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Effects of protective clothing on cardiovascular and thermal responses to heat stress

Alison Louise Fogarty

University of Wollongong

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EFFECTS OF PROTECTIVE CLOTHING
ON CARDIOVASCULAR AND THERMAL
RESPONSES TO HEAT STRESS

A thesis submitted in partial fulfilment of the requirements for the award of the degree

Masters of Science (Hons)

from

University of Wollongong.

by

Alison Louise Fogarty, BExSc (Hons).

Department of Biomedical Science
2002
I, Alison Louise Fogarty, declare that this thesis, is submitted in partial fulfilment of the requirements for the award of Master of Science (Hons), in the Department of Biomedical Science, University of Wollongong, is wholly my own work unless otherwise referenced or acknowledged. The document has not been submitted for qualifications at any other academic institution.

Date: 20/11/02

Alison Louise Fogarty
Abstract

Research into the physiological impact of thermal protective clothing is dominated by field studies, limiting the number of variables that could be measured. This project sought to investigate the underlying mechanisms of heat strain in firefighters. Thus, a laboratory-based examination was undertaken, where seven males completed two trials of semi-recumbent, intermittent cycling (39.6°C, 45% relative humidity), wearing either the NSW Fire Brigades' thermal protective ensemble, or shorts (control). Mean core temperature (\(T_c\): oesophagus, auditory canal, rectum), mean skin temperature (\(T_{sk}\)), cardiac frequency (\(f_c\)), stroke volume (\(Q\)), cardiac output (\(\dot{Q}\)), mean arterial pressure (MAP), forearm blood flow (\(Q_f\)), change in plasma volume (\(\Delta PV\)), chest mean sweat rate (\(m_{sw}\)), and overall sweat loss were measured. In the uniform trials, subjects experienced significantly shorter times to fatigue (52.5 versus 58.9 min), which occurred at lower peak work rates (204.3 versus 277.4 watts). Furthermore, greater average \(T_c\) (37.9 versus 37.5°C) and \(T_{sk}\) (37.3 versus 36.9°C) \((P<0.05)\) were observed. Despite a greater overall gross sweat loss (923.0 versus 547.1 g·m\(^{-1}\)·hr\(^{-1}\)) in the uniform trial \((P<0.05)\), \(\Delta PV\) remained equivalent. There was a significant interaction between time and clothing on \(f_c\), indicating that as time progressed, the effect of the uniform on \(f_c\) became more powerful. Clothing had a significant effect on average \(f_c\), (133.8 versus 120.5 b·min\(^{-1}\)), and average \(\dot{Q}\) (14.3 versus 12.2 l·min\(^{-1}\)). However, there was no main effect on \(Q\) (107.5 versus 100.2 ml), indicating that the higher \(\dot{Q}\) was chronotropically driven. Furthermore, there was no main effect on \(m_{sw}\), or average \(\dot{Q}_f\). During the uniform experiment, subjects experienced greater thermal and cardiovascular strain. However, while the uniform reduced exercise tolerance, it did not affect the exercise mode, posture, or climate-specific (temperature and humidity) cardiovascular responses observed at the point of volitional fatigue. These results indicated that, when performing work at high intensities in hot conditions, homeostasis is already significantly compromised and that the additional stress imposed by the thermal protective ensemble presents a negligible, further impact upon physiological control.
Acknowledgements

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• My seven subjects who willingly gave up their time to be prodded and probed in the name of science.

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CHAPTER ONE: INTRODUCTION

1.1 Introduction

Firefighting places a significant physiological strain on the body. This strain has several components. First, the environment in which firefighters are working is uncontrolled with high ambient temperatures, high radiant heat and possibly toxic fumes. Second, for protection from this environment, firefighters must wear protective uniforms. These uniforms further increase the physiological load (such as thermal and cardiovascular load) on the body. Finally, to complete the tasks required, firefighters must work at a high intensity, often for extended periods of time. It is essential that the physiological effects of firefighting are understood so that recommendations can be made to limit strain-related injuries (e.g. cardiovascular collapse, and heat stroke). If a firefighter collapses while fighting a fire, not only is his own life in jeopardy, so too are the lives of his colleagues.

The body produces heat through several mechanisms including basal cellular metabolism, muscular activity, and the effects of temperature, epinephrine and thyroxine on cells. To ensure thermal homeostasis, heat loss must equal heat production. During exercise, for every litre of oxygen consumed, approximately 20 kJ of energy is produced from oxidative catabolism of fats and carbohydrates. Only 20% of this energy may be used for useful mechanical work, while the remaining 80% is converted to heat energy. Thus, for someone working at an oxygen consumption rate of 2 l·min⁻¹, the rate of heat production in the body would be about 800 J·s⁻¹ (Gleeson, 1998). If this heat was not dissipated to the environment, core temperature of a 70-kg man would rise 1°C in under ten minutes. Only a small amount of the heat produced by active skeletal muscle is lost through the overlying skin. Instead, most of the heat is transferred by convection to the venous blood returning to the heart. Gleeson (1998) reported that the rate of temperature increase in the quadriceps muscle at the start of high-intensity cycling may be as high as 1°C·min⁻¹. This rate cannot continue as denaturing, and thus, inactivation, of the muscle contractile proteins and enzymes would occur within 10 min. Therefore, most of the heat produced in the muscle is transported to the body’s core.
Despite the broad range of temperatures in which humans can survive, central body temperature is usually regulated within a very narrow range (35° to 41°C). There are four mechanisms by which humans can exchange heat with the environment: radiation, convection, conduction and evaporation. As air temperature (T_a) increases, mean skin temperature (\( \bar{T}_{sk} \)) also rises but core temperature (\( \bar{T}_c \)) remains relatively stable. As T_a converges with \( \bar{T}_{sk} \) and the temperature gradient between T_a and \( \bar{T}_c \) decreases, the contribution of conduction, convection and radiation in heat dissipation (dry heat exchange) from the body is reduced. This results in evaporation being the predominant avenue for heat loss under such circumstances (Pascoe et al., 1994).

The primary purpose of firefighting uniforms is protection from the hostile working environment. Nunneley (1989) suggests however, that thermal protective clothing has a significant, detrimental effect on work in the heat. First, it increases the metabolic rate required to perform a task, by adding mass and restricting movement. Extra movement may also be required to counteract decreased visual fields, or loss of manual dexterity. The restriction of movement is especially pertinent to firefighters (Gordon et al., 1983). Second, the clothing decreases the body’s ability to lose heat via evaporation. Sweat produced may evaporate from the skin surface, or be wicked into layers of the clothing. From either the skin surface, or the clothing, sweat may drip, remain trapped or evaporate. Yet, for maximal heat loss, sweat must vaporise from the skin surface (Nunneley, 1989). When sweat evaporates from the clothing, most of the dissipated heat comes from the environment and not the skin (Craig and Moffitt, 1974; Candas et al., 1987).

For evaporative heat loss to occur, heat produced must first be transported to the skin. Changes in skin blood flow (\( Q_{sk} \)) are controlled by \( \bar{T}_c \) and \( \bar{T}_{sk} \) (Wurster et al., 1966). A \( \bar{T}_c \) threshold must first be reached before an increase in \( Q_{sk} \) will occur (Kenney and Johnson, 1992). Mean skin temperature offsets this threshold without influencing the slope of the relationship between \( Q_{sk} \) and \( \bar{T}_c \) (Wenger et al., 1975). Exercise also has an influence on this relationship, delaying the threshold at which \( Q_{sk} \) increases (Kellogg et al., 1991b). Three major mechanisms are responsible for the increase in
\( \dot{Q}_{sk} \) observed during thermal stress: decreased sympathetic vasoconstriction, increased sympathetic cutaneous active vasodilation and the direct, local effects of skin temperature (Johnson and Proppe, 1996). This increase in \( \dot{Q}_{sk} \) is not limitless, however. As first demonstrated by Brengelmann and colleagues (1977), once \( T_c \) approaches 38°C, the increase in \( \dot{Q}_{sk} \) is attenuated. To date, no \( \dot{Q}_{sk} \) research has been undertaken in an industrial setting or under conditions in which the impact of clothing is evaluated on physiological performance; thus this project aims to examine the impact of wearing thermal protective clothing on \( \dot{Q}_{sk} \).

When exercise is undertaken, there is an increase in heat production, due to skeletal muscle contraction. This heat production may be up to ten times greater than that observed at rest. For thermal balance to be restored, heat loss must be increased, and \( \dot{Q}_{sk} \) must increase proportionally (Sawka et al., 1996). Initially, however, moderate to high intensity exercise generally evokes cutaneous vasoconstriction (Brengelmann, 1977; Kellogg et al., 1991a; Johnson and Proppe, 1996). Concurrently, there is an increase in perfusion of the active muscles (Sawka and Wenger, 1985). As exercise continues there is competition between the skin and the active muscles for perfusion (Brengelmann et al., 1977; Kellogg et al., 1993; Johnson and Proppe, 1996).

During prolonged exercise in high temperatures, the circulatory system must simultaneously perfuse the exercising muscle and cutaneous vascular beds, to continuously support both metabolism and heat dissipation (Sawka et al., 1996). The increased blood volume in the cutaneous vascular beds may induce reduced cardiac filling and stroke volume (Rowell, 1977; Sawka and Wenger, 1985; Sawka et al., 1996). In addition, sweat secretion can result in a reduction in blood volume (Sawka et al., 1984; Sawka et al., 1996). Consequently, a higher cardiac frequency \( (f_c) \) is required to maintain an adequate blood pressure. A further compensatory measure is reduced perfusion of the splanchnic and renal beds, allowing a redistribution of blood to skeletal muscle and cutaneous sites (Rowell, 1977; Sawka et al., 1996). If however, adequate perfusion of the skin and active skeletal muscles cannot be maintained, reduced work performance and possibly hyperthermia may ensue (Sawka et al., 1996).
If, in addition to prolonged exercise and high temperatures, protective clothing is worn, the challenge to the circulatory system should be augmented (Barnard and Duncan, 1975; Manning and Griggs, 1983; Smith et al., 1994). The current investigation will explore the relationship between wearing thermal protective clothing whilst exercising in the heat, and the physiological function of the circulatory system.

It has been shown in endurance athletes exercising at 70-80% of their peak oxygen consumption ($V_{O2peak}$), that when either dehydration or hyperthermia are experienced in isolation, stroke volume ($Q$) decreased by 7-8% but $f_c$ increased sufficiently to maintain cardiac output ($\dot{Q}$) (Gonzalez-Alonso et al., 1997). When dehydration and hyperthermia were both present, however, $Q$ decreased by 20% and $f_c$ was unable to increase sufficiently resulting in a 13% decrease in $\dot{Q}$ (Gonzalez-Alonso et al., 1997). It is foreseeable that, when thermal protective clothing is worn while working in a hot environment, both dehydration and hyperthermia will occur. Furthermore, Smith et al. (2001) measured $Q$ while subjects performed simulated fire drills. Subjects repeated the 7-min fire drill three times with minimal rest. They reported significant decreases in $Q$ after each trial. Unfortunately, thermal strain was measured with an infrared tympanic thermometer only, so minimal conclusions can be drawn regarding the thermal strain encountered. The effects of these three variables (hyperthermia, dehydration and protective clothing) in unison have yet to be fully explored.

While not investigating the magnitude of the effect of thermal protective clothing, Cheung and McLellan (1998) studied the effect of hydration status and fluid replacement on heat tolerance in men wearing nuclear, biological or chemical protective clothing. This and the previously discussed study by Smith et al. (2001) appear to be the only studies that have measured $\dot{Q}$ while protective clothing was worn. Subjects performed a heat-strain test on a treadmill until exhaustion in 40°C (30%RH). When the subjects performed the trial dehydrated, $f_c$ was significantly higher from 25-min onwards. In both hydration states $Q$ decreased steadily throughout the trials with the $Q$ in the dehydrated state being significantly lower. As $f_c$ was increased however, $\dot{Q}$ was maintained and increased throughout the trials in both
Haemoconcentration occurs during exercise in the heat. The magnitude of this change is partially dependent on body posture, with Diaz et al. (1979) reporting greater haemoconcentration in an upright posture than for semi-recumbent or supine postures at rest. In the same study, when exercise was undertaken, an initial, rapid loss of plasma volume (PV) occurred when semi-recumbent or supine. As exercise continued, the rate of haemoconcentration was reduced. Nevertheless, after 45 min of light to moderate exercise PV had decreased by 7.1 and 11% respectively. Furthermore, Nadel et al. (1979) reported a 16.5% decrease after 20 min of heavy exercise at 36°C. The only study (to the best of our knowledge) that measured PV while wearing thermal protective clothing was conducted by Duncan et al. (1979) who observed a 3.3% decrease in PV following 15 min of moderate exercise at 42°C. They did not, however, control for postural changes, which could contribute to the changes observed.

Three different methods of evaluating the physiological effects of protective clothing in the heat can be employed. One method is to expose human subjects, wearing thermal protective clothing, to a hot environment, and measure pertinent physiological variables. The second and third methods do not require human subjects. Instead, guarded hot plate testing and manikin testing, or computer modelling, are used to predict the physical and physiological responses to a particular uniform (Levine et al., 1998). The object of all methods is to determine physiological limits so that dangerous heat strain does not occur, yet, there are several systematic errors when using computer models (Nunneley, 1989). For instance, for computer modelling to be effective and reliable, certain limitations need to be overcome. Nunneley (1989) reports consistent underestimation by modelling equations, when predicting the amount of sweat evaporation in subjects wearing heavy uniforms walking on a treadmill. Another limitation is the exclusion of the effect of body movement in hot plate and manikin testing and computer modelling. Body movement increases heat exchange by increasing convection through the microclimate. This can change the
vapour-barrier characteristics and the insulation provided. The convectional heat exchange between the microclimate and the external environment can also be modified by individual factors such as garment fit, permeability, porosity, and size, as well as positioning of apertures in the uniform (Nunneley, 1989; Xu and Werner, 1997). In contrast, controlled laboratory trials provide a comprehensive evaluation of the total physiological impact of the protective clothing on the wearer.

Several studies investigating the physiological effects of firefighting have already been undertaken. O'Connell et al. (1986) demonstrated that for the same work (stepping at 60 steps·min\(^{-1}\), for 5-min) a firefighter’s personal protective equipment ensemble increased the mean \(V_o2\) from 45\% of \(V_{o2peak}\) without the clothing ensemble to 80\% of \(V_{o2peak}\) with gear. A similar trend was seen with \(f_c\), increasing from 71\% of \(f_{cmax}\) without the gear, to 95\% of \(f_{cmax}\) with gear. Other researchers have also demonstrated that firefighters do attain close to maximal \(f_c\) while working (Barnard and Duncan, 1975; Lusa et al., 1993; Smith and Petruzzello, 1998). In an applied setting, Barnard and Duncan (1975) monitored \(f_c\) in firefighters while working. They reported \(f_c\) values between 175-195 beats·min\(^{-1}\) during the first 3-5 min of a fire. High \(T_c\) and \(T_{sk}\) have also been recorded in firefighters (Faff and Tutak, 1989; Montain et al., 1994; Smith et al., 1997) White and colleagues (1991) compared light work clothing and a chemical protective suit in cool, thermoneutral and hot environments. They found that only \(T_a\) had a significant effect on \(T_c\), while both the chemical suit and increased \(T_a\), increased \(T_{sk}\) significantly. Several studies have been performed in simulated or real fire environments (Barnard and Duncan, 1975; Lusa et al., 1993; Smith and Petruzzello, 1998). These provide invaluable insight into the conditions firefighters have to work in, but do not investigate the mechanisms underlying the physiological responses. For example, the nature of the applied designs and methods used, limits the frequency of data collection, and the types of variables which can be measured. The majority of studies have limited themselves to three or four measures, used non-reproducible protocols and uniforms with specific properties. Therefore, it is not possible to compare studies and to draw general conclusions concerning the mechanisms causing heat strain in firefighters. This project will explore these
mechanisms. To date, no study has simultaneously measured local and overall sweat
rates; mean body temperature (from three sites), $\bar{T}_{sk}$, numerous cardiovascular factors
(such as $f_c$, $Q_{sk}$, $Q$ and $Q$) and changes in plasma volume in a controlled environment.
Until the relationships between protective clothing and physiological strain are
understood, recommendations to minimise heat injuries in firefighters cannot be
optimised.

