Physiological Responses of Fire Service Training Instructors to Live Fire Training.

Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor in Philosophy.

By

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TEXT BOUND CLOSE TO THE SPINE IN THE ORIGINAL THESIS

Declaration.

The work in this thesis is original and has not been submitted previously in support of any qualifications or course.

Signed:

Date:

Abstract.

The present study has shown that during live fire training exercises breathing apparatus instructors (n = 6) who were instructing trainee fire fighters were placed under short term stress. The factors that contribute to this included; 1) the necessary workload that is carried out during live fire training exercises (24.4% increase compared to resting heart rate); 2) weight from wearing protective clothing and self contained breathing apparatus (further increase of 6.9%); 3) heat storage from wearing protective clothing (additional increase in heart rate of 8.9%) and 4) heat radiated from the fire (heart rate increased by 3.5%, although peak heart rate responses further increased by 25.8%).

During days when breathing apparatus instructors drank during the live fire training exercises, they were in positive fluid balance (220.8 (± 448.4) ml) compared to being in negative fluid balance (-1009.2 (± 1629.6) ml) during days when euhydrated prior to exposure. There were lower skin (-0.7 (± 0.9) %) and micro-climate (-4.2 (± 4.3) %) and mean heart rates (-23.2 (± 13.8) %) when breathing apparatus instructors exited the fire flashover unit to drink 200 ml of water compared to when they were euhydrated prior to exposure. This suggests leaving the fire flashover unit to drink during the live fire training exercises lowers the stress response. The study showed that hydrating the breathing apparatus instructors prior to exposure did not appear to lower the short term stress responses produced during the live fire training exercises.

The increase in heart rate on exposure to live fire training exercises (as measured by heart rate variability) was mediated by a 21.5 (± 32.7) % increase in the sympathetic control and a -53.6 (± 69.8) % decrease in the parasympathetic control of heart rate. There were no beneficial effects of hydrating breathing apparatus instructors prior to exposure on the sympathetic or parasympathetic control of heart rate (there were still increases in the sympathetic indicators of heart rate control (13.4 (± 28.6) %) and decreases in the parasympathetic indicators (-75.0 (± 45.2) % between pre and post exposure measurements). However, less stress was placed on the breathing apparatus instructors when they drank during exposure compared to when they were either hydrated or euhydrated prior to exposure. This was evidenced from the abolition of significant changes in the sympathetic or parasympathetic indicators of heart rate control. In addition, exposure to heat appeared to produce the same heart rate variability responses that post myocardial infarction patients exhibit. It could be hypothesised that repeated exposure to severe heat stress over long periods of time may lead to increased prevalence of heart conditions. Underlying biological factors are usually associated with the aetiology of myocardial infarction. However, there is no reason to suspect that these factors are relevant to the breathing apparatus instructors. However, increased heart rate over time would suggest that further research may be of benefit in this respect.

Salivary cortisol significantly increased immediately post exposure (42.4 (± 32.9) $nmol \cdot litre^{-1}$) when compared to pre exposure (21.6 (± 13.4) $nmol \cdot litre^{-1}$) to the live fire training exercises. Hydrating the breathing apparatus instructors prior to exposure or drinking during the live fire training exercises did not significantly lower this response.

The present study has shown that during exposure to live fire training exercises there were significant increases in the heat stored within the protective clothing. Therefore, to alleviate this heat storage the study recommends that the breathing apparatus instructors exit the Fire Flashover Unit every 10 minutes to drink 200 ml of water during live fire training exercises. However, alternative methods to reduce the heat storage which require further investigation include the wearing of cool-vests and wicking garments to increase evaporative heat loss. The study also recommends that breathing apparatus instructors should ensure they hydrate prior to, during and after live fire training exercises avoiding alcohol, and caffeinated drinks.

Acknowledgements.

My express thanks go to the Breathing Apparatus Instructors of the Greater Manchester Fire Service who were so willing to give up their time, dignity (all that naked weighing!) and bodily fluids in the name of research. This study would not have been possible with out their co-operation and undying sense of humour.

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Gazza, the technical wizard from the Biology department has been with me through thick and thin, with the good elements of the research (getting burnt in the FFU and nearly throwing up!) and the bad elements (too many to mention). Cheers big ears!

Si and Bec. Thank you for just being at the end of the phone and giving me a relaxed safe haven to come to when things got too much. I look forward to seeing much more of you now this is in!

I would also like to take this opportunity to thank those at College not directly involved in the PhD but were always there for me. Thank you Dave Kellett, Pandy Brodie, Kate Irving, Sarah Andrews, the porters (for all those unofficial gym workouts that kept me sane), and Henri Blackman and Gary Lloyd for giving vital CPR to my laptop that inevitably could not keep up with the pace!

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Glossary.

Anti-chamber – Chamber in the FFU where the BAIs debrief the students.

BAIs – Breathing Apparatus Instructors

BIA – Bioelectrical Impedance Analysis

During – where the BAIs were not actively encouraged to drink for 2 hours prior to exposure but exited the FFU to drink 200 ml of water every 10 minutes during the LFTEs.

ECW – Extracellular water

Euhydrated – where the BAIs were not actively encouraged to drink for 2 hours prior to exposure.

FFU – Fire Flashover Unit

Fire-chamber – Chamber in the FFU where the fire exercises are carried out

GK – Gym kit (shorts, t-shirt and trainers)

HF Norm – represents the parasympathetic nervous system

HR – Heart Rate (Beats minute^{-1})

HRV – Heart rate variability

Hydrated – drinking 600 ml of water 1 hour prior to exposure to the LFTEs

ICW – Intracellular water

IM – Ideal mixture – if gases are at their IM, this could potentially lead to ignition of these gases.

LF Norm – represents the sympathetic nervous system

LF:HF Ratio – represents the sympathetic nervous system

LFTEs – Live Fire Training Exercises – carried out in the presence of fire and students.

Mock Training Exercises – Exercises carried out in the absence of fire and students.

PC+SCBA – Protective Clothing and Self Contained Breathing Apparatus

pNN50 – represents the parasympathetic nervous system

Protocol 1 – Investigated the physiological responses to LFTEs compared to mock training exercises.

Protocol 2 and Protocol 3 – Investigated the hypothesis that fluid ingestion would lower the physiological responses to LFTEs when BAIs were either hydrated prior to exposure, euhydrated prior to exposure (Protocol 2) or euhydrated prior to exposure but drank during the LFTEs (Protocol 3).

RER – Respiratory exchange ratio

RMSSD – represents the parasympathetic nervous system

TBW – Total body water

WGK – Gym kit (shorts, t-shirt and trainers) and weighted rucksack (equivalent to the weight of PC+SCBA)

Chapter 1

Introduction.

1.1 Study rationale.

The operational work of the fire service is performed in an environment that is constantly changing and can be inherently hazardous. The purpose of operational training is to contribute to the safe and effective working practices of fire fighters. This is achieved through the regular undertaking of scenario based exercises such as drills and live fire training exercises (LFTEs). The Fire Services therefore have to place great emphasis on ensuring safe systems of work, equipment and a competent workforce.

LFTEs are a regular feature of fire fighter operational training. LFTEs are conducted in a variety of fire-ground scenarios and many Fire Services are now using bespoke Fire Flashover Units (FFU) for simulated training exercises. In this particular context, this type of training is normally conducted by Breathing Apparatus Instructors (BAIs), who, in addition to verbally providing specific instruction and advice, ensure that students carry out exercises in a safe and controlled manner.

The BAIs are not operational as they are not assigned to a fire station and are not on active duty where they answer emergency calls. Instead, they are assigned to a training unit where they act solely as instructors. Whilst there are clear guidelines for instructors on observing their students for symptoms of fatigue and heat stress during LFTEs, these guidelines are less specific in relation to the instructors themselves. During LFTEs the BAIs tend to rely on their personal knowledge and experience in recognising the symptoms of fatigue and heat stress. However, there have been incidents of BAI fatalities within the fire service which have also been documented. The role of the BAIs involves close supervision of their students' welfare whilst undertaking LFTEs. However, there does not currently appear to be a system in place within the Fire Service that monitors the general health, safety and stress levels of these BAIs. Therefore, BAIs were monitored as opposed to fire fighters. The study aims to address whether BAIs have significant stress levels and identify sources of this stress.

1.2 The history of Live Fire Training Exercises (LFTEs).

LFTEs were incorporated into fire fighters' training following a fire in Blaina, South Wales, when two fire fighters died during a routine fire incident (Fire Service College (FSC), 1998). An investigation was launched by the Health and Safety Executive (HSE) into the standards of training that the fire fighters were given to cope with the 'standard' fire incident. They worked from the Health and Safety act (1974) that stated:

> "An employer, so far as is reasonably practicable, is required to provide such information, instruction, training and supervision as is necessary to enable the health and safety at work of employees" (section 2 (2) (c)).

In addition, fire fighters should be exposed to situations that give rise to fears they may potentially experience during a real fire. In turn, fire fighters should be trained to be able to recognise, cope and deal with these fears (HSE, 29th July, 1996).

The HSE document (1996) advocated that the Fire Service should provide sufficient training in ensuring fire fighters are prepared for operational situations that they are likely to encompass in active duty (as far as practically possible). The HSE document (1996) also stated that the Fire Service should not only continue to provide the basic operational training, but should also include training which provides realistic conditions that fire fighters are likely to encounter.

The HSE (1996) concluded that the Fire Service training did not adequately prepare fire fighters to recognise or deal with the incident at Blaina. They issued an improvement notice demanding that Fire Services investigate what future developments were required to establish a coherent program of training. This would ensure fire fighters were adequately equipped to deal with situations during fires. The result was the development of LFTEs, where instructors were required, using live fire, to replicate fire scenarios and teach fire fighters the most up to date and advanced fire fighting techniques. This had to be undertaken within a metal cargo container box known as a Fire Flashover Unit (FFU) or 'hot box'.

1.3 Fire Flashover Unit (FFU).

LFTEs were originally developed by the Swedish Fire Research Department (Fire Service College (FSC), 1998). Typical compartment specifications are defined in Figure 1.1. Typical temperature in the anti-chamber at mid level (1.2 metres from the FFU floor) was 48.3 (± 4.0) ^{0}C and at ceiling level was 54.5 (± 4.7) ^{0}C. In the fire chamber at mid level typical temperature was 107 (± 32.9) ^{0}C and at ceiling level 114 (± 20.4) ^{0}C.

Three instructors are required to ensure the safety of students during training exercises. The BAI in the control room (Figure 1.3) is protected from the heat whilst observing the other instructor and the students carrying out the LFTEs. In conjunction with the instructor within the FFU, it is the role of the BAI within the control room to ignite and extinguish the fire. Figure 1.4 shows the chair which is ignited during LFTEs to demonstrate fire behaviour and Figure 1.5 shows the anti-chamber of the FFU.

The second BAI situated next to the fire appliance (fire engine) during the LFTEs ensures that there is a consistent water supply. The third BAI is located in the FFU with the fire fighter recruits (students). The LFTEs are divided into fire exercises and debriefs. The fire exercises are based on real life situations that are re-created within the FFU, often using critical incidences whereby fire fighters have died. Figure 1.6 shows the BAI standing to the rear of the student whilst the student fights the fire.

Once the fire exercise is completed, the fire is extinguished (controlled by the BAI in the control room or sometimes by the BAI within the FFU who has a remote control in the anti-chamber). This is immediately followed by a debrief session within the anti-chamber compartment of the FFU. This debrief session provides BAIs with the opportunity to feed back to students concerning how their techniques could be improved. Figure 1.7 shows the BAI giving feedback to the students concerning their progress during the previous fire exercise. During the debriefs, the fire is off and the door connecting the fire chamber and the anti-chamber is closed.

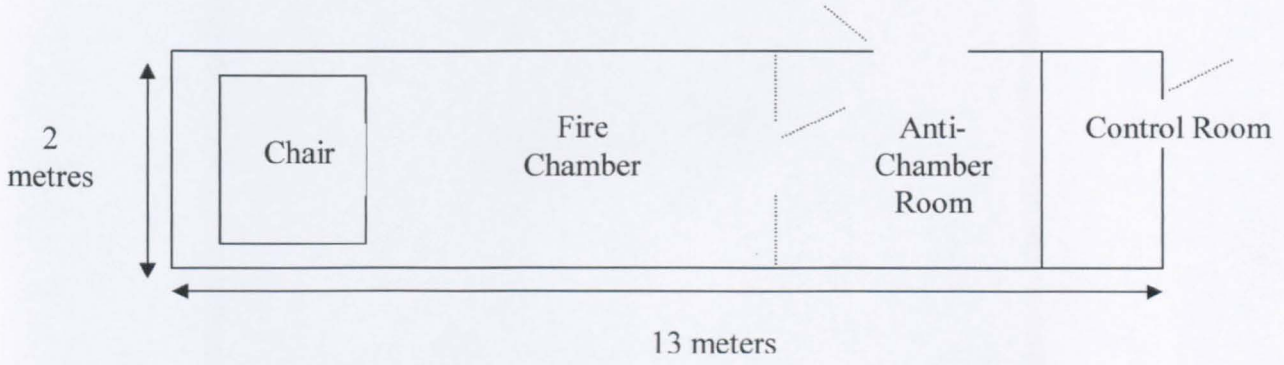


Figure 1.1 – Dimensions of a typical liquid petroleum gas (LPG) FFU.

Figure 1.2 – FFU at Greater Manchester Fire Training Headquarters.

Figure 1.3 – Control room of the FFU.

Figure 1.4 – Chair inside the FFU used to demonstrate typical fire responses if a chair was ignited.

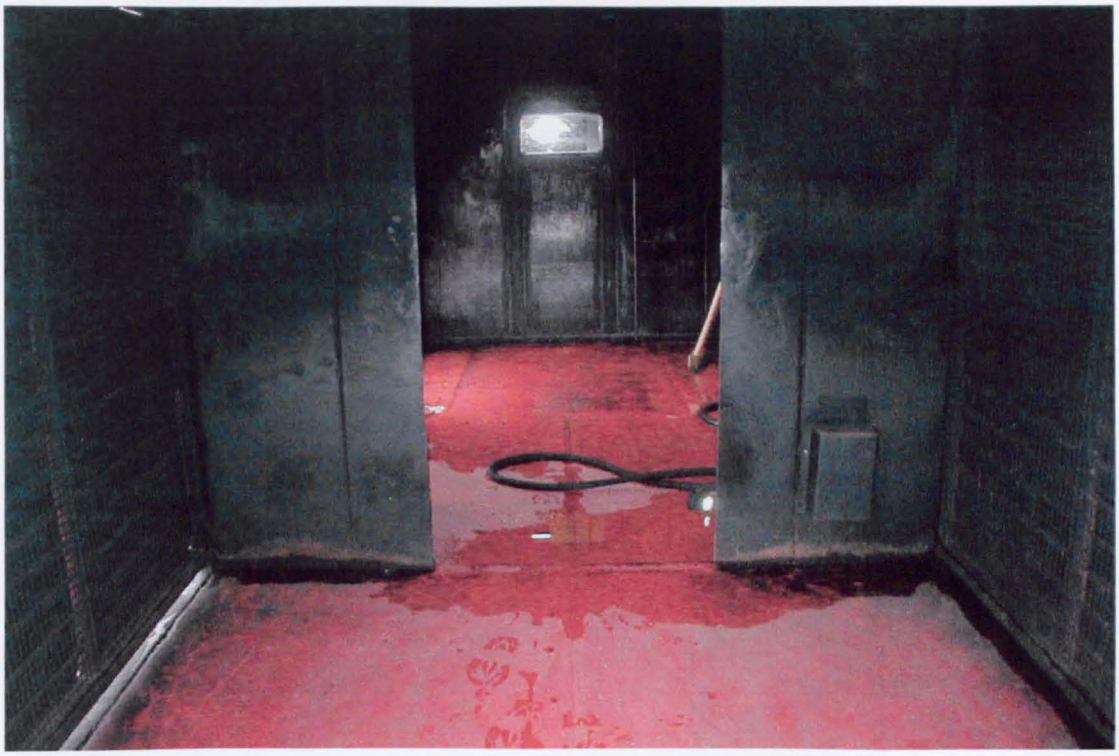


Figure 1.5 – looking towards the control room with the fire chair behind the photographer inside the FFU.

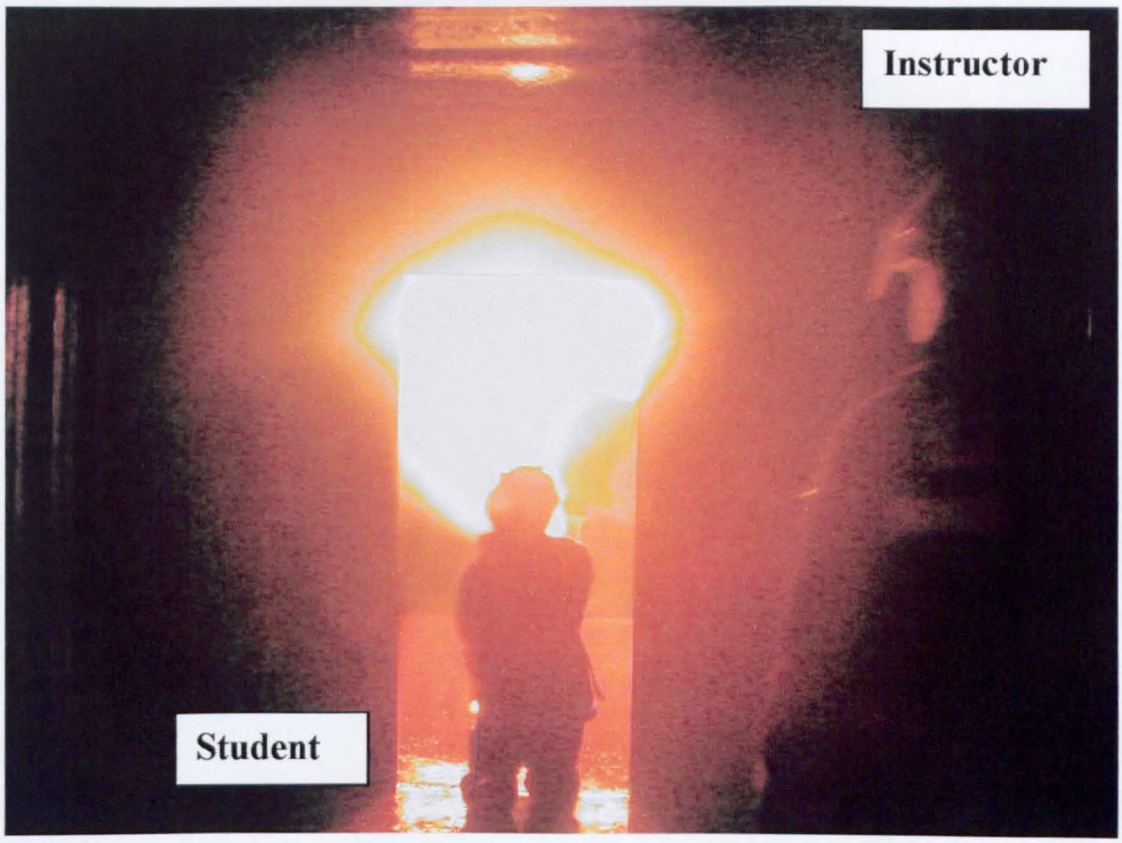


Figure 1.6 – Student fighting a fire known as a flashover.

Figure 1.7 – Instructor providing feed back to the students during a debrief.

The different types of fire that the BAIs introduce the students to within the FFU are outlined below.

1.4 Fire Theory: Early development of a fire.

According to the Fire Service College (FSC) (1998), in a room or compartment where there is a fire, all combustible materials (anything that will contribute to the fire growing) should be considered a source of flammable gas. These gases are produced when the fuel is heated. For example, when a sheet of chipboard is heated there is gas produced from the resin on the surface of the sheet. The pyrolysis, or decomposition of the hydrocarbons in the resin exudes the gas (FSC, 1998). These gases rise in the fire plume (smoke from the fire) and mix with the air. If the air and gas mixture is within its ideal mixture (IM), any ignition source e.g., embers from partially burnt substances, will trigger the combustion process. The IM refers to the gas content of the room that has been burning. Gases have a flammable range which consists of an upper and lower explosive limit, beyond and below which the gas will not ignite. The IM is often in the middle of the flammable range and is the point at which the percent of the flammable gas compared to the air of the room is most likely to ignite with an ignition source. This process becomes self-sustaining as the combustible gases fuel the ignition source. Flames and radiated heat are the products of the combustion process. The plume that is formed through this process can be seen as having an upper and lower section. In the upper part of the plume there is no flame. This is because the flammable gases in this section are at too low a concentration to ignite. However, the plume is filled with unburned gases, soot, entrained air and non-flammable products of combustion. The gases in the lower part of the plume have a rich concentration of combustible gases and therefore are surrounded by flames (FSC, 1998). During the LFTEs this fire is simulated through the chair (Figure 1.4) being set alight and is referred to as a 'chair fire' or 'Number one fire' (Figure 1.8).

The progression of the fire through a compartment is caused by radiated heat from the flume. Objects (for example tables and chairs) that surround the plume are heated to such an extent that they too begin to produce flammable gases. There is an increase in the amount of heat generated from the fire plume. The heat intensity

increases causing an increase in the temperature of the surrounding walls and ceiling. This will result in the heat beginning to radiate back into the room (FSC, 1998). This leads to more objects in the room beginning to produce combustible gases from not only the fire plume, also the surrounding compartment. This process will continue as long as there is a continued supply of oxygen (FSC, 1998).

Gradually, the amount of combustible gases produced, becomes greater than the amount that the fire can consume. Such gases have been warmed by the amount of radiated heat from the plume and surrounding structures. They are therefore at a higher temperature than the air in the lower part of the room (region of lower pressure) and thus, rise to the ceiling (region of higher pressure).

There is an area in the compartment called the neutral plane (NP) which refers to the area where the higher and lower pressure regions meet (Figure 1.9). It can be clearly seen as the bottom of the combustible gas layer. As the fire develops the amount of combustible gases produced will also increase. This will collect in the high-pressure region. The more gas that is produced will increase the size of the high-pressure region and alter the position of the NP (FSC, 1998). Therefore, the students are instructed to observe the position of the NP as an indicator of the stage of a fires' development (FSC, 1998). More importantly, the position of the NP can also indicate the potential the fire has to cause what is known as a Flashover or Backdraught.

Figure 1.8 – The chair seen in Figure 1.4 set alight during a 'Number one' fire.

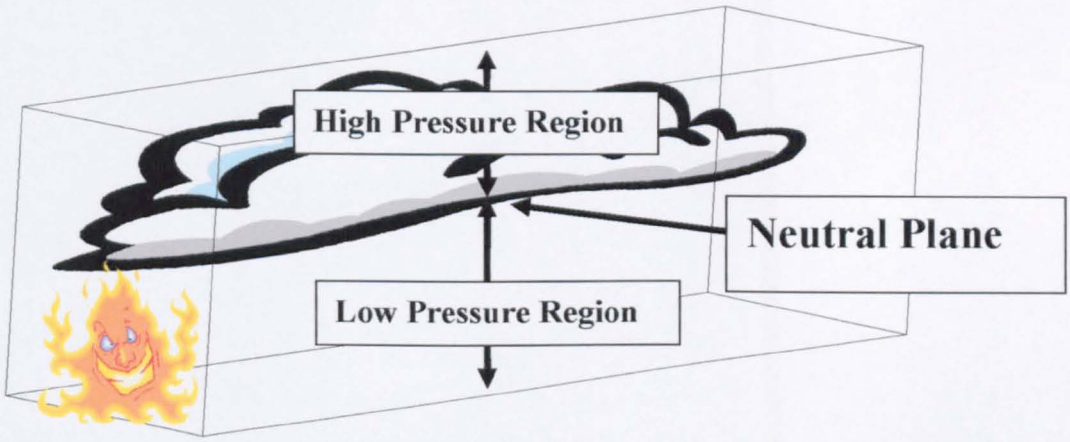


Figure 1.9 – Compartment with fire plume and clear low and high pressure regions. The neutral plane (NP) is where the low and high regions meet.

1.4.1 Flashover.

A compartment fire can progress to the point where there is a high volume of combustible gases which have collected in the high pressure region. This is often in conjunction with a large amount of heat being radiated from not only the fire plume and the high pressure region, but also the walls and ceiling of the room (FSC, 1998). The increase in radiated heat from these areas results in the generation of flammable gases from any exposed flammable materials. If a further source of ignition presents itself at this time, there will be a sudden transition from the situation of a growing fire to a state of fully developed fire (FSC, 1998) where the compartment, will in essence, become engulfed in flames. This is known as a flashover.

During a LFTE a simulated flashover is known as a 'Number two' fire. Students during a LFTE will be aware of this situation through a vast increase in temperature and also by the large flame that appears across the ceiling (see Figure 1.6). The students are trained to observe this visual change which consists of tongues of flames that are visible in the high pressure area. Furthermore, the increase in downward radiated heat, results in combustible materials in the room producing flammable gases and smoke.

It is the duty of the BAI's to highlight the symptoms of the flashover and educate the students with the most effective techniques to safeguard themselves, members of their team, and potentially control any further spread of the fire throughout the compartment or building.

1.4.2 Student responses during training.

Cooling the high pressure region of the compartment is the most efficient way of preventing the occurrence of a flashover (FSC, 1998). Through cooling, the students reduce the intensity of radiated heat and size of the flames. The NP will also lift causing the smoke layer to rise.

The BAIs also educate the students to cool the high pressure region in the most effective way. Applying a spray/fog stream into the high pressure region at ceiling level in short duration pulses, prevents large amounts of water generating an

extensive volume of steam. This would bring the smoke layer down from ceiling level obscuring visibility (FSC, 1998). Furthermore, the students' fire fighter uniform protects them from the radiated heat, but not from the steam produced when excess volumes of water are placed on the fire. Therefore, creating steam would result in an increase in the stress placed on the student and BAIs during LFTEs.

1.4.3 Backdraught.

A backdraught is the product of a sequence of events that culminates in an explosion due to limited ventilation in a fire compartment. Over time gases (comprised of unburnt pyrolysis and partially combusted gases) accumulate within the compartment. On introduction to an air supply, an explosion may result that works its way through the compartment and out of the opening (FSC, 1998).

It is the role of the BAIs' to present to students the theory and practical explanations of backdraught without one actually occurring. The products exuded by a fire form a gas layer (region of high pressure). This gas layer has a flammable range (FSC, 1998). This flammable range will vary depending on the contents of the room that have been burning. An example would be the production of carbon monoxide. Figure 1.10 shows that when the atmosphere consists of carbon monoxide between 12 % (known as the lower explosive limit (LEL)) and 74 % (known as the upper explosive limit (UEL)) this determines the explosive force of these gases if an ignition source is introduced.

A backdraught occurs when the gases reach the IM, which can occur whilst the fire fighters are in the middle of the compartment. During a backdraught fire fighters would be engulfed in flames and the result would probably be fatalities.

Therefore, BAIs ensure that students are educated in not only the most effective methods of fire fighting to combat the different fires they may encounter, but also the signs and symptoms. In addition, the BAIs ensure the students are aware of the heat stress symptoms that occur during fire fighting ensuring they have adequate breaks and are hydrated adequately. However, as previously stated there is no system in place to monitor the BAIs during LFTEs.

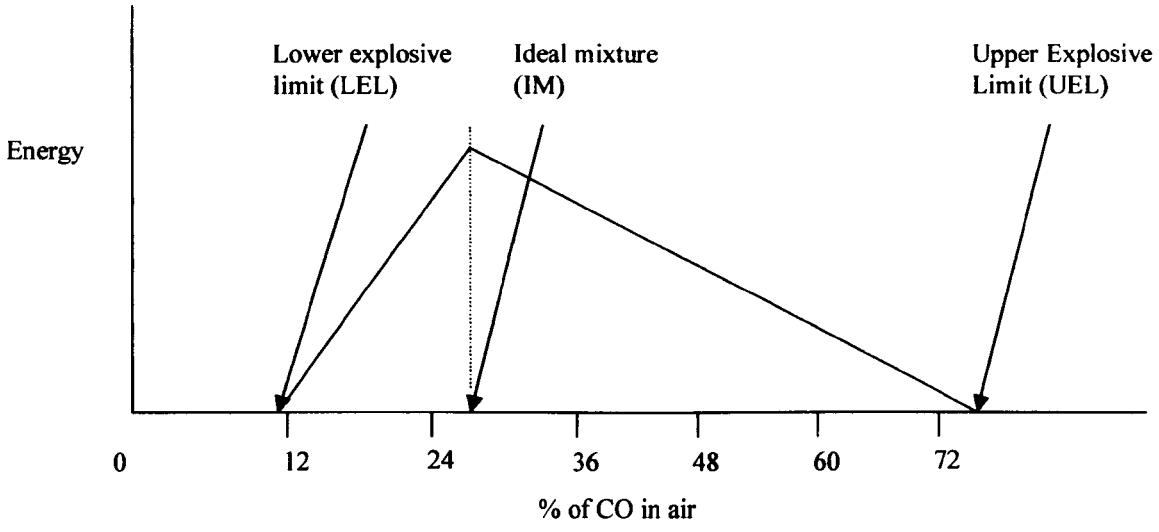


Figure 1.10 – The flammable range of CO.

1.5 Study constraints.

This study has had the exclusive opportunity to have access to the live fire training facilities and the BAIs of the Greater Manchester Fire Service. There is a shortage of fire fighters on active duty, both within the Fire Service nationally, and in particular, the Greater Manchester Fire Service, placing heavy work demands on the BAIs. As such, testing periods for the present study could only be conducted at certain times of the year when the workload allowed. The nature of the BAI employment has necessarily limited some aspects of the study i.e., monitoring was only possible pre, during and post exposure to the LFTEs. All BAIs from Greater Manchester Fire Service were used as subjects (n = 6) within the study. Due to the department being under staffed it was not possible to increase the subject number further.

The constraints placed on the study from the subjects and the nature of the LFTEs high ambient temperatures, negated the possibilities of laboratory-based testing, and as such the study focuses heavily on field-based research. The inventory of tests have been designed and selected with this in mind.

Chapter 2

Review Of Literature.

2.1 Overview.

Fire fighting has long been recognised as a profession that places a high physical demand on the body, although there appears to be no information about the level of work the BAIs carry out during LFTEs. It is important to differentiate between the types of work BAIs conduct and the types of work the recruits/operational fire fighters (students) undertake during LFTEs. Students have one exposure to the LFTE during which they perform a greater amount of physical work than BAIs. However, BAIs have one exposure to the LFTEs every other day over a period of a week. There is a plethora of research into the physiological responses of fire fighters to fire exercises, but there is a significant lack of research into the physiological responses of BAIs to LFTEs. Therefore throughout the thesis, examples used inevitably come from fire fighter research as opposed to research specific to BAIs.

Both BAIs and students have to deal with the energy cost of working in a very hot environment with the same heavy and impermeable protective clothing (PC). The PC places a great demand on their physiological responses (Williams, Petersen and Douglas, 1996). In particular, the impermeable nature of the fire fighting PC inhibits the ability to successfully evaporate sweat and thus cool the body. The failure of the evaporative process results in an increased oxygen consumption, heart rate and core temperature coupled with an increase in thermal stress (Duncan, Gardner, Barnard, 1979; Faff and Tutak, 1986). The amount of physical stress is further compounded by the wearing of heavy SCBA (self-contained breathing apparatus and cylinder) (refer to Figure 2.1).

Figure 2.1 – BAI in Protective Clothing and Self Contained Breathing Apparatus (PC+SCBA) prior to a LFTE.

The methods by which student fire fighters are trained necessitate a realistic as possible environment, but are constrained by the requirements of health and safety. However, even in an experimentally contained environment, realism can only be achieved by exposing the BAIs and students to considerable heat stresses. Fatalities have occurred amongst BAIs through continuous exposure to LFTEs. In an attempt to monitor the potentially hazardous situation, students are continuously observed by the BAIs during the test procedures. However, BAIs must rely solely on their own experience of knowing when the level of heat stress to which they are subjected, is approaching a level which would be deemed as hazardous. At this stage, the normal course of action would be to terminate the LFTE.

2.2 Stress and the fire fighting occupation.

The present study defines stress based on the general adaptation syndrome (Selye, 1976). As the body fails to meet the demands placed on it in response to a stressor, it does not maintain homeostasis or steady state (Plowman and Smith, 2001).

Fodor *et al* (1998) conducted a study to ascertain the prominent causes of stress within the daily lives of Canadian fire fighters. It is evident from Figure 2.2 that 35% of fire fighters perceived their job to be undoubtedly the most stressful part of their life. In comparison, the Health and Safety Executive (2000) reported 20% of office workers in the UK reported either high or extremely high levels of stress at work.

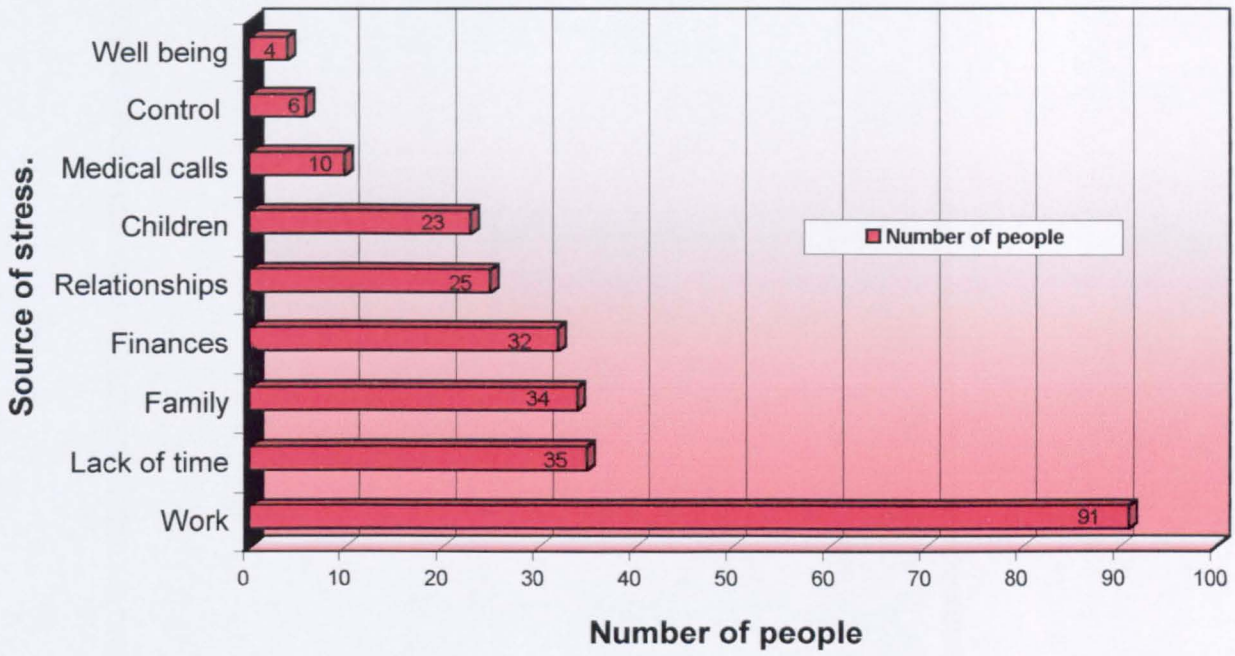


Figure 2.2 – sources of stress in a group of Canadian fire fighters (Taken from Fodor et al, 1998).

Fodor *et al* (1998) did not differentiate between psychological and physiological stress. However, a previous study carried out by Lemon and Hermiston (1977) identified the metabolic costs of fire fighting, in terms of specific tasks, produced a high percentage of their subjects' aerobic capacity (between 60-80% of $VO_{2\ max}$). However, factors affecting the physiological responses whilst carrying out fire fighting duties were omitted from the studies. For example increased CO_2 production, emotional stress and heat and humidity (Barnard and Duncan, 1975) were not accounted for.

Romet and Frim (1987) investigated the physically demanding tasks (consistent with Barnard and Duncan (1975)), taking into account the less demanding tasks. Such tasks are undertaken by fire fighters who operate the water hoses outside the building and drive the appliance (fire engine). They concluded that stress on fire fighters can be partially relieved by frequent rotation between the heavier and lighter tasks during an emergency.

Research carried out by Washburn and Harlow (1982) investigated the causes of premature death in fire fighters from the U.S.A (Figure 2.3). This shows that fire fighter deaths occurred most frequently during fire fighting incidents (63%), with car accidents accounting for 7% and water rescues 16%. Working on maintenance of equipment and working at the fire station accounted for 10% of deaths, whilst 4% of deaths occurred during training (all of which were heart attacks). Although Washburn and Harlow (1982) state that 123 fire fighters died as a result of fire service duties in 1981, it was unclear as to the percentage of the U.S.A Fire Service this figure represented. Also, figure 2.3 does not include the number of fire fighter fatalities that occurred whilst not on duty, therefore under estimating the total number of deaths.

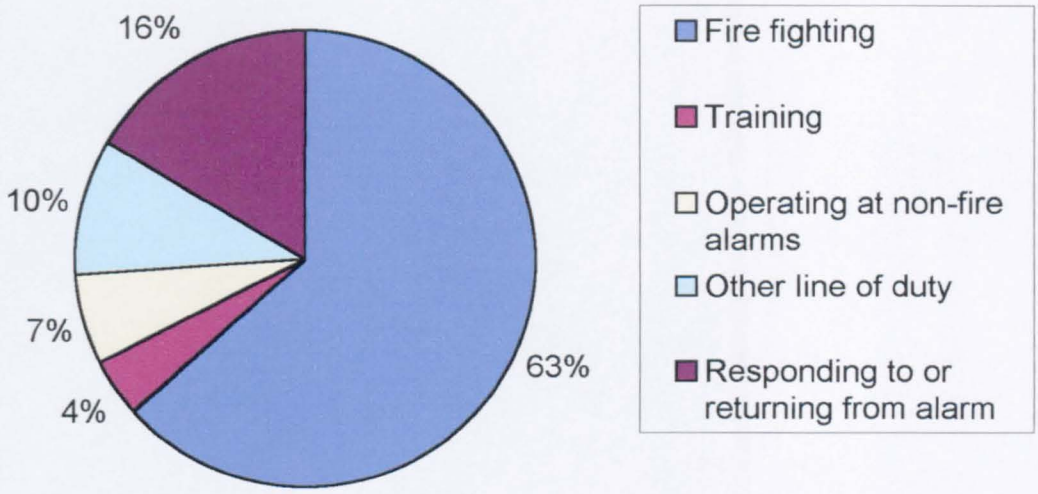


Figure 2.3 – Fire fighter deaths by type of duty; Fire fighting (fire fighter deaths occurred most frequently during fire fighting incidents); Training (deaths occurring during training (all of which were heart attacks, although the circumstances surrounding the deaths were not stipulated); Operating at non-fire alarms (deaths occurring through car accidents and water rescues); Other lines of duty (deaths occurring whilst fire fighters worked on maintaining equipment and working at the fire station); Responding to or returning from alarm (deaths occurring through responding to or returning from fire incidences). Taken from Washburn and Harlow (1982).

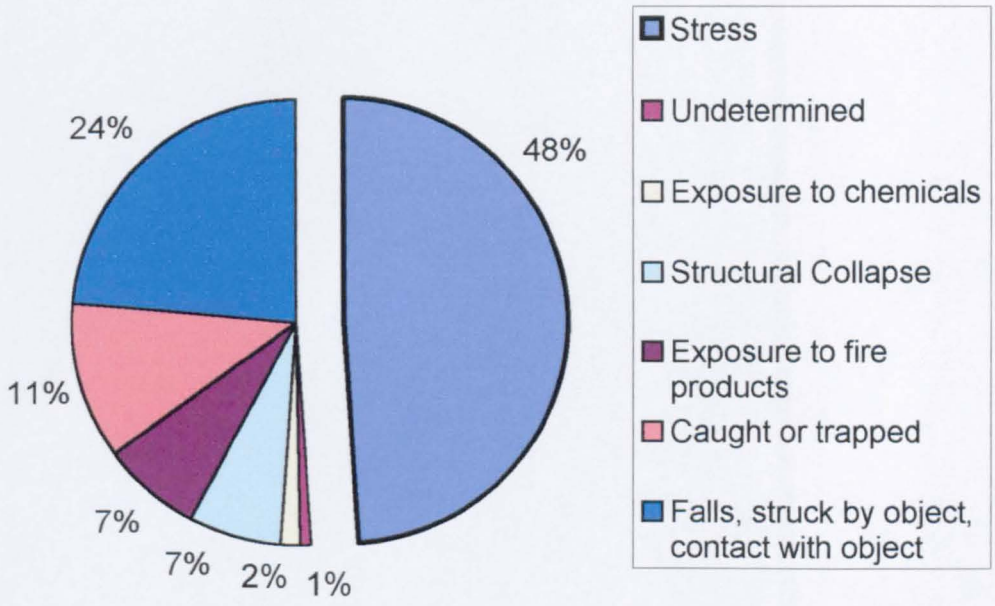


Figure 2.4 – Causes of fatal injuries to fire fighters. Fatal injuries were defined as Stress (Washburn and Harlow (1982) fail to describe this any further); Undetermined (One fire fighter died from internal bleeding of a medically undetermined cause); Exposure to chemicals (death by exposure to poisonous chemicals); Structural collapse (death caused by collapse of building whilst fire fighters are in, next to or on top of the building); Exposure to fire products (fire fighters burning to death or smoke inhalation); Caught or trapped (included fire fighters drowning); Falls, struck by object, contact with object (these included motor vehicle accidents, aircraft crashes, crossing roads, electrocution, falling from machine/apparatus and murders). Taken from Washburn and Harlow (1982).

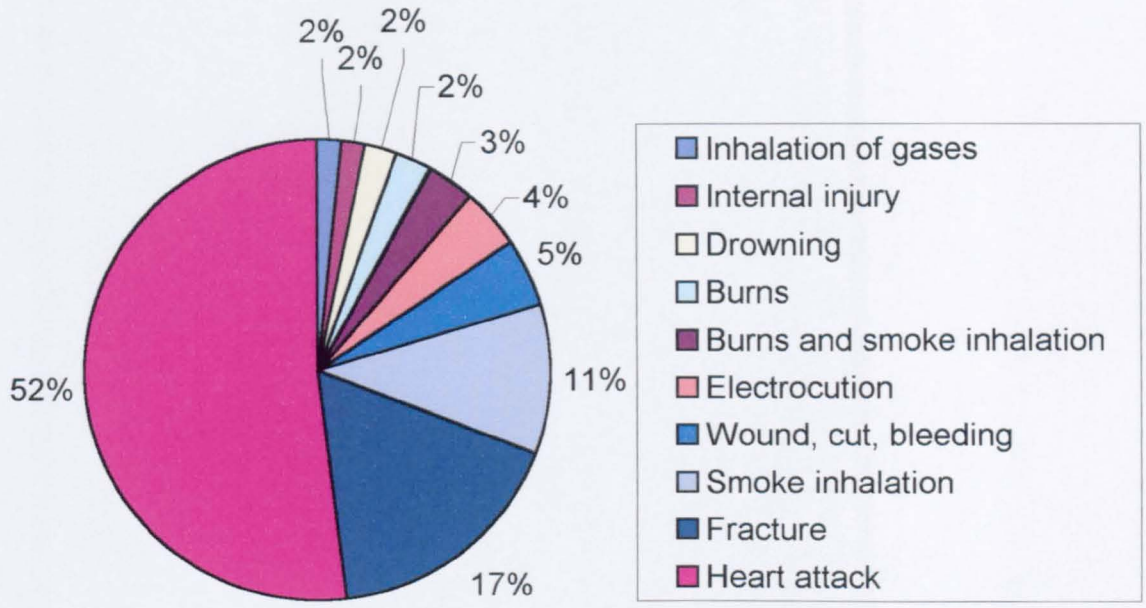


Figure 2.5 – The nature of fatal injuries to fire fighters. Taken from Washburn and Harlow (1982).

In accordance with Fodor *et* al, (1998), Washburn and Harlow (1982), also identified stress as a major factor in fire fighters lives. In support of the findings from Fodor *et al* (1998), stress was found to be the major cause of premature death in fire fighters (Figure 2.4). However, further clarification of 'stress' was not presented, although Washburn and Harlow (1982) were able to identify heart attack as the major cause of death, which accounted for 52% of the fatalities (Figure 2.5).

2.3 Fire fighting and fitness.

It is widely recognised that fire fighters should possess physiological characteristics that allow them to withstand the high levels of physical and heat stress (Davis and Dotson, 1986, Peate *et al*, 2002) caused by working with fire (typical ceiling temperatures in the fire chamber ranged from 100-280^{0}C). This will reduce the increased risk of injury whilst on duty, decrements in chronic health (Davis and Dotson, 1986) and improve a worker's ability to perform (Peate *et al*, 2002). There are additional stresses from the weight of the protective clothing (PC) and self contained breathing apparatus (SCBA) (both together weighing some 20.4 (± 1.5) kg), and the non-permeable properties of the PC.

This suggests that there is an evident need for a basic level of aerobic fitness to withstand the stresses placed on the body. This is supported by the findings of Gledhill and Jamink (1992). They observed that fire fighters having $VO_{2\ max}$ values less than 35 ml $O_2 kg^{-1} min^{-1}$ failed specific fire fighting tasks, and those with a greater $VO_{2\ max}$ than this value, passed the tests.

Lemon and Hermiston (1977) investigated the physiological profile (aerobic capacity, anaerobic capacity, muscular strength and body composition) of 45 professional fire fighters aged between 23 and 49 years. Despite the findings of Davis and Dotson (1986) the results obtained for aerobic capacity, anaerobic capacity, muscular strength and body composition were not significantly different when comparing fire fighters to inactive individuals. This was consistent with the findings of Clark *et al* (2002). They observed 80.7% of the fire fighters measured (n = 49) were categorised as being overweight, obese or morbidly obese.

The findings of Lemon and Hermiston (1977) stated that the intensity of fire fighting duties is undertaken between 60-80 % $VO_{2\,max}$. Therefore, fire fighters should ideally have a superior level of general fitness when compared to a sedentary population. This suggests a plausible reason as to why fire fighters perceive their job as 'stressful'. It was suggested that, fire fighters may benefit from a physical training program specifically designed to maintain a higher level of personal fitness (Lemon and Hermiston, 1977). This is supported by evidence provided by Cheung, McLellan, and Tenaglia (2000) who state that long term (rather than short term) physical fitness programs will provide some protection against the intense physical demands of the profession. This will also provide protection when exposed to extreme heat, hence lowering the physical demands of fire fighting.

2.4 Simulated fire fighting tasks and heat stress.

Lusa *et al* (1993) observed that peak heart rates elicited during a cycle ergometer test in thermoneutral conditions, were exceeded during fire fighter specific drills known as 'smoke diving'. Smoke diving is carried out in a smoke filled building with a fire lit on the ground floor, whereby fire fighters search and rescue dummies from within the building. Lusa *et al* (1993) believe this was due to the heat augmenting the stress on the cardiovascular system resulting in a decrease in stroke volume, through an increase in subcutaneous blood flow to allow for heat dissipation. This decrease in stroke volume, in turn, caused cardiac output to decrease. As such, the body responded to the decrease in stroke volume by increasing heart rate. These findings are consistent with those of Pirnay, Deroanne and Petit (1970), who also observed increases in heart rates in hot conditions during maximal exercise tests. The study of Pirnay *et al* (1970) highlighted decreases approximating to 5 beats·minute^{-1} when exercising on a cycle ergometer compared to smoke diving. Thus, wearing PC+SCBA combined with heat exuded from fire, appear to create a greater stress on the cardiovascular system, and as such, increase HR further than a cycle exercise test undertaken in thermoneutral conditions (Louhevaara *et al*, 1984, Louhevaara, 1984, Lusa *et al*, 1993, Serra *et al*, 1998, Louhevaara *et al*, 1995).

The results from Lusa *et al* (1993) and Pirnay *et al* (1970) may be explained by cycle ergometry recruiting a smaller muscle mass. This, in turn, creates a lower maximal

heart rate when compared to full body exercise during smoke diving (Lusa *et al*, 1993). In addition, Lusa *et al* (1993) defined the cessation criteria for the $VO_{2\ max}$ test as attainment of predicted maximal heart rate. This suggests that the subjects used within this study may not have been working maximally. This may explain why the HR increased further during 'smoke diving'.

Research carried out by Davis *et al* (1982) investigated the relationship between simulated fire fighting tasks and physical performance measures. They observed that there was a significant ($p<0.05$) relationship between fitness and effectiveness on fire fighting jobs. However, their studies do not allow for the additional stress that heat would place on the fire fighters. This was due to all exercises being undertaken in thermoneutral conditions, and were therefore not a true reflection of the responses the fire fighters would produce under hot conditions.

Desiree *et al* (1989) observed the potential effects of acclimation between volunteer fire fighters and professional fire fighters when they exercised on a cycle ergometer in a temperate environment (36^0-38^0C). There were no significant physiological differences between the volunteer fire fighters and professional fire fighters. However, Desiree *et al* (1989) observed an increase in the rectal and skin temperatures in the volunteer fire fighters when compared to the professional fire fighters, which became more accentuated as the experiment proceeded. Desiree *et al* (1989) observed significant ($p<0.05$) differences in rectal and skin temperatures between the two groups at a power output of 100 watts. Although the heart rates were higher in the volunteer group, they were found not to be significant. In addition, sweating rates were also higher in the volunteers when compared to the professional fire fighters, but again not significantly. Also, there were no obvious differences observed for total work time or blood lactate concentration post exercise. However, perceived exertion was significantly higher in the volunteer group. Desiree *et al* (1989) state that in conjunction with the study of Shvartz *et al* (1977), the disparity between the volunteer and professional fire fighters can only be explained by differences in acclimation to heat exposure. This was concluded from the professional fire fighters reporting a greater use of sauna facilities in comparison to the volunteer fire fighters.

Acclimation to heat increases the plasma volume and also sweating rate (Nielsen, 1998, Sawka and Montain, 2000). However it seems unlikely that professional fire fighters will demonstrate any physiological adaptations to heat acclimation as the exposure times to temperature excess is limited. According to Terrados and Maughan (1995), acclimation occurs after 10 to 14 consecutive days of heat exposure. It is unlikely that the professional fire fighters have this amount of exposure as they spend a very small amount of time fighting fires (Louhevaara and Kinnunen, 1994). There has been no research undertaken to investigate the effects of acclimation on BAIs.

2.5 Stress and PC+SCBA.

Undertaking active work in a hot environment is, in general, a more stressful challenge to the maintenance of normal body temperature than performing similar work in a thermoneutral environment (Havenith, 1999). The cardiovascular responses that often accompany exposure to the heat may compromise workers health and safety. In addition, if there is the necessity to wear protective clothing (PC), as in the case of fire fighters and BAIs, this may increase the likelihood of heat strain. This is because the PC causes a reduction in the environmental temperature at which the heat stress occurs (Barnard (1999), Havenith (1999)).

Skoldstrom (1987) suggests that the stress placed on fire fighters is from a combination of both heavy workload and heat stress. The workload is generated from not only carrying the heavy equipment associated with fire fighting (water filled hoses, ladders etc.), but also from the weight from wearing the heavy protective clothing and self contained breathing apparatus and cylinder (PC+SCBA). Carter *et al* (1999) also reported increased core temperature, skin temperature and heart rate when wearing PC+SCBA.

Heat stress is generated primarily from the radiated heat from the fire, but there is also an element of metabolic heat storage from the PC+SCBA (Skoldstrom, 1987). Although Skoldstrom (1987) did not observe the physiological responses to wearing PC+SCBA in BAIs, it is possible to assume they will be affected in a similar fashion as the same uniform is used by both fire fighters and BAIs. The concept that wearing PC+SCBA generated an additional workload was supported by Bone *et al* (1994).

They observed significantly higher ($p<0.05$) skin temperatures (+ 1.5^0C) and rectal temperatures (+ 0.9^0C) when their subjects were dressed in PC+SCBA when compared to wearing light clothing (gym kit). Research carried out by Bishop *et al* (1994) support these findings by reporting an increase in heat storage when wearing PC. The increase in heat storage was observed through an increase in core and skin temperature.

Manning and Griggs (1983) investigated the effects of wearing different protective clothing (PC but no SCBA, PC and a light weight SCBA, or PC and a heavy SCBA) on the heart rate responses of fire fighters whilst carrying out a fire fighting drill. They observed that during the drills when wearing one of the three ensembles, heart rate increased rapidly to 70-80% of maximum within the first minute then plateaued at approximately the maximal HR (90-100 % of maximum HR) in each case. Manning and Griggs (1983) concluded that it is not an influencing factor whether the SCBA is worn or not, as the physical activity appears to elicit enough stress on its own to elicit maximal heart rates. This drill, although carried out at 36^0C, was not carried out under extreme temperatures.

Montain *et al*, (1994) investigated the effects of wearing protective clothing in heated environments. They observed the differences in physiological responses to heat whilst wearing protective clothing and partial protective clothing at 43^0C. They observed a significantly ($p<0.05$) higher core temperature (+ 1.6^0C) whilst wearing the fully protective clothing compared to wearing the partially protective clothing. They concluded that full protective clothing reduced physiological tolerance when exercising in heat due to an increase in core temperature. This was in agreement with other research that also observed increased heat strain whilst wearing protective clothing, as indicated by an increase in core temperature (Mihal, 1981, Payne *et al*, 1994 and Levine *et al*, 2001).

Havenith and Vrijkotte (1994) investigated the effect of wearing chemical protective equipment during moderate to hard exercise and hard exercise. They observed that in thermoneutral conditions, heat strain occurred below 20^0C. The observations are significant to the present study as the BAIs were exposed to temperatures within the fire flashover unit (FFU) in excess of 120^0C during LFTEs.

White, Vercruyssen and Hodous (1989) investigated the effects of wearing different types of protective ensembles. The trials utilised gym kit (wearing t-shirts and shorts) as the control, whilst the other ensembles included wearing gym kit and SCBA (including the cylinder), PC+SCBA (which included the cylinder), and also the chemical protective clothing (which included the SCBA and cylinder). The trials carried out low (30% $VO_{2\ max}$) and high (60% $VO_{2\ max}$) intensity work. They observed that PC and SCBA together elicited the greatest stress on their subjects through volitional fatigue occurring at 25.0 (± 9.0) minutes during the low intensity exercise and at 4.0 (± 1.0) minutes at high intensity exercise. This trial was the quickest to reach volitional fatigue for both the low and high intensity trials (refer to Table 4.1). This suggests that the BAIs are placed under substantial stress from wearing PC+SCBA even at low intensities of exercise over that of other forms of protective clothing such as chemical protective clothing (Chemklos II coveralls and SCBA). When chemical protective clothing was worn, volitional fatigue occurred at 72.0 (± 18.0) and 13.0 (± 13.0) minutes during the low and high intensity exercise trials respectively (White and Hodous, 1987, White *et al*, 1989).

Table 2.1 – The mean time to reach fatigue wearing different protective clothing ensembles (taken from White *et al* (1989)). Data is presented as the mean and ± SD (n = 9).

	Time to reach volitional fatigue (minutes)	
Clothing Trial	**Low Intensity**	**High Intensity**
Control (Gym Kit)	164.0 (± 17.0)	91.0 (± 45.0)
Gym Kit and SCBA	129.0 (± 33.0)	23.0 (± 24.0)
PC+SCBA	25.0 (± 9.0)	4.0 (± 1.0)
Chemical protective clothing + SCBA	72.0 (± 18.0)	13.0 (± 13.0)

Advances in protective clothing technology have meant that fire fighters and BAIs can now be exposed to higher temperatures for longer periods of time. Research carried out by Foster and Roberts (1994), suggest that wearing modern protective equipment, may safeguard fire fighters (and BAIs) from the external temperatures in

excess of 200°C for short periods of time. However, the encapsulating nature of the PC impedes the body's natural cooling mechanisms, evaporation (White and Hodous, 1988) and radiation. Hence, the PC is effective at halting the fire fighters (and BAIs) internally generated heat from escaping, thus creating a secondary heat environment (Duncan, Gardner and Barnard, 1979). Therefore, the heat from the fire combined with the heat stored from wearing the PC+SCBA, results in an increase in the amount of heat stress placed on fire fighters (White and Hodous, 1988) and one can presume, BAIs.

Wearing PC+SCBA negates an individuals' capacity to lose heat through evaporation. This suggests increased sweating through acclimation will not decrease the level of heat strain during exposure to hot environments (Sawka and Montain, 2000). Therefore, increasing the sweating responses of BAIs through acclimation would not be effective in reducing heat stress within a working environment. There appears to be no previous research observing the effects of wearing PC+SCBA at low intensities of exercise (< 20 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) on BAIs. However, it may be postulated that during LFTEs the PC, SCBA and heat from the fire, together constitute significant stress on the individual BAI. This may result in the BAI suffering from performance decrement, heat illness or general discomfort (Duncan *et al* (1979), Smolander *et al* (1985), Ilmarinen and Makinen (1992)).

The research suggests that fire fighters are placed under considerable stress from heat from the fire, weight of the PC+SCBA, psychological stress, the physical activity and the heat storage from wearing the PC. Therefore, when these are combined fire fighters and BAIs would be placed under great amounts of stress. Therefore, the study focuses on identifying the major contributing factor to the stress placed on BAIs when wearing PC+SCBA. From this, the study then attempts to lower this identified stressor.

2.6 Fire fighter stress responses to emergencies.

Research has investigated heart rate (HR) responses to actual emergencies, and fire fighting tasks whilst wearing PC+SCBA (Manning and Griggs, 1983 and Sothmann, *et al*, 1992). However, this research was not conducted in hot environments but in

thermoneutral conditions (15^0-27^0C). As such, it would be incorrect to assume the vast majority of the fire fighters occupation is conducted fighting fires (Lusa *et al*, 1994). In reality there is only a very small percentage of a fire fighters time actually spent fighting fires. Considerably more time is spent walking, running, lifting as well as being sedentary (Baker *et al*, 2000).

Barnard and Duncan (1975) studied fire fighter electrocardiograph (ECG) responses to fire incidences. They observed that performing sudden exercise, without the benefit of prior exercise, can produce an ischemic condition in the heart. This was observed from S-T depression greater than 1mm. The subjects used in the study were in good physical condition, and as such, the observed ischemic responses may be best explained by carrying out job related tasks. These findings may also suggest an explanation for the high incidence of heart attacks in professional fire fighters (Gardner *et al*, 1974 and Barnard and Duncan, 1975) as reported by Washburn and Harlow (1982).

2.7 Heart Rate Variability (HRV).

2.7.1 HRV and cardiac disease.

The office for national statistics (2001) identified heart attacks as the largest cause of death in males between the ages of 45 and 54 years of age. Heart attacks claimed 23.5 % of the total number of deaths reported in 2001. Washburn and Harlow (1982) also identified heart attack as the major cause of death in fire fighters from the USA. The findings reported by the office for national statistics (2001) and Washburn and Harlow (1982) suggest it was necessary to monitor HRV. HRV was used to indicate the balance between sympathetic and parasympathetic heart rate control, thus inferring the possible risks of sudden cardiac death (Van Ravenswaaij-Arts *et al*, 1993). Thus, the current study used HRV during controlled conditions as a precursor to using HRV under live fire training conditions.

HRV is a measure of fluctuations that the heart makes around the mean HR (Van Ravenswaaij-Arts *et al*, 1993). Monitoring HRV is a method that can be used to indicate the parasympathetic and sympathetic function of the autonomic nervous system. Thereby, HRV can give possible indications about the risks of sudden

cardiac death (VanRavenswaij-Arts *et al*, 1993). This is through an alteration in the balance between the sympathetic and parasympathetic influence of HR control (Van Ravenswaaij-Arts *et al*, 1993).

Beat-to-beat variations, when observing the cardiac cycle length, have been studied for many years. Early research was carried out by Hon and Lee (1965) on foetal distress, who observed that a distressing situation led to an alteration in the inter-beat interval. More recent research investigated the effect of training on sinus bradycardia, and observed what is thought to be a slowing of the heart rate at rest through an increase in vagal stimulation (Kotana *et al*, 1982). They observed that the reduction in resting HR was due to a reduction in intrinsic cardiac rate and not through an increase in parasympathetic tone.

Originally, pharmacological blockade was the criterion method used to study autonomic responses. This method uses a dual blockade system utilising propranolol and atropine in order to abolish β-sympathetic and parasympathetic cardiac control respectively. However, the dual blockade system utilised by Kotana *et al* (1982), only allows an assessment of either the parasympathetic activity or the sympathetic activity at one time. Therefore, the synergistic action of the parasympathetic and sympathetic control of heart rate can not be observed under a single set of conditions. In addition, Brenner *et al*, (1998) suggest the level of blockage that pharmacological blockade produces on the sympathetic and parasympathetic innervation has never been categorically shown. Therefore, the completeness of blockade, and in turn the use of a dual blockade system to monitor HRV, may be questionable.

In addition to the questionable completeness of blockage from pharmacological blockade, it is also a highly invasive method. Brenner *et al* (1998) believe this method can be surpassed using more modern and less invasive techniques. Less invasive techniques have been developed which incorporate recent advances in telemetry, electrocardiographic recording and their associated highly powerful computer-based analysis systems (Brenner *et* al, 1998). One such system is the Polar system (Polar Electro Oy, Kempele, Finland). The Polar system uses a telemetric system that analyses beat-to-beat variations in R-R intervals. It records the

sympathetic and parasympathetic indicators of heart rate control under a single set of conditions, where pharmacological blockade could not. The evidence that HRV is a valid method of obtaining the balance of obtaining the sympathetic and parasympathetic balance can be found in Table 2.2 and 2.3. Karvonen *et al*, (1984) and Delaney, Leong and Brodie (2001), observed high correlations between the Polar system and a standard three-lead electrocardiograph (ECG) (Biopac MP100, Biopac Systems Inc.) in all variables in the frequency domain. This suggests that the Polar performance software system is a valid and reliable method of monitoring HRV.

The Polar system is easy to use and is a non-invasive method of obtaining HRV data. It uses a chest strap that acts as the transmitter of the R-R interval information and a wrist watch that acts as a receiver. The R-R interval information is data logged onto the wrist watch and can be downloaded to the Polar Precision Performance Software package (Polar Electro Oy, Kempele, Finland) at a later stage. It was not possible to use laboratory based ECG measurements due to the field based nature of the study. It was for this reason that the non-invasive telemetric method was utilised.

2.7.2 Measurement of HRV.

The research from the present study analysed the R-R intervals utilising both the frequency and time domain responses. Time domain analysis, according to the Task Force (1996), is one of the simplest methods of analysing HRV. This method measures intervals between successive normal-to-normal (NN) intervals (intervals between adjacent QRS complexes). Thus, the time domain analysis observes the standard deviation and length of R-R intervals. Results are interpreted as predominance of vagal activity eliciting a long resting R-R interval in conjunction with a large variance of the R-R interval (Brenner *et al,* 1998). In addition, large variance of the mean NN interval and mean heart rate also indicate the predominance of vagal activity (Task Force, 1996). Heart rate and cycle length are how differences are invariably observed (Task Force, 1996). Table 2.2 summarises the time domain indices used within the present study.

Table 2.2 – Time Domain Analysis.

Variable	Units	Description
NN intervals	msec	The intervals between adjacent QRS complexes (also known as R-R intervals).
SD	msec	Standard deviation of all R-R intervals. The SD indicates overall heart rate variability.
SD as a % of Mean HR	%	The standard deviation of all R-R intervals expressed as a percent of the mean HR (msec). Used to observe intra-subject variability.
pNN50	%	The number of adjacent pairs of R-R intervals differing by more than 50 ms divided by the total number of R-R intervals. The pNN50 indicates the parasympathetic branch of cardiac control (Task Force, 1996). According to Eckberg (1997) the criterion method that indicates vagal nervous impulses to the heart is through the R-R interval shortening after atropine is given to β-adrenergically blocked subjects. Rimoldi *et al* (1990) showed, in conscious dogs, that the normalised HF component of R-R variability disappeared after atropine infusion (acts as a parasympathetic blocker). Therefore, HF can be considered a marker of vagal activity. In turn, the normalised HF correlates well to pNN50, suggesting pNN50 is a marker of parasympathetic control of HR (Delaney and Brodie, 2000).
RMSSD	msec	The square root of the mean of the sum of the squares of differences between adjacent R-R intervals The RMSSD indicates the parasympathetic branch of cardiac control. (Delaney and Brodie, 2000). According to the findings of Eckberg (1997) and Rimoldi *et al* (1990) showing the normalised HF correlating well to RMSSD, this suggests RMSSD is a marker of parasympathetic control of HR (Delaney and Brodie, 2000).

Frequency domain analysis is more complicated than the time domain analysis. There are two methods that are commonly utilised. They include fast fourier transformation and autoregression analysis (Brenner *et al*, 1998). The autoregressive method is the preferred method of analysis as it is still reliable using a small number of samples (the Task Force (1996) do not give evidence to support this). The autoregressive analysis also produces smoother spectral components with easy post-processing of the spectrum with automatic calculations of low and high frequencies (Task Force, 1996). The power or variance is subdivided into frequency ranges (Table 2.3). There are 3 major frequency peaks in healthy adults. They include high frequency (HF) (in the range of 0.15 Hz – 0.40 Hz (Delaney and Brodie, 2000) or 0.15 Hz – 0.50 Hz (Brenner *et al*, 1998), low frequency (LF) (in the range of 0.04 Hz – 0.15 Hz) and very low frequency (VLF) (0.00 – 0.04 Hz) (Brenner *et al*, 1998). However, the fast fourier transform (FFT) method was also used in order to obtain a

graphical representation of the power spectral density (refer to Figures 7.12 and 7.13). The FFT breaks down the R-R intervals into different sine waves, this in turn reflects the power spectral density and how power (variance) is distributed as a function of frequency of the changes in R-R intervals (Task Force, 1996).

Table 2.3 – Frequency Domain Analysis using autoregression analysis.

Variable	Units	Description	Frequency Range
Total Power	$Msecs^2$	Sum of all power frequencies	$\approx\leq 0.4$ Hz
VLF	$Msecs^2$	Power in the VLF range	≤ 0.04 Hz
LF	$Msecs^2$	Power in the LF range	0.04 - 0.15 Hz
LF Norm	Nu	LF power in normalised units (LF/(Total Power-VLF)x100. Indicates sympathetic control of HR. Evidenced from sympathetic excitation elicited by transient coronary occlusion, which resulted in significant increases in the normalised LF component of R-R variability (Rimoldi *et al*, 1990). In addition, Pomeranz *et al* (1985) reported that normalised LF was reduced when propranolol (sympathetic blocker) was infused to subjects, which indicates sympathetic influence on HR. This suggests that normalised LF indicates sympathetic control of HR (Malliani *et al*, 1991 and Pomeranz *et al*, 1985).	
HF	$Msecs^2$	Power in the HF range	0.15 - 0.4 Hz
HF Norm	Nu	HF power in normalised units (HF/(Total Power-VLF)x100. Indicates parasympathetic control of HR. Evidenced from research from Rimoldi *et al* (1990) who showed, in conscious dogs, the normalised HF component of R-R variability disappeared after atropine infusion, thus HF can be considered a marker of vagal activity (Rimoldi *et al*, 1990 and Pomeranz *et al*, 1985).	
LF:HF ratio		Ratio LF (ms^2) / HF (ms^2)	

The Task Force (1996) suggest that the optimum time for short-term R-R recordings should be obtained over a period of 5 minutes. Brenner *et al* (1998) state that ideally the measurement should be taken when the HR has reached a near as possible steady state. However, if this is not possible, the measurements should be taken during restricted 'windows'. Therefore, in conjunction with the advice from both the Task Force (1996) and Brenner *et al* (1998), the present study monitored HRV during time 'windows' that equated to periods of 5 minutes. Movement artifact is a major limitation to measurement of HRV (Brenner *et al*, 1998) and should be avoided where possible. Therefore, subjects remained innate throughout all HRV testing periods.

2.7.3 Controlled and spontaneous breathing on HRV.

A further source of contention when measuring HRV is whether to control breathing frequency, or maintain spontaneous breathing frequencies. Stark *et al*, (2000) report breathing frequency (BF) should be controlled when interpreting HRV data due to increases in BF. This, in turn, results in a decrease in the HF component. Failure to control breathing will cause interpretations of results between treatment conditions to be difficult. This is because it will prove difficult to distinguish between treatment effect and the result of a change in BF (Stark *et al*, 2000). Other researchers have also controlled BF, therefore, purporting that any changes in HRV are from the treatment and not BF changes (Hirsch and Bishop, 1989).

Continuous change in the sympathetic and parasympathetic balance induces the sinus rhythm to fluctuate around the mean HR. The HR shows fluctuations that are equal in frequency to the respiratory rate because of inspiratory inhibition of the vagal tone (VanRavenswaij-Arts *et al*, 1993). This inhibition of the vagal tone is evoked largely by impulses sent from the medulla respiratory centre to the cardiac centre. The HF component of HRV has the frequency range that corresponds to respiration rate and thus reflects respiratory sinus arrhythmia (RSA). The parasympathetic branch of the autonomic nervous system is ultimately responsible for transmitting the RSA (Akselrod *et* al, 1981, Task force, 1996, Brenner *et al*, 1998, Delaney and Brodie, 2000 and Stark *et al*, 2000).

Porges and Byrne (1992) suggest that the cortical effort of matching an externally-paced rhythm for BF induces changes in the autonomic control of the heart. In addition, research is contradictory on the effects that paced breathing has on HRV. Patwardhan *et al*, (1995) showed no significant changes in HF, and therefore vagal control, when paced breathing was compared to un-paced breathing. This was also true for Stark *et al* (2000) who observed significant decreases ($p<0.05$) in HRV but did not observe any significant decreases in the HF component of HRV.

Section 7.3 of the present study aimed to observe whether there was a cortical affect of controlling breathing to 0.2 Hz, when compared to a matched spontaneous breathing frequency of 0.2 Hz. Therefore, if there was a significant reduction in the HF component, this would highlight the need to control breathing during other

studies (when observing interventions such as heat exposure). However, if there was no significant reduction in the HF component, this would negate the requirement to control for breathing during other trials.

2.7.4 The effect of sauna exposure on HRV.

According to Brenner *et al*, (1998), there is minimal information available on the effect of adverse conditions such as heat exposure, on the responses of the sympathetic and parasympathetic branches of the autonomic responses of heart rate as assessed by HRV. Thus, this section of the present study aimed to advance previous research that investigated the effects of psychological stress (Boutcher, *et al*, 1998 and Sacknoff, *et al*, 1993) in thermoneutral conditions. This would be conducted through investigating the effects of exposure to extreme heated environments and the resultant effect on HRV. Therefore, in order to investigate the effect of heat exposure on HRV, the present study examined the HRV responses of 10 healthy male subjects on exposure to a Finnish sauna. The sauna was incorporated into the study to investigate the sensitivity of HRV responses during heated conditions.

2.7.5 HRV and LFTEs.

There has been no research to date investigating the effects of LFTEs on HRV. The present study measured HRV immediately pre and post exposure to LFTEs. It was not possible to monitor HRV during LFTEs as a sufficient length of time did not elapse whereby the BAIs remained static to obtain meaningful results. Therefore, the final HRV section observed the effects of exposing BAIs to LFTEs and the resultant HRV responses.

