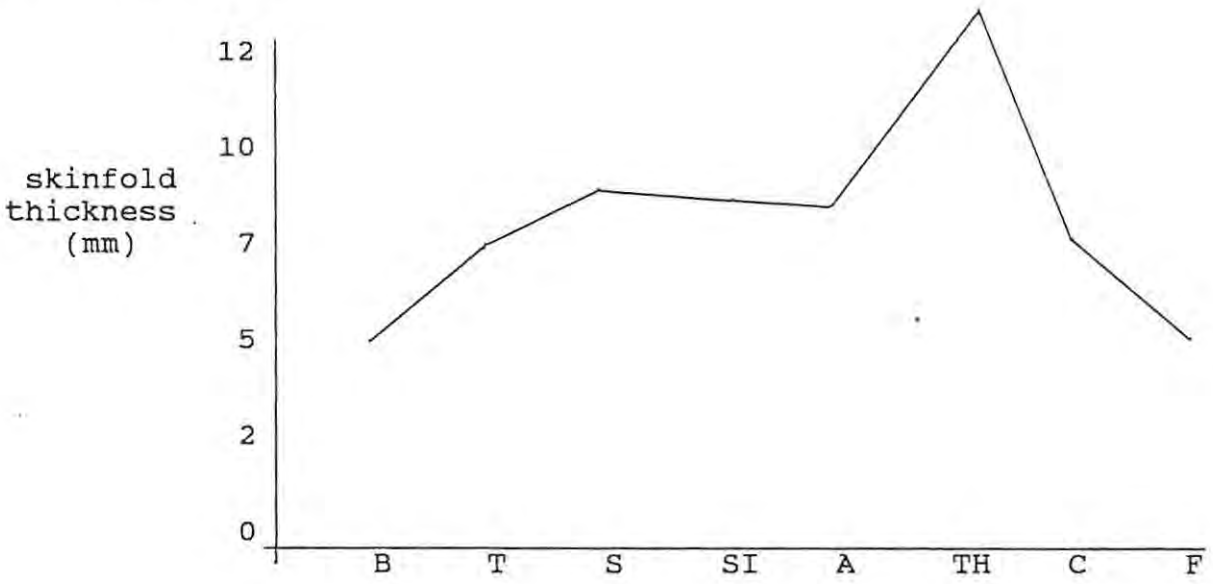
Many thanks and all the best for the future

SKINFOLD PROFILE

Your skinfold ratings and graphical profile are an indication of how your fat is distributed on your body.

	Your measure	Rating
Biceps (B)	5.2	Plump
Triceps (T)	7.2	Average
Subscapular (S)	8.4	Ideal
Suprailiac (SI)	8.3	Ideal
Abdominal (A)	8.1	Ideal
Thigh (TH)	12.5	Average
Calf (C)	7.3	Average
Forearm (F)	5.0	Average

Skinfold Graph



Additional Comments:

BODY COMPOSITION ANALYSIS

Total measured body mass	:	100.0%	69.6 kg
Fat Mass	:	9.3%	6.5 kg
Muscle Mass	:	45.9%	31.9 kg
Bone Mass	:	17.2%	12.0 kg
Residual Mass	:	25.3%	17.6 kg

Total estimated body mass: 68.0 kg
Error of estimation : -2.4%

Acceptable percentages of body fat for relatively inactive males are: under 30 years of age, 13-18% and over 30 years of age, 14-24%. The values for females are: under 30 years of age, 20-28% and over 30 years of age, 25-31%.

Note that the body requires a certain amount of fat for healthy functioning. It is NOT advisable for fat percentages to be below 3% in a male and 12% in a female.

Body fat may be classified into two categories, namely essential and storage fat. Essential fat is stored in the heart, lungs, liver, bones, spleen, kidneys, intestines, muscles and the central nervous system. This fat is required for normal physiological processes. Storage fat is the nutritional reserve which is used as a source of energy and which includes the fatty tissue that protects the various organs and that fat which is stored beneath the surface of the skin.

Relative body fat norms for a variety of activities are presented below:

Activity	%fat	
	Males	Females
Basketball	13.2	20.8
Distance running	7.5	13.3
Diving	14.8	22.3
Gymnastics	9.0	12.9
Hockey	11.5	22.2
Karate	13.2	22.2
Rowing	13.9	28.0
Rugby	11.2	-
Soccer	9.8	-
Squash	11.8	22.8
Swimming	7.5	18.9
Tennis	11.8	22.1
Volleyball	13.4	25.3

Additional Comments:

MAXIMAL AEROBIC POWER

Maximal aerobic power ($\dot{V}O_2$ max) can be defined as the maximum amount of oxygen that can be consumed per unit of time. It is an indication of the functional capacity of the cardiovascular and respiratory systems. Since it represents the maximal rate at which energy can be released in the presence of oxygen, it may serve as an indication of one's potential for endurance-type activities. Bearing in mind that other energy pathways are also used during activity, it is obviously not a measure or predictor of performance as such.

Your $\dot{V}O_2$ max value: .....$ml.kg^{-1}.min^{-1}$

Normative data ($ml.kg^{-1}.min^{-1}$):

$\dot{V}O_2$ max	FEMALES	$\dot{V}O_2$ max	MALES
<30	POOR	<32	POOR
30-34	FAIR	32-34	FAIR
35-41	AVERAGE	41-51	AVERAGE
42-50	GOOD	52-60	GOOD
>50	VERY GOOD	>61	VERY GOOD