1.2 Aims and hypotheses
This project was designed to explore the physiological mechanisms contributing to the
physiological strain experienced when wearing thermal protective clothing.
Experiments were performed with subjects in both fully-clothed (firefighter protective
ensemble) and semi-clothed states. As this project focussed upon the mechanisms
underlying field-based observations, all trials were performed within a controlled
testing environment, and were not designed to replicate the working environment.

It was hypothesised that:
(i) Thermal strain, as measured by core temperature, skin temperature, $f_c$ and sweat
rate, would be greater while wearing the thermal protective ensemble, than during the
control trial.
(ii) There would be a smaller decrease in plasma volume throughout exercise when
subjects performed the control trial.
(iii) Forearm blood flow, when compared across both trials, would be lower during
the control trials than the clothing trials. Furthermore, during the rest periods,
forearm blood flow would remain elevated when subjects were wearing thermal
protective clothing.
(iv) Cardiac output would increase with exercise intensity in both conditions. The
peak cardiac output observed for the control trials would exceed that of the clothing
trials. However, during submaximal exercise, cardiac output would be greater when
subjects were wearing thermal protective clothing.
CHAPTER TWO: METHODS

2.1 Subjects
Seven physically active males volunteered to participate in this project. The subjects were required to come to the laboratory three times. The initial visit was for a familiarisation session and the subsequent visits were for a control exercise-heat stress trial, wearing minimal clothing, and a clothing exercise-heat stress trial, wearing the NSW Fire Brigades' protective uniform. Table 2.1 summarises their physical characteristics. Women were excluded due to hormonal fluctuations (Tenaglia et al., 1999). Subjects were screened to eliminate those with cardiovascular risk factors and were given a Subject Information Package, and they provided informed consent in accordance with the guidelines of the University of Wollongong Human Ethics Committee before participating in the study.

2.2 Personal protective equipment
During the clothing trial, subjects wore a personal protective equipment ensemble. This included wool pants, a cotton t-shirt, a firefighter's turnout coat (outer: PFZ twill, inner: dual layer wool, PFZ), over-pants and gloves. A firefighter's helmet and flash hood (balaclava) were also worn. The standard breathing mask employed by the firefighters was replaced by a Hans Rudolph oronasal mask (Hans Rudolf inc., Kansas City, U. S. A.) as the standard mask could not be used without extensive instruction (Figure 2.1). Subjects wore sneakers and sports socks during the clothing trials. During the control trials, subjects wore shorts, socks and sneakers only.

2.3 Experimental conditions
All exercise tests were conducted in a climate chamber maintained at 39.6°C (±0.02) and 45.0% (±0.08) relative humidity, in which the black globe temperature averaged 40.0°C (±0.01) and wind velocity was less than 0.05 m·s⁻¹. Baseline data were collected in an air-conditioned laboratory prior to entering the climate chamber.
Table 2.1: Physical characteristics of subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age (years)</th>
<th>Mass (kg)</th>
<th>Height (cm)</th>
</tr>
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<td>S1</td>
<td>20</td>
<td>72.02</td>
<td>174.8</td>
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<tr>
<td>S2</td>
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<td>12.76</td>
<td>8.4</td>
</tr>
</tbody>
</table>

Abbreviations: \( \sigma \) = standard deviation
Figure 2.1: Subject dressed in personal protective equipment with a laboratory mask replacing the standard breathing mask.
2.4 Experimental procedures

2.4.1 Experimental standardisation

Prior to each trial, subjects were asked to avoid strenuous exercise, consumption of caffeine and alcohol 12-h prior to each trial. Fluid intake and diet closely followed a prescribed regimen ensuring low fat intake, uniform carbohydrate intake and adequate hydration during the 12-h period preceding each session. To control for circadian shifts in core temperature, the test sessions for each subject were performed at the same time of day. The two exercise-heat stress trials were at least seven days apart as the literature clearly shows that trials conducted on a weekly basis are not associated with significant thermal adaptation (Barnett and Maughan, 1993; Cotter et al., 1997). The order in which these trials were completed was balanced across subjects, to minimise order effects. During both trials an exercise-heat stress test was performed.

2.4.2 Protocol

2.4.2.1 Familiarisation session

During the familiarisation session, anthropometric measures were taken and the ergometer settings were set and recorded. Subjects were also introduced to the measures being taken in the exercise-heat stress trials. Finally, the subjects were asked to cycle in the clothing ensemble at 50 watts for 5-min in the climate chamber set at 40°C (50% RH). This session was to ensure that subjects were comfortable with the measures to be taken and the thermal strain induced by the high temperatures and the uniform.

2.4.2.2 Subject preparation

Subjects arrived at the laboratory in a rested state. Upon arrival, subjects were asked to void before an initial weight was obtained. Following 25 min of seated rest, subjects were prepared for blood withdrawal and the determination of plasma volume. A 20 gauge catheter, attached to 15 cm of teflon tubing, was inserted into the left antecubital vein and secured with a plastic covering (Opsite Flexigrind, Smith & Nephew, Hull, U.K.). The end of the teflon tubing was also secured with an elastic bandage (Tubifast, Seton Health Care, U.K.). The dead space of the combined
catheter and tubing was less than 1 ml. Ten ml of blood was withdrawn (Li-heparin, 15 I. U. heparin·ml⁻¹), without stasis for the plasma volume background reference. Before each blood sample, 3 ml of fluid was removed from the catheter and discarded. Following each sample, the catheter was flushed with 5 ml of isotonic saline (sodium chloride injection BP 0.9%). A butterfly needle was then inserted into the right antecubital vein for administration of Evan's blue dye (2.5 ml; Evan's Blue Injection 25 mg, New World Trading Corporation U. S. A.) for the determination of plasma volume. A three-way tap valve was attached to the end of the butterfly needle so that both the line and the Evan's blue dye syringe could be flushed with a minimum of 20 ml of isotonic saline (sodium chloride injection BP 0.9%). As the plunger of the syringe, injecting the Evan's Blue dye reached the end of the syringe barrel, a 10-min time was started. The butterfly needle was then removed from the right arm. At 10 min, 10 ml of blood was removed without stasis from the cannula in the left arm; 5 ml of blood (Li-heparin, 15 I. U. heparin·ml⁻¹) for the Evan's blue dye determination and 5 ml (potassium-EDTA 1.6 mg·ml⁻¹ of blood) for the determination of haematocrit and haemoglobin concentration. The catheter was flushed with 5 ml of isotonic saline (sodium chloride injection BP 0.9%), followed by 2 ml of heparinised saline (50 I. U. ·5ml) to prevent blood clotting within the line.

Subjects were then prepared for the measurement of core and skin temperatures, cardiac frequency, cardiac output and stroke volume, sweat rate, and forearm blood flow. Core temperature was measured at three sites; auditory canal, oesophagus and rectum. Subjects inserted their own rectal thermistor to a predetermined depth of 10 cm. An auditory canal thermistor was inserted to a depth of 1 cm, insulated with cotton wool and held in place by waterproof tape. The oesophageal probe was then inserted by a trained technician (see section 2.5.1.1) before skin thermistors were attached at eight sites with a single layer of waterproof tape, with dimensions of approximately 5 cm by 5 cm. For the measurement of cardiac output and stroke volume via impedance cardiography, tape and ECG electrodes were attached and the heart sounds microphone was situated on the left medial pectoralis, adjacent to the nipple and held in place with an elastic band encircling the torso. A $f_c$ monitor was
then fitted around the chest and a sweat capsule was attached using Collodion glue (Mavidon Medical Products, U. S. A.) to the right side of the chest. To measure forearm blood flow, the largest circumference of the right forearm was also measured and marked for strain gauge placement for venous occlusion plethysmography. During a clothing trial, subjects were then aided in putting on the clothing, attaching 3 thermistors within the layers of the uniform. The sweat capsules for measuring clothing humidity had been attached prior to the trial. If it was a control trial, subjects imitated putting on the clothing ensemble, to negate the effect this action had on plasma volume.

2.4.2.3 Exercise-heat stress protocol
During each exercise-heat stress trial, subjects performed intermittent, stepped, exercise to volitional fatigue on a cycle ergometer (cycle ergometer: Lode Excalibur Sport, Groningen, The Netherlands). Cycle ergometry has minimal relevance to the firefighter, however, this project focusses more on physiological function than it does on real-world simulation. Since the variables being investigated required both minimal body motion and workload precision, semi-recumbent cycling was the method of choice (Figure 2.2). Figure 2.3 shows the work protocol employed. Each trial had three stages, each lasting approximately 20 min. Between each stage, there was a 5 min rest. The first two stages were performed at constant workloads (100 W and 150 W, respectively). Within each stage, the workload was increased to the set workload over 30 s, then maintained for 5 min before being stepped down to rest over 30 s. Each rest period lasted 30 s, permitting measurement of skin blood flow and stroke volume without motion artefact. After three 5 min stages at a set workload, a longer, 5-min rest period was undertaken. This was designed to mimic the standard rest periods employed in the industry. It also allowed blood to be taken. The workload of the third stage started at 100 W (2.5 min) and then increased to 150 W (2.5 min), before ramping at 8 W·min⁻¹ to volitional fatigue.
Figure 2.2: An example of a subject completing the control (minimal clothing) and the clothing (wearing a protective clothing ensemble) trials while cycling on a recumbent ergometer.
Figure 2.3: The work protocol used during an exercise-heat stress protocol, in which subjects undertook two trials; one wearing a protective clothing ensemble and one wearing only shorts.
2.5 Experimental measurements

2.5.1 Body temperatures
Core, cutaneous and clothing temperatures were measured continuously during each trial. All temperatures were recorded at 0.25 Hz using a data logger (1206 Series Squirrel, Grant Instruments Pty Ltd., Cambridge, U. K.) and later downloaded to a computer for analysis. Core temperature was measured at three sites: oesophagus, rectum and auditory canal. The average of these three temperatures was used to calculate mean core temperature ($T_c$).

2.5.1.1 Oesophageal temperature
An oesophageal thermistor ($T_o$) (Type 401, Yellow Springs Instruments Co. Ltd., Yellow Springs, U. S. A.) was inserted through the nose, to the level of the atria. The insertion length was determined using the following equation (Mekjavic and Remple, 1990):

\[ L = 0.479 \times (\text{sitting height (cm)}) - 4.44 \text{ cm} \]

Prior to insertion, the naro-pharyngeal mucosa was anaesthetised with a topical anaesthetic (Xylocaine, Astra Pharmaceuticals, Sydney, Australia). The thermistor was then inserted by a trained technician. Once the thermistor had passed through the nasal region, the subjects drank water through a straw to prevent the probe entering the trachea, and to facilitate the swallowing of the probe. The volume of water consumed was recorded for the determination of mass changes during the trial.

2.5.1.2 Rectal temperature
Rectal temperature ($T_r$) was measured using a thermistor (Type FF, Yellow Springs Instruments Co. Ltd., Yellow Springs, U. S. A.), inserted 10 cm past the anal sphincter.

2.5.1.3 Auditory canal temperature
Auditory canal temperature ($T_a$) was measured using an ear moulded plug, with a
thermistor protruding 1 cm from the mould. A large piece of cotton wool was secured
over the ear to minimise the effect of the environmental temperature.

2.5.1.4 Skin temperatures

Skin temperatures were measured at eight sites using thermistors (Type EU Yellow
Springs Instruments Co. Ltd., Yellow Springs, U. S. A.) attached with waterproof
tape. Mean skin temperature ($\bar{T}_{sk}$) was determined by the following equation (ISO,
1992):

\[
\bar{T}_{sk} = (T_1*0.07) + (T_2*0.175) + (T_3*0.175) + (T_4*0.07) \\
+ (T_5*0.07) + (T_6*0.05) + (T_7*0.19) + (T_8*0.2)
\]

\text{where:}

\[
\begin{align*}
\bar{T}_{sk} &= \text{mean skin temperature (°C)}, \\
T_1 &= \text{forehead temperature (°C)}, \\
T_2 &= \text{scapular temperature (°C)}, \\
T_3 &= \text{chest temperature (°C)}, \\
T_4 &= \text{upper-arm temperature (°C)}, \\
T_5 &= \text{forearm temperature (°C)}, \\
T_6 &= \text{hand temperature (°C)}, \\
T_7 &= \text{thigh temperature (°C)}, \text{ and} \\
T_8 &= \text{calf temperature (°C)}. 
\end{align*}
\]

2.5.1.5 Mean body temperature

Mean body temperature ($\bar{T}_b$) was determined from the weighted summation of $\bar{T}_c$ and
$\bar{T}_{sk}$, using the following equation (Vallerand et al., 1992):

\[
\bar{T}_b = 0.9 \bar{T}_c + 0.1 \bar{T}_{sk}
\]

\text{where:}

\[
\begin{align*}
\bar{T}_b &= \text{mean body temperature}, \\
\bar{T}_c &= \text{mean core temperature}, \text{ and} \\
\bar{T}_{sk} &= \text{mean skin temperature}. 
\end{align*}
\]
2.5.1.6 Clothing temperatures

The combined influence of the metabolic heat production, the thermal environment and the protective clothing ensemble on the thermal gradient between the core and the environment was assessed using $T_c$ and $T_{sk}$ plus temperatures recorded from three thermistors (Type EU, Yellow Springs Instruments Co Ltd., Yellow Springs, U. S. A.) placed on the outer surface of the t-shirt and the inner liner and the outer surface of the tunic.

2.5.1.7 Calibration

All thermistors used to measure $T_b$ and $T_{sk}$ as well as ambient and clothing temperatures, were calibrated before testing. All probes were placed in a 38-litre water bath (Grant Instruments, Cambridge, U. K.) with a NATA (National Association of Testing Authorities) certified thermometer (Dobbie Instruments, Sydney, Australia). Thermistors used to record $T_{ex}$, $T_{re}$, $T_{sk}$ and clothing temperatures were calibrated over a range from 21°C to 46°C with values being recorded with each 2°C increment. The values were recorded after the temperature had stabilised for 5 minutes. The $T_{sa}$ thermistors were calibrated over a range from 30°C to 40°C. A linear equation was then calculated using the recorded thermistor data and the known temperatures from the NATA certified thermometer ($r=0.99$). These equations, one for each thermistor, were then used to correct the thermistor data recorded during the trials.

2.5.2 Sweat rate

Sweat rate ($\dot{m}_{sw}$) was monitored continuously at 0.25 Hz via a sweat capsule attached on the left side of the chest, approximately 8 cm above the nipple. The sweat measurement system (Sweat Monitor, Clinical Engineering Solutions, Australia) pumped air through a sealed container containing a saturated lithium chloride solution (Figure 2.4). This solution was employed because of its stability and low humidity (11-12% over a temperature range of 25 to 50°C). Once leaving the salt container, the air passed through a rotameter (Platon, Duff and Mactintosh, Australia) where the air flow reaching the sweat capsule was regulated. The air flow was maintained at 0.6
**Figure 2.4:** Schematic of the sweat system. Lines which connect components are air lines (thick) or electrical connections (thin). Only one channel is drawn, beginning with the saturated salt solution and ending with the relative humidity monitor.
l·min\(^{-1}\). At the sweat capsule, eight small holes dispersed the air over a skin area of 3.15 cm\(^2\) (±0.04). The humidity and temperature of the air leaving the capsule was measured downstream (capacitance hygrometer). Sweat rate was calculated from the change in relative humidity of the air leaving the capsule, using the following equation (Taylor et al., 1997):

\[
\dot{m}_{sw} = (\text{rh}_* \times P_{H2Oa} \times m/100 + T_{exp} * k) - (\text{rh}_{exp} \times P_{H2O} \times m/100 \times T_a \times k) / A \quad \text{Equation 4}
\]

where:

- \(\dot{m}_{sw}\) = mass flow of water off the skin (g·cm\(^{-2}\)·min\(^{-1}\)),
- \(\text{rh}_*\) & \(\text{rh}\) = relative humidity entering and leaving the capsule (%),
- \(P_{H2Oa}\) = partial pressure of water vapour of air entering the capsule if it were 100% saturated (mmHg),
- \(\dot{m}\) = airflow through capsule rotameter (l·min\(^{-1}\)),
- \(k\) = 3.464 = water vapour gas constant (mmHg·l\(^{-1}\)·K\(^{-1}\)) and
- \(A\) = 3.15 area of skin under the sweat capsule (cm\(^2\)).