2.8 Live fire training and heat stress.

Foster and Roberts (1994) monitored fire fighters in determining time limits of exposure to fires. Fire fighters were exposed to a fire exercise using a crib fire (fire constructed from paper, straw and wood) whilst in a fire house (training building to simulate housing). They were monitored using a humidity probe, thermocouples, and air flow transducer that were attached to the outside of their tunics.

Foster and Roberts (1994) proposed exposure time limitations to different ambient temperatures. At lower temperatures, limits for exposure to routine conditions (typically 100^0C) should not exceed 25 minutes. At higher ambient temperatures, called 'hazardous conditions', they suggested exposure to 160^0C should be for no longer than 1 minute (Foster and Roberts, 1994). They also suggested that under extreme conditions (typically between 160^0C and 235^0C) fire fighters should only be exposed long enough to rescue an individual (no longer than 1 minute). Finally they defined temperatures in excess of 235^0C as critical conditions and stated fire fighters would not be expected to operate within these conditions. In addition, Foster and Roberts (1994) conclude that there is a deficit of physiological, psychological and biochemical data from fire fighters during fire exercises.

The limits proposed by Foster and Roberts (1994) were based on the data from the external temperature, humidity and airflow probes. They did not obtain physiological measurements obtained from the subjects themselves. Foster and Roberts (1994) reported that instrumentation necessary for these measurements was too heavy for operational use but concluded that

> 'the information gained from the study may provide a basis of discussion and, possibly, further elaboration in the future' (page 5).

There appears to be some research into the physiological responses of fire fighters to heated environments, such as the research carried out by Bennett *et al* (1995). They investigated tolerance levels of fire fighters carrying out physical activity whilst simulating shipboard fire fighting drills. Bennett *et al* (1995) stated that there appears to be very little information on the physiological responses of BAIs to LFTEs. As previously mentioned, the BAIs undertake very different roles to those performed by fire fighters and fire fighter students. During LFTEs the BAIs perform very little physical work, tending to remain at a low level of intensity, but are necessarily exposed to a hot environment (107 (± 32.9) ^{0}C). As such, the current study has focussed on this aspect of fire fighting, in addition to the more obvious effects of heat stress.

2.9 Psychological measurements.

In order to investigate the psychological affects of LFTEs on BAIs, the study incorporated two psychological tests. The first was the rate of perceived exertion (RPE) scale (Borg, 1982). This was a 15 point scale rating usually utilised in exercise tests. However, the RPE scale was used to obtain a subjective view of how hard the BAIs perceived they were working at different times throughout the LFTEs.

An adapted version of the Stroop colour word interference test (Stroop, 1935) was also used within the study (refer to section 3.4.13i in the Methods). The adapted Stroop test was tested against the original to verify the adaptation (refer to Appendix B). The time to complete the adapted Stroop test, per word, was not significantly different from the time to complete the original Stroop test. This suggests that validity was not affected when adapting the original Stroop test. The original Stroop test contained 200 word characters (red, blue, green, brown and purple) which could potentially increase the time for completion. This in turn would interrupt the LFTEs and render the Stroop test too invasive to use during exposure. A modified version (containing nine word characters – Table 3.8 in the Methods) was therefore utilised. Stroop (1935) stated that congruent colours should not follow one another when completing a Stroop test. The nine word characters used within the adapted Stroop test were the minimal number of words that allowed this rule to be adhered to.

The Stroop test was used to observe the effect of heat on concentration at different times during exposure to a sauna and also LFTEs. The time to carry out the adapted Stroop Test was recorded. The stress placed on the subjects by undertaking the adapted Stroop test resulted in minimal effects on the HRV responses. This was confirmed by other research (Delaney and Brodie, 2000) that observed small changes in HRV. Figure 2.6 shows a BAI carrying out the adapted Stroop test during a LFTE.

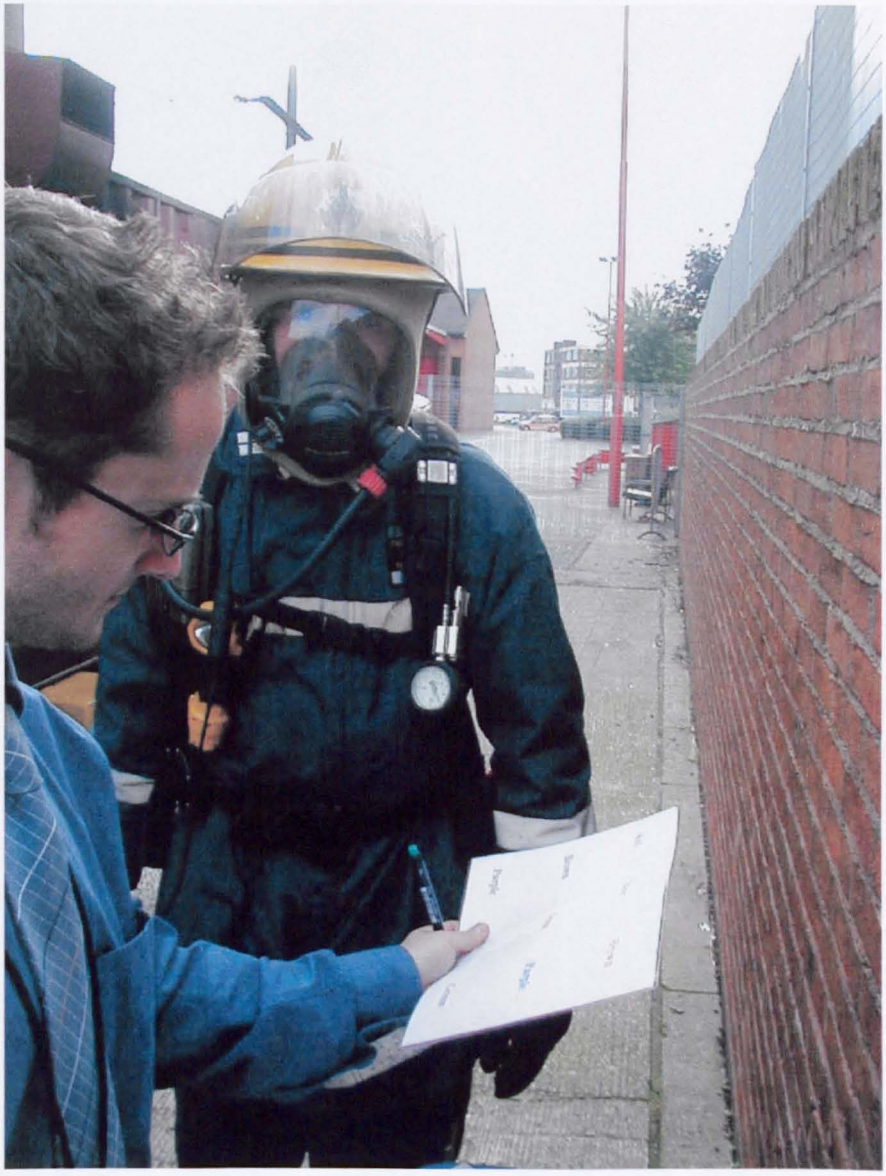


Figure 2.6 – A BAI carrying out the adapted Stroop test (psychological test to measure concentration) prior to a LFTE.

2.10 Blood glucose measurements.

To observe whether BAIs were hypoglycaemic as a result of exposure to LFTEs, blood glucose was monitored immediately pre and post exposure to LFTEs.

Research investigating the effect on blood glucose when subjects were exposed to heated environments is contradictory. Nielsen (1994), observed no significant differences in glucose utilisation as a result of exercising in temperate conditions (34^0C) when compared to exercising in thermoneutral conditions (25^0C) (0.4 $mmol \cdot min^{-1} \cdot kg^{-1}$ and in the thermoneutral conditions 0.6 $mmol \cdot min^{-1} \cdot kg^{-1}$). Nielsen (1994) suggests there were no significant metabolic changes in the exercising muscles of their subjects. This signifies heat does not alter muscle metabolism and hence glucose levels, which confirmed the findings of earlier research by Nielsen *et al*, (1990).

However, the results from Nielsen (1994) are in contrast with others. Fink *et al* (1975) observed significant increases ($p<0.05$) in glucose levels during temperate conditions (40^0C), when compared to exercising in environmental conditions of 9^0C. The results from the research by Fink *et al* (1975), are also in agreement with the research carried out by Parkin *et al*, (1999), who also observed significant increases ($p<0.05$) in glucose concentration at the cessation of maximal exercise when temperate conditions (32^0C) were compared to thermoneutral environments (16^0C).

Therefore, the BAIs blood glucose was recorded to observe whether their levels would deviate from the normal range between 4.0 mmol·Litre to 6.1 mmol·Litre (Colberg, 2001) through exposure to LFTEs.

2.11 Blood lactate measurements.

Blood lactate was observed in ascertaining the workload encountered by BAIs during LFTEs. In addition, research is contradictory on the lactate responses to temperate (30^0-42^0C) conditions compared to thermoneutral (15^0-29^0C) conditions. Nielsen (1994), observed that exposure to temperate conditions may induce metabolic changes within the exercising muscle. Research carried out by Fink *et al*, (1975) and Kozlowski *et al* (1985) observed that the increase in muscle temperature may contribute to the increased lactate production. Furthermore, Fink *et al* (1975) found

the increased lactate production may be a result of a decrease in blood flow to the working muscles during exercise in temperate environments. This is a result of an increased blood flow to the subcutaneous layer in order to increase heat dissipation from the body's core. However, Nielsen (1994) did not observe a significant decrease in muscle blood flow or, significant increase in lactate when temperate conditions were compared to thermoneutral conditions.

Therefore, the study observed whether exposure to the hot conditions of the LFTEs significantly altered the levels of blood lactate.

2.12 Hydration, plasma volumes and body water analysis.

In resting humans there is an intermittent loss of water and electrolytes from the kidneys and the gastro-intestinal tract, in conjunction with a constant loss of fluid from the respiratory tract and skin. In addition, there is an increased loss of fluid through sweating when exposed to heat and/or exercise (Greenleaf, 1992).

Under normal, or thermoneutral conditions, thirst, as a mechanism of fluid ingestion, ensures adequate fluid intake (Greenleaf, 1992). However, under conditions that are physiologically and/or psychologically stressful, this reflex may not elicit a sufficient stimulus to maintain body fluid balance. Greenleaf (1992) terms this imbalance between hydration and the voluntary response to drink as involuntary dehydration. Hence, an individual may drink until satiety has been reached although the body is still in a dehydrated state. Without careful monitoring, this dehydrated state may lead to a loss of concentration (Brooks *et al*, 1996) during LFTEs leading to decrements in the BAIs performance.

According to Bray *et al* (1986), the human body is comprised of 60% water (Total Body Water - TBW), where 54% of this volume is in the cells (intracellular - ICW). The remaining 46% is outside the cells and represents the extracellular water (ECW). For a 70 kg man these values equate to approximately 42 litres for TBW, with ICW constituting 23 litres and ECW equating to 19 litres. There are approximately 3 litres of the extracellular fluid that constitute the blood plasma whilst the rest (interstitial fluid) is used to provide the cells with an aquatic environment (Bray *et al*, 1986).

According to Manore and Thompson (2000), the body is constantly monitoring the hydration status through adjusting intake and output. The concentration of blood increases with increases in fluid losses through the sweating process. The body responds through reducing the water loss from the kidneys, stimulates the thirst process and vasoconstricts the blood vessels to increase blood pressure. Water loss from the kidneys is reduced through the action of anti-diuretic hormone (ADH). An increase in osmolality of the plasma stimulates the osmoreceptors in the hypothalamus to release ADH. This causes an increase in water reabsorbtion in the kidney, thus attempting to maintain plasma volume (Wade, 1984).

BAIs have to work in environments that are thermally stressful. During exposure to these environments there is a high sweat rate that attempts to obtain thermoregulation in order to maintain body temperature (Pandolf *et al*, 1988, Noakes *et al*, 1985). However, through wearing PC+SCBA, thermoregulation and thus maintenance of body temperature is unlikely to occur as the process of evaporation becomes impaired (Havenith, 1999).

Sawka and Greenleaf (1992), reported that during extremely hot conditions, dehydration can occur. According to Brooks *et al*, (1996), fluid losses that equate to 2% of body mass can lead to a decrement in performance. Body mass losses, as little as 1%, will lead to a loss of concentration and may also represent the threshold for impaired exercise thermoregulation. This may lead to a decrement in physical work capacity (Greenleaf, 1992). A loss of concentration whilst the BAIs are working during the LFTEs would be unacceptable.

In order to ascertain whether exposure to LFTEs caused the BAIs to become dehydrated, they were analysed for changes in body water, fluid balance and estimated plasma volumes. In addition, the study observed whether hydrating the BAIs prior to exposure or during exposure, maintained their fluid balance during the days when they undertook LFTEs.

The Dual Scan 2005 (Bodystat, Isle of Man, England)) was used to monitor the changes in body water. Estimated body water was recorded at the beginning and end

of each shift. Readings were not recorded immediately pre and post exposure to the heat due to an over-estimation of total body water results (refer to Appendix C). Bioelectrical Impedance Analysis (BIA) is a widely used method to estimate body composition. It uses ranges from hospitals and health clubs to use by private clinicians'. According to the National Institutes of Health Technology Assessment Conference Statement (NIHTACS, 1994), it can be used to assess a wide variety of body weights, ages, and disease states utilising the electrical impedance of body tissues. According to NIHTACS, (1994), BIA provides an accurate estimation of total body water (TBW).

Fluid balance was ascertained from the fluid intake subtracted from the fluid lost during the days LFTEs were undertaken. Fluid intake was monitored by the BAIs keeping a diary of the total amount of fluid they consumed. Total losses in fluid were calculated from the amount of urine produced throughout the days when LFTEs were undertaken and from the fluid losses that were calculated from sweat losses (obtained from weight losses) when subjects were exposed to LFTEs.

Plasma volumes were estimated using the equation from Van Beaumont *et al* (1972) (refer to section 3.4.5ii in the methods) using haematocrit volumes obtained pre and post exposure to LFTEs.

2.13 Cortisol and Osmolality.

It was hypothesised that exposure to the LFTEs and the resultant heat stress that the subjects would be exposed to, would in turn, create a stress response. This stress response would be highlighted through an increase in the salivary cortisol levels.

The stress hormones (cortisol, adrenaline and growth hormone) play a salient role in meeting the demands of the circulatory systems during a stress response. The secretion of adrenaline appears to be very sensitive, with an increase in stress creating an increase in the sympathetic nervous system response. This, in turn, creates a release of adrenaline within a few seconds of exposure to a stressor. However, the hypothalamic-pituitary-adrenal axis system (which is responsible for the release of cortisol) is much slower with a release of cortisol delayed anywhere

between 20 to 30 minutes (Brenner *et al*, 1998) post exposure to a stressor. Cortisol is bound to corticosteroid-binding globulin and the effects of this glucocorticoid are not seen until the capacity of the globulin is exceeded (20 $g \cdot ml^{-1}$). This results in the 20-30 minute response time (Brenner *et al*, 1998). This suggests that short term stresses may induce little or no change in cortisol secretion. The LFTEs were on average in excess of 30 minutes, suggesting changes in cortisol concentration should be detected between the pre and post exposure readings.

Newsholme and Leech (1994) report that stress caused by anxiety or aggression elevates adrenaline production, which stimulates the activity of triacylglycerol lipase and creates an increase in blood free fatty acid (FFA) concentration. Pollard (1995), also suggests that cortisol modifies fat metabolism, which leads to an overall elevated FFA release from the adipose tissue. This 'fight' or 'flight' response appeared to be a necessity for primitive man where the increased production of FFAs would be used for energy production. However, modern day man appears to be placed in situations that lead to a stress response which are followed by periods of inactivity. This may have health implications through the elevation of the FFAs in the blood remaining high for hours after exposure to the stressor. The increase in FFAs may contribute to the development of such cardiovascular diseases as atherosclerosis. Office for National Statistics (2001) state there is an increase in cardiac related deaths compared to figures obtained from previous years. This increase appears to be paralleled by the increase in stress of the modern working world (Newsholme and Leech, 1994).

As previously reported, Washburn and Harlow (1982), reported that 52% of American fire fighter deaths were caused by heart attacks. In addition, heart attacks claimed more lives of males between the ages of 35 and 45 years in the UK in 2001, than any other cause of death (Office for National Statistics, 2001). It would therefore, seem appropriate to monitor stress levels pre and post exposure to LFTEs to observe the combined effects of wearing PC+SCBA, carrying out the necessary low intensity exercise, the heat stress, and also hydration on the BAIs. Williams, Petersen and Douglas (1996) investigated the heart rate responses of BAIs to LFTEs. They concluded that, in order to further investigate the BAIs responses to heat stress on exposure to LFTEs, appropriate biochemical markers of stress should be

monitored. They also reported that biochemical analysis was beyond the scope of their study.

It was beyond the scope of the present study to measure adrenaline responses. The present study assessed the use of glycerol as an indirect measure of a stress response. However, the method of assessing plasma glycerol was not sensitive enough to detect changes in glycerol concentration. Therefore, investigation of the stress responses of BAIs, in response to exposure to LFTEs, used biochemical analysis of salivary cortisol concentration.

2.13.1 Cortisol.

Cortisol is a glucocorticoid. These hormones are so named because they increase blood glucose concentration through an increased production of glucose by the liver and a reduction in use by other tissues (Newshome and Leech, 1994). There is also an increase in the rate of gluconeogenesis which occurs, in part, through cortisol increasing the concentration of the key gluconeogenic enzymes (fructose bisphosphatase and phosphoenolpyruvate carboxykinase) (Newsholme and Leech, 1994). According to Powers and Howley (2001), the increased production of glucose is a result of cortisol stimulating free fatty acid (FFA) and glycerol mobilisation (which is transported to the liver for gluconeogenesis). The FFA's and glycerol are transported from adipose tissue through increasing the sensitivity of lipolytic hormones (catecholamines).

Whether the source of stress is vigorous physical activity, psychological stress or exposure to an adverse environment (all of which are present during LFTEs), the body produces a co-ordinated response involving an increase in brain levels of dopamine. Dopamine, in turn, causes the hypothalamus to secrete corticotropin-releasing factor (CRF), which stimulates the anterior pituitary to release adrenocorticotropin (ACTH) (Brooks, Fahey and White, 1996). ACTH (through secondary messenger systems) causes the adrenal cortex of the adrenal gland to release cortisol (Brooks *et al* (1996) and Brenner *et al*, (1998)). In addition, there is an activation of the sympathetic nervous system that stimulates the release of catecholamines from the adrenal medulla.

Salivary cortisol concentrations have been reported as a valid and reliable measure and representation of plasma levels (Kugler *et al*, 1996). Research by Lac and Berthon (2000) demonstrated a 1.5-fold increase in salivary cortisol between pre 10 kilometre race values and those obtained during the race. This suggests salivary cortisol is sensitive to exercise stress. Brenner *et al*, (1998) showed that salivary cortisol was sensitive to heat stress through observing an increase from 12.3 (± 8.5) nmol·litre^{-1} pre exposure to 23.6 (± 20.2) nmol·litre^{-1} post exposure. Salivary cortisol peak values are reached 1-2 minutes after the maximal plasma cortisol values, suggesting that, given sufficient time, either salivary or plasma values can be used to measure cortisol concentration (Pollard, 1995). Salivary cortisol was the preferred method of sample collection due to the field testing nature of the study.

2.13.2 Osmolality.

Ship and Fischer (1997), showed osmolality increased with dehydration, suggesting that osmolality may indirectly indicate a decrease in hydration state. They also observed that salivary flow rate decreased when their subjects were dehydrated. Costa *et al* (1980), also observed that when osmolality was increased during dehydration, salivary flow rate was decreased. However, the equilibrium between saliva and plasma cortisol concentrations remains, even when the salivary flow rates are altered (Ellison, 1988). Blannin *et al* (1998) reported that the increase in osmolality during their study was a result of an increased loss of saliva water by evaporation. This occurred as the exercise intensity increased and their subjects were breathing through their mouths. However, due to the low intensity of exercise BAIs carry out during the LFTEs, it is unlikely that a decrease in salivary flow rate or increase in osmolality would be due to an exercise induced loss of salivary water. Rather, alteration of hydration state through exposure to the heat during LFTEs would result in an increased water loss and thus, increased osmolality. During a stress response there is an increase in adrenaline secretion which, in turn is responsible for a decrease in salivary flow rate and an increase in salivary osmolality (Selye, 1976).

Therefore, saliva osmolality was monitored as an indicator of hydration. However, Selye (1976) suggests it may also be an indirect indication of a stress response

through increased adrenaline production. This increase in adrenaline creates a decrease in salivary flow from the parotid salivary gland.

2.14 Thesis Aims.

The overall aim of the study was to obtain an enhanced physiological understanding of BAIs responses during LFTEs using a range of physiological, psychological and biochemical parameters. In summary, the main output is aimed to provide recommendations for the Fire Service to reduce any potential stressors.

2.15 Research Questions:

Therefore, the study attempted to answer these research questions:

1. Is there additional stress placed on individuals when wearing protective clothing? If so, is it possible to:
    a. Quantify what contributes to the HR responses incurred whilst wearing PC+SCBA?
    b. Identify what causes the additional stress?

2. Can incorporating hydration strategies prior to, or during LFTEs:
    a. Reduce the physiological, psychological and biochemical responses of BAIs during LFTEs?
    b. Result in the BAIs maintaining fluid balance throughout the days of LFTEs?

3. When using heart rate variability (HRV) to detect changes in the sympathetic and parasympathetic balance of HR control:
    a. Is the HRV method sensitive enough to detect these changes: when breathing is controlled; during hot environments; when different hydration strategies are implemented during LFTEs; and identify whether BAIs are stressed as a result of exposure to LFTEs?

4. Is salivary cortisol a suitable measurement of short term interrupted stress as found during LFTEs? If this method is viable, can it be adapted as a useful field-based test in this respect?

Chapter 3

Methods.

3.1 Breathing Apparatus Instructors during Live Fire Training Exercises (Protocols 1-3).

3.1.1 Subjects.

The subjects that participated in the study were 6 male BAIs (mean age 46 (± 5.4) years, weight 81.8 (± 14.6) kg, and height 176.3 (± 9.5) cm and $VO_{2\ max}$ 39.9 (± 7.8) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) from Greater Manchester Training Headquarters. The subjects represented the total BAI population from the Greater Manchester Training Headquarters. Subjects had at least 1 years experience in carrying out BAI duties and were from various watches within the Greater Manchester Training Headquarters. All subjects were required to give their written informed consent before participating within the study.

Both the Chester College Ethics Committee and the Greater Manchester Fire Safety Officer approved all studies.

3.1.2 Pre-test conditions.

Subjects were required initially to have emptied their bladder, not eaten for at least 4 hours, not ingested caffeine for at least 2 hours and not carried out any physical exercise for at least 12 hours prior to all testing.

3.1.3 Testing Period

Due to health and safety regulations, subjects were not allowed to be exposed to LFTEs on consecutive days. Hence, they were monitored on alternate days through out the week, for example, one was monitored on Monday morning and Wednesday morning, whilst the other subject was monitored on the Tuesday and Thursday morning. This test pattern was also followed during the mock training exercises.

3.2 Physiological Responses During Live Fire Training Exercises (LFTEs) (Protocol 1).

3.2.1 Aim/Objective of Study.

In comparing the physiological responses of subjects during live fire training (when under live fire conditions) to mock (simulated) training exercises, the contribution of fire and students to the overall stress placed on subjects was assessed.

3.2.2 Mock Fire training exercises - 34 minute exposure to the FFU without fire or students present.

3.2.2i Mock Training Exercises.

The subjects conducted the 'mock' fire exercises as they would the LFTEs, carrying them out once every other day for approximately 34 minutes. They followed a set schedule, which was a typical training exercise (Table 3.2) that had been designed by the BAIs. When the subjects were exposed to the mock training exercises, they carried out duties as normal, whilst dressed in PC+SCBA, but the fire was not present and the students were also absent. The subjects were exposed to the FFU on the same days of the week for all weeks and the times of the mock training exercises were the same for all the days of testing.

Refer to Table 3.1 for details on the parameters that were measured immediately pre, during and immediately post exposure to the mock training exercises.

3.2.3 LFTEs.

3.2.3i Thirty four minute exposure to the FFU with fire and students present.

The LFTEs followed the schedule which was carried out during the mock training exercises with the exception of the inclusion of the fire and students. Refer to Table 3.1 for details on the parameters that were measured immediately pre, during and immediately post exposure to the LFTEs.

Table 3.1 – Physiological and Psychological Parameters measured pre, during and post Mock and LFTEs for all 3 Protocols.

Immediately Pre exposure	**During exposure**	**Immediately Post exposure.**
Body mass	Heart Rate	Body mass
Body water*	Skin, aural and micro-climate temperature	Body water*
Blood pressure	Adapted Stroop Test	Blood pressure
Haematocrit volumes	RPE scale	Haematocrit volumes
Lactate	Changes in bar pressure readings between each exercise.	Lactate
Glucose		Glucose
Resting Heart Rates		Resting Heart Rates
HRV		HRV
Aerobic capacity**		Cylinder volumes
Cylinder volumes		Skin, aural and micro-climate temperature
Skin, aural and micro-climate temperature		Saliva (for Cortisol)
Saliva (for Cortisol)		RPE Scale
RPE Scale		Adapted Stroop Test
Adapted Stroop Test		
These parameters were taken immediately before the instructors entered the FFU for both Mock and LFTEs.	These parameters were taken during the time when the instructors were actually in the FFU for both Mock and LFTEs. The heart rate and body temperature was continually monitored throughout exposure to both the Mock and LFTEs. The adapted Stroop test and RPE was carried out once for each instructor at the half way point of each Mock and LFTE.	These parameters were taken immediately on leaving the FFU for each instructor for every exposure to the Mock and LFTEs.

Exposure time – Approximately 34 minutes

**** - This parameter was measured at the beginning and again at the end of the shift, not immediately pre and post exposure to the Mock and LFTEs.***

***** - This parameter was measured a week prior to the pre measurements only.***

Table 3.2 – Schedule followed by the BAIs during all Mock (BAIs conducted mocks as if students were present) and LFTEs for all protocols.

Time (mins)	Exercise	Description
1:00-2:43	Demo	Demonstration to students of typical fires they will encounter during exercise.
2:43 – 4:14	Debrief	BAIs discuss how to fight the fire in the anti-chamber whilst the fire is off (although some heat will remain in the anti-chamber).
4:14 – 5:21	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
5:21 – 7:28	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
7:28 – 8:14	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
8:14 – 9:47	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
9:47 – 11:07	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
11:07 – 16:05	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
16:05 – 17:52	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
17:52 – 21:25	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
21:25 – 22:20	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
22:20 – 24:30	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
24:30 – 26:12	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
26:12 – 28:58	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
28:58 – 29:29	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
29:29 – 30:31	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
30:31 – 31:30	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
31:30 – 32:50	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
32:50 – 34:13	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
34:13 – 36:10	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
36:10 – 36:58	Exercise	BAI monitor 2 students fighting fire in both the anti-chamber and fire chamber.
36:58 – 38:10	Debrief	Debrief of students performance with fire switched off in anti-chamber only.
38:10	Exit	Students and BAI leave fire chamber.

3.3 Study To Investigate The Effects Of Hydration On The Physiological Responses Of Breathing Apparatus Instructors (BAIs).

3.3.1. Aims/Objectives of study:-

The aim of the study was to observe differences between the physiological responses of hydrated subjects (they were required to consume 600 ml of water 1 hour prior to the LFTEs) (mean urine osmolality 246.8 (± 135.2) mOsmols·kg^{-1}, urine volume 304.2 (± 156.9) ml and colour 1 (± 0.4)), when compared to euhydrated subjects (mean urine osmolality 486.5 (± 169.4) mOsmols·kg^{-1}, urine volume 275.8 (± 83.1) ml and colour 2.0 (± 0.5)) (i.e., the subjects were neither dehydrated nor hyperhydrated) (Protocol 2).

During Protocol 3, euhydrated subjects (mean urine osmolality 394.2 (± 293.8) mOsmols·kg^{-1}, urine volume 262.5 (± 80.2) ml and colour 2.0 (± 1.0)) and hydrated subjects (mean urine osmolality 214.5 (± 194.0) mOsmols·kg^{-1}, urine volume 395.8 (± 222.7) ml and colour 1.0 (± 0.5)), were also compared to the responses elicited by euhydrated subjects (but drank during the LFTEs) (mean urine osmolality 475.3 (± 364.2) mOsmols·kg^{-1}, urine volume 283.3 (± 68.3) ml and colour 2.0 (± 0.8)).

3.3.2. Live fire Training Exercises.

The LFTEs during Protocols 2 and 3 followed the schedule undertaken during the mock and LFTEs (Table 3.2). Refer to Table 3.1 for details on the parameters that were measured immediately pre exposure, during exposure (approximately 15 minutes into the LFTEs) and immediately post exposure to the FFU.

3.3.2i. LFTEs where the subjects were either euhydrated prior to exposure, hydrated prior to exposure, or, euhydrated prior to exposure but drank during the LFTEs.

Subjects were randomly assigned to either one of three groups; either the LFTEs when euhydrated prior to exposure (not hydrated (urine colour of 1.0) or dehydrated (mean

urine colour of 3.0 or more)), hydrated prior to exposure (consumption of 600 ml of water 1 hour prior to exposure to the LFTEs with a urine colour of 1, and osmolality values equating to less than 320 $mOsmols{\cdot}kg^{-1}$), or, when they were euhydrated prior to exposure but drank during the LFTEs.

The subjects who carried out the LFTEs when hydrated prior to exposure were required to drink 600 ml of water 1 hour prior to exposure to the FFU. Euhydrated subjects were not actively encouraged to consume fluid during the 2 hour period before exposure. Water consumed after the first 10 minutes of the LFTE consisted of 200 ml and an additional 200 ml every 10 minutes until a total of 600 ml had been drunk. Any fluid consumed was recorded in fluid diaries, however, at no time was water denied to the subjects.

Table 3.3 – Timing of fluid ingestion during Protocols 1, 2 and 3.

	Prior to exposure		During exposure***
	Euhydrated*	Hydrated**	
Protocol 1	0 ml	0 ml	0 ml
Protocol 2	0 ml	600 ml	0 ml
Protocol 3	0 ml	600 ml	200 ml (x3)

Exposure time – 34 minutes

* - BAIs consumed no fluid 2 hours prior to exposure.
** - BAIs consumed 600 ml of water 1 hour prior to exposure
*** - BAIs consumed no fluid for 2 hours prior to exposure but consumed 200 ml of water every 10 minutes throughout exposure to the LFTEs.

3.4 - Parameters.

Prior to testing the subjects were given the opportunity to familiarise themselves with all of the procedures listed below:

3.4.1 Body Mass and Height.

Height was measured using a free-standing stadiometer (Leicester height measures, Seca Ltd, UK). Body mass was measured (pre and post exposure) using the Salter scales (Model N° 109, England) whilst subjects were either nude or in protective clothing and self contained breathing apparatus (PC+SCBA) (trousers, t-shirt, helmet, A26 tunic and trousers, boots, gloves, SCBA and the lightweight cylinder). Nude body mass was obtained post-exposure after a vigorous towel down.

3.4.2 Body Water using Bioelectrical Impedance Analysis.

Body water was analysed at the beginning and at the end of the subjects shift. Measurements were obtained after subjects had been lying in the supine position for a period of 5 minutes. The electrodes were placed on the hand and foot and impedance measured using the Dual Scan 2005 body water analyser (BodyStat, Isle Of Man, England).

3.4.3 Blood Pressure.

Blood pressure was analysed using a manual sphygmomanometer (Spengler, Model N° CE0459, France) and stethoscope. Results were recorded immediately pre and immediately post-exposure thus observing the cardiovascular responses to the mock and LFTEs. Blood pressure was obtained after the subject had rested for 10 minutes. The blood pressure cuff was then placed on the right arm of the subject, approximately 2.5 cm from the antecubital space, with the centre of the cuff's bladder over the brachial artery. The stethoscope diaphragm was placed over the brachial artery in the antecubital space. The cuff was inflated to 160 mm Hg, after which the air was released approximately 5 mm Hg per second. On hearing two consecutive beats (Korotkoff sounds) this was taken as the systolic blood pressure, whilst disappearance of the Korotkoff sounds was taken as the diastolic blood pressure (Adams, 1998).

3.4.4 Blood analysis.

Blood sampling was obtained by venepuncture by a qualified phlebotomist immediately pre and post-exposure to the FFU, collected in centrifuge tubes (Costar, England) and placed immediately on ice until they were stored in a freezer until required. All samples were defrosted at room temperature and batch analysed by the researcher. Health and safety precautions were adhered to at all times. A blood sample (5 ml) was obtained immediately pre and post exposure to training exercises. Blood samples obtained post-exposure to the training exercises were taken at the same time.

3.4.4i Haematocrit Volumes.

Haematocrit values were obtained immediately pre and post exposure to all of the training exercises. Blood samples were placed on ice for 6 minutes. Haematocrit volumes were ascertained 'in the field' following the protocol of Dacie and Lewis (1984), using Wintrobe's tubes with an internal diameter of 2.5-3 mm and 110 mm in length, that were filled with 50 µl of whole blood. The samples were spun at 2000 **g** for 15 minutes. The Wintrobe tube was placed along side a standard scale in order for haematocrit to be obtained.

3.4.4ii Plasma Volumes.

Plasma volumes were estimated from the haematocrit values using the equation of Van Beaumont, Greenleaf and Juhos (1972)

$$\%\,\Delta PV = \frac{100}{100\text{-}Hct_{pre}} \times \frac{100\,(Hct_{pre} - Hct_{post})}{Hct_{post}}$$

The Hct_{pre} was the haematocrit obtained prior to exposure and the Hct_{post} was the haematocrit obtained post exposure to the LFTEs.

3.4.4iii Blood Lactate.

A 50 µl sample of whole blood was applied to the Accusport lactate analyser®. The timing of when the samples were obtained for analysis was kept constant throughout all testing.

3.4.4iv Blood Glucose.

A 50 µl sample of whole blood was applied to the Accusport glucose analyser®. The timing of when the samples were obtained for analysis was kept constant throughout all testing.

3.4.5 Resting Heart Rate.

A Polar heart rate monitor (PE 5000) was attached to the BAIs in conjunction with a coded transmitter, which was moistened prior to application (to improve conductance). HR was recorded every 5 seconds for the 5 minute monitoring period. The transmitter was positioned immediately below the xiphoid process and tightened so that a good connection was obtained. Resting heart rate was recorded over a 5 minute period after 10 minutes of seated rest whilst subjects wore underwear and then whilst wearing PC+SCBA. Subjects were encouraged not to talk and instructed to remain still. The HR data was then downloaded to the Polar software (Poland Electro Oy, Kempele, Finland) immediately afterwards. Resting HR was determined from the mean of the final 20 seconds when it was apparent that a steady state had been achieved.

3.4.6 Heart Rate Variability.

Subjects were instructed to wear a Polar heart rate monitor (PE 5000) set to measure the R-R intervals for every beat for a period of 5 minutes as described earlier. This was carried out in a seated position in order to minimise the effects of movement artifact. Subjects were encouraged not to talk to the researchers and remain still throughout the measurements. After measurements had been obtained the heart rate variability (HRV) data was downloaded to the Polar software (Polar Electro Oy, Kempele, Finland).

3.4.6i Controlled and spontaneous breathing on HRV.

Eighteen subjects from Chester College of Higher Education (11 female; mean age 19.5 (± 0.7) years, weight 63.4 (± 6.6) kg, height 161.2 (± 4.5) cm and 7 male; mean age 21 (± 1.3) years, weight 78.7 (± 6.2) kg and height 181.6 (± 4.9) cm) participated within the study. Each subject arrived at the laboratory in a 4 hour post-prandial

condition, had not exercised, consumed caffeine, or alcohol for at least 12 hours prior to all testing.

The Chester College Ethics Committee approved the study. Subjects participating in the study were screened using a medical questionnaire, thus ensuring only healthy individuals were used. All subjects gave their informed consent prior to commencement.

The investigation was a randomised cross-over study with subjects assigned to the controlled breathing (CB) or spontaneous breathing (SB).

Room temperature was controlled and maintained between 22°C and 24°C. Subjects were seated in a comfortable position which was maintained throughout the trials. The subjects were then required to have 15 minutes of seated rest, during which time they were required not to talk or move. This was followed by a further 5 minutes during which time the heart rate and breathing frequency were monitored.

This procedure was replicated following a 10-minute interval during which time subjects were allowed to converse in quiet conversation with the researchers. Once the 10 minutes had elapsed they were required to breathe at a set rate for 15 minutes. Subjects used a stopwatch to monitor their breathing. The rhythm was maintained at one cycle every five seconds therefore breathing 12 cycles·minute^{-1} (0.2 Hz). The researchers closely monitored breathing rate at all times. Following the 15 minutes, heart rate was monitored for a further 5 minutes.

Table 3.4 – Heart Rate Variability Parameters measured when breathing was either controlled or spontaneous.

Spontaneous Breathing
Heart Rate
Parasympathetic indicators (pNN50, HF Norm and RMSSD)
Sympathetic indicators (LF Norm and LF:HF Ratio)
TP, SD and absolute LF and HF.

Subjects were seated in a comfortable position that they would be able to maintain through out the trials. The subjects were then required to have 15 minutes of seated rest, during which time they were required not to talk or move. This was followed by a further 5 minutes during which time the heart rate and breathing frequency were monitored.

During exposure
Between trials there was a 10-minute interval during which time subjects were allowed to converse in quiet conversation with the researchers.

Controlled breathing
Heart Rate
Parasympathetic indicators (pNN50, HF Norm and RMSSD)
Sympathetic indicators (LF Norm and LF:HF Ratio)
TP, SD and absolute LF and HF.

This procedure was replicated once the 10 minutes had elapsed. Subjects were required to breathe at a set rate for 15 minutes. Subjects used a stopwatch to monitor their breathing. The rhythm was maintained at one cycle every five seconds therefore breathing 12 cycles·minute^{-1} (0.2 Hz). The researcher's closely monitored breathing rate at all times. Following the 15 minutes, heart rate was monitored for a further 5 minutes.

3.4.6ii The effect of sauna exposure on HRV.

Participating subjects consisted of 10 healthy males from Chester College of Higher Education. Ages ranged from 18-42 years of age (M: age 24 (± 4.7) years, weight 73.9 (± 11.1) kg, and height 177.3 (± 9.0) cm). All subjects were required to give their written informed consent before participating within the study. The subjects were instructed not to eat or drink caffeine for 4 hours prior to all testing, or have carried out any physical exercise for 12 hours prior to testing.

Body mass and height was recorded and a Polar heart rate monitor was attached (refer to sections 3.4.1 and 3.4.6).

Three temperature probes were attached to the subjects (refer to section 3.4.10). The change in aural temperature was observed between points A and B (refer to Figure 7.5).

The time taken to complete the adapted Stroop test was used as an indication of mental concentration (refer to section 3.4.13). Subjects were required to carry out the adapted Stroop test immediately before and after exposure to the sauna.

Subjects were also required to give a rating of perceived discomfort from an adapted version of the rate of perceived exertion scale (RPE) (Borg, 1982) (Appendix A). Each subject was instructed on how to use the scale and was given anchor statements that allowed the subject to know what the top and bottom figures in the scale represented. A 2 ml blood sample was taken via venopuncture by a qualified phlebotomist from the right antecubital vein immediately pre and post-exposure to the sauna.

The subjects were then seated for a period of 10 minutes. During this time subjects were required to refrain from talking and moving. HRV was monitored after this 10 minutes period for a further 5 minutes. Room temperature was 27.0 (± 0.7) ^{0}C.

The subjects were then moved into the sauna where they sat in a similar position to that elicited during the previous 15 minutes. The sauna was set to a temperature of

74.3 (± 5.9) ^{0}C. All subjects sat in the sauna for 10 minutes during which time they were encouraged not to move or talk. At the end of the 10 minutes, HRV was monitored for a further 5 minutes.

On exit from the sauna, subjects immediately gave a discomfort rating, carried out the adapted Stroop test and had a further blood sample taken. This was followed by a thorough towel down and nude body mass was obtained to calculate fluid loss.

Table 3.5 – Heart Rate Variability, Physiological and Psychological Parameters measured pre, during and post exposure to a sauna.

Immediately Pre exposure	**During exposure**	**Immediately Post exposure.**
Body mass	Heart Rate	Body mass
RPE Scale	Skin, aural and micro-climate temperatures	RPE scale
Haematocrit volumes		Haematocrit volumes
Skin, aural and micro-climate temperature	HRV*	Adapted Stroop Test
Resting Heart Rates	Parasympathetic indicators (pNN50, HF Norm and RMSSD)	
Adapted Stroop Test	Sympathetic indicators (LF Norm and LF:HF Ratio)	
HRV	TP, SD and absolute LF and HF.	
Parasympathetic indicators (pNN50, HF Norm and RMSSD)		
Sympathetic indicators (LF Norm and LF:HF Ratio)		
TP, SD and absolute LF and HF.		

Exposure time – 15 minutes

** – HRV measurements taken in the final 5 minutes of the 15 minute exposure.*

3.4.6iii HRV and LFTEs.

Pre exposure to the training exercises the subjects wore heart rate monitors and temperature probes as previously described. Subjects were rested in a seated position dressed in PC+SCBA for a period of 10 minutes. During this time they remained still and refrained from talking. HRV was monitored immediately following the 10 minutes for a further 5 minutes, during which time the subjects were controlled in an identical manner as the 10 minutes previously.

It was not possible to monitor HRV during the training exercises as previously stated. Therefore, HRV was monitored immediately post exposure to the training exercises for a period of 5 minutes whilst the subjects were seated. During the 5 minutes the BAIs were instructed to remain still and not verbally communicate.

3.4.7 Aerobic Capacity.

Aerobic capacity was assessed using the Chester Step Test. The Step Test was carried out using a 30 cm step. The subjects stepped in time to a set metronome beat. The timing device used was a Seiko Quartz Metronome (Model N° SQ-44) rather than the Chester Step Test pre-recorded stepping rate. Each level was 2 minutes in duration and the stepping rate was 15, 20, 25, 30 and 35 steps·minute^{-1} for levels 1 through to 5 respectively. Thus, the Step Test was 10 minutes in duration.

A Step Test was used on the basis that it is a valid method of obtaining aerobic capacity (Sykes, 1995), the subjects were conversant with the form of test, and it was also best suited to the field-testing nature of the study. Subjects carried out the Step Test on 4 separate occasions (subjects were randomly assigned to each of the groups to eradicate order affects). They carried out each of the test conditions at the same time of day for each of the four days whilst dressed in either gym kit (GK), gym kit with weighted rucksack (WGK) (equivalent to the weight of the PC+SCBA) or PC+SCBA (helmet, A26 tunic and trousers, boots, gloves, self-contained breathing apparatus (SCBA) and the lightweight cylinder) during thermoneutral conditions (room temperature during the GK trial was 21.8 (± 0.8) ^{0}C, WGK trial 22.5 (± 1.0) ^{0}C and PC+SCBA trials 21.0 (± 1.5) ^{0}C). There were no significant differences in room temperature observed between any of the trials.

a) b)

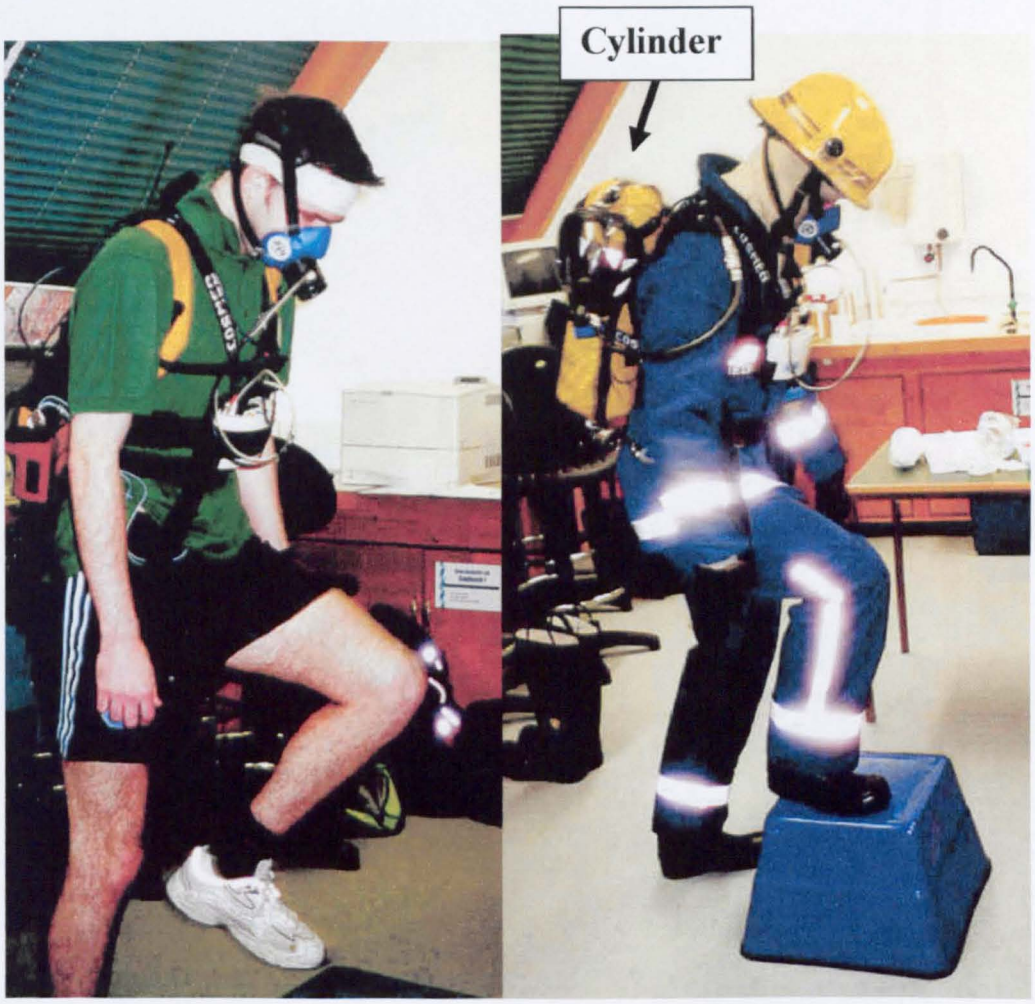


Figure 3.1 – Subjects dressed in (a) gym kit and weighted rucksack and (b) in PC+SCBA.

Figure 3.2 – Shows front and side view of BAI wearing Cosmed K4^2 facemask.

One of the PC+SCBA trials was carried out whilst wearing the on line gas analyser (Cosmed $K4^2$, Italy) and the other trial subjects were 'under air' (when subjects breathed the air from the cylinder).

Subjects were required to step with a weighted rucksack to highlight the differences between the HR and oxygen cost responses. Such responses could be due to the increase in weight of the PC+SCBA and/or the increases in heat storage due to the encapsulative properties of wearing the PC+SCBA. All subjects were required to give their written informed consent before participating within the study.

HR was monitored (refer to section 3.4.6). They were also required to wear the Cosmed $K4^2$ (Italy) portable metabolic gas analyser and facemask. Prior to all testing, the Cosmed $K4^2$ was calibrated using known gases (15.05 % O_2, 5.4202 % CO_2 and nitrogen balance). The volume transducer was calibrated using a 3 litre syringe. The Cosmed $K4^2$ has been reported to be a valid method of obtaining oxygen consumption (Hausswirth, Bigard and Le Chevalier, 1997). Readings of VO_2, VE·minute^{-1}, RER and VE/VO_2 were obtained breath by breath during rest and test conditions. The mean values were obtained from the last 20 seconds of data from each of the levels. Temperature was also measured (as described in section 3.4.10), and the mean values obtained from the last 20 seconds of data from each of the levels.

The subjects continued to step until they either reached the end of the test or volitional fatigue forced them to stop. On cessation of the test, the HR monitor, temperature data logger and Cosmed $K4^2$ stopped recording and subjects asked to cool down.

Table 3.6 – Physiological and Psychological Parameters measured when subjects carried out a Step Test dressed in either gym kit, gym kit with weighted rucksack or PC+SCBA.

Resting Parameters	Measurements obtained during Step Test
HR	HR
VO_2	VE
Skin, aural and micro-climate temperatures	VE/VO_2
RPE	VO_2
Bar pressure readings*	Skin Aural and MC temperature
	RPE
	Bar pressure readings

Bar pressure readings were only taken whilst subjects were dressed in PC+SCBA and under air (when they were breathing from the cylinder).
Bar pressure and RPE readings were taken at the beginning and end of each level.
HR, VE, VE/VO_2, VO_2 and the skin, aural and micro-climate temperature measurements were obtained from the mean of the final 20 seconds of each level.

3.4.8 Workload Equivalent.

When the BAIs wore the face mask used in conjunction with the SCBA, they breathed air from the cylinder (refer to Figure 3.1 (b)). The more air breathed from the cylinder reduced the pressure in the cylinder. Noting the changes in cylinder pressure (measured in bars) allowed ventilation to be calculated. Cylinder pressure readings were recorded immediately pre, during and immediately post exposure to the LFTEs. The equation below was taken from Louhevaara *et al* (1994). The nominal water capacity was the volume of water required to fill the cylinder, giving an indication of the volume of the cylinder.

Prediction of oxygen cost (Prediction equation obtained from drop in cylinder pressure).

Calculation of ventilation (V_E):

$$V_E \text{ (litres·minute}^{-1}) = \frac{\text{Cylinder pressure drop in bar pressure x nominal water capacity (9 litres).}}{\text{Duration of Exercise (minutes).}}$$

From the differences between the pre and post exposure bar pressure readings, the oxygen cost of working in the FFU were obtained utilising the formula below.

Calculation of VO_2:

$$VO_2 \text{ (litres·minute}^{-1}) = \frac{\text{Cylinder pressure drop in bar pressure x nominal water capacity (9 litres).}}{\text{22.5 litres of pulmonary ventilation}}$$

$$VO_2 \text{ (ml } O_2 \cdot kg^{-1} \cdot min^{-1}) = \frac{VO_2 \text{ (litres·minute}^{-1})}{\text{Body Weight (kg)}} \times 1000$$

The ventilation equivalent (VE/VO_2) used in the equation was a constant (22.5 litres·minute^{-1}) taken from the findings of Cotes (1975). During the Step Test the VE/VO_2 values were obtained from an on-line gas analyser (Cosmed K4^2 (Italy)) and were compared to the VE/VO_2 constant obtained by Cotes (1975). During levels 1 and 2 of the Step Test (refer to Figure 3.3) the VE/VO_2 values (23.9 (± 1.8) litres·minute^{-1} and (23.1 (± 2.0) litres·minute^{-1} for levels 1 and 2 respectively) were

not significantly different when compared to the constant of 22.5 litres·minute^{-1} (used during other studies such as Louhevaara *et al*, 1984). However, VE/VO_2 values for all other levels (3 through to 5) were significantly different ($p<0.05$) from the 22.5 litres·minute^{-1}. This suggests that at low intensities (equivalent to stepping at 15 or 20 step·minute^{-1}) the VE/VO_2 constant from Cotes (1975) is valid.

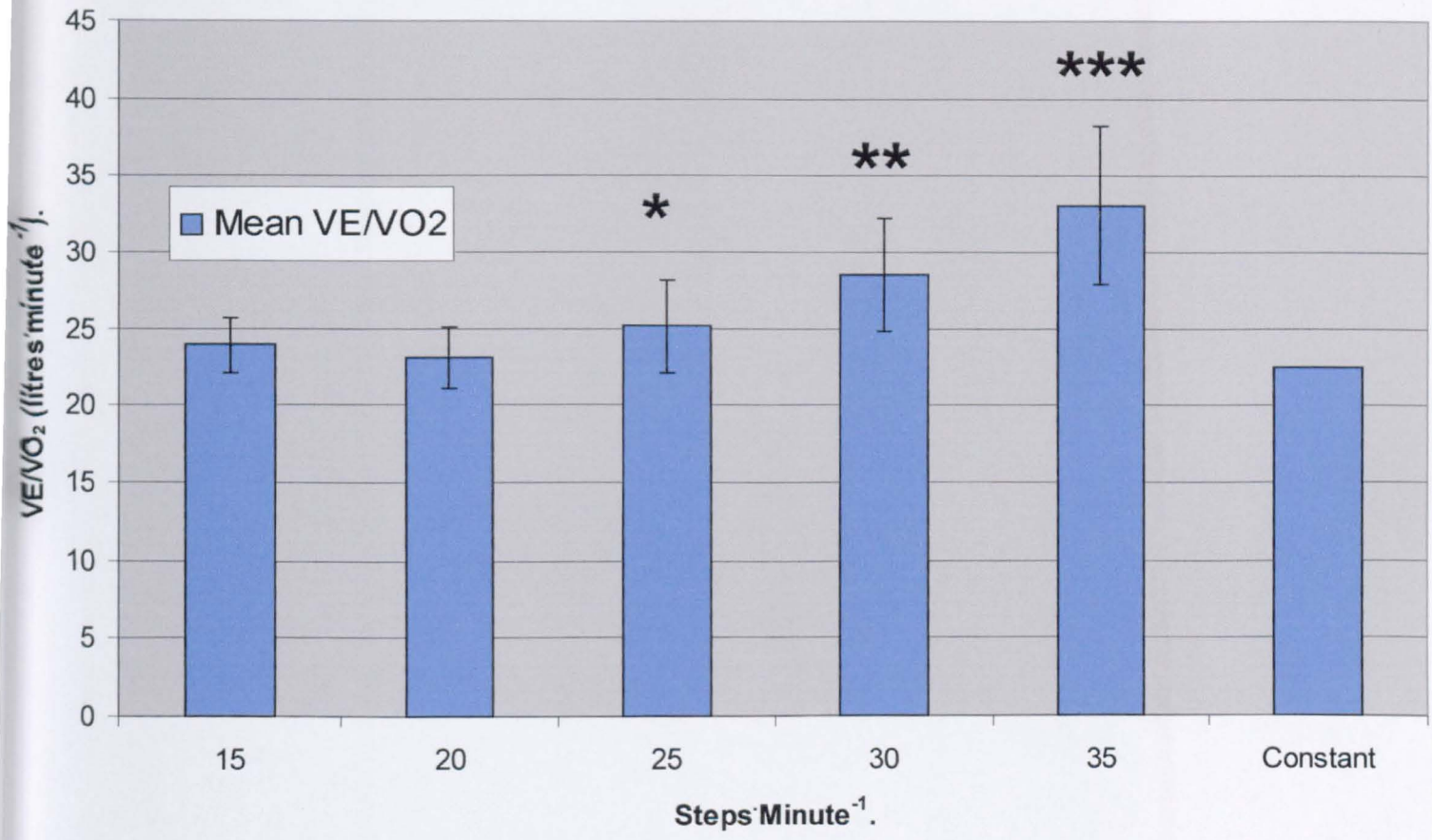


Figure 3.3 – Mean VE/VO_2 values measured directly from the Cosmed K4[2] when performing a Step Test (step rates of 15, 20, 25, 30 and 35 steps·minute^{-1} were equivalent to levels 1 2, 3, 4 and 5 respectively) and also the constant from Astrand and Rodahl (1986). Data is presented as the mean and ± SD (n = 6). Significance was tested using a paired samples t-test with a post hoc Bonferroni. * - significance between the mean VE/VO_2 values obtained when stepping at 25 steps·minute^{-1} compared to the constant (22.5 litres·minute^{-1}) ($p<0.05$). - significance between the mean VE/VO_2 values obtained when stepping at 30 steps·minute^{-1} compared to the constant (22.5 litres·minute^{-1}) ($p<0.05$). *** - significance between the mean VE/VO_2 values obtained when stepping at 35 steps·minute^{-1} compared to the constant (22.5 litres·minute^{-1}) ($p<0.05$).**

3.4.9 Temperature.

The temperature outside the FFU was monitored using a high accuracy digital thermometer (Solex, Model N° ST-3300, England). Temperature inside the FFU was monitored using K type thermocouples (Pyrotenex, England) and data logged using the Fix system (Fix Systems, USA). Skin, aural and micro-climate temperature was monitored using a 4 channel data logger (Ecolog TN4, Buchs SG, Switzerland) and three temperature probes. In order to verify the calibration of the Ecolog TN4, the researcher placed the probes in a water bath (Grant Instruments Y28, Cambridge, UK) at varying temperatures. The results are summarised in Table 3.7. The table shows the Ecolog TN4 was able to measure temperature with minimal deviations from the water bath temperature.

Table 3.7 – Summary of temperatures to calibrate the Ecolog TN4.

Water bath temperature (^{0}C)	Skin temperature probe (^{0}C)	Aural temperature probe (^{0}C)	Microclimate temperature probe (^{0}C)
21.0	21.3 (± 0.2)	21.1 (± 0.1)	21.0 (± 0.0)
25.0	25.7 (± 0.2)	25.7 (± 0.1)	25.8 (± 0.1)
30.0	30.3 (± 0.0)	30.1 (± 0.0)	30.3 (± 0.0)
35.0	35.3 (± 0.1)	35.2 (± 0.0)	35.2 (± 0.0)

Figure 3.4 shows the response time of the temperature probes to changes in water bath (Grant Instruments Y28, Cambridge, UK) temperatures. Each water temperature was monitored for 5 minutes and showed that the temperature probes response was immediate.

When the temperature probes were monitored in an incubator (Orbital, Stuart Scientific, UK) the probes adjusted to the change in incubator temperature within approximately 60-90 seconds and also reverted back to room temperature on removal from the incubator with in 60-120 seconds (Figure 3.5). Temperatures were confirmed using a thermometer (Thermosystem, England).

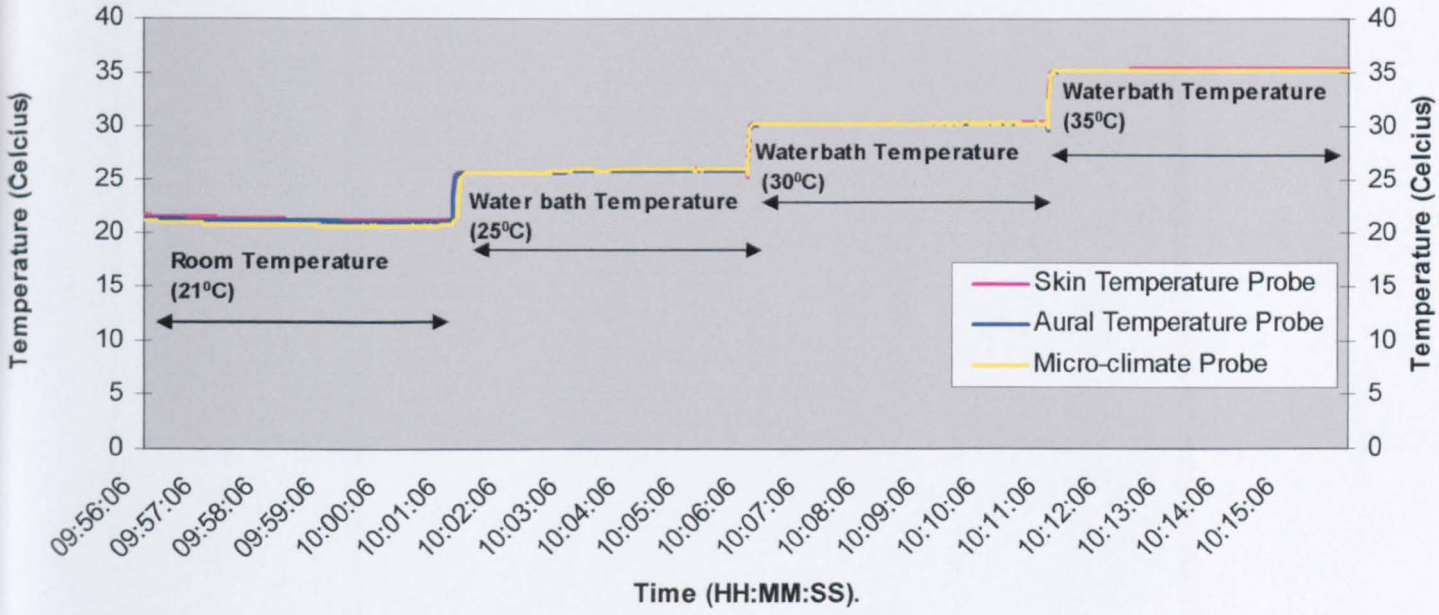


Figure 3.4 – Temperature measurements obtained at room temperature (21^0C) and when in a water bath recorded at 25^0C, 30^0C and 35^0C.

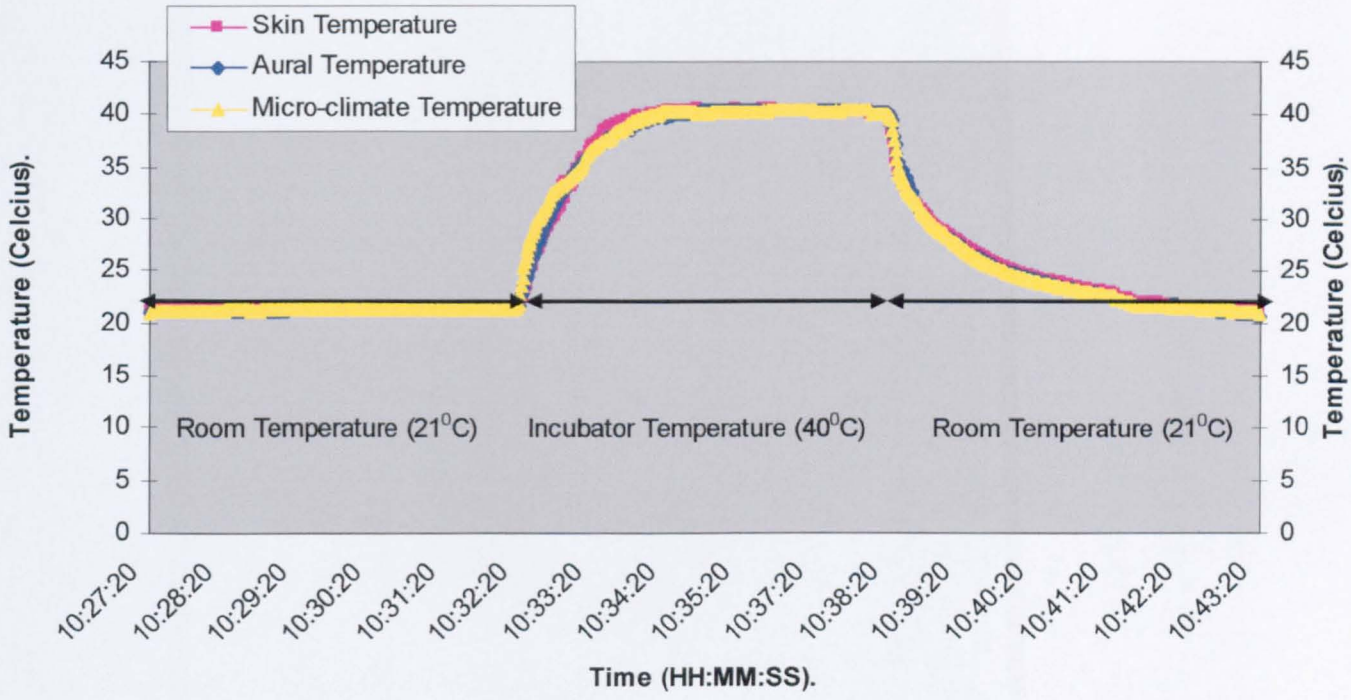


Figure 3.5 – Temperature measurements obtained at room temperature (21^0C) and in an incubator set at 40^0C.

The data logger monitored the BAIs' temperature for 5 minutes prior to all of the training exercises whilst dressed in PC+SCBA, and during all the training exercises (the data logger was started immediately prior to the start of the exercises and stopped immediately post exposure to all training exercises). Temperature was monitored for a further 5 minutes post exposure to the training exercises with the BAIs dressed in PC.

(i) Skin temperature

The skin temperature probe was attached to the pectoralis major (Liang and Norris, 1993) with surgical tape.

(ii) Micro-climate temperature

The micro-climate probe lead was threaded underneath the heart rate transmitter (that was attached underneath the xiphoid process) leaving the probe itself to hang freely between the BAIs' skin and uniform. In addition, the probe was covered in a drilled silicone sheath. This ensured the probe only recorded micro-climate temperature and not the temperature of the skin or clothing. Muir, Bishop and Kozusko (2001) observed no significant differences between the placement of micro-climate temperature probes at the shoulder, hip and thigh. This suggests that the placement of the probe in line with the xiphoid process would represent micro-climate through out the PC.

(iii) Aural temperature

The aural temperature probe (Nielsen *et al*, 1990) was inserted into the ear by the researcher and the BAIs were instructed to push the probe as far into the ear as possible so that it was not uncomfortable or painful. The probe was then covered in cotton wool and securely kept in place by surgical tape. To minimise the effect of the external environment on the aural probe, a bandage with a padded area was also wrapped around the head, which was secured with surgical tape (Taylor, Fogarty and Armstrong, 2000).

3.4.10 Urine analysis.

During the days of mock training exercises and LFTEs, each excretion of urine was analysed for colour, volume and osmolality.

(i) Urine Volume.

The urine volume (ml) was obtained by the subjects urinating straight into a jug provided.

(ii) Urine Colour.

From the jug the subjects were required to pour a sample into a 7 ml bijou bottle (Sterilin, England). The subjects monitored the urine colour from the bijou bottle using the urine colour chart taken from Armstrong, *et al* (1994) (refer to Appendix A for an example). The urine sample was then placed on ice until the samples were stored in a freezer.

(iii) Urine Osmolality.

The urine samples were defrosted at room temperature and batch analysed. A 100 µl sample was pippetted into a 1.7 ml centrifuge tube (Costar, England) and osmolality was monitored using an osmometer (Roebling, Model N° 9609011).

3.4.11 Saliva

(i) Saliva collection.

Immediately pre and post exposure to all of the training exercises, the BAIs were required to thoroughly rinse their mouths out twice with water. They were then required to accumulate saliva in their mouths for a further 4 minutes. During the 4 minutes the BAIs were required not to talk and to remain still. They were also instructed not to actively draw saliva into the mouth, rather, let it accumulate naturally. Once the 4 minutes had elapsed, the BAIs were required to spit the saliva they had collected into a bijou bottle provided. The samples were then placed on ice until they were stored in a freezer.

(ii) Cortisol Analysis (Based on Da Costa, Coleman, Jones and Williams (2002)).

Preparation -Day Before.

Prior to testing the Immulon 4 Elisa plates (Thermo Labsystems, England) were coated using anti-cortisol antibody (1/50 stock) (Dr Corelie Monroe, Vancouver, USA). Then 100 µl of the coating solution was pipetted (Finnipipette, Thermo Labsystems, England) into the inside wells of the ELISA plate and 100 µl of carbonate buffer was pipetted into the outside wells. The ELISA plate was then covered with cling-film and stored at 4°C overnight.

Preparation - On the day

Cortisol-HRP (Dr Corelie Monroe, Vancouver, USA) was diluted using EIA-PBS (0.1% BSA (Sigma, United Kingdom)) using a 1 in 60,000 dilution. A standard curve was prepared utilising a serial dilution from 20.000 $ng \cdot ml^{-1}$ to 0.039 $ng \cdot ml^{-1}$ (Figure 3.6).

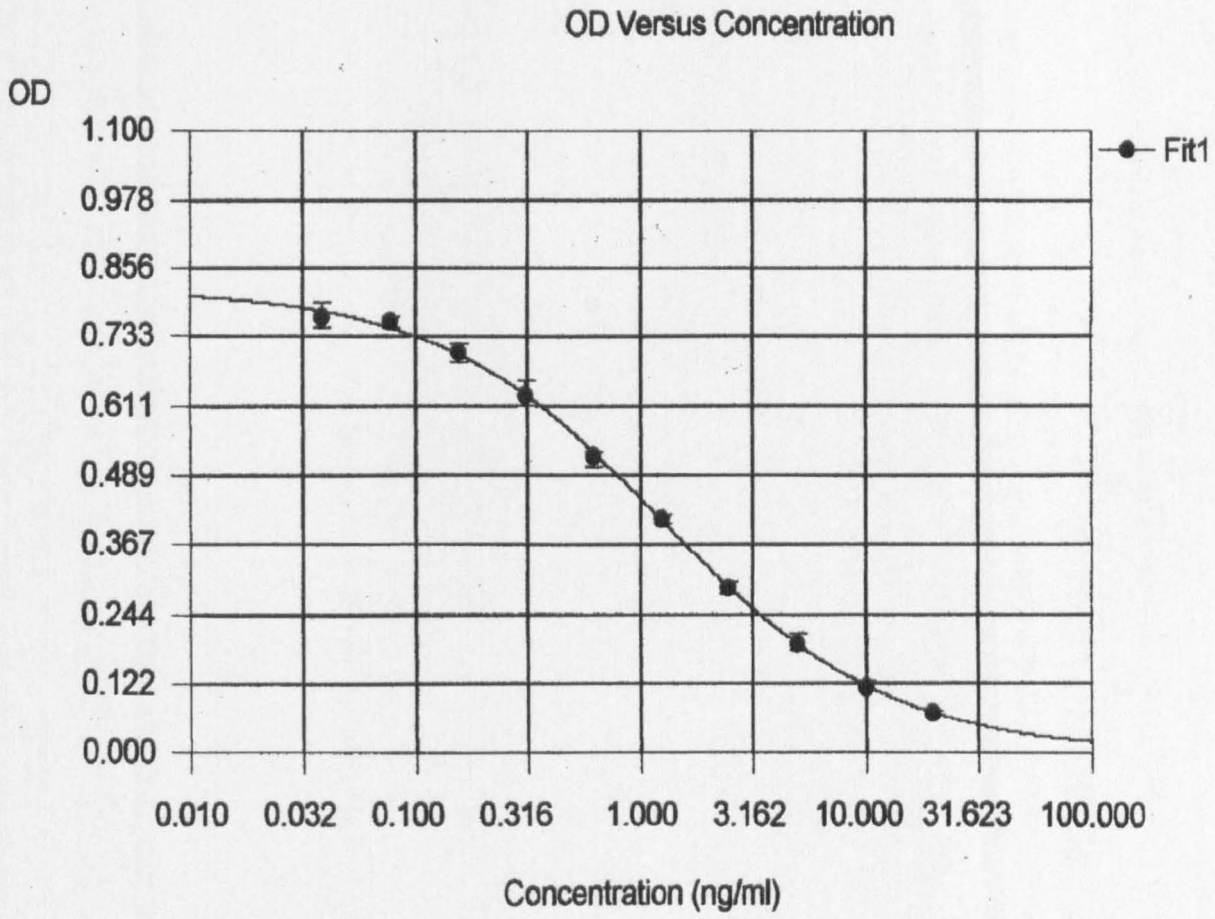


Figure 3.6 – Typical standard curve used during the Cortisol assay.

Saliva samples were defrosted at room temperature and a 1 ml sample was pipetted into a 1.7 ml centrifuge tube (Costar, England) and centrifuged (Micro-centrifuge, Hawksley, England) at 2000 *g* (centrifugal force) for 5 minutes. The supernatant was removed in readiness to be used in the cortisol ELISA. An appropriate dilution of the salivary samples was determined using distilled water prior to analysing. This was typically between 1/4 and 1/8 for saliva.