### 2.5.2.1 Determination of the sweat threshold and sensitivity

The sweating threshold was calculated for each subject from the chest \(\dot{m}_{sw}\). The threshold was determined as follows:

(i) 15 second data was graphed against time,
(ii) the average of the first minute was taken as a representation of zero.
(iii) a point was chosen where \(\dot{m}_{sw}\) began to rise above the baseline,
(iv) a linear function was fitted to the data after this point, and a second function was fitted to the baseline data.
(v) simultaneous equations were used to obtain the time (s) where these functions intercepted, the time was taken as the sweating threshold,
(vi) the \(\bar{T}_c\), \(\bar{T}_{sk}\) and \(\bar{T}_b\) that corresponded with this time was taken as the \(\bar{T}_c\), \(\bar{T}_{sk}\) and \(\bar{T}_b\) threshold for sweating.

Sweat rate sensitivity was determined from the \(\dot{m}_{sw}\) data, from onset of sweating until the end of the initial rise in sweating, against the \(\bar{T}_c\). A linear function was fitted to obtain the slope.
2.5.2.2 Protective clothing ensemble humidity

To enable the measurement of sweat as it passed through the layers of the protective clothing ensemble, three channels of the sweat system were modified to draw air from the clothing instead of from the saturated solution container. Figure 2.5 shows a schematic of the system. Air was sucked through the system by a pump outside the chamber. Air travelled from sweat capsules, placed on the outside layer of the t-shirt, the inside liner and the outside layer of the tunic at chest level, to a hygrometer (Sweat Monitor, Clinical Engineering Solutions, Australia), which measured the relative humidity of this air. Between the hygrometer and the pump was a rotameter (Platon, Duff and Mactintosh, Australia) which controlled this airflow. The data were recorded at 0.25 Hz and then manually converted to relative humidity using the calibration data.

2.5.2.3 Calculation of vapour pressure

The vapour pressure of each layer of the clothing was calculated using the following equation (Santee and Gonzales, 1988):

\[ VP = (\exp(16.6536 - 4030.15/t + 235)) \times (RH/100) \]

where:

\[ VP = \text{vapour pressure (kPa)}, \]
\[ t = \text{temperature of layer (°C)} \text{ and} \]
\[ RH = \text{relative humidity of layer (%)} . \]

2.5.2.4 Calibration

The humidity sensors were calibrated prior to testing, at three different relative humidities: 6.6%, 35.9% and 74.8%. A saturated solution of lithium chloride at 21°C was used for the low humidity point. A saturated solution of sodium chloride was used for the mid and high-humidity points as its relative humidity changes dramatically with temperature. The mid and high-humidity points were recorded at 21°C and 40°C respectively. To ensure that the system was completely stable, data were recorded 60
Figure 2.5: Schematic of the sweat system modified to measure clothing humidity. Lines which connect components are air lines (thick) or electrical connections (thin). Only one of the three channels modified is drawn, beginning with the sweat capsule and ending with the pump.
min after the required solution temperature was attained. The values obtained from the calibration were used to derive a calibration curve for each channel, so that actual relative humidity values could be derived. The equations of these curves were then generated, allowing derivations of humidities within the range of 6-75% relative humidity.

2.5.2.5 Determining gross sweat loss
Gross sweat and evaporative losses were estimated from changes in nude and clothed masses respectively (uncorrected for metabolic or respiratory losses; A and D electronic balance, Model No. fw-150k, CA, USA).

2.5.3 Cardiovascular responses

2.5.3.1 Cardiac frequency
Cardiac frequency \( (f_c) \) was obtained from ventricular depolarisation using a Polar heart rate monitor (Model PE3000, or Model Advantage, Polar Electro Sport Tester, Kempele, Finland) and later downloaded to a computer. Data were recorded at 0.25 Hz. Validation of the polar sports testers had previously been performed in our laboratory against a five-lead electrocardiogram during incremental cycling (Figure 2.6; Quinton Q5000; Osborne, 1994).

2.5.3.2 Blood pressure
Systolic and diastolic blood pressure were measured at regular intervals using manual auscultation (Freestyle, Accoson Pty. Ltd., West Sussex, U. K.). Mean arterial pressure was then derived using the following equation:

\[
\text{MAP} = \text{DP} + \left(\frac{1}{3} * [\text{SP} - \text{DP}]\right)
\]

\textit{where:}

- \( \text{MAP} \) = mean arterial pressure,
- \( \text{DP} \) = diastolic pressure, and
- \( \text{SP} \) = systolic pressure.

\textit{Equation 6}
Figure 2.6: Comparison of a Sports Tester to a 5 lead ECG during seated rest, cycle exercise at 150 W, and seated recovery (Osborne, 1994).
2.5.3.3 Cardiac output and stroke volume

Stroke volume (Q) and cardiac output (\(\dot{Q}\)) were quantified indirectly and non-invasively using impedance cardiography (Model 304B, Surcom Inc., U.S.A.), where Q is determined from changes in transthoracic impedance and \(\dot{Q}\) is calculated by multiplying Q and \(f_c\). From the impedance cardiograph two signals were recorded: basal transthoracic impedance (\(Z_0\)); and the first derivative of the change in transthoracic impedance (\(dZ/dt\)). A tetrapolar tape (Cardiograph electrode tape, Instrumentation for Medicine, U.S.A.) system was employed, where 4 tape electrodes, 2 inner and 2 outer were used. The two inner electrodes were placed around the base of the neck and around the thorax at the level of the xiphoid process, and the two outer electrodes were placed at least 3 cm above or below the inner electrodes. The outer electrodes emitted a constant current of 4 mA at a sinusoidal frequency of 100 kHz while the two inner electrodes detected \(Z_0\) and \(dZ/dt\). A 3-lead electrocardiograph signal and phonocardiogram (Model 21050A, Hewlett Packard Inc., Andover, U.S.A.) were also recorded. Data were collected for 15 s at a minute and a half before the end of each exercise period (in the last exercise period, readings were taken every five minutes) and at the start of each rest period. Movement during a recording period can render the signal unanalysable, hence some recordings occurred during the rest periods (Muzi et al., 1985).

The signal was then analysed using a computer program (Impedance Plethysmography, developed by the University of Sydney, using Lab View). One respiratory cycle per recording was analysed and the computer program calculated Q and \(\dot{Q}\) using equation 7 (Kubicek et al., 1966; Kubicek et al., 1970):

\[
Q = \rho \ast \left(\frac{l}{Z_0}\right)^2 \ast \frac{dZ}{dt} \ast LVET
\]

Equation 7

where:

\[
\begin{align*}
Q & = \text{stroke volume (ml)}, \\
\rho & = \text{resistivity of blood (}\Omega\text{cm)}, \\
l & = \text{inner distance between the voltage detecting electrodes (cm)}, \\
Z_0 & = \text{baseline thoracic impedance (}\Omega\text{)}, \\
dZ/dt & = \text{maximum rate of change of impedance during systole (}\Omega\text{·s}^{-1}\text{)}, \text{ and} \\
LVET & = \text{left ventricular ejection time (s)}. \\
\end{align*}
\]
In the analysis, three points per cardiac cycle were selected manually, the B point, which occurred immediately after the aortic valve opened, the X point, which corresponded with the closing of the aortic valve and the top of the C wave. The entire C wave reflects the rate of blood flow ejected from the left ventricle (see Figure 2.7). To determine Q and $\dot{Q}$, left ventricular ejection time (LVET), $Z_0$ and $dZ/dt$ were calculated (by the computer program). The LVET was measured as the time between the B point and the X point. The vertical difference between the top of the C wave and the B point represented $dZ/dt$ while $Z_0$ was taken directly from the waveform. The electrocardiogram and the phonocardiogram aided identification of the above points when noise was present in the signal. The distance between the inner electrodes was measured anteriorly and posteriorly with the average entered into the program. The computer program calculated the resistivity of blood from a manually-entered haematocrit value. The haematocrit value was calculated from the first blood sample taken during each trial.

There is some controversy as to the accuracy of impedance cardiography in measuring absolute stroke volumes. Sherwood et al. (1990) and Hatcher and Srb (1986) found that impedance cardiography accurately recorded relative but not absolute changes in stroke volume when compared with another indirect method, $CO_2$ rebreathing. Other researchers, however, have found that this method does correlate with both absolute and relative values obtained with other accepted methods (Du Quesnay et al., 1987; Belardinelli et al., 1996).

2.5.3.4 Forearm skin blood flow

Forearm blood flows ($Q_f$) were derived using venous occlusion plethysmography (EC 4 Plethysmography, D.E. Hokanson, Inc., Bellevue, U. S. A.). Forearm blood flow was measured by stopping venous return from the right forearm while allowing a constant arterial inflow. This was achieved using a pressure cuff (E20 Rapid cuff inflator, Hokanson Inc., U. S. A.) placed around the upper arm, and inflated to 50 mmHg. The cuff was automatically inflated (AG 101 Cuff inflator air source, D. E.)
Abbreviations: ECG = electrocardiogram; \(\frac{dZ}{dt}\) = first derivative of the thoracic impedance signal; PCG = phonocardiogram; B point = point occurring immediately after the aortic valves open; X point = point corresponding with the closing of the aortic valves. C wave = reflects the rate of blood flow ejection from the left ventricle.

Figure 2.7: Example of the cardiac impedance waveform adapted from Sherwood (1990).
Hokanson, Inc. (U. S. A.) for 8-s and deflated for 12-s for a total of 3 inflations per minute. As blood flowed into the limb with each cardiac cycle, it caused the limb to swell. This increase in limb size was measured by a mercury-in-silastic strain gauge (Hokanson Inc. U. S. A.) placed around the forearm at its greatest circumference. The arm was placed above heart level by resting it on a support. This maximised venous drainage, while ensuring movement was kept to a minimum. Data were sampled three times a minute, for a period of approximately 2 min, starting 1 min before the end of each exercise period and continuing during the 30-s rest. To eliminate the effects of transient blood flow changes to the hand, a blood pressure cuff was placed around the wrist and inflated to at least 180 mmHg to stop arterial and venous flow. Johnson and Rowell (1975) have demonstrated that blood flow changes in the resting forearm correlate highly with changes in skin blood flow ($Q_s$).

As the strain gauge was stretched, the electrical resistance of a reference current passing through the mercury increased. Analog output from the plethysmography system, sampled at 20 Hz, was transmitted to an IBM compatible computer (Total Peripherals, Notebook 386SX, Sydney, Australia) via an eight channel, 12-bit analog-to-digital convertor (Computer Boards Inc., PPIO-A18, Mansfield, U. S. A.). The changes in the circumference of the strain gauge were transferred to a computer and later converted to volume curves and manually analysed. The $Q_t$ was calculated from the initial slope of the volume curve.

Blood flow was measured by determining the initial slope of the volume curve after any cuff artefact (see Figure 2.8). The cuff artefact is seen as the rapid rise in the curve immediately post-inflation. This is due to blood being pushed back down the arm. A line was drawn through the peaks of the first pulses after the cuff artefact, extending completely across the graph. The volume change from the bottom to the top of the chart is equivalent to the range setting (sensitivity) for that reading. The slope of the line was then determined by calculating the change in volume per unit time. This was calculated by finding the time needed for the line to cross the graph by drawing a vertical line from the point where the line crossed the top of the graph, then
Bits at 5 volts = 448, at -5 volts = -477
Durations from A-B about 42 seconds
Range was set at 29%
Therefore 60/42*2 = 2.9
Blood flow = 2.9 ml/100ml².min⁻¹

Figure 2.8: Example of the method for determining forearm blood flow from venous-occlusion plethysmography.
measuring the time horizontally to where the line crossed the bottom of the chart. The number of times the line would have crossed the chart in a minute is then calculated and multiplied by the range.

2.5.4 Blood analyses

2.5.4.1 Resting plasma volume

Resting plasma volume (PV) was measured using the Evan’s blue dilution technique. After the subject had rested in a reclining chair for 30-min a background blood sample was collected (Li-heparin, 15 I. U. heparin·ml⁻¹). Following this collection, a known pre-weighed volume of Evan’s blue dye (2.5 ml; Evan’s Blue Injection, 25 mg, New World Trading Corporation, U. S. A.) was injected via a cannula. Ten minutes after the dye injection, a blood sample was taken from the opposite arm (Li-heparin, 15 I. U. heparin·ml⁻¹). These samples were stored at 4°C until the end of the trial. Immediately after the trial, the samples were centrifuged at 3500 rev·min⁻¹ for 15 min. Plasma was then separated into 5 ml containers for analysis (See Appendix A for detail).

The analysis of PV is based upon the binding of Evan’s blue dye to plasma albumin and measured using a spectrophotometer (Greenleaf et al., 1979). The absorbances of the background, the 10-min sample and a standard were determined in a spectrophotometer at a wavelength of 615 nm (Shimadzu UV-1601, UV-visible spectrophotometer, Tokyo, Japan). The standard sample was 1 ml of background plasma plus 0.2 ml of an Evan’s blue standard. The Evan’s blue standard contained 1 ml of dye and 49 ml of distilled water. The absorbances were then used in equation 8 (after Campbell et al., 1958):
PV = V * D * v * ε_{std} / (ε_{pl} - ε_{b}) * 1.03

*Equation 8*

where:

PV = plasma volume,
V = volume of Evan’s blue dye injected,
D = dilution of the standard sample,
v = volume of sample extracted (1 ml),
ε_{std} = absorbance of the standard sample,
ε_{pl} = absorbance of the test blood sample,
ε_{b} = absorbance of the blank blood sample, and
1.03 = correction factor for uptake of dye by the tissues (3%).

Prior to the commencement of the trials, an error analysis was performed on the above technique to identify the source of greatest error, so this could be addressed if necessary. Expected values for each component in the equation were decreased by 5%. The results are presented in Table 2.2. The resulting change in the PV obtained was 5% or less. Furthermore, standard food dye was used to mimic the Evan’s blue dye. Approximately 2.5 ml of the food dye was drawn into a syringe, a butterfly needle and a three-way tap valve were attached, and the dye was injected into a beaker. It was then determined how much saline needed to be injected through this system to flush out all visible signs of the dye. The volume of saline required was 20 ml. The change in PV was then determined by changes in Hct and [Hb].

2.5.4.2 Haematocrit and haemoglobin concentration

Blood was collected in K-EDTA (1.6 mg·ml⁻¹ of blood) at the same time as the 10-min Evan’s blue sample, at minutes 20 and 44 of the exercise trial and at the cessation of exercise. One ml of blood was removed from each tube and frozen at -20°C for later analysis of haemoglobin concentration ([Hb]). The K-EDTA tubes were then stored at 4°C for determination of haematocrit (Hct) later that day.