The ELISA plates were removed from storage and washed using a plate washer (Ultrawash Plus (Dynex Technologies, United Kingdom)) with wash buffer (PBS + 0.05% TWEEN 20) 3 times and tapped dry. Then 50 µl of EIA PBS was added to all wells. This was followed by 50 µl of standards, samples and controls being added to the appropriate wells. Cortisol HRP (50 µl) was also added to all the wells except the substrate control. The ELISA plates were gently tapped to ensure efficient mixing. Once the samples had been mixed, the ELISA plates were incubated at room temperature for 1.5 hours. Just before the incubation period was complete the substrate solution was prepared using a 1:10 ratio of Veterquinol TMB chromogen (Biovet, Canada) to substrate buffer (Biovet, Canada). Once the incubation period was completed the ELISA plates were washed 3 times and tapped dry. Then 100 µl of substrate solution was added to all wells. A further incubation period at room temperature was carried out for 1 hour. Once the incubation period had finished, 50 µl of 1M phosphoric acid (Fisher, England) was added to all the ELISA plate wells. The ELISA plate was then read at 450 nm using the MRX II (Dynex Technologies, United Kingdom).

Saliva samples were obtained immediately pre and post exposure and also every hour for five hours post exposure to the LFTEs. Cortisol has a relatively long circulatory half-life, suggesting that peak values are often reached well into recovery (Brenner *et al*, 1998). It was for this reason that cortisol values were obtained every hour for 5 hours post exposure to the LFTEs. The salivary cortisol would not have been affected by the delay in freezing as cortisol levels in saliva have been reported to remain stable for up to 30 days at room temperature (Nicolson, deVries and van Poll, 1992).

According to Blannin *et* al (1998) the most appropriate method of expressing changes in cortisol is accounting for osmolality. Osmolality increases when exposed to either exercise or heat (Pollard, 1995). Hence, by dividing the cortisol concentration by the osmolality it is possible to take into account the cortisol increases in proportion to the change in salivary osmolality.

(iii) Salivary osmolality.

Osmolality was measured in a 100 µl volume using an osmometer (Roebling, Model N° 9609011).

3.4.12 Psychological parameters.

(i) The Adapted Stroop Test.

An adapted version (Table 3.7) of the Stroop test (Stroop, 1935) was incorporated pre exposure, during exposure (at the mid way point through each exposure approximately 15 minutes in the exercise) and post exposure to each of the mock and LFTEs. The time taken to complete the test was recorded and used as an indication of concentration.

In undertaking the adapted Stroop test subjects were required to read out aloud the colour that each of the words was printed in and not say the colour the words were describing (Table 3.8). For example, the word in the top left hand box says red, but is printed in blue. Subjects were required to say 'Blue'. The time taken to complete the test was recorded. Subjects were given time to practice the adapted Stroop test prior to testing to avoid a practice effect.

Table 3.8 – Example of the adapted Stroop test.

Red		**Green**
	Brown	**Purple**
Green	Yellow	Red

(ii) The Rating of Perceived Exertion (RPE) Scale.

An RPE scale (Borg, 1982) reading was taken pre exposure, during exposure (at the mid-point of the training exercise) and then immediately post exposure to the LFTE for each of the BAIs. Refer to Appendix A for an example of the RPE scale.

3.5 - Statistical Analysis.

Data are presented as mean ± SD in the text, tables and figures.

Paired sample t-tests were used within the study as was deemed appropriate to test the differences between the means of two sets of scores from the same subjects (Thomas and Nelson, 1996).

When analyses between three variables were required a one way ANOVA was utilised (Norman and Streiner, 1997). In order to avoid a Type I error, post hoc analysis using the Tukey technique was utilised (Thomas and Nelson, 1997).

It was deemed appropriate to ascertain the power of the statistical analysis. Power is directly related to sample size and is important to calculate when using small sample numbers (Norman and Streiner (1997) and Vincent (1999)).

The equation used to determine power was taken from Howell (2002).

$$\text{Effect size} = \frac{M_1 - M_2}{\text{SD of the mean of the differences}}$$

$$\text{Power} = \text{Effect size} \times \sqrt{n}$$

Power should be approximately 0.8 when testing the difference between two matched samples (Welkowitz, Ewen and Cohen (2000) and Howell (2002)).

Significance was declared at the 5% probability levels ($p<0.05$). The statistical package for the social sciences (SPSS) version 10.0 was used within the present study to analyse the data.

Chapter 4.

Physiological Responses to Exercise whilst Wearing Protective Clothing and Self Contained Breathing Apparatus (PC+SCBA).

4.1 Introduction.

The capacity of the body to lose heat to the environment depends on a number of external parameters:

- The higher the ambient temperature, the less heat can be lost to the external environment. In turn, this means that heat could not be lost through the cooling processes of evaporation, conduction, convection or radiation. Therefore, as the external temperature increases, so too would the heat storage of the body increase. Hence, the amount of heat lost to the environment would decrease. (Havenith, 1999).
- Humidity will affect evaporative heat loss. The moisture content in the environment's air determines the degree of moisture that can evaporate from the skin to the environment or vice versa. Havenith (1999) states the skins moisture level is invariably higher than that of the environment, thus allowing evaporative heat loss from the skin. However, if the moisture content of the environment is greater than that of the skin, in conjunction with the temperature of the environment exceeding skin temperature, the process of evaporation is impeded (Havenith, 1999). Evaporation is the predominant process of heat loss in temperate environments, therefore, if this process becomes impaired, the stress on the body is magnified (Havenith, 1999).

Impeding the evaporative process whilst wearing PC+SCBA appears to increase the stress that is imposed upon the wearer. As such, the present study aimed to quantify the combined effects of wearing PC and SCBA together during exercise in thermoneutral conditions. Consequently, exercising in thermoneutral conditions allowed comparison with the components of the HR responses produced during the heat stress of LFTEs.

Therefore, the aims of this chapter are to quantify what contributes to the HR responses when wearing PC+SCBA and identify the cause of any additional stress.

4.2. Methods.

Refer to Methods chapter (Section 3.4.8).

4.3 Results.

4.3.1 Step Test and PC+SCBA.

During seated rest there were small but significant increases ($p<0.05$) in oxygen cost between the gym kit (GK) and weighted gym kit (WGK) trials and also between the GK and PC+SCBA (Table 4.1). There were no significant increases in the temperature readings between any of the clothing trials at rest (Table 4.1). This suggests that the increase in oxygen cost may have been as a result of metabolic costs, as opposed to increased heat storage through wearing the weighted rucksack or PC+SCBA.

Table 4.1 – Mean resting HR, oxygen cost, and skin, aural and microclimate temperature values prior to exercising over a 5 minute period when the BAIs were either dressed in gym kit (GK), gym kit with a weighted rucksack (WGK) or protective clothing and self contained breathing apparatus (PC+SCBA) . Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test. * - significant difference ($p<0.05$) between the trials when the BAIs were dressed in GK and WGK. ** - significant difference ($p<0.05$) between the trials when the BAIs were dressed in GK and PC+SCBA.

	GK	WGK	PC+SCBA
HR (beats·minute^{-1})	69.0 (± 6.4)	74.0 (± 8.9)	74.0 (± 13.0)
Oxygen Cost (ml O_2·kg^{-1}·min^{-1})	3.6 (± 0.6)	4.5 (± 0.5)*	4.3 (± 0.5)**
Skin temperature (^{0}C)	33.6 (± 0.7)	33.5 (± 1.1)	34.2 (± 1.0)
Aural temperature (^{0}C)	36.0 (± 0.3)	36.0 (± 0.3)	36.0 (± 0.5)
Micro-climate temperature (^{0}C)	33.0 (± 1.0)	32.9 (± 1.2)	32.3 (± 1.1)

All subjects completed the five stepping levels successfully. The results from Figure 4.1 show that there were significant increases ($p<0.05$) in HR between the GK and the WGK (the mean of the mean change in HR for the 5 levels was 23.9 (± 5.2) beats·minute^{-1}) and also GK and PC+SCBA (the mean of the mean change in HR for the 5 levels was 26.5 (± 5.2) beats·minute^{-1}) for all five levels. There were no significant increases in HR between the WGK and PC+SCBA trials.

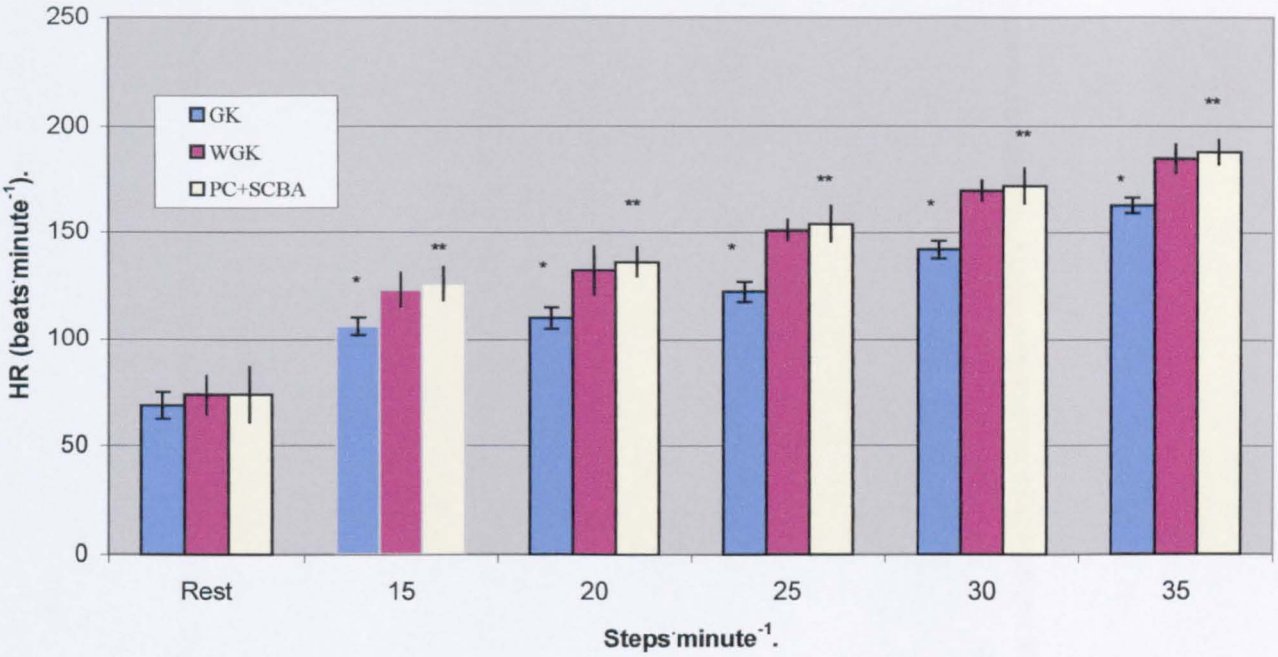


Figure 4.1 – Mean HR whilst dressed in GK (gym kit), WGK (gym kit with weighted rucksack) and PC+SCBA (protective clothing and self contained breathing apparatus) whilst at rest and when performing a Step Test (step rates of 15, 20, 25, 30 and 35 steps·minute^{-1} were equivalent to levels 1 2, 3, 4 and 5 respectively). Data is presented as the mean ± SD (n = 6). Significance was tested using a paired samples t-test. * - significant difference ($p<0.05$) between the GK and WGK trials. ** - significant difference ($p<0.05$) between the GK and PC+SCBA HR trials.

Table 4.2 – Mean skin, aural and micro-climate temperature (^{0}C) values during the Step Test when the BAIs were either dressed in gym kit (GK), gym kit with a weighted rucksack (WGK) or protective clothing and self contained breathing apparatus (PC+SCBA). Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA with a post hoc Tukey test. * - significant differences ($p<0.05$) between the trials when the BAIs were dressed in GK and WGK. ** - significant difference ($p<0.05$) between the trials when the BAIs were dressed in GK and PC+SCBA.

ST Level	Gym kit			Gym kit and Weighted Rucksack			PC+SCBA		
	Skin	Aural	Micro-climate	Skin	Aural	Micro-climate	Skin	Aural	Micro-climate
Level 1 (Low)	33.4 (±0.4)*	36.1 (±0.3)	31.8 (±1.3)	33.1 (±1.4)	36.2 (±0.3)	31.8 (±2.0)	34.4(±0.8)**	36.2 (±0.4)	32.7 (±1.2)
Level 2	33.1 (±0.4)*	36.1 (±0.3)	31.0 (±2.0)	32.6 (±1.2)	36.1 (±0.3)	31.1 (±2.5)	34.3(±0.8)**	36.2 (±0.4)	32.7 (±1.1)
Level 3	33.1 (±0.4)*	36.1 (±0.3)	31.0 (±2.1)	32.5 (±1.1)	36.0 (±0.3)	30.8 (±2.7)	34.4(±0.8)**	36.3 (±0.9)	32.6 (±1.1)
Level 4	33.1 (±0.4)*	36.2 (±0.3)	31.1 (±2.2)	32.5 (±1.0)	36.1 (±0.3)	30.8 (±2.8)	35.1(±0.8)**	36.4 (±0.4)	32.7 (±1.0)
Level 5 (High)	33.6 (±0.3)*	36.4 (±0.3)	31.0 (±2.3)	33.2 (±1.4)	36.4 (±0.3)	30.1 (±2.9)	36.7(±1.2)**	36.7 (±0.4)	33.0 (±0.9)

Mean aural and micro-climate temperature results showed no significant differences between any of the clothing trials at any of the stepping levels (Table 4.2).

The mean skin temperature responses were significantly increased ($p<0.05$) for all levels of stepping when WGK was compared to PC+SCBA (Table 4.2), as were responses when the PC+SCBA was compared to GK. When the mean skin temperature responses were compared between the GK and WGK trials, there were no significant differences at levels 1 and 2. However, when subjects stepped dressed in WGK, the mean skin temperature responses were significantly increased ($p<0.05$) when compared to stepping in GK at levels 3, 4 and 5 (Table 4.2).

The mean Rate of Perceived Exertion (RPE) responses between GK and WGK showed no significant differences at levels 1 and 2. However, the WGK responses were significantly increased ($p<0.05$) when compared to the GK responses at the end of levels 3, 4 and 5 (Figure 4.2). Mean GK and PC+SCBA RPE responses were not significantly different at the end of level 1, but did produce significant increases ($p<0.05$) in perceived exertion whilst wearing PC+SCBA at the end of levels 2, 3, 4 and 5 (Figure 4.2). There were no significant differences between the RPE responses for WGK and PC+SCBA for levels 1, 2 and 3 but there were significant increases ($p<0.05$) in the RPE responses when subjects wore PC+SCBA at the end of levels 4 and 5 (Figure 4.2).

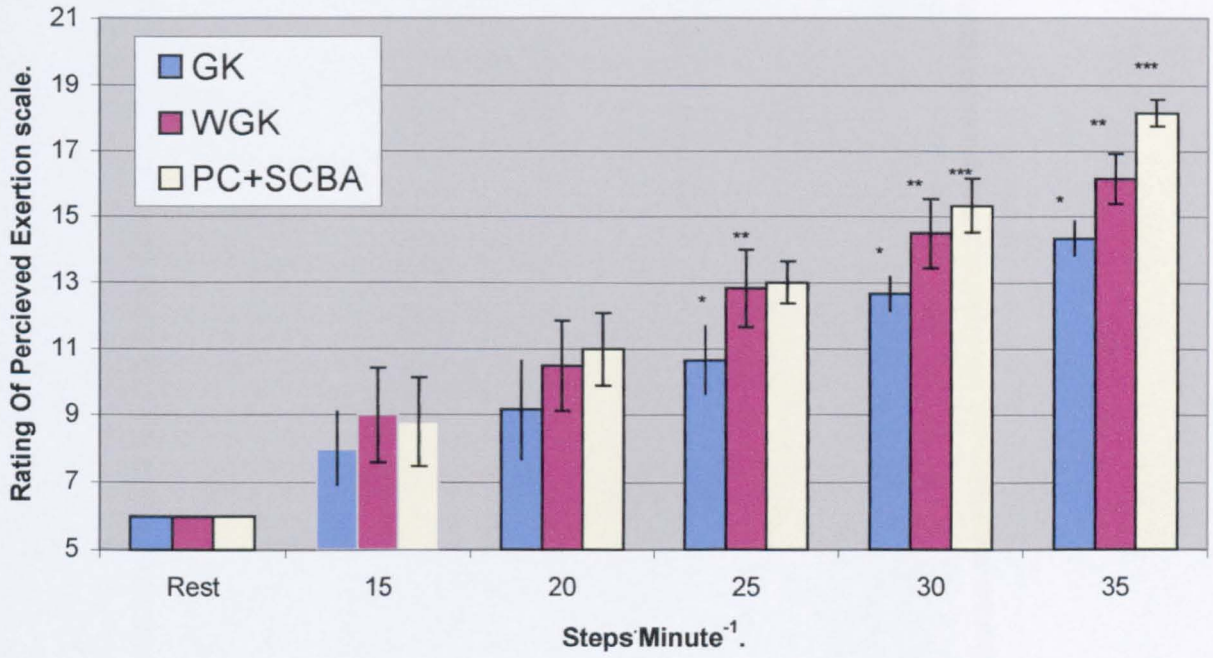


Figure 4.2 – Mean Rate of Perceived Exertion responses whilst dressed in GK (gym kit), WGK (gym kit with weighted rucksack) and PC+SCBA (protective clothing and self contained breathing apparatus) whilst at rest and when performing a Step Test (step rates of 15, 20, 25, 30 and 35 steps·minute^{-1} were equivalent to levels 1 2, 3, 4 and 5 respectively). Data is presented as the mean ± SD (n = 6). Significance was tested using a paired samples t-test with a post hoc Bonferroni. * - significant differences ($p<0.05$) between the GK and WGK. ** - significant differences ($p<0.05$) between the GK and PC+SCBA. * - significant differences ($p<0.05$) between the WGK and PC+SCBA HR trials.**

Oxygen cost (ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) whilst wearing PC+SCBA was significantly increased ($p<0.05$) when each corresponding level was compared to wearing GK alone (Figure 4.3). The oxygen cost of stepping whilst dressed in gym kit with a weighted rucksack (WGK), when compared to stepping dressed in GK, produced significant increases ($p<0.05$) between levels 1 through to 3. However, oxygen cost between GK and WGK for levels 4 and 5 demonstrate a greater increase in oxygen cost, but were not significant. When the subjects dressed in PC+SCBA were compared to the WGK trial, there were no significant differences in the mean oxygen cost during levels 1, 2, 3 and 4. However, the oxygen cost elicited during level 5 when subjects wore PC+SCBA was significantly increased ($p<0.05$) when compared to the oxygen cost when dressed in WGK.

In order to ascertain why there were no significant differences in oxygen cost between GK and WGK at levels 4 and 5 of the Step Test, the present study investigated the effect of workload on the anaerobic contribution to energy production. Increased anaerobic contribution was represented by a significant increase in RER (Figure 4.4). Figure 4.4 shows there to be significant increases ($p<0.01$) in RER at level 4 during the WGK trial (1.1 (± 0.1)) compared to the GK trials (0.9 (± 0.0)). There were also significant increases ($p<0.01$) in the RER results at level 5 during the WGK trial (1.2 (± 0.1)) compared to the GK trial (1.0 (± 0.1)). Figure 4.5 shows the ventilatory threshold obtained from the mean VE and VO_2 values at rest and from each level of the Step Test whilst dressed in gym kit with a weighted rucksack.

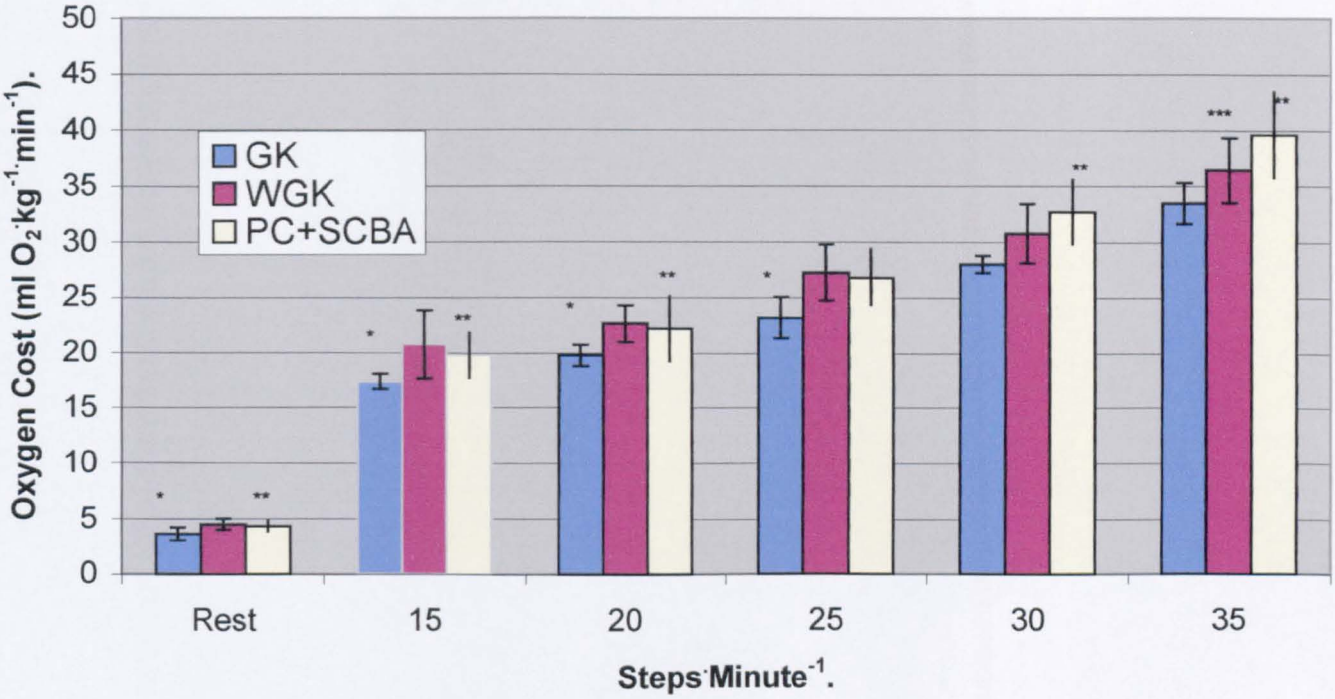


Figure 4.3 – Mean oxygen cost (ml O_2 kg^{-1} min^{-1}) responses whilst dressed in GK (gym kit), WGK (gym kit with weighted rucksack) and PC+SCBA (protective clothing and self contained breathing apparatus) whilst at rest and when performing a Step Test (step rates of 15, 20, 25, 30 and 35 steps minute^{-1} were equivalent to levels 1 2, 3, 4 and 5 respectively). Data is presented as the mean ± SD (n = 6). Significance was tested using a paired samples t-test with a post hoc Bonferroni. * - significant differences ($p<0.05$) between the GK and WGK. ** - significant differences ($p<0.05$) between the GK and PC+SCBA. * - significant differences between the WGK and PC+SCBA.**

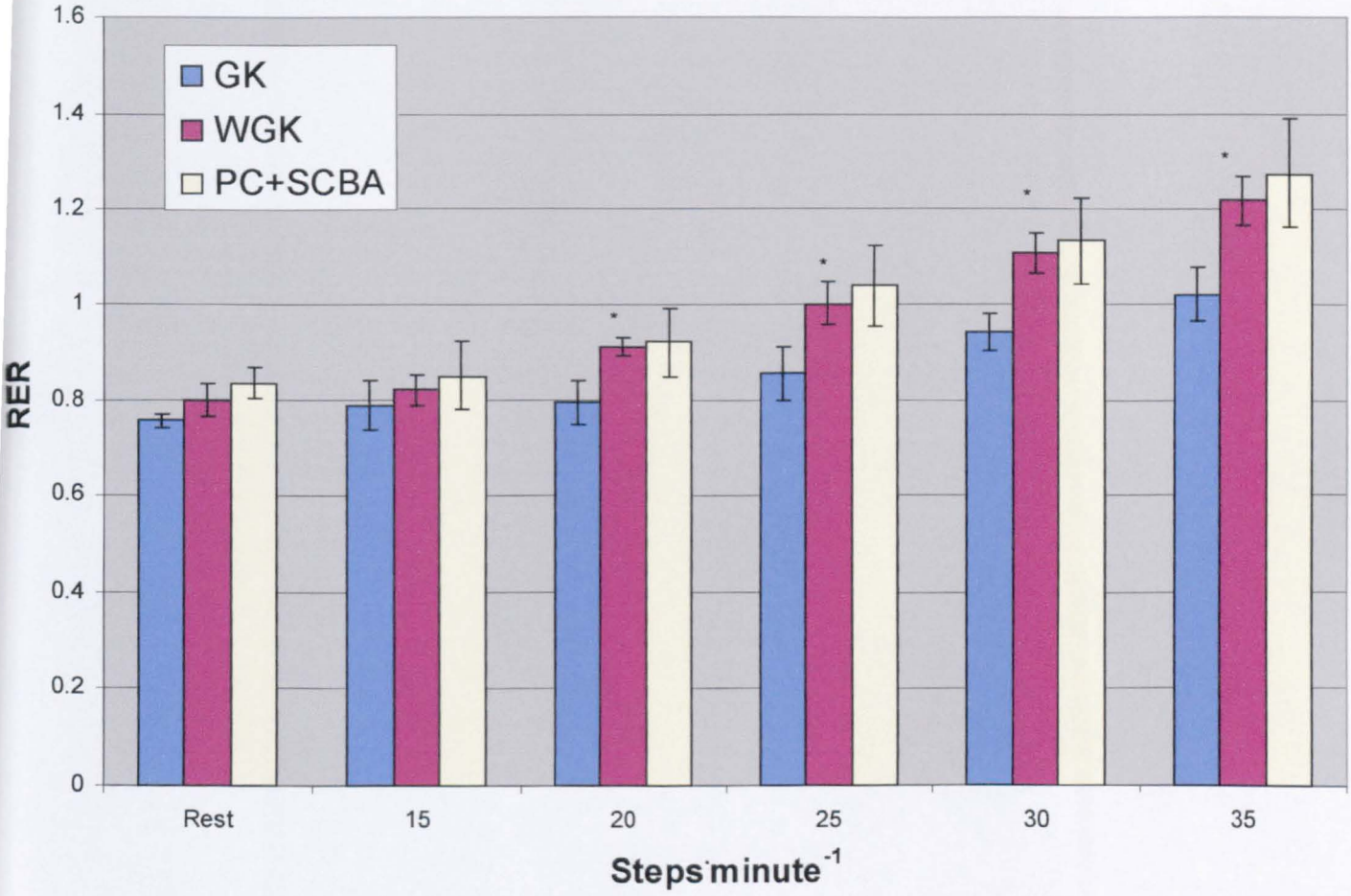


Figure 4.4 – Mean Respiratory Exchange Ratio responses whilst dressed in GK (gym kit), WGK (gym kit with weighted rucksack) and PC+SCBA (protective clothing and self contained breathing apparatus) whilst at rest and when performing a Step Test (step rates of 15, 20, 25, 30 and 35 steps·minute^{-1} were equivalent to levels 1 2, 3, 4 and 5 respectively). Data is presented as the mean ± SD (n = 6). Significance was tested using a paired samples t-test with Post Hoc Bonferroni. * - significant differences ($p<0.05$) between the GK and WGK.

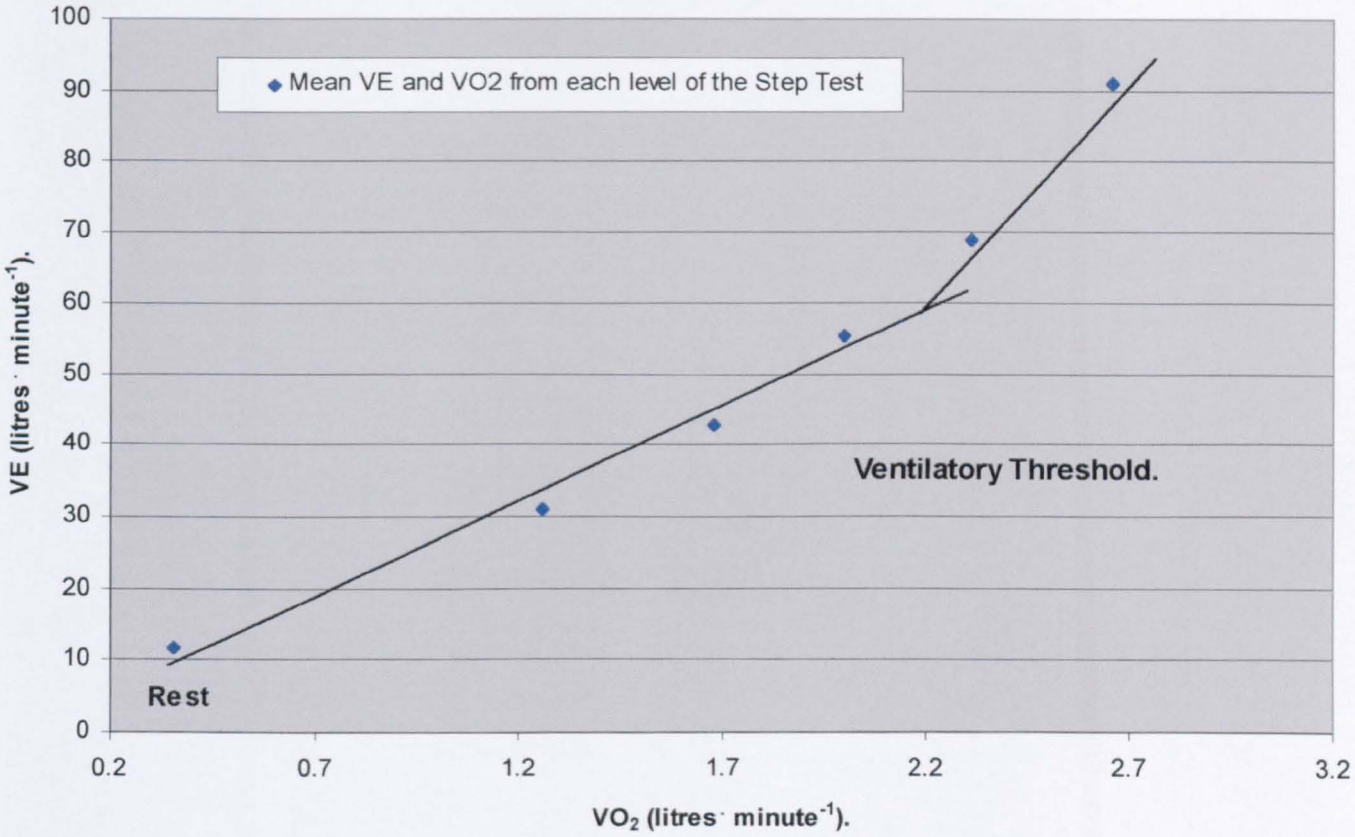


Figure 4.5 – Mean VE and VO_2 values at rest and from each level of the Step Test whilst dressed in gym kit with a weighted rucksack (n = 6).

4.3.2 Step Test and LFTEs.

The following section discusses predicting oxygen cost from the drop in the SCBA cylinder pressure) during LFTEs (refer to 3.4.9 in the methods). The extreme temperature (ceiling temperature often in excess of 150^0C) BAIs encountered during LFTEs did not allow the direct measurement of oxygen consumption using the Cosmed K4[2]. Louhevaara et al (1984) and Smolander *et al* (1985) used indirect methods to ascertain oxygen cost during simulated fire fighting tasks using a ventilation equivalent (VE/VO_2) value of 22.5 litres. The ventilation equivalent was developed from the work of Cotes (1975) and Astrand and Rodahl (1986). The VE/VO_2 is the ratio between the volume of air ventilated and the amount of oxygen consumed by the tissues and is a measure of breathing economy (Wilmore and Costill, 1999).

In addition, this section also attempts to quantify the HR responses and oxygen cost elicited by BAIs during LFTEs (HR and an indirect measurement of oxygen cost (using the equation given in section 3.4.6 of the methods) were monitored). The HR and indirect oxygen cost measurements were used in conjunction with the HR and oxygen cost (measured directly using the Cosmed K4[2]) obtained during the Step Test.

4.3.2i Prediction of oxygen cost (Prediction equation obtained from drop in cylinder pressure).

Refer to method section 3.4.9.

4.3.2ii Developing the equation.

The indirect equation used widely (Smolander *et al*, 1985, and Louhevaara *et al*, 1994) was developed from laboratory based studies. During these studies the subject's mode of exercise was cycle ergometry and they were dressed in GK and not PC+SCBA (Astrand and Rodahl, 1986). As such, this section of the study investigated whether the additional weight from wearing the PC+SCBA during exercise in the 'field environment', affects the ventilation equivalent (VE/VO_2) value rendering the equation unsuitable to use outside of laboratory conditions. Therefore, section 4.3.2 investigated whether the constant used in the equation (22.5 litres·minute^{-1}) was different from actual readings

obtained during the Step Test when subjects were dressed in PC+SCBA. The study also investigated whether there were differences between the direct measurements of oxygen cost obtained from the Cosmed K4[2] and the predicted oxygen cost obtained using the equation. The cylinder pressure readings were observed at the beginning and end of each level during the Step Test during the PC+SCBA trial when subjects were under air (breathing air from the cylinder).

4.3.2iii. Ventilation Equivalent (VE/VO_2).

During the Step Test the VE/VO_2 values were obtained from an on-line gas analyser (Cosmed K4[2] (Italy)). The VE/VO_2 values (23.9 (± 1.8) litres·minute^{-1} and (23.1 (± 2.0) litres·minute^{-1} for levels 1 and 2 respectively) were not significantly different when compared to the constant of 22.5 litres·minute^{-1} (used during other studies such as Louhevaara *et al*, 1984). However, VE/VO_2 values for all other levels (3 through to 5) were significantly different ($p<0.05$) from the 22.5 litres·minute^{-1}(refer to Figure 3.3 in the Methods).

4.3.2iv Prediction of oxygen cost.

Figure 4.6 shows the mean oxygen cost of each level of the Step Test. The actual oxygen cost was obtained using the Cosmed K4[2]. The Cosmed K4[2] has been previously reported to be a valid method of obtaining oxygen consumption (Hausswirth, Bigard and Le Chevalier, 1997).

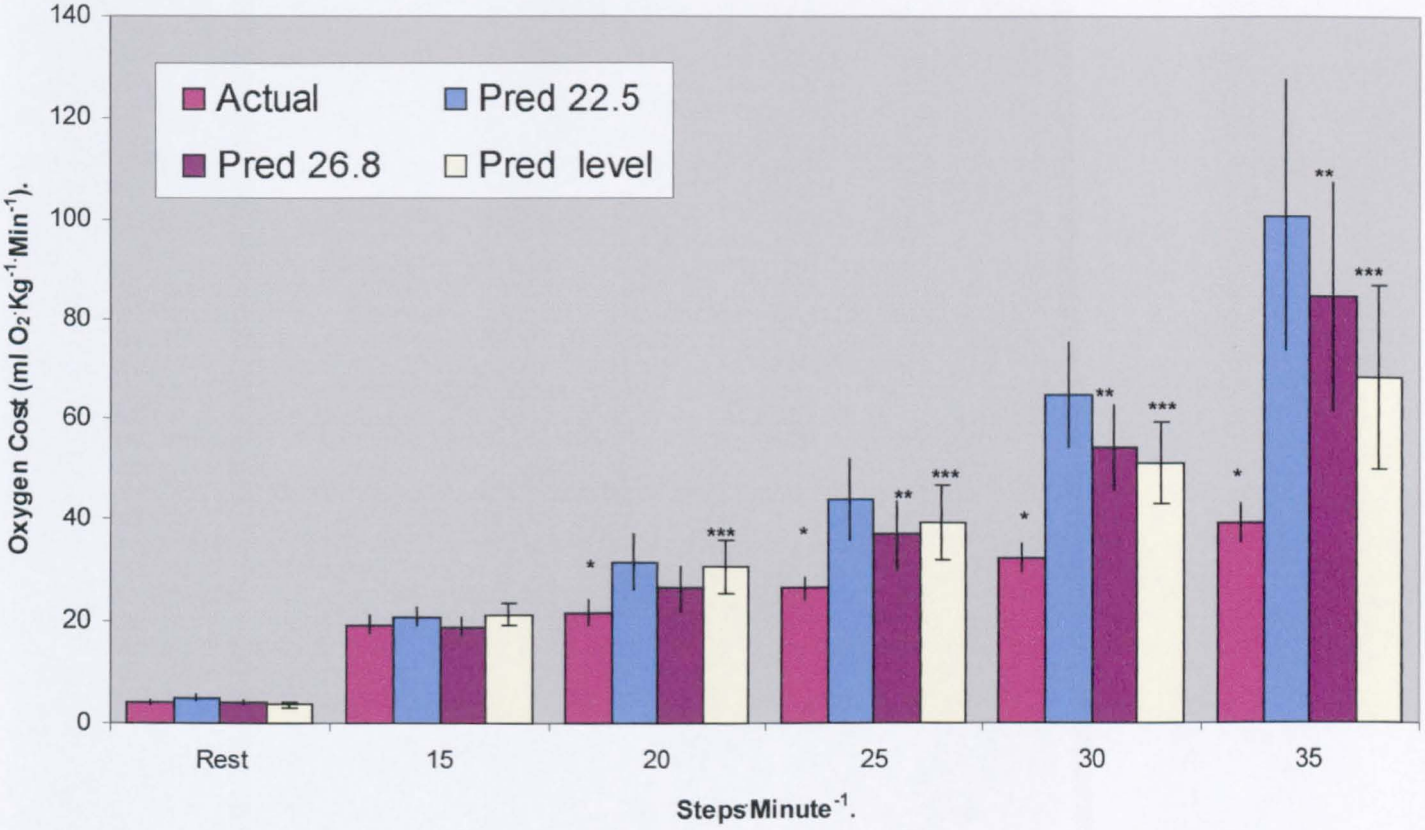


Figure 4.6 – Mean values for the actual oxygen cost and predicted oxygen cost when performing a Step Test (step rates of 15, 20, 25, 30 and 35 steps·minute^{-1} were equivalent to levels 1 2, 3, 4 and 5 respectively). Oxygen cost was obtained using the Cosmed K4^2 (Actual) and predicted using; the 22.5 litres·minute^{-1} constant obtained from Astrand and Rodahl (1986) (Pred 22.5); the 26.8 litres·minute^{-1} obtained from the mean VE/VO_2 from all 5 levels of the Step Test (Pred 26.8); and the mean VE/VO_2 of each Step Test level was used to obtain the corresponding levels oxygen cost (Pred level). Data is presented as the mean ± SD (n = 6). Significance was tested using a paired samples t-test with a post hoc Bonferroni. * - significant differences ($p<0.05$) between the Actual oxygen cost and Pred 22.5 oxygen cost. ** - significant differences ($p<0.05$) between the Actual oxygen cost and Pred 26.8 oxygen cost. * - significant differences ($p<0.05$) between the Actual oxygen cost and Pred level oxygen cost.**

The oxygen cost (ml $O_2 kg^{-1} min^{-1}$) observed in Figure 4.6 was predicted using three different constants in conjunction with the equation (section 3.4.9 in the Method chapter) specified by Louhevaara *et al* (1984). Oxygen cost (ml $O_2 kg^{-1} min^{-1}$) was predicted using the VE/VO_2 of 22.5 litres minute^{-1} in conjunction with the equation, which was consistent with Louhevaara *et al* (1984). Also, the mean VE/VO_2 of each level was used with the equation to predict the oxygen cost of the corresponding Step Test level. For example, the mean VE/VO_2 for level 1 (23.8 litres minute^{-1}) was used as the constant (instead of the 22.5 litres minute^{-1}) to obtain the oxygen cost for level 1, and the mean VE/VO_2 of level 2 (23.1 litres minute^{-1}) was used to obtain the oxygen cost for level 2 and so on for all of the other levels. Also the mean VE/VO_2 of all the 5 levels (26 litres minute^{-1}) was also used as a constant within the equation to ascertain the oxygen cost of each level. Figure 4.6 also shows the actual mean oxygen cost obtained from the readings of the Cosmed K4^2.

The results showed there were no significant differences between the actual mean oxygen cost (obtained from the Cosmed K4^2) and the three methods of obtaining predicted mean oxygen cost when they were compared at level 1 of the Step Test. There were also no significant differences in the actual mean oxygen cost (obtained from the Cosmed K4^2) and the predicted (using the mean of all 5 levels VE/VO_2 – 26.8 litres minute^{-1}) mean oxygen cost at level 2 of the Step Test.

The actual mean oxygen cost (obtained from the Cosmed K4^2) was significantly lower ($p<0.05$) when compared to the original constant (22.5 litres minute^{-1}) at levels 2 through to 5. The actual mean oxygen cost was significantly lower ($p<0.05$) compared to the predicted (using the mean of all 5 levels VE/VO_2 – 26.8 litres minute^{-1}) oxygen cost for levels 3 through to 5. There were also significant increases ($p<0.05$) in predicted oxygen cost (constant was obtained from using the mean VE/VO_2 from the corresponding Step Test level) for levels 3 through to 5 when compared to the actual mean oxygen cost (obtained from the Cosmed K4^2). This shows that for levels 3, 4 and 5 of the Step Test the mean oxygen cost obtained from the Cosmed K4^2 was lower than the predicted methods of obtaining oxygen cost.

Subjects reported that they had reached near maximal effort and would have found it difficult to continue exercising after the end of level 5 of the Step Test. This was supported by the RPE readings obtained at the termination of the Step Test (18 (± 1.0)).

4.4 Discussion.

The chapter attempts to identify whether there is additional stress from wearing PC+SCBA, identify what causes the additional stress and quantify what contributes to the HR responses whilst wearing PC+SCBA.

4.4.1 Step Test and PC+SCBA

There were small but significant differences in oxygen cost at rest between the GK and WGK trials and GK and PC+SCBA trials (Figure 4.3). This suggests that the increase in weight from wearing the weighted rucksack created an increase in metabolic cost and therefore an increase in oxygen cost.

There were no significant increases when at rest in HR, skin, aural or micro-climate temperature readings between any of the clothing trials. This also suggests that the increase in oxygen cost at rest, when wearing the weighted rucksack or PC+SCBA, may have been as a result of metabolic costs from the weight of the PC+SCBA. This was opposed to an increase in heat storage, as would have been evidenced from significant increases in the temperature readings.

However, when the subjects wore PC+SCBA there were patterns showing increases in HR and micro-climate, when compared to wearing the WGK during levels 4 and 5 of the Step Test. The patterns suggested that wearing PC+SCBA during thermoneutral conditions (room temperature during the GK trial 21.8 (± 0.8) ^{0}C, WGK trial 22.5 (± 1.0) ^{0}C and PC+SCBA trial 21.0 (1.5) ^{0}C ($p>0.05$ between the trials)) may create an increase in HR, partly due to an increase in heat storage (refer to Figure 4.1 and Table 4.2). The weight of the PC+SCBA and the rucksack were the same, suggesting it was the

heat encapsulating properties of the PC+SCBA that consistently caused the increase in HR.

The HR responses whilst dressed in PC+SCBA during level 3 of the Step Test (154 (± 8.5) beats·minute^{-1}) were consistent to those produced by other research during fire exercises such as smoke diving (searching for dummies in a smoke filled room). Smolander, Louhevaara and Korhonen, (1985) observed heart rates of 155 beats·minute^{-1} whilst carrying a 70 kg dummy for 10 minutes, Lusa, *et al*, (1993) observed HR responses of 150.0 (± 13.0) beats·minute^{-1} when subjects carried out smoke diving, whilst Baker *et al*, (2000) observed HRs of 139.0 (± 2.8 SEM) beats·minute^{-1} when subjects exercised on a treadmill at 5 km·hour^{-1}. The HR results of the present study were also consistent with White *et al*, (1989), who also observed an increase in HR during exercise dressed in PC+SCBA when compared to wearing SCBA with air permeable overalls (APO+SCBA). APO+SCBA allowed air to penetrate the protective garments allowing evaporation to occur, whereas PC+SCBA did not. The study of White *et al* (1989) demonstrated that HR responses at lower intensities (30% of $VO_{2\ max}$) were lower when exercising in APO+SCBA compared to PC+SCBA. Although significance was not reported, the present study showed at lower intensities (50% $VO_{2\ max}$, during level 1 of the Step test) there were lower HR responses (1.8 (± 5.5) %) during the WGK trials compared to the PC+SCBA trials.

As a result of the increased workload incurred by the increase in weight of the different ensembles (increase in 6 kg when wearing PC+SCBA compared to APO+SCBA), peak HR of 136.0 (± 23.0) beats·minute^{-1} and 172.0 (± 9.0) beats·minute^{-1} for APO+SCBA and PC+SCBA respectively, were produced. However, White *et al* (1989) failed to report any p values within their results. In addition to the findings of White *et al* (1989, 1991), White and Hodous (1987) suggest that the increase in HR may also be contributed to by the heat insulating properties of the PC. These findings would appear to be consistent with the present study that showed a 1.8 (± 5.5) % increase in HR during the PC+SCBA trials compared to the WGK trials.

The suggestion that an increase in heat storage is consistent with an increase in HR is further supported by the aural temperature readings and RPE responses (Table 4.2 and Figure 4.2 respectively). Subjects wearing WGK when compared to PC+SCBA showed no significant differences for HR, aural temperature and RPE responses. However, there were consistent patterns demonstrated to suggest small increases in HR, aural temperature and RPE responses during the PC+SCBA trial when compared to the WGK trial (Figure 4.1, Table 4.2 and Figure 4.2 respectively). These results suggest that as the intensity of the workload increases, so does the difference between the WGK and PC+SCBA aural and skin temperatures (refer to Table 4.2). The micro-climate (Table 4.2) also showed these patterns suggesting that in addition to the weight, there was a small contribution, although not significant, made by the heat storage from wearing the PC+SCBA. Muir, Bishop and Kozusko (2001) also observed increased micro-climate temperatures resulting from an increase in heat storage from wearing PC+SCBA.

Faff and Tutak (1989) and Tanaka, Brisson and Volle (1978) reported trends that show increased core temperature between PC+SCBA and light uniform (shirt and trousers) whilst exercising on a cycle ergometer during the initial 15 minutes of exercise. They observed significant increases ($p<0.05$) in core temperature following 15 minutes of exercise. The initial 15 minutes showed core temperatures to be the same for both clothing trials. Therefore, there appears to be a delay in the rise in core temperature, suggesting that the 10 minutes exercise used in the present study was not long enough in duration to observe a significant increase in core temperature (represented by aural temperature) in PC+SCBA over the other two clothing trials. These findings are in agreement with others (Baker *et al*, 2000) who observed no significant increases in core temperature until after 12 minutes of incremental exercise between gym kit and PC+SCBA.

Faff and Tutak, (1989) also observed consistent increases, although not significant, in HR between PC+SCBA and fire fighters uniform (cotton shirt and polyester trousers only) during 30 minutes of cycling at an intensity of 1.5 $W \cdot kg^{-1}$. However, because their subjects exercised on a cycle ergometer, these differences can be attributed to heat

storage from wearing the PC, as opposed to the effect of the additional weight from wearing the PC+SCBA. In addition, Faff and Tutak (1989) failed to report the difference in weight between the fire fighters' uniform and PC+SCBA trials. It can be postulated that they assumed the effect of the additional weight from the PC+SCBA was negated through sitting on the cycle ergometer.

There were significant increases ($p<0.05$) observed in skin temperature when subjects exercised in PC+SCBA compared to WGK. These results were consistent with Havenith (1999) who showed the evaporative capacity becomes compromised when wearing impermeable protective clothing. The increase in skin temperatures in the present study suggest that the evaporative capacity did, indeed, become inhibited as the metabolic heat produced from the working muscles was transported from the working muscles' capillary beds to the subcutaneous layer for heat exchange to occur (Forney and Vromen, 1985).

However, heat exchange was compromised whilst wearing PC+SCBA, thus creating an increase in skin temperature. In turn, this may have had the effect of increasing core temperature as heat was not efficiently lost from the skin surface through evaporation and instead was stored in the core. Table 4.2 shows this was reflected in the increased aural temperature as the intensity increased whilst wearing PC+SCBA over that of the WGK. In addition, the increase in skin temperature was not as evident during the GK and WGK trials, suggesting air flow over the skin allowed an exchange of heat through the evaporation system as the subjects wore t-shirts that were not encapsulating. Consistent with Taylor *et al* (2000), these results suggest that by lowering skin temperature may reduce the stress placed on BAIs when wearing PC+SCBA.

The mean oxygen cost (ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) responses when wearing PC+SCBA were significantly increased ($p<0.05$) when each level was compared to the corresponding level whilst wearing GK alone (refer to Figure 4.3). This was presumably through the increase in workload placed on the subjects from the additional weight of wearing the PC+SCBA. In addition, there may have been a small contribution in increased oxygen

cost from the increase in heat storage from wearing the PC+SCBA. This was evidenced from the increased micro-climate (Table 4.2) and skin temperature (Table 4.2) responses in comparison to wearing GK.

Mean oxygen cost was significantly increased ($p<0.05$) at levels 1, 2 and 3 when WGK was compared to GK. However, it was unclear as to why the WGK responses did not continue to elicit significant increases in oxygen cost during the final two levels of the Step Test. The temperature responses showed no significant differences between the GK and WGK trials, suggesting there was little heat storage from wearing the weighted rucksack. Aural temperatures did not increase significantly, possibly due to the short duration of the Step Test (10 minutes). However, HR responses were significantly ($p<0.05$) higher during the WGK trial than the GK trial as were the RPE responses. This showed that the WGK trial was indeed of a greater workload than the GK trial. Research carried out by Borghols *et al* (1978) and Faff and Tutak (1986) also observed no significant increases in oxygen cost whilst standing still with loads up to 30 kg. They do acknowledge that walking or climbing with PC+SCBA increased oxygen cost up to 20%. Oxygen cost during Level 1 of the Step Test (15 Steps·Minute^{-1}) was increased by 21.9 (± 2.2) % compared to the resting oxygen cost which was consistent with the findings of Faff and Tutak (1986).

Figure 4.4 shows an increase in RER suggesting that the amplified work output obtained during the WGK trial over that of the GK in levels 4 and 5 was obtained with an increase in anaerobic metabolism. This was supported by Figure 4.5, which shows after level 3 of the Step Test the relationship between the mean VE (litres·minute^{-1}) and VO_2 values (litres·minute^{-1}) demonstrates a disproportionate increase in VO_2 compared to VE. This suggests that the ventilatory threshold has been reached, thus explaining the oxygen cost for levels 4 and 5 of the Step Test (equivalent to 78.0 (± 5.2) % and 92.3 (± 5.5) % $VO_{2\,max}$ respectively) not being significantly different between the GK and WGK trials.

Therefore, the results of the present study (summarised in Table 4.3) are consistent with White, *et al*, (1989), Louhevaara *et al* (1984) and Lusa *et al*, (1993)), who have

demonstrated the effect of PC+SCBA in eliciting a significant cardiorespiratory and thermoregulatory stress to the wearer. These results suggest that the ability to dissipate heat from the PC, thus lowering the skin and micro-climate temperature and HR, may be imperative in reducing the stress placed on BAIs during LFTEs.

Table 4.3 – Summary of the physiological responses to the Step test when wearing GK, WGK and PC+SCBA.

	HR	Oxygen Cost	Skin Temperature	Aural Temperature	Micro-climate Temperature
GK and PC+SCBA	Significant ↑ between GK and PC+SCBA for all 5 levels	Significant ↑ between GK and PC+SCBA for all 5 levels	Significant ↑ between GK and PC+SCBA for all 5 levels	No significant differences between any of the trials	No significant differences between any of the trials
GK and WGK	Significant ↑ between GK and WGK for all 5 levels	Significant ↑ between GK and WGK for levels 1-3	Significant ↑ between GK and WGK for levels 3-5		
WGK and PC+SCBA		Significant ↑ between WGK and PC+SCBA for level 5 only	Significant ↑ between WGK and PC+SCBA for all 5 levels		

4.4.2 Predicting Oxygen Cost

The extreme heat encountered during LFTEs did not allow direct measurement of oxygen cost, therefore a predicted ventilatory equivalent was used. The VE/VO_2 results (Figure 3.3 in the Methods) suggest the predicted ventilation equivalent (22.5 litres·minute^{-1}), whilst exercising when wearing PC+SCBA, is appropriate to use at low levels of exercise intensity. However, when exercising at higher intensities (e.g. levels 3 through to 5 of the Step Test), the 22.5 litres·minute^{-1} appears to be significantly ($p<0.05$) lower than the directly measured VE/VO_2. This suggests a ventilation equivalent of 22.5 litres·minute^{-1} is not appropriate to use at higher intensities of exercise (equivalent to level 3 or more on the Step Test). Therefore, the present study used the ventilation equivalent of 22.5 litres·minute^{-1} during LFTEs. This was due to the BAIs exercising at low intensities of exercise, equivalent to Level 1 of the Step Test.

4.4.2i Oxygen Cost.

Morgan and Raven (1985) reported that wearing SCBA whilst 'under air' (when subjects wear the face mask and breathe using the air from the cylinder) had little or no effect on subjects' breathing at low intensities of exercise. During LFTEs the BAIs were under air. The exercise intensity was low during LFTEs (17.4 (± 2.8) ml $O_2 kg^{-1} min^{-1}$) which was equivalent to exercising at 100 watts on a cycle ergometer (Astrand and Rodahl, 1986)). It is therefore unlikely, according to Morgan and Raven (1985), to have any significant effect on breathing. However, the study by Morgan and Raven (1985) was carried out in thermoneutral conditions, whilst the LFTEs are under hot conditions. This may suggest further research is required into the perception of breathing during LFTEs. In addition, Patton, Bidwell, Murphy, Mello and Harp (1995) compared VO_2 readings whilst under air and when not under air. They observed when 'under air' their subjects' VO_2 was unaffected. This suggests that during the LFTEs, the values obtained were a reliable representation of oxygen cost, even when the BAIs were under air.

The oxygen cost results (Figure 4.6) suggest that the equation incorporating the VE/VO_2 prediction of 22.5 litres $minute^{-1}$ is applicable at lower intensities of exercise (equivalent to levels 1 and 2). However, as intensity increases (levels 3 through to 5) the prediction equation becomes less effective. A comparison between the oxygen cost obtained from direct analysis (Cosmed K4[2]) and the prediction equation using the 22.5 litres $minute^{-1}$ ventilation equivalent for level 1 of the Step Test highlighted no significant differences. This suggests that at lower intensities of exercise (level 1 elicited 19.4 (± 1.7) ml $O_2 kg^{-1} min^{-1}$) this is an acceptable method of predicting oxygen consumption. Low intensity exercise (17.4 (± 2.8) ml $O_2 kg^{-1} min^{-1}$) was carried out by the BAIs during LFTEs which was not significantly lower than the mean oxygen cost of 19.4 (± 1.7) ml $O_2 kg^{-1} min^{-1}$ ascertained during level 1 of the Step Test. Therefore, it would appear that the prediction equation, 22.5 litres $minute^{-1}$, is an acceptable method of predicting oxygen cost for BAIs during low intensity LFTEs.

4.4.3 Initial Observations of HR and Oxygen Cost Responses to LFTEs.

The HR and indirect oxygen cost measurements (using the equation from Section 3.4.9 in the Methods) were used in conjunction with the HR and oxygen cost (measured directly using the Cosmed K4[2]) obtained during the Step Test. They were used to ascertain the constituent parts of the HR responses during LFTEs.

Initial observations of typical HR and oxygen cost responses to LFTEs showed the oxygen cost of carrying out the LFTEs was low, with a typical oxygen cost being approximately 16 ml $O_2 kg^{-1} min^{-1}$ and the peak heart rates elicited reaching 150 beats $minute^{-1}$ (equivalent to 75% of the predicted maximum heart rate). When the oxygen cost responses of the BAIs during this LFTE was compared to the equivalent oxygen cost produced when carrying out the Step Test, the HR was much lower. For example, the equivalent HR response to an oxygen cost of 16.46 ml $O_2 kg^{-1} min^{-1}$ during the Step Test was 90 beats $minute^{-1}$ (Figure 4.7). However, when the peak HR of 150 beats $minute^{-1}$ was compared to the equivalent oxygen cost whilst carrying out the Step Test dressed in gym kit, represented an oxygen cost of 31 ml $O_2 kg^{-1} min^{-1}$ (65% VO_2 max.).

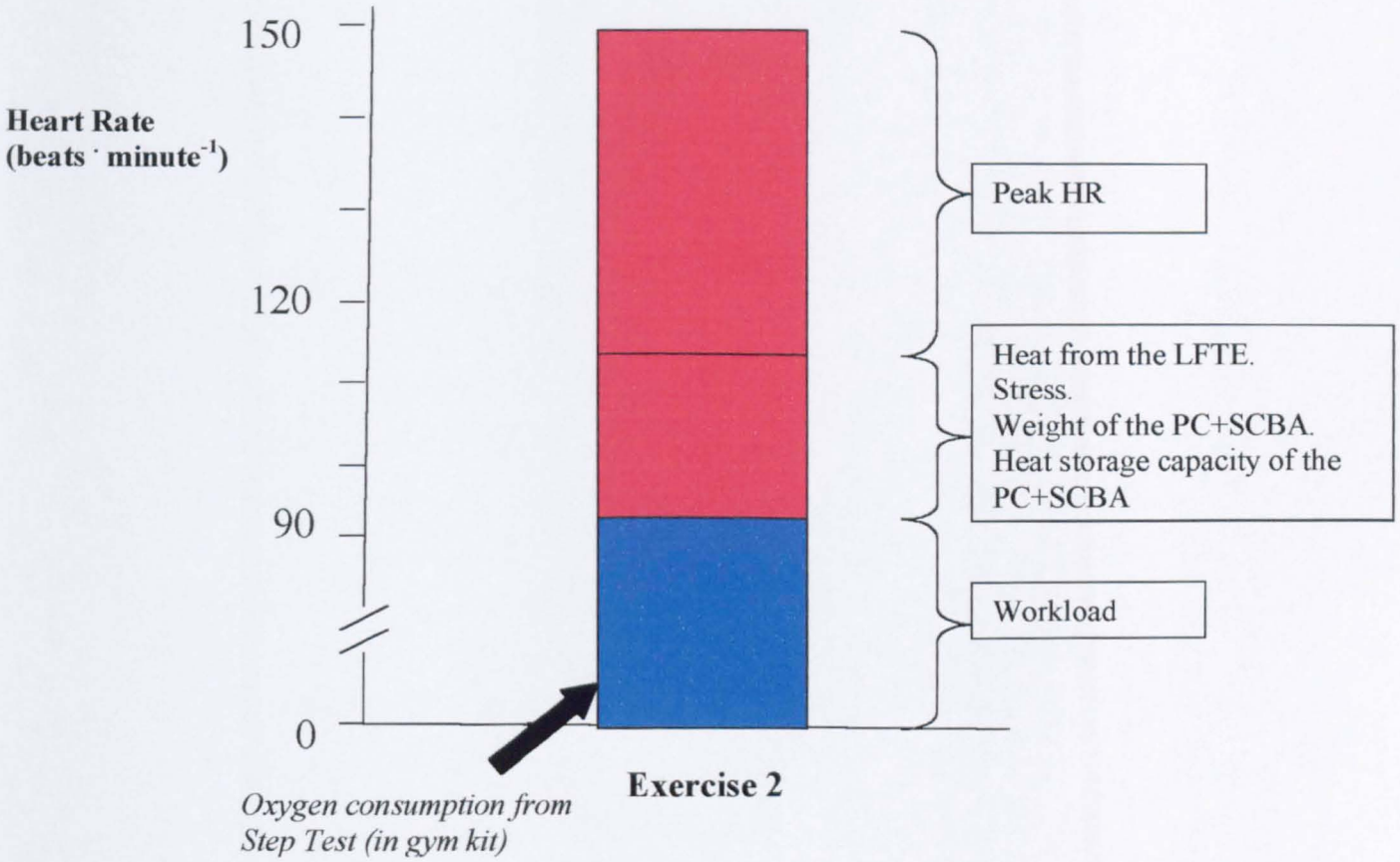


Figure 4.7 - the effect of a specified workload (16.46 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) on HR response. The HR response results from a combination of heat/stress and workload placed on Subject 1 during the second live fire training exercise. 150 beats·minute^{-1} was the peak HR response observed during the penultimate fire exercise whilst 90 beats·minute^{-1}represents the mean HR response during Level 1 of the Step Test (which has the equivalent oxygen cost of LFTEs) in gym kit.

The increase in HR and oxygen cost elicited by the BAIs during the Step Test and the FFU, suggests exposure to typical temperatures in excess of 100^0C may be the causal factor here. However, the effect of wearing PC+SCBA may have also added to this stress, which was not highlighted in Figure 4.7. For this reason, the study utilised (as reported earlier) the Step Test dressed in gym kit, gym kit with a weighted rucksack and PC+SCBA, thus fully examining the composite parts of the HR response during LFTEs.

4.4.3i Quantifying HR responses from wearing PC+SCBA during LFTEs.

Previous research has focussed on the effect of wearing PC+SCBA when exercising (Louhevaara *et al*, 1984, Louhevaara, 1984, Lusa *et al*, 1993, Serra *et al*, 1998, Louhevaara *et al*, 1995), but has not presented information quantifying the components of the increase in HR as is reported here.

Figure 4.8 shows the mean HR and oxygen cost values obtained during the Step Test study (reported earlier). The Step Test results are intrapolated using a line of best fit to intercept the mean oxygen cost and HR responses produced during LFTEs (HR: 116 (± 18.8) beats·minute^{-1} and oxygen cost 17.4 (± 2.8) ml O_2·kg^{-1}·min^{-1}). This allowed further quantification of the contributory factors to the HR responses produced during LFTEs in addition to those observed in the initial findings (Figure 4.7).

The areas that contribute to the HR responses during LFTEs are labelled 1-5 on Figure 4.8. Block 1 was obtained from where the line of best fit for the Step Test (whilst dressed in gym kit) intercepted the mean HR and oxygen cost responses from the LFTEs. This represented the HR response that the BAIs would have produced if they had carried out the LFTEs in gym kit (95 beats·minute^{-1}) in thermoneutral conditions (room temperature).

Block 2 was obtained from the point at which the line of best fit for the Step Test (whilst dressed in gym kit with a weighted rucksack loaded to the equivalent weight of the PC+SCBA) intercepted the mean HR and oxygen cost responses from the LFTEs. This represented the HR response that the BAIs would have elicited if they had carried out

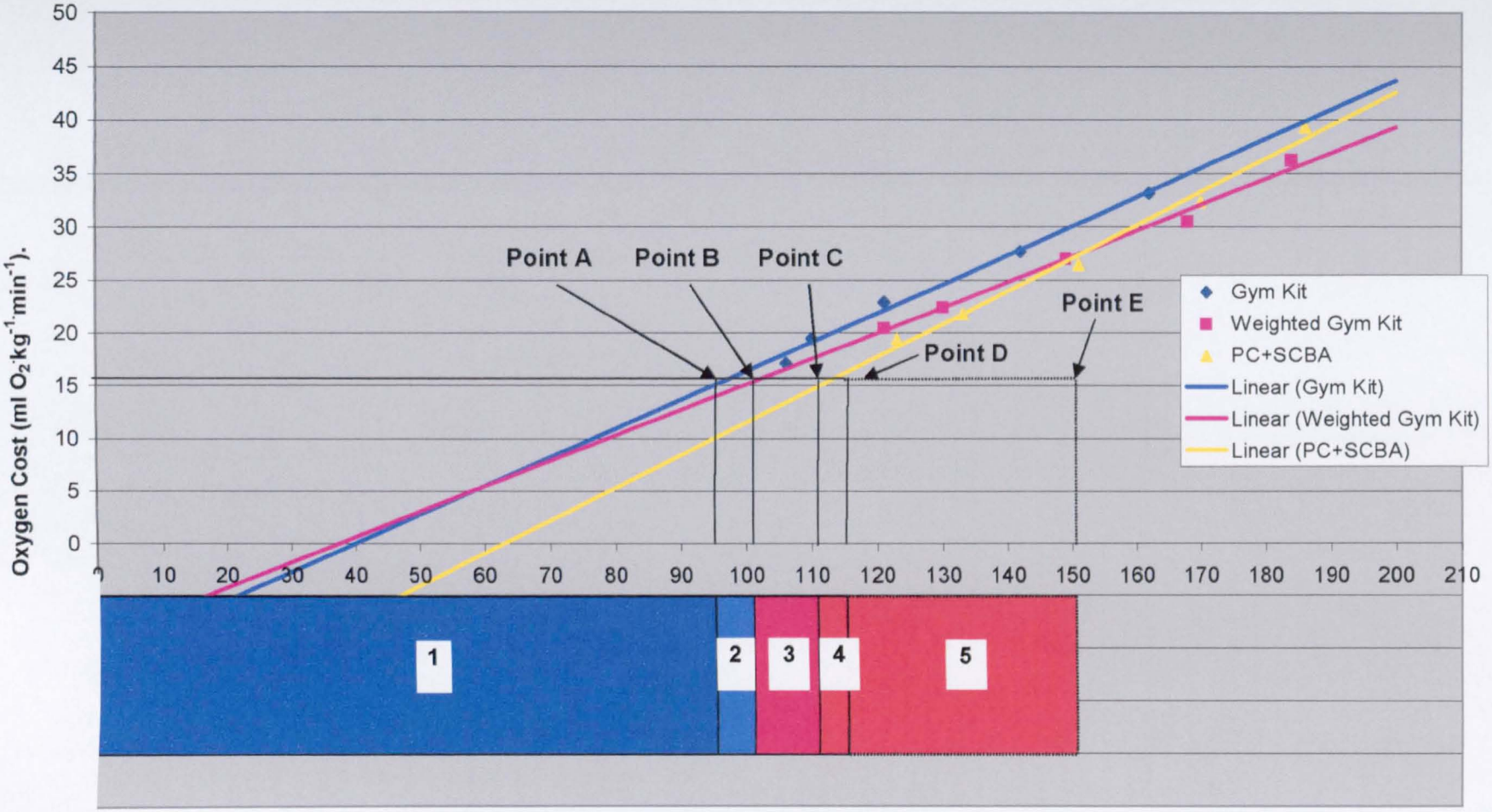
the LFTEs in gym kit and weighted rucksack in thermoneutral conditions. Wearing the weighted rucksack represented the HR contribution that would have been made from wearing the PC+SCBA (20.4 (± 1.5) kg) but without the effects of the possible heat storage from wearing the PC+SCBA (White, Vercruyssen and Hodous, 1989). The calculated HR response was 102 beats·minute^{-1}, therefore the weight (and not the heat storage) from wearing the PC+SCBA contributed a further increase in 7 beats·minute^{-1}.

Block number 3 was obtained from the point at which the line of best fit for the Step Test (whilst dressed in PC+SCBA) intercepted the mean HR and oxygen cost responses from the LFTEs. This represented the HR response that BAIs would have produced if they had carried out the LFTEs in PC+SCBA in thermoneutral conditions. Therefore, the results suggest there is a further increase of 10 beats·minute^{-1} to 112 beats·minute^{-1} as a direct result of heat storage from wearing the PC+SCBA.

The study has quantified the HR and VO_2 responses to low intensity exercise whilst dressed in PC+SCBA during thermoneutral conditions. This was to investigate the workload and heat storage components of the HR exhibited during the LFTEs. Therefore, block number 4 represents the mean HR responses to 12 LFTEs (n = 6) when the BAIs carried out low intensity exercise and were dressed in PC+SCBA. The difference between number 3 and 4 is the addition of heat stress from the LFTEs, hence the increase (4 beats·minute^{-1}) in HR is indicative of the heat stress placed on the BAIs.

Block number 5 shows the increase in HR that is representative of the mean peak HR responses to LFTEs (151 (± 20.5) beats·minute^{-1}). These results suggest that the stress produced as a result from exposure to the heat increases HR by at least 35 beats·minute^{-1}.

Figure 4.8 - Graph to observe the constituent parts of typical HR responses during LFTEs using the mean HR and oxygen cost values extrapolated from the three trials of the Step Test.



1 – HR response interpolated from the line of best fit when carrying out the step test in GK.	**2** – HR response interpolated from the line of best fit when carrying out the step test in WGK.	**3** – HR response interpolated from the line of best fit when carrying out the step test in PC+SCBA.	**4** – Mean HR response to LFTEs dressed in PC+SCBA (n=6).

5 – Mean peak HR responses from LFTEs dressed in PC+SCBA (n=6)

Point A – is the point where the interpolated line of best fit for the mean HR and oxygen cost values, when the subjects stepped whilst dressed in gym kit during the Step Test, intercepts the mean HR and oxygen cost observed during LFTEs. This represents the workload carried out by the BAIs during LFTEs with out the added stress of wearing the PC+SCBA, the heat storage through wearing the PC, or being exposed to fire.

Point B - is the point where the interpolated line of best fit for the mean HR and oxygen cost values, when the subjects stepped whilst dressed in gym kit with a weighted rucksack during the Step Test, intercepts the mean HR and oxygen cost observed during LFTEs. This represents the workload carried out by the BAIs during LFTEs with the additional stress response from weight of wearing the PC+SCBA but without the added stress of the heat storage through wearing the PC, or being exposed to fire.

Point C - is the point where the interpolated line of best fit for the mean HR and oxygen cost values, when the subjects stepped whilst dressed in PC+SCBA during the Step Test, intercepts the mean HR and oxygen cost observed during LFTEs. This represents the workload carried out by the BAIs during LFTEs with the additional stress response from weight of wearing the PC+SCBA and the added stress of the heat storage through wearing the PC, but does not include the stress of being exposed to fire.

Point D – is the mean HR and oxygen cost of carrying out 12 LFTEs (n = 6). This represents the accumulative effect on HR through the workload carried out during LFTEs, the effect of wearing PC+SCBA, the heat storage from wearing PC and the effect of heat from the fire.

Point E – is the mean peak HR responses elicited as a result of carrying out 12 LFTEs (n = 6). This represents the further physiological effect heat from the fire can have on the BAIs HR responses in addition to the factors listed in point D.

4.5 Conclusions.

The chapter has quantified the contributions to the HR responses incurred whilst wearing PC+SCBA. Figure 4.8 showed there was an increase in HR due primarily to the workload imposed on the BAIs through carrying out the LFTEs (28.4% increase over that of the resting HR). There was further stress placed on the BAIs by the additional weight from wearing the PC+SCBA (6.3% increase over that of the workload). There may also be a contribution to the increase in HR from heat storage from wearing the PC+SCBA (9.8% increase in HR over the increase caused by the weight from wearing the PC+SCBA). Further to these stressors, there was an additional increase in HR from exposure to the heat from the fire (mean HR showed a 3.6% increase and peak HR showed a 25.8% increase in HR during the LFTEs, over that of wearing PC+SCBA during thermoneutral conditions).

The results obtained from the Step Test suggest the additional stress incurred as a result of wearing PC+SCBA (compared to WGK) was highlighted by the significant increases ($p<0.05$) in skin temperature. This strongly suggests it is the increase in heat storage that

had caused this skin temperature response. This further suggests that heat dissipation from the PC may be the most significant factor in reducing heat stress during LFTEs.

In addition, it was not possible to monitor oxygen cost directly during the LFTEs and, as such, a prediction equation was required. The results showed that owing to the low intensity of exercise carried out during the LFTEs, the prediction equation was confirmed as being suitable for predicting oxygen cost during LFTEs.

Therefore, the results presented suggest reducing the stress placed on the BAIs during LFTEs would require a reduction in the effect of wearing the PC+SCBA. In turn, this may reduce the HR response and increase cooling. The heat storage from within the PC is the most realistic component that can be reduced during LFTEs. The workload intensity was very low suggesting that a further reduction would be unrealistic. Also altering the weight of the PC+SCBA was beyond the scope of the study. In addition, reducing the heat of the fire was not possible and may contravene the HSE guidelines. The guidelines suggest fire fighters are required to be exposed to realistic situations in order to prepare them for events that occur during fire incidences (HSE, 1996). The way in which these results impact on working practices is through the manner in which BAIs lower their skin, aural and micro-climate temperature, in turn lowering the HR responses to LFTEs. This will be discussed in greater detail in Chapter 6.

Chapter 5.

Physiological Responses During Live Fire Training Exercises (LFTEs) (Protocol 1).

5.1 Introduction.

Wearing PC+SCBA places an increased metabolic demand on the body even during thermoneutral conditions (conditions without any additional heat source) (Baker *et al*, 2000). This was observed in Chapter 4 whereby subjects wearing PC+SCBA produced increases in HR by 15.5 (± 3.0) % and oxygen cost by 18.4 (± 11.8) % when compared to wearing gym kit whilst exercising at 35 steps·minute^{-1} (level 5). Therefore, this protocol (Methods, sections 3.1 and 3.2) was designed to observe the physiological effects of wearing PC+SCBA during thermoneutral conditions (between 15^0-29^{0}C) without the influence of fire and the presence of students (mock training exercises). This was then compared to hot environments with the inclusion and influence of fire and students (LFTEs). The present study isolated the effect of heat and students through carrying out the mock training exercises and LFTEs identically. Therefore, the study observed the effects of heat and students on the BAIs during LFTEs.

There appears to be limited research which has observed the effects of wearing PC+SCBA at low intensities of exercise on BAIs (< 20 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) as occurred during mock and LFTEs.

5.2 Methods.

Refer to Methods (Sections 3.1 and 3.2)

5.3 Results:

Physiological responses.

Heart Rate (HR).

Mean heart rates were recorded during the LFTEs over a 34 minute period. Peak heart rates were also measured (expressed as a percent of maximum HR obtained from a maximum exercise test (refer to section 3.4.8 in the Methods)).

Table 5.1 – Mean heart rate responses between mock and LFTEs. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock training exercises and LFTEs readings.

	Mock Fire Training Exercises	Live Fire Training Exercises (LFTEs)
Mean HR (beats·minute^{-1})	84.2 (± 11.7)	116 (± 18.8)*
As a % of Maximum HR	48.1 (± 7.0)	66.6 (± 10.4)*
Peak HR (beats·minute^{-1})	102.7 (± 13.4)	149.8 (± 20.8)*
As a % of Maximum HR	58.9 (± 7.7)	85.9 (± 11.3)*

There was a significant increase ($p<0.01$) of 32.7 (± 15.5) beats·minute^{-1} which equated to a 39.8 (± 20.0) % increase in mean HR between the mock and LFTEs (Table 5.1). In addition, there were significant increases ($p<0.01$) of 47.1 (± 18.8) beats·minute^{-1} in the peak HR which was a 47.4 (± 21.9) % increase in the peak HR when LFTEs were compared to mock training exercises. The HR distribution for each individual was clearly different between mock fire and LFTEs (Figure 5.1).

Figure 5.1 shows that during LFTEs when both fire and students were present the distribution of HR was increased.

Figure 5.2 shows there were increases in the mean BAI HR responses during LFTEs when compared to trainee fire fighter students. This comparison was used to observe the different affects of stress on BAIs and students. During LFTEs students are stressed due to the inexperience of wearing PC+SCBA and being exposed to LFTEs, whereas the BAIs may be stressed due to monitoring the safety of students. The lower cardiac stress responses of BAIs (97 (± 17) beats·minute^{-1}) compared to students (121.0 (± 30.0) beats·minute^{-1}) can be clearly seen from Figure 5.2. In addition, the students were required to go further into the fire chamber than the BAIs, which meant they carried out more physical activity and were exposed to the fire to a greater degree than the BAIs.

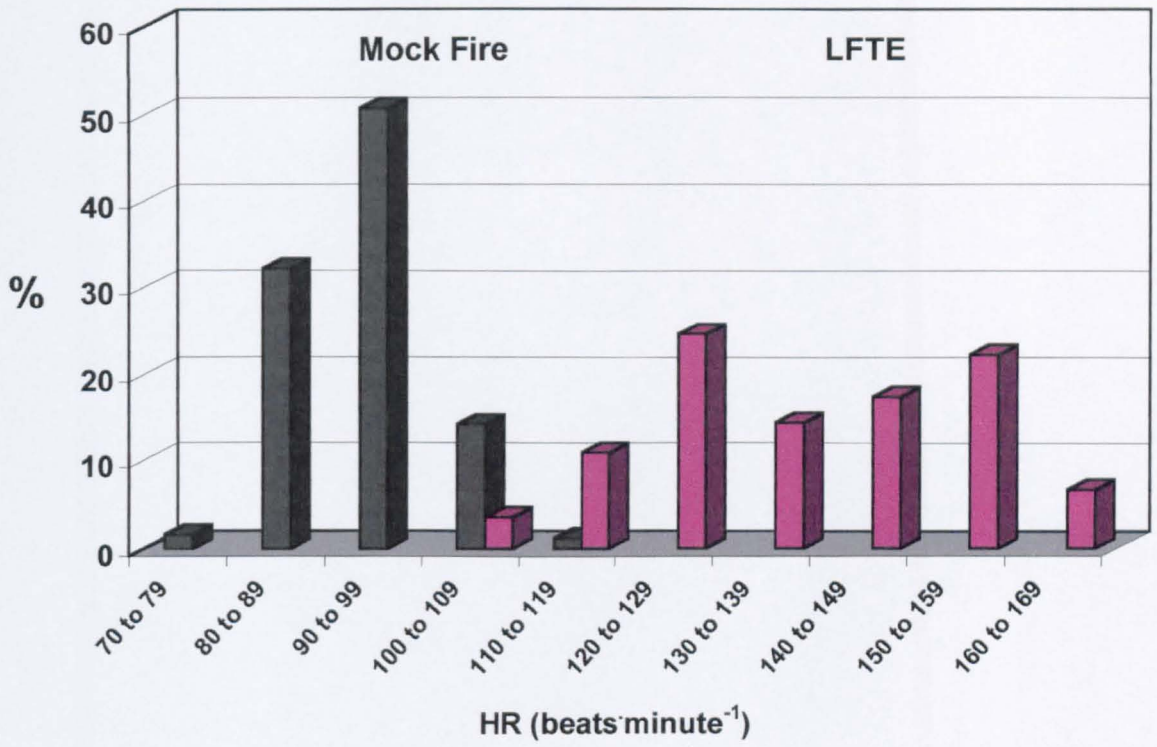


Figure 5.1 – Typical heart rate distribution differences between a mock training exercise and a LFTE. Values are the HR responses of a single BAI during a mock training exercise and a LFTE (n = 1). HR is categorised into bands that show what percent of the total HR response, elicited over the period of the mock training exercise and LFTE, falls into each of the categories.

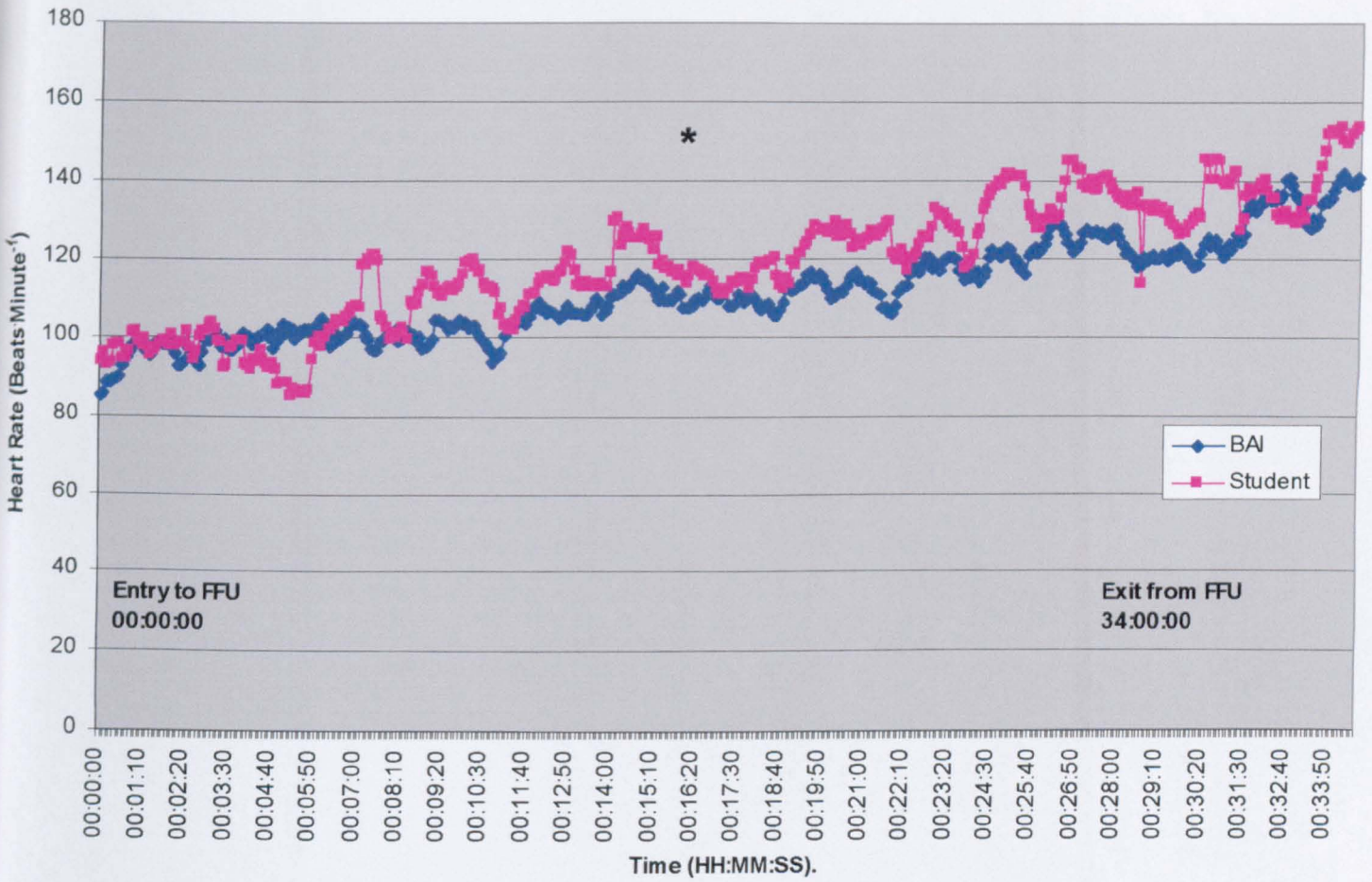


Figure 5.2 – BAIs (BAI) and fire fighter students (Student) mean HR responses to LFTEs. Data is presented as the mean (n = 6). Data points were obtained every 5 seconds during exposure to the LFTEs. For a description of the training exercises that occurred during the LFTEs refer to the methods chapter. Significance was tested using an unpaired t-test. * - significant differences ($p<0.05$) between the BAIs and students HR readings.

Figure 5.3 depicts significant increases ($p<0.01$) in the mean BAI HR responses during LFTEs (109.0 (± 15.0) beats·minute^{-1}) compared to the mock fire training exercises (82.0 (± 5.0) beats·minute^{-1}). This clearly shows the increase in HR which was predominantly caused by the inclusion of the fire and students.

Figure 5.4 shows the mean BAI HR responses to LFTEs when exposed to both heat and students (122.0 (± 19.0) beats·minute^{-1}). HR responses are also depicted when the BAIs were in the control room (and therefore not exposed to heat or students) (89.0 (± 4.0) beats·minute^{-1}). Whilst located in the control room during the LFTEs, BAIs were required to operate the computer system that controlled the fire. During this time BAIs carried out no physical activity. The BAIs in the control room were used as the control group (wore PC but not SCBA and were not exposed to the fire or students).

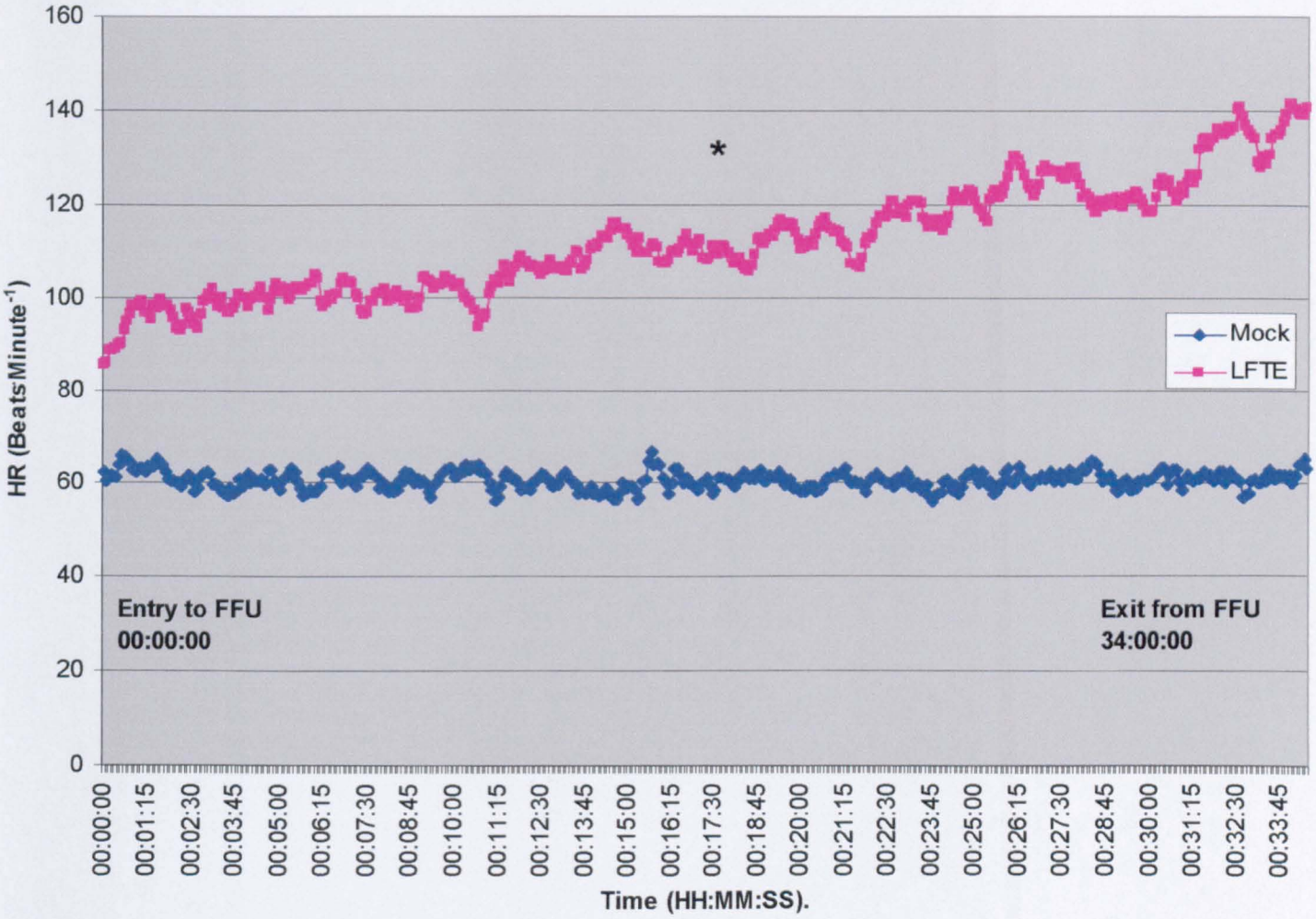


Figure 5.3 – Mean BAI HR responses to mock training exercise and LFTEs. Data is presented as the mean (n = 6). Data points were obtained every 5 seconds during exposure to the LFTEs. For a description of the training exercises that occurred during the LFTEs refer to the methods chapter. Significance was tested using a paired t-test. * - significant differences ($p<0.05$) between the BAIs mock training exercises and LFTEs HR responses.

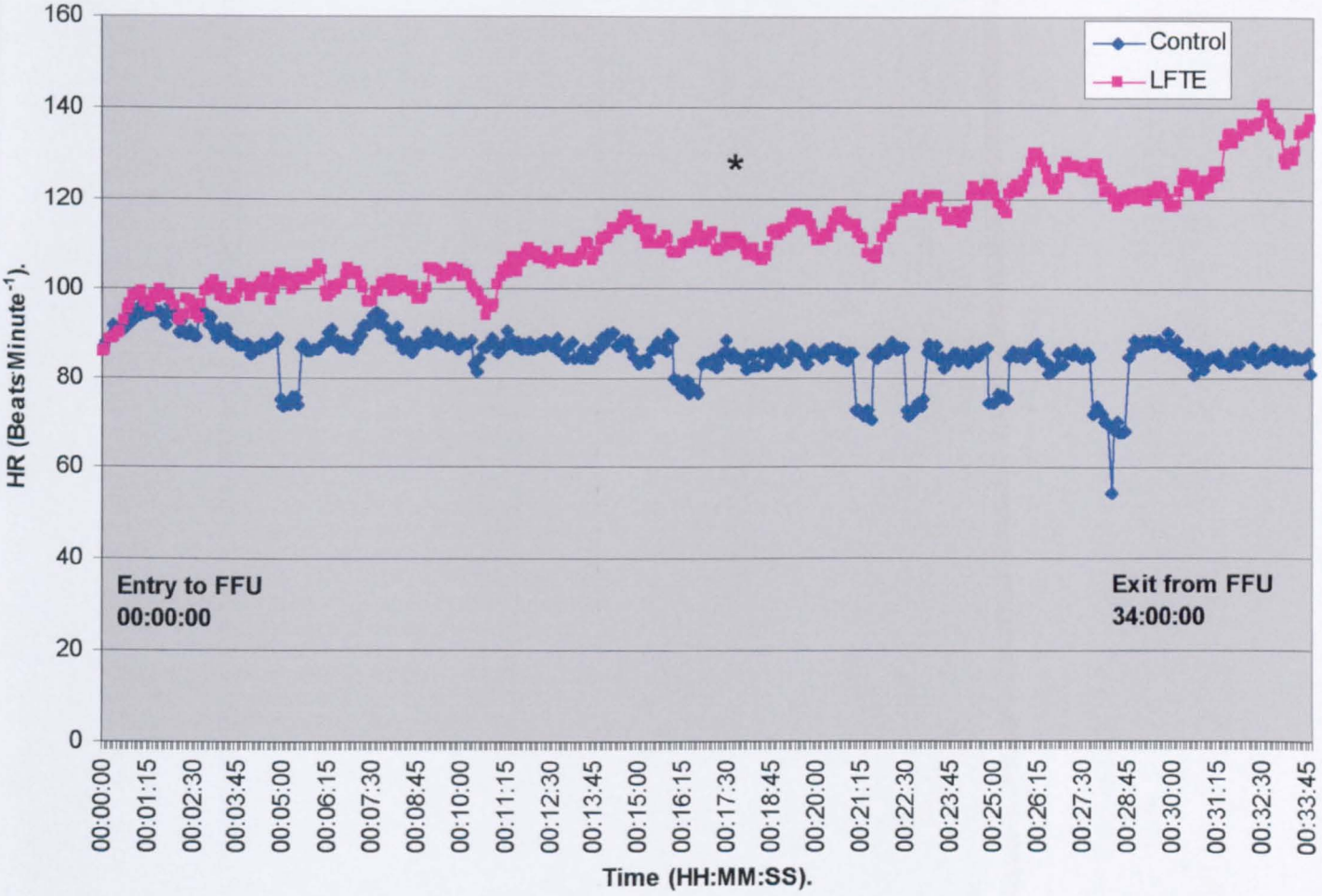


Figure 5.4 – Mean BAI HR responses whilst carrying out LFTEs inside the FFU and also when in the control room. Data is presented as the mean (n = 6). Data points were obtained every 5 seconds during exposure to the LFTEs. For a description of the training exercises that occurred during the LFTEs refer to the methods chapter. Significance was tested using a paired t-test. * - significant differences ($p<0.05$) between the BAIs HR whilst inside the FFU compared to HR responses whilst in the control room.

Oxygen Cost obtained during mock training exercises and LFTEs.

Oxygen cost was obtained (Section 3.4.9 in the Methods) from the drop in cylinder pressure (the cylinder is from where the BAIs breathe their air) that occurred during the 34 minute duration of the mock and LFTEs (Section 3.2 in the Methods).

Table 5.2 – Mean oxygen cost responses between mock and live fire training exercises (obtained from the drop in cylinder pressure). Maximum oxygen cost obtained from maximal test wearing PC+SCBA using modified Step Test (refer to Methods Chapter). Data is presented as the mean ± SD (n= 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock training exercises and LFTEs readings.

	Mock Fire Training Exercises	Live Fire Training Exercises
Mean O_2 Cost ($ml\ O_2 \cdot kg^{-1} \cdot min^{-1}$)	13.8 (± 2.3)	17.4 (± 2.8)*
% Maximum O_2 Cost	38.1 (± 9.3)	47.8 (± 10.7)*

There were significant increases ($p<0.01$) in mean oxygen cost and mean per cent of maximum oxygen cost during the LFTEs when compared to the mock training exercises. This suggests that the increase in temperature from the inclusion of fire and students within the FFU created an increase in oxygen demand on the BAIs. The BAIs carried out low levels of exercise during the LFTEs which was defined by the mean oxygen cost (17.4 (± 2.8) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$). According to Astrand and Rodahl (1986), 17.4 (± 2.8) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$ was equivalent to exercising on a cycle ergometer at 100 watts. During each of the mock training exercises and LFTEs there were a series of different low intensity exercises executed which are summarised in Table 5.3.

Table 5.3 – Definitions of different exercises executed during the training exercises.

	Definition
Demo	A demonstration was given to the students showing the different types of fire exercises they might encounter (a number 1 fire followed by a number 2 and then a number 3 fire).
Number 1	Initially the door was closed between the fire chamber and anti-chamber. When the door was opened the chair fire was the only fire that was ignited as the students entered the fire chamber. The students were required to put the fire out using the correct technique.
Number 2	Initially the door was closed between the fire chamber and the anti-chamber. When the door was opened the chair fire was ignited but there was also a flashover present when the students entered the fire chamber.
2 Delayed (2D)	Initially the door was closed between the fire chamber and anti-chamber. When the door was opened and the students entered the room, there was no fire present. As students made their way forward, the flashover was then ignited over their heads. BAIs were limited in their movement during this exercise as they had to hold the control box* to control the ignition and extinguishing of the fire.
2 Double Delayed (2DD)	Same as 2D except once the flashover had been extinguished the students began to reverse out of the fire chamber where upon another flashover was ignited. BAIs were limited in their movement during this exercise as they had to hold the control box* to control the ignition and extinguishing of the fire.
Number 3	This was where the door was open between the fire chamber and anti-chamber and remained open throughout the exercise. There was a chair fire ignited and the students were required to make their way through the anti-chamber and into the fire chamber where a flashover was ignited. BAIs were limited in their movement during this exercise as they had to hold the control box* to control the ignition and extinguishing of the fire.

* - the control box was a button that, once pressed, allowed the BAIs to initially signal to the control room that they were in control of the ignition of the fire. By pressing the button on the control box again the BAIs ignited the fire. Releasing the button would extinguish the fire.

Figure 5.5 shows that during the LFTEs there were significant increases ($p<0.05$) in oxygen cost specifically throughout exercises number 1 and 2 when compared to the mock exercises. The demo, number 2D, 2DD and 3 training exercises were not significantly different ($p>0.05$) between the mock and LFTEs.

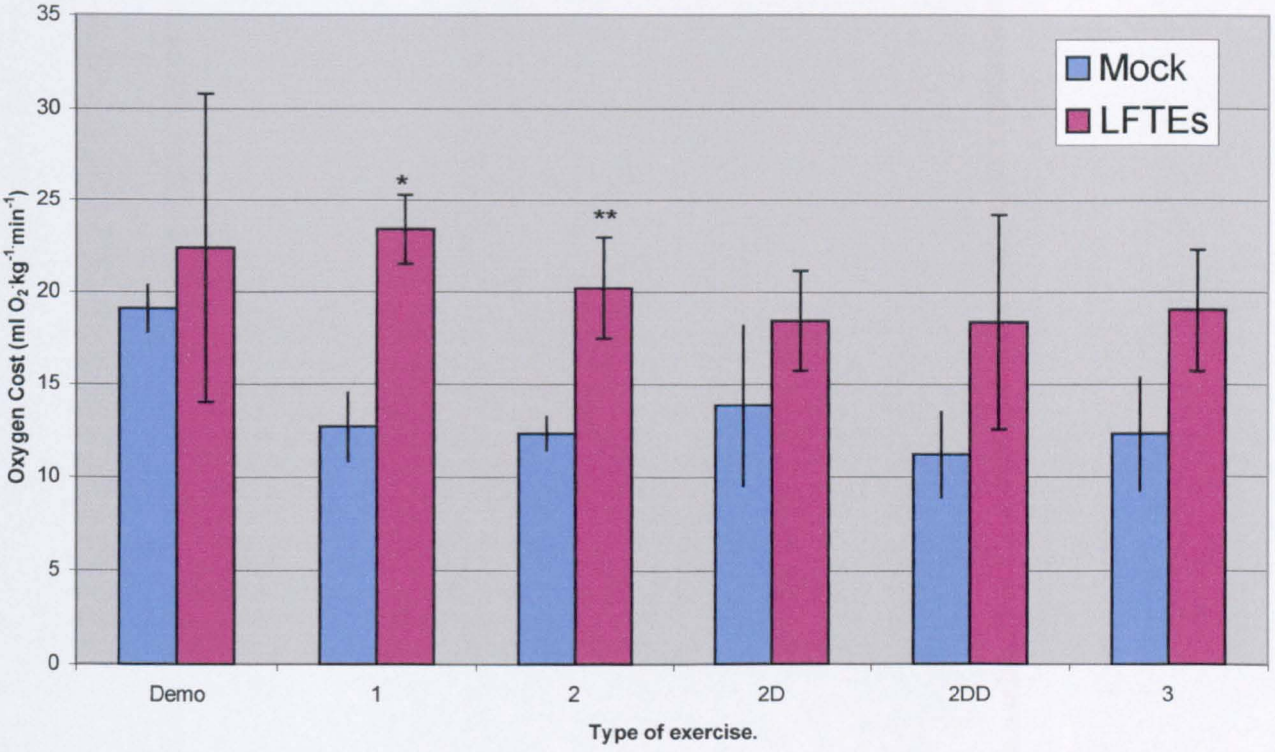


Figure 5.5 – Mean oxygen cost of the BAIs from the different types of fire exercises (refer to Table 5.3 for description of fire exercises) carried out during the mock training exercises and LFTEs. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock training exercises and LFTEs for the number 1 fire exercises. ** - significant differences ($p<0.05$) between the mock training exercises and LFTEs for the number 2 fire exercises.

Temperature during mock training exercises and LFTEs.

Mean temperature responses to the mock and LFTEs produced by the BAIs are highlighted in Figures 5.6, 5.7 and 5.8 for skin, aural and micro-climate respectively. To observe the schedule that the BAIs followed during all of the training exercises refer to Table 3.2 in the Methods.

Figure 5.8 shows the increase in temperature within the PC and its escalation throughout the LFTEs when compared to the mock training exercises. This is also true of both the skin temperature (Figure 5.6) and aural temperature (Figure 5.7). This also shows increases in temperature throughout the LFTEs when compared to the mock training exercises. It was unclear as to why there was a difference between the starting temperature for micro-climate for mock and LFTEs (Figure 5.8). However, this could be explained by differences in the temperature outside the FFU (12.4 (± 0.4) ^{0}C during the mock training exercises and 15.0 (± 0.2) ^{0}C during the LFTEs).

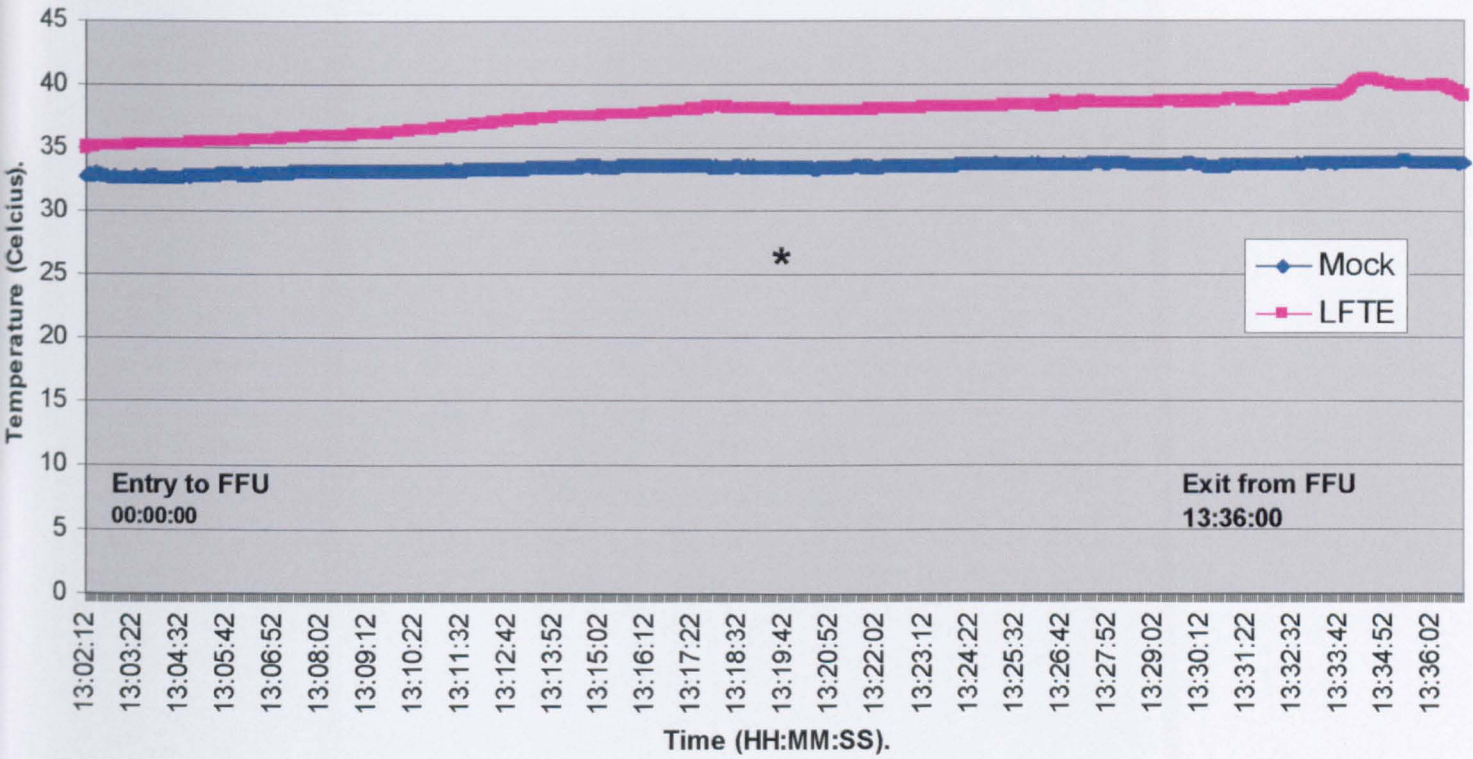


Figure 5.6 – Mean skin temperature in response to mock training exercises and LFTEs. Values were obtained every second through out the exercises (n = 6). Significance was tested using a paired samples t-test. * - significant differences ($p<0.05$) between the mock training exercises and LFTEs.

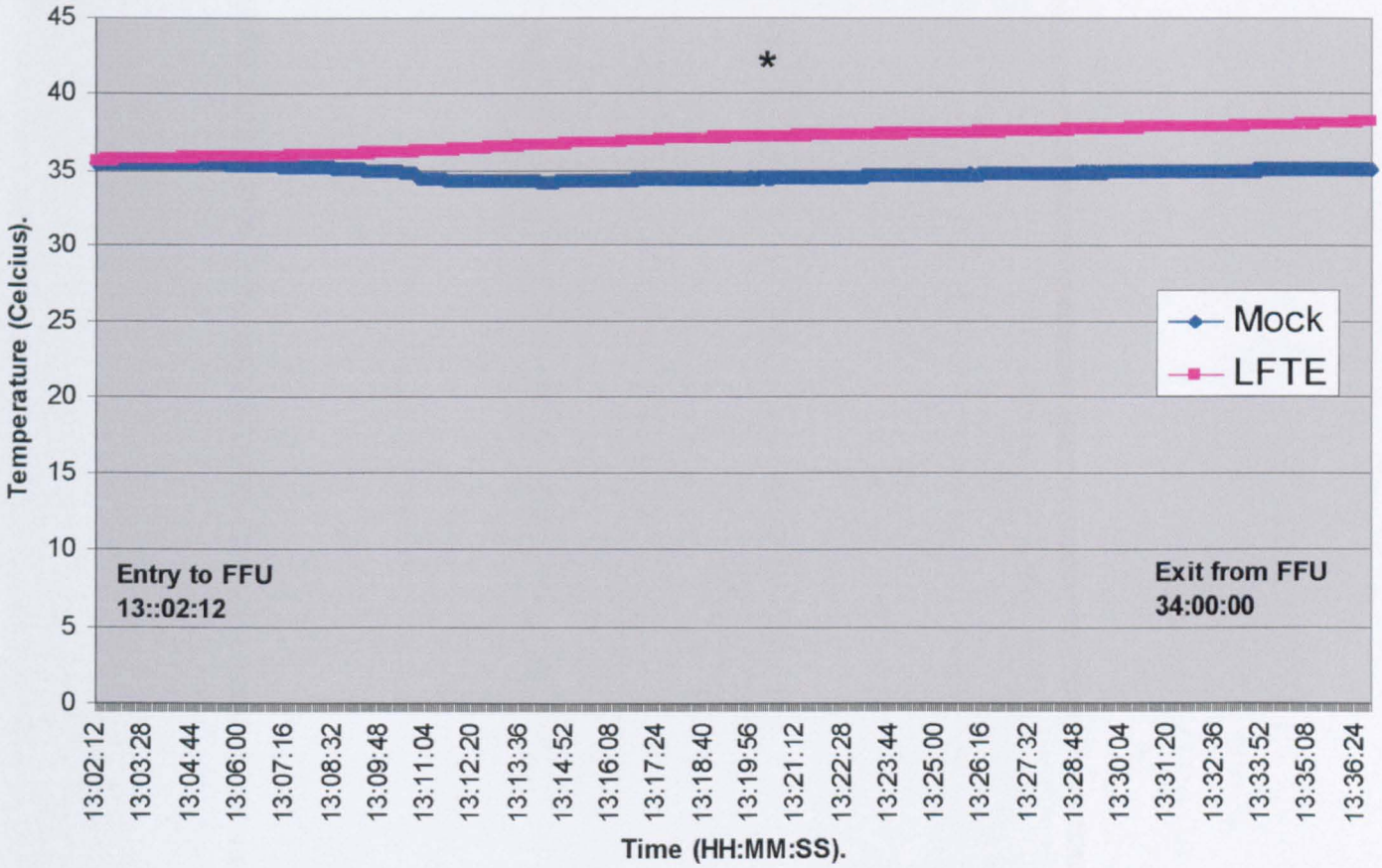


Figure 5.7 – Mean aural temperature in response to mock training exercises and LFTEs. Values were obtained every second through out the exercises (n = 6). Significance was tested using a paired samples t-test. * - significant differences ($p<0.05$) between the mock training exercises and LFTEs.

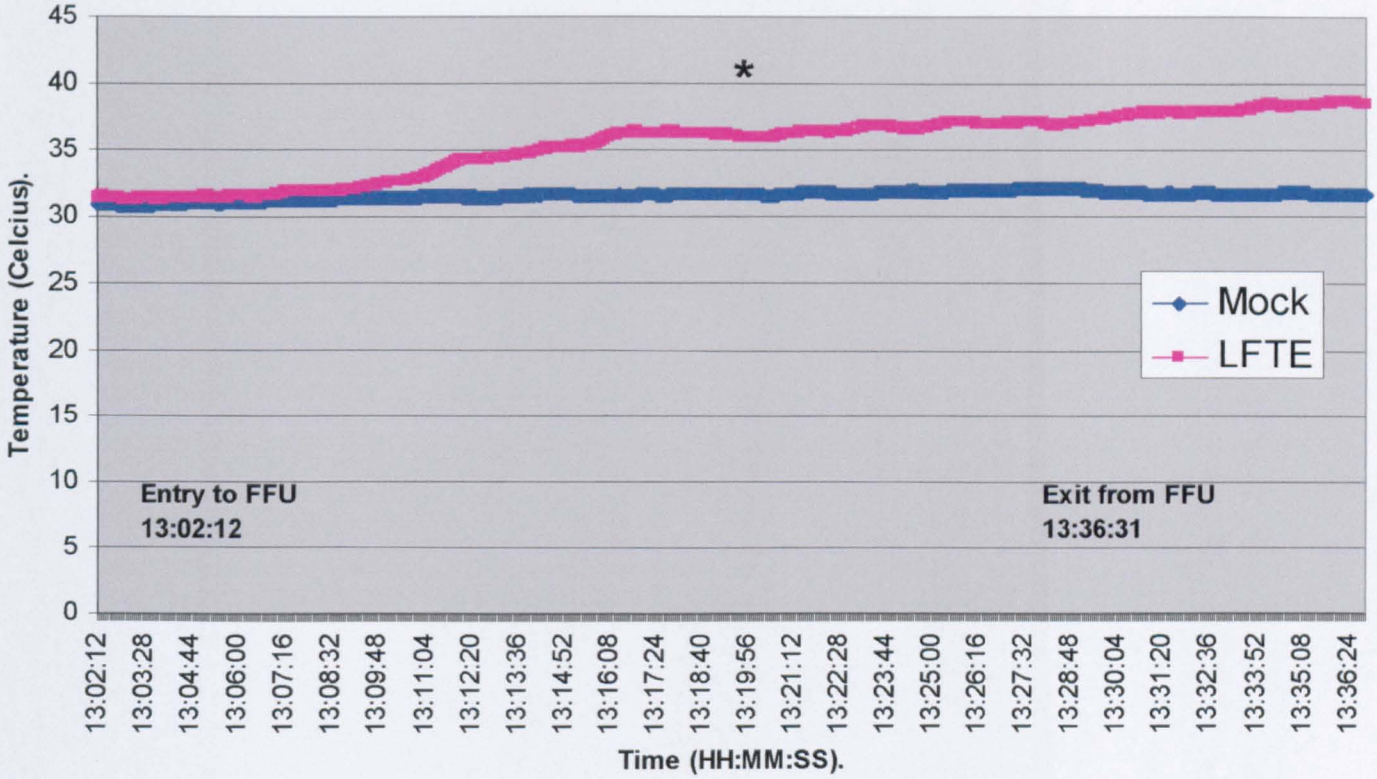


Figure 5.8 – Mean micro-climate temperature in response to mock training exercises and LFTEs. Values were obtained every second through out the exercises (n = 6). Significance was tested using a paired samples t-test. * - significant differences ($p<0.05$) between the mock training exercises and LFTEs.

Table 5.4 shows the significant ($p<0.05$) increase in temperature inside the FFU between the mock training exercises and the LFTEs.

Table 5.4 – Mean environmental temperature readings inside the Fire Flashover Unit during mock and LFTEs. Temperature readings were obtained in the anti-chamber and the fire chamber. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock and LFTEs.

	Anti-chamber Temperature (^{0}C)		Fire Chamber Temperature (^{0}C)	
	Ceiling	Mid level	Ceiling	Mid level
Mock	15.9 (± 2.6)*	15.7 (± 2.2)*	15.1 (± 2.3)*	15.3 (± 2.5)*
LFTE	54.5 (± 4.7)	48.3 (± 4.0)	114.1 (± 20.4)	107 (± 32.9)

Table 5.5 shows the increases in temperature for BAIs for the 3 temperature probes. The mean temperature of the initial 5 minutes of the exercise was then compared to the mean of the final 5 minutes.

Table 5.5 – The mean temperature readings (^{0}C) of the initial and last 5 minutes during exposure to the mock training exercises and LFTEs. Data is presented as the mean (of 5 minutes worth of data) ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock and LFTEs.

	Mock Exercises				LFTEs			
	Beginning	Termination	Δ Temp.	P Value	Beginning	Termination	Δ Temp.	P Value
Skin	32.8 (± 1.7)	33.7 (± 1.5)	0.8 (± 0.6)	0.015*	35.0 (± 2.2)	38.9 (± 1.1)	3.8 (± 1.7)	0.003*
Aural	35.7 (± 0.2)	36.1 (± 0.2)	0.4 (± 0.2)	0.004*	36.0 (± 0.6)	38.3 (± 0.8)	2.2 (± 0.5)	0.000*
Micro-climate	31.5 (± 0.8)	32.3 (± 1.1)	0.8 (± 0.4)	0.006*	32.7 (± 2.6)	38.7 (± 2.1)	6.0 (± 0.6)	0.000*

Table 5.4 shows the small, but significant increases ($p<0.05$) in skin, aural and micro-climate temperatures between the initial 5 minutes and last 5 minutes of the mock training exercises. These increases were as a result of wearing the PC+SCBA and carrying out low intensity exercise. In addition, Figure 5.9 shows the effect of wearing PC+SCBA pre exposure to the mock training exercises when compared to wearing gym kit (t-shirt and shorts). There were significant increases ($p<0.05$) in

skin (3.0 (± 2.8) %), aural (3.0 (± 2.1) %) and micro-climate (20.5 (± 16.3) %) temperatures.

These temperatures were significantly increased ($p<0.05$) during the mock exercises by 4.0 (± 2.7) % for skin temperature, 1.3 (± 0.8) % for aural and 4.9 (± 2.1) % for micro-climate temperature. This was presumably due to the low intensity workload (17.4 (± 2.8) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) and the wearing of the PC+SCBA. On termination of the mock training exercises, skin, aural and micro-climate temperatures returned to the equivalent of the pre exposure gym kit readings. This occurred once the PC+SCBA had been removed and only gym kit was worn. Differences between the pre and post gym kit skin, aural and micro-climate temperatures were not significantly different.

Figure 5.10 shows significant increases ($p<0.05$) in skin, aural and micro-climate temperatures when the BAIs wore PC+SCBA in comparison to the wearing of gym kit prior to exposure to LFTEs. There was a further increase in skin, aural and micro-climate temperature by 10.8 (± 3.3) %, 3.8 (± 1.6) % and 3.8 (± 0.4) % respectively, when the BAIs were exposed to the LFTEs. On termination of the LFTEs, the skin and micro-climate temperatures did not return to the equivalent of the pre exposure gym kit temperature readings until the BAIs had removed their PC+SCBA and were wearing gym kit post exposure to the LFTEs. However, post gym kit aural temperatures were still significantly ($p<0.05$) higher than the pre exposure gym kit aural temperature.

During LFTEs the increase in temperature was further exacerbated over that of the mock fire training exercises through the inclusion of the fire ($p<0.01$). There were significant increases ($p<0.05$) in skin, aural and micro-climate temperatures during LFTEs when compared to the mock training exercises. Figure 5.11 shows typical HR and temperature responses to LFTEs observed within the present study for a BAI. A steady increase in aural temperature is evident from 35.0 to 36.5 °C, microclimate from 30.5 to 35.9 °C and skin from 31.2 to 36.8 °C

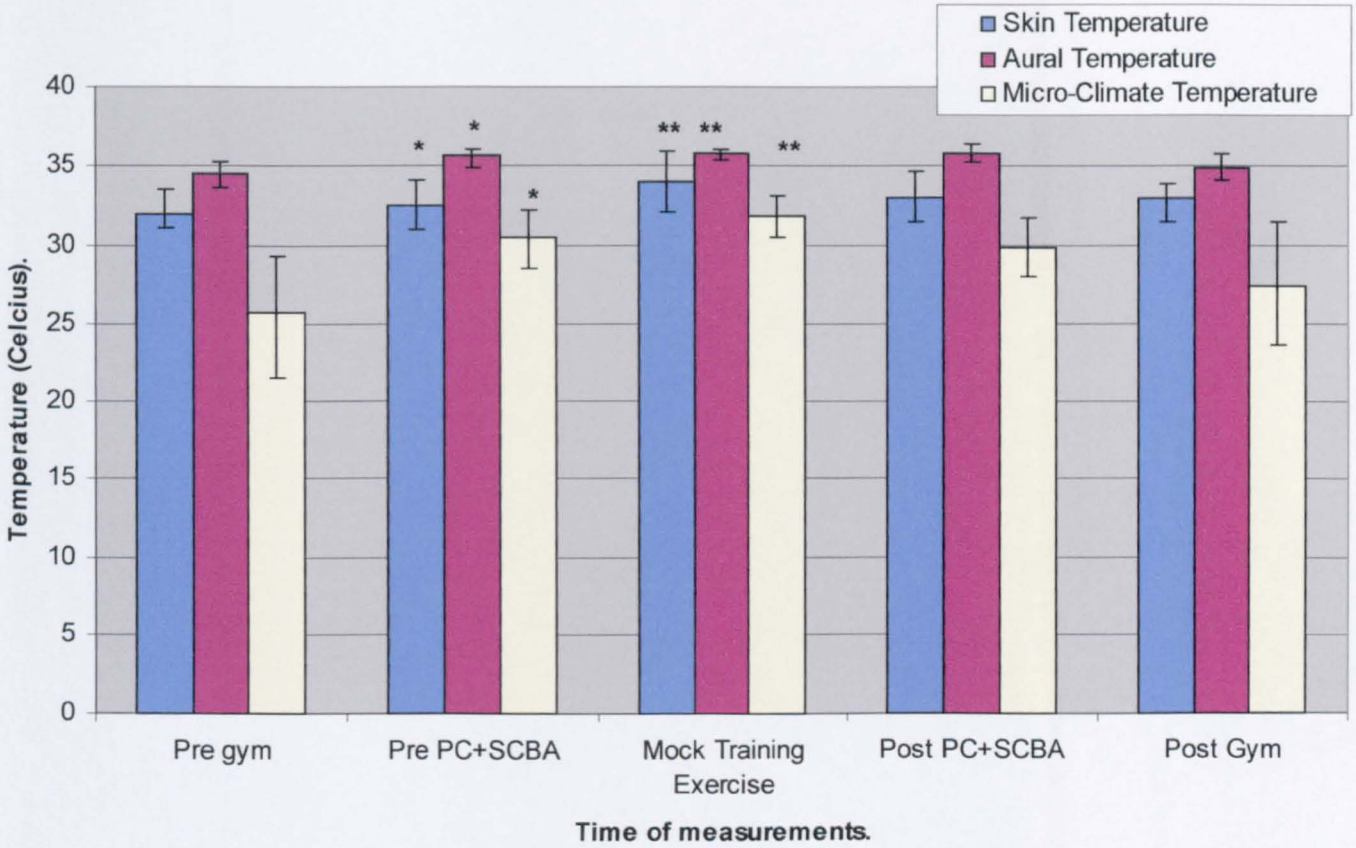


Figure 5.9 – Mean skin, aural and micro-climate temperatures pre exposure (dressed in gym kit (Pre gym) and PC+SCBA (Pre PC+SCBA)), during exposure (Mock training exercise) and post exposure dressed in PC+SCBA (Post PC+SCBA) and also gym kit (Post gym kit). Values were the means (± SD) obtained over 5 minutes for all pre and post readings. During the mock training exercises the data is the mean (± SD) of a 34 minute exposure (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre gym and pre PC+SCBA temperature readings. ** - significant differences ($p<0.05$) between the pre PC+SCBA and the mock training exercises temperature readings.

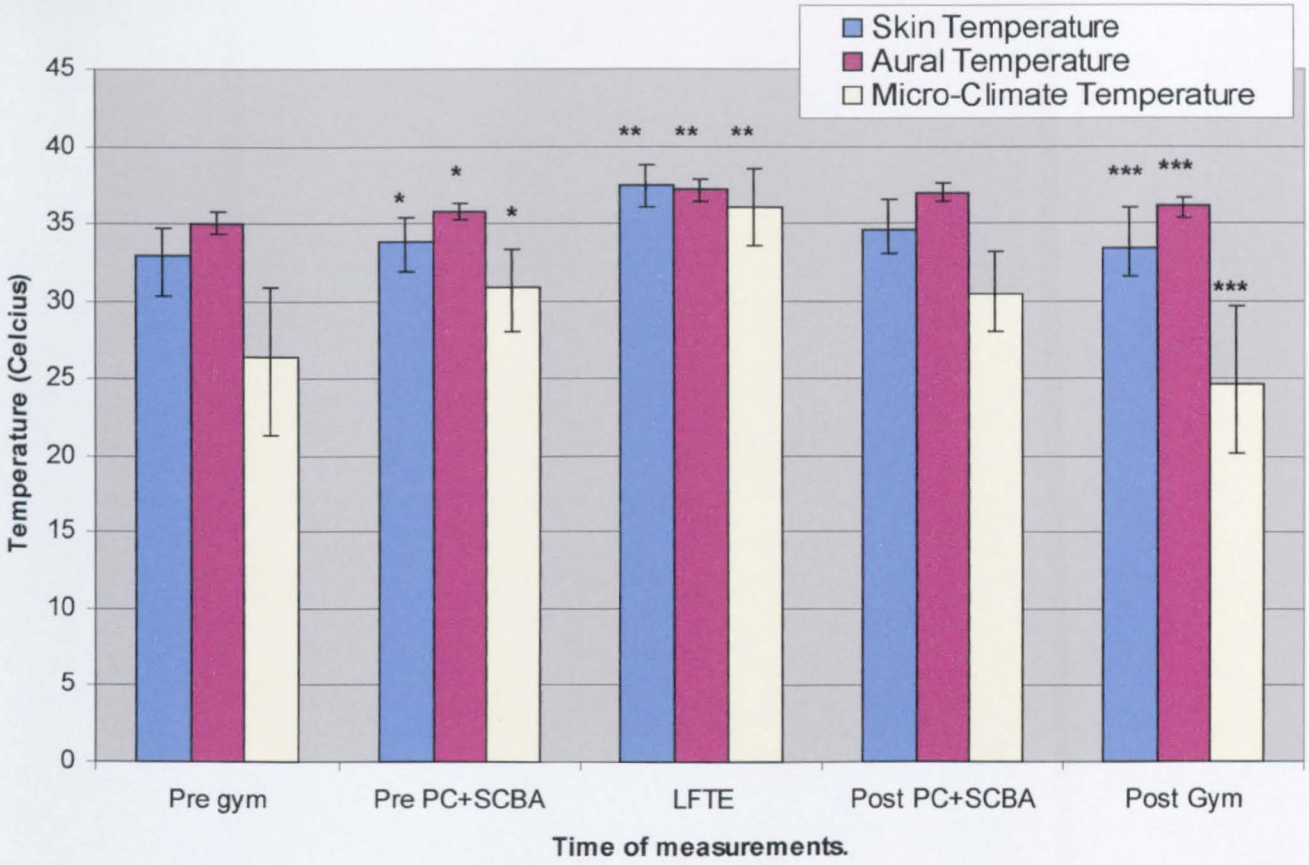
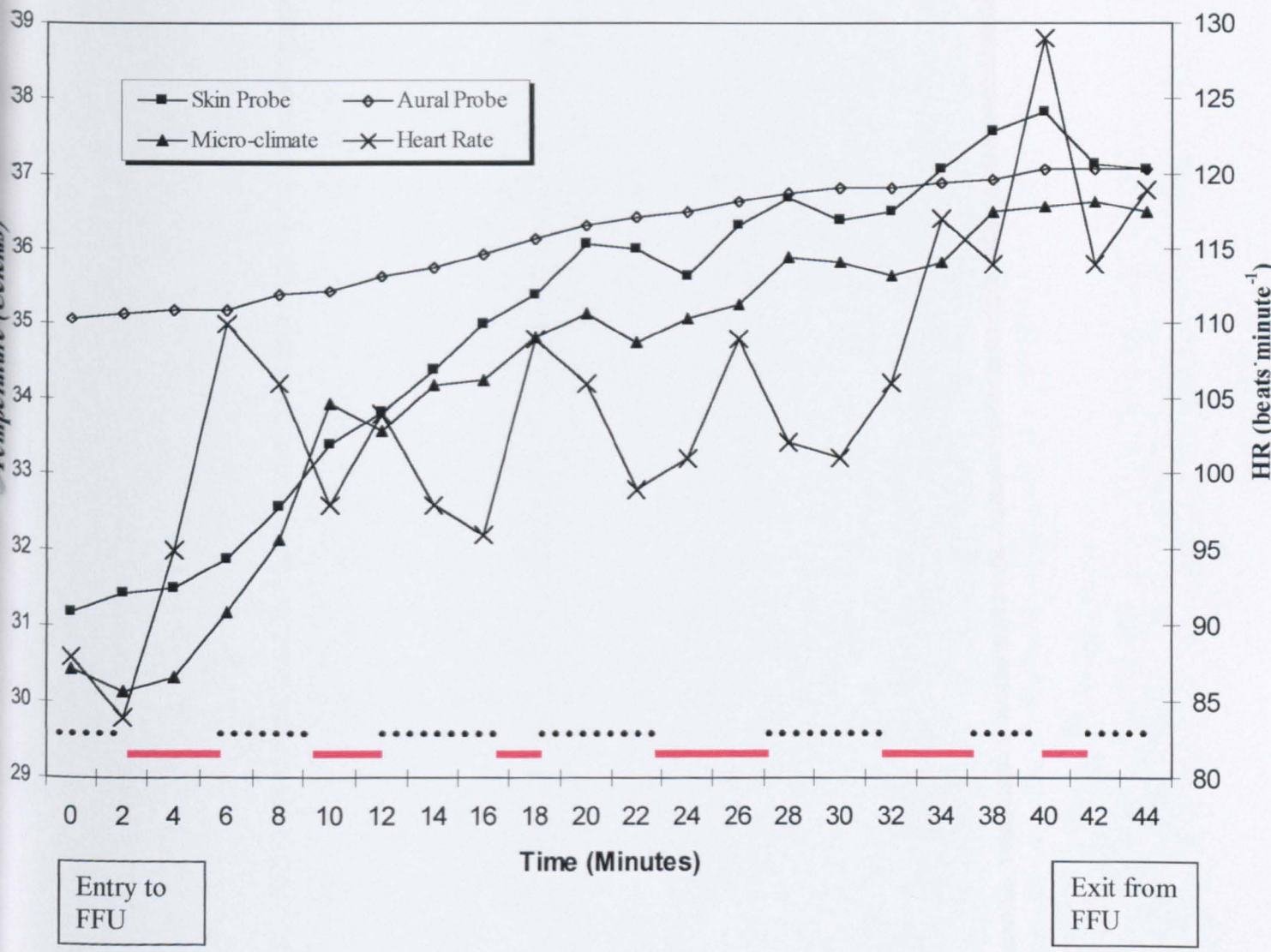


Figure 5.10 – Mean skin, aural and micro-climate temperatures pre exposure (dressed in gym kit (Pre gym) and PC+SCBA (Pre PC+SCBA)), during exposure (LFTE (Live fire training exercise)) and post exposure dressed in PC+SCBA (Post PC+SCBA) and also gym kit (Post gym kit). Values were the means (± SD) obtained over 5 minutes for all pre and post readings. During the LFTEs the data is the mean (± SD) of a 34 minute exposure (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre gym and pre PC+SCBA temperature readings. ** - significant differences ($p<0.05$) between the pre PC+SCBA and the LFTEs temperature readings. * - significant differences ($p<0.05$) between the pre gym and post gym temperature readings.**



..... - *Denotes 'debrief'' when fire was not ignited and instructor was giving feeding back to the students.*

——— - *Denotes when live fire exercises occurred.*

Figure 5.11 – Typical HR, skin, aural and micro-climate temperatures during exposure to a LFTE (live fire training exercise). Values were obtained every 2 minutes.

Figure 5.11 shows the peak and trough effect on temperature and HR responses from the exposure to the fire training exercises and cooler debriefs. The heat from the fire appears to create a progressive increase in the micro-climate temperature and skin temperature. Over the course of the LFTE there appears to be a progressive increase in the heat stored in the body. This is ascertained from the progressive increase in the core temperature (represented by aural temperature). Increases in the skin and micro-climate temperatures appear to result in the progressive increase in core temperature. This amplified heat storage in the body core is consistent with a progressive increase in the HR response over the course of the LFTE.

Blood Pressure.

Figure 5.12 shows the mean systolic pressure pre and post exposure to both the mocks and LFTEs. The increases in systolic blood pressure during the mock training exercises were not significant. However, there were significant increases ($p<0.01$) between the pre and post exposure results when exposed to the LFTEs. Diastolic pressure was not significantly changed between the pre and post measurements for either the mock or LFTEs.

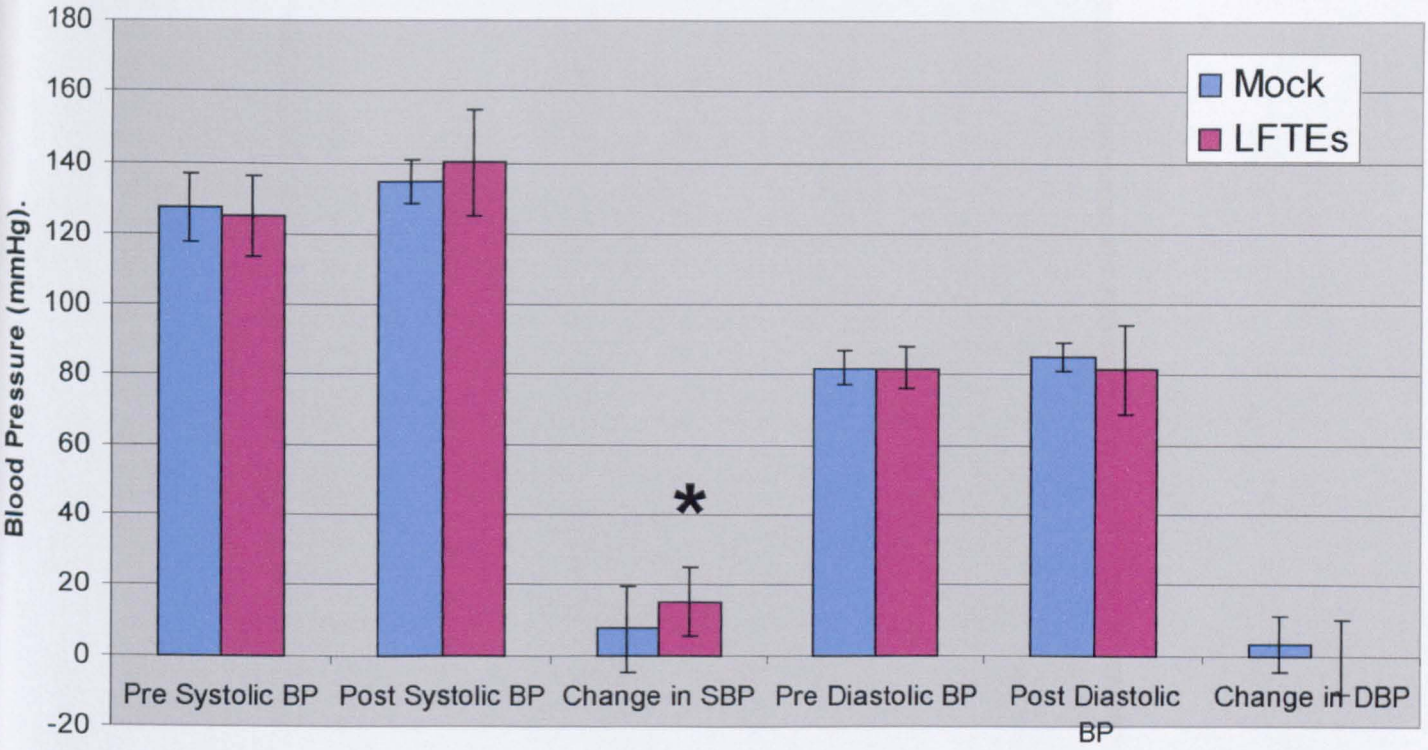


Figure 5.12 – Mean and mean changes in blood pressure (BP) in response to mock and live fire training exercises (LFTEs). Blood pressure was obtained pre exposure (Pre systolic BP and Pre diastolic BP) and post exposure (Post systolic BP and Post diastolic BP). Changes between the pre exposure and post exposure blood pressure values for systolic (change in SBP) and diastolic (change in DBP) blood pressure were obtained. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre and post LFTE systolic blood pressure readings.

Blood Lactate.

Figure 5.13 shows the mean lactate results from the mock and LFTEs. The mean lactate measurements show significant ($p<0.05$) increases between pre and post exposure for both exercises. Lactate production was not excessively high due to the low levels of physical exertion (equivalent to $\leq$ 100 Watts of cycling ergometry) undertaken by the BAIs during the exercises. The results showed no significant differences between the blood lactate levels produced between the mock and LFTEs. This suggests that the workload carried out was similar.

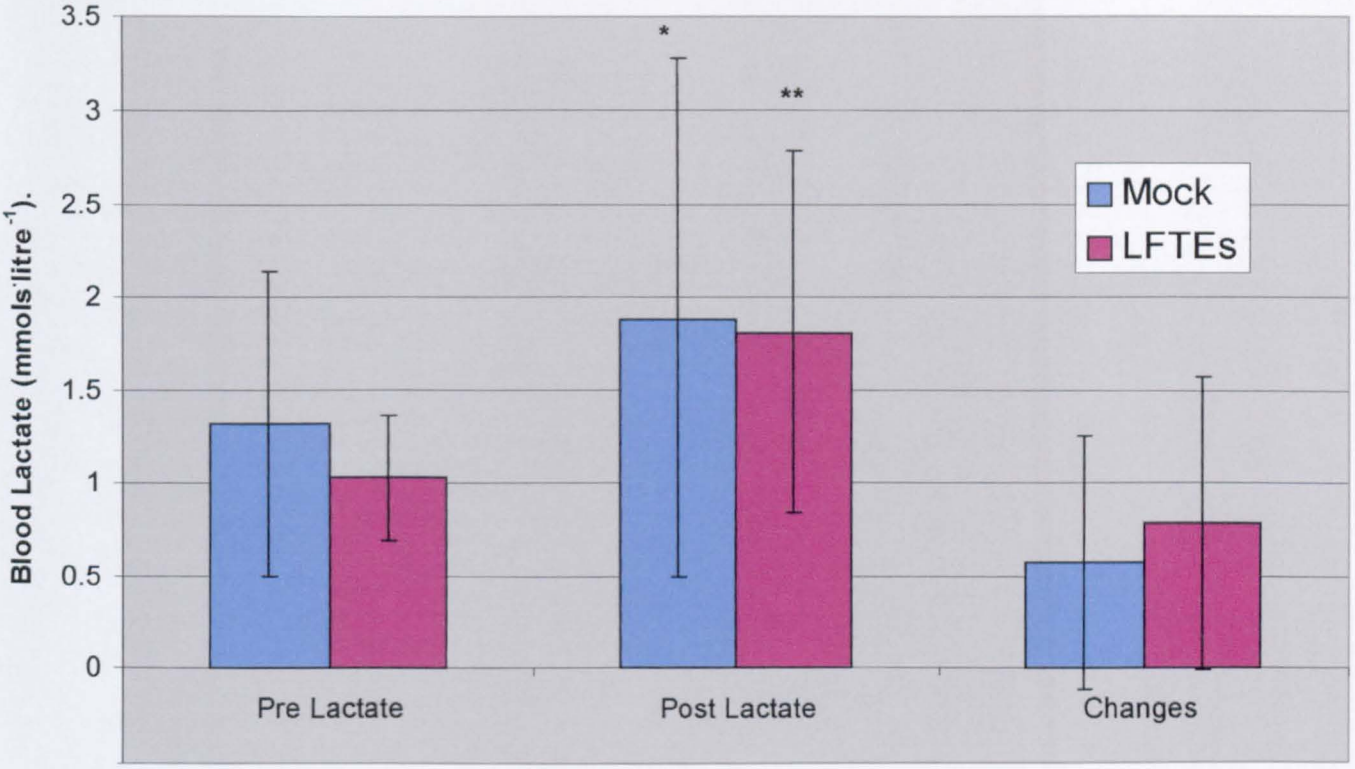


Figure 5.13 – Mean and mean changes in blood lactate in response to mock and live fire training exercises (LFTEs). Blood lactate was obtained pre exposure (Pre Lactate) and post exposure (Post Lactate). Changes between the pre exposure and post exposure blood lactate values (changes) were obtained. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre and post mock blood lactate readings ($p<0.05$). ** - significant differences ($p<0.05$) between the pre and post LFTE blood lactate readings.

Blood Glucose.

Table 5.6 shows the blood glucose values pre exposure to the mock training exercises and LFTEs.

Table 5.6 – Blood glucose values obtained pre and post exposure to either the mock training exercises (Mock) or live fire training exercises (LFTEs). Data is presented as the mean ± SD (n= 6).

	Mock training exercises	LFTEs
Pre exposure blood glucose (mmol·litre^{-1})	5.3 (± 0.9)	5.4 (± 0.8)
Post exposure blood glucose (mmol·litre^{-1})	6.0 (± 0.9)	5.3 (± 0.4)

Body Mass.

According to Brooks *et al* (1996), monitoring pre and post body mass in the absence of fluid intake, is an effective method in observing the amount of fluid lost through heat exposure. Figure 5.14 represents the amount of fluid lost through exposure to the mock training exercises and the LFTEs. The pre and post differences were not significant for the mock training exercises, but were significant for the LFTEs ($p<0.01$). The differences between pre and post exposure to the mock training exercises and the LFTEs are highlighted in Figure 5.14. The mean losses for the LFTEs was -1.1 (± 0.3) kg which equated to a mean loss of -1.3 (± 0.4) % of body mass.

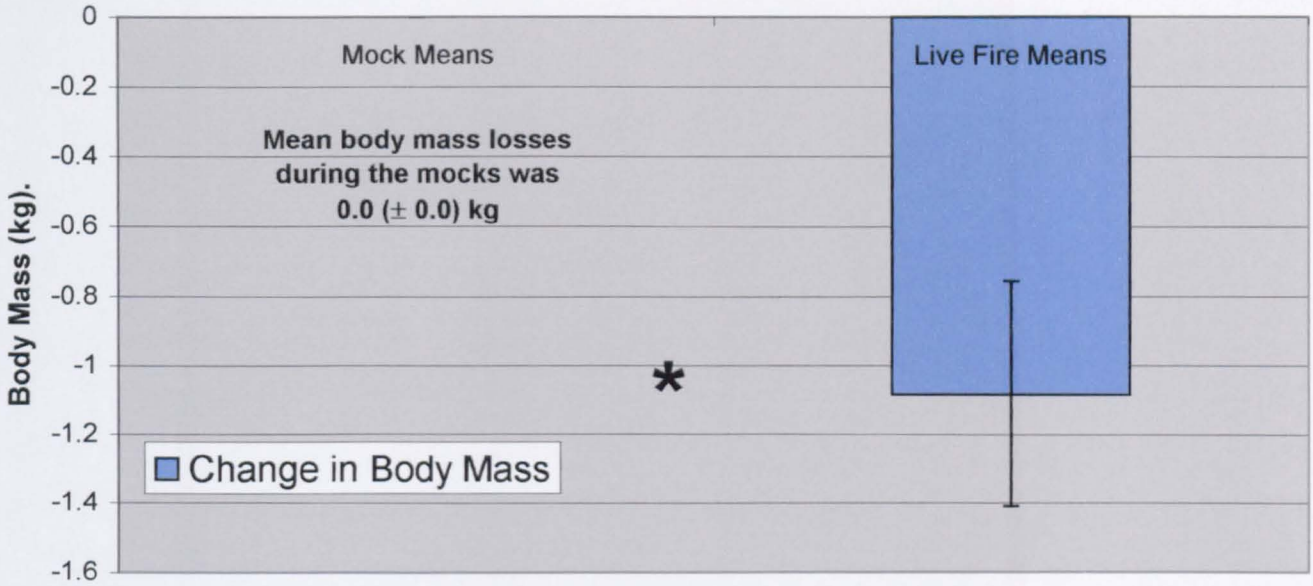


Figure 5.14 – Mean change in body mass between the pre exposure and post exposure readings when exposed to mock training exercises and LFTEs. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test. * - significant differences ($p<0.05$) between the changes in body mass (between pre and post exposure body mass) between the mock and LFTEs readings.

Haematocrit.

The results obtained during the mock training exercises showed there were no significant differences between the pre and post haematocrit values (Table 5.7).

This suggests that there was not a significant decrease in the estimated plasma volume. Therefore, it could be implied that by wearing PC+SCBA without the presence of heat is not a significant stressor to create an increase in percentage of haematocrit post exposure to the mock fire, and, thus a decrease in plasma volume. There was a significant increase ($p<0.05$) between pre and post LFTE haematocrit percentages (mean pre: 43.46 (± 1.88) % and post: 45.58 (± 1.74) %) (Table 5.7). This was consistent with the body mass losses suggesting there were fluid losses through exposure to the LFTE (1.3 (± 0.4) % body mass).

Table 5.7 – Mean haematocrit (%) and estimated plasma volume (Section 3.4.5ii in the Methods) in response to mock and LFTEs. Data is presented as the mean ± SEM (n= 6). Significance was tested using a paired samples t-test. * - significant differences ($p<0.05$) between the pre and post exposure haematocrit measurements. ** - significant differences ($p<0.05$) between the mock and LFTE estimated plasma volumes readings.

	Pre Exposure	**Post Exposure**
Mock training exercises Haematocrit (%)	44.6 (± 0.9)	44.8 (± 1.0)
LFTEs Haematocrit (%)	43.5 (± 1.9)	45.6 (± 1.7)*
	Mock Plasma volume losses	**LFTEs plasma volume losses.**
Mean estimated % reduction in plasma volume.	-0.4 (± 0.6)	-8.1 (± 2.2)**

Fluid balance during mock training exercises and LFTEs.

The amount of fluid consumed and urine produced by the BAIs during the mock and LFTEs was monitored. The BAIs were instructed not to alter their fluid intake, rather, they were told to drink as they would normally. This allowed the fluid balance of the BAIs to be calculated during the days of the mock training exercises and the LFTEs (Table 5.8).

Table 5.8 – Mean daily fluid consumed (mean of the sum of daily fluid consumed), urine produced (mean of the sum of daily urine produced), sweat loss (mean sweat lost (obtained from body mass lost) as a result of exposure to the LFTEs), total fluid losses (obtained from the mean daily urine produced and sweat losses through exposure to the LFTEs added together), fluid balance (mean daily fluid consumed subtracted from the total losses), mean urine osmolality and mean urine colour; intracellular water (ICW), extracellular water (ECW) and total body water (TBW) changes (Dual Scan 2005 (Bodystat, Isle of Man, England)). Body water changes were obtained from the changes between the readings taken in the morning and at the end of the shifts of the days when the BAIs were exposed to the LFTEs. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock and LFTE estimated plasma volumes readings.