Packed cell volume was determined using the microhematocrit technique (Hct: IEC MB Centrifuge, International Equipment Co., Needham Heights, U. S. A). Plain capillary tubes, 75 mm in length with an internal diameter of 1 mm were used. The
Table 2.2: Error Analysis of the Evan’s blue dye technique for determination of plasma volume. Row one of the table shows expected values for each variable. Rows 2-6 show values with one variable decreased by 5%, the values modified are highlighted.

\[
\text{PV} = \frac{V \times D \times St \times v}{(T \times 1.03)}, \text{ where } V = \text{volume (wt) of Evan's blue dye injected; } D = \text{dilution of standard sample (1:250); } St = \text{absorbance of standard; } v = \text{volume of dye extracted (1 ml); } T = \text{absorbance of TEST sample minus the absorbance of BLANK; and } 1.03 = \text{factor to correct for slow dye uptake by the tissues.}
\]

<table>
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<th></th>
<th>V</th>
<th>D</th>
<th>St</th>
<th>v</th>
<th>T</th>
<th>PV</th>
<th>%</th>
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<td>1</td>
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<td>1</td>
<td>0.027</td>
<td>3171.52</td>
<td>-5.00</td>
</tr>
<tr>
<td>T</td>
<td>2</td>
<td>250</td>
<td>0.168</td>
<td>1</td>
<td>0.027</td>
<td>2876.66</td>
<td>4.76</td>
</tr>
</tbody>
</table>
capillary tubes were filled with blood by capillarity, leaving approximately 15 mm unfilled. One end was sealed with plasticine (Sigillu, Herler, Copenhagen, Denmark). From each blood sample, three tubes were filled to enable determination of Hct in triplicate. Capillary tubes were then centrifuged at 3500 g for 10 min. Once centrifuged, the length of the packed red cells, and the length of the combined packed red cells and plasma sections were measured in mm, using digital calipers with an accuracy of 0.1 mm. Haematocrit was calculated as the ratio of packed red cells to the combination of red cells and plasma. This was expressed as a percentage.

Whole blood was used to determine [Hb] in duplicate using the cyanmethaemoglobin method (Proc. 525, Sigma Diagnostics ®, St Louis, U. S. A.). The reproducibility of the kit had a coefficient of variation of 0.4% (Sigma Diagnostics). Twenty µl of blood were added to 5 ml of Drabkin’s solution. Drabkin’s solution contains sodium bicarbonate, potassium ferricyanide and potassium cyanide. During a 15-min incubation period (at room temperature), the haemoglobin and all its derivatives (except sulthaemoglobin) in the unknown sample were oxidised to methaemoglobin to form cyanmethaemoglobin, using potassium ferricyanide. Methaemoglobin reacts with potassium cyanide to form cyanmethaemoglobin which has maximum absorption at 540 nm, with the colour intensity being related to [Hb]. A series of haemoglobin standards (0, 6, 12 and 18 g·dl⁻¹) were used to quantify [Hb]. The spectrophotometer (Shimadzu UV-1601, UV-visible spectrophotometer, Tokyo, Japan) automatically determined the four-point calibration curve (see Figure 2.9) based on the concentration versus absorbance from the standards and the subsequent samples were automatically converted to concentrations (g·dl⁻¹).

2.5.4.3 Plasma volume changes
Plasma volume changes during the exercise test were calculated using percent change in PV (ΔPV), relative to the basal sample collected, as calculated from changes in Hct and [Hb]. The absolute PV was then calculated, where possible, from the percent change and the resting PV measured with the Evan’s blue dye technique. The ΔPV was calculated using equation 9 (after Strauss et al., 1951):
Figure 2.9: Calibration curve generated from a series of human haemoglobin standards (0, 6, 12 and 18 g·dl⁻¹) that were used to quantify [Hb]. The spectrophotometer (Shimadzu UV-1601, UV-visible spectrophotometer, Japan) automatically determined the four-point calibration curve based on the concentration versus absorbance from the standards and the subsequent samples were automatically converted to concentration (g·dl⁻¹).
\[
\Delta PV = \{([Hb_b] * (1 - Hct_b) / [Hb_a] * (1 - Hct_a)) - 1\} * 100% \\
\text{where:}
\]\n
\[
[Hb_b] = \text{initial haemoglobin concentration},
\]

\[
[Hb_a] = \text{sample haemoglobin concentration},
\]

\[
Hct_b = \text{initial haematocrit, and}
\]

\[
Hct_a = \text{sample haematocrit.}
\]

Subsequently, absolute plasma volume was calculated:

\[
P V = (\Delta PV / 100) \times \text{baseline PV}
\]

\text{where:}

\[
PV = \text{plasma volume, and}
\]

\[
\Delta PV = \% \text{ change in plasma volume.}
\]

This plasma volume was then added or subtracted from the resting PV to calculate the absolute PV for that particular point during the exercise test\(^1\).

2.5.5 Psychophysical variables

Three psychophysical measurements were taken throughout the trial: thermal sensation; thermal discomfort; and rating of perceived exertion. All subjects were provided with the scales prior to the start of the trial, with written and oral instruction on how to use each scale. Recordings were taken during baseline, and at 3, 10, 15, 20, 26, 33, 39, 44 and 51 min and then every 5 min until the end of exercise. The recordings at baseline, 20 and 44 min were for thermal sensation and discomfort only.

2.5.5.1 Rating of perceived exertion

Rating of perceived exertion was measured using a scale developed by Borg (1962). The question was asked, “How hard does the exercise feel in your whole body?” \(\ldots\)

\(^1\) For one subject their duplicate baseline [Hb] was very high in the control trial. When the exercise samples for this trial were compared with their clothing exercise samples, the numbers were similar. Furthermore, as subjects followed rigorous standardisation directions (in regards to hydration and diet) prior to both trials, it was concluded that the high [Hb] was due to measurement error and thus it was replaced with the baseline [Hb] value from the clothing trial for that subject.
and in your legs?" Subjects responded on a scale of 6 to 20 (6 = very, very light; 20 = very, very hard).

2.5.5.2 Thermal sensation
Thermal sensation was monitored using a scale produced by Gagge et al. (1967). The question was asked “How does the temperature of your body feel?”, from which the subject responded on a scale of 1 to 13 (1 = unbearably cold; 13 = unbearably hot).

2.5.5.3 Thermal discomfort
Thermal discomfort was monitored on a scale modified by Gagge et al. (1967). The question was asked “How does the temperature of your body feel?”, from which the subject responded on a scale of 1 to 5 (1 = comfortable; 5 = extremely uncomfortable).

2.6 Data analysis
This experiment was a two-factor (clothing and time) repeated measures design, with two levels of the main effect: clothing ensemble and minimal clothing. When the effects had multiple time points they were analysed using a two-way analysis of variance with repeated measures (ANOVA) procedure, otherwise a paired t-test was used. Alpha was set at 0.05 for all analyses. Variables are reported as means with standard errors of the means (±sem) unless stated otherwise.
CHAPTER THREE: RESULTS

Due to between-subject variations in thermal tolerance, both within and between conditions, not all subjects completed both trials. Six of the seven subjects commenced the final exercise phase during the control trial, while only five commenced this phase in the clothing trials. The duration of the control trial was always significantly greater ($P<0.05$) than the clothing trial, with average durations of 58.9 min ($\pm 3.8$) and 52.5 min ($\pm 4.1$) for the control and clothing trials respectively. A similar significant trend ($P<0.05$) was observed with maximum work-rates of 277.4 watts ($\pm 25.06$) in the control trial, and 204.3 watts ($\pm 17.91$) in the clothing trial.

Subjects started the two trials from a common, resting physiological base, and did not reveal significant differences, throughout the trials, for the following variables: stroke volume$^2$ (101.57±2.71 ml (control) versus 109.92±3.29 ml (clothed)); mean arterial pressure (92.6±0.59 mmHg (control) versus 92.7±0.89 mmHg (clothed)); sweat rate (1.92±0.03 mg·min$^{-1}$·cm$^{-2}$ (control) versus 2.45±0.04 mg·min$^{-1}$·cm$^{-2}$ (clothed)) and rating of perceived exertion for the whole body and legs only (12.6±0.27 (control-body) and 13.5±0.26 (control-legs) versus 13.1±0.27 (clothed-body) and 14.3±0.31 (clothed-legs)). The resting data from these trials were comparable to normal physiological data (Table 3.1).

### 3.1 Body temperature responses

#### 3.1.1 Skin temperatures

##### 3.1.1.1 Local skin temperatures

Local skin temperatures at the nine sites were more uniform during the clothing trials than the control trials (Figure 3.1), and the temperatures averaged across the trial at the scapula, arm and hand sites were all significantly higher ($P<0.05$) during the clothing trials.

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$^2$ Values given here are the averages across the entire trial.

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Table 3.1: Comparison of baseline values with data from the literature.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Experimental</th>
<th>From literature</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>Clothing</td>
<td></td>
</tr>
<tr>
<td>$T_c$ (°C)</td>
<td>36.90 (±0.02)</td>
<td>36.81 (±0.02)</td>
<td>36.8 - 37.1</td>
</tr>
<tr>
<td>$f_c$ (beats·min$^{-1}$)</td>
<td>63.14 (±1.13)</td>
<td>71.67 (±1.22)</td>
<td>66.7 - 89.0</td>
</tr>
<tr>
<td>$Q$ (ml)</td>
<td>82.79 (±6.95)</td>
<td>79.49 (±6.84)</td>
<td>46 - 95</td>
</tr>
<tr>
<td>SBP (mmHg)</td>
<td>119.57 (±2.79)</td>
<td>119.57 (±4.19)</td>
<td>99 - 158</td>
</tr>
<tr>
<td>DBP (mmHg)</td>
<td>73.86 (±1.98)</td>
<td>71.43 (±2.50)</td>
<td>57 - 99</td>
</tr>
<tr>
<td>PV (ml)</td>
<td>3536.59 (±283.10)</td>
<td>3491.84 (±313.90)</td>
<td>3065 - 3856</td>
</tr>
</tbody>
</table>

Abbreviations: $T_c$ = mean oesophageal temperature, $f_c$ = cardiac frequency, $Q$ = stroke volume, SBP = systolic blood pressure, DBP = diastolic blood pressure, and PV = plasma volume.

Figure 3.1 Mean and local skin temperatures averaged across an intermittent rest and exercise trial in 39.6°C, 45% RH. Subjects completed the trial twice, while wearing either minimal clothing (control) or a protective clothing ensemble. Legend: ■ = control mean skin temperature, □ = clothed mean skin temperature, ● = control local skin temperature, △ = clothed local skin temperature. *signifies a significant difference between trials (P<0.05).
3.1.1.2 Mean skin temperature

Between-condition differences in $T_{sk}$ were significant during baseline (overall ANOVA $F = 389.74; 6, 2; P<0.05$), with the clothing $T_{sk}$ being 5.77% ($\pm 0.26$) greater than the control $T_{sk}$ (Figure 3.2A). The higher $T_{sk}$ during the clothing baseline can be attributed to the insulative qualities of the ensemble, thereby minimising the body heat that could be transferred to the environment. The sudden increase in $T_{sk}$ at the start of phase II, is attributed to the entry into the climate chamber. Due to differences in the time taken for this transition, the data from the end of baseline to the start of exercise were eliminated. To demonstrate the dramatic increase in $T_{sk}$ on entry into the climate chamber, Figure 3.2B has been included. Once inside the chamber and exercising, the differences in the $T_{sk}$ response in the two conditions disappeared, with both increasing at a steady rate during the first exercise phase (phase II). From phase III onwards, the rate of increase in $T_{sk}$ was attenuated in both conditions, with temperatures almost reaching a plateau. Although no significant differences ($P>0.05$) were observed, there was a trend for the control values to be slightly lower than the clothing values. The final minute $T_{sk}$ were identical, at 38.6°C ($\pm 0.08$) and 38.7°C ($\pm 0.06$) for the control and clothing trials respectively, despite the control trials continuing for a further 6.4 min ($\pm 1.2$). The $T_{sk}$ averaged across the entire trial was significantly higher ($P<0.05$) in the clothing trials, with the clothing mean 1.13% ($\pm 0.11$) higher than the control value.

As a result of the much lower baseline $T_{sk}$ in the control trials, and the $T_{sk}$ from both trials converging in the final minute, the change in $T_{sk}$ over the entire trial in the clothing trials was significantly lower ($P<0.05$). If the baseline data were removed, however, this change was not significantly different ($P>0.05$).

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3 Unless stated otherwise, the figures presented show the time course of the data averaged across subjects for each condition. In addition, a simple mean and standard error of the mean for both conditions are presented. These values were obtained by averaging over the entire trial phase, and thus do not always fall on the data curve for that condition.
Figure 3.2: Mean skin temperature response (A) during trials, in which subjects performed six phases of alternating exercise (phases II, IV and VI) and rest (phases I, III and V) in 39.6°C, 45% RH. Subjects completed the trial twice, once wearing a protective clothing ensemble and once wearing minimal clothing. Inset graph is mean skin temperature averaged across the entire trial. Data are presented as means and standard errors of the means during each phase. Graph B illustrates the change in mean skin temperature of one subject (control and clothed trials) during the transition from baseline, in an air-conditioned laboratory, to the start of exercise, in a climate chamber. When subject numbers deviated from the original sample size, due to missing data or subject attrition, the sample size is specified adjacent to phase-specific means. * signifies a significant difference between trials ($P<0.05$). Legend: —, ○ and ⌂ = clothed.—, ▲ and ■ = control.
3.1.2 Core temperatures

3.1.2.1 Local core temperature indices

The two conditions resulted in different responses in the three core temperature indices, with Figure 3.3A illustrating these responses during the control trial, and Figure 3.3B illustrating these responses during the clothing trial. In both conditions, $T_{es}$ and $T_{au}$ demonstrated greater sensitivity to the metabolic heat production; increasing during the work phases of the trials (phases II, IV and VI), and decreasing during the rest phases (phases III and V). In contrast, $T_{re}$ showed an almost consistent increase throughout the trials. The response in all three core temperature indices was generally increased in the clothed trials, although, not all subjects showed this response pattern. Figure 3.4A and 3.4B illustrate a typical response observed in most subjects, whereas Figure 3.4C and 3.4D present a subject who had a slightly blunter response, especially $T_{re}$, to the clothing trial, with only slight increases in the three indices.

3.1.2.2 Mean core temperature

Mean core temperature ($T_c$) increased from baseline in both conditions, with the average increase for control and clothing not being significantly different ($P > 0.05$): $2.1^\circ C (\pm 0.15)$ and $2.5^\circ C (\pm 0.17)$ respectively (Figure 3.5). The jump in $T_c$ from phase I to phase II was due to the change in thermal state from the air-conditioned laboratory (approximately $21^\circ C$), where baseline measurements were taken, to the climate chamber. These transient data were removed to normalise for time differences in transferring the subject from the laboratory to the climate chamber and commencing exercise. Upon entering the chamber and commencing the experimental protocol, $T_c$ increased due to the combined effects of metabolic work and the hot environment. During the rest periods (phases III and V), $T_c$ plateaued.

From approximately 15 min, the data for the control and clothing trials separate, with $T_c$ in the clothing trials increasing at a significantly faster rate ($P < 0.05$). Statistical comparisons revealed significant ($P < 0.05$) between-condition differences during the second rest phase, (phase V). The average clothing $T_c$ recorded during phase V was
Figure 3.3: The core temperature response as measured at the oesophagus, rectum and auditory canal during a trial wearing minimal clothing (A), or a protective clothing ensemble (B). Subjects performed an alternating rest (phases I, III and V) and exercise (phases II, IV and VI) in 39.6°C, 45% RH. Legend: = oesophageal temperature, = aural temperature, = rectal temperature.
Figure 3.4: Comparison of a typical response (A and B) with a blunter response (C and D) of two subjects who completed an intermittent rest (phases I, III and V) and exercise (phases II, IV and VI) while wearing either minimal clothing or a protective clothing ensemble in 39.6°C, 45% RH. Legend: = oesophageal temperature, = aural temperature, = rectal temperature.
Figure 3.5: Mean core temperature was computed during control and clothed trials performed in 39.6°C, 45% RH while intermittently exercising on a recumbent cycle ergometer (phases II, IV and VI). Inset graph is the mean core temperature averaged across the entire trial. Data are expressed as means and standard errors of the means for all subjects across each phase. Legend: ○ and □ = clothing, ▲ and ■ = control. * signifies a significant difference between trials ($P < 0.05$).
1.72% (±0.06) higher than the corresponding control values. The final minute $T_c$ did not differ between the two conditions. The average $T_c$ for the entire trial was significantly lower in the control trials ($P<0.05$) at 37.5°C (±0.07). The average $T_c$ during the clothing trials was less than 1% greater at 37.9°C (±0.02).