	Mock	LFTEs
Mean daily fluid consumed (ml)	2333.3 (± 1237.3)	2154.2 (± 1051.9)
Urine Produced (ml)	1003.6 (± 326.5)	1822.3 (± 1014.3)
Sweat Loss (ml)	0.0 (± 0.0)	1083.3 (± 325.7)*
Total losses (ml)	1003.6 (± 326.5)	2905.6 (± 1074.9)*
Fluid balance (ml)	1329.8 (± 1186.9)	-751.4 (± 704.6)*
Mean Urine Osmolality ($mOsmol \cdot kg^{-1}$)	354.1 (± 77.4)	420.3 (± 136.8)
Mean Urine Colour	2.0 (± 0.7)	3.0 (± 0.7)
Body water changes between the beginning and end of the shift on the days of mock and LFTEs.		
ECW changes between the beginning and end of the shift (ml)	141.7 (± 271.2)	33.3 (± 349.9)
ICW changes between the beginning and end of the shift (ml)	33.3 (± 296.4)	-58.3 (± 460.2)
TBW changes between the beginning and end of the shift (ml)	175.0 (± 508.3)	-25 (± 781.8)

The mean amount of weight lost by sweating during LFTEs (1.08 (± 0.33) kg between pre and post exposure, which equated to 1083.3 (± 325.7) ml), was observed. The sweat losses were significantly increased ($p<0.05$) when the BAIs were exposed to the LFTEs in comparison to the mock training exercises. This was due to the significant ($p<0.05$) increase in temperature inside the FFU. The increase in sweat was accompanied by significant ($p<0.05$) increases in urine osmolality and

colour between pre and post exposure to the LFTEs. The mean total fluid losses (obtained from the addition of the mean sweat losses, to the mean of the sum of urine lost) were subtracted from the mean total fluid intake (obtained from the mean daily fluid intake). This produced the fluid balance of the BAIs during the days of the mock training exercises and LFTEs. The fluid balance results showed the BAIs were in negative fluid balance during the days of the LFTEs (-751.4 (± 704.6) ml), which was significantly lower ($p<0.05$) than the positive fluid balance of the BAIs during the days of the mock training exercises (1329.8 (± 1186.9) ml). There were no significant differences between the mean daily urine osmolality or urine colour between the mock and LFTEs.

Body water (total body water (TBW), extracellular water (ECW) and intracellular water (ICW)) was obtained using the Dual Scan 2005 Body water analyser (BodyStat, Isle of Man, England) which used the method of bioelectrical impedance analysis. The changes between the commencement and the completion of the BAIs shift for TBW, ECW and ICW, were not significantly different between the mock and LFTEs.

The mean fluid consumed 2 hours prior to exposure to the LFTEs was 431.3 (± 381.3) ml, which compared to the fluid lost during the LFTEs. This meant that the BAIs were in an average fluid deficit of 652.1 (± 560.3) ml. The mean fluid consumed 2 hours post exposure to the LFTEs was 481.3 (± 396.0) ml, which compared to the fluid lost during the LFTEs meant the BAIs were in an average fluid deficit of 602.1 (± 436.1) ml. When the mean fluid consumed 2 hours prior to exposure (431.3 (± 381.3) ml) was subtracted from the mean fluid lost during the LFTEs (1083.3 (± 325.7) ml), the resultant median fluid loss was 637.5 ml. This suggests that consuming 600 ml of water prior to exposure, may maintain fluid balance of the BAIs on exposure to the LFTEs.

Psychological Responses to mock training exercise and LFTEs.

Rate of Perceived Exertion (RPE).

The RPE scale was incorporated into the study to observe the BAIs perception of how hard they were working (although the oxygen cost was low (17.4 (± 2.8) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$)). The results for the mock training exercises showed small but significant increases ($p<0.05$) in the RPE responses obtained during exposure (mid way through – approximately 15 minutes into the mock training exercises) (7.6 (± 1.4)) compared to the responses obtained pre exposure (6.0 (± 0.0)). There were also significant increases ($p<0.05$) between the RPE responses obtained during the mock training exercises (7.6 (± 1.4)) and the post exercise RPE measurements (8.0 (± 1.7)) (Figure 5.15).

There were significant ($p<0.05$) increases between the RPE responses measured during exposure (9.0 (± 1.1)) when compared to the RPE measurements obtained pre exposure (6.0 (± 0.0)) to the LFTEs. There were also significant increases ($p<0.05$) in the RPE responses obtained post exposure (12.0 (± 1.8)) in comparison to the RPE responses obtained during exposure (9.0 (± 1.1)) to the LFTEs (Figure 5.15).

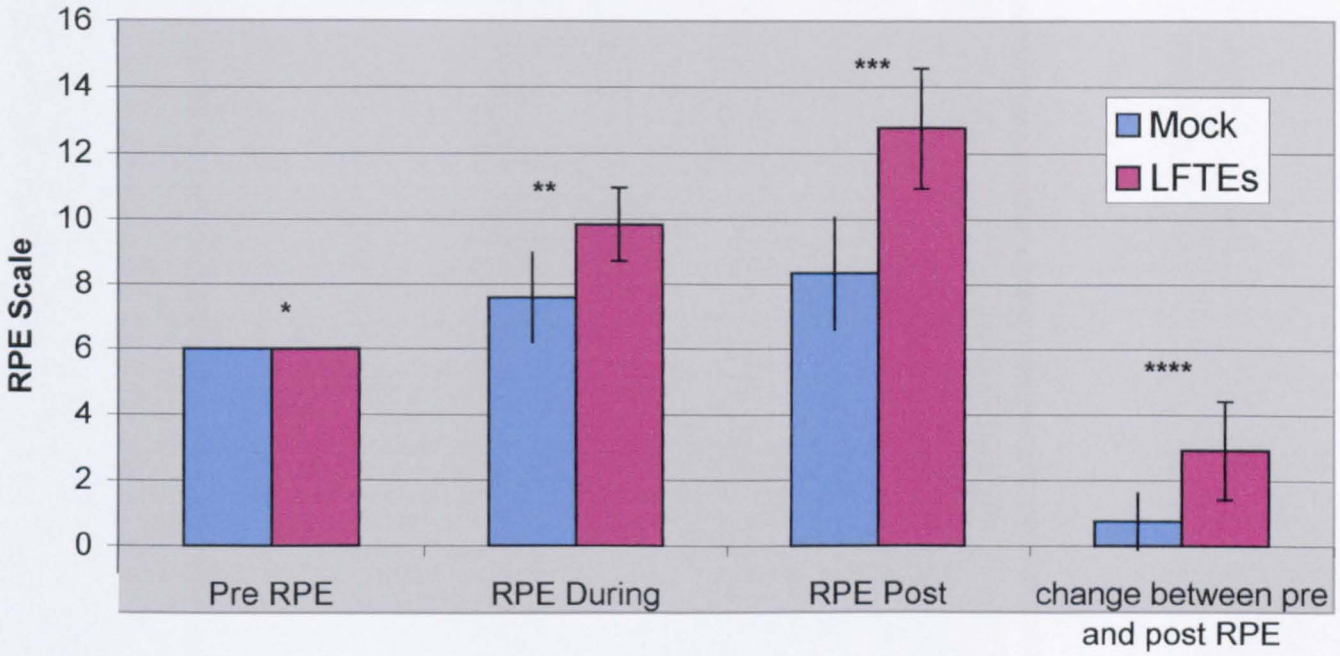


Figure 5.15 – Mean and mean change (between the pre and post exposure measurements) in perceived exertion ratings pre (carried out immediately prior to entering the FFU), during (carried out at the mid-point which was approximately 15 minutes into the LFTE) and post (carried out immediately after leaving the FFU) exposure to the LFTEs. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre and post exposure readings for the mock training exercises and the LFTEs. ** - significant differences ($p<0.05$) between the pre and during exposure readings for the mock training exercises and the LFTEs. * - significant differences ($p<0.05$) between the during and post exposure readings for the mock training exercises and the LFTEs. **** - significant differences ($p<0.05$) between the changes (between the pre and post exposure readings) between the mock training exercises and the LFTEs.**

Adapted Stroop Test.

The adapted Stroop test was incorporated into the study in order to test whether there were decreases in BAI concentration as a result of exposure to LFTEs. It was hypothesised that the more stressed and dehydrated the BAIs became, the longer it would take to carry out the adapted Stroop test.

There were no significant changes in the time for the BAIs to complete the adapted Stroop test between pre exposure and during exposure measurements, nor between the pre exposure and post exposure measurements during the mock training exercises (Figure 5.16). The adapted Stroop test times for the LFTEs were significantly increased ($p<0.05$) between the pre and post exposure readings. There were no significant differences between the pre exposure measurements and the measurements obtained during the LFTEs (obtained approximately 15 minutes into the LFTEs). In addition, there were no significant differences between the measurements obtained during the LFTEs and the post exposure measurements (Figure 5.16).

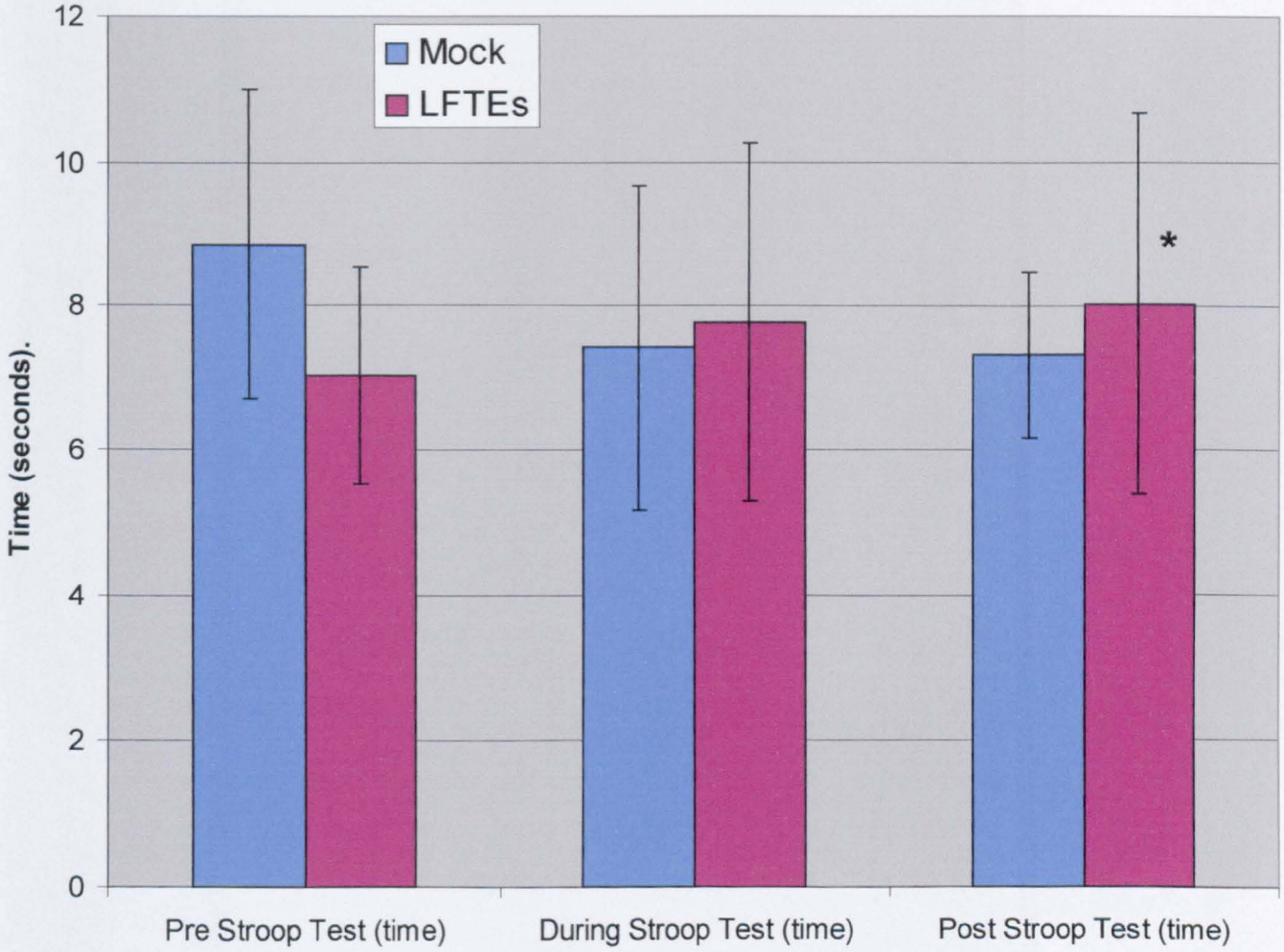


Figure 5.16 – Mean time to complete the adapted Stroop test pre (carried out immediately prior to entering the FFU), during (carried out at the mid-point which was approximately 15 minutes into the LFTE) and post (carried out immediately after leaving the FFU) exposure to the LFTE. Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA with post hoc Tukey. * - significant differences ($p<0.05$) between the pre and post LFTE readings.

5.4 Discussion.

The aim of this work was to observe the contribution heat and students had on the physiological and psychological responses of BAIs when exposed to LFTEs whilst wearing PC+SCBA.

Heart Rate (HR)

Part of the increase in heart rate (observed from Figure 5.1) was a result of the heat from the fire. However, a certain amount of this 'stress' may be attributable to the psychological stress of having to monitor the safety of students within this environment. There were increases in HR caused by wearing the PC+SCBA. In addition, HR was also increased through the storage of heat from wearing the PC and carrying out the necessary workload during mock training exercises and LFTEs.

An increase in HR as a result of exposure to the LFTEs would be expected due to the increase in demand for blood flow being shared with several vital organs. In particular there is a diversion of the blood supply to the working muscles and an increase in blood flow to the skin, in order to dissipate the heat from the body's core (Nadel, 1980). According to Fortney and Vroman (1985), the body is highly adept at tolerating most naturally occurring heat stresses through both physiological and behavioural responses. During exercise the venous drainage carries heat produced from the working muscles to the body's core, thus elevating the core temperature. This was observed in the present study when the mean aural temperatures from the mock and LFTEs were compared (Figure 5.8).

This rise in core temperature is integrated with input from the peripheral thermal receptors by the hypothalamus. As the body's core temperature increases, there is a drive to dissipate the heat. There are two major efferent outputs – increased sweat output and an inhibition of cutaneous vasomotor tone (Fortney and Vroman, 1985). This results in an increase in cardiac output as the body attempts to provide sufficient circulation to the combined demands of both the muscles and the skin or compromise one in lieu of the other (Nadel, 1980). Selective blood flow to either of the areas would result in a negative outcome, as a reduced blood flow to the skin means that there is a reduction in the amount of heat dissipation, thus creating an increase in

core temperature. A decrease in the blood flow to the muscles may lead to anaerobiosis and therefore an impairment of physical performance (Nadel, 1980).

The body attempts to increase blood flow by increasing cardiac output. This is achieved through increasing HR which, in turn, allows an increased blood flow to both the cutaneous layer and working muscles (Fortney and Vromen, 1985).

The increases in HR observed during the LFTEs, when compared to the mock training exercises, are supported by earlier works such as Shaffrath and Adams (1984). They observed that an increase in environmental temperature increased the progressive distribution of blood into cutaneous capacitance vessels and to the working muscle capillary beds. The progressive distribution of blood caused a reduction in central blood volume, in turn, creating an increase in HR to increase the filling pressure (Shaffrath and Adams, 1984). This was observed from a significantly higher heart rate ($p<0.05$) when their subjects exercised at 43.4% and 62.2% of $VO_{2\ max}$ at higher ambient temperatures (39.5 ($\pm$ 2.4) ^{0}C) when compared to lower ambient temperatures (24 ($\pm$ 0.8) ^{0}C).

The increase in HR observed during the mock training exercises and LFTEs could also be attributed to an increase in heat storage from wearing the PC. This was consistent with the HR responses observed in Chapter 4. The increase in HR during the present study was consistent with the increases in micro-climate observed between the pre exposure gym kit and pre exposure PC+SCBA measurements. Such measurements were obtained prior to exposure to the mock training exercises (Figure 5.11) (20.5 ($\pm$ 16.3) % increase) and the LFTEs (Figure 5.12) (18.9 ($\pm$ 15.9) % increase).

Time constraints were placed on the study by Greater Manchester Fire Service. This made it impossible to carry out an additional study to investigate HR responses of BAIs in conjunction with the addition of students when not exposed to a LFTE. However, it is more likely that the amount of stress attributable to the presence of the students would be minimal. From personal communication with BAIs, they felt they were not affected by the inclusion of the students due to the safety precautions that

exist (the fire can be shut off at the press of a button) and their considerable experience in live fire training. Figure 5.2 shows the mean HR response produced by the BAIs during exposure to a LFTE compared to the mean student HR responses. The students HR responses show the effect of not only the heat but also the additional stress of dealing with the new situation of being in a LFTE. The BAIs HR response did not increase to the same degree as the students, possibly further suggesting that the stress from the addition of students on the BAIs during LFTEs was negligible.

The increase in cardiac stress observed in students over that of BAIs was consistent with Taylor, Fogarty and Armstrong (2000). They also observed increases in trainee fire fighters HR responses (reported mean HR 148 beats·minute^{-1} (SD not reported)) through anxiety, due to the unfamiliarity of exposure to LFTEs.

Oxygen Cost.

The mean oxygen cost, obtained from the drop in cylinder pressure over the 34 minutes of exposure was significantly increased ($p<0.05$) during the LFTEs when compared to the mock training exercises. Powers, Howley and Cox (1982), state that the increase in oxygen cost may be due to increased metabolic demands to allow a greater peripheral circulation, greater sweat gland activity and also increase in stress hormone activity. As previously reported, the BAIs followed a scripted exercise schedule ensuring they carried out the mock training exercises and LFTEs identically (Table 3.2 in the Methods). This strongly suggests the increase in oxygen cost was not a result of the BAIs carrying out additional activity during the LFTEs.

The oxygen cost from carrying out the demo, 2D, 2DD and 3 (exercises carried out during the mock and LFTEs) were not significantly different between the mock training exercises and the LFTEs. This may have been caused by two factors. Firstly, the BAIs may have increased the amount of oxygen used through excessive talking and shouting. It was not possible to control the amount the BAIs talked during their debrief sessions. The BAIs were timed on how long each exercise and debrief took, but it would have been beyond the scope of the study to script the verbal instructions given throughout the fire exercises and would have interfered with the LFTEs to an unacceptable level.

Secondly, the lack of significant difference in the oxygen cost for the demo, 2D, 2DD and 3 between the mock and LFTEs may be caused through the amount of control the BAIs had over the individual exercises. For example, fire exercises 1 and 2 were controlled by the BAI in the control room. This allowed the BAIs greater freedom to move with the students, which would have placed a greater amount of heat stress on the BAIs during the LFTEs when compared to the mock training exercises. However, the 2D, 2DD and Number 3 fire exercises were all directly controlled by the BAIs within the chamber. The BAIs in the FFU hold a control box which is attached to the chamber via a lead which controls the fire, this therefore restricts the BAIs to the distance they are able to move. This would suggest, by limiting the BAIs movement they were not subjected to as much heat stress.

Therefore, the BAIs carried out low levels of physical activity during LFTEs which was supported by the oxygen cost (17.4 (± 2.8) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$). This equated to 47.8 (± 10.7)) % of $VO_{2\ max}$, which was obtained from a maximal test (Section 3.4.8 in the Methods). The oxygen cost values were not reflected by the HR responses of 116 (± 18.8) beats·minute^{-1} which equated to 66.6 (± 10.4) % maximum HR (Section 3.4.8 in the Methods). Astrand and Rodahl (1986) report that the 47.8 (± 10.7) % of $VO_{2\ max}$ observed during the LFTEs is equivalent to 60% of HR maximum. This shows there is an increase in the HR response of 6% which could be accounted for by the increase in heat when the BAIs were exposed to the LFTEs. This suggests that it is indeed the heat stress that increased the HR, as opposed to an increase in workload.

Temperature.

It appears the intensity of carrying out the mock training exercises was low (13.8 (± 2.3) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$, which was equivalent to ≤ 100 Watts (Astrand and Rodahl, 1986)). However, the additional weight from wearing the PC+SCBA was large enough to produce an increase in heat storage in the core as represented by the significant increase ($p<0.05$) in core temperature (represented by aural temperature, Figure 5.7). Also, wearing the PC+SCBA and carrying out the workload that was

necessary during the mock training exercises created significant increases ($p<0.05$) in skin, aural and micro-climate temperature (Table 5.5).

There was an additional significant increase ($p<0.01$) in skin, aural and micro-climate temperature observed when the initial 5 minutes of the LFTEs were compared to the last five minutes of the LFTEs. In order to observe the accumulative effect of being exposed to LFTEs, the initial 5 minutes of temperature data obtained during the LFTEs was compared to the last 5 minutes of temperature data obtained. The increase in micro-climate and skin temperature, as evident in Figure 5.10, was in accordance with the findings of Duggan (1988), Aoyagi *et al* (1994) and Taylor *et al* (2000). The significant increase in micro-climate ($p<0.05$) appears to suggest that this may cause an increase in skin temperature. The impermeable nature of the PC does not allow the exchange of heat from the skin to the environment through the process of evaporation. This was observed from the significant increases ($p<0.05$) in skin and micro-climate temperatures between pre gym kit and pre PC+SCBA measurements for the mock training exercises (Figure 5.9) and the LFTEs (Figure 5.10).

Figure 5.17 clearly shows that wearing PC+SCBA increases skin, aural and micro-climate when compared to when the BAIs wearing gym kit only. There is an increase in skin, aural and micro-climate during the mock training exercises through carrying out the workload necessary to complete the mock training exercises, carrying the weight of the PC+SCBA and the increase in heat storage from wearing the PC. There is a further increase in these temperatures when there is the addition of fire and therefore, heat during the LFTEs. With the addition of heat during the LFTEs, the increase in micro-climate and skin temperatures are consistent with the core temperature (represented by the aural temperature). This suggests there is not a thermal gradient that would allow heat to leave the core. Instead, the core temperature would continue to rise (as represented by Figure 5.11). This suggests lowering the micro-climate and skin temperature may create a thermal gradient, thus lowering the amount of heat stored in the core as opposed to increasing it.

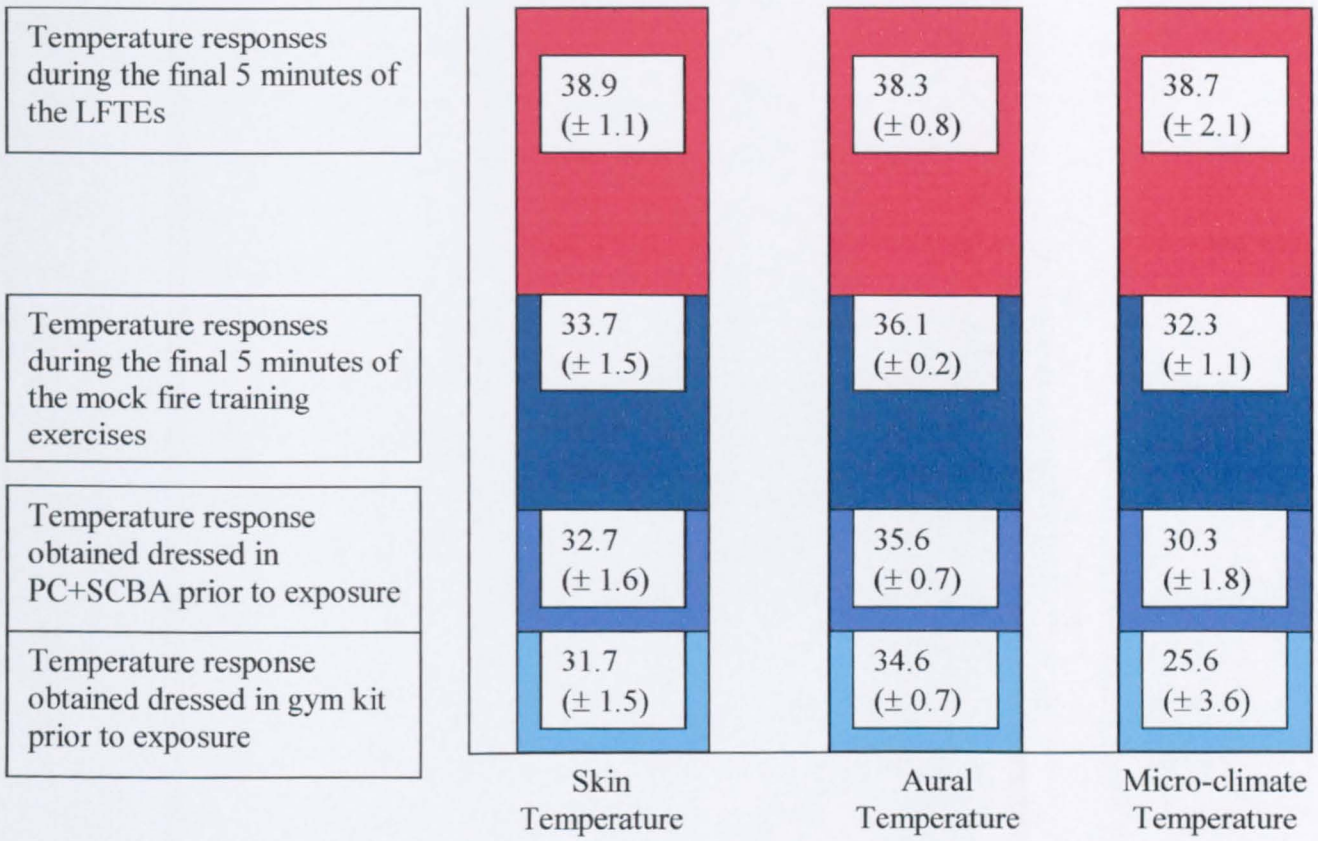


Figure 5.17 – Temperature responses (^{0}C) when wearing gym kit and protective clothing and self-contained breathing apparatus (PC+SCBA) during mock training exercises and live fire training exercises (LFTEs). Data is presented as the mean ± SD (n= 6).

Thus, as the body fails to cool itself down greater stress will be placed on the BAIs during LFTEs. The aural temperatures observed in Table 5.5 at the commencement and termination of the LFTEs, are consistent with Taylor *et al* (2000). They observed core temperature readings (using aural temperature) of 35^0C at the beginning and 38^0C at the termination of LFTEs. Therefore, during the LFTEs there was considerable heat storage within the PC from the heat exuded from the fire. This suggests that a rise in temperature within the PC may also be responsible, in part, for the increase in HR.

As a result of exposure to the LFTEs, there were significant increases ($p<0.05$) in aural temperature between the pre gym kit and the post gym kit measurements. This suggests the BAIs had not reduced their core temperature even on removal of the PC+SCBA post exposure to the LFTEs. This implies that the effects of exposure to LFTEs may continue even after the BAIs have exited the fire flashover unit.

From Figure 5.11 it is also possible to observe the peak and trough nature of the HR and temperature responses during LFTEs. Such responses are caused by the exercises being interspersed with debrief sessions where the BAIs feed back on the student's performance. During the debriefs, the fire was extinguished but heat was retained in the container as the doors on the fire flashover unit (FFU) remained closed. It is also evident that the temperature inside the PC (evident from the increase in micro-climate) and HR responses progressively increased. This also suggests the heat storage within the PC may be significant in placing greater stress on the BAIs.

It could be argued that the significant increases in skin, aural and micro-climate temperature between the mock exercises and LFTEs, were caused solely by the increase in environmental temperature within the FFU. This is because the work rate carried out during the LFTEs was identical during the mock training exercises (Table 3.2 in the Methods). Assuming there are no other factors involved, the differences shown in Table 5.5 highlight that the heat stress is caused by the increase in environmental temperature, which in turn increased the micro-climate temperature (Muir, Bishop and Kozusko, 2001). The increase in micro-climate (6.0 ($\pm$ 0.6) ^{0}C) may have increased the skin temperature by 3.8 ($\pm$ 1.7) ^{0}C and in turn, increased core

temperature (represented by aural temperature) by 2.2 (± 0.5) ^{0}C respectively. The increases in temperature occur as the body is unable to cool itself due to the impermeable nature of the PC+SCBA.

Blood Pressure.

As a result of the mock training exercises systolic blood pressure increased (although not significantly). This suggests that wearing PC+SCBA and the low intensity of exercise carried out during the mock training exercises, may place a small amount of cardiovascular stress on the BAIs even before the inclusion of heat.

The significant increase ($p<0.05$) in systolic blood pressure (12.3 (± 7.9) %) obtained between pre and post exposure to the LFTEs, suggests that the heat from the fire and the presence of students stresses the cardiovascular system of the BAIs over the weight of the PC+SCBA alone.

Research carried out by Volek *et al* (2001) investigated blood pressure responses to a temperate environment (37^{0}C) for 30 minutes whilst exercising at 60-70% VO_{2peak}. Their blood pressure responses are summarised below (Table 5.9).

Table 5.9 – Table to summarise findings from (A) Volek *et al* (2001), (B) Gonzalez-Camarena *et al* (2000) and (C) the present study pre and post LFTEs. Data is presented as mean (± SD).

(A)	Systolic BP	Diastolic BP
At 10 minutes	157 (± 5)	82 (± 3)
At 20 minutes	155 (± 6)	79 (± 4)

(B)	Systolic BP	Diastolic BP
6 mins	173.9 (± 14.6)	97.7 (± 12.1)

(C)	Systolic BP	Diastolic BP
0 mins	124.9 (± 11.4)	82.2 (± 6.0)
35 mins	140.2 (± 15.1)	81.8 (± 12.9)

The mean post exposure systolic readings for BAIs (140.2 (± 15.1) mmHg) was lower than the results produced by Volek *et al* (2001) and also Gonzalez-Camarena *et al* (2000). They observed blood pressure in response to exercise (60% $VO_{2\ max}$) in thermoneutral conditions. Both studies elicited higher systolic responses through exercising their subjects continuously and at a higher intensities, whereby the BAIs

worked intermittently and at lower intensities which was reflected in the lower oxygen cost responses (47.8 (± 10.7) % of $VO_{2\ max}$).

According to both Brooks *et al* (1996) and Astrand and Rodahl (1986), the diastolic blood pressure results are typical responses of diastolic pressure to exercise. The diastolic is not likely to change significantly but will decrease due to the heat and exercise stresses. This causes a decrease in peripheral resistance that develops due to the increase in vasodilatation to the muscle capillary beds (McArdle, Katch and Katch, 2001). This is evident from Table 5.9.

Differences between the blood pressure responses between Gonzalez-Camarena *et al* (2000) and Volek *et al* (2001), may be due to Gonzalez-Camarena *et al* (2000) using sedentary subjects. This would increase the likelihood of increased blood pressure responses to exercise, as it would have been a more stressful experience when compared to the trained subjects in the study by Volek *et al* (2001). The BAIs in the preset study produced a $VO_{2\ max}$ of 39.9 (± 7.8) ml $O_2 kg^{-1} min^{-1}$. According to Cooper (1977) (cited in Powers and Howley, 2000), their aerobic capacity is graded as 'fair'. It could be postulated that this category places them between the sedentary subjects of Gonzalez-Camarena *et al* (2000) and the trained subjects of Volek *et al* (2001). This may suggest that BAIs with a low aerobic fitness level may find exposure to LFTEs more stressful than those with a higher aerobic fitness level. Therefore, in order to lower the cardiovascular stress placed on the BAIs during LFTEs, BAIs should have a sound level of aerobic fitness.

Blood Lactate.

The blood lactate concentration showed significant increases ($p<0.05$) between pre exposure and post exposure to the mock training exercises. This may suggest that the effect of wearing the PC+SCBA and the intensity of the mock training exercises, created an increase in muscle glycolysis. This, in turn, produced an increase in blood lactate concentration. The increase in blood lactate observed between pre and post exposure to the mock training exercises was also observed between pre and post exposure to the LFTEs ($p<0.05$). The changes between pre and post blood lactate concentrations for the mock and LFTEs were not significantly different. This

suggests blood lactate was not affected by the addition of heat from the fire during LFTEs. There were no significant differences between the mock and LFTEs, confirming the weight of the PC+SCBA and also the exercise intensity during the mock and LFTEs remained the same. The blood lactate levels obtained pre and post exposure to LFTEs were low (pre exposure 1.0 (± 0.3)) mmol·litre^{-1} and post exposure 1.8 (± 1.0) mmol·litre^{-1}). This suggests that the BAIs were not working as hard as the subjects that carried out the Step Test (Chapter 4) who reached their ventilatory threshold. Nielsen (1994) also did not observe a significant increase (1.3 (± 0.1) to 2.2 (± 0.2) mmol·litre^{-1}) in blood lactate during thermoneutral conditions (20^{0}C) when compared to temperate (40-42^{0}C) conditions. Subjects exercised at 60% of $VO_{2\ max}$ (BAIs exercised at 47.8 (± 10.7) % $VO_{2\ max}$ during LFTEs) for 30 minutes (walking on a treadmill), they were then required to move to an adjacent heated room and continue walking for a further 60 minutes or until exhaustion, or whichever was reached first.

Research carried out by Tatterson *et al* (2000), investigated the effect of cycling in temperate and thermoneutral environments. Their findings showed lactate concentrations were significantly higher ($p<0.05$) during the temperate (32^{0}C) environment when compared to the thermoneutral environment (23^{0}C). This was within the initial 10 minutes of a 30 minute bout of exercise. However, during the final 10 minutes the lactate values were significantly lower ($p<0.05$) due to a lower power output ($p<0.05$). This suggests that heat stress induced a decrease in power output and in turn a lower lactate production. Therefore, the higher intensity of exercise appears to cause an increase in body temperature that, according to Tatterson *et al* (2000), is an associated factor with the increased lactate production.

Febbraio *et al* (1996) were in agreement with Tatterson *et al*, (2000) who also observed significant increases ($p<0.05$) in lactate production during temperate environments. Febbraio *et al* (1996) believe that it is the increase in muscle temperature that results in an increased rate of glycolysis during exposure to a temperate environment. This in turn increases the rate of lactate production. Febbraio *et al* (1996) utilised intensities that were equivalent to 115% of $VO_{2\ max}$ for a period of 2 minutes.

The findings of Febbraio *et al* (1996) were not consistent with the present study's findings that showed no significant differences in lactate concentrations between the mock and LFTEs. The differences in exercise intensity may explain the disparity in findings when the results from the present study are compared to others examining continuous protocols. In addition, Febbraio *et al* (1996) exercised their subjects continuously. The BAIs during LFTEs did not exercise continuously due to debriefs between the exercises, whereby they stopped to instruct the students on fire science and fire fighting techniques, thus, creating peak and troughs of exercise intensity.

Therefore, blood lactate was increased significantly between the pre and post exposure measurements for both the mock and LFTEs through the BAIs wearing PC+SCBA and exercising at a low intensity. However, the addition of heat from fire during the LFTEs did not increase blood lactate significantly when compared to the mock training exercises. This suggests the heat did not adversely affect the BAIs muscle metabolism or blood flow to the working muscles, and that the work rate between the mock training exercises and LFTEs was the same.

Blood Glucose.

The blood glucose levels prior and post exposure to the LFTEs suggest that the BAIs were neither hypoglycaemic, nor hyperglycaemic. According to Colberg (2001) a normal blood glucose range is between 4.0 mmol·Litre and 6.1 mmol·Litre, which was in agreement with the present study's findings. This suggests that exposure to the LFTEs did not significantly affect glucose levels allowing the BAIs to maintain them within the 'normal' range. Therefore, the study does not recommend any additional guidelines for the BAIs regarding the consumption of food prior to exposure to LFTEs.

Body Mass.

According to Brooks *et al* (1996), during work periods in a hot environment, losses of body mass as little as 1% may reduce an individuals working capacity and also compromise their concentration. During the LFTEs the BAIs were required to maintain concentration at all times in order to maintain a safe training environment for both themselves and the fire fighter students. Therefore, the mean losses of 1.3 (±

0.4) % of body mass lost through sweating during the LFTEs, signifies that the BAIs may have lost concentration. This may have compromised their ability to maintain a safe training environment during the time exposed to the heat. There were no significant increases in the changes (between the pre and post measurements) in time taken for the BAIs to complete the adapted Stroop test during the mock training exercises. However, there were significant increases ($p<0.05$) in the changes (between the pre and post measurements) in time taken for the BAIs to complete the adapted Stroop test during the LFTEs. The differences in time to complete the adapted Stroop test between the mock and LFTEs supports the hypothesis that losses of body weight as little as 1% affect concentration.

Haematocrit.

The present study reported an 8.1 (± 2.2) % decrease in estimated plasma volume. The original study by Van Beaumont *et al* (1972), also reported that their subjects elicited an -8.3% change in estimated plasma volume as a result of 13 days of bed rest. Other studies investigating the effects of exercise/heat and its effect on plasma volume include research undertaken by Ashenazi and Ephshtein (1998). They investigated the effects of a continuous 110 kilometre march with a 20 kg backpack load and observed a -6.7% change in plasma volume pre and post march (taking into account the amount of fluid taken on board). The 20 kg pack was the equivalent weight to the PC+SCBA that the BAIs wore, but the exercise was far greater in duration (35 (± 2.0) minutes compared to 16 hours). Ashenazi and Ephshtein (1998) reported the change in plasma volume was less than the plasma volumes reported in the present study. This suggests that the additional heat experienced by the BAIs during LFTEs (35 (± 2.0) minutes) produced a greater loss of plasma volume compared to subjects who exercised for 16 hours without the addition of heat.

However, maximal exercise protocols appear to elicit greater percentage change in plasma volume than low intensity high ambient temperatures as experienced by the BAIs. Novosadova (1977) reported a percentage change in plasma volume of -13% during maximal exercise protocols when compared to lower intensities (60% $VO_{2\ max}$) that produced a -6.7% change in plasma volume. Gore *et al* (1992) also observed a change in plasma volumes of –6.5% during thermoneutral conditions

whilst running for 60 minutes at 65% $VO_{2\ max}$. However, Jimenez *et al* (1999) observed changes in plasma volume of –11.4% when their subjects exercised at 60% of their $VO_{2\ max}$ at 30 (± 0.5) ^{0}C. These findings are supported by Fortney *et al* (1988) who also observed changes in plasma volumes of –13% when their subjects exercised at 65% of their $VO_{2\ max}$ at 30^{0}C for 30 minutes. This suggests that when both heat and heavy physical exercise are combined, there is a greater stress placed on the body through a greater loss of plasma volume. This reduction in plasma volume occurs through an increase in the sweating process and leads to a decrease in the central filling pressure. This, in turn, causes an increase in HR to compensate for the reduction in stroke volume, thus placing an increased stress on the cardiovascular system (Brooks, Fahey and White, 1996). However, the decrease in estimated plasma volume during the LFTEs was not as great as the reductions reported by Jimenez *et al* (1999), possibly due to the lower intensity of exercise the BAIs carried out during the LFTEs. This suggests that the mean haematocrit results obtained from exposure to the LFTEs (2.1 (± 1.5) % increase) and the reduction in estimated plasma volume (-8.1 (± 2.2) %) was not great enough to invoke an additional increase in cardiac stress.

Fluid Balance.

The fluid balance (obtained from the mean daily fluid consumed, subtracted from the total losses) was significantly ($p<0.05$) increased during the mock training exercises when compared to the LFTEs. This was because the sweat losses (evidenced by weight lost between the pre and post exposure measurements), as a result of exposure to the LFTEs, were not evident during the mock training exercises. The fluid consumed was not significantly different between the mock training exercises and the LFTEs. This suggests the BAIs did not perceive it necessary to alter their fluid ingestion as a result of exposure to the LFTEs, highlighting the need for increasing the education of BAIs on hydration strategies.

The mean amount of fluid lost by sweating during LFTEs was 1.08 (± 0.33) kg (lost between pre and post exposure to the LFTEs) which equated to 1083.3 (± 325.7) ml. To rehydrate successfully a quantity equivalent to 150% of the amount of fluid lost (Brooks *et al,* 1996 and Manore and Thompson, 2000) should be consumed. This is

to compensate for not only the amount of fluid lost through sweating as a result of exposure to the LFTEs, but also the amount lost through the production of urine. In order to obtain successful rehydration the BAIs would have been required to consume approximately 1625 ml of water and/or cordial (this was equivalent to 150% of the fluid lost during the days of the LFTEs). The mean daily water consumption added to the mean daily cordial consumption was only 995.8 (± 157.0) ml, suggesting the BAIs were not consuming sufficient beneficial fluid during the days of LFTEs. This meant the BAIs were returning to other duties, and indeed, would be finishing their shift in a dehydrated state (Table 5.8). When the mean fluid consumed 2 hours prior to exposure (431.3 (± 381.3) ml) was subtracted from the mean fluid lost during the LFTEs (1083.3 (± 325.7) ml), the resultant median fluid loss was 637.5 ml. In addition, the fluid balance observed on days of the LFTEs (-751.4 (± 704.6 ml) had a median fluid loss of 504 ml which suggests an additional volume of 600 ml would maintain fluid balance during the days of LFTEs.

The TBW changes (-25 (± 781.8) ml) were obtained from the changes between the beginning and end of the BAIs shifts, using the Dual Scan 2005 body water analyser (BodyStat, Isle of Man, England). The TBW losses did not support the extent of the losses obtained after the mean fluid intake had been subtracted from the mean fluid losses (-751.4 (± 704.6) ml). It was not possible to monitor body water immediately pre and post exposure to the LFTEs due to the Dual Scan 2005 over-estimating TBW (refer to Appendix C).

There were distinct differences between the mean TBW losses between the mock and LFTEs, although they were not significant. The TBW changes showed decreases between the beginning of the shift and the end of the shifts when LFTEs had been carried out. This was not consistent with the mock training exercises that showed increases in TBW between the beginning and end of the shifts. This suggests the BAIs were finishing their shifts without fully replenishing their fluid levels as a result of exposure to the LFTEs. These results suggest the BAIs did not consume adequate appropriate fluid (water and cordial) during their shifts, which placed them in a dehydrated state at the end of their shifts (Table 5.8).

During the days of the LFTEs there were differences between the changes in TBW values (observed at the beginning and end of the BAI's shift), and those demonstrated by the fluid balance (Table 5.8). This may be as a result of the increased in skin blood flow when the TBW readings were obtained. Scheltinga *et al* (1991), suggest that the method of bioelectrical impedance analysis (BIA) is indeed sensitive enough to detect changes in TBW. However, Caton *et al* (1988) observed significant increases ($p<0.05$) in TBW values when their subjects were exposed to a temperate environment (35^0C) in comparison to an ambient temperature of 14.4^0C. Caton *et al* (1988) believe the differences occurred through the amplified heat increasing peripheral skin blood flow which in turn caused an increase in the body water readings. They concluded that BIA is a useful tool for the estimation of TBW, although measurements should only be taken under well standardised conditions.

Therefore, the Dual Scan 2005 was not sensitive enough to detect changes as a result of exposure to LFTEs that resulted in an over-estimation of TBW values. This suggests the Dual Scan 2005 is not suitable to use on BAIs on days when they have been exposed to LFTEs. A more suitable method of monitoring fluid balance may be to utilise the urine colour charts.

The Fire Service Manual (2000), guidelines state BAIs should not carryout LFTEs on consecutive days, but may carry out LFTEs every other day. This break is evidently required as the BAIs completed the LFTE shifts in a state of negative fluid balance (Table 5.8). However, the fluid balance results suggest the lack of recovery in TBW by the end of the BAIs shifts was caused by the lack of hydration strategies adopted, rather than the lack of recovery time between LFTEs. This could be remedied by fluid intake being monitored more rigorously by consuming a greater percentage of water and cordial, instead of other drinks such as tea and coffee that have diuretic properties.

Psychological Parameters.

Rate of Perceived Exertion (RPE).

There were significant increases ($p<0.05$) observed between the measurements obtained pre exposure and those obtained during the mock exercises and also

between the measurements obtained during the exposure and the post exposure measurements for RPE. This suggests that wearing the PC+SCBA did increase the BAIs perception of their workload throughout the mock training exercises. However, the RPE descriptions equated to 'very light' signifying that although the PC+SCBA had an effect on the BAIs during low intensity work, they did not perceive it as overtly stressful work.

There were significant increases ($p<0.05$) in RPE values between the measurements obtained pre exposure and those obtained during the LFTEs. There were significant differences between the measurements obtained pre exposure and those obtained post exposure to the LFTEs and also between those obtained during exposure and post exposure. This suggests the increases in HR (Figures 5.2, 5.3 and 5.4) and temperature responses (Figure 5.6, 5.7 and 5.8) to the hot environment during the LFTEs, increased the BAIs perception of work rate. This is consistent with Maw, Boutcher and Taylor (1993), who reported an upward resetting of RPE responses when heat stressed.

Further evidence of this upward resetting of RPE when heat stressed was from the mean RPE responses obtained post exposure to the LFTE when compared to the RPE responses during the Step Test (whilst dressed in PC+SCBA) (refer to Chapter 4). The RPE responses show the mean level 1 RPE response was 8 (± 1) which was equivalent to the post mock RPE. However, the mean post LFTE RPE was equivalent to level 3 on the Step Test although the workload during the LFTEs was much lower (27.4 (± 3.9) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$ during level 3 of the Step Test compared to 17.3 (± 2.8) ml $O_2 \cdot kg^{-1} \cdot min^{-1}$ during the LFTEs).

The mean post LFTE RPE equated to 'somewhat hard' which was significantly higher ($p<0.05$) than the perceived post mock training exercise RPE responses. This may suggest that although the PC+SCBA adds to the stress it appears that it is the environmental temperature within the FFU that creates the greatest amount of perceived stress.

This signifies that in order to reduce the psychological stress placed on the BAIs by the heat a reduction in the amount of heat storage within the protective clothing may, in turn, reduce the amount of heat stress placed on the BAIs during LFTEs. Therefore, reducing the heat storage from within the PC may, in part, help reduce the increase in HR and temperature responses and therefore reduce the perceived increased workload and actual thermoregulatory responses that the BAIs felt during LFTEs.

Adapted Stroop Test.

The differences between the times to complete the adapted Stroop test between the measurements obtained pre exposure, during exposure and post exposure to the mock training exercises were not significant. This suggests that wearing the PC+SCBA without an adverse heated environment (during the mock training exercises) does not affect the BAIs concentration.

Figure 5.16 shows the mean times to complete the adapted Stroop during the LFTEs showing that there were significant increases in the time to complete the adapted Stroop test between pre exposure measurements and post exposure measurements. Therefore, the continuous exposure to LFTEs for a period of 35.0 (± 2.0) minutes to the heat from the fire, dehydration and the inclusion of the fire fighter students did appear to affect the BAIs concentration during LFTEs. Brooks *et al* (1996) state that losses as little as 1% of body weight will result in losses of concentration. The BAIs mean loss of body mass amounted to 1.3 (± 0.4) % which would concur the findings of Brooks *et al* (1996).

These results imply that the adapted Stroop test is a useful method in monitoring the loss of concentration in BAIs during LFTEs, when they are exposed to hot environments in the presence of students and have lost weight through the sweating process. However, even though the increase in time taken to complete the adapted Stroop was significant, the difference in time was relatively small, thus, it is unclear if the increase was significant in terms of a decrease in mental agility/cognitive function.

5.5 Conclusions.

The results show that the inclusion of the fire and students, over that of simply wearing PC+SCBA, creates significant increases in the stress placed on the BAIs during LFTEs when compared to mock training exercises in the short term. This was shown through the significant increases in HR, oxygen cost, skin, aural and micro-climate temperatures, haematocit, body mass losses and systolic blood pressure changes when the mock exercises were compared to the LFTEs.

In addition, there were significant increases in the BAIs perceived exertion when the mock values were compared to the LFTE values. This was even though the workload remained the same through the BAIs undertaking identical exercise schedules in both the mock and LFTEs. There were significant increases in the time to complete the adapted Stroop test between the pre exposure and post exposure adapted Stroop test measurements when the BAIs had been exposed to LFTEs. However, when exposed to the mock training exercises there were no significant increases in time to complete the adapted Stroop test. This suggests the addition of fire, students and dehydration during the LFTEs causes an increased loss of concentration, which was not evident during the mock training exercises.

In order to reduce these responses it is suggested that they increase the amount of water and cordial (possibly using reduced sugar cordial so gastric emptying and fluid absorption is not impeded) consumed pre exposure and post exposure. In addition, during the days the BAIs undertake LFTEs they should reduce the amount of fluid consumed that has diuretic properties.

Chapter 6.

The Physiological Responses of BAIs:

1. **When Hydrated Prior to LFTEs Compared to being Euhydrated Prior to LFTEs (Protocol 2)**

   and

2. **When Different Timings of Fluid Ingestion were Incorporated (Protocol 3).**

6.1 Introduction.

In the previous chapter some indicators of short term stress were identified. The aim of this chapter was to determine whether hydration would reduce these indicators.

The study observed the effect of the timing of fluid ingestion on the physiological and psychological responses to LFTEs. The BAIs carried out three trials to observe the different fluid ingestion timings. Firstly, the BAIs carried out LFTEs when euhydrated prior to exposure, (euhydrated being defined as 'in normal fluid balance' (Manore and Thompson, 2000)), whereby the BAIs were not hydrated or dehydrated (dehydrated defined as urine colour of 3 or greater (Armstrong *et al*, 1998)) and had not consumed any fluid for 2 hours prior to the LFTEs. The BAIs also carried out LFTEs when hydrated prior to exposure (BAIs consumed 600 ml of water 1 hour prior to the LFTEs). The BAIs were defined as being hydrated when urine colour was 1 (Armstrong *et al*, 1998) and osmolality was less than 320 mOsmols·kg^{-1}. The BAIs also carried out LFTEs when euhydrated prior to exposure but drank during exposure (BAIs drank 200 ml every 10 minutes during the LFTEs).

Therefore, this chapter seeks to confirm the results obtained in Chapter 5 but also observe the effects of either hydrating BAIs prior to exposure or administering fluid during LFTEs.

6.2 Methods.

(Refer to Methods chapter, Section 3.3)

6.3 Results.

Murray *et al* (1995) showed drinking every 5 minutes during exercise and exposure to heat was beneficial in lowering HR and RPE responses. However, this was deemed to be too intrusive during LFTEs, thus, drinking every 10 minutes was adopted.

The control results during Protocols 1, 2 and 3 have produced the same physiological responses, thus confirming the consistency of the BAIs during LFTEs.

Heart Rate (HR).

The mean and peak HR results obtained during exposure to LFTEs throughout Protocols 2 and 3 (Table 6.1), show there were no significant decreases in HR as a result of consuming water prior to, or during LFTEs, when compared to the BAIs being in a euhydrated state prior to undertaking LFTEs.

Table 6.1 – Mean HR, per cent of maximum HR, peak HR and peak HR as a per cent of maximum HR. BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) (Protocol 2), or drinking 200 ml every 10 minutes during exposure (During) (Protocol 3). Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test.

		Mean HR	% Max	Peak HR	% Max
Protocol 2	Euhydrated	115.3 (± 16.8)	65.8 (± 9.6)	173.5 (± 32.5)	98.6 (± 16.6)
	Hydrated	120.4 (± 24.0)	69.0 (± 13.4)	157.8 (± 20.1)	90.4 (± 11.6)
Protocol 3	Euhydrated	110.3 (± 15.5)	62.5 (± 8.7)	157.8 (± 12.6)	89.8 (± 6.8)
	Hydrated	115.2 (± 16.7)	65.7 (± 9.8)	155.2 (± 15.8)	88.3 (± 10.2)
	During	104.7 (± 25.1)	89.3 (± 15.2)	138.7 (± 33.7)	79.1 (± 20.9)

Table 6.1 shows that the mean HR responses during Protocol 3, when the BAIs were euhydrated prior to exposure and hydrated prior to exposure, produced similar HR responses to that of the BAIs throughout Protocol 2 (when euhydrated and hydrated prior to undertaking LFTEs).

There were no significant changes in the mean HR between the 3 trials. However, the HR was significantly ($p<0.05$) decreased (by -23.2 (± 13.8) %) when BAIs exited the FFU to drink, compared to the HR obtained at the equivalent times during the LFTEs

when BAIs were euhydrated prior to exposure (increased by 28.1 (± 28.3) %) (Table 6.2).

Table 6.2 – Mean change in HR when BAIs exited the FFU to drink during LFTEs, compared to the HR obtained at the equivalent times during the LFTEs when BAIs were euhydrated prior to exposure. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test. * - significant differences ($p<0.05$) between the euhydrated and during LFTEs HR responses.

	HR (Beats·minute^{-1})	HR (% change)
Euhydrated	27.7 (± 24.3)	28.1 (± 28.3)
During	-32.3 (± 27.8)*	-23.2 (± 13.8)*

Oxygen Cost obtained during mock training exercises and LFTEs.

There were small but significantly higher ($p<0.05$) mean oxygen costs produced during LFTEs when BAIs were hydrated prior to exposure, compared to when BAIs were euhydrated. There were no significant differences between any of the fluid timing trials for the mean oxygen cost during Protocol 3. However, there were no significant differences in the percentage of maximum oxygen cost between the LFTEs when BAIs were hydrated prior to exposure or euhydrated, or when the BAIs drank during the LFTEs throughout Protocols 2 and 3 (Figure 6.1). To ensure there were no differences between the workload carried out by BAIs during LFTEs, BAIs followed the same schedule during both the LFTEs when euhydrated or hydrated prior to exposure (refer to Table 3.2 in the Methods).

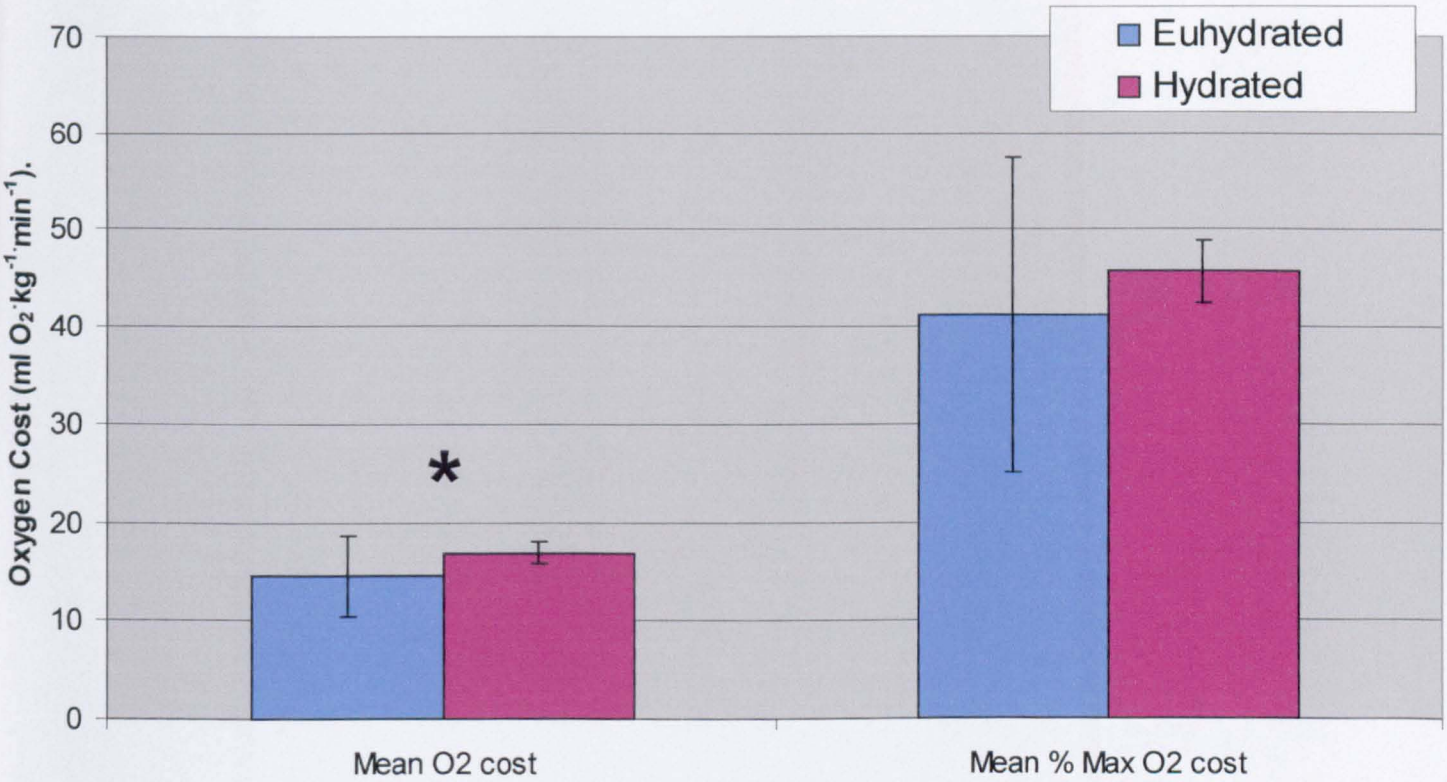


Figure 6.1 – Mean oxygen cost and percentage of maximum oxygen cost during Protocol 2. BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated) or consuming 600 ml of fluid 1 hour prior to exposure (Hydrated). Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test. * - significant differences ($p<0.05$) between the euhydrated and hydrated oxygen cost readings.

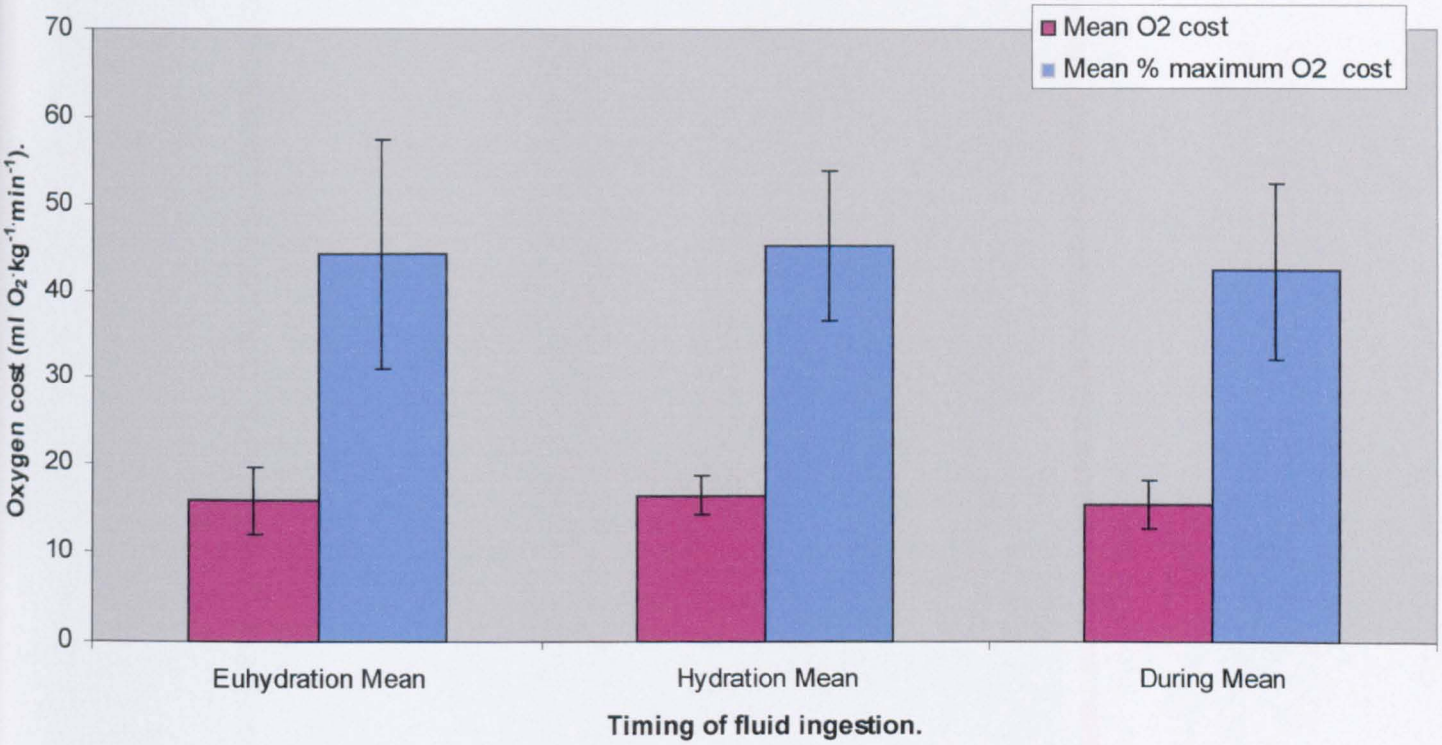


Figure 6.2 – Mean oxygen cost and percentage of maximum oxygen cost during Protocol 3. BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During). Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA.

Temperature.

Temperature outside the FFU.

During Protocol 2 there were small but significant increases in the temperature outside the FFU during LFTEs when BAIs were hydrated prior to exposure (19.8 (± 2.5) ^{0}C) compared to when they were euhydrated prior to exposure (17.7 (± 2.4) ^{0}C) and also inside the FFU (116 (± 18.5) ^{0}C when BAIs were euhydrated prior to exposure and 133.9 (± 27.7) ^{0}C) when hydrated prior to exposure). However, there was little physiological evidence to suggest this significantly affected the BAIs during the LFTEs. In Protocol 3 there were no significant differences in temperature outside the FFU between drinking prior to exposure (16.3 (± 1.1) ^{0}C) compared to when the BAIs were euhydrated prior to exposure (16.3 (± 1.8) ^{0}C) or the environmental temperature inside the FFU (Figure 6.3) when hydrated prior to exposure (54.5 (± 7.3) ^{0}C in the anti-chamber and 120.8 (± 21.3) ^{0}C in the fire chamber) and when euhydrated prior to exposure (57.2 (± 7.1) ^{0}C in the anti-chamber and 111.0 (± 20.6) ^{0}C in the fire chamber).

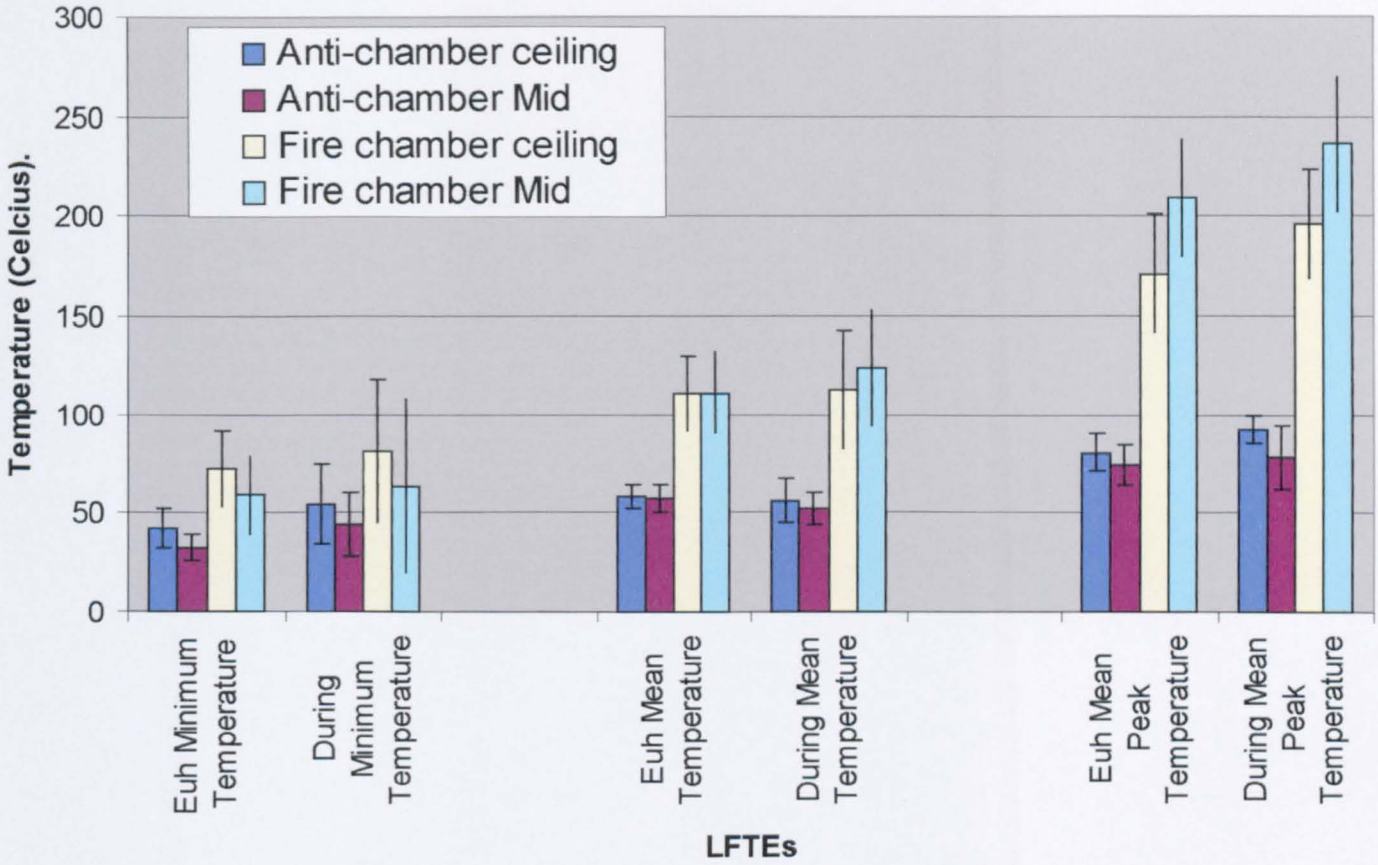


Figure 6.3 – Mean temperatures within the FFU during the LFTEs (Protocol 3). Temperature readings were obtained in the anti-chamber (room within the FFU that did not contain fire, and was where the debriefs occurred) and the fire chamber (the compartment in the FFU where the fire was ignited and where the fire fighting occurred). Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA.

As was consistent with Chapter 5, Figures 6.4, 6.5 and 6.6 show significant increases ($p<0.05$) in skin, aural and micro-climate temperature respectively between the pre exposure gym kit and pre exposure PC+SCBA during Protocols 2 and 3. During such protocols these temperatures were all significantly increased ($p<0.05$) further during exposure to both LFTEs when BAIs were either hydrated or euhydrated prior to exposure, or drank during the training exercises. There were no significant differences in the BAIs skin (Figure 6.4), aural (Figure 6.5) or micro-climate (Figure 6.6) temperatures during Protocols 2 and 3 when the BAIs were either hydrated or euhydrated prior to exposure, or drank during the LFTEs. The skin, aural and micro-climate temperature responses were similar with those produced in Protocol 1 (Chapter 5).

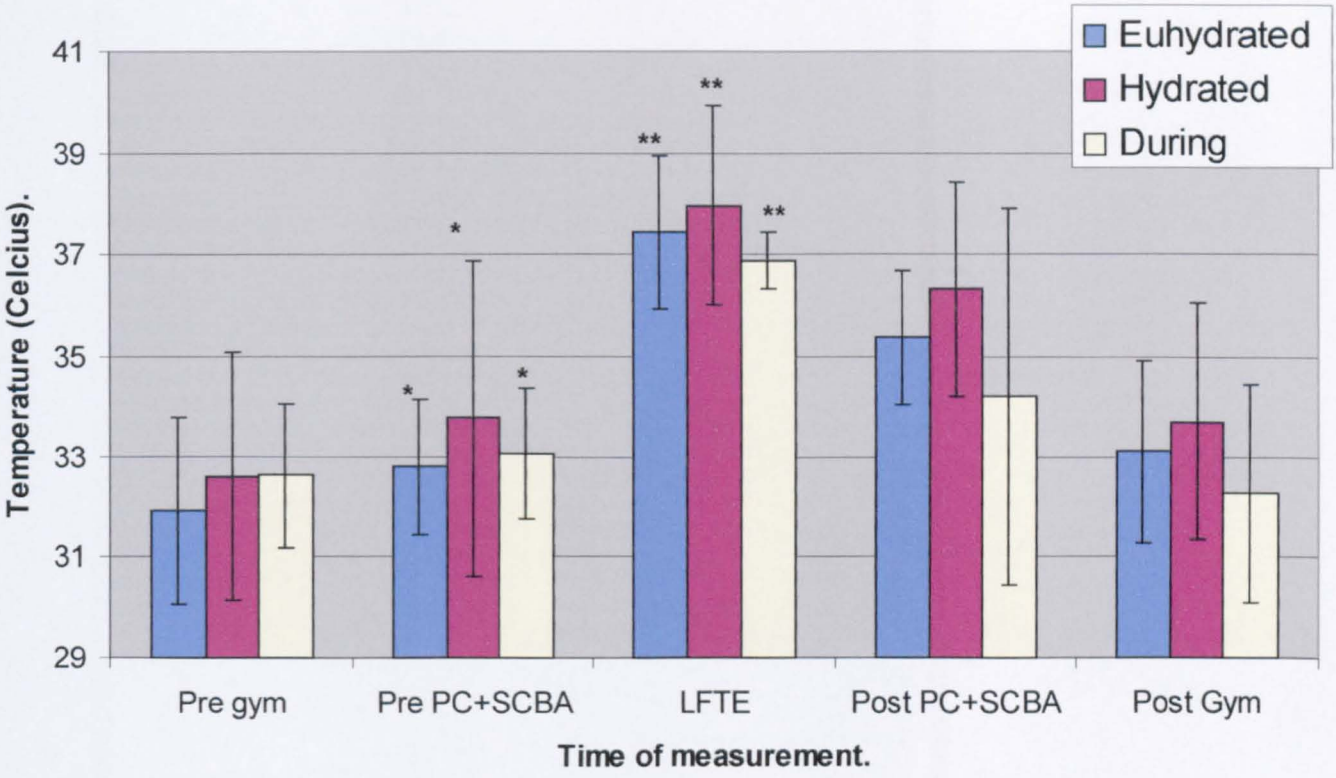


Figure 6.4 – Mean skin temperatures pre exposure (dressed in gym kit (Pre gym) and PC+SCBA (Pre PC+SCBA)), during exposure (LFTE (Live fire training exercise)) and post exposure dressed in PC+SCBA (Post PC+SCBA) and also gym kit (Post gym kit) (Protocols 2 and 3). Values were the means (± SD) obtained over 5 minutes for all pre and post readings. During the LFTEs the data is the mean (± SD) of a 34 minute exposure (n = 6). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre gym kit and pre PC+SCBA skin temperatures. ** - significant differences ($p<0.05$) between the pre PC+SCBA and the LFTEs skin temperatures.