3.1.3 Mean body temperature
As mean body temperature ($T_b$) is a function of $T_{sk}$ and $T_c$, it reflected the patterns observed in these two measures (Figure 3.6). Thus, the average $T_b$ over the entire trial was significantly higher ($P<0.05$) in the clothing trials. In addition, the control $T_b$ during baseline and phase V, were significantly below ($P<0.05$) the clothing values, with the control values being approximately 1.5% lower.

3.1.4 Thermal gradients
The core to skin thermal gradient was calculated at each data point. Although not significant, this gradient was higher for the control trials during baseline (Figure 3.7A). Upon entry to the chamber, where $T_{sk}$ increased dramatically without a corresponding increase in $T_c$, a reciprocal decrease in thermal gradient was seen. The thermal gradient in both conditions continued to decrease for approximately the first 10 min of exercise, before reaching a plateau, which lasted until the end of the first rest phase (phase III). From this point onwards, a slight increase in the thermal gradient occurred under both conditions, reflecting the increase in $T_c$. For the greater part of both the control and clothing trials, the thermal gradient was above zero indicating potential heat loss to the environment. However, at times, in certain subjects and in both conditions, the thermal gradient fell below zero, indicating heat gain from the environment. Figure 3.7B illustrates a thermal gradient below zero in one subject in both the control and clothing trial. In the control trials, thermal gradients below zero were recorded in four subjects, with the duration below zero averaging 24.3 min (±8.19). In contrast, heat absorption was recorded in five subjects during the clothing trials, but the average duration below zero was only 7.0 min (±3.53). It cannot be assumed, however, that greater heat loss occurred in the clothing trials, as the thermal gradient is simply a reflection of the direction of heat.
Figure 3.6: Mean body temperature was calculated during intermittent rest (phases I, III and V) and exercise (phases II, IV and VI) trials performed in 39.6°C, 45% RH. Subjects undertook the trials in either minimal clothing or a protective clothing ensemble. Inset graph is the mean body temperature averaged across the entire trial. Data are presented as means and standard errors of the means across each phase. Legend: ○ and ⬇ = clothing, ▲ and ■ = control. * signifies a significant difference between trials (P < 0.05).
Figure 3.7: The core-skin thermal gradient (A) during trials conducted at 39.6°C, 45% RH during which subjects performed alternating phases of work (phases II, IV and VI) and rest (phases I, III and V) while wearing either minimal clothing or a protective clothing ensemble. Data are expressed as means and standard errors of the means across each phase. Figure 3.7B illustrates the gradient below zero in one subject in both conditions. Legend: o and — = clothed, ▲ and — = control.
flow between the skin and the core. For heat loss to occur, the heat transferred to the skin, must then be dissipated into the environment.

3.1.5 Summary of body temperature responses

The average $T_{sk}$, $T_c$ and $T_b$ for the entire trial were significantly higher ($P<0.05$) during the clothed trials. Furthermore, $T_c$ was significantly higher during the second rest phase, whereas $T_{sk}$ was significantly higher (both $P<0.05$) for the baseline only. These trends were reflected in the $T_b$ response which was significantly higher ($P<0.05$) during baseline and the second rest phase.

3.2 Cardiovascular responses

In conjunction with the significant between-condition differences ($P<0.05$) in the body temperatures, significant differences were observed in some of the cardiovascular responses, particularly cardiac frequency, and cardiac output.

3.2.1 Cardiac frequency

Cardiac frequency ($f_c$), during both conditions, followed similar patterns, showing a sharp elevation at the start of exercise in phase II (Figure 3.8). At minutes 11 and 16.5 (phase II) and at minutes 35.5 and 42 (phase IV), $f_c$ decreased during the 30-s rest periods, which were incorporated to allow for venous occlusion plethysmography and cardiac impedance recordings. Despite these pauses, $f_c$ continued to rise throughout both exercise phases (phases II and IV), implying that steady state criteria were never met. During the first rest phase (phase III), $f_c$ decreased, but did not return to baseline. At the start of the second exercise phase (phase IV), $f_c$ increased to exceed previous exercising levels, presumably due to the higher work rate. Unlike the previous exercise phase, however, the exercising $f_c$ did not continue to increase with each sub-stage of exercise, suggesting a steady state was achieved. During the second rest phase (phase V), the control $f_c$ decreased to within 5 beats·min$^{-1}$ of the first rest phase $f_c$ (phase II). In the clothing trial, however, the $f_c$ during the second rest phase was almost 20 beats·min$^{-1}$ higher than the first rest phase. Control $f_c$ was consistently
Figure 3.8: The cardiac frequency response for trials conducted at 39.6°C, 45% RH during which subjects completed six phases of intermittent work wearing either minimal clothing (control) or a protective clothing ensemble (clothed). Inset graph is the cardiac frequency averaged for the entire trial. Data are means and standard errors of the means derived within phases and across subjects. Legend: ○ and □ = clothing, ▲ and ■ = control. * signifies a significant difference between trials (P<0.05).
below clothing $f_c$ for phases I-V, and this difference reached significance in the second rest phase (phase V, $P<0.05$). During the final minute (phase VI), there was no significant difference between the two conditions ($P>0.05$), and subjects were working at approximately 98% ($\pm 2.9$) and 93% ($\pm 3.3$) of their age-predicted maximal heart rate, for the control and clothing conditions respectively. Moreover, when averaged across the trial, $f_c$ was significantly higher in the clothing trials. There was a significant interaction between time and clothing on $f_c$. This indicates that, as time progressed, the effect of the uniform on $f_c$ became more powerful. Thus, the longer the duration of the heat exposure, whilst wearing a protective clothing ensemble, the greater the cardiovascular strain.

3.2.2 Stroke volume and cardiac output

Baseline stroke volumes ($Q$) were similar ($P>0.05$) in both the control and clothing trials (Figure 3.9A). During the first exercise phase (phase II), $Q$ increased sharply as a result of commencing exercise, which acted to increase venous return. In both the control and clothing trials, $Q$ continued to increase during this phase. In the second exercise phase (phase IV), initial $Q$ was similar to the initial $Q$ in the first exercise phase. The $Q$ in this phase increased initially and then decreased at the end of this phase. This may imply that the increase in body temperature, and the increase in work rate, resulted in the $Q$ being compromised, perhaps due to peripheral venous pooling. This is supported by the final minute $Q$ which, despite an increase in work rate, was similar to the final $Q$ during the second exercise phase.

Stroke volume was measured during the rest periods immediately after exercise ceased. Therefore these values would not be a true indication of the average values for these rest periods. Although there were no significant differences during the rest periods, there was a trend for the clothing $Q$ to increase while the control values decreased.

Cardiac output ($\dot{Q}$) data are summarised in Figure 3.9 and Figure 3.10. The latter illustrates the raw $Q$, the corresponding $f_c$, and the resultant $\dot{Q}$ for a single subject.
Figure 3.9: Stroke volume (A) and cardiac output (B) were computed during an alternating exercise (phases II, IV and VI) and rest (phases I, III and V) trials at 39.6°C, 45% RH. Subjects wore either minimal clothing or a protective clothing ensemble. Inset graphs are the stroke volume (A) and cardiac output (B) averaged across the entire trial. Data are expressed as means and standard errors of the means across subjects. Legend: ○ and □ = clothing, ▲ and ■ = control. * signifies a significant difference between trials (P<0.05).
Figure 3.10: Typical response data for stroke volume (A), cardiac frequency (B) and calculated cardiac output during an alternating rest (phases I, III and V) and exercise (phases II, IV and VI). Subjects completed the trial twice wearing either minimal clothing (control) or a protective clothing ensemble in 39.6°C, 45% RH. Legend: ○ = clothing, ▲ = control.
showing a typical response. Baseline $\dot{Q}$, during the control and clothing trials, were 4.8 l·min$^{-1}$ ($\pm 0.32$) and 5.6 l·min$^{-1}$ ($\pm 0.67$) respectively (Figure 3.9B). This increased throughout the first exercise phase (phase II) and initially in the second exercise phase (phase IV), before reaching a plateau. This plateau implies that the increase in $f_c$ (Figure 3.8) during this period compensated, at least partially, for the concurrent decrease in $Q$. Cardiac output continued to increase after the second rest period (phase V), with the final $\dot{Q}$ attained 20.4 l·min$^{-1}$($\pm 1.83$) and 19.9 l·min$^{-1}$ ($\pm 2.13$) for the control and clothing trials respectively. Once again, it would appear that $f_c$ increased to compensate for the concurrent decrease in $Q$, with $f_c$ increasing by 62.3 beats·min$^{-1}$ ($\pm 10.50$) and 31.0 beats·min$^{-1}$ ($\pm 8.98$) for control and clothed trials respectively. In the first rest period (phase III), $\dot{Q}$ decreased in both conditions. In the second rest phase (phase V), however, the clothing $\dot{Q}$ continued to increase while the control $\dot{Q}$ decreased. As a result, the clothing $\dot{Q}$ was significantly higher ($P<0.05$) than the control $\dot{Q}$ during this phase. Furthermore, the average $\dot{Q}$ for the entire trial was significantly higher ($P<0.05$) in the clothed trials.

### 3.2.3 Forearm blood flow

Baseline forearm blood flow ($\dot{Q}_f$) was 3.6 ml·100 ml$^{-1}$·min$^{-1}$ ($\pm 0.30$) and 4.7 ml·100 ml$^{-1}$·min$^{-1}$ ($\pm 0.40$) for control and clothing trials respectively (Figure 3.11A). An increase was observed during the first exercise phase (phase II) before reaching a plateau at the beginning of the first rest phase (phase III). In the control trial, $\dot{Q}_f$ continued to increase throughout the second exercise phase (phase IV). In the latter stages of phase V, $\dot{Q}_f$ started to plateau and this plateau was maintained to the end of the trial in both conditions. Furthermore, the final minute $\dot{Q}_f$ were similar at 16.3 ml·100 ml$^{-1}$·min$^{-1}$ ($\pm 2.59$) and 18.8 ml·100 ml$^{-1}$·min$^{-1}$ ($\pm 3.15$) respectively. Although $\dot{Q}_f$ in the clothing trials were consistently above the control values, this difference was significant only during the last five min of the first exercise phase (phase II). Additionally, the change in $\dot{Q}_f$ across the trial was not significantly different ($P>0.05$) between trials (Figure 3.11B).

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Figure 3.11: Forearm blood flow (A) collected during intermittent exercise (phases II, IV and VI) and rest (phases I, III and V) completed in 39.6°C, 45% RH. Subjects wore either minimal clothing (control) or a protective clothing ensemble. Inset graph is the forearm blood flow averaged over the entire trial. Overall change in forearm blood flow (B) was calculated from the difference between the first and last blood flow values. Data are means and standard errors of the means derived across subjects. Legend: ○ and □ = clothing, ▲ and ■ = control. * signifies a significant difference between trials ($P<0.05$).
3.2.4 Plasma volume responses

The change in plasma volume ($\Delta PV$), calculated from the concomitant change in Hct/[Hb], demonstrated reductions at each stage in both conditions (Figure 3.12A). In the first and second stage of exercise, haemoconcentration occurred to a slightly greater extent in the clothing trials. However, this pattern was reversed immediately after the termination of exercise. Overall the $\Delta PV$ during the trials reflected a generalised haemoconcentration, averaging $-11.65\% \pm 3.26$ and $-10.79\% \pm 1.90$ for control and clothing conditions respectively. There were no significant differences in the $\Delta PV$ between conditions ($P>0.05$). Due to dietary lipid contamination of blood samples, it was only possible to calculate absolute plasma volumes (PV) in 4 subjects (Figure 3.12B). The lipid contamination occurred despite subjects being carefully instructed about dietary fat restrictions for the 12 h prior to each trial. The average baseline plasma volumes were 3536.6 ml ($\pm 283.10$) and 3491.8 ml ($\pm 313.90$) for the control and clothing conditions respectively. In the control condition, PV remained unchanged until the sample taken immediately after the subject terminated the trial. The final control PV was 338.0 ml ($\pm 172.06$) less than the control baseline PV. In the clothing trials there was a slight haemodilution after the first exercise phase. This was reversed, however, by the end of the second exercise phase with values at this stage and the end of the trial both showing haemoconcentration. The final clothing PV was 355.4 ml ($\pm 69.65$) less than the clothing baseline value. There were no significant differences between the PV response in the control and clothing conditions ($P>0.05$).

3.2.5 Blood pressure responses

The systolic blood pressure (SBP) response (Figure 3.13A) in both conditions were not significantly different at any point during either trial ($P>0.05$). SBP increased during the first exercise phase (phase II), before almost returning to baseline during the first rest period (phase III). It then increased to a higher level with the higher work rate during the second exercise phase (phase IV) and decreased during the second rest phase (phase V) but did not return to baseline. The final minute SBP were almost identical, however, the duration of the control trials was 6.4 min ($\pm 1.20$) longer.
Figure 3.12: The comparison of the percent change in plasma volume (A) and the absolute plasma volume (B) measured during intermittent work and rest trials conducted in 39.6°C, 45% RH during which subjects wore either minimal clothing (control) or a protective clothing ensemble. Data are expressed as means and standard errors of the means.

Legend: 
- = clothed, 
= control.
Figure 3.13: Systolic (A) and diastolic (B) blood pressure was recorded during intermittent work (phases II, IV and VI) and rest (phases I, III and V) trials in 39.6°C and 45% RH during which subjects wore either minimal clothing or a protective clothing ensemble. Inset graphs are systolic (A) and diastolic (B) blood pressure averaged across the entire trial. Data are presented as means and standards errors of the means across subjects. Legend: ⓞ and = clothing, ♂ and = control.
As with SBP, there were no significant differences between conditions with diastolic blood pressure (DBP) responses \( (P > 0.05) \) (Figure 3.13B). DBP consistently decreased throughout each exercise phase (phases II and IV), implying a decrease in peripheral vascular resistance. The final minute DBPs recorded were similar in both trials, nevertheless, the duration of the clothing trials was considerably less.

Mean arterial pressure (MAP), a better indicator of blood pressure regulation (Sherwood, 1993), increased during both exercise phases (phases II and IV), and returned to baseline values during rest periods (phases III and V), in both the control and clothing trials (Figure 3.14). The final minute values in both conditions were higher than recorded during either of the exercise phases. There were no significant differences in the MAP responses to the two conditions \( (P > 0.05) \) indicating that, under both conditions, despite slightly different \( Q \), \( \dot{Q} \) and \( f_c \) responses adequate MAP was maintained. That is, cardiovascular function was not compromised.

### 3.2.6 Summary of cardiovascular responses

Four key cardiovascular changes were observed. First, \( f_c \) and \( \dot{Q} \) were significantly lower in the control trials when averaged across the entire trial. Second, there were no significant differences between conditions for \( Q \), \( \dot{Q} \), and PV. Third, the \( Q \) response became attenuated in the latter stages of both conditions, with \( f_c \) approaching age-predicted maximal levels to defend \( \dot{Q} \). Finally, adequate MAP was maintained in both conditions.

### 3.3 Sudomotor responses

#### 3.3.1 Local sweat rate

Chest sweating \( (\dot{m}_{sw}) \), started increasing in both trials approximately 2 min after the start of exercise (Figure 3.15A). During the control trial, the \( \dot{m}_{sw} \) responded to changes in metabolic heat production, increasing at the beginning of the two exercise periods, and decreasing during both the two rest periods and the 30-s pauses. In contrast, the clothing ensemble blunted this sensitivity, with the \( \dot{m}_{sw} \) generally increasing throughout the whole trial. Although not significant, the \( \dot{m}_{sw} \) during the
Figure 3.14: Mean arterial pressure was derived during alternating exercise (phases II, IV and VI) and rest (phases I, III and V) trials in 39.6°C, 45% RH. Subjects completed the trial twice, wearing minimal clothing (control) or a protective clothing ensemble. Inset graph in the mean arterial pressure averaged across the entire trial. Data are means and standard errors of the means derived across subjects. Legend: ○ and ⬇️ = clothing, ▲ and ⬆️ = control.
Figure 3.15 Sweat rate (A) was recorded during intermittent exercise (phases II, IV and VI) and rest (phases I, III and V) trials completed in 39.6°C and 45% RH, while wearing either minimal clothing (control) or a protective clothing ensemble. The inset graph shows the average sweat rate across the entire trial. Sweat produced and sweat evaporated (B) were also calculated from mass changes over the trials. Data are expressed as means and standard errors of the means within phases across subjects. Legend: ○ and □ = clothing, ▲ and ■ = control. * signifies a significant difference between trials ($P<0.05$).
clothing trials was consistently higher until the final minute. In the clothing condition at 37-min and 45-min, there were sudden increases in $\dot{m}_{sw}$, partially due to subjects terminating the trial, and the exclusion of their data from that point onwards. During the final rest period (phase V), $\dot{m}_{sw}$ in the clothing condition reached a plateau while the control $\dot{m}_{sw}$ decreased slightly. The final minute $\dot{m}_{sw}$ for both conditions were identical and lower than the $\dot{m}_{sw}$ in phase V. This is due to the inclusion of the final minute data of the two subjects that finished during phase IV.

### 3.3.2 Sweating threshold and sweat rate sensitivity

Figure 3.16 illustrates a typical sweat rate response, and the calculation of the sweating threshold and sweat rate sensitivity. The sweat threshold occurred significantly earlier ($P<0.05$) in the clothing trials. In contrast, the $T_c$, $T_b$ and $T_{sk}$ at which sweating commenced were identical across conditions. Thus, sudomotor control was not modified, but body temperatures were merely displaced upwards when wearing the protective clothing ensemble. In addition, the protective clothing ensemble did not significantly ($P>0.05$) affect the sweat rate sensitivity.

### 3.3.3 Gross sweat loss

Gross sweat losses were significantly greater ($P<0.05$) in the clothing trials when compared to the control trials, subjects losing 1.42% of body weight (±0.23) and 2.14% of body weight (±0.12) during the control and clothing trials respectively (Figure 3.15B). In contrast, the evaporative sweat loss was significantly less during the clothing trials ($P<0.05$), indicating that a considerable amount of sweat was trapped within the layers of the clothing.

### 3.3.4 Summary of sudomotor responses

There were no significant differences in the mean sweat rate as measured at the chest. Sweating commenced significantly earlier ($P<0.05$) in the clothing trials, although the threshold was at the same $T_c$, $T_{sk}$ and $T_b$. Furthermore, there were no significant differences in the sweat rate
Figure 3.16: Typical response data for chest sweat rate (A) sweating threshold (B) and sensitivity determination (C) during an intermittent exercise (phases II, IV and VI) and rest (phases III and V) protocol in 39.6°C, 45% RH.
sensitivity. In contrast, when gross sweat loss was compared, subjects sweated significantly more \( (P < 0.05) \) in the clothing trials but significantly less \( (P < 0.05) \) of this sweat was evaporated when compared to the control trials.

3.4 Psychophysical responses

3.4.1 Rating of perceived exertion

Ratings of perceived exertion (RPE), for both the whole body and legs, were very similar across trials and conditions. Leg RPE was recorded because the leg muscles were the main muscle group used in cycle ergometry and thus indicated local fatigue. During both exercise phases (phases II and IV), RPE scores increased throughout the phase (Figure 3.17A & 3.17B). The final RPE scores were similar across conditions for both whole body and legs at 16.6 \( (\pm 0.65) \), 16.6 \( (\pm 0.65) \), 16.6 \( (\pm 0.43) \) and 16.1 \( (\pm 0.60) \) for control/whole body, control/legs, clothing/whole body and clothing/legs respectively. It must be remembered, however, that the control trials continued for an additional 6.4 min \( (\pm 1.20) \). As the final RPE scores were so similar in the legs in both conditions, it implied that local fatigue was not the cause of the shorter tolerance time during the clothing trials. Furthermore, a score of 16 is only in the range of 'hard' to 'very hard'. Subjects were instructed to exercise to exhaustion, if local fatigue was indeed the limiting factor, it would be expected that the subjects would report RPE scores closer to 20.

3.4.2 Thermal sensation and discomfort

Thermal sensations at baseline, in both conditions, were almost identical, and in the 'slightly warm' to 'warm' category (Figure 3.18A). During the first exercise phase (phase II), thermal sensation scores increased in both conditions and at the end of the phase, the scores were 9.46 \( (\pm 0.19) \) and 10.36 \( (\pm 0.21) \) for the control and clothing trials respectively. During the first rest phase (phase III), decreases in the thermal sensation scores occurred during both conditions, before increasing again in phase IV. Throughout this second exercise phase, the clothing scores increased, and ended this phase at a subjective sensation of 'very hot'; while in the control trial, thermal
Figure 3.17: Rating of perceived exertion for the whole body (A) and the legs (B) during work phases (phases II, IV and VI) of intermittent work and rest trials conducted at 39.6°C and 45% RH. Subjects completed the trial twice wearing either minimal clothing (control) or a protective clothing ensemble. Inset graphs are the rating of perceived exertion for the body (A) and legs (B) averaged across the whole trial. Data are means and standard errors of the means across subjects. Legend: ○ and □ = clothing, ▲ and ■ = control.
Figure 3.18: Thermal sensation (A) and thermal discomfort (B) during intermittent work and rest trials, whilst wearing either minimal clothing (control) or a protective clothing ensemble in 39.6°C, 45% RH. Legend: ○ = clothing, • = control. Inset graphs are the thermal sensation (A) and the thermal discomfort (B) averaged across the whole trial. Data are means and standard errors of the means across subjects. Legend: ○ and ■ = clothing, • and □ = control. * signifies a significant difference between trials (P<0.05).
sensation increased at a slower rate, finishing at 10.08 (±0.20) indicating a rating of ‘hot’. The greater increase in the clothing thermal sensation scores were significantly different \((P<0.05)\) from midway through phase IV and into phase V, indicating that the subjects perceived that the clothing ensemble was hotter. The differences between the control and clothing scores had disappeared by the final minute which were 11.1\((±0.23)\) and 11.6 \((±0.24)\) for the control and clothing phase, respectively. This is probably due to the subjects working at a significantly higher \((P<0.05)\) work rate at the end of the control trials, and thus, reaching similar levels of heat strain.

The average thermal sensation score across the entire trial was significantly \((P<0.05)\) higher in the clothing trial. This was also the case with the thermal discomfort scores where a similar pattern was observed. Thermal discomfort was in the ‘comfortable’ to ‘slightly uncomfortable’ range at baseline in both conditions (Figure 3.18B). In the clothing condition, thermal discomfort increased during the first exercise phase, and ended this phase at a discomfort score of ‘uncomfortable’. In contrast, in the control trials, thermal discomfort increased initially, before plateauing from 15 min until the start of the second exercise phase (phase IV) at 2.06 \((±0.05)\). In the second exercise phase, thermal discomfort increased under both conditions, but the clothing thermal discomfort was significantly higher \((P<0.05)\) throughout ranging from ‘uncomfortable’ to ‘very uncomfortable’, compared to the control ‘slightly uncomfortable’ to ‘uncomfortable’ range. The ratings of thermal discomfort continued to be significantly higher \((P<0.05)\) during phase V, as well as during the final minute of exercise, despite the longer exercise time and higher final exercise intensity in the control trials. The final minute clothing score fell within the ‘very uncomfortable’ to ‘extremely uncomfortable’ range, while the final minute control value was within the ‘uncomfortable’ to ‘very uncomfortable’ range.

3.4.3 Summary of psychophysical responses

There were no significant differences between the two conditions in the rating of perceived exertion for the whole body or legs. In contrast, thermal sensation was significantly higher \((P<0.05)\) in the clothing trials.
during phase IV and V. Furthermore, thermal discomfort was significantly higher ($P<0.05$) in the clothing trials during phases IV, V and VI.

### 3.5 Effects of personal protective clothing

#### 3.5.1 Clothing temperatures

Mean skin, t-shirt ($T_{\text{shirt}}$), inner liner ($T_{\text{inner}}$) and outer surface of tunic ($T_{\text{outer}}$) temperatures all increased upon entry into the climate chamber (Figure 3.19A). The $T_{\text{outer}}$ increased within the first 5 min to air temperature, and then matched ambient temperature for the rest of the trial. The oscillations in $T_{\text{outer}}$ were due to the climate chamber oscillating around the pre-set temperature. These oscillations were also seen to a lesser extent in $T_{\text{inner}}$. These oscillations cannot be seen in the $T_{\text{shirt}}$ implying that the tunic provided considerable thermal insulation. In the second exercise phase (phase IV), $T_{\text{outer}}, T_{\text{inner}}$ and $T_{\text{shirt}}$ reached a plateau and then started to decrease. $T_{\text{sk}}$ however, continued to increase. The higher temperatures within the clothing ensemble inhibiting conductive, convective and radiant heat loss, as well as the increased work-rate, may explain the continual increase in $T_{\text{sk}}$. In the final rest period (phase V), $T_{\text{sk}}$ reached a plateau, while $T_{\text{outer}}, T_{\text{inner}}$ and $T_{\text{shirt}}$ all decreased. This plateau may be a result of the huge reduction in metabolic heat production with cessation of exercise, and to a lesser extent the slight decrease in the clothing ensemble temperatures.

#### 3.5.2 Clothing relative humidity

The relative humidity of the t-shirt ($RH_{\text{shirt}}$), inner surface of the liner ($RH_{\text{inner}}$), and the outer surface of the tunic ($RH_{\text{outer}}$) were all similar during baseline at 57.3% RH ($\pm 2.34$), 56.1% RH ($\pm 1.53$) and 55.9%RH ($\pm 1.05$) for $RH_{\text{shirt}}, RH_{\text{inner}}$, and $RH_{\text{outer}}$ respectively (Figure 3.19B). All clothing RH measures increased upon entry into the chamber. Throughout the entire trial, the relative humidity of all the layers was above the ambient humidity, demonstrating an evaporative gradient away from the body, theoretically facilitating evaporative heat loss. The $RH_{\text{shirt}}$ had the highest humidity throughout the trial due to the sweat production of the body. It reached 85.0% RH ($\pm 1.75$) by the end of the second exercise phase (phase IV). The $RH_{\text{outer}}$ increased more gradually, reaching 67.3% RH ($\pm 1.27$) at the end of the second exercise phase.
Figure 3.19: Temperature (A), relative humidity (B) and vapour pressure (C) measured in different layers of a protective clothing ensemble conducted at 39.6°C, 45% RH during which subjects completed six phases of intermittent work (phases II, IV and VI) and rest (phases III and V). Data are expressed as means of the subjects across subjects at 15-s intervals. Legend: — = shirt, — = inner liner of tunic, — = outer layer of tunic, and •••• = mean skin temperature.

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4 The notches seen in the graph (A) in phase IV are a result of a subject finishing early. As subjects continued until exhaustion the time they finished varied considerably.
(phase IV). The lowest humidity was seen in RH_{inner}, which rose more gradually than RH_{shirt}, and RH_{outer}, reaching only 72.3% RH (±1.19) by the end of the second exercise phase.

3.5.3 Clothing vapour pressure

Upon entry to the chamber, the initial lower temperature of the shirt caused the vapour pressure of the shirt to be the lowest (Figure 3.19C). After 10 min of exercise the vapour pressure of the shirt increased above the vapour pressures of both the liner and the outer tunic, due to an increase in shirt temperature, resulting from increased metabolic heat production, and an increase in humidity due to increased sweating. From this point onwards, the vapour pressure of the shirt was always higher than the liner. This would, therefore, result in a vapour pressure gradient away from the shirt, allowing water vapour to move away from the body. This vapour pressure gradient did not continue through the uniform to the outside environment, however, as the vapour pressure of the outer tunic was always below the pressure of the liner. The vapour pressure of the air, however, was always below the vapour pressure of the uniform at all three measuring sites. The reduction in the temperatures at all three sites during the rest periods (phases III and IV) resulted in a concurrent levelling off of all three vapour pressures.
CHAPTER FOUR: DISCUSSION

The present study was conducted to investigate the mechanisms contributing to the physiological strain encountered when wearing thermal protective clothing. As this project concentrated upon the mechanisms underlying field-based observations, all trials were conducted within a controlled testing environment, and were not designed to replicate the working environment. There were minimal significant, between-condition differences, with the main exceptions being: trial duration, maximal work rate achieved, mean core temperature ($T_c$); mean skin temperature ($T_{sk}$); mean body temperature ($T_b$), cardiac frequency ($f_c$) and thermal sensation and discomfort. This suggests that, when performing work at high intensities in hot conditions, homeostasis is already significantly compromised, and that the additional stress imposed by the thermal protective ensemble presents a negligible, further impact upon physiological control.

4.1 Tolerance times

In this study, the duration of the clothing trials was more than 10% shorter than the control trials. This decrement in tolerance time was much smaller than the 25% reported by Louhevaara et al. (1995) in their study, where subjects performed a maximal test on a treadmill while wearing a fire protection ensemble and breathing apparatus. Furthermore, the authors attributed the reduced tolerance time entirely to the weight of the uniform and breathing apparatus. If this was correct, as the exercise mode employed in the current study was non-weight bearing, no difference in trial duration would be expected. Instead, this discrepancy between results may be due to the differences in the experimental environment. Louhevaara et al. (1995) conducted their study in 18°C, 50% RH, to minimise the effect of high air temperatures. In contrast, the current study was completed in much higher air temperatures (39.6°C and 45.0% RH), with a corresponding increase in environmentally-induced thermal strain.

A study by McLellan et al. (1993) supports the idea that differences in experimental environment can influence responses under such conditions. They investigated the
influence of temperature and metabolic rate on tolerance time when wearing nuclear, biological and chemical protective clothing. They tested three clothing ensembles: combat clothing; combat clothing with a semi-permeable overgarment; and combat clothing with a semi-permeable overgarment and respirator while performing either light or heavy exercise in cool (18°C, 50%RH) or warm (30°C, 50%RH) conditions. It was concluded that, as metabolic rate and environmental temperature increased, work times were progressively reduced, with the work time most reduced when wearing the heaviest clothing.

4.2 Body temperatures

4.2.1 Skin temperatures

Mean skin temperature rose sharply at the start of exercise due to a combination of metabolic heat production and environmental conditions. A slight dampening of this effect can be seen in the clothing trials, due to the insulation within the clothing delaying the effects of the high air temperatures. The lack of significant differences between conditions at each time point for the remainder of the trial indicates that either, under such high air temperatures, the insulative qualities of the uniform had a minimal effect on $T_{sk}$, or, for some reason, the $T_{sk}$ response during the control trials was unusually high. When compared with some studies (Smolander et al., 1984; Nielsen et al., 1990), the $T_{sk}$ during the control trials was somewhat high. However, the hot conditions and the high workloads could account for the high $T_{sk}$ seen in the control trial (Bothorel et al., 1991). Furthermore, these values are not dissimilar to those reported by Gavhed and Holmer (1989) when professional and volunteer firefighters (wearing minimal clothing) performed an incremental cycle ergometer test for 90 min in similar conditions. Moreover, the $T_{sk}$ averaged across the trial was significantly higher in the clothing trials, despite the lack of significant differences at each time point.

In the current study, mean forehead skin temperatures were identical between conditions. This is contrary to Montain et al. (1994) who suggested that the increase in heat around the face was a major contributing factor to the volitional cessation of
exercise. As the head is a major avenue for heat loss, and with the very hot conditions employed in this study, perhaps the maximum amount of heat loss through the head was already occurring in the control studies. This study found that the thermal protective clothing had the most effect on the hands, arms and scapulas. In the case of the hand, this is not surprising as heavy gloves were worn during the clothing trials.