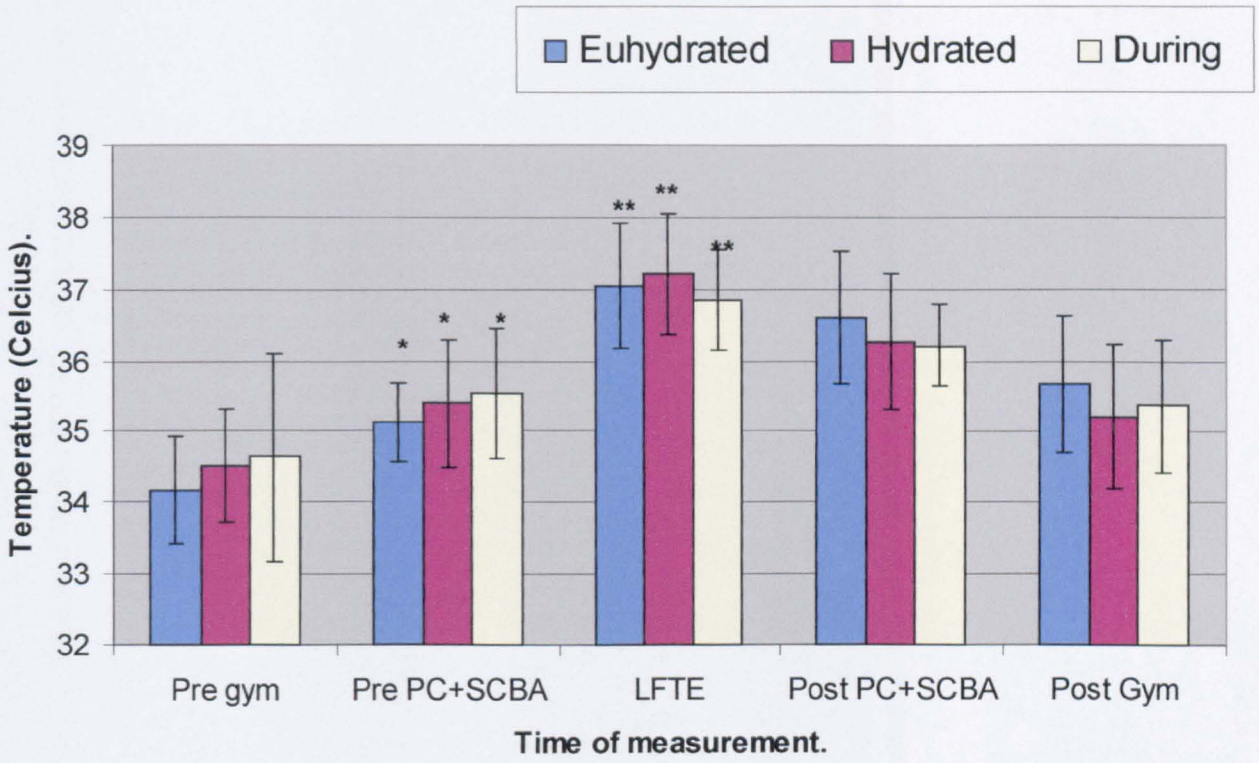


Figure 6.5 – Mean aural temperatures pre exposure (dressed in gym kit (Pre gym) and PC+SCBA (Pre PC+SCBA)), during exposure (LFTE (Live fire training exercise)) and post exposure dressed in PC+SCBA (Post PC+SCBA) and also gym kit (Post gym kit) (Protocols 2 and 3). Values were the means (± SD) obtained over 5 minutes for all pre and post readings. During the LFTEs the data is the mean (± SD) of a 34 minute exposure (n = 6). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre gym kit and pre PC+SCBA aural temperatures. ** - significant differences ($p<0.05$) between the pre PC+SCBA and the LFTEs aural temperatures.

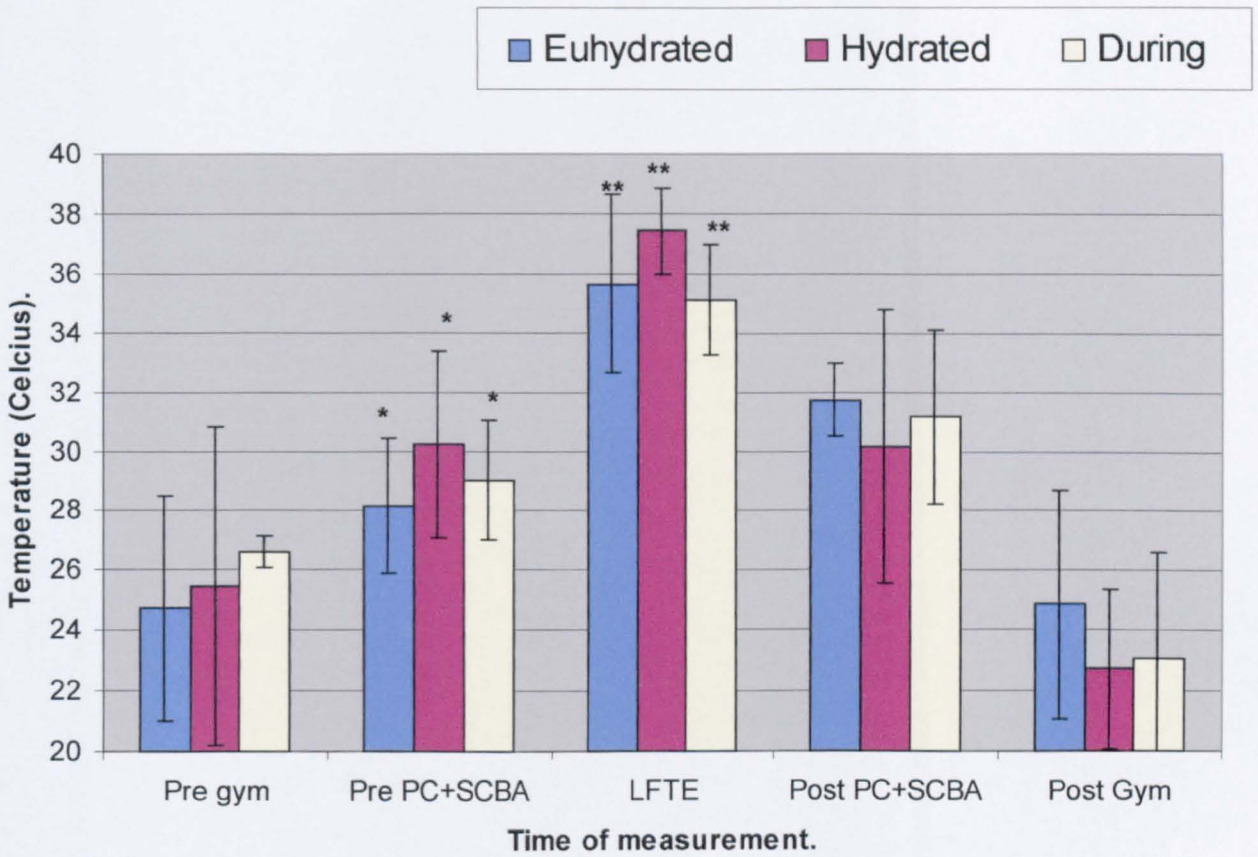


Figure 6.6 – Mean micro-climate temperatures pre exposure (dressed in gym kit (Pre gym) and PC+SCBA (Pre PC+SCBA)), during exposure (LFTE (Live fire training exercise)) and post exposure dressed in PC+SCBA (Post PC+SCBA) and also gym kit (Post gym kit) (Protocols 2 and 3). Values were the means (± SD) obtained over 5 minutes for all pre and post readings. During the LFTEs the data is the mean (± SD) of a 34 minute exposure (n = 6). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre gym kit and pre PC+SCBA micro-climate temperatures. ** - significant differences ($p<0.05$) between the pre PC+SCBA and the LFTEs micro-climate temperatures.

Figure 6.7 shows the reductions in skin and micro-climate temperature when BAIs exited the FFU to consume fluid (at points A, B and C) during Protocol 3.

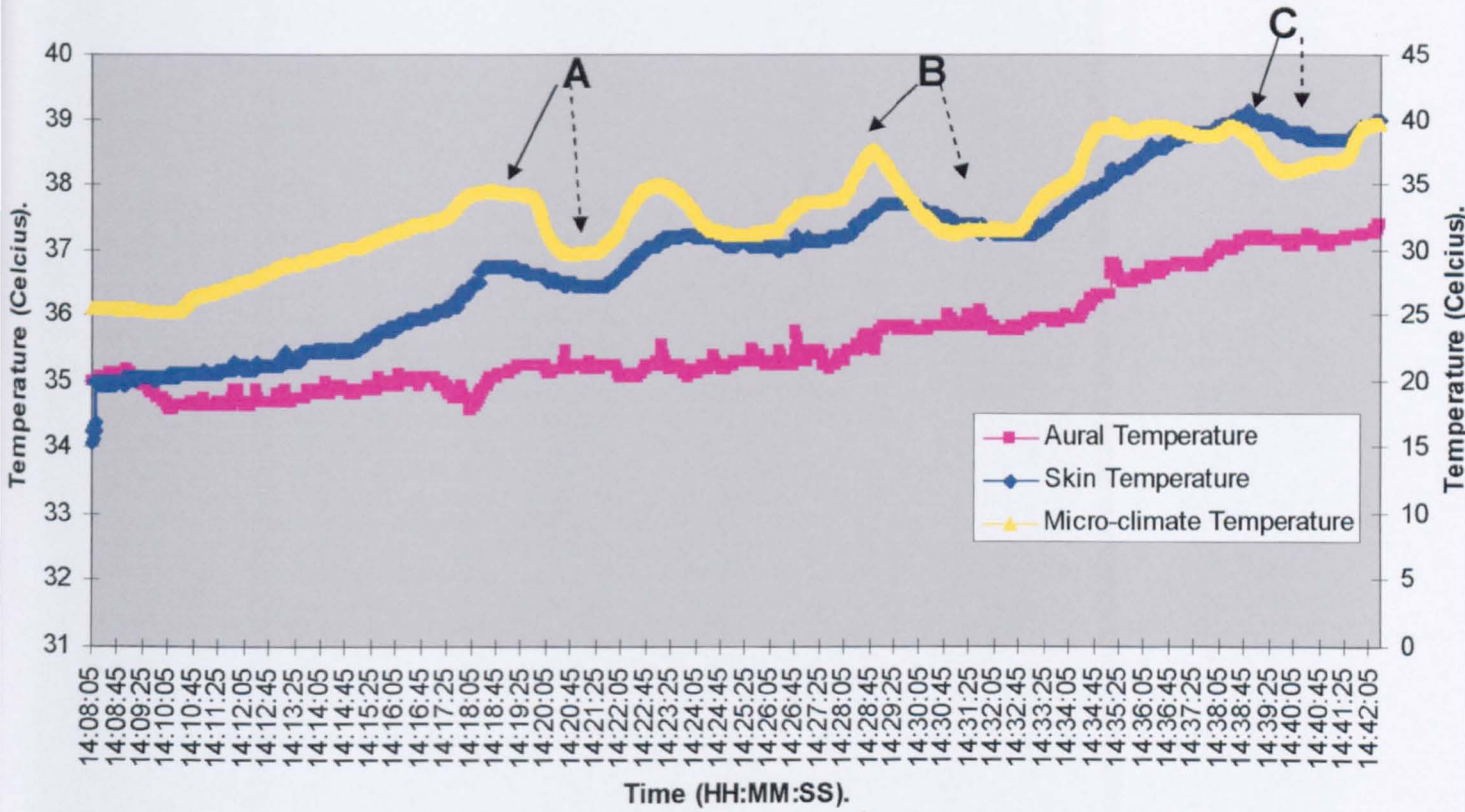


Figure 6.7 – Typical temperature responses to a LFTE when the BAI exited the FFU to consume 200 ml of fluid (Protocol 3).

A ——→ Denotes the first time that the BAI exited the FFU to drink 200 ml of fluid.
A - - - - → Denotes the BAIs re-entry back into the FFU.
B ——→ Denotes the second time that the BAI exited the FFU to drink 200 ml of fluid.
B - - - - → Denotes the BAIs re-entry back into the FFU.
C ——→ Denotes the third time that the BAI exited the FFU to drink 200 ml of fluid.
C - - - - → Denotes the BAIs re-entry back into the FFU.

There were significant ($p<0.05$) reductions in skin temperature (-0.7 (± 0.9) % decrease) and micro-climate temperature (-4.2 (± 4.3) % decrease) between the intervals when BAIs exited the LFTEs to consume fluid and re-entered the FFU. This was compared to the equivalent times during the LFTEs when BAIs were euhydrated prior to exposure and produced a 1.2 (± 1.1) % increase in skin temperature and 1.5 (± 2.3) % increase in micro-climate temperature.

Blood Pressure.

During Protocols 2 and 3, systolic blood pressure was significantly increased ($p<0.05$) between the pre and post measurements for the LFTEs when BAIs were euhydrated or hydrated prior to exposure, or drank during the LFTEs (Figure 6.8). There were no significant differences in systolic pressure between any of the fluid trials.

There were no significant differences between the pre exposure diastolic blood pressure readings and the post exposure diastolic blood pressure readings for any of the fluid trials. There were no significant differences in the changes between the pre exposure and post exposure readings when BAIs were either hydrated or euhydrated prior to exposure, or drank during the LFTEs (Figure 6.8) during Protocols 2 and 3. Both the diastolic and systolic blood pressure responses were consistent with the findings produced in Protocol 1.

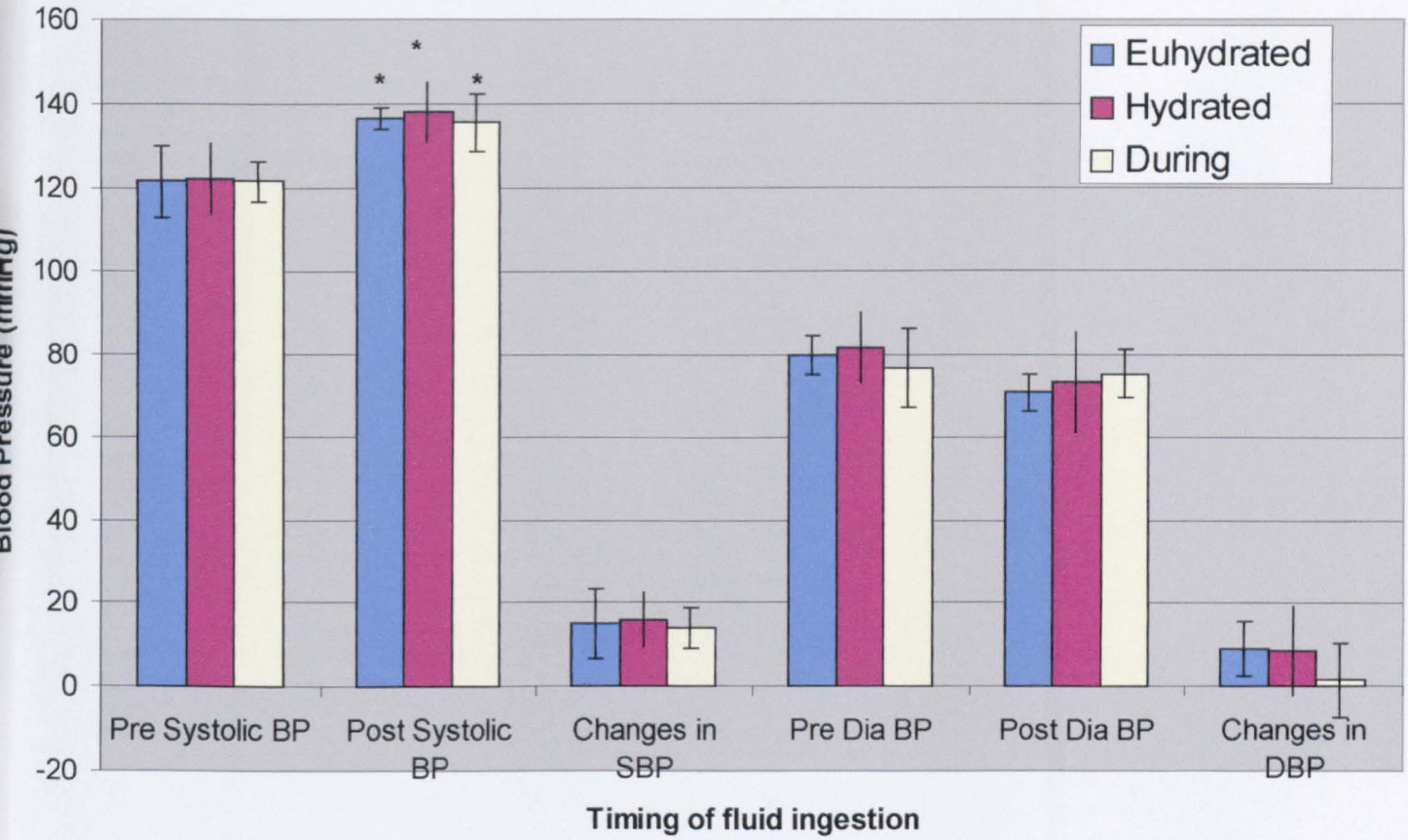


Figure 6.8 – Mean and mean changes in blood pressure (BP) in response to live fire training exercises (LFTEs). Blood pressure was obtained pre exposure (Pre systolic BP and Pre diastolic BP) and post exposure (Post systolic BP and Post diastolic BP). Changes between the pre exposure and post exposure blood pressure values for systolic (change in SBP) and diastolic (change in DBP) blood pressure were obtained. BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocols 2 and 3). Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA with post Tukey. * - significant differences ($p<0.05$) between the pre and post LFTE systolic blood pressure readings.

Blood Lactate.

During Protocol 2 and 3, blood lactate values showed significant increases ($p<0.05$) between the pre exposure and post exposure measurements when BAIs were either euhydrated or hydrated prior to exposure, or drank during the LFTEs (Figure 6.9). These increases were consistent with the blood lactate results observed during Protocol 1. There were no significant differences between the changes between pre and post readings in blood lactate between the LFTEs when BAIs were either hydrated or euhydrated prior to exposure, or drank during the LFTEs (Figure 6.9).

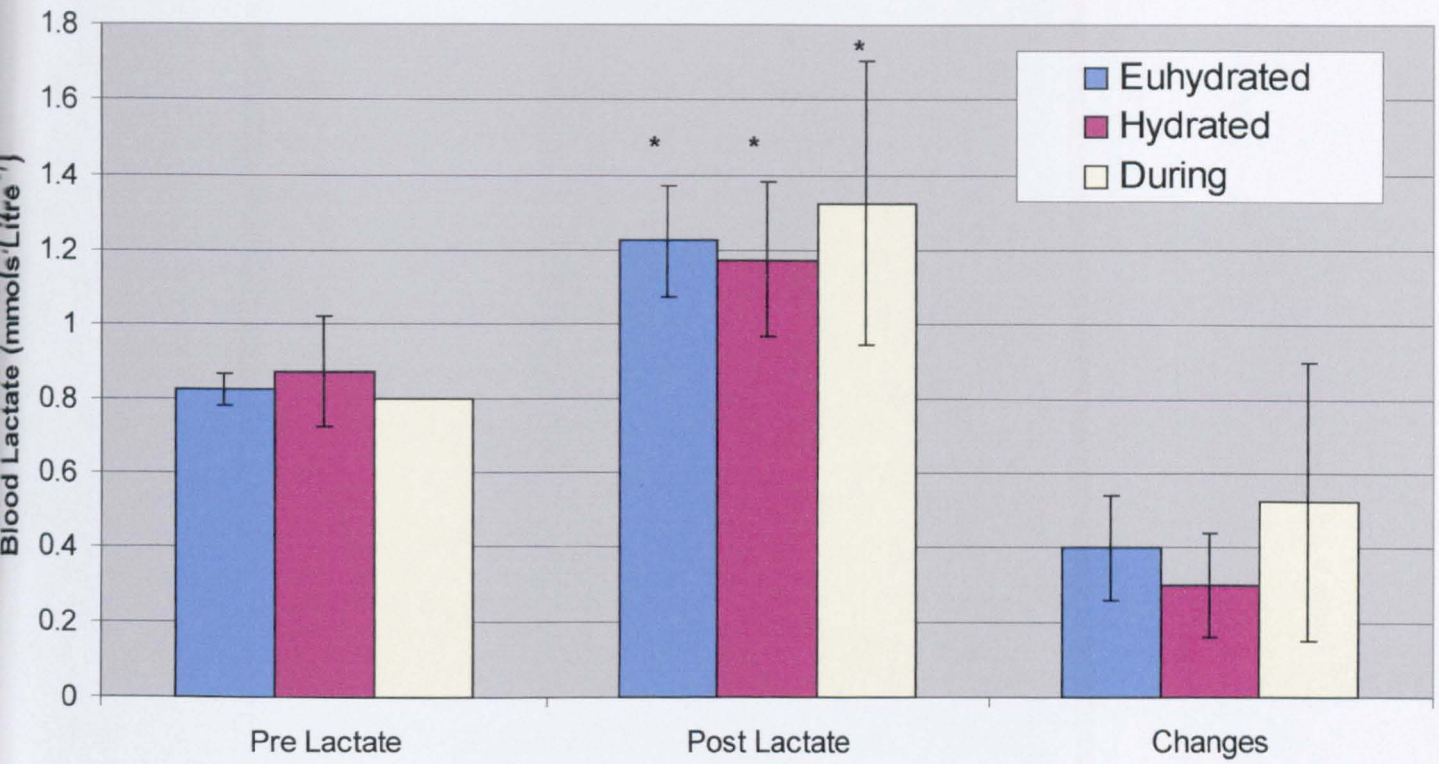


Figure 6.9 – Mean and mean changes in blood lactate in response to live fire training exercises (LFTEs). Blood lactate was obtained pre exposure (Pre Lactate) and post exposure (Post Lactate). Changes between the pre exposure and post exposure blood lactate values (changes) were obtained. BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) (Protocol 2) or drinking 200 ml every 10 minutes during exposure (During) (Protocol 3). Data is presented as the mean and ± SD (n= 6). Significance was tested using a one way ANOVA with post hoc Tukey. * - significant differences ($p<0.05$) between the pre and post blood lactate readings.

Body Mass.

Mean post body mass was significantly ($p<0.05$) decreased when compared to pre body mass for the LFTEs when BAIs were euhydrated (-1.3 (± 1.1) kg) prior to exposure, hydrated prior to exposure (-0.8 (± 0.3) kg) and were euhydrated prior to exposure but drank during the LFTEs (-0.5 (± 0.3) kg) (Figure 6.10). These results are consistent with the findings of Protocol 1 (-1.1 (± 0.3) kg). There were no significant differences in the body mass losses between any of the fluid trials during Protocols 2 or 3 respectively.

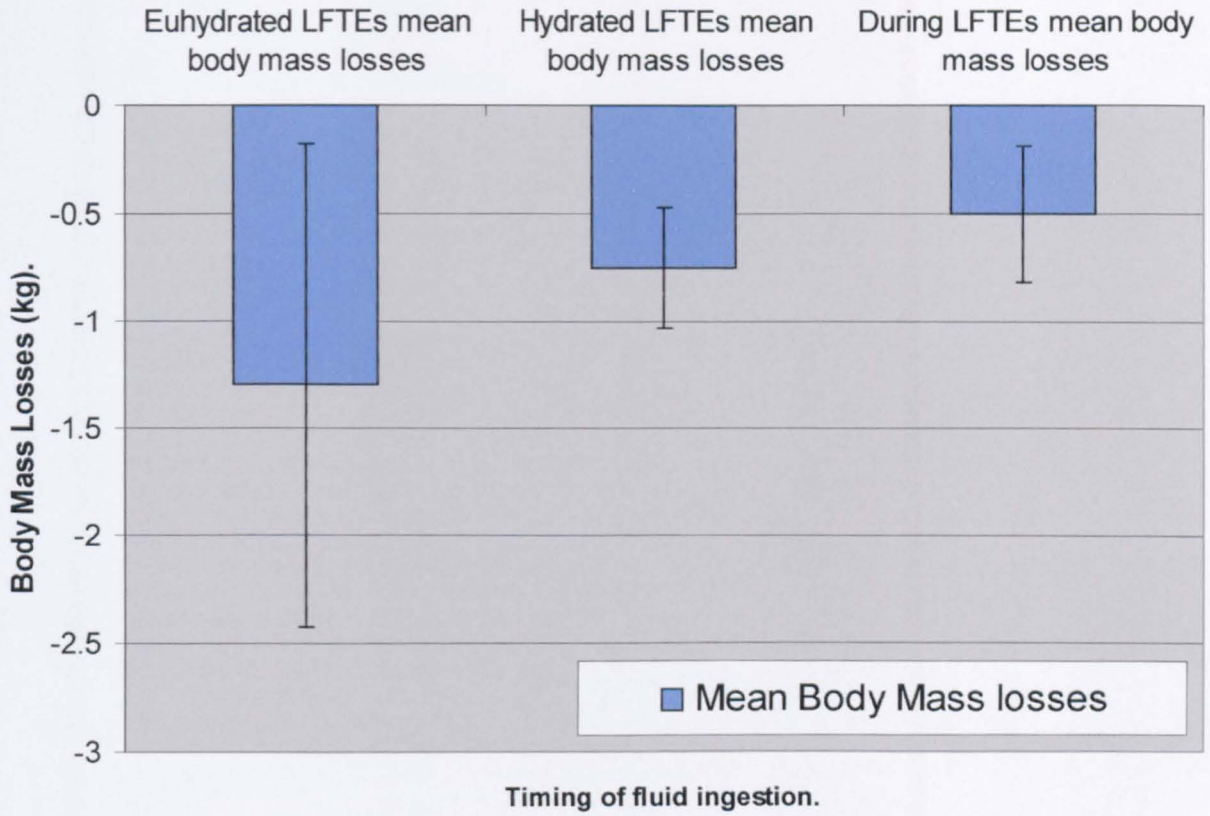


Figure 6.10 – Mean change in body mass between the pre exposure and post exposure readings when exposed to LFTEs. BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocols 2 and 3). Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA.

Haematocrit.

When the BAIs were either hydrated prior to exposure, euhydrated or drank during the LFTEs there were significant increases ($p<0.05$) in haematocrit values when the pre exposure and post exposure haematocrit values were compared (Figure 6.11). However, there were no significant differences in the changes between the pre and post exposure haematocrit values for any of the fluid trials during Protocols 2 or 3. In addition, there were no significant differences between the plasma volume changes when BAIs were hydrated prior to exposure, euhydrated or drank during LFTEs (Table 6.3).

Table 6.3 – Mean percent changes in estimated plasma volume (using the equation from Van Beaumont *et al* (1972)) as a result of exposure to LFTEs whilst incorporating different timings of fluid ingestion for Protocols 1, 2 and 3. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test.

	Euhydrated	Hydrated	During
Mean % Change in Plasma Volume (Protocol 1)	-8.1 (± 2.2)	-	-
Mean % Change in Plasma Volume (Protocol 2)	-9.1 (± 4.2)	-9.2 (± 5.2)	-
Mean % Change in Plasma Volume (Protocol 3)	-8.6 (± 4.0)	-8.3 (± 5.4)	-7.4 (± 3.4)

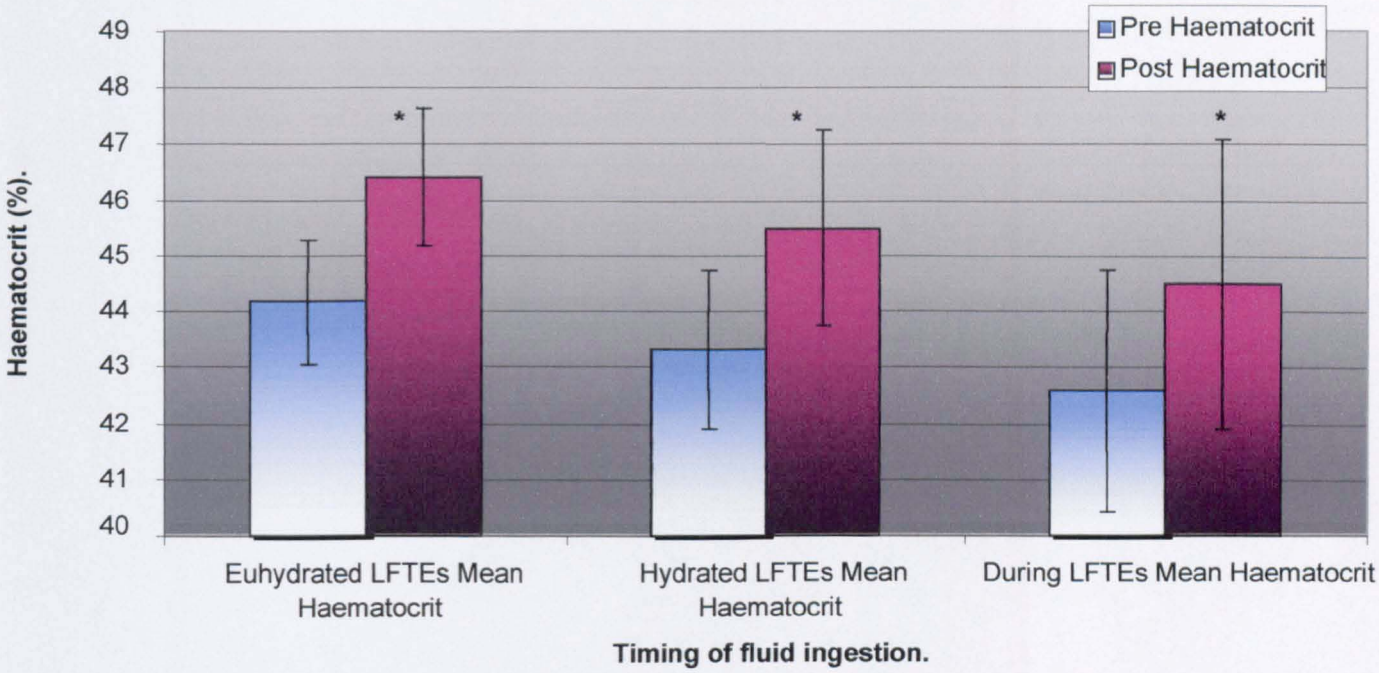


Figure 6.11 – Mean haematocrit in response to live fire training exercises (LFTEs). Haematocrit was obtained pre exposure (Pre Haematocrit) and post exposure (Post Haematocrit). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocols 2 and 3). Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with Post Hoc Bonferroni. * - significant differences ($p<0.05$) between the pre and post haematocrit readings.

Fluid balance.

The mean urine results obtained prior to the LFTEs are summarised in Table 6.3. The urine colour and osmolality results obtained prior to the LFTEs when BAIs were hydrated prior to exposure, showed that the BAIs were indeed hydrated, (with a urine colour of 1 and mean urine osmolality less than 320 mOsmols·kg^{-1}). BAIs were defined as euhydrated prior to exposure when their urine colour was 2. However, they were not dehydrated as the urine colour readings would have been between 3 and 8 (Armstrong *et al*, 1998).

As in Protocol 1, Table 6.4 shows significant increases ($p<0.05$) between pre and post exposure for urine osmolality and urine colour when the BAIs were euhydrated prior to exposure. When BAIs were hydrated prior to exposure there were significant ($p<0.05$) increases in urine osmolality and colour and significant ($p<0.05$) decreases in urine volume between pre and post exposure. There were no significant changes in urine osmolality, volume or colour between pre and post exposure when BAIs drank during LFTEs.

There were significant decreases ($p<0.05$) in the pre exposure urine colour between the LFTEs when BAIs were hydrated or euhydrated prior to exposure. This substantiates that the BAIs consumed 600 ml of water prior to exposure. There were no other significant differences between the trials for urine osmolality, volume and colour.

When BAIs were hydrated prior to exposure there were significant increases ($p<0.05$) in the post exposure urine colour and osmolality. This suggests that the fluid consumed prior to exposure (600 ml of water) was conserved within the BAIs body's as it was not being produced post exposure. This was evidenced by the significant increases ($p<0.05$) in urine colour (117.0 (± 68.3) %) and in osmolality (420.0 (± 605.9) %) and significant decrease ($p<0.05$) in urine volume (-42.0 (± 13.4) %).

Table 6.4 – Mean urine osmolality, volume and colour obtained pre exposure and post exposure to the LFTEs. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between pre exposure urine colour when the BAIs were either hydrated or euhydrated prior to exposure. ** – significant differences ($p<0.05$) between pre and post exposure measurements.

	Urine Osmolality	Urine Volume	Urine Colour
Pre Euhydrated LFTEs	394.2 (± 293.8)	262.5 (± 80.2)	2.0 (± 1.0)*
Pre Hydrated LFTEs	214.5 (± 194.0)	395.8 (± 222.7)	1.0 (± 0.5)
Pre 'During' LFTEs	475.3 (± 364.2)	283.3 (± 68.3)	2.0 (± 0.8)
Post Euhydrated LFTEs	606.8 (± 346.3)**	250.0 (± 100.0)	3.0 (± 0.8)**
Post Hydrated LFTEs	410.2 (± 182.7)**	212.5 (± 86.2)**	3.0 (± 0.5)**
Post 'During' LFTEs	361.2 (± 312.6)	338.3 (± 145.7)	2.0 (± 0.8)

When the BAIs were either euhydrated prior to exposure, hydrated or were euhydrated prior to exposure but drank during the LFTEs, they consumed, on average, a daily amount of fluid equating to 1678.3 (± 635.7) ml, 2433.3 (± 937.4) ml and 2050.8 (± 488.8) ml respectively (Table 6.5). The typical fluids consumed by the BAIs were water, cordial, tea, coffee and lemonade.

Table 6.5 – Mean daily fluid consumed (mean of the sum of daily fluid consumed), urine produced (mean of the sum of daily urine produced), sweat loss (mean sweat lost (obtained from body mass lost) as a result of exposure to the LFTEs), total fluid losses (obtained from the mean daily urine produced and sweat losses through exposure to the LFTEs added together), fluid balance (mean daily fluid consumed subtracted from the total losses), mean urine osmolality and mean urine colour; intracellular water (ICW), extracellular water (ECW) and total body water (TBW) changes (Dual Scan 2005 (Bodystat, Isle of Man, England)). Body water changes were obtained from the changes between the readings taken in the morning and at the end of the shifts of the days when the BAIs were exposed to the LFTEs. Data is presented as the mean ± SD (n= 6). Significance was tested using a paired samples t-test.

	Euhydrated	Hydrated	Drinking During
Mean Daily Fluid Consumed (ml)	1678.3 (± 635.7)	2433.3 (± 937.4)	2050.8 (± 488.8)
Urine Produced (ml)	1354.2 (± 575.4)	1529.2 (± 844.7)	1330 (± 356.3)
Sweat losses through exposure to the LFTEs (ml)	1333.3 (± 1125.5)	750 (± 273.9)	500 (± 316.2)
Total losses (ml)	-2687.5 (± 1216.3)	-2279.2 (± 828.4)	-1830.0 (± 190.9)
Fluid Balance (ml)	-1009.2 (± 1629.6)	+159.2 (± 696.0)	+220.8 (± 448.4)
Body water changes between the beginning and end of the shift on the days of LFTEs.			
ECW changes (ml)	-383.3 (± 116.9)	1183.3 (± 1794.9)	1316.6 (± 1975.2)
ICW changes (ml)	-100.0 (± 209.8)	500.0 (± 3679.1)	2500.1 (± 3397.6)
TBW changes (ml)	-483.3 (± 263.9)	1683.3 (± 5122.7)	3816.7 (± 5016.1)

Table 6.5 shows the amount of daily fluid consumed (water, cordial, tea, coffee and lemonade), the fluid lost from sweating when exposed to LFTEs and also the fluid lost when urine was produced, which together represents the BAIs fluid balance. The fluid balance showed that the BAIs were on average in negative fluid balance by -1009.2 (± 1629.6) ml during the days when the BAIs were euhydrated prior to the LFTEs. However, during days when the BAIs were hydrated prior to exposure or were euhydrated prior to exposure but drank during the LFTEs, the BAIs were in positive fluid balance by 159.2 (± 696.0) ml and 220.8 (± 448.4) ml respectively. There were no significant increases in the fluid balance on the days when the BAIs were either euhydrated prior to the LFTEs (-1009.2 (± 1629.6) ml), hydrated prior to

the LFTEs (159.2 (± 696.0) ml) or when the BAIs were euhydrated prior to exposure but drank during the LFTEs (220.8 (± 448.4) ml).

The mean changes in body water were obtained from the differences between the measurements recorded at the commencement of the shift and those recorded at the end. The mean changes in ECW showed significant decreases ($p<0.05$) during the days when BAIs were euhydrated prior to LFTEs. The mean changes in TBW, obtained on the days of the LFTEs when the BAIs were euhydrated prior to exposure where significantly decreased ($p<0.05$). There were no significant differences in the mean changes in either ECW or TBW during the days BAIs were hydrated prior to exposure or euhydrated prior to exposure but drank during LFTEs. In addition, there were no significant differences in the changes in ICW measurements between the days when BAIs were euhydrated or hydrated prior to exposure, or drank during the LFTEs.

Psychological Responses to mock training exercise and LFTEs.

Rate of Perceived Exertion (RPE).

During Protocols 2 and 3, the mean RPE responses were significantly increased ($p<0.05$) when the pre exposure (obtained before exposure to LFTEs) and during exposure (obtained 15 minutes into the LFTEs) responses were compared for all fluid trials. When the BAIs were either euhydrated or hydrated prior to exposure there were significant ($p<0.05$) increases in the RPE responses obtained during the LFTEs, compared to the post exposure responses (Figure 6.12). In addition, the mean post exposure RPE responses were significantly higher ($p<0.05$) when compared to the pre exposure RPE responses for all fluid trials.

There were no significant differences in the mean changes (between the pre exposure and post exposure responses) in the RPE responses between LFTEs when BAIs were euhydrated prior to exposure, hydrated prior to exposure or drank during LFTEs throughout any of the fluid trials.

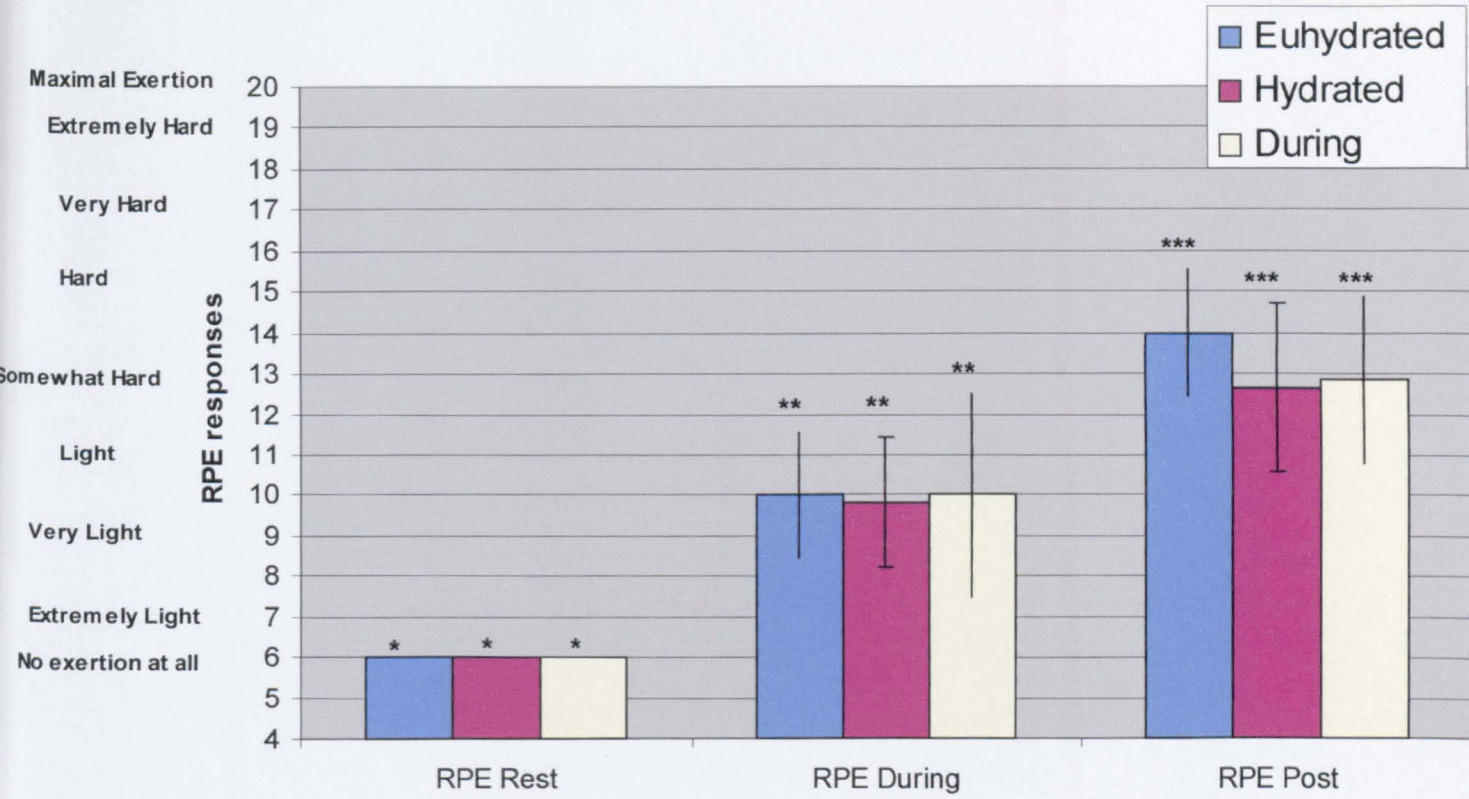


Figure 6.12 – Mean and mean change (between the pre and post exposure measurements) in perceived exertion ratings pre (carried out immediately prior to entering the FFU), during (carried out at the mid-point which was approximately 15 minutes into the LFTE) and post (carried out immediately after leaving the FFU) exposure to the LFTEs. BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocols 2 and 3). Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA with post hoc Tukey. * - significant differences ($p<0.05$) between the pre and post exposure readings. ** - significant differences ($p<0.05$) between the pre and during exposure readings. * - significant differences ($p<0.05$) between the during and post exposure readings.**

The Adapted Stroop Test

Figure 6.13 shows that during Protocol 2, whereby BAIs were euhydrated prior to exposure to the LFTEs, there were significant increases ($p<0.05$) in the time to complete the adapted Stroop test between the pre exposure measurements and post exposure measurements. This was consistent with Protocol 1.

When BAIs were hydrated prior to the LFTEs there were significant increases ($p<0.05$) in time to complete the adapted Stroop test between the pre exposure and during exposure measurements. However, there were no significant differences between the pre exposure measurements and post exposure measurements, nor were there significant differences between the measurements obtained during exposure and the post exposure measurements.

When BAIs were either hydrated or euhydrated prior to exposure there were no significant differences in the time to complete the adapted Stroop test between the LFTEs. This shows that by hydrating BAIs prior to exposure, did not abate the loss of concentration.

Figure 6.14 shows that during Protocol 3, when BAIs were euhydrated prior to exposure, there were significant increases ($p<0.05$) in the time to complete the adapted Stroop test between the pre exposure measurements and the post exposure measurements. This was consistent with the results observed in Protocols 1 and 2. There were no significant differences in the time to complete the adapted Stroop test between the pre exposure measurements and the measurements obtained during the LFTEs. Also there were no significant differences between the measurements obtained during the LFTEs and the post exposure measurements.

There were no significant differences in the time to complete the adapted Stroop test between any of the fluid ingestion trials, suggesting that fluid ingestion prior to exposure or during exposure did abate the loss of concentration.

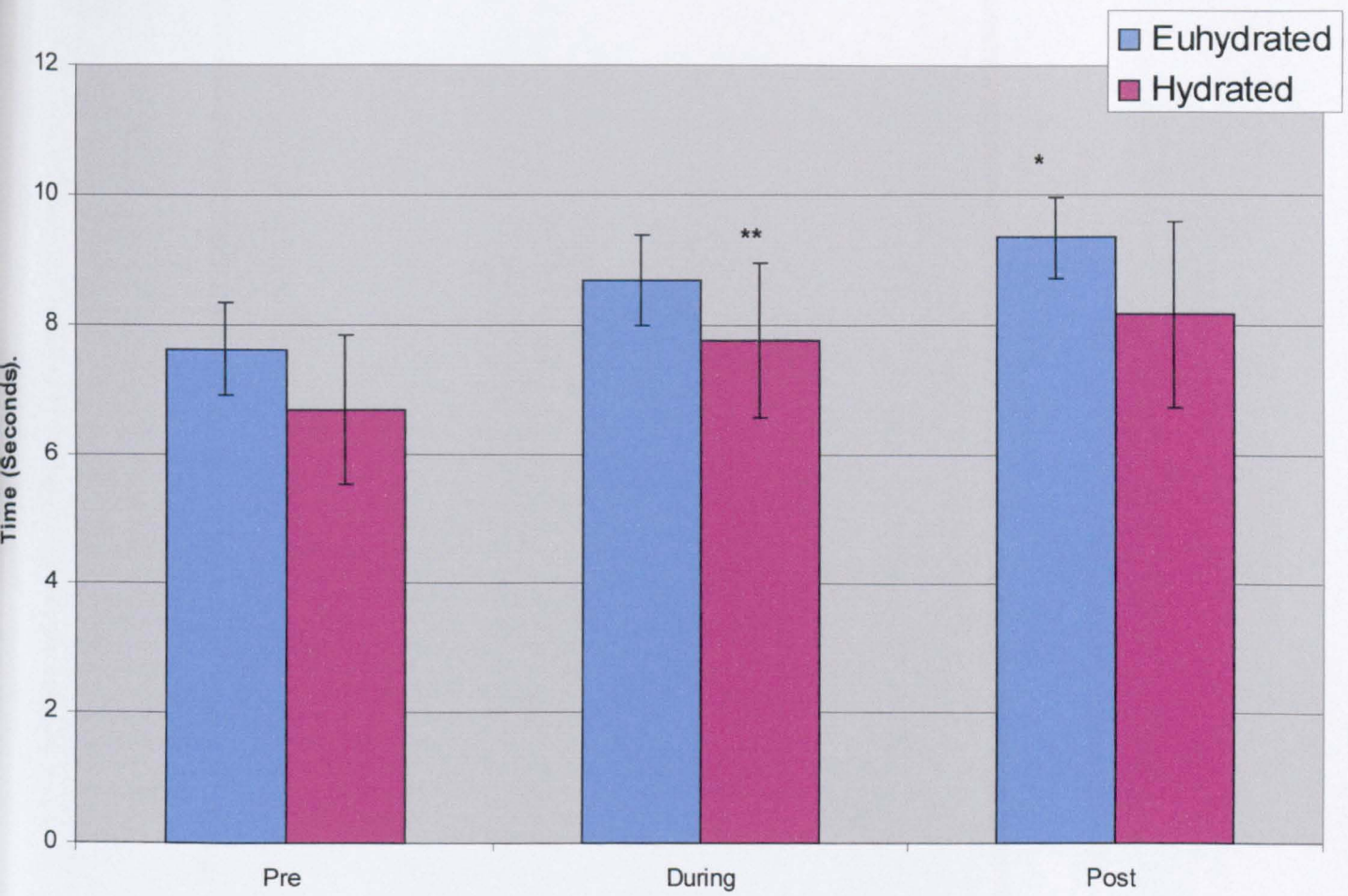


Figure 6.13 – Mean time to complete the adapted Stroop test pre (carried out immediately prior to entering the FFU), during (carried out at the mid-point which was approximately 15 minutes into the LFTE) and post (carried out immediately after leaving the FFU) exposure to the LFTE (Protocol 2). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated) or consuming 600 ml of fluid 1 hour prior to exposure (Hydrated). Data is presented as the mean ± SD (n = 6). Significance was tested using a one way ANOVA with post hoc Tukey. * - significant differences ($p<0.05$) between the pre and post euhydrated readings. ** - significant differences ($p<0.05$) between the pre and during hydrated readings.

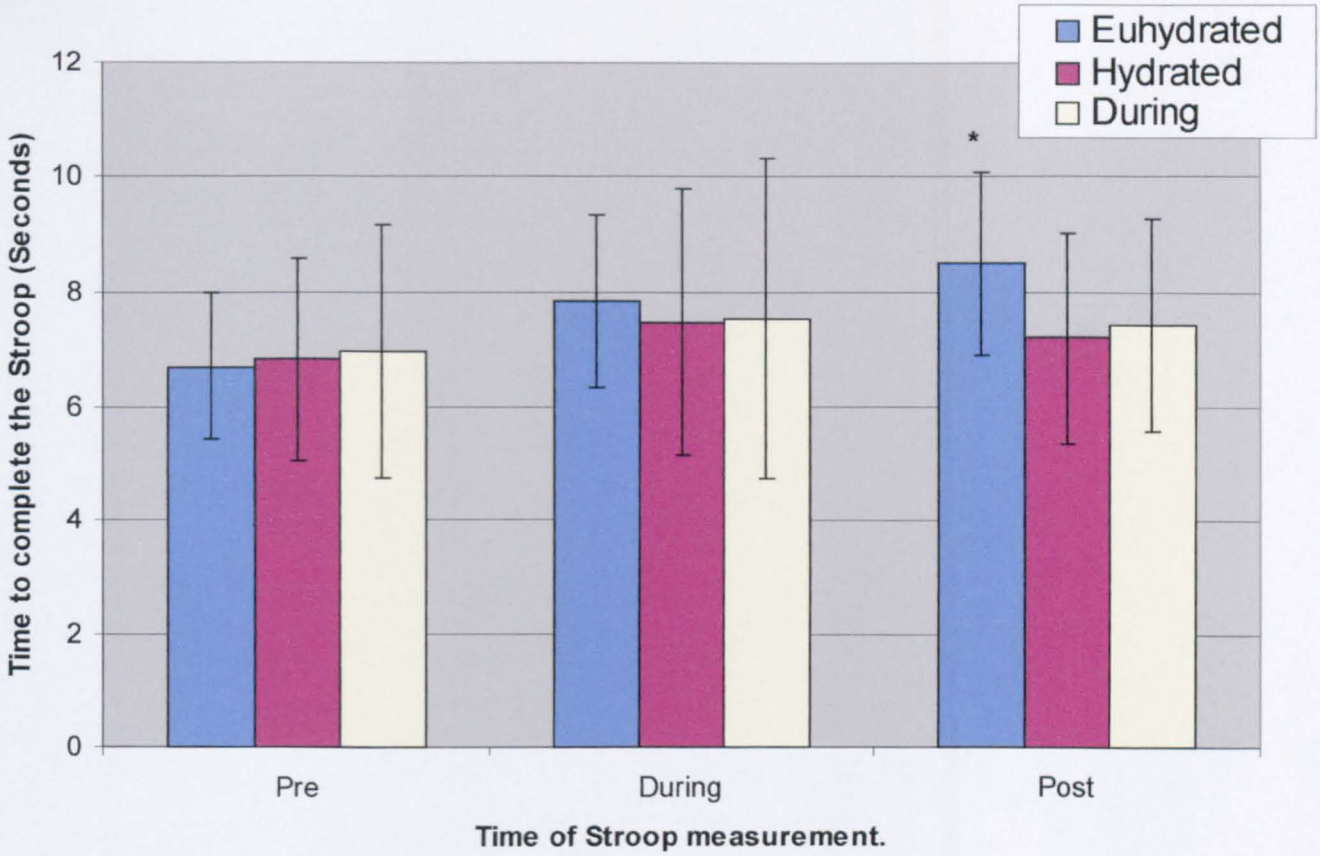


Figure 6.14 – Mean time to complete the adapted Stroop test pre (carried out immediately prior to entering the FFU), during (carried out at the mid-point which was approximately 15 minutes into the LFTE) and post (carried out immediately after leaving the FFU) exposure to the LFTE (Protocol 3). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated), or drinking 200 ml every 10 minutes during exposure (During). Data is presented as the mean ± SD (n= 6). Significance was tested using a one way ANOVA with post hoc Tukey. * - significant differences ($p<0.05$) between the pre and post euhydrated readings.

6.4 Discussion.

Heart Rate.

There were no significant differences between the LFTEs when BAIs were either hydrated or euhydrated prior to exposure, or when they drank during the LFTEs. This suggests that hydrating the BAIs 1 hour prior to exposure did not result in a lower HR response, when compared to BAIs in a euhydrated state prior to exposure.

When the BAIs were euhydrated prior to exposure during Protocol 2, the mean HR was 115.3 (± 16.8) beats·minute^{-1} which was the same as the mean HR produced during Protocol 1 (116.8 (± 18.8) beats·minute^{-1}) and similar to those produced during Protocol 3 (110.3 (± 15.5) beats·minute^{-1}). This suggests the Protocols were carried out consistently.

Murray *et al* (1995) observed significant decreases ($p<0.05$) in HR when 100 ml of fluid was consumed every 5 minutes during exercise when compared to no fluid consumed. Their subjects exercised on a cycle ergometer at 50% $VO_{2\ max}$ during temperatures of 32.0^{0}C (± 1.5) and 70.0 (± 3.0) % humidity. One exercise trial involved no fluid being ingested prior to or during exercise, whilst another trial involved consuming 400 ml of fluid prior to exercise and then 100 ml was ingested every 5 minutes during exercise. These results show drinking prior to and during exercise trials in heated environments may result in a decrease in HR responses to exercise (Heaps *et al*, 1994, Murray *et al,* 1995 and Ryan *et al*, 1989) and was used as the rationale for BAIs drinking during the LFTEs in Protocol 3.

The HR results from the present study compared with research carried out by Costill, Kammer and Fisher (1970). They also observed fluid intake had very little influence on HR responses when their subjects exercised at 70% of $VO_{2\ max}$ on a treadmill during environmental temperatures between 24.8^{0}C and 25.6^{0}C and relative humidity between 49 % and 55 %. Their subjects carried out 2 separate trials during which they were either administered with fluid or not administered with fluid throughout the exercise. Typical HRs observed for no fluid and fluid trials were 154 beats·minute^{-1} and 150 beats·minute^{-1} respectively.

However, the differences in findings elicited by the different studies may be explained by diverse environmental temperatures. Costill *et al* (1970) exercised their subjects during temperatures between 24.8^{0}C and 25.6^{0}C, whilst Murray *et al* (1995) exercised their subjects in temperatures of 32.0 (± 1.5) ^{0}C. In addition, the differences in intensity and mode of exercise may have also affected the HR responses. Costill *et al* (1970) exercised their subjects at higher intensities (70.0 % $VO_{2\ max}$) on a treadmill, whilst Murray *et al* (1995) exercised their subjects on a cycle ergometer at lower intensities (55% $VO_{2\ max}$).

The results from the studies of Costill *et al* (1970) and Murray *et al* (1995), suggest that fluid intake may only be effective during exposure to environments whereby the temperature was in excess of 30.0^{0}C and of moderate intensity (approximately 55% $VO_{2\ max}$). This was similar to the intensity undertaken during the present study (41.2 (± 16.2) % $VO_{2\ max}$) although the temperature was far greater (53.1 (± 7.8) ^{0}C in the anti-chamber). In addition, the effect of fluid ingestion may be negated by intense exercise that is equivalent to, or higher than 70% of $VO_{2\ max}$ as suggested from the results reported by Costill *et al* (1970) or high environmental temperatures, such as those observed during LFTEs.

During Protocols 2 and 3, the HR results showed drinking prior to exposure to LFTEs did not induce a significant decrease in HR responses when compared to BAIs being euhydrated prior to exposure. Therefore, this suggests that drinking prior to LFTEs does not lower the stress response to LFTEs. However, other studies have reported that drinking prior to and during exposure to temperate environments does lower the stress response (Murray *et al*, 1995).

Although the mean HR responses were not significantly different between the 3 fluid trials, the HR responses when BAIs exited the FFU to drink 200 ml of water, resulted in a significantly lower HR response (-23.2 (± 13.8) %) compared to the equivalent time during LFTEs when BAIs were euhydrated prior to exposure. This was consistent with the skin and micro-climate temperatures that were also significantly

lowered when BAIs left the FFU to drink compared to the LFTEs whereby the BAIs were euhydrated.

Manore and Thompson (2000) suggest that gastric volume is the most potent regulator of gastric emptying which, in turn, reflects the rate of rehydration. The average person empties approximately 40 ml·minute^{-1} from the stomach. Thus consuming 600 ml of water 1 hour prior to the LFTEs allowed sufficient time for the rate of gastric emptying to allow the water consumed to be emptied from the stomach and be absorbed. This shows that the rate of gastric emptying can not explain why hydrating the BAIs prior to exposure to LFTEs had no effect on HR responses compared to BAIs that were euhydrated prior to exposure.

Oxygen Cost.

The mean oxygen cost was obtained from the drop in cylinder pressure over the 34 minutes of exposure. During Protocol 2 BAIs were hydrated prior to exposure and compared to BAIs who were euhydrated. The results showed significant increases ($p<0.05$) in oxygen cost. There were no differences in the workload between the LFTEs as BAIs followed the schedule detailed in the Methods (Table 3.2). This suggests the increase in oxygen cost was not due to an increased workload. The mean oxygen cost obtained from the cylinder pressure drop during exposure to the LFTEs in Protocol 3 produced no significant differences between the LFTEs when the BAIs were hydrated prior to exposure, euhydrated prior to exposure or euhydrated prior to exposure but drank during the LFTEs.

Heaps, Gonzalez-Alonso and Coyle (1994) investigated the effect of dehydration on cardiovascular drift (increases in HR and reductions in stroke volume). They observed that during 20 minutes of cycling, the VO_2 produced by the subjects after they had consumed fluid pre-exercise, exhibited lower mean VO_2 values (2.78 (± 0.09) Litres·Minute^{-1} when no fluid was consumed and 2.67 (± 0.09) litres·minute^{-1} when fluid equivalent to the body weight lost was consumed). Heaps *et al* (1994) failed to report whether significance was obtained. Their subjects cycled until they reached body weight losses equivalent to 2.5% of body weight. After exercising they rested for 60 minutes. This was immediately followed by either a 2 hour period

whereby they consumed fluid equivalent to that lost, or they did not consume any fluid during this time. Subjects were then required to cycle at 65% of peak VO_2 for 20 minutes.

Although Heaps *et al* (1994) used a different protocol to the present study, their results do not agree with the present study, where the latter suggests drinking prior to exercise does not lower VO_2 responses. The differences between the results reported by Heaps *et al* (1994) and the present study are probably explained through the differences in ambient temperature. Subjects involved in the research by Heaps *et al* (1994) cycled in ambient temperatures of 21^0C. However, the mean temperature at shoulder height in the fire chamber was 120.8 (± 21.3) ^{0}C and in the anti-chamber was 54.5 (± 7.3) ^{0}C. This suggests that the increased FFU temperature elicited during LFTEs compared to studies conducted in thermoneutral conditions, increased the blood flow to the subcutaneous layer and in turn created a greater increase in vasodilatation (Fortney and Vromen, 1985). According to Powers, Howley and Cox (1982) increases in heat load can result in increases in VO_2 responses, thus, negating any positive effects of hydrating BAIs prior to LFTEs.

Temperature.

During Protocols 2 and 3, prior to exposure to the LFTEs, there were significant increases ($p<0.05$) in the skin (Figure 6.4), aural (Figure 6.5) and micro-climate (Figure 6.6) temperatures between the gym kit measurements and PC+SCBA measurements. This occurred prior to the LFTEs when BAIs were hydrated prior to exposure, when euhydrated prior to exposure and when euhydrated prior to exposure but drank during LFTEs. This was consistent with the findings of Protocol 1, suggesting that hydrating BAIs through consuming 600 ml of water 1 hour prior to exposure does not stem the increase in skin, aural and micro-climate through wearing PC+SCBA. This increase in skin, aural and micro-climate was significantly ($p<0.05$) exacerbated further when the BAIs were exposed to LFTEs. This was irrespective of whether the BAIs were hydrated, euhydrated prior to exposure, or drank during the LFTEs. This also suggests that hydrating BAIs prior to LFTEs does not halt the significant increases in skin, aural and micro-climate when BAIs are exposed to LFTEs.

Havenith (1999) reported that the evaporative capacity of the skin becomes compromised when wearing PC, thus suggesting this could explain the increases in skin temperature whilst wearing PC+SCBA. This may also suggest that BAIs should avoid wearing the PC+SCBA until the last available moment prior to LFTEs, therefore, lowering the heat stress response, leading to lower skin temperature responses prior to exposure. This is evidenced from the significant increases in skin, aural and micro-climate temperatures when the BAIs were dressed in gym kit compared to when they wore PC+SCBA.

There was a small but significant increase ($p<0.05$) in the temperature outside the FFU during the LFTEs when BAIs were hydrated prior to exposure compared to when they were euhydrated prior to exposure. However, there was little evidence of this increase within the PC+SCBA when the skin, aural and micro-climate temperatures were observed.

Significant increases ($p<0.05$) in core temperature (represented by rectal temperatures) were reported by Riebe *et al* (1997) when subjects were in a euhydrated state, compared to subjects who consumed fluid prior to exercise in a temperate environment (that was the same for both the trials (35.9 (± 0.0) ^{0}C). These findings are not consistent with the present study as there were no significant differences in aural temperature between the LFTEs when BAIs were hydrated or euhydrated prior to exposure. Therefore, it may be postulated that the differences between Riebe *et al* (1997) and the present study was the high temperature in the FFU, during the LFTEs when the BAIs were hydrated prior to exposure, which led to the positive benefits of hydrating prior to exposure not being observed.

There were significant reductions in skin temperature (-0.7 (± 0.9) % decrease) and micro-climate temperature (-4.2 (± 4.3) % decrease) between the intervals when BAIs exited the LFTEs to consume fluid and re-entered the FFU. This suggests that exiting the FFU to drink 200 ml every 10 minutes reduced the heat stored within the PC and thus the heat stress placed on the BAIs.

Blood Pressure.

Exposure to LFTEs produced significant increases ($p<0.05$) in systolic blood pressure between the pre exposure measurements and post exposure measurements when BAIs were hydrated or euhydrated prior to exposure, or drank during the LFTEs during Protocols 2 and 3. These findings were consistent with the systolic blood pressure responses produced by BAIs during the LFTEs in Protocol 1. Crandall *et al* (2000) have also shown increases ($p<0.05$) in systolic blood pressure as a result of exposure to temperate environments. Richardson and Capra (2001) observed significant increases ($p<0.05$) in systolic blood pressure between pre and post measurements when fire fighters were exposed to temperate conditions whilst wearing PC+SCBA. They concluded that the increase in systolic blood pressure was directly related to the increase in environmental temperature. This suggests that the increase in temperature when BAIs were exposed to LFTEs, compared to prior to exposure, induced the significant increases in systolic blood pressure.

However, the mock training exercises during Protocol 1 showed there were small increases in systolic blood pressure from wearing the PC+SCBA and undertaking the workload necessary to complete the mock training exercises. This suggests the significant increase in systolic blood pressure observed during the LFTEs during Protocols 2 and 3 was contributed to by the wearing of PC+SCBA and the workload required to carry out the LFTEs, but was primarily from the heat from the fire. There were no significant differences in the changes (between pre and post exposure to the LFTEs) in systolic blood pressure between any of the fluid trials during Protocols 2 or 3. This suggests that hydrating BAIs prior to, or during exposure, did not attenuate the increase in systolic blood pressure when exposed to extreme heat during LFTEs.

During Protocols 2 and 3 there were no significant differences between the diastolic pressure between the pre exposure and post exposure measurements. There were no significant differences in the diastolic blood pressure changes (between the pre exposure and post exposure measurements) between any of the fluid trials. The diastolic blood pressure results from Protocol 1 showed that exposure to hot environments (LFTEs) when compared to thermoneutral conditions (the mock training exercises) did not elicit significant differences in the diastolic blood pressure values. The diastolic pressure results from Protocols 2 and 3 suggest that hydrating

BAIs prior to exposure also does not affect the diastolic blood pressure. According to Astrand and Rodahl (1986), the diastolic responses reported in Protocols 2 and 3 are typical of an exercise response, although it does suggest diastolic blood pressure was unaffected by the fluid interventions.

Blood Lactate.

There were significant increases ($p<0.05$) in blood lactate when the pre exposure measurements were compared to the post exposure measurements during Protocols 2 and 3. This suggests wearing PC+SCBA and carrying out the workload necessary to carryout LFTEs creates an increase in muscle glycolysis. The increases in blood lactate between the pre and post exposure measurements were consistent with those observed in Protocol 1.

There were no significant differences in blood lactate concentration between the LFTEs when BAIs were hydrated prior to exposure, euhydrated prior to exposure or when they drank during LFTEs. This suggests that hydrating BAIs prior to exposure or, consuming fluid during LFTEs did not reduce the blood lactate concentration during exposure to LFTEs.

Xavier Bigard *et al* (2001) investigated the effects of dehydration on fatigue of muscular contractions and observed results that were consistent with the present study. Their subjects were dehydrated until they lost 3% of body mass (BAIs when euhydrated prior to exposure produced mean body mass losses of 1.4 (± 0.8) % and when hydrated prior to exposure produced losses of 1.2 (± 0.4) %) through intermittent exposure to a sauna (80-85^{0}C). They concluded there were no significant increases in blood lactate concentration between the dehydrated and euhydrated trials.

The significant increases in temperature outside the FFU and also the temperature inside the FFU during LFTEs when BAIs were hydrated prior to exposure compared to when they were euhydrated prior to exposure, appears to have not significantly affected the blood lactate concentration despite the findings of Fink *et al* (1975).

These findings are consistent with Nielsen (1994) who also did not observe a significant increase (1.3 (± 0.1) to 2.2 (± 0.2) mmol·litre^{-1}) in blood lactate, during thermoneutral conditions (20^{0}C) compared to temperate (40-42^{0}C) conditions.

Daries, Noakes and Dennis (2000) also observed no significant increases in lactate concentrations when subjects either consumed 0.9 litres·hour^{-1} compared to 0.4 litres·hour^{-1} as a result of running at 65% of VO_2 peak at 25^{0}C for 90 minutes. This suggests that fluid intake appears to have no affect on lactate production as shown in the present study.

Body Mass.

During Protocol 2 when BAIs were either hydrated or euhydrated prior to exposure they lost, through the sweating process, 0.9 (± 0.2) kg and 1.1 (± 0.7) kg respectively. The body mass losses were not significant between LFTEs when BAIs were hydrated or euhydrated prior to exposure. Hydrating BAIs prior to exposure was still inadequate to stem their body weight loss to less than 1% of mean body weight. When hydrated prior to exposure BAIs produced mean body mass losses of 1.2 (± 0.4) %). Losses greater than 1% may result in a decrement in physical work capacity and loss of concentration (Riebe *et al* 1997 and Brooks *et al*, 1996).

During Protocol 3 the BAIs lost on average 1.3 (± 1.1) kg, 0.8 (± 0.3) kg and 0.5 (± 0.3) kg for the BAIs when they were euhydrated prior to LFTEs, hydrated prior to the LFTEs and when they were euhydrated prior to exposure but drank during the LFTEs respectively (Figure 6.10). There were no significant differences between the body mass losses produced by exposure to the different fluid trials. There were also no differences between the temperature outside the FFU, or inside the FFU and no difference between the workload between the LFTEs that incorporated the different timings of fluid ingestion. This suggests the fluid interventions were the only difference between the LFTEs. Therefore, drinking during the LFTEs appears to produce smaller losses in body mass (Figure 6.10), thus, lessening the likelihood of decrements in physical work capacity and concentration (Riebe *et al* 1997 and Brooks *et al*, 1996).

Haematocrit.

During Protocols 2 and 3 there were significant increases ($p<0.05$) in haematocrit when the pre exposure values were compared to the post exposure values for LFTEs when BAIs were either hydrated prior to exposure, euhydrated prior to exposure or drank during LFTEs. This shows that hydration did not stop the changes in haematocrit and plasma volume, nor did it stop the loss of concentration as evidenced by the increased time to complete the adapted Stroop test.

However, there were no significant differences in changes (between the pre exposure and post exposure values) in haematocrit values between LFTEs when BAIs were hydrated prior to exposure, compared to when they were euhydrated prior to exposure. This suggests that hydrating BAIs prior to exposure does not alleviate the stress response of exposure to LFTEs. There were also no significant differences in the reductions in plasma volume between the LFTEs when BAIs were either hydrated or euhydrated prior to exposure. This was not consistent with the findings of Jimenez *et al* (1999) who reported that hydrating their subjects induced increases in plasma volume when compared to when their subjects were in a euhydrated condition. However, their subjects were not exposed to heat when hydrated, suggesting that hydrating with 600 ml of fluid prior to exposure to the LFTEs, was not sufficient to stem the 9.2 (± 5.2) % reduction in plasma volume observed due to exposure to LFTEs.

Mean percent changes in plasma volume are shown in Table 6.3. There were no significant differences between any of the timings of fluid ingestion on percent plasma volume changes. This suggests fluid consumption that occurred prior to exposure or during exposure did not stem the decrease in plasma volume values between the pre exposure and post exposure values.

The mean haematocrit results from the present study when BAIs were hydrated or euhydrated prior to the LFTEs, were in agreement with those elicited by Heaps *et al* (1994). Heaps *et al* (1994) also observed no significant differences between their fluid and no fluid trials. However, there were significant increases ($p<0.05$) in haematocrit values between pre (42.6% (± 0.7)) and post (46.5% (± 0.7)) exercise values when fluid was withheld (no fluid trial) in response to cycling at 65% of peak

VO_2 for 20 minutes at 21^0C. In addition, when 1.9 (± 0.1) litres of water was ingested prior to exercise (fluid trial), the haematocrit values were also significantly increased ($p<0.05$) between the pre (43.5 % (± 1.0)) and post (47.1 % (± 0.9)) exercise values.

Fluid Balance.

There were significant decreases ($p<0.05$) in urine colour when the mean pre exposure values of the BAIs when hydrated prior to exposure were compared to the urine colour pre exposure to LFTEs when BAIs were euhydrated prior to exposure. This shows that when the BAIs consumed 600 ml of fluid 1 hour prior to exposure, they were hydrated prior to entering the LFTEs (Table 6.4).

During the LFTEs when BAIs were euhydrated prior to exposure, they were in negative fluid balance by -1009.2 (± 1629.6) ml. The BAIs were in positive fluid balance by 159.2 (± 696.0) and 220.8 (± 448.4) ml for the LFTEs when they were hydrated prior to exposure and when they drank during the LFTEs respectively. This was irrelevant of the diuretic drinks they consumed (beverages such as tea, coffee and lemonade). This suggests that drinking prior to exposure and during exposure will ensure the BAIs remain in positive fluid balance during the shifts when they carry out LFTEs. Therefore, beverages that contain diuretic properties would not have aided the hydration status of the BAIs, thus should be avoided. Also, the BAIs reported drinking alcohol during the evenings prior to and also following the LFTEs. This may be contra-indicatory to a successful rehydration programme and should be avoided on the evening following and preceding LFTEs.

There were no significant differences between the sum of the mean fluid consumed or the sum of the mean urine produced between any of the different fluid trials. This suggests there were no lasting benefits of altering the fluid timing on hydration status and urine output through out the days of the LFTEs when BAIs were hydrated prior to exposure or when BAIs drank during LFTEs.

The changes in ECW, ICW and TBW (Table 6.5) are in agreement with Protocol 1. The results suggest the Dual Scan 2005 over-estimates body water values (obtained

at the start and termination of the shift) when BAIs have been exposed to LFTEs. This supports the findings of Caton *et al* (1988) who suggest that exposure to heated environments increased the peripheral skin blood flow that resulted in an over-estimation of body water. These results suggest the Dual Scan 2005 is not suitable to monitor body water on the days when BAIs are exposed to LFTEs.

Psychological Responses to LFTEs.

Rate of Perceived Exertion (RPE).

During Protocols 2 and 3 the RPE readings were significantly increased ($p<0.05$) between pre exposure and during exposure measurements, pre exposure and post exposure RPE measurements and the measurements obtained during exposure and post exposure RPE responses for all fluid trials. These findings are consistent with the RPE responses reported in Protocol 1. There were no significant differences in the RPE responses between any of the fluid trials for either the RPE responses obtained pre, during or post exposure.