4.2.2 Core temperatures

Core temperature was measured at three sites: the rectum, oesophagus and auditory canal. The latter two measures demonstrated greater sensitivity to changes in metabolic rate throughout the trials. This is in agreement with the literature. It has already been shown that oesophageal temperature responds more rapidly to changes (within approximately 1 min) in central blood temperature than does rectal temperature, with rectal temperature taking up to 11 min longer to respond to a change in core temperature (Cooper and Kenyon, 1957; Aulick et al., 1981). This delay is thought to be due to the comparatively low blood flow around the rectum, which increases the thermal inertia of this tissue bed (Molnar and Read, 1974; Aulick et al., 1981). In contrast, auditory canal temperature responds more rapidly (approximately 4 min), provided it is not influenced by environmental temperature (Piironen, 1970). In addition, auditory canal temperature may be a better indicator of brain temperature (Cabanac, 1986).

The results from the current study demonstrated greater thermal strain, as indicated by higher core temperatures, in the clothing trials. These differences were only apparent in the latter rest stage of the trials. To date, no trials have been performed in similar conditions with either the level of clothing (and hence insulation), the ambient conditions, the work rate or trial duration differing and, thus, effecting the physiological strain imposed. Nevertheless, some comparisons can be made. Core temperatures at exhaustion during the clothing trials were similar to those reported by Montain et al. (1994), despite the shorter duration (180 min versus 25-60 min) and higher work rates (30-43% $V_{O2max}$ versus 50 W to max) of the current study. A study by Smolander et al. (1984) investigated the cardiorespiratory and thermal effects of
gas protective clothing, with subjects completing 30 min on a treadmill at 21 and 41% of their $V_{O2max}$ wearing either shorts (control) or an impermeable gas protection clothing ensemble (clothing). The $T_c$ response at both work rates during both the control and clothing trials was attenuated when compared with the current study. This is somewhat surprising as the permeability of the gas protection ensemble is considerably less than the ensemble used in the current study. The temperate conditions used by Smolander et al. (1984) could account for the lower $T_c$ response, however. The much lower air temperatures would potentially enable greater convective and conductive heat flow away from the body compared to the current study, where the air temperature was higher than the body temperature, thus favouring heat gain from the environment. Disputing this, is a further finding in the same study (Smolander et al., 1984) that, when the suits were ventilated with air at 28 l·min$^{-1}$, to increase evaporative and convective heat loss, there was not a significant decrease in any of the physiological responses at the higher work rate. This suggests that the predominant factor in the differing $T_c$ responses could be the lower work rate employed by Smolander and colleagues (1984).

4.3 Cardiovascular responses

4.3.1 Cardiac frequency

During each rest period, $f_c$ decreased, but did not return to baseline levels. This is due to a combination of the endogenous and exogenous heat loads. When heat stress is encountered at rest, $f_c$ increases by 10-30 beats·min$^{-1}$ (Rowell, 1986b). In addition, once exercise ceased the body was still dissipating the metabolic heat produced during the exercise phase. This is supported by the $\dot{Q}_r$ observed at the start of the rest periods, which was either the same, or higher than the $\dot{Q}_r$ observed just prior to the end of each exercise phase. Unfortunately, it is not possible to say if the level of blood flow to the periphery, as indicated by $\dot{Q}_r$, was maintained during the entire rest period, as $\dot{Q}_r$ was only measured immediately after exercise cessation, and was not measured again until after the next exercise period had commenced.

The $f_c$ response during the clothing trial was significantly greater than the control trial
response in the second rest phase, and when averaged across the whole trial. This is in agreement with the findings of Baker et al. (2000), who observed significantly higher \( f_c \) when wearing a fire-fighter kit while working on a treadmill in a temperate environment. Furthermore, the maximal \( f_c \) recorded during the current study were similar to those recorded by firefighters working at actual fires (Sothman et al., 1992), indicating that cardiovascular strain encountered in the current study was closely aligned to the cardiovascular strain faced in a firefighter's working environment.

It has long been recognised that firefighters do attain near maximal \( f_c \) while combatting fires (Barnard and Duncan, 1975). The work rate employed in this study, as indicated by \( f_c \), is in accordance with \( f_c \) recorded during trials simulating real-fire situations. Several researchers, when performing simulated firefighter tasks, have reported \( f_c \) from between 72\%-109\% of age-predicted maximal heart rates (Romet and Frim, 1987; Bennett et al., 1995; Smith et al., 1997). In the current study, subjects were working up to 93\% and 98\% of their age-predicted maximal heart rates during the clothing and control trials respectively. A similar trend was seen in core temperature responses with the same researchers recording peak core temperatures (measured at the rectum or auditory canal) between 38.5 and 39.8°C. The present study's range of peak core temperature was 38.4-39.2°C and 38.7-39.6°C for the control and the clothing trials respectively.

### 4.3.2 Stroke volume and cardiac output

Although there were no significant differences in \( Q \), \( \dot{Q} \) was significantly higher when averaged across the trial and during the second rest phase (phase \( V \)). Despite the significantly higher \( \dot{Q} \) in phase \( V \), final minute values of stroke volume (\( Q \)) and cardiac output (\( \dot{Q} \)) in both conditions were almost identical. These values, however, were slightly lower than those recorded by Montain et al. (1998) during their study in which heat-acclimated subjects performed exercise at 65\% of their \( V_{O_2\text{max}} \) for 50 min

\(^5\) Where age-predicted max heart-rates were not presented in the article, they were approximated from the average max \( f_c \) and the average age of the subjects.
in a hot climate (30°C, 50% RH). After 40 min of treadmill exercise, Q and \( \dot{Q} \) were 119±18 ml and 19.7±2.5 l·min\(^{-1} \) respectively. The slightly lower values recorded in the current study may be a result of the heat acclimation, as stroke volume is increased when acclimated, (Wyndham et al., 1968), as well as the different exercise modes used.

In both conditions Q started to decrease towards the end of the second exercise phase and did not increase during the final exercise stage, despite the increase in work rate. Notwithstanding the decrease in Q, the heart rate reserves of the subjects were sufficient, so that \( f_c \) could be increased to maintain \( \dot{Q} \) in both conditions. This is contrary to the findings of some authors, who reported reduced Q in conjunction with maximal \( f_c \), resulting in a decreased \( \dot{Q} \) in firefighters performing fire training drills (Smith et al., 2001a) and men performing supine cycling in 40°C (Suzuki et al., 1980). In contrast, a study by Cheung and McLellan (1998) supports the findings of the present study. They investigated the effects of hydration and fluid replacement while wearing nuclear, biological and chemical clothing in 40°C, 30% RH. Subjects completed six trials performing either light or heavy exercise at three hydration states: euhydrated with fluid replacement during exercise, euhydrated without fluid replacement during exercise or dehydrated with fluid replacement during exercise. In all six conditions, Q decreased with a corresponding increase in \( f_c \) in order to maintain \( \dot{Q} \).

A decrease in Q while exercising in the heat is not uncommon. As core temperature rises, cutaneous blood vessels dilate and cutaneous blood flow increases (Johnson et al., 1974; Rowell, 1986a). Simultaneously, the active muscles require oxygen to meet metabolic demands. This oxygen requirement can be met, to some extent, by increasing the oxygen extraction of the muscles, but an increase in blood flow is also necessary. With skin blood flow capable of reaching over 4 l·min\(^{-1} \) while exercising in the heat, and an increasing work rate, requiring higher muscle blood flows, venous return is reduced, filling pressure is reduced and thus Q is decreased (Rowell, 1974; Rowell, 1986a). This phenomenon is referred to as cardiovascular drift and is usually
accompanied by corresponding decreases in central venous pressure, central blood volume and pulmonary and systemic arterial pressures as well as $f_c$ creep (Rowell, 1986a). In the current study, $f_c$ creep was observed with $f_c$ increasing during the first and second exercise phases, although there was no change in work rate. There was not, however, a corresponding decrease in mean arterial pressure, although there was a plateau during the second exercise phase. As central venous pressure, central blood volume and pulmonary arterial pressures were not measured, it is impossible to ascertain if indeed cardiovascular drift did occur.

In order to defend $Q$ despite decreases in $Q$, $f_c$ increased. Although not measured in this experiment, it has also been shown that as exercise in the heat continues, splanchnic and renal blood flow continue to vasoconstrict in direct proportion to increasing $f_c$. It has consistently been shown that this decrease commences when $f_c$ reaches 100 beats·min$^{-1}$. It has further been demonstrated that a concurrent increase in norepinephrine concentration occurs, indicating greater sympathetic activity (Rowell, 1986a). The relationship between heart rate and splanchnic and renal blood flows is not modified, however, if the level of sympathetic activity is altered, such as during high body temperature (Rowell et al., 1965; Rowell et al., 1987), prolonged exercise or progressively increasing exercise intensity (Rowell, 1983).

4.3.3 Forearm blood flow

Under both conditions, forearm blood flow ($Q_f$) reached a plateau towards the end of the second exercise phase and did not increase further, despite a continual increase in core temperature. This is in agreement with Brengelmman et al. (1977), who investigated $Q_f$ during exercise at high oesophageal temperatures ($T_{es}$). They reported an attenuated increase in $Q_f$ per degree increase in $T_{es}$ above 38°C. They further suggested that at this point the need to maintain mean arterial pressure begins to outweigh the thermally-induced cutaneous vasodilation (Brengelmann et al., 1977). This phenomenon was further investigated by Nielsen et al. (1984). They minimised the displacement of blood into the cutaneous veins, when exercising in the heat, by immersing subjects in hot water. The hydrostatic pressure of the water enabled the
maintenance of a higher Q and, thus, the circulatory system was more able to cope with the combined heat and exercise stress.

4.3.4 Plasma volume responses

Final plasma volumes in both conditions decreased from baseline values (by approximately 12% and 11% for the control and the clothing trials respectively) indicating haemoconcentration. To the best of our knowledge, the only other study that has measured plasma volume changes while wearing thermal protective clothing in a controlled environment was conducted by Duncan et al. (1979). They recorded a 5.2% decrease in subjects wearing a firefighter’s turnout suit while undertaking moderate exercise for 15 min in 42°C. This is a greater reduction, when compared with the 2.7% decrease observed after 20 min of exercise, in the current study. As Duncan et al. (1979) did not control for changes in posture, it is difficult to attribute the plasma changes observed in their study to the effect of exercise and heat alone. In addition, the different exercise modes employed may also be an influencing factor. It is also not clear whether the second blood sample was taken at the cessation of exercise or after the 5 min recovery period. Plasma volume levels can start to return to pre-exercise levels immediately after exercise cessation (Harrison et al., 1975). In a recent study by Smith et al. (2001b), plasma volume was measured during live-fire fighting drills lasting approximately 28 min (3 x 5.5-6.5 min drills, with 10 min rest). They found a comparable (15%) decrease in plasma with the current study. Unfortunately, due to the uncontrolled nature of the drills undertaken, this decrease in plasma volume cannot be attributed to dehydration alone. This highlights the difficulties of measuring plasma volume in an applied setting, as it is difficult to eliminate the effects of posture, exercise type and intensity, and hydration state (Gaebelein and Senay, 1980). In the current study, however, these four variables were controlled.

The influence of posture on the PV response in exercise was investigated by Diaz et al. (1979). When moderate exercise was performed in 49.5°C (31% RH) they reported significant differences in the supine and semi-recumbent postures with
reductions of 11% and 7.2% respectively. In the current study, postural influence was minimised by subjects resting in a seated posture for 30-45 min before the initial blood sample was taken. Subjects were transferred to the cycle ergometer by a wheelchair and a semi-recumbent cycle was used to ensure that a similar posture was maintained throughout the trial. The only unavoidable change in posture was when the subject dressed in the clothing ensemble 10 min before commencing data collection. To minimise this effect, during the control trials subjects imitated this motion.

4.3.5 Blood pressure responses
The current study found no significant differences in the blood pressure response across conditions. This is in agreement with Louhevaara et al. (1995), although their values were somewhat higher. The recumbent cycle posture, as well as the much hotter air temperatures in the current study, would account for this discrepancy. This lack of difference between the blood pressure response indicates that, despite the added thermal strain imposed by the clothing, adequate mean arterial pressure (MAP) was maintained under both conditions.

It would appear that, under both conditions, the cardiovascular system was reaching the upper limit of being able to maintain adequate MAP. Firstly, Q was reduced, and $f_c$ had to approach maximal values so that $\dot{Q}$ could be maintained. Secondly, plasma volume decreased by 10-11%, thereby decreasing the volume of blood available for circulation. In both conditions, however, $Q_r$ decreased at much the same time as Q. This decrease in $Q_r$ indicates that, in order for $\dot{Q}$ to increase to deliver sufficient blood to the active muscles, as work rate increased in the final exercise stage, thermally-induced cutaneous vasodilation had to be reduced. This also suggests that when there is a competition between active muscles and the cutaneous vascular beds for perfusion, the active muscles have priority: a state, which has been well-established for many years (Rowell, 1974).

Another possibility is that when prolonged exercise and heat stress are combined, it is neither the active muscles nor the cutaneous beds that dominate. Instead, the
cardiovascular system attempts to regulate blood pressure (Rowell, 1986a). Our data supports this, as MAP was maintained during both conditions, despite the increasing work rate and continued thermal strain. Nielsen (1984) suggested that baroreflexes impose an upper limit on skin blood flow, and it has also been suggested that as maximal cardiac output is approached, active skeletal muscles vasoconstrict (Rowell, 1986a), thus defending MAP.

4.4 Sudomotor responses
To the best of our knowledge, continuous sweating has not been previously measured while wearing a protective clothing ensemble. The present study indicated, that in high air temperatures, the clothing ensemble did not have a significant effect on the chest $\dot{m}_{sw}$, or the sensitivity of the sweat response. There was a trend however, for the $\dot{m}_{sw}$ to be higher during the clothing trials. This trend is supported by the significantly higher gross sweat loss during the clothing trials. The greater sweat loss in the clothing trials was due to an imbalance between the rate of metabolic heat production and the amount of heat lost via sweat evaporation. In the clothing conditions, the clothing ensemble inhibited sweat evaporation from the skin resulting in an accumulation of heat within the body, and consequently the higher core temperatures (Kenney et al., 1987) and fatigue occurring sooner.

The $T_c$, $T_{sk}$ and $T_b$ at which sweating commenced did not change, although the time taken to reach this point was sooner in the clothing condition. It has been shown that the $T_c$, $T_{sk}$ and $T_b$ sweating thresholds are under central nervous control (Sawka, 1985). We had anticipated that the protective clothing ensemble would not have an effect on central control of sweating, as indicated by sweating threshold, but that it would decrease the sweating sensitivity. It has been shown that pre-heating can modify the central control of sweating (Booth, 2000), however, our subjects started from a common physiological base and, at the commencement of exercise, $T_b$ was not significantly different between conditions. We had predicted that the clothing ensemble would be unable to wick the sweat away from the skin surface, with the skin surface becoming saturated, with sweat production decreasing as a result of
hidromeiosis, thus reducing the sweat rate for a given $T_c$ (Sawka and Wenger, 1985). It would appear, however, that hidromeiosis did not occur, at least on the basis of the current data, as there was no change in sweat sensitivity. This was somewhat surprising as the relative humidity of the shirt approached 85% at the end of the clothing trials. However, the relative humidity of the inner and outer layers of the clothing ensemble were lower than the relative humidity of the shirt, therefore there was a gradient present for sweat movement away from the skin. However, any sweat absorbed into the clothing and not evaporated would not contribute to body heat loss.