These results are consistent with research carried out by Riebe *et* al (1997). They observed no significant differences in RPE responses when subjects abstaining from fluid ingestion were compared to the subjects who drank fluid orally during exercise (90 minutes of treadmill walking at 50% of $VO_{2\ max}$ during 36^0C). They also reported that it was only at 40 minutes that the differences in the RPE responses were significantly different ($p<0.05$) between the no fluid and fluid trials. When the BAIs were euhydrated or hydrated prior to exposure, they were only exposed for 35.6 ($\pm$ 4.0) minutes and 34.7 ($\pm$ 3.2) minutes respectively. This suggests the duration of the LFTEs were not great enough to produce significant differences.

Murray *et al*, (1995) observed there were differences between the RPE responses when their subjects drank 100 ml every 5 minutes compared to not drinking fluid during the trials. Their subjects were required to carry out 3 trials, each involved cycling at 50% $VO_{2\ max}$ for 60 minutes, during which subjects either consumed no fluid, 100 ml of fluid every 5 minutes or 200 ml of fluid every 10 minutes. However, significant differences between the RPE responses between drinking 100 ml every 5

minutes and not drinking during the trials was only assumed after 25 minutes. This may explain why the short duration of the LFTEs did not produce any differences between the fluid trials for the measurements obtained pre exposure, during exposure and post exposure.

Adapted Stroop Test.

During Protocol 2 there were significant increases in time to complete the adapted Stroop test between the pre exposure and during exposure measurements for the LFTEs when the BAIs were hydrated or euhydrated prior to exposure. There were also significant increases in time to complete the adapted Stroop test between the pre exposure and post exposure measurements. In addition, there were significant increases between the measurements obtained during exposure and the post exposure measurements when BAIs were euhydrated prior to exposure. This suggests there was a loss of concentration as a result of exposure to LFTEs which may in part, be caused by the mean body mass losses. When BAIs were hydrated or euhydrated prior to exposure they lost 1.2 (± 0.4) % and 1.4 (± 0.8) % of their body mass respectively. These results are consistent with the findings reported by Brooks *et al* (1996) that losses of 1% of body mass result in decreases in concentration.

There were no significant differences between the time to complete the adapted Stroop test for the pre exposure measurements, during exposure measurements and the post exposure measurements when BAIs were hydrated prior to exposure compared to when they were euhydrated prior to exposure. This suggests that hydrating BAIs prior to the LFTEs did not prevent body mass losses in excess of 1%. In turn, this did not prevent losses of concentration as demonstrated by the significant increases in time to complete the adapted Stroop test during the LFTEs when BAIs were hydrated prior to exposure.

During Protocol 3 when the BAIs had drunk during the LFTEs, they exhibited completion times that did not significantly increase as a result of exposure to the LFTEs. However, when the BAIs were euhydrated prior to the LFTEs, the time to complete the adapted Stroop test did significantly increase between the pre exposure measurements and the during exposure measurements (Figure 6.14). This suggests

that by ingesting fluid during exposure, reduces the likelihood of a reduction in concentration. This is consistent with the losses of body mass reported in Figure 6.9. This showed when BAIs were hydrated prior to exposure and when they drank during the LFTEs, the amount of body mass lost was lower compared to the LFTEs when the BAIs were euhydrated prior to exposure. Brooks, *et al* (1996) state that losses as little as 1% of body weight will result in losses of concentration. However, the BAIs mean loss of body mass amounted to 0.6 (± 0.4) % when they drank during exposure, which was lower than the 1% of body mass concurring the findings of Brooks *et al* (1996).

Enander and Hygge (1990) suggested that concentration is not affected when heated environments fail to increase the core temperature. The mean aural temperature during the LFTEs increased by 2.8 (± 1.4) ^{0}C when BAIs were euhydrated prior to exposure. This increase in aural temperature, in conjunction with the loss of body mass through sweating, may have contributed to the significant increase ($p<0.05$) in time to complete the adapted Stroop test (between the pre exposure measurements and during exposure measurements). This suggests there was an increase in a loss of concentration observed during the LFTEs when the BAIs were euhydrated prior to exposure. Therefore, hydrating BAIs prior to exposure or drinking during the LFTEs appears to halt the loss in concentration of BAIs during LFTEs.

6.5 Conclusions.

The aim of this chapter was determine whether hydration could reduce the short term stress identified in Chapter 5. The data confirmed the effect of temperature on the short term stress in Chapter 5. This was evidenced through the significant increases between the pre exposure and post exposure body mass losses, haematocrit, blood lactate, systolic blood pressure, RPE, and the time to complete the adapted Stroop test. Hydrating BAIs prior to the LFTEs, did not alleviate the significant increases in skin, aural and micro-climate temperatures when PC+SCBA was worn prior to exposure and also during exposure to LFTEs. Hence, hydrating the BAIs prior to exposure did not lower the stress placed on them during LFTEs.

The results have also shown that leaving the FFU to drink during LFTEs lowered the responses to LFTEs through dissipation of heat from the PC, compared to when BAIs were hydrated or euhydrated prior to exposure. This in turn significantly lowered micro-climate and skin temperature responses, which placed less heat stress upon the BAIs (Figure 6.7).

Chapter 7

Heart Rate Variability (HRV)

7.1 Introduction.

Previous studies indicate that a number of stress indicators are affected by heat. The aim of this chapter is to further investigate one of these parameters, heart rate variability (HRV). This chapter investigates three different areas. The first area examines the effects on HRV through controlled breathing when compared to spontaneous breathing. This section of the study thus aimed to observe whether there was a cortical affect of controlling breathing to 0.2 Hz, when compared to a matched spontaneous breathing frequency of 0.2 Hz.

The second area of the study investigates whether the method of monitoring HRV was sensitive enough to detect any changes in the sympathetic and parasympathetic control of HR during exposure to heat. During this study subjects were exposed to a Finnish sauna for a period of 15 minutes.

The third area investigates whether during LFTEs BAIs were stressed. This was indicated through a reduction of the influence of the parasympathetic and increase in the sympathetic influence on HR control. In addition, the study also investigates whether fluid interventions had any effect on the sympathetic and parasympathetic control of HR during exposure to LFTEs during a field-based protocol.

7.2 Methods.

Refer to Methods chapter (Section 3.4.7).

7.3 Results.

7.3.1 Controlled and spontaneous breathing on HRV.

Figure 7.1 shows the mean results representing the sympathetic indicators of HR control. When breathing was maintained at 0.20 (± 0.0) Hz (12.0 (± 0.0) cycles·minute^{-1}) during the controlled breathing (CB) trials or at 0.20 (± 0.9) Hz (12.3 (± 0.9) cycles·minute^{-1}) during the spontaneous breathing (SB) trials, there were no significant differences between the CB and SB trials.

Table 7.1a (Appendix E) shows the complete data obtained from the CB and SB trials. The most important data is discussed within this chapter. However, due to the large volume of data obtained from the HRV measurements, there is additional material (e.g., absolute values (where the values have not been normalised) for the frequency domain) placed in Appendix E. Appendix E contains all the additional data from all the trials discussed within this chapter.

Figure 7.2 shows the mean results for indicators of parasympathetic influences on HR control. There were no significant differences between the parasympathetic indicators of HR control between the CB and SB trials.

Figures 7.3 and 7.4 are power spectral density graphs (PSD) that show the frequency domain data, analysed using fast fourier transform (FFT) analysis. These graphs compare Subject 10 when breathing frequency (BF) was controlled (Figure 7.3) and when BF was spontaneous (Figure 7.4). Controlled breathing can be observed from Figure 7.3 through an increase in the size of the spike at 0.2 Hz, which is equivalent to 12 breaths·minute^{-1}. The other components of HRV (VLF and LF) were not affected through controlling breathing when compared to spontaneous breathing.

Figures 7.5 and 7.6 show typical R-R interval tachograms for a single subject during the CB and SB trials. The similarities in the mean interval time between the tachograms suggest there were no differences between controlled and spontaneous breathing on the R-R intervals.

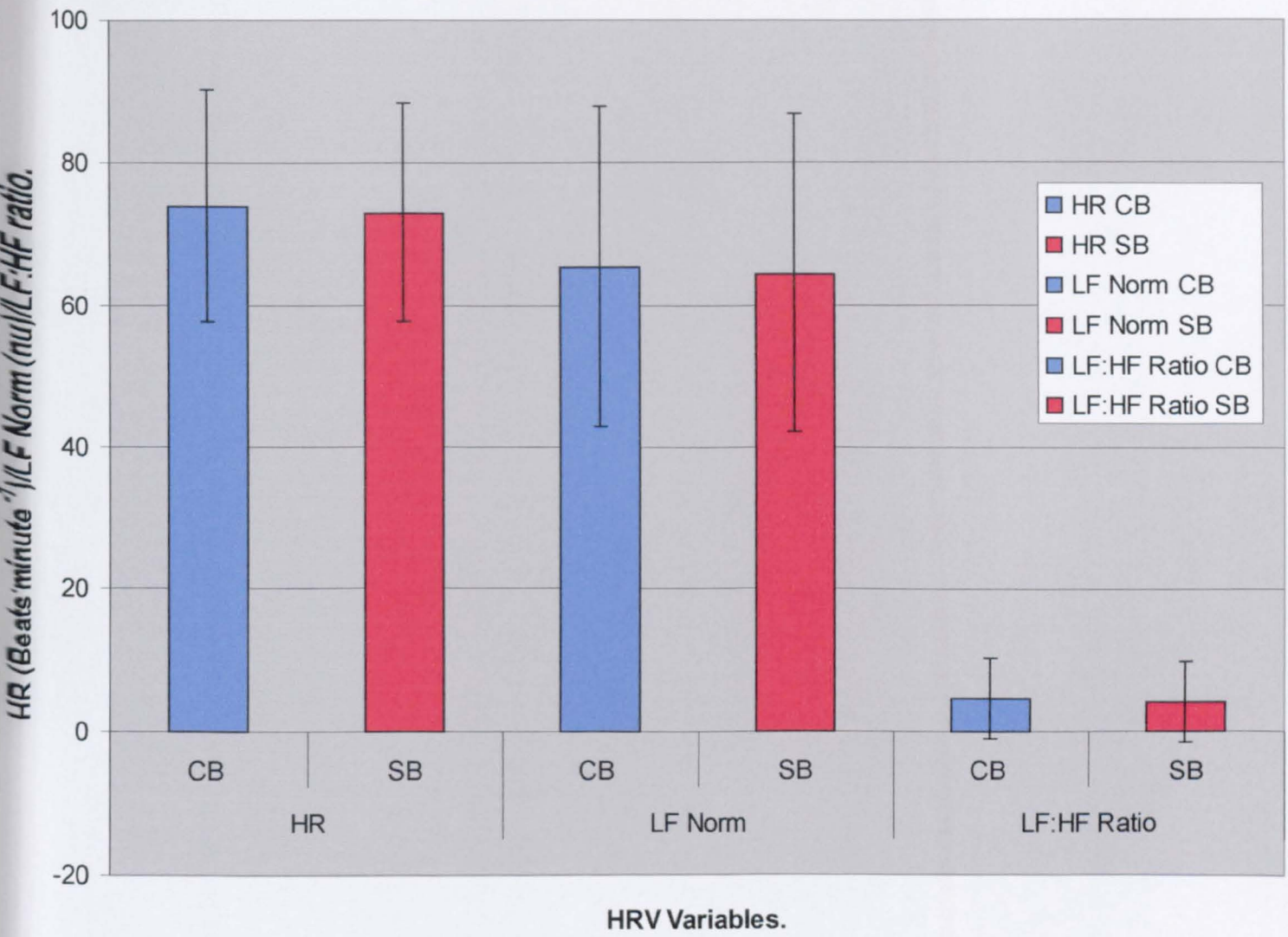


Figure 7.1 – The effect of controlled breathing (CB) and spontaneous breathing (SB) on heart rate (HR), and the indicators of sympathetic control of cardiac function; LF Norm (low frequency (LF) values in normalised units); and LF:HF Ratio (ratio between normalised low frequency (LF) and normalised high frequency (HF) data). Results were obtained during seated rest when breathing frequency was controlled (CB) at 0.20 (± 0.0) Hz and when breathing frequency was spontaneous (SB) (0.20 (± 0.9) Hz). Data is presented as the mean ± SD (n = 18). Significance was tested using a paired t-test.

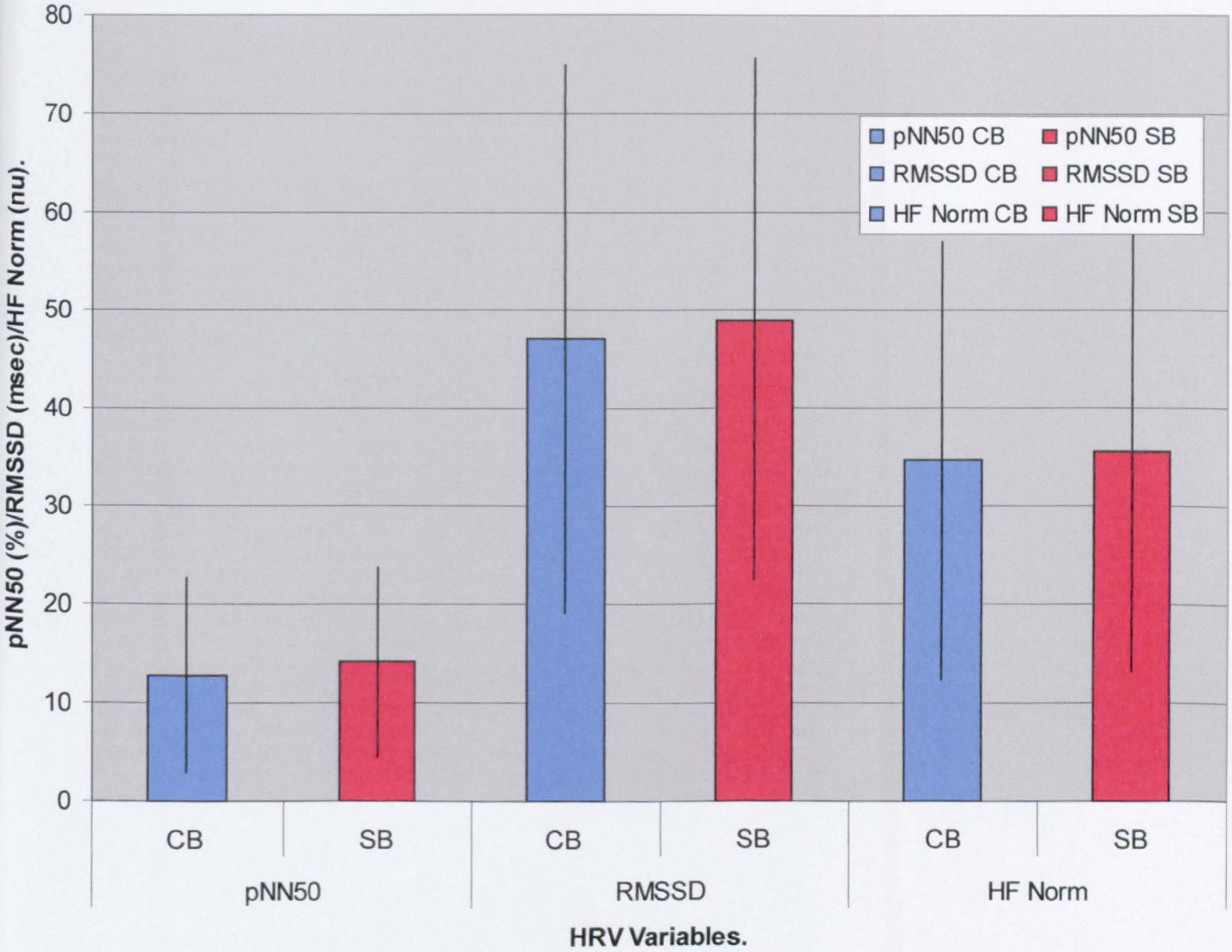


Figure 7.2 – The effect of controlled breathing (CB) and spontaneous breathing (SB) on the indicators of parasympathetic control of cardiac function; pNN50 (the number of adjacent pairs of R-R intervals differing by more than 50 ms divided by the total number of R-R intervals); RMSSD (the square root of the mean of the sum of the squares of differences between adjacent R-R intervals); and HF Norm (power in the high frequency (HF) normalised). Results were obtained during seated rest when breathing frequency was controlled (CB) at 0.20 (± 0.0) Hz and when breathing frequency was spontaneous (SB) (0.20 (± 0.9) Hz). Data is presented as the mean ± SD (n = 18). Significance was tested using a paired t-test.

Key –

VLF (0.00 - 0.04 Hz).

LF (0.04 - 0.15 Hz)

HF (0.15 – 0.40 Hz)

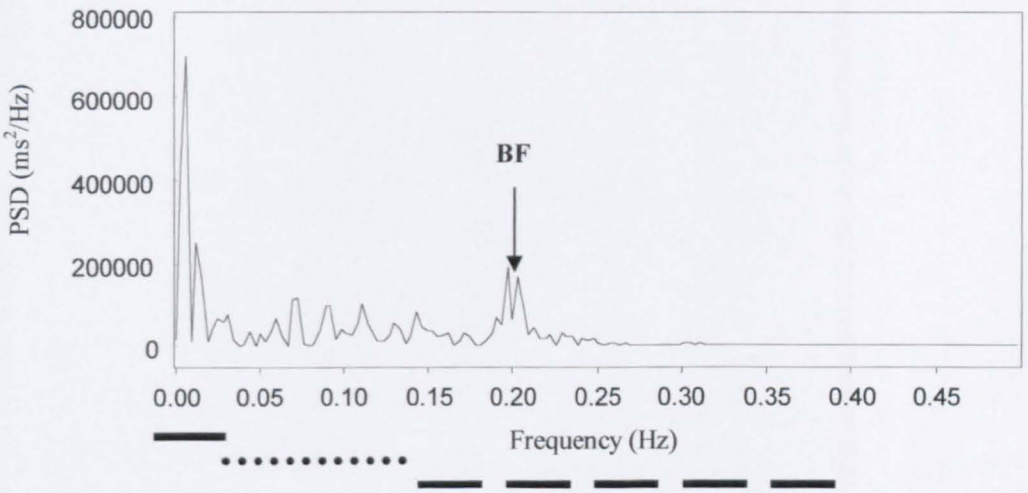


Figure 7.3 – The effect of controlled breathing (CB) using the fast fourier method of obtaining a power spectral density (PSD) graph in thermoneutral conditions (room temperature maintained between 22^0 and 24^0C) for a period of 5 minutes. These graphs depict where the distribution of power has occurred within the HRV components. The areas on the graphs that are represented by the VLF, LF and HF frequencies are depicted by the different lines (refer to the key). Results were obtained during seated rest when breathing frequency was controlled (CB) at 0.20 (± 0.0) Hz (n = 1).

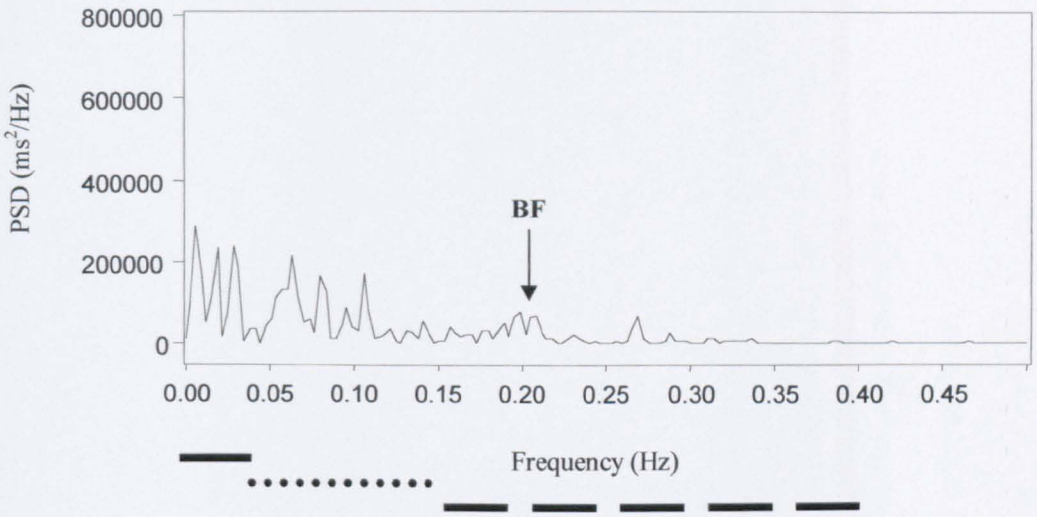
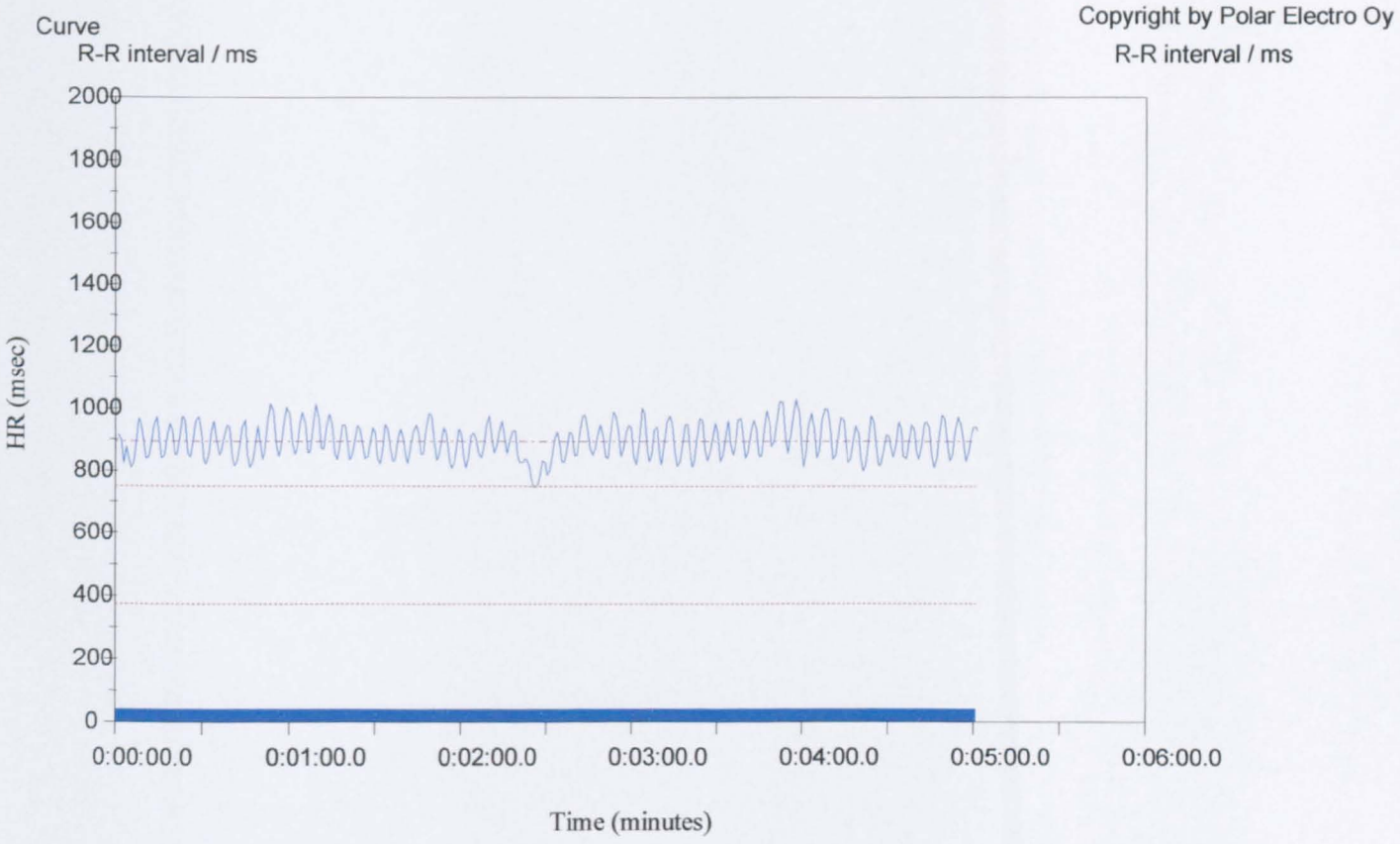
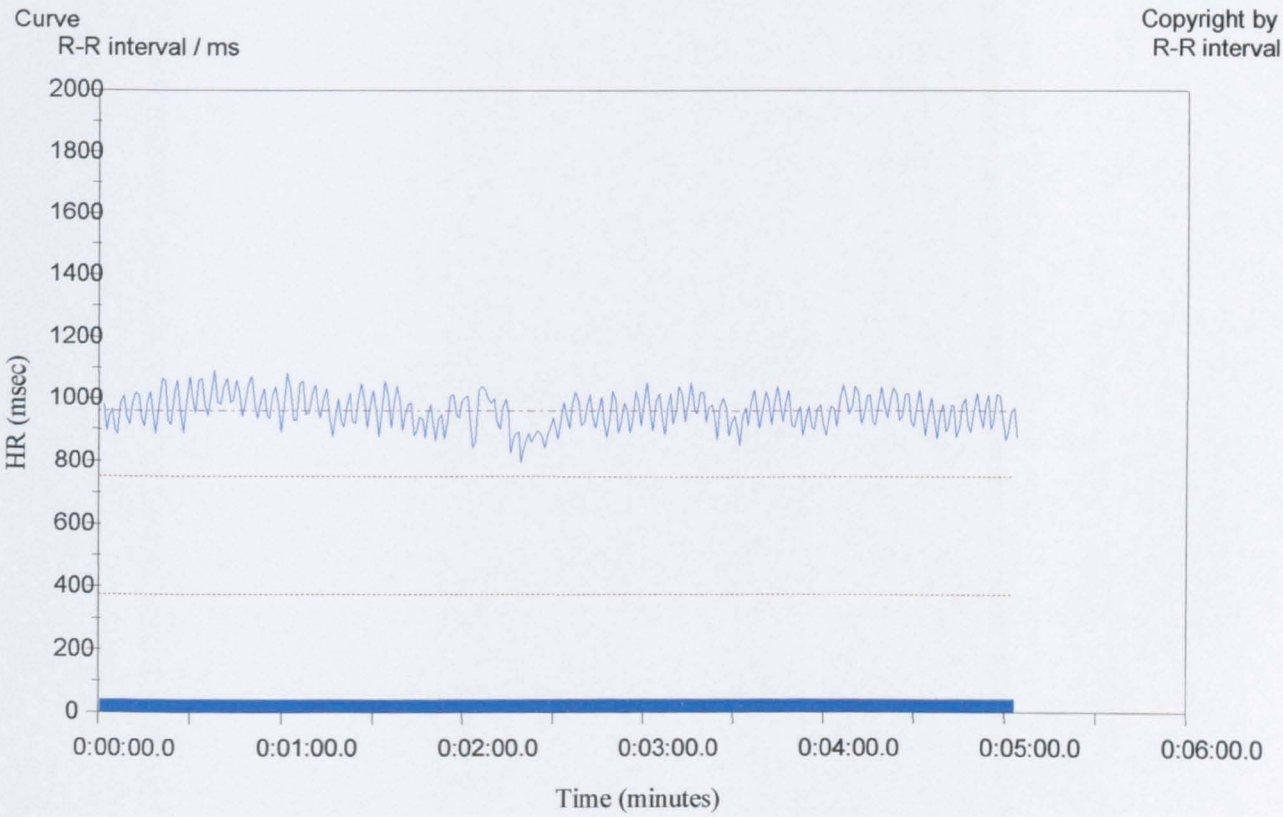


Figure 7.4 – The effect of spontaneous breathing (SB) using the fast fourier method of obtaining a power spectral density (PSD) graph in thermoneutral conditions (room temperature maintained between 22^0 and 24^0C) for a period of 5 minutes. These graphs depict where the distribution of power has occurred within the HRV components. The areas on the graphs that are represented by the VLF, LF and HF frequencies are depicted by the different lines (refer to the key). Results were obtained during seated rest when breathing frequency was spontaneous (SB) at 0.20 (± 0.9) Hz (n = 1).



				Average	895 ms		
				Duration of exercise: 0:05:00.0			

Figure 7.5 – The effect of controlled breathing (CB) using a heart rate (HR) R-R interval tachogram (Polar software (Finland, Oy)). Data was obtained in thermoneutral conditions (room temperature maintained between 22^0 and 24^0C) during seated rest for a period of 5 minutes. Breathing frequency was controlled at 0.20 (± 0.0) Hz (n = 1).



				Average	910 ms		
				Duration of exercise: 0:05:00.4			

Figure 7.6 – The effect of spontaneous breathing (SB) using a heart rate (HR) R-R interval tachogram (Polar software (Finland, Oy)). Data was obtained in thermoneutral conditions (room temperature maintained between 22^0 and 24^0C) during seated rest for a period of 5 minutes. Breathing frequency was controlled at 0.20 (± 0.9) Hz (n = 1).

Figure 5.5 and 5.6 suggest that there were no differences between the R-R interval tachograms when breathing was controlled or spontaneous.

7.3.2. The effect of sauna exposure on HRV.

Table 7.1 shows the mean body mass losses obtained immediately pre and post exposure to the sauna were 0.6 (± 0.3) kg (equating to 0.9 (± 0.4) % of the subject's body weight). Haematocrit was also significantly increased ($p<0.05$) between the pre exposure values and post exposure values. Using the equation from Van Beaumont *et al* (1972), the mean haematocrit changes suggested a mean reduction of plasma volume of –4.9 (± 3.3) %. The time taken to complete the Stroop test also increased significantly ($p<0.05$) by 17.2 (± 24.5) %.

The mean skin and environmental temperature obtained in the final 5 minutes of sauna exposure were significantly increased ($p<0.05$) (26.8 (± 7.2) % and 175.9 (± 27.5) % respectively) when compared to the pre exposure readings. Also the aural temperature during the sauna (aural temperature change in the sauna obtained from the mean of the first compared to the last 5 minutes of exposure) increased significantly ($p<0.05$) by 4.8 (± 1.1) %. This increase can be observed from points A and B in Figure 7.7.

Figure 7.8 shows that the indicators of sympathetic HR control increased significantly ($p<0.05$), when pre readings were compared to the last 5 minutes of exposure. The mean heart rate (beats·minute^{-1}) increased 60.7 (± 18.3) %, LF Norm (nu) increased 84.5 (± 76.5) % and the LF:HF ratio increased 10.9 (± 10.8) %.

The indicators of parasympathetic influence on heart rate control are depicted in Figure 7.9. The mean HF Norm (nu) significantly decreased ($p<0.05$) by 34.9 (± 19.4) %, mean pNN50 (%) decreased significantly by 89.1 (± 31.3) %, mean RMSSD (msec) decreased significantly ($p<0.05$) by 83.4 (± 7.0) %. Also the indicator of overall HR variability, mean SD (msec), decreased significantly ($p<0.05$) by 60.8 (± 16.4) %.

Figure 7.10 and 7.11 are typical R-R interval tachograms for a single subject whilst sat in thermoneutral conditions and for the same subject whilst sat in the sauna. The differences in the traces clearly show the reduction in overall HRV, but increase in HR.

Table 7.1 – Mean temperature, body mass, haematocrit and time to complete the adapted Stroop test, prior to and during the sauna exposure. Values are means (M), standard deviations (SD) with n = 10. The change and % change are also presented for each parameter. All temperature measurements were monitored prior to, and during the last 5 minutes of sauna exposure; environmental, aural (ear) and skin (pectoralis major). Aural change in sauna was obtained from the mean of the initial 5 minutes compared to the mean of the last 5 minutes of sauna exposure. Body mass, haematocrit values and mean time to complete the adapted Stroop test were observed prior to the sauna exposure (Pre) and post exposure to the sauna (Sauna). Significance was tested using a paired samples t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre and sauna readings. ** - significant differences ($p<0.05$) between the mean aural temperature obtained from the initial 5 minutes compared to the mean of the final 5 minutes of the sauna exposure.

Variable	Units	Pre		Sauna			
		M	SD	M	SD	Change	%Change
Environmental Temperature	^{0}C	27.0	0.7	74.3	5.9	47.3 (±6.5)	175.9 (±27.5)*
Temperature							
Skin	^{0}C	33.0	0.9	41.9	1.6	8.8 (±2.1)	26.8 (±7.2)*
Aural	^{0}C	35.9	0.6	38.8	0.5	2.8 (±0.7)	7.9 (±2.1)*
Aural change in sauna	^{0}C	37.0	0.6	38.8	0.5	1.8 (±0.4)	4.8 (±1.1)**
Body mass	kgs	73.9	11.1	73.3	11.3	-0.6 (±0.3)	-0.9 (±0.5)*
Haematocrit	%	45.2	2.5	46.4	2.0	1.3 (±0.9)	2.8 (±2.1)*
Time to complete Stroop test	Seconds	6.7	1.5	7.7	1.6	1.0 (±1.4)	17.2 (±24.5)*

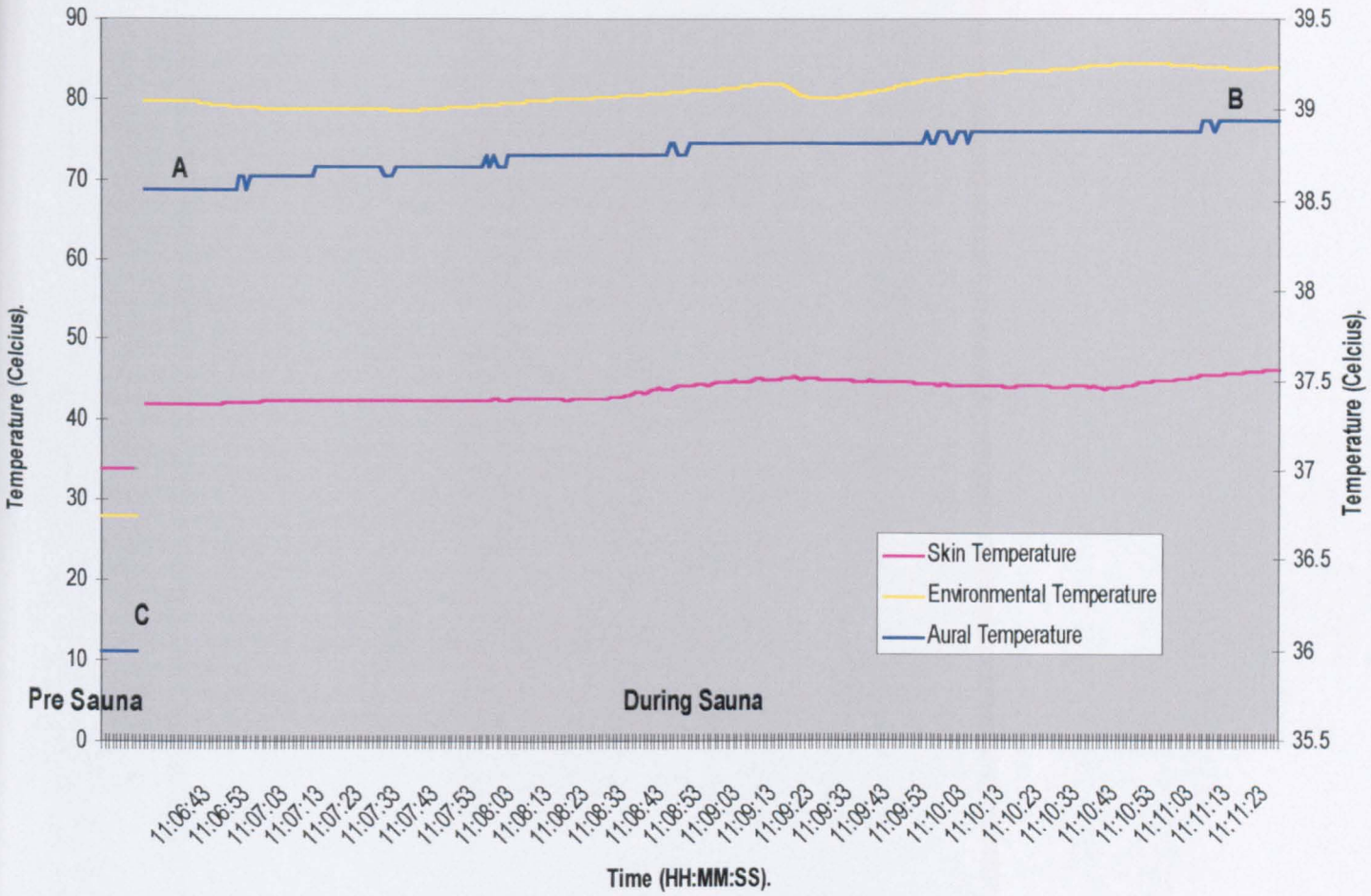


Figure 7.7 – Typical example of skin, aural and climate responses for Subject 2 obtained prior to sauna exposure (Pre sauna) (mean of 5 minutes data collection) and during the sauna (measurements taken every second). Point A represents the mean of the first 5 minutes of aural temperature data. Point B represents the final 5 minutes of aural temperature data. Point C represents the mean of 5 minutes aural temperature data obtained prior to exposure (n = 1).

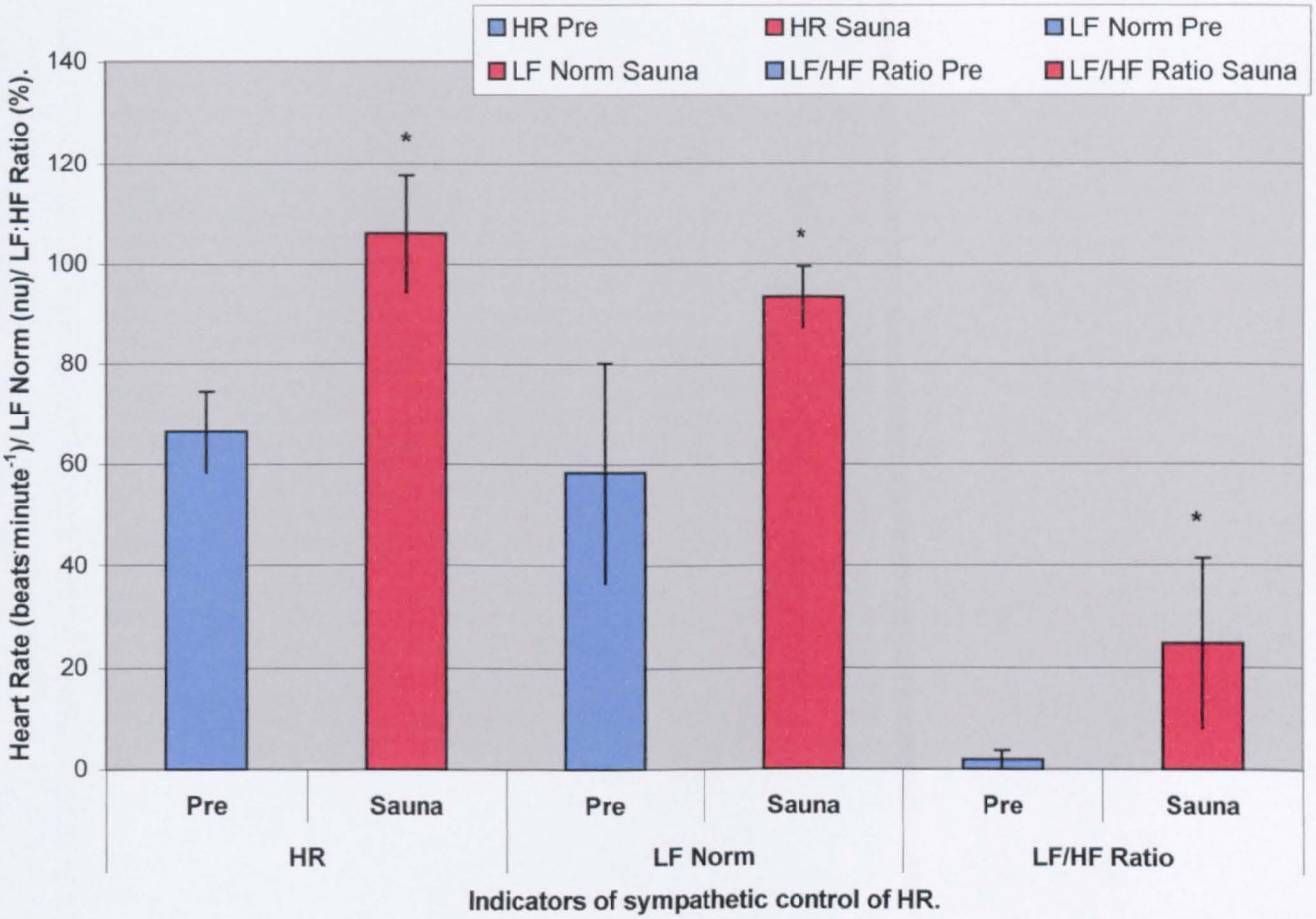


Figure 7.8 – Heart rate (HR) and the indicators of sympathetic control of cardiac function; LF Norm (low frequency (LF) values in normalised units); and LF:HF Ratio (ratio between normalised low frequency (LF) and normalised high frequency (HF) data) when exposed to a sauna. Results were obtained during 5 minutes of seated rest prior to exposure (Pre) and during the final 5 minutes of a 15 minute sauna exposure (Sauna). Data is presented as the mean ± SD (n = 10). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre and sauna measurements.

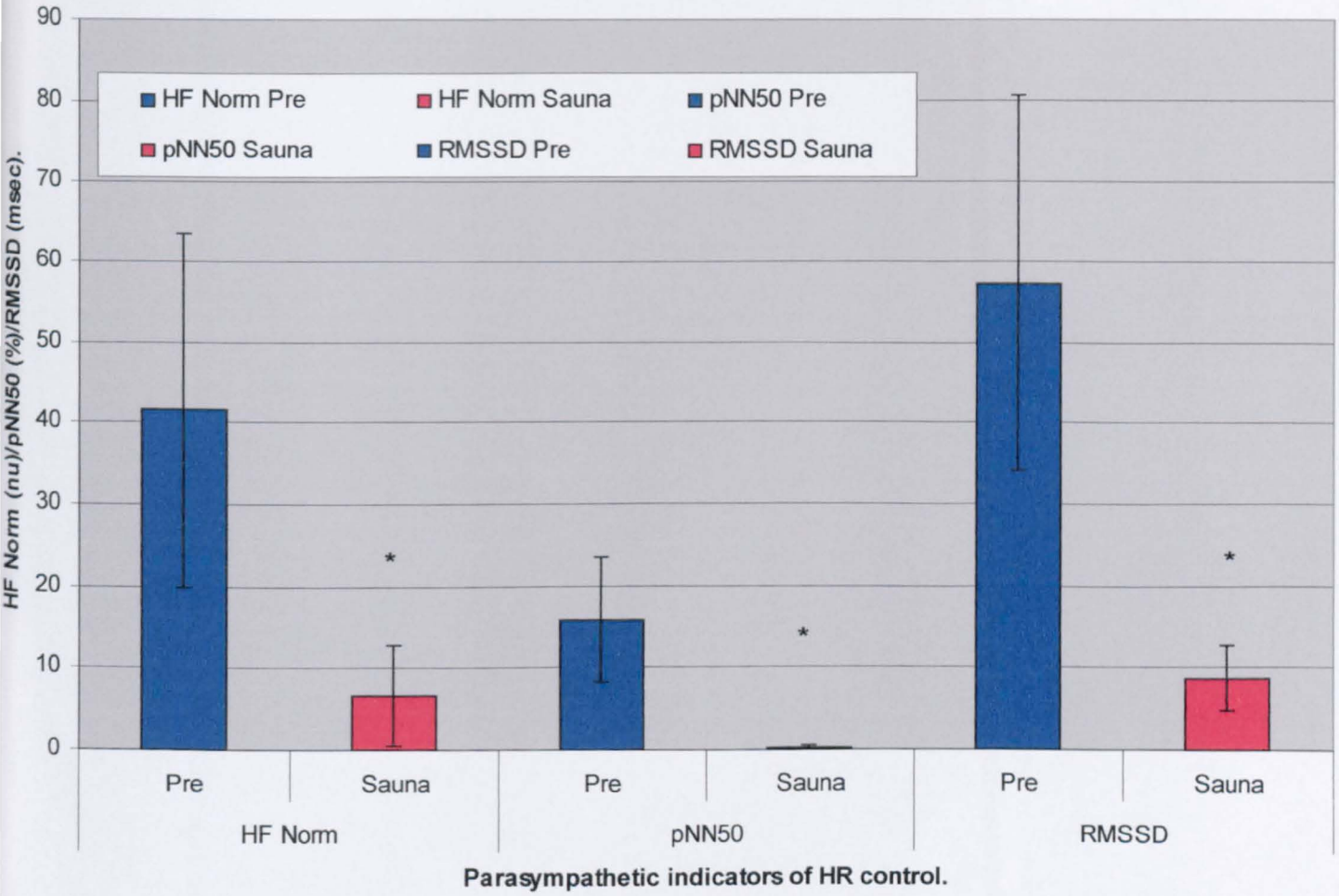


Figure 7.9 – The indicators of parasympathetic control of cardiac function; pNN50 (the number of adjacent pairs of R-R intervals differing by more than 50 ms divided by the total number of R-R intervals); RMSSD (the square root of the mean of the sum of the squares of differences between adjacent R-R intervals); and HF Norm (power in the high frequency (HF) normalised) when exposed to a sauna. Results were obtained during 5 minutes of seated rest prior to exposure (Pre) and during the final 5 minutes of a 15 minute sauna exposure (Sauna). Data is presented as the mean ± SD (n = 10). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the pre and sauna measurements.

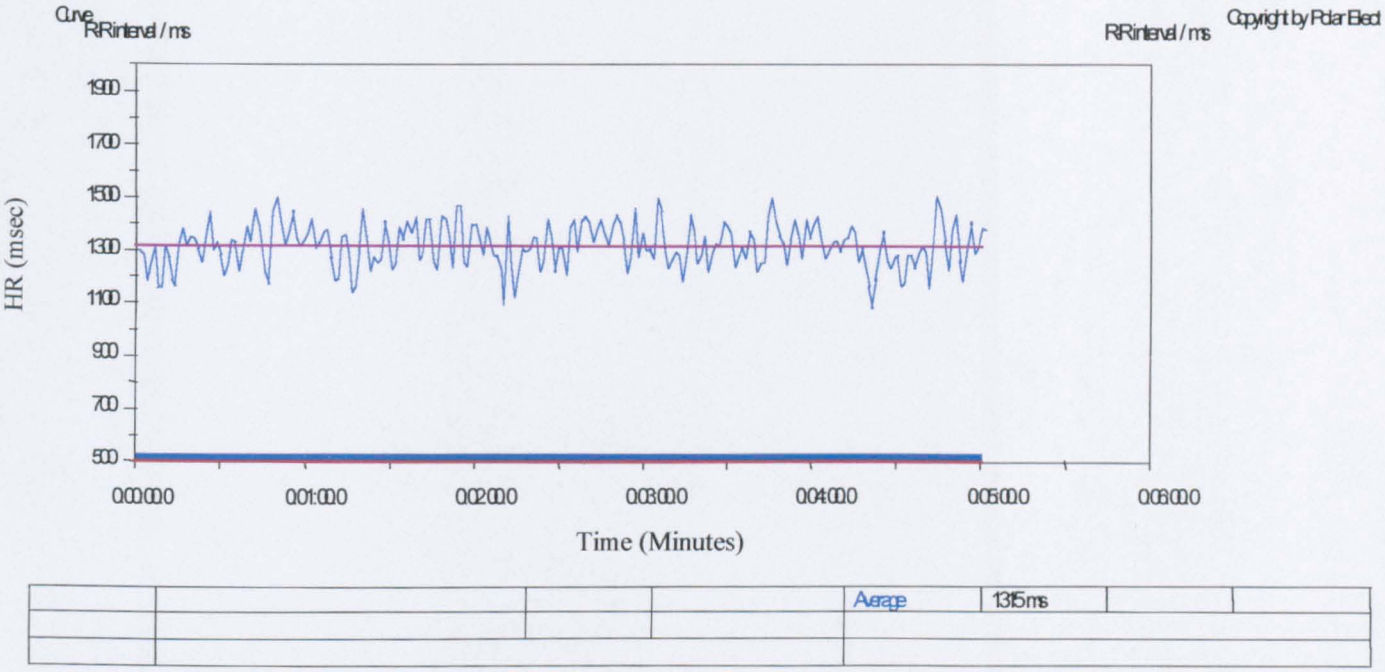


Figure 7.10 – A heart rate (HR) R-R interval tachogram (Polar software, Finland, Oy) obtained in thermoneutral conditions (room temperature was maintained at 27 (± 0.7) ^{0}C) during seated rest for a period of 5 minutes prior to sauna exposure (n = 1).

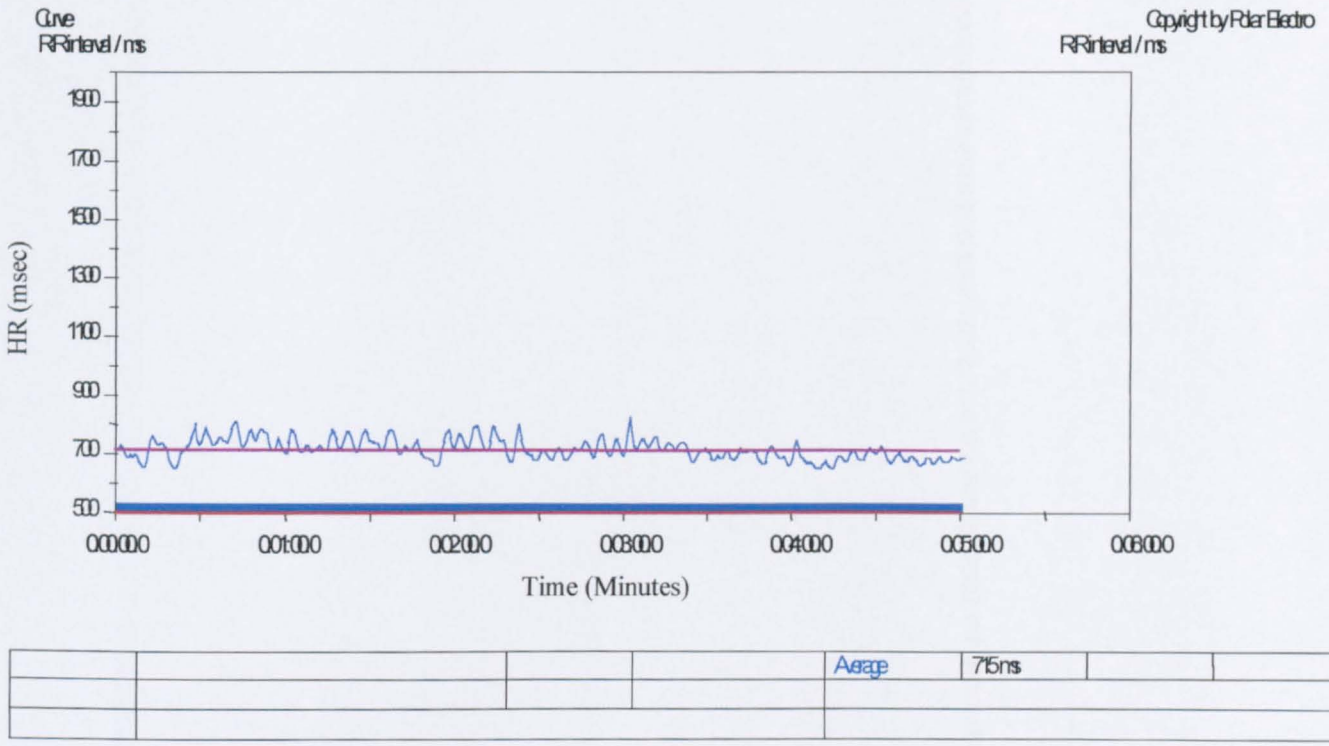


Figure 7.11 – A heart rate (HR) R-R interval tachogram (Polar software, Finland, Oy) obtained during the final 5 minutes of a 15 minute sauna exposure. Although HR has increased as a result of exposure to the sauna (66.5 (± 8.1) beats·minute^{-1} prior to exposure compared to 106.0 (± 11.7) beats·minute^{-1} during the sauna), Figure 5.11 when compared to Figure 5.10 shows that HRV has decreased (n = 1).

Figures 7.12 and 7.13 are power spectral density (PSD) graphs that represent the power or variance of the R-R intervals that are subdivided into frequency ranges. The areas on the graphs that are represented by the VLF, LF and HF frequency ranges are depicted by the different lines (refer to the key below).

The change in the tachograms in Figures 7.10 and 7.11 is further supported by the power spectral density graphs (PSD) (Figures 7.12 and 7.13) that show the reduction in the HRV components (reduction in overall power and thus HRV). Power is represented by the area under the line of the PSD graph, so when exposed to the sauna, Figure 7.13 clearly shows a reduction in the area under the line, thus representing a reduction in overall power.

Therefore, exposing subjects to a sauna for 15 minutes resulted in a response that could only be perceived as a considerable stress placed on the body from the heat. This was evidenced from a decrease in overall power (Figure 7.13), increase in the indicators of sympathetic control of HR (LF Norm and the LF:HF ratio) and a decrease in the indicators of parasympathetic control of HR (HF Norm, pNN50 and RMSSD).

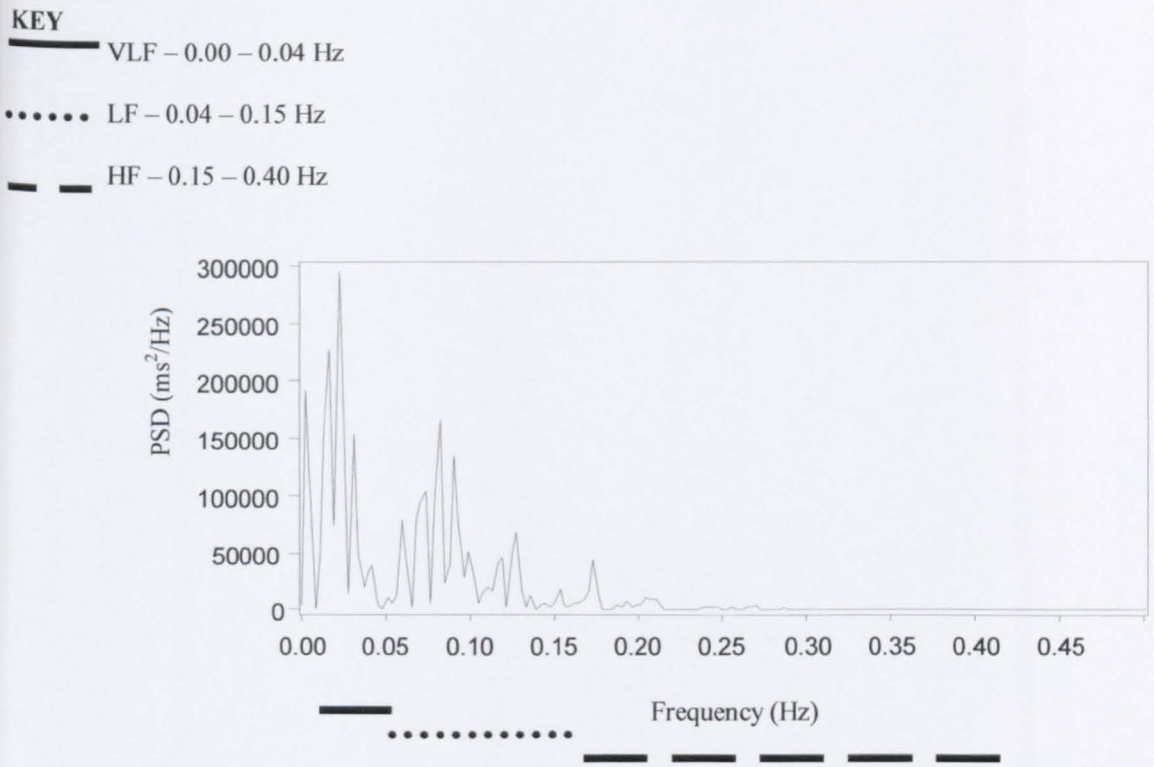


Figure 7.12 – A 5 minute sample taken after 10 minutes of seated rest prior to sauna exposure using the fast fourier method of obtaining a power spectral density (PSD) graph in thermoneutral conditions (room temperature maintained at 27 (± 0.7) ^{0}C) (n = 1). These graphs depict where the distribution of power has occurred within the HRV components. The areas on the graphs that are represented by the VLF, LF and HF frequencies are depicted by the different lines (refer to the key).

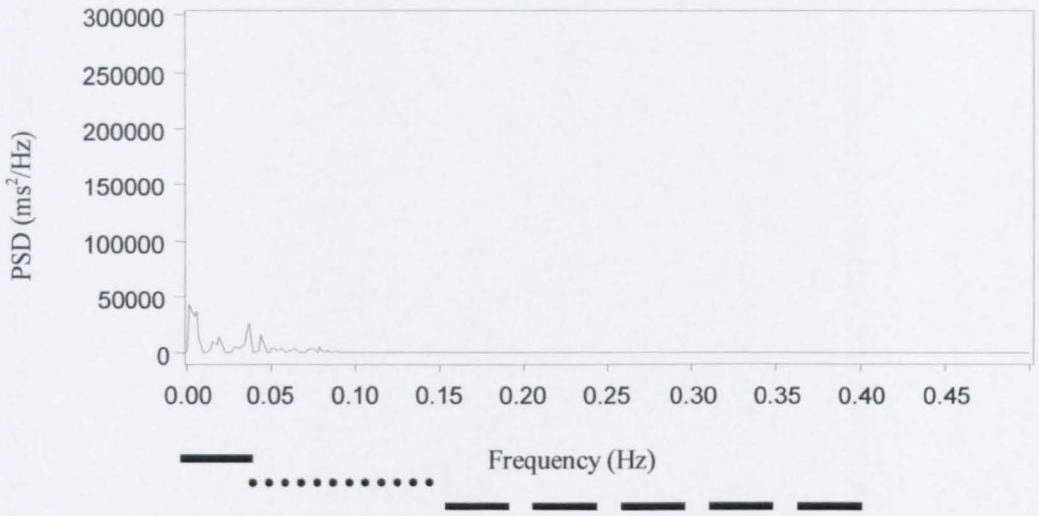


Figure 7.13 – A 5 minute sample obtained during the final 5 minutes of a 15 minute sauna exposure using the fast fourier method of obtaining a power spectral density (PSD) graph in at 74.3 (± 5.9) ^{0}C) (n = 1). These graphs depict where the distribution of power has occurred within the HRV components. The areas on the graphs that are represented by the VLF, LF and HF frequencies are depicted by the different lines (refer to the key).

7.3.3 HRV and LFTEs

The effect of exposure to mock fire training exercises and the changes caused by exposure to the LFTEs on HRV can be observed from Tables 7.5 and 7.6 (Appendix E). The results are the means of six subjects (mean age 46 (± 5.4 SD) years, weight 81.8 (± 14.6) kg, and height 176.3 (± 9.5) cm). The subjects carried out 2 mock training exercises each (training exercise where the fire was switched off and there were no students present) and 2 LFTEs each (training exercises where the fire was switched on and students were present). A training exercise schedule was developed by BAIs (Table 3.2 in the Methods), suggesting the workload was identical between both the mock exercises and LFTEs. This ensured that the only difference between the mock training exercises and LFTEs was the addition of the fire and students.

7.3.3i Physiological Responses to Mock Training Exercises and LFTEs (Protocol 1).

As expected, the results (Table 7.5, Appendix E), showed there were no significant differences between the pre mock and pre LFTE indicators of sympathetic control of HR (LF Norm and LF:HF ratio), or indicators of parasympathetic control of HR (HF Norm, pNN50 and RMSSD) whilst wearing PC+SCBA. This suggests a stress response did not occur prior to LFTEs through an anticipatory rise in HR. The experience of BAIs to carrying out LFTEs may constitute an explanation of these results.

There were no significant differences between pre and post exposure for either the indicators of sympathetic control of HR or parasympathetic indicators of HR during the mock training exercises. As a result of exposure to the LFTEs, there was a significant increase ($p<0.05$) in the post exposure sympathetic indicator of HR control (LF Norm increased by 21.5 (± 32.7) %). Also there were significant decreases ($p<0.05$) in the post exposure parasympathetic indicators of HR control (pNN50 decreased by -53.6 (± 69.8) % and RMSSD decreased by -5.3 (± 61.1) %) when compared to the pre exposure values.

There were significant increases ($p<0.05$) in HR (23.1 (± 25.2) %) between the mock and LFTEs post exposure, whilst dressed in PC+SCBA. This increase in HR through

exposure to LFTEs, appears to be mediated by a withdrawal of the parasympathetic nervous system (Fig 7.14), represented by a significant reduction ($p<0.05$) in the HF Norm values, pNN50 and RMSSD. In addition, there appears to be an increase in the predominance of the sympathetic nervous system that is represented by the significant increase ($p<0.05$) in LF Norm values by 8.5 (± 16.1) % (Figure 5.15).

There were significant decreases ($p<0.05$) in total power (TP), absolute VLF, LF, HF and SD values. This illustrates an overall decrease in HRV which was also observed during exposure to the sauna.

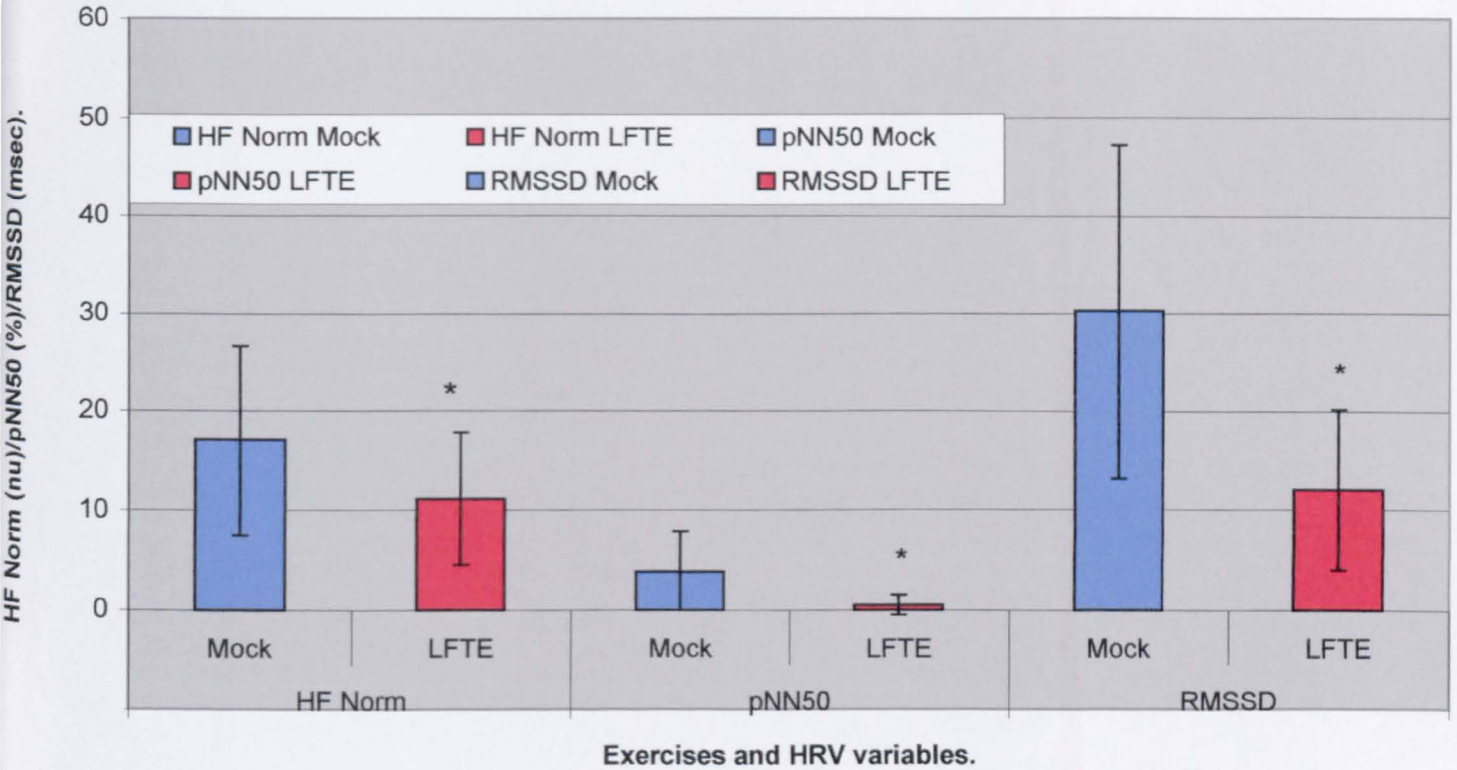


Figure 7.14 – The indicators of parasympathetic control of cardiac function; pNN50 (the number of adjacent pairs of R-R intervals differing by more than 50 ms divided by the total number of R-R intervals); RMSSD (the square root of the mean of the sum of the squares of differences between adjacent R-R intervals); and HF Norm (power in the high frequency (HF) normalised) when exposed to a mock training exercise (Mock) when the fire and students were absent from the exercises and live fire training exercises (LFTE) where the fire and students were present. Results were obtained during 5 minutes of seated rest immediately post exposure to the mock and LFTEs. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock and LFTE post exposure measurements.

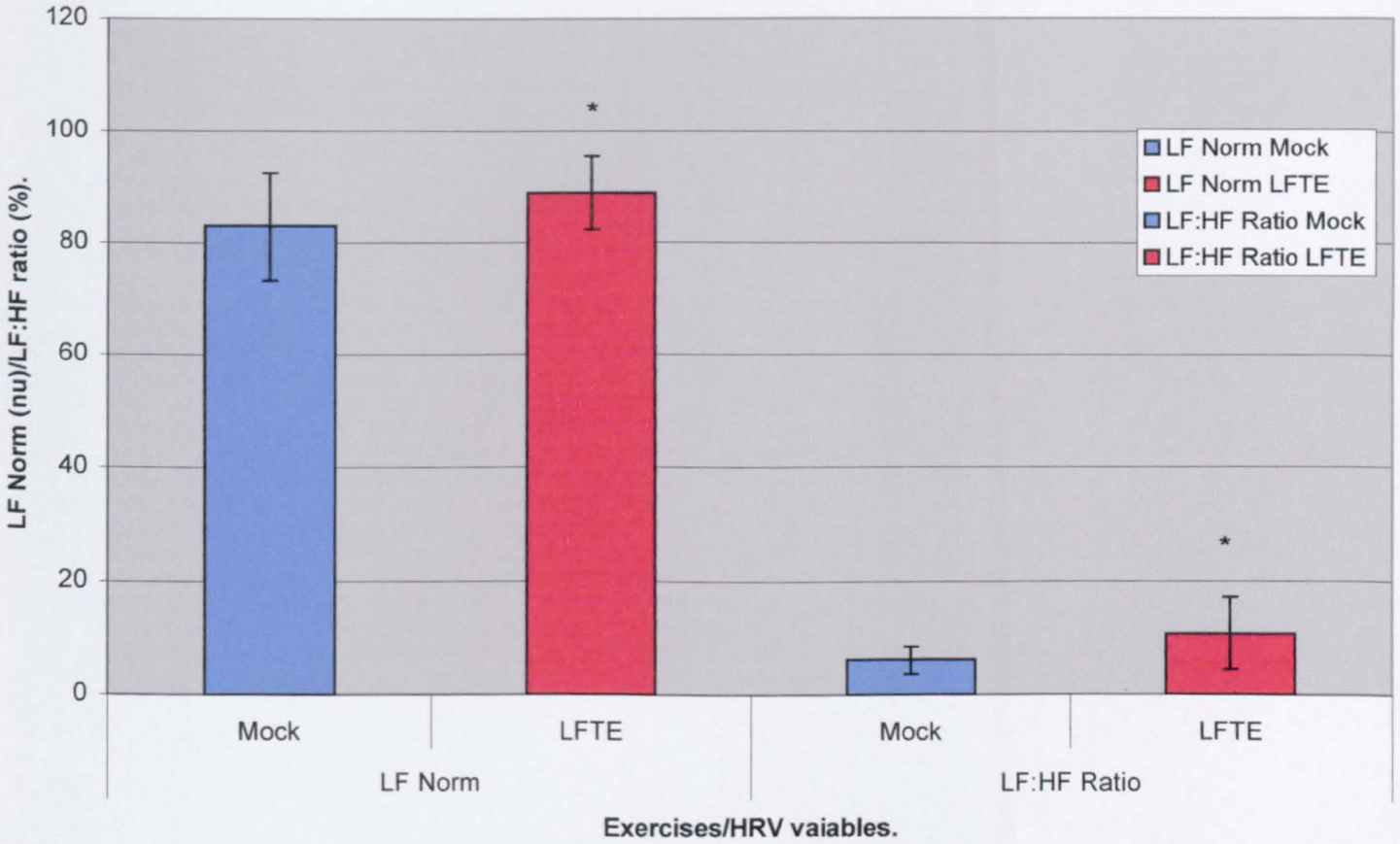


Figure 7.15 – The indicators of sympathetic control of cardiac function; LF Norm (low frequency (LF) values in normalised units); and LF:HF Ratio (ratio between normalised low frequency (LF) and normalised high frequency (HF) data) when exposed to a sauna when exposed to a mock training exercise (Mock) when the fire and students were absent from the exercises and live fire training exercises (LFTE) where the fire and students were present. Results were obtained during 5 minutes of seated rest immediately post exposure to the mock and LFTEs. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mock and LFTE post exposure measurements ($p<0.05$).

7.3.3ii Comparison of HRV responses of BAIs when they were either hydrated prior to exposure, euhydrated prior to exposure to the LFTEs (Protocol 2) or euhydrated prior to exposure but drank during the LFTEs (Protocol 3).

The results obtained prior to exposure during Protocol 2 (Table 7.8 (Appendix E)) and 3 (Table 7.10, 7.12 and 7.14 in Appendix E) showed there to be no significant differences between any of the sympathetic indicators of HR control (LF Norm and LF:HF ratio) or parasympathetic indicators of HR control (HF Norm, pNN50 and RMSSD) between the LFTEs when BAIs were either hydrated prior to exposure, euhydrated prior to exposure or drank during the LFTEs whilst dressed in PC+SCBA. The HRV results obtained post exposure to the LFTEs during Protocol 2 and 3, whilst dressed in PC+SCBA, produced no significant differences in HR between the LFTEs for any of the fluid trials.

During Protocol 2 there were significant ($p<0.05$) reductions in SD, TP, VLF, and absolute LF and HF values indicating a reduction in HRV as HR was greater post exposure to the LFTEs when BAIs were hydrated prior to exposure than the LFTEs when they were euhydrated prior to exposure. In addition, there were significant decreases ($p<0.05$) when hydrated compared to euhydrated, in the indicators of parasympathetic withdrawal (pNN50 and RMSSD) (Figure 7.16) but no significant differences in HF Norm values between the post LFTEs readings when BAIs were hydrated or euhydrated prior to exposure. There were no significant increases in LF Norm values between the LFTEs when BAIs were euhydrated or hydrated prior to exposure (Figure 7.17). Consistent with Protocol 1, there was a significant increase ($p<0.05$) in LF Norm (27.5 (± 33.3) %) and a significant decrease ($p<0.05$) in pNN50 (-55.7 (± 35.4) %) and RMSSD (-33.6 (± 44.7) %) between the pre and post exposure values when the BAIs were euhydrated prior to exposure during Protocol 2. In addition, when the BAIs were hydrated prior to exposure, there were no significant differences between pre and post exposure for the sympathetic indicators of HR control. However, there were significant decreases ($p<0.05$) in the parasympathetic indicators of HR control (pNN50 decreased -75.0 (± 45.2) % and RMSSD decreased -76.3 (± 8.6) %).