The trend for $\dot{m}_{sw}$ to be higher in the clothing condition may not have reached significance because of the location of the sweat capsule. Nadel et al. (1971) investigated regional sweating responses for a given $T_c$. They found that the back and chest had the highest $\dot{m}_{sw}$ for a given $T_c$, whereas the limbs did not start sweating until $T_c$ had reached a higher temperature. As $\dot{m}_{sw}$ was measured at the chest, and given the high levels of thermal strain encountered, perhaps $\dot{m}_{sw}$ at the chest, under both conditions, approached maximal levels. During the clothing condition, however, due to the significantly higher $T_c$, higher $\dot{m}_{sw}$ occurred at other skin regions and hence overall sweat loss was significantly greater.

In addition, it has also been shown that local skin temperature can modify the rate of local sweat production (Ogawa, 1984). When $T_{sk}$ was averaged across the entire trial, it was significantly higher in the clothing conditions. Furthermore, there were significant differences between the following individual skin sites: hand, arm and scapula. Therefore, these higher local skin temperatures could have caused an increased sweat production at these sites, causing the significantly higher gross sweat production in the clothing conditions. It is not clear why local skin temperature effects the rate of sweat production: one theory is that at higher temperatures there is an increase in neurotransmitter released for each neural impulse arriving at the neuroglandular junction (Ogawa, 1984). It is also possible that at higher temperatures, the responsiveness of the sweat gland to the released neurotransmitter is increased (Ogawa, 1970; Ogawa and Asayama, 1986).
In phase III of the trial, control $\dot{m}_{sw}$ was more sensitive to the metabolic heat production, decreasing during this rest pause but increasing as soon as exercise recommenced. Simultaneously, $T_b$ becomes significantly lower in the control trials, indicating that greater evaporative cooling was occurring in that state. In contrast, the protective ensemble inhibited evaporative cooling in the clothing trials. Once again, this is supported by the significantly lower evaporative sweat loss recorded in the clothing trials, and is in agreement with other studies which calculated evaporative sweat loss from changes in body mass while wearing different clothing ensembles (Smolander et al., 1984; Sköldström, 1987).

Due to the exercise mode employed and, thus, the posture adopted by subjects, the rate of evaporative heat loss may have been under-estimated. An important factor in evaporative heat loss when wearing clothing is the flushing of air through the ensemble (den Hartog, 2000). As $\dot{m}_{sw}$ and the clothing humidities and temperatures were measured on the chest, and the torso was kept as stationary as possible, minimal flushing would have occurred. This may, however, be realistic as firefighters often have to wear a breathing apparatus with a harness that would hamper flushing around the torso.

4.5 Psychophysical responses
Both thermal sensation and discomfort were significantly higher in the latter stages of the clothing trial, indicating that the subjects were feeling hotter, and more uncomfortable in the clothing trials. The considerably higher thermal sensation measured during the clothing trials was somewhat surprising as the $T_{sk}$ recorded simultaneously were not significantly higher and it is accepted that thermal sensation is principally related to $T_{sk}$ (Gagge et al., 1967; Gagge et al., 1969). In this investigation, thermal sensation appeared to be more closely influenced by $T_c$. This suggests that the cues for thermal sensation may be different when wearing protective clothing during heavy work in the heat, and thus, the relationship between $T_{sk}$ and thermal sensation may be altered. It has previously been shown by Boutcher et al. (1995) that the relationship between $T_{sk}$ and thermal sensation may not be so concise.
They investigated the effects of individual skin site temperatures on thermal sensation and found that forehead skin temperature had a significantly greater contribution to thermal sensation than other skin sites during exercise in cool conditions.

4.6 Effects of clothing ensemble on physiological responses: Evaluating hypotheses

In the clothing condition, all the temperature indices ($\bar{T}_{sk}$, $\bar{T}_c$, $\bar{T}_b$) were significantly higher across the trial. Furthermore, $f_c$ and, subsequently $\dot{Q}$, were significantly higher to possibly transport the heat from the core to the shell. If this was the case, then we would expect to see significantly higher $\dot{Q}_f$, which was not observed. There was a trend, however, for $\dot{Q}_f$ to be higher in the clothing trials. This may not have reached significant levels for a couple of reasons. First, we measured $\dot{Q}_f$ at only one site, and therefore, it was assumed that forearm cutaneous blood flow was a valid index of $\dot{Q}_{sk}$ throughout the body. This may not be the case, as $\dot{Q}_{sk}$ is a function of local $\bar{T}_{sk}$ and therefore, variations in $\bar{T}_{sk}$ will effect $\dot{Q}_{sk}$. As previously discussed, $\bar{T}_{sk}$ was significantly higher in the clothing trials, and it was also more uniform across the local skin sites. Therefore, it is possible that in the control trials, there was considerable variation in $\dot{Q}_{sk}$ across body regions, resulting in $\dot{Q}_f$ not being a reliable indicator of overall $\dot{Q}_{sk}$. Second, it is assumed with venous occlusion plethysmography that the blood flow to the muscles of the forearm remains constant. Some researchers have shown this to be the case (Detry et al., 1972), while others disagree (Smolander and Kolari, 1985). Therefore, it is possible that the current $\dot{Q}_f$ data is not a true reflection of whole-body $\dot{Q}_{sk}$.

When averaged across the trial, $\bar{T}_{sk}$, $\bar{T}_c$, $f_c$ and sweat rate (as measured by weight changes) were significantly higher in the clothing trials. Therefore, our first hypothesis that thermal strain, would be greater while wearing the thermal protective ensemble, than during the control trial can be accepted.

The lack of significant differences observed in $\Delta PV$ between the clothing and control conditions causes our second hypothesis that a smaller decrease in PV throughout exercise would be observed when subjects performed the control trial to be rejected.
This is somewhat surprising as there was a significantly greater amount of sweat lost during the clothing trials and it has been shown that this can cause a reduction in PV (Sawka, 1985).

Our third hypothesis that forearm blood flow, when compared across both trials, would be lower during the control trials than the clothing trials and that furthermore, during the rest periods, forearm blood flow would remain elevated when subjects were wearing thermal protective clothing can be rejected, although there does appear to be a trend towards this occurring.

The first part of our forth hypothesis can be accepted with cardiac output increasing with exercise intensity in both conditions. The second and third part, however, has to be rejected, as the peak cardiac output observed for the control trials did not exceed that of the clothing trials. It is possible that this is due to the exercise protocol used, and the lack of a finite ending point. This is supported by the clothing \( \dot{Q} \) becoming significantly higher in the second rest phase, although it is impossible to say if this trend continued into the third exercise phase. The third part of hypothesis four said that during submaximal exercise, cardiac output would be greater when subjects were wearing thermal protective clothing. This was not a consistent observation throughout the trials, although it did occur in the second rest phase, and also when averaged across the trial, \( \dot{Q} \) was significantly higher in the clothing condition.

4.7 Thermal properties of the clothing ensemble

The humidity and temperature measurements taken within the clothing were not fundamental to this investigation. They were undertaken to provide an indication of water vapour and heat flow through the protective clothing ensemble. The modified sweat system that was employed sucked air from the area below the attached capsule, with the air obtained taking the path of least resistance. Thus, depending on the properties of the material on which the capsule was attached, the air may have been coming from within the material itself, or being drawn from the air layer on the other side of the material, and passing through the material. We are confident that, due to
the weave of the shirt and the outer layer of the tunic, that this did not occur at the capsules on these sites. In contrast, the inner liner was of a more loose weave, and of a lighter fabric, thus some of the air may have been coming from the layer of air between the liner and the shirt. For a more comprehensive understanding of the dynamics of the heat and water vapour flow, further investigation is warranted.

The temperatures between each layer of the clothing indicate a thermal gradient away from the body, with the temperature increasing to equilibrate with ambient temperature on the outer surface of the clothing ensemble. This would add to the thermal strain by preventing convective and conductive heat loss from the body. In addition, with the t-shirt relative humidity approaching 85% by the end of the trial, and thus high water vapour pressure next to the skin, minimal evaporative heat loss could take place.

Calculating water vapour pressure from the humidity and temperature measurements allowed a greater appreciation of the potential vapour movement through the layers of clothing. For the greater part of the trial, the water vapour pressure of the shirt was higher than the vapour pressure of the tunic and liner. Furthermore, the vapour pressure of the outer layer was consistently higher than the vapour pressure of the liner. The ambient vapour pressure, however, was lower than all three sites throughout the trial. As water passes down its water vapour pressure gradient, the sweat from the shirt should have passed down its vapour pressure gradient to the liner. It could then be concluded that the vapour remained there, as the outer tunic had a higher vapour pressure than the liner. It is our belief, however, that this was not the case. In the final stages of manufacture, the liner was immersed in a water-repellant solution making it hygroscopic. Thus, the hygroscopic properties of this layer wicked the vapour through to the outer layer. As the vapour pressure of the outer layer was higher than that of the ambient air, the vapour could then pass into the environment.

From a practical perspective, to maximise subject comfort, skin wettedness needs to be minimised (Berglund and Gonzales, 1977). Therefore, the material closest to the
skin should not be saturated, so that the sweat produced can be readily absorbed by this material. For this to occur, outer layers need to be able to wick the moisture away from the layer next to the skin to allow for continued evaporation. The use of a hygroscopic agent on the liner of the current ensemble helped to promote this movement of moisture away from the skin.

4.8 General conclusions
This project was designed to explore the mechanisms contributing to the physiological strain experienced when wearing thermal protective clothing. It was found that thermal strain, as measured by $\overline{T_c}$, $\overline{T_{sk}}$, $f_c$, and sweat produced and $\dot{Q}$ averaged across the trial, was greater when wearing protective clothing. However, when looking at the time courses of these variables, the differences only became significant in the final rest stage of the trial, with the final values being similar. Furthermore, the subjects felt significantly hotter and more uncomfortable when wearing the clothing ensemble. Despite this greater thermal strain, there were no significant differences between conditions for either the plasma volume response or the forearm blood flow response. It can be concluded that, under the present conditions of high temperatures and high work rates, the thermal strain upon the body is already high and the additional stress imposed by the thermal protective ensemble presents a negligible, further impact upon physiological control.

4.9 Future research
The current study has confirmed that some degree of thermal strain is encountered while wearing a firefighter's protective clothing ensemble when working at high intensities under hot conditions. However, the underlying mechanisms remain unclear. Due to a lack of significant differences in $\dot{Q}$, PV and $\dot{m}_{sw}$, it was not possible to determine conclusively if dehydration or unsustainable cutaneous blood flow were the cause of the shorter tolerance times in the clothing trials.

It is believed that the hot environment and high work rates may have contributed to the lack of differences between the clothing and control trials. It is therefore,
necessary to examine the firefighter’s protective clothing ensemble under different conditions so that its full impact is understood. Furthermore, the interaction between clothing insulation, hydration status, and fitness status needs to be examined in a controlled environment.

Several researchers have completed studies in lower temperatures (e.g. Baker et al., 2000; Duggan, 1988). None of these studies were comprehensive, however, with either minimal physiological measures recorded, an uncontrolled environment used or the period of exercise so short, that it did not reflect the time a fire fighter would be expected to work in a protective ensemble. Therefore, a series of trials should be done in a controlled environment, taking a full battery of measures, with the air temperature set at several different levels.

Future studies should also attempt to collect $\dot{Q}_{sk}$ and $\dot{m}_{sw}$ at more than one site. First, it would appear from the current study that sweat loss was greater in the clothing trials, although the $\dot{m}_{sw}$ at the chest was not different between conditions. It would be useful to measure $\dot{m}_{sw}$ at several sites, to better understand sweat distribution, so that clothing ensembles could be modified to better facilitate evaporative heat loss. Concurrently, if $\dot{Q}_{sk}$ was measured at several locations the impact of cutaneous vascular demands on evaporative, conductive and convective heat losses could be better examined and protective ensembles modified accordingly.
CHAPTER FIVE: REFERENCES


APPENDIX A: EVAN'S BLUE DYE DILUTION TECHNIQUE

Pre-injection sample (blank)

Equipment
1. Gloves
2. Tourniquet
3. 20 gauge catheter
4. Luer lock
5. Alcohol swab
6. 7 ml heparinised tube
7. Centrifuge
8. Disposable pipette
9. 5 ml vial

Procedure
• Prepare antecubital vein for blood withdrawal.
• Withdraw 7 ml of blood without stasis.
• Mix heparinised tube.
• Centrifuge sample (3500 rpm for 15 min).
• Draw off plasma and transfer to 5 ml vial (1 ml for blank and 1 ml for standard preparation required).

Injection and post-injection procedure

Equipment
1. Powder-free gloves
2. 5 ml vial of Evan’s blue dye
3. 5 ml syringe
4. 20 gauge needle
5. 20 gauge catheter
6. Three-way tap valve
7. Alcohol swabs
8. Lint-free cloth
9. Tourniquet
10. Calibrated scales (4 decimal places)
11. 5 ml heparinised tube
12. Stop watch
13. Centrifuge
14. 5 ml vial

**Preparation**
- Attach 20 gauge needle (with cover) to 5 ml syringe, wipe both with lint-free cloth.
- Weigh needle and syringe to 4 decimal places and record mass.
- Drain dye to bottom of vial and wipe the outside seal with an alcohol swab.
- Open vial in safety cabinet and draw approximately 2.5 ml of the dye into the syringe.
- Remove the needle from the syringe, expel all air from the barrel and push the dye to the tip of the syringe.
- Attach a clean 20 gauge needle (with cover) to the syringe.
- Wipe the syringe, and weigh to 4 decimal places, record mass.
- Remove the needle and cap and inject the dye through three-way tap valve into the butterfly needle.
- Attach a 20 ml syringe of sterile saline to the other tap of the valve.
- Turn the valve so that approximately 5 ml of saline flushes the EB syringe.
- Inject this into butterfly needle.
- Repeat the above.
- With the remaining 10 ml of saline flush butterfly needle and line.
- Wipe syringe, attach needle and cover, then reweigh.
- Subtract the empty weight from the pre-injection weight to determine the amount of dye injected.

**Post-injection sample**
- At 9 min post-injection prepare antecubital vein on the arm opposite to that
used for the EB injection.

- At ten minutes exactly withdraw 3 ml of blood without stasis.
- Transfer blood to a heparinised tube and mix gently.

Plasma samples

- Centrifuge samples (3500rpm for 15 min at 4°C), then draw off the plasma into separate labelled vials.

Standard Solution

- Prior to testing put 1 ml of EB from vial and make up to 50 ml with distilled water in a 50-ml volumetric flask. This can be stored at 4°C for several weeks.

Sample preparation

- In 1st cuvette, place 1 ml of plasma pre-injection (background).
- In 2nd cuvette, place 1 ml of pre-injection plasma and 0.2 ml of EB standard. Mix well and let stand for 2 min (standard).
- In 3rd cuvette, place 1 ml of plasma from the 10 min sample (EB10).

Analysis

- Turn the Spectrophotometer (Shimadzu UV-1601, UV-visible spectrophotometer, Tokyo, Japan) on 30 min prior to reading samples.
- Prepare 4 cuvettes with 1 ml of distilled water in each (these will be used to auto zero the spectrophotometer).
- Select photometric option.
- Set the wavelength at 615nm.
- Autozero the spectrophotometer using the distilled water samples.
- Measure the absorbance of the blank, standard and the EB10 samples.
Calculation

The absorbances were then used in the following equation (after Campbell et al., 1958):

\[
PV = V \times D \times v \times \epsilon_{\text{std}} \div ((\epsilon_{\text{pl}} - \epsilon_{\text{b}}) \times 1.03)
\]

\textit{Equation 11}

where:

- \( PV \) = plasma volume,
- \( V \) = volume of Evan's blue dye injected,
- \( D \) = dilution of the standard sample,
- \( v \) = volume of sample extracted (1ml),
- \( \epsilon_{\text{std}} \) = absorbance of the standard sample,
- \( \epsilon_{\text{pl}} \) = absorbance of the test blood sample,
- \( \epsilon_{\text{b}} \) = absorbance of the blank blood sample, and
- 1.03 = correction factor for uptake of dye by the tissues (3%).