Protocol 3 was also consistent with Protocols 1 and 2. Between pre and post exposure to the LFTEs when BAIs were euhydrated prior to exposure, there were significant increases ($p<0.05$) in the sympathetic indicator of HR control (LF Norm) by 12.0 (± 8.1) %. In addition, there were significant decreases ($p<0.05$) in the parasympathetic indicators of HR control (HF Norm decreased by -46.7 (± 20.7) % and RMSSD decreased by -53.7 (± 26.2) %). When the BAIs were hydrated prior to exposure, there were significant increases ($p<0.05$) in the sympathetic indicator of HR control (LF:HF ratio) by 137.1 (± 147.6) %. Also there were significant decreases between the parasympathetic indicator of HR control (RMSSD) by -54.1 (± 26.4) % between the pre and post exposure measurements. However, there were no significant differences between the pre and post exposure measurements for either the sympathetic indicators or parasympathetic indicators of HR control when the BAIs were euhydrated prior to exposure, but drank during the LFTEs.

Post exposure (during Protocol 3), the HR and HRV variables (Tables 7.11, 7.13 and 7.15 in Appendix E) for both the sympathetic (LF Norm and LF:HF ratio) (Figure 7.18) and parasympathetic indicators of HR control (HF Norm, pNN50 and RMSSD) (Figure 7.19) were not significantly different between any of the fluid trials. However, there were significant increases ($p<0.05$) in TP and absolute VLF and LF values when the BAIs drank during the LFTEs, compared to when they were hydrated prior to exposure to the LFTEs (Table 7.13 Appendix E). Also, the indicators of parasympathetic control of HR (pNN50, and RMSSD) significantly increased ($p<0.05$) when the BAIs drank during the LFTEs, compared to when they were hydrated prior to exposure to the LFTEs, although HF Norm increased, but not significantly.

In addition, the temperature outside the FFU was not significantly different when the BAIs were hydrated prior to exposure (16.3 (± 1.1) ^{0}C) and when they euhydrated prior to exposure (16.3 (± 1.9) ^{0}C) or drank during the LFTEs (16.1 (± 1.8) ^{0}C). In turn, there were no significant differences between the temperature within the FFU during the LFTEs when the BAIs were euhydrated prior to exposure (anti-chamber 57.2 (± 7.1) ^{0}C), hydrated prior to exposure (anti-chamber 54.5 (± 7.3) ^{0}C) or drank during the LFTEs (anti-chamber 56.3 (± 11.2) ^{0}C). In addition, the skin, aural and

micro-climate temperatures were also not significantly different pre exposure to the LFTEs when the BAIs were hydrated or euhydrated prior to exposure, or drank during the LFTEs.

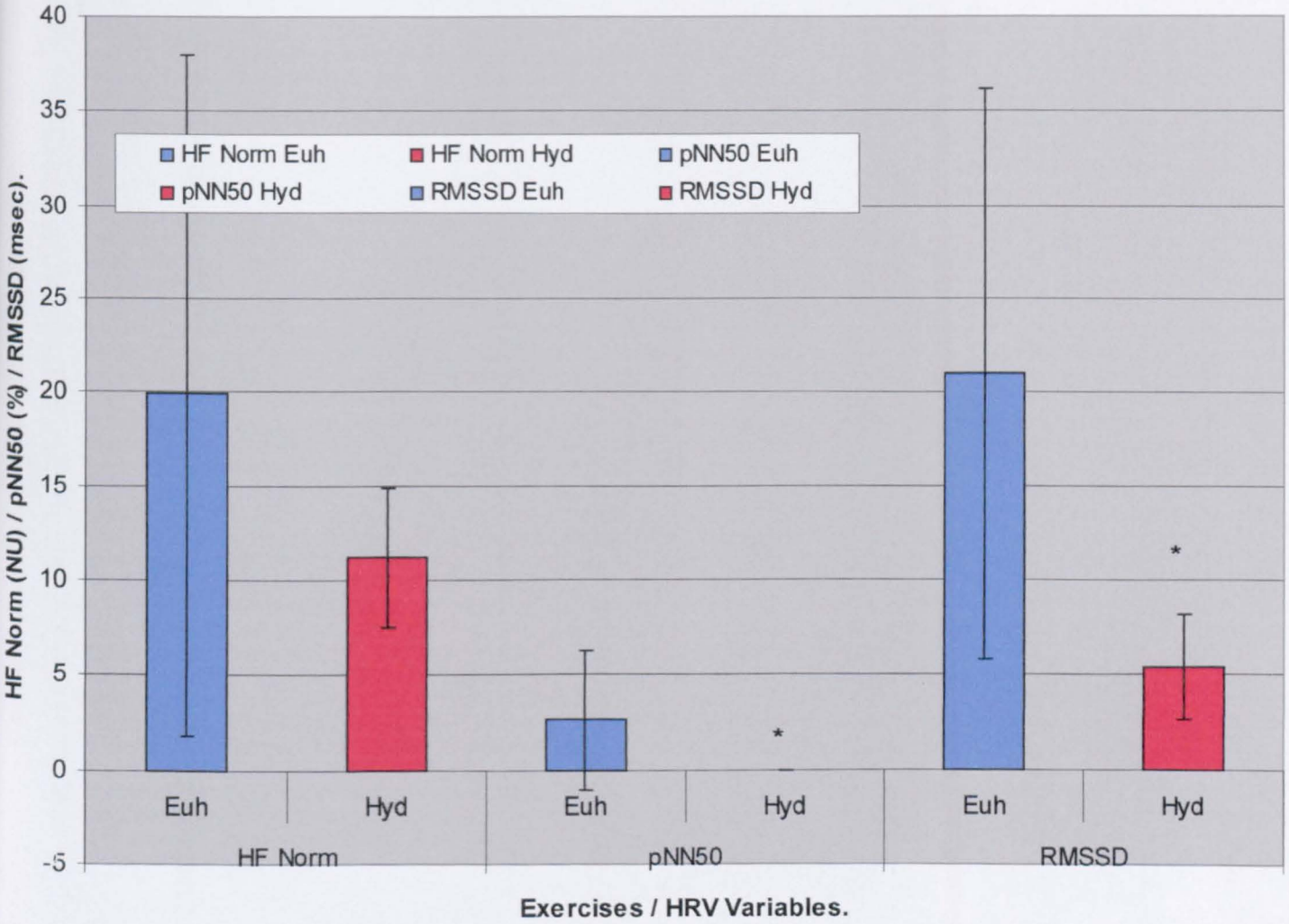


Figure 7.16 – The indicators of parasympathetic control of cardiac function; pNN50 (the number of adjacent pairs of R-R intervals differing by more than 50 ms divided by the total number of R-R intervals); RMSSD (the square root of the mean of the sum of the squares of differences between adjacent R-R intervals); and HF Norm (power in the high frequency (HF) normalised) when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated) or consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) (Protocol 2). Results were obtained during 5 minutes of seated rest immediately post exposure to the LFTEs. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the Euhydrated and Hydrated post exposure measurements.

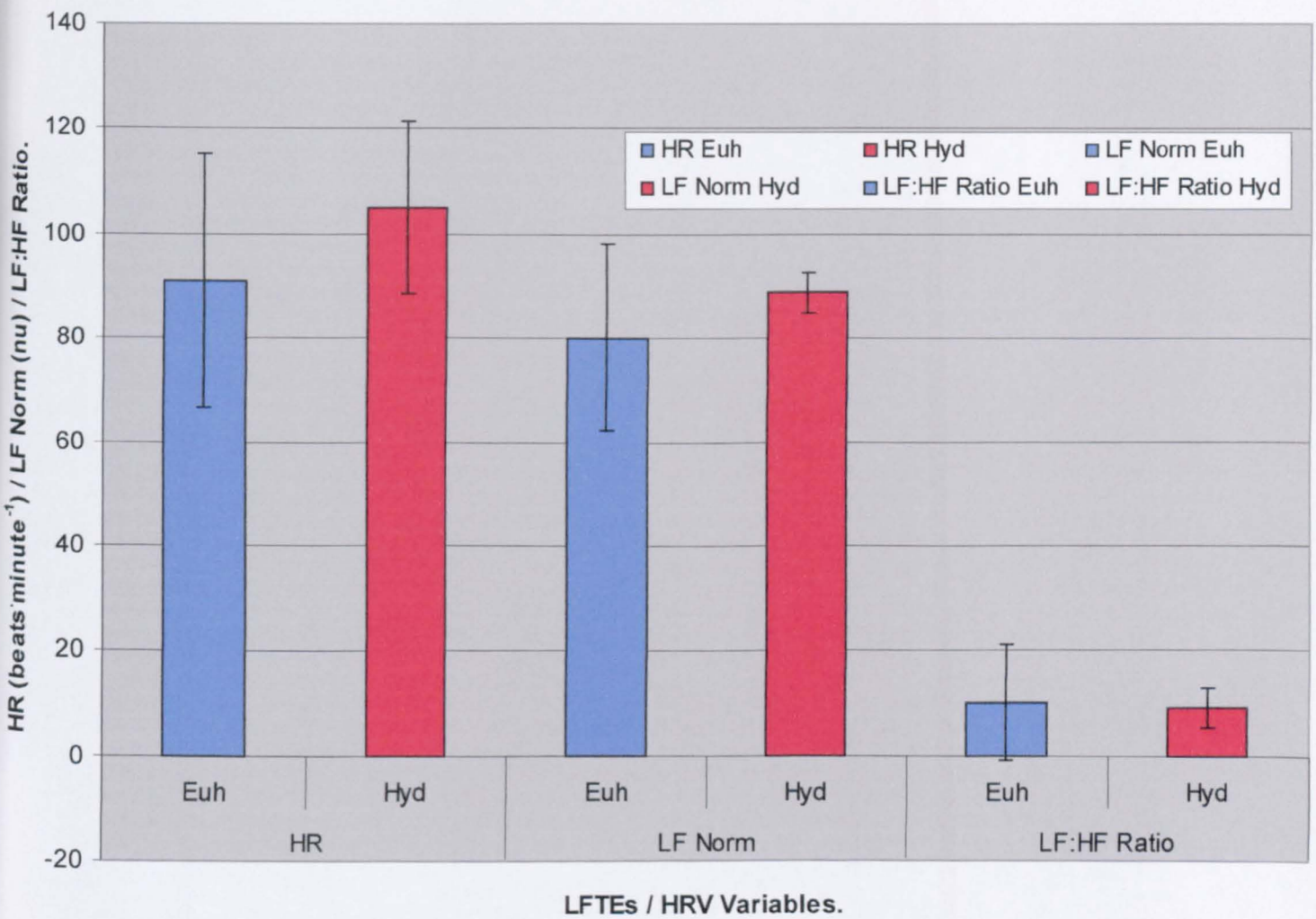


Figure 7.17 – Heart rate (HR) and the indicators of sympathetic control of cardiac function; LF Norm (low frequency (LF) values in normalised units); and LF:HF Ratio (ratio between normalised low frequency (LF) and normalised high frequency (HF) data) when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated) or consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) (Protocol 2). Results were obtained during 5 minutes of seated rest immediately post exposure to the LFTEs. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test.

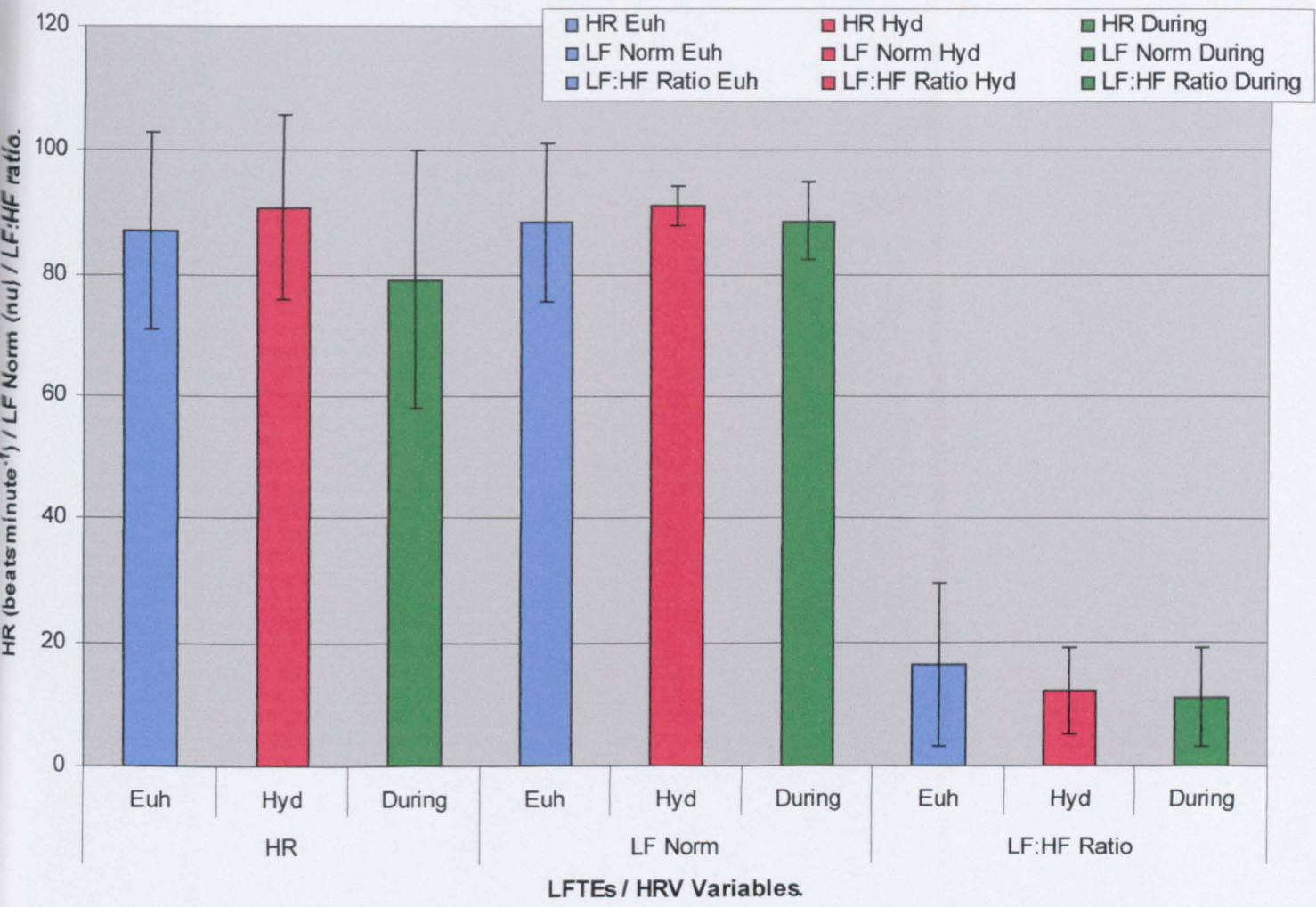


Figure 7.18 – Heart rate (HR) and the indicators of sympathetic control of cardiac function; LF Norm (low frequency (LF) values in normalised units); and LF:HF Ratio (ratio between normalised low frequency (LF) and normalised high frequency (HF) data) when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocol 3). Results were obtained during 5 minutes of seated rest immediately post exposure to the LFTEs. Data is presented as the mean and ± SD (n = 6). Significance was tested using a paired t-test.

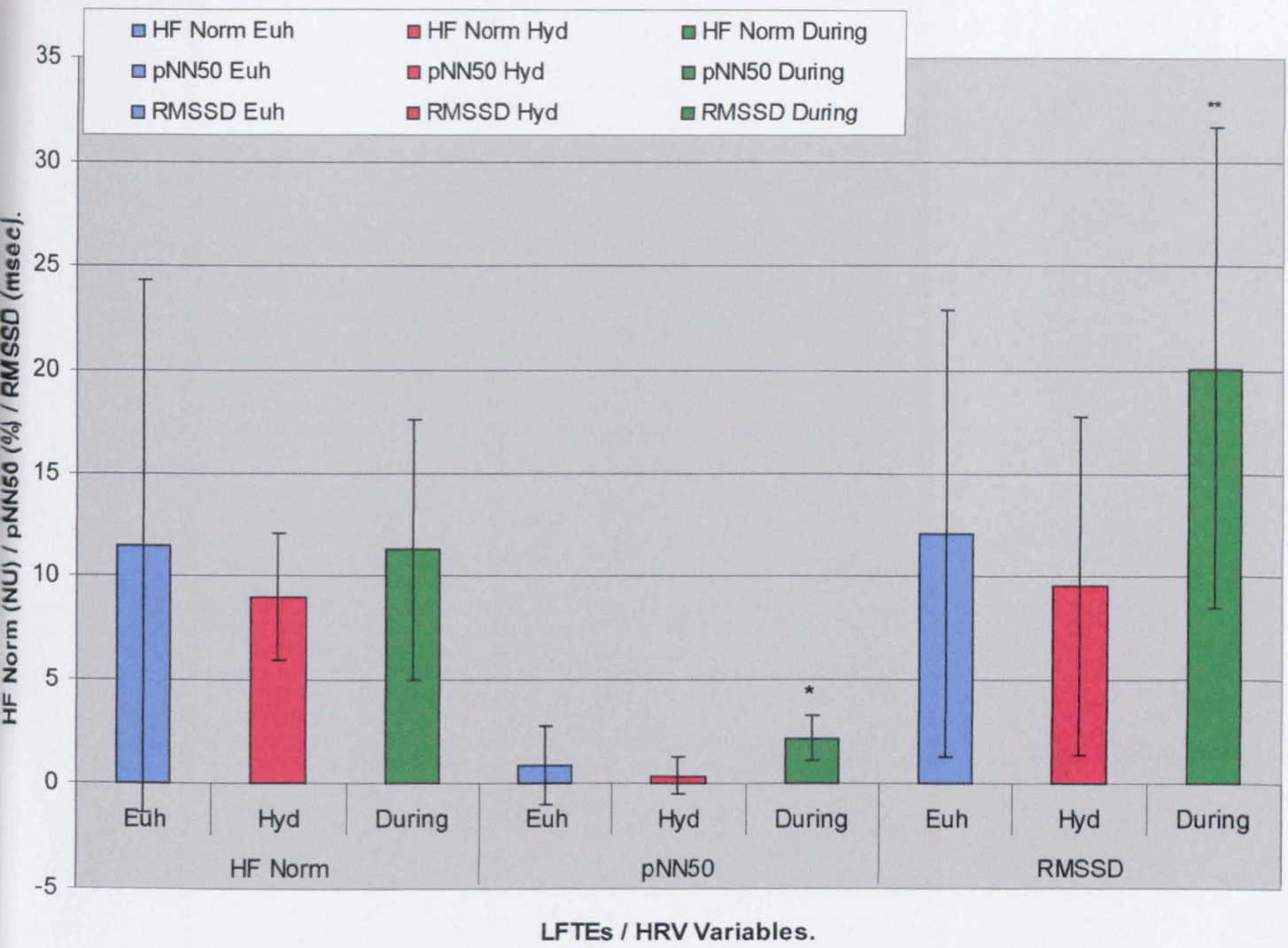


Figure 7.19 – The indicators of parasympathetic control of cardiac function; pNN50 (The number of adjacent pairs of R-R intervals differing by more than 50 ms divided by the total number of R-R intervals); RMSSD (The square root of the mean of the sum of the squares of differences between adjacent R-R intervals); and HF Norm (power in the high frequency (HF) normalised) when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocol 3). Results were obtained during 5 minutes of seated rest immediately post exposure to the LFTEs. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the Hydrated and During post exposure pNN50 measurements. ** - significant differences ($p<0.05$) between the Hydrated and During post exposure RMSSD measurements.

7.4 Discussion.

7.4.1 Controlled and spontaneous breathing on HRV.

The results obtained suggest that it is not necessary to control breathing when assessing HRV. This is consistent with the findings of Hirsch and Bishop (1989), who also observed no significant differences between controlled and spontaneous breathing on HRV. However, Stark *et al* (2000), suggest that the cortical effect of controlling breathing, irrelevant of the frequency, will reduce the HF component of HRV. The results from the present study suggest the absolute HF (Table 7.1a Appendix E) and the indicators of parasympathetic influence on heart rate (Figure 7.2) show there were non significant patterns towards a decrease in HF that was consistent with the findings of Patwardhan *et al* (1995a) and Stark *et al* (2000).

No significant differences were observed between CB and SB during this investigation for any of the indicators of parasympathetic or sympathetic control of HR. Therefore, the findings are in agreement with earlier research conducted by (Patwardhan *et* al, 1995a), whom also observed no effect on HRV from controlling breathing. Significant differences between CB and SB were not observed as the CB of 0.2 Hz did not significantly deviate from the normal metabolic needs of the subjects. Thus, the present study has shown that the cortical effect of CB does not significantly affect HRV. This suggests it is not necessary to control breathing during the other experiments e.g., when subjects are exposed to the sauna and the LFTEs (even though Patwardham *et al* (1995) have reported the need to control breathing).

During exposure to a sauna there was an increase in breathing frequency (pre-exposure 0.12 (± 0.03) Hz to 0.18 (± 0.05) Hz during exposure to the sauna ($p<0.05$)). This suggests if breathing had been controlled this may have masked the true effects of heat exposure through increasing the stress placed on the subjects. Therefore, breathing was not controlled.

7.4.2 The effect of sauna exposure on HRV.

When exposed to the sauna there was an increase in stress placed on the subjects, as measured by an increase in the skin and aural temperatures. Skin temperature

increased from 33.0 (± 0.9) ^{0}C prior to sauna exposure to 41.9 (± 1.5) ^{0}C during the final 5 minutes of sauna exposure and aural temperature increased from 37.0 (± 0.6) ^{0}C during the initial 5 minutes when compared to 38.8 (± 0.5) ^{0}C during the final 5 minutes of sauna exposure. There was also a significant increase ($p<0.05$) in the time taken to carry out the adapted Stroop test between pre and post-exposure to the sauna. Muller and Schumann (1894) recognised the need to be aware of the practice effect when dealing with tests such as these. Therefore, each subject in the present study was given opportunity to practice the adapted Stroop prior to the sauna exposure. Thus, the increase in temperature and time taken to complete the adapted Stroop test suggest it was the effect of the heat and/or the dehydration that resulted in the slower completion time.

It is known that dehydration affects psychological performance (Greenleaf, 1992, Brooks *et al*, 1996). During the present study body mass losses were 0.65 (± 0.3) kg that equated to 0.9 (± 0.5) % of the subjects body weight. According to Brooks *et al* (1996), body mass losses of as little as 1% could result in losses of concentration. The present study's findings confirm that may well be the case with losses as little as 0.9 (± 0.5) % of body mass.

The increase in HR through a thermoregulatory effect is well documented (Fortney and Vroman, 1985). This increase in HR is usually accompanied by other thermoregulatory responses that include redistribution of blood through vasodilatation. The process of vasodilatation involves the opening of new skin capillary beds and dilatation of cutaneous veins which allows a greater time for the transfer of heat from the blood to the cooler skin, potentially through a cholinergic mechanism (Fortney and Vroman, 1985). However, when ambient temperature is greater than skin temperature, heat transfer is reversed, resulting in an increase in heat storage. The increase in temperature was highlighted in Figure 7.7, whereby the ambient temperature was significantly increased (room temperature was 27 (± 0.7) ^{0}C and increased to 74.3 (± 5.9) ^{0}C during the final 5 minutes of sauna exposure) over that of the skin temperature. The skin temperature increased from 33.0 (± 0.9) ^{0}C prior to sauna exposure to 41.9 (± 1.5) ^{0}C during the final 5 minutes of sauna exposure. This, in turn, created an increase in aural temperature which increased

from 37.0 (± 0.6) ^{0}C during the initial 5 minutes compared to 38.8 (± 0.5) ^{0}C during the final 5 minutes of sauna exposure. The increases in these temperatures produced a corresponding increase in heart rate. The increase in blood flow increases HR through the intensified requirement on cardiac output which is driven from the increases in core temperature and skin temperature (Fortney and Vromen, 1985).

The present study elicited higher values for the HRV components than those reported by Delaney and Brodie (2000), who observed the effect of mental stress during thermoneutral conditions. The present study was carried out identically to that of Delaney and Brodie (2000). Therefore, the results indicate that the differences have been caused by the significantly different temperatures the subjects were exposed to between the present study (74.3 (± 5.9) ^{0}C) and that of Delaney and Brodie (2000) (22-25^{0}C).

According to the Task Force (1996), the standard deviation (SD) is an indication of overall HR variability. The SD values obtained during the present study show a significant decrease ($p<0.05$). Delaney and Brodie (2000) state a reduction in the SD is indicative of the overall reduction in autonomic nervous system activity, hence a reduction in HRV (Task Force, 1996). The significant decrease ($p<0.05$) in SD may be caused by the increase in heat placed on the body from exposure to the sauna and, in turn, would reflect changes in the areas of the autonomic system that the present study observed (HR and temperature). A significant reduction in overall HRV was also observed by Parsons *et al* (1992) when they exposed subjects to an environment of 27^{0}C compared to when they were exposed to 17^{0}C.

Figure 7.9 shows pNN50 and RMSSD have lowered significantly ($p<0.05$) as a result of exposure to the sauna. This signifies a reduction of the influence of the parasympathetic nervous system on HR, thus, allowing an increase in the HR response during exposure to the sauna.

Klieger, *et al* (1987) and Delaney and Brodie (2000), argue that HF values are highly correlated to pNN50 and RMSSD values as they are all estimates of the HF variations (Task Force, 1996). The HF values from the present study were correlated

to pNN50 and RMSSD at rest (r=0.8 for both parameters). During exposure to the sauna the correlations for HF to pNN50 and RMSSD were r=0.6 for both parameters suggesting the findings from the present study are in agreement with others (Klieger, *et al*, 1987, Brenner *et al*, 1998, Delaney and Brodie, 2000). The results strongly suggest the pNN50 and RMSSD are indeed indicators of HF variations, which in turn, is an indicator of parasympathetic control of HR.

The absolute LF values (msec) showed significant decreases ($p<0.05$) between rest in thermoneutral conditions and rest in the sauna (refer to Table 7.4a Appendix E). This was also observed from the changes between Figures 7.12 and 7.13. The absolute values when taken at face value suggest erroneous results (Delaney and Brodie, 2000). The Task Force (1996) suggests the absolute LF may also be measured in normalised units (nu), thus representing the balanced and controlled behaviour of the autonomic nervous system, minimising the effects of changes in the total power on the absolute LF and HF. However, both the absolute and normalised values should be reported (Task Force, 1996). When the absolute LF results were normalised the results showed significant increases ($p<0.05$) for LF Norm. Brenner *et al* (1998), state the ratio of LF to HF (LF:HF) is a more conclusive indicator of sympathetic influence than purely the LF Norm value. From the results of the present study there was a dramatic increase in the ratio ($p<0.01$). This strongly suggests that there was an increase in the sympathetic drive, which, in turn increased the HR in response to the heat of the sauna. The absolute HF values (msec), like the absolute LF values, also showed significant decreases ($p<0.05$) between rest in thermoneutral conditions and rest in the sauna (refer to Table 7.4a Appendix E). However, when the absolute HF values are normalised there were significant decreases ($p<0.05$) in the HF Norm values (Figure 7.8).

The HF Norm and LF Norm suggest that the increase in HR is not only from an increase in vagal withdrawal (HF Norm was reduced by 82.7 (± 11.4) %), but also an increase in stimulation from the sympathetic nervous system (LF Norm was increased by 84.5 (± 76.5) %). These findings are consistent with Brenner *et al* (1997). Indicators of an increase in sympathetic predominance and parasympathetic

withdrawal as a result of sauna exposure can be observed from Figures 7.8 and 7.9 respectively.

The subjects in the present study were exposed to a greater physiological stress when compared to those in the study by Delaney and Brodie (2000) who were exposed only to a psychological test. As such, the percent change in HF Norm and LF Norm (see above) appear to be in excess of those reported by Delaney and Brodie (2000). Their study reported a decrease in HF Norm by 30% and LF Norm increased by 9.1%. It can be inferred that the adapted Stroop test did not affect the HRV data as subjects were allowed to practice the test prior to monitoring, thus minimising the effect on HRV.

Brenner *et al* (1997) reported that there is very little research into the effects of heat exposure on HRV. However, the Task Force (1996), state that light exercise will induce increases in LF Norm and decreases in HF Norm (Brenner *et al*, 1998). This is consistent with the findings of the present study when exposed to heat.

Brenner *et al* (1998) also observed an increase in LF Norm values after intense exercise that remained elevated. This suggests there was an input from both the sympathetic and parasympathetic influences of cardiac control. Fagraeus and Linnarsson (1976), observed at the onset of light dynamic exercise (50 watts at 60 revolutions·minute^{-1}) the initial increase in HR occurred through a rapid vagal withdrawal, followed by an increase in sympathetic mediation. However, they were unable to conclusively show the origin of this increase in HR at the onset of exercise.

Gorman and Proppe (1984) investigated the effect of heat stressed conscious baboons to observe the changes in sympathetic activity by elevating the arterial blood temperature by 1-2°C over a period of 1-2 hours. The baboons produced tachycardic responses (increase in 20 (± 1.2 (SEM)) beats·minute^{-1}) where non-autonomic mechanisms accounted for 40% of these responses, 40% from vagal withdrawal and 20% from an increase in sympathetic stimulation of the HR. There was increase in HR of 60.7 (± 18.3) % in response to the sauna in the present study. This was mediated by an -82.7 (± 11.4) % decrease in HF Norm, and therefore

parasympathetic influence, and an 84.5 (± 76.5) % increase in LF Norm, and therefore sympathetic influence on cardiac control. During heat exposure, the vagal withdrawal and concomitant increase in sympathetic stimulation has also been reported elsewhere (Kinugasa and Hirayanagi, 1999).

Research is still unclear as to the extent of the parasympathetic and sympathetic drives that contribute to the LF Norm readings (Brenner *et al*, 1998). However, the present study's findings support the studies of Delaney and Brodie (2000) and Task Force (1996) who also state that the HF Norm readings predominately indicate parasympathetic drive, and the LF Norm is indicated predominately by the sympathetic drive.

Previous research is still ambiguous as to the significance of the VLF readings. The very low frequency (VLF) values significantly decreased ($p<0.05$) (Table 7.4a Appendix E) when subjects were exposed to a hot environment. This was depicted by the differences between Figures 7.12 and 7.13. However, the VLF component is less well defined. Research has been unable to establish a specific physiological process that it can be attributed to (Task Force, 1996). Kinugasa and Hirayanagi (1999) state that the VLF frequency is not very well described in the research and therefore requires further research in order to successfully interpret the data. In addition, the Task Force (1996), state that VLF readings should be avoided during short term readings such as 5 minute test periods. This is due to the VLF frequency having a non-harmonic component which is affected by trend removal and does not have any coherent properties. Although VLF results are reported, due to the inconsistent findings on the significance of the VLF readings, they have not been included in the analysis.

The results from the study suggest there is an increase in HR when subjects were exposed to heat. This occurs through an initial reduction in parasympathetic drive (indicated by a reduction in HF Norm, pNN50 and RMSSD) that is followed by a predominance of sympathetic drive (indicated by increases in LF Norm and LF:HF ratio) of the autonomic system. These results suggest there is a stress response experienced on exposure to hot environmental conditions. It appears that this

response may, in part, be triggered by the increase in skin temperature as a result of the significant increase in environmental temperature during exposure to the sauna.

Therefore, the gradient for heat exchange during the pre exposure measurements that allowed heat from the core to dissipate was reversed during the sauna exposure. Consequently, heat was not dissipated from the skin but stored. This in turn increased the core temperature (from 37.0 (± 0.6) ^{0}C during the initial 5 minutes compared to 38.8 (± 0.5) ^{0}C during the final 5 minutes of sauna exposure). Heat stress, as measured by HRV, has shown a decrease in parasympathetic and increase in the sympathetic indicators of HR control which is consistent with other methods of measuring autonomic activity to the heart during heat stress.

7.4.3 Physiological Responses to Mock Training Exercises and LFTEs (Protocol 1).

The increased levels of stress placed on BAIs as a result of the addition of fire and students during the LFTEs, was observed by a significant increase in HR ($p<0.05$). The increase in HR appears to have occurred through an increase in vagal withdrawal indicated by a significant decrease ($p<0.05$) in HF Norm, pNN50 and RMSSD values. In addition, there was a significant increase in the sympathetic control of HR, as evidenced from the significant increase in LF Norm between pre and post exposure to the LFTEs. This was in comparison to no significant changes in LF Norm between the pre and post exposure readings during the mock training exercises.

The decreases in HF Norm values (24.5 (± 47.2) %) are similar to the findings of Delaney and Brodie (2000) who reported a 30% reduction in HF Norm, (but are less than the decrease elicited during the sauna exposure). The differences could be accounted for by the environmental temperature during the LFTEs equating to 48.3 (± 4.0) ^{0}C (in the anti-chamber) where they were intermittently exposed to extreme heat. However, during exposure to the sauna the temperature was 74.3 (± 5.9) ^{0}C whereby the subjects were constantly exposed to the heat stress. Also, the subjects in the sauna study were measured during the exposure and BAIs were monitored post exposure to the LFTEs for HRV. There was not a 5 minute interval during the LFTEs

whereby BAIs were physically inert to allow HRV measurements to be obtained. This was also supported by the skin temperatures, which were significantly greater ($p<0.05$) during the sauna exposure (41.9 ($\pm$ 2.0) ^{0}C) when compared to post LFTEs (34.9 ($\pm$ 2.1) ^{0}C). Mean skin temperatures measured post exposure to the LFTEs were 37.9 ($\pm$ 1.3) ^{0}C and were also significantly lower ($p<0.05$) than those elicited during the sauna exposure. This was due to the BAIs being monitored post exposure to the LFTEs.

The increased HR during LFTEs was also due to an increase in sympathetic predominance indicated by significant increases ($p<0.05$) in both the LF Norm values and the LF:HF ratio. This was also in conjunction with a significantly higher ($p<0.05$) skin, micro-climate and aural temperature when the mock training exercises were compared to the LFTEs. The large increase in skin, micro-climate and aural temperatures during LFTEs suggests the need to lower these temperatures, which, in turn, would decrease the cardiorespiratory stress placed on BAIs. The results from Chapter 4 also support reduction of skin temperature in relation to reducing the cardiorespiratory stress of BAIs when exercising.

Therefore, results from both Chapters 4 and 5 and also the HRV data, imply that lowering the skin temperature, may, in turn lower the amount of increased sympathetic drive and vagal withdrawal during heat exposure. This would lessen the stress placed on BAIs during LFTEs.

The Task Force (1996) suggest the use of HRV for diagnosis of a variety of heart conditions. Table 7.2 shows the example of a typical HRV response after a Myocardial Infarction (MI), which is also used for a prognosis of heart failure after a MI. These same indicators of MI were also observed during heat stress, observed from exposure to both the sauna and post exposure to LFTEs. However, these indicators are also seen post exposure to physical exercise and psychological stress.

Table 7.2 – Prediction of mortality of patients post MI using HRV variables compared to HRV responses to heat stress observed during the sauna exposure, LFTEs, physical exercise and psychological stress. ↓ - Denotes decrease in HRV variable / ↑ - Denotes increase in HRV variable.

HRV Variables	MI	Heat Stress	Physical Exercise	Psychological Stressor
LF Norm	↑	↑	↑	↑
HF Norm	↓	↓	↓	↓
LF:HF Ratio	↑	↑	↑	↑
TP	↓	↓	↓	↓
Absolute LF	↓	↓	↓	↓
Absolute HF	↓	↓	↓	↓

Therefore, it could be hypothesised that if BAIs were regularly exposed to these 'heat-induced' stressful situations where they are exhibiting all the typical HRV responses that are observed after a MI, they may be more prone to MI attacks. This supports the need to lower the LF Norm values and LF:HF ratio, whilst increasing the HF Norm values. This would be to reduce the likelihood of health problems occurring in BAIs throughout their future profession. Underlying biological factors are usually associated with the aetiology of MI. However, there is no reason to suspect that these factors are relevant to the BAIs. However, increased HR over time would suggest that further research may be of benefit in this respect.

7.4.4 Comparison of the HRV responses of BAIs when either hydrated prior to exposure, euhydrated prior to exposure (Protocol 2), or euhydrated prior to exposure but drank during the LFTEs (Protocol 3).

During Protocol 2, the hypothesis that drinking prior to LFTEs would lower the HR responses, which may attenuate the negative effects of fluid loss during heat exposure, was tested. Therefore, there would be an increase in the indicators of parasympathetic influence and decrease in the sympathetic influence of HR control.

The HR results obtained post exposure to the LFTEs showed no significant differences when BAIs were in a hydrated state prior to exposure, compared to when they were euhydrated prior to exposure. There were significant decreases ($p<0.05$) in the parasympathetic indicators of HR control (pNN50 and RMSSD) suggesting an increase in vagal withdrawal. This signifies that there were no beneficial effects of

hydrating the BAIs prior to exposure to LFTEs when compared to the BAIs being euhydrated prior to exposure. In addition, when comparing the sympathetic indicators of HR control, there were no significant differences between the LFTEs when BAIs were hydrated or euhydrated prior to exposure. The small increase in HR during the LFTEs when BAIs were hydrated prior to exposure, when compared to the LFTEs when BAIs were euhydrated prior to exposure, appeared to be mediated by the decrease in parasympathetic control of HR as indicated by the reduction in pNN50 and RMSSD. The results suggest that hydration alone cannot negate the effects of the high ambient temperatures that BAIs were exposed to during the FFU. These results are consistent with McLellan *et al* (1999), who reported that uncompensatable heat stress negates the positive physiological responses of hydration. Therefore, 600 ml of water drank prior to the LFTEs, was not adequate to significantly reduce the stress placed on the BAIs, as evidenced by the HRV results.

The results from Protocol 3 confirmed that hydrating the BAIs prior to exposure appears to have no effect on pre exposure HRV variables when compared to euhydrated BAIs prior to exposure.

There were no significant differences in the responses of the sympathetic or parasympathetic indicators of HR control between the LFTEs when the BAIs were hydrated prior to exposure, compared to when the BAIs were euhydrated prior to exposure to the LFTEs. However, when the BAIs were euhydrated prior to exposure, there were significant increases in the sympathetic indicator (LF Norm) of HR control (12.0 (± 3.3) %) between the pre and post exposure measurements that were consistent with both Protocol 1 and 2. This suggests that when no fluid was consumed either prior to or during LFTEs, created an increase in the sympathetic indicators of HR control which were mediated by adrenaline release, suggesting an increase in stress. However, when the BAIs drank during the LFTEs, this produced no significant differences between the pre and post exposure measurements for either the sympathetic or parasympathetic indicators of HR control.

There does appear to be a relationship between increases in skin temperature (Figure 6.4 Chapter 6) and an increased stress response as indicated by an increase in

sympathetic mediation of HR control (Figure 7.18). This has been reported earlier in this chapter when HRV was monitored during exposure to a sauna. The post exposure HRV readings showed there to be a significant increase in parasympathetic predominance of HR control (pNN50 and RMSSD) when the BAIs drank during the LFTEs, compared to drinking prior to the LFTEs. This suggests there was an increase in vagal tone through drinking during the LFTEs, over that of drinking prior to exposure. Therefore, the decrease in HR observed when the BAIs drank 200 ml of water every 10 minutes during the LFTEs, compared to drinking before the LFTEs, was predominately as a result of an increase in the parasympathetic drive.

Chapter 6 suggests there were lower skin and micro-climate temperatures (Figure 6.7) when the BAIs drank during the LFTES. This allowed heat stored within the PC to dissipate in comparison to the BAIs who were hydrated or euhydrated prior to exposure. This was consistent with the parasympathetic and sympathetic indicators of HR control that also suggested that drinking during the LFTEs induced lower stress levels.

There were no significant differences between any of the sympathetic (Figure 7.18) or parasympathetic (Figure 7.19) indicators of HR control between the LFTEs when BAIs drank during the LFTEs and when they were euhydrated prior to exposure. There were significant increases in the pNN50 and RMSSD when BAIs drank during the LFTEs, compared to being hydrated prior to exposure. This suggests less stress was placed on the BAIs when they drank during the LFTEs. Also, there were clear patterns to suggest that drinking during the LFTEs did elicit lower stress responses. For example, the post LFTE HR was lower (9%) and there were decreases in the indicators of sympathetic control of the autonomic nervous system LF Norm and LF:HF ratio values (Task Force (1996) and Brenner *et al* (1998)).

When BAIs drank during the LFTEs, the post exposure urine colour resulted in a low urine colour (2 (± 0.8)). This suggested better hydration than the post LFTEs urine colour (3 (± 0.8)) when the BAIs were euhydrated prior to the LFTEs. Neither the temperature outside the FFU nor the temperature inside the FFU were significantly different between drinking protocols, nor were the workloads different between the

three fluid trials. This suggests, by intermittently leaving the hot environment of the FFU to a cooler environment to drink 200 ml of water every 10 minutes appears to alleviate in part, the stress incurred during LFTEs.

7.5 Conclusions.

In summary, the results suggest that when subjects are continuously subjected to extreme heat, as experienced during exposure to a sauna (74.3 (± 5.9) ^{0}C), the heat induces a stress response as indicated by a significantly increased HR. This increase in HR appears to occur through a reduction in parasympathetic control of HR (indicated by reduced HF Norm, pNN50, and RMSSD) with a simultaneous significant increase in sympathetic control of HR, (indicated by an increased LF Norm and LF:HF ratio).

During Protocol 1 the results obtained post exposure to the mock exercises showed very little change in LF Norm, with a 2.4 (± 11.4) nu increase and a similar 2.4 (± 11.4) nu decrease in the HF Norm. These results suggest there is very little stress placed on BAIs during the mock training exercises through wearing the PC+SCBA and carrying out the low intensity exercise necessary during training exercises.

However, with the addition of fire and students to the LFTEs, post exposure parasympathetic (HF Norm decreased -24 (± 47.2) % and pNN50 by -53.6 (± 69.8) %) and sympathetic (LF Norm increased 8.5 (± 16.1) %) indicators of HR control were significantly different compared to the post exposure mock training exercises. This suggests the inclusion of heat and students caused a heat stress response. These results were consistent with those observed within the sauna, suggesting a heat stress response occurred during exposure to the LFTEs. Therefore, the addition of heat creates a large amount of stress on the BAIs above that of wearing the PC+SCBA and the workload of carrying out the LFTEs.

When HRV was monitored pre and post exposure to LFTEs, the parasympathetic and sympathetic indicators of HR control did not show as great a percentage change as the sauna results. These differences between the LFTEs and sauna results can be explained through increased skin temperature during the sauna exposure (8.8 (± 2.1)

^{0}C) compared to typical increases in skin temperatures during the LFTEs (3.8 (± 1.7) ^{0}C). The differences in skin temperature can be explained by the subjects' continual exposure to the heat during the sauna, whereas, during the LFTEs, the BAIs interspersed heat exposure to the fire with debriefs where the fire was switched off. This may suggest that debriefs act as a useful interlude to the BAIs, thus, reducing the level of stress placed upon them.

The results in Protocol 2 showed there to be no significant differences in mean HR during the LFTEs when BAIs were hydrated prior to exposure, compared to the LFTEs when BAIs were euhydrated prior to exposure. This suggests that hydrating BAIs prior to exposure to LFTEs does not lower the heat induced stress responses.

During Protocol 3, there were significant increases in TP and absolute VLF and LF values when the BAIs drank during the LFTEs, compared to when they were hydrated prior to exposure to the LFTEs. Also, the indicators of parasympathetic control of HR (pNN50, and RMSSD) significantly increased when the BAIs drank during the LFTEs, compared to when they were hydrated prior to exposure to the LFTEs. Therefore, it was possible to lower HR responses, lower the sympathetic control on HR and increase the parasympathetic control on HR when the BAIs drank during the LFTEs.

In addition, the lower HR responses were often accompanied by lower skin temperature responses. This was caused by BAIs exiting from the FFU intermittently to drink, which allowed heat from the PC to escape, which also lowered the micro-climate temperature. The lower micro-climate, which in turn lowered skin temperature, was also accompanied by a lower aural temperature response. This suggests that there was less heat being stored in the body which resulted in a lower HR response.

Also, there were no significant differences between the pre and post exposure measurements for either the sympathetic indicators or parasympathetic indicators of HR control when BAIs drank during the LFTEs. However, there were significant increases in the sympathetic and parasympathetic indicators of HR control when

BAIs were either hydrated or euhydrated prior to exposure. This suggests that drinking during exposure lowers the stress response to the LFTEs when compared to either not drinking (euhydrated) or drinking prior to exposure (hydrated).

Urine colour increased (meaning the BAIs were more dehydrated) as a result of exposure to the LFTEs when BAIs were euhydrated prior to exposure (when BAIs did not dink prior to exposure). However, there were no changes between the pre and post exposure urine colour results when BAIs drank during the LFTEs. This further supports the findings which suggest there was less stress placed on BAIs when they drank during the LFTEs. Therefore, removing BAIs from the FFU to drink intermittently during LFTEs has the effect of reducing the effects of heat stress placed upon them.

As previously stated, exposure to heat appears to produce the same HRV responses that post MI patients exhibit (Table 7.3). However, drinking during exposure to the LFTEs alters the HRV values from those that are similar to the response observed post MI. These responses include increased sympathetic indicators of HR control and decreased parasympathetic indicators of HR control. Instead, the HRV responses produced when BAIs drank during the LFTEs exhibit decreases in the sympathetic indicators of HR control and increased parasympathetic indicators of HR control. This, in turn, may reduce the likelihood of BAIs having an increased prevalence of heart conditions over the long term period. At present there is no data available on this prevalence. Also, drinking during LFTEs appears to lower skin temperature and therefore the amount of heat stress placed on BAIs during LFTEs (Table 7.4).

Additional research is required to investigate the use of HRV monitoring in providing important information about the health status of BAIs during LFTEs, in particular the likelihood of monitoring myocardial infarctions. Despite the complex analysis post test, HRV can be monitored using the Polar (Poland Electro Oy, Kempele, Finland) watch and software and is therefore a suitable method for field testing.

Table 7.3 – Summary of parasympathetic and sympathetic indicators of HR control responses to heat stress situations and a post MI HRV response. Changes are between pre exposure and post exposure measurements (or pre and during exposure for the sauna study). Sauna (n = 10), LFTEs when the BAIs were euhydrated prior to exercise (n = 6), hydrated prior to exercise (n = 6) and when the BAIs were euhydrated prior to exposure but drank during the LFTEs (n = 6). The information presented on MI (Myocardial Infarction) is taken from the Task Force (1996).

Sauna		Euhydrated prior to LFTEs		Hydrated prior to LFTEs		Drinking During LFTEs		MI	
Parasympathetic	Sympathetic	Parasympathetic	Sympathetic	Parasympathetic	Sympathetic	Parasympathetic	Sympathetic	Parasympathetic	Sympathetic
↓SD	↑LF Norm	↓SD	↑LF Norm	↓SD	↑LF Norm	↓SD	↑LF Norm	↓SD	↑LF Norm
↓pNN50	↑LF:HF Ratio	↓pNN50	↑LF:HF Ratio	↓pNN50	↑LF:HF Ratio	↓pNN50	↑LF:HF Ratio	↓pNN50	↑LF:HF Ratio
↓RMSSD		↓RMSSD		↓RMSSD		↓RMSSD		↓RMSSD	
↓HF Norm		↓HF Norm		↓HF Norm		↓HF Norm		↓HF Norm	

Table 7.4 – Summary of post exposure parasympathetic and sympathetic indicators of HR control, when BAIs were euhydrated prior to exposure but drank during the LFTEs, compared to when the BAIs were hydrated prior to exposure and when they were euhydrated prior to exposure. LFTEs when the BAIs were euhydrated prior to exposure but drank during the LFTEs (n = 6).

Drinking During LFTEs	
Parasympathetic	Sympathetic
↑SD	↓ LF Norm
↑pNN50	↓ LF:HF Ratio
↑RMSSD	
↑HF Norm	

Chapter 8.

Cortisol as a Biochemical Marker of Stress.

8.1 Introduction.

In previous chapters a number of physiological stress indicators were found to be increased from exposure to the LFTEs. The aim of the study was to determine whether a biochemical field based test would be suitable when monitoring BAIs. This chapter investigates whether BAIs experienced stress when exposed to LFTEs. This was evidenced through significant increases in salivary cortisol concentrations between the measurements obtained pre and post exposure to LFTEs.

8.2 Method

Refer to the Methods (Section 3.4.12).

8.3 Results.

The results between the salivary cortisol concentrations obtained prior to exposure to the mock training exercises and the LFTEs during Protocol 1 showed no significant differences (Figure 8.1). There were also no significant increases recorded in salivary cortisol concentration between the pre and immediately post exposure measurements of the mock training exercises. Significant increases ($p<0.05$) in salivary cortisol were observed in the immediately post exposure cortisol concentrations (42.4 (± 32.9) $nmol \cdot litre^{-1}$) when compared to the pre exposure concentrations (21.6 (± 13.4) $nmol \cdot litre^{-1}$) of the LFTEs. There were no significant differences in salivary cortisol concentration when the pre exposure measurements were compared to those obtained 1, 2, 3, 4 or 5 hours following the cessation of the LFTEs.

The relationship between osmolality and cortisol was also investigated (Figure 8.2). Both dehydration and catecholamine release in BAIs as a result of exposure to the LFTEs would be expected to decrease the salivary flow rate and increase the osmolality. The osmolality increased ($p<0.05$) from 57.4 (± 10.0) $mOsmol \cdot kg^{-1}$ prior to exposure to 88.5 (± 26.2) $mOsmol \cdot kg^{-1}$ post exposure. In an attempt to standardise the cortisol concentration, it was divided by the osmolality.

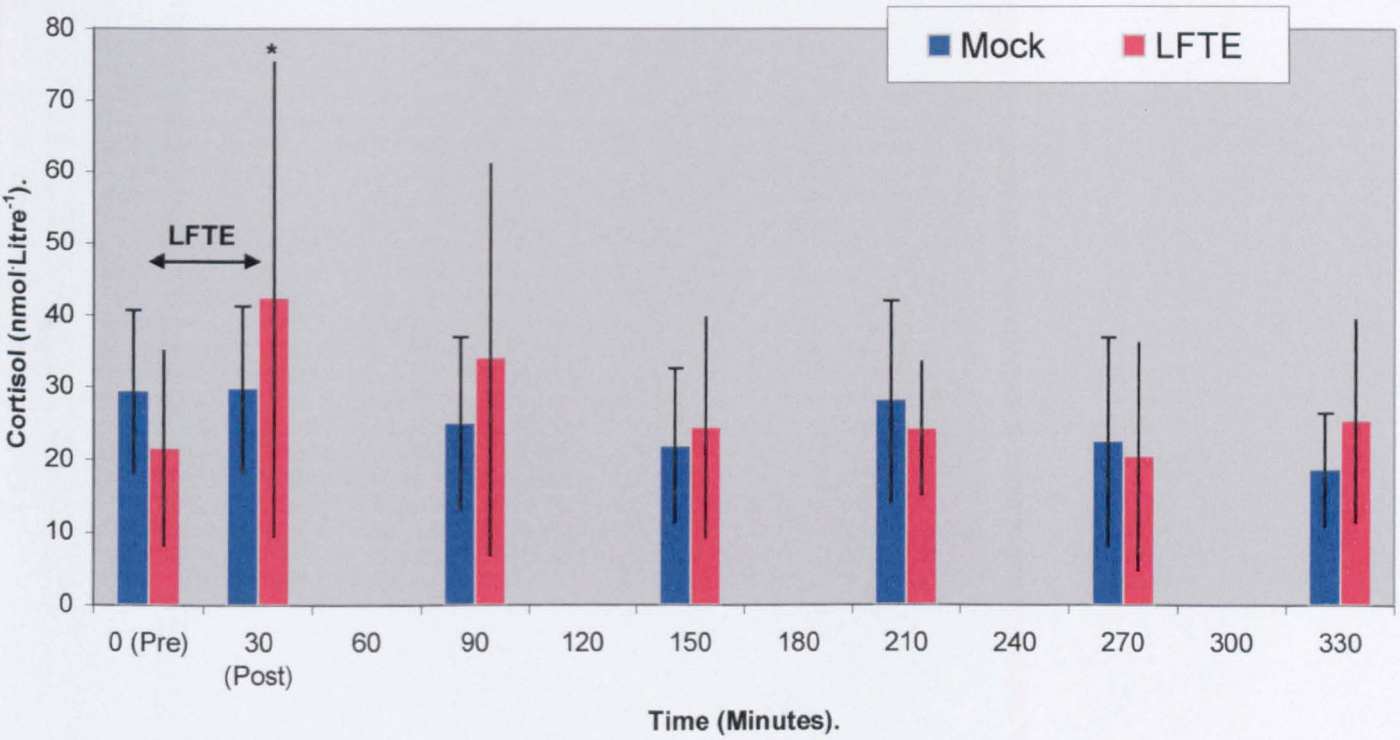


Figure 8.1 – Mean salivary cortisol produced by BAIs when exposed to mock training exercises (Mock), when the fire and students were absent from the exercises, and live fire training exercises (LFTE), when the fire and students were present. Saliva samples were obtained immediately pre exposure, immediately post exposure and every hour post exposure to the exercises for five hours. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the LFTE pre and immediately post exposure salivary cortisol measurements.

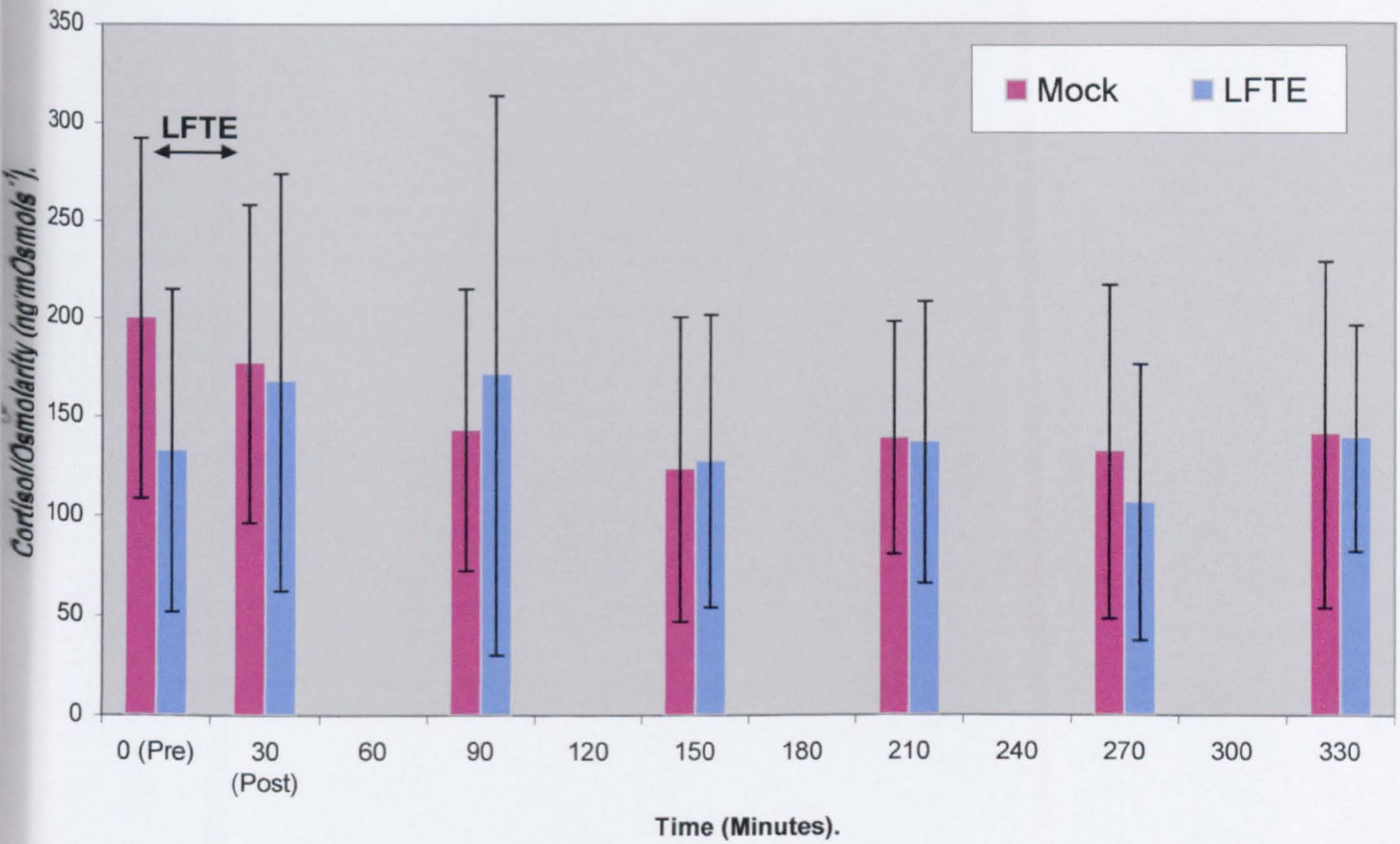


Figure 8.2 – Mean salivary cortisol (normalised by osmolality) produced by BAIs when exposed to mock training exercises (Mock), when the fire and students were absent from the exercises, and live fire training exercises (LFTE), when the fire and students were present. Saliva samples were obtained immediately pre exposure, immediately post exposure and every hour post exposure to the exercises for five hours. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test.

There were no significant differences in the pre and post cortisol (normalised by osmolality) concentrations between mock and LFTE values. Nor were there significant differences between any of the values at 1, 2, 3, 4 or 5 hours post exposure when compared to the pre exposure cortisol (normalised by osmolality) for either the mock or LFTEs.

Mean osmolality showed no significant increases between pre and immediately post exposure to the mock training exercises. However, significant increases ($p<0.05$) were evident between the measurements obtained pre exposure and immediately post exposure to the LFTEs.

In an attempt to determine the stress response directly attributed to hydration state, BAIs were either euhydrated prior to exposure, hydrated prior to exposure (Protocol 2) or were euhydrated prior to exposure but drank during the LFTEs (Protocol 3). It was envisaged that dehydration (exacerbated through starting the LFTEs in a euhydrated state as opposed to a hydrated state) would cause an increase in stress. Therefore, through hydrating the BAIs prior to exposure to the LFTEs, or, drinking during the LFTEs, would induce a lower stress response when compared to the euhydrated LFTEs.

During Protocol 2 the results show that there were no significant differences in cortisol concentration between hydrated and euhydrated states when BAIs were exposed to LFTEs for any of the time points (Figure 8.3).

Salivary osmolality (Figure 8.4) results indicated no significant differences between the LFTEs when BAIs were either hydrated or euhydrated prior to exposure for any of the sampling periods. There were also no significant differences when the pre exposure values were compared to any of the other time points post exposure when BAIs were either hydrated or euhydrated prior to exposure.

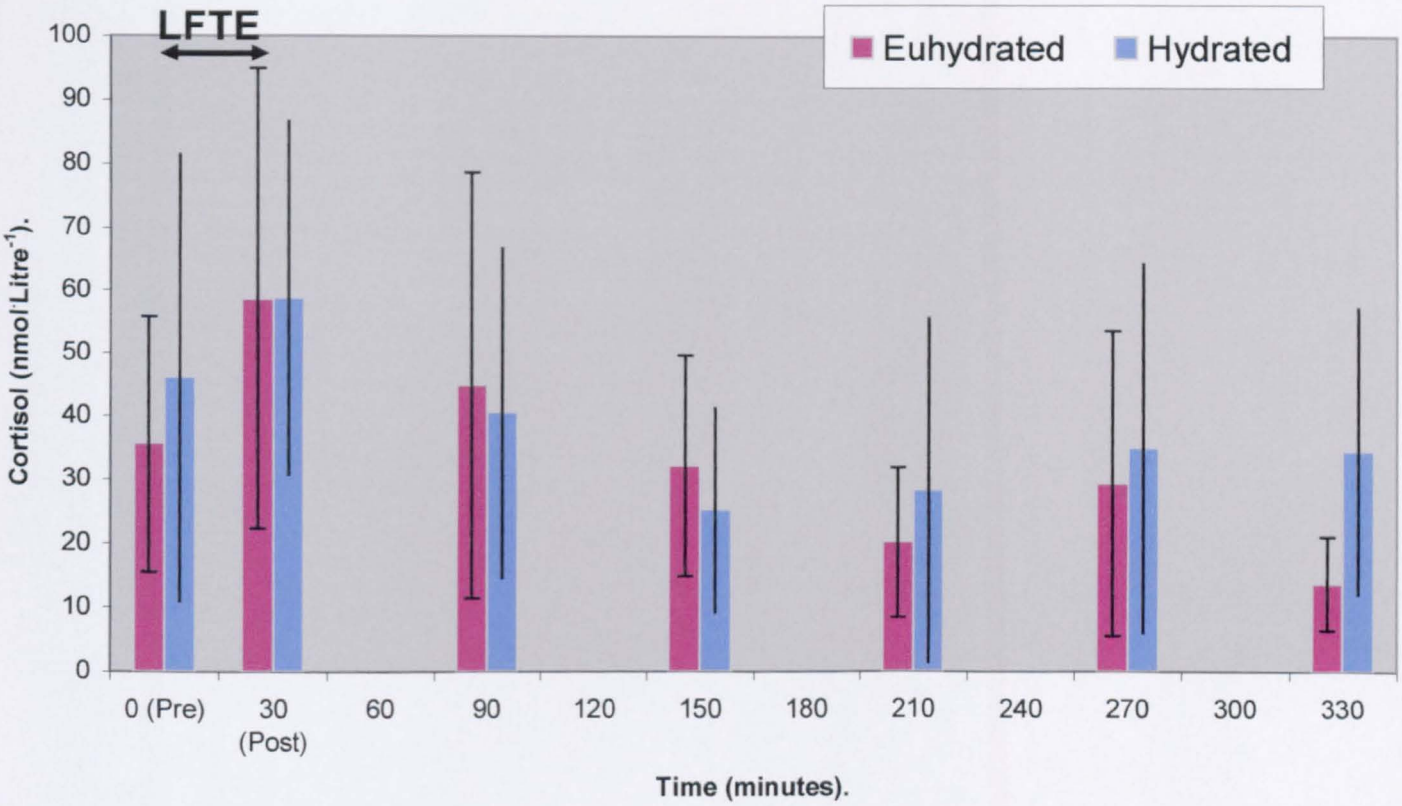


Figure 8.3 – Mean salivary cortisol produced by BAIs when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated) or consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) (Protocol 2). Saliva samples were obtained immediately pre exposure, immediately post exposure and every hour post exposure to the exercises for five hours. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test.

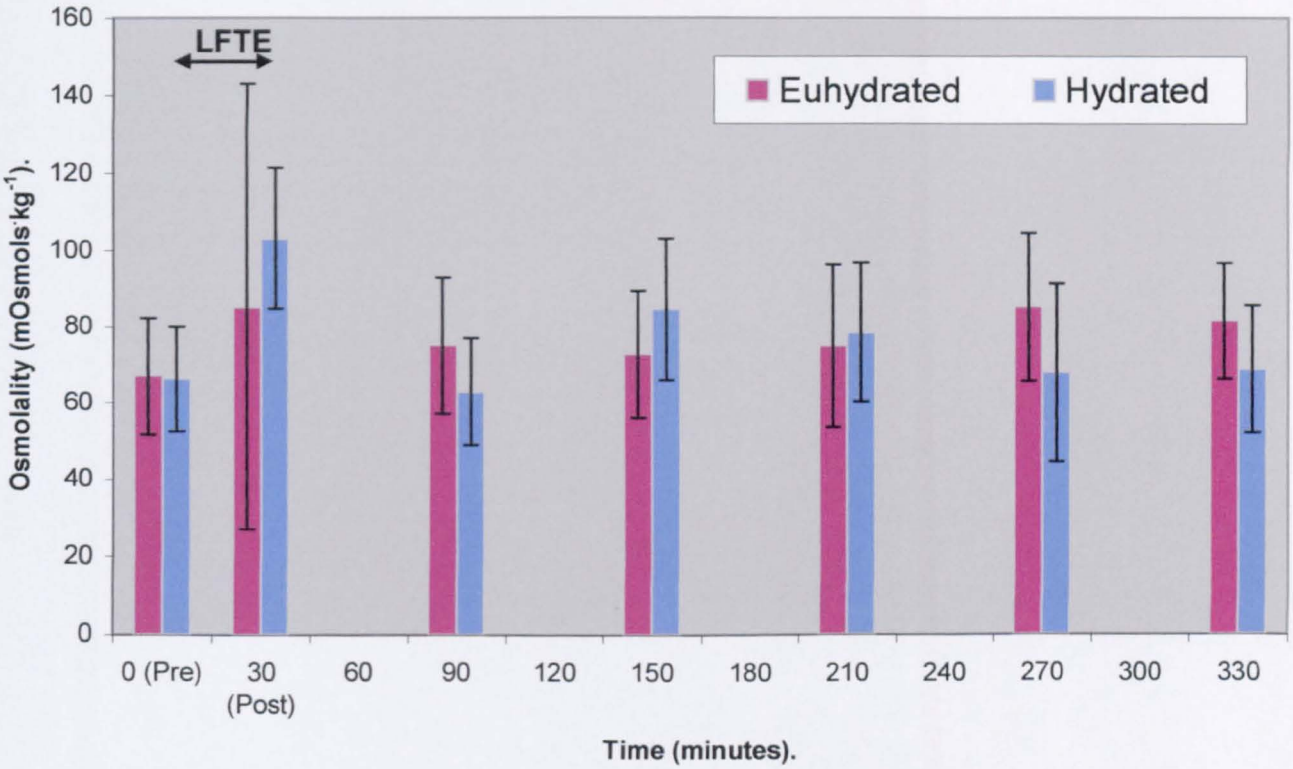


Figure 8.4 – Mean salivary osmolality produced by BAIs when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated) or consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) (Protocol 2). Saliva samples were obtained immediately pre exposure, immediately post exposure and every hour post exposure to the exercises for five hours. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test.

The results for cortisol (normalised by osmolality) showed there were no significant differences between the LFTEs when the BAIs were either hydrated or euhydrated prior to exposure for any of the times of cortisol concentrations. There were also no significant differences when the pre exposure values were compared to any of the time points post exposure when the BAIs were either hydrated or euhydrated.

During Protocol 3 there were no significant differences in cortisol concentration between the time points for any of the three timings of fluid ingestion (when the BAIs were either hydrated prior to exposure, euhydrated prior to exposure, or euhydrated prior to exposure but drank during LFTEs). Also there were no significant increases in cortisol concentration between the pre exposure values compared to any of the post exposure values for the three drinking trials (Figure 8.5). These findings are consistent with Protocol 2.

When the salivary cortisol concentrations (normalised by osmolality) were considered (Figure 8.6), there were no significant differences between any of the fluid ingestion times between the pre and immediately post values. Also there were no significant differences between the three fluid ingestion trials at any of the time points post exposure to the LFTEs.

The mean osmolality results (Figure 8.7), showed there were significant increases ($p<0.05$) between pre and post exposure to the LFTEs when BAIs were hydrated prior to undertaking the LFTEs. However, there were no significant differences observed for the LFTEs when BAIs were either euhydrated prior to exposure or euhydrated prior to exposure but drank during the LFTEs. In addition, there were significantly lower post exposure osmolality results when BAIs were euhydrated but exited the FFU to drink during the LFTEs when compared to the other trials. No other differences were observed between any of three fluid ingestion trials at any of the time points.

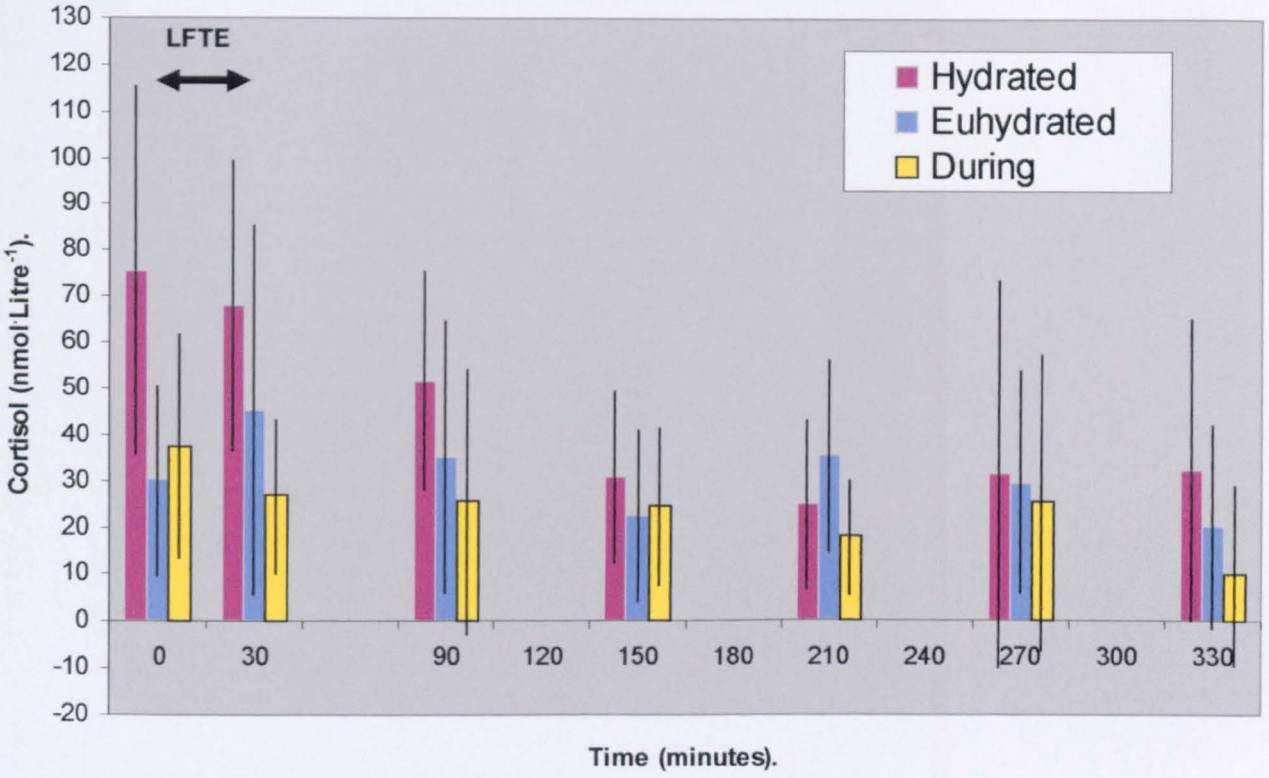


Figure 8.5 – Mean salivary cortisol produced by BAIs when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocol 3). Saliva samples were obtained immediately pre exposure, immediately post exposure and every hour post exposure to the exercises for five hours. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test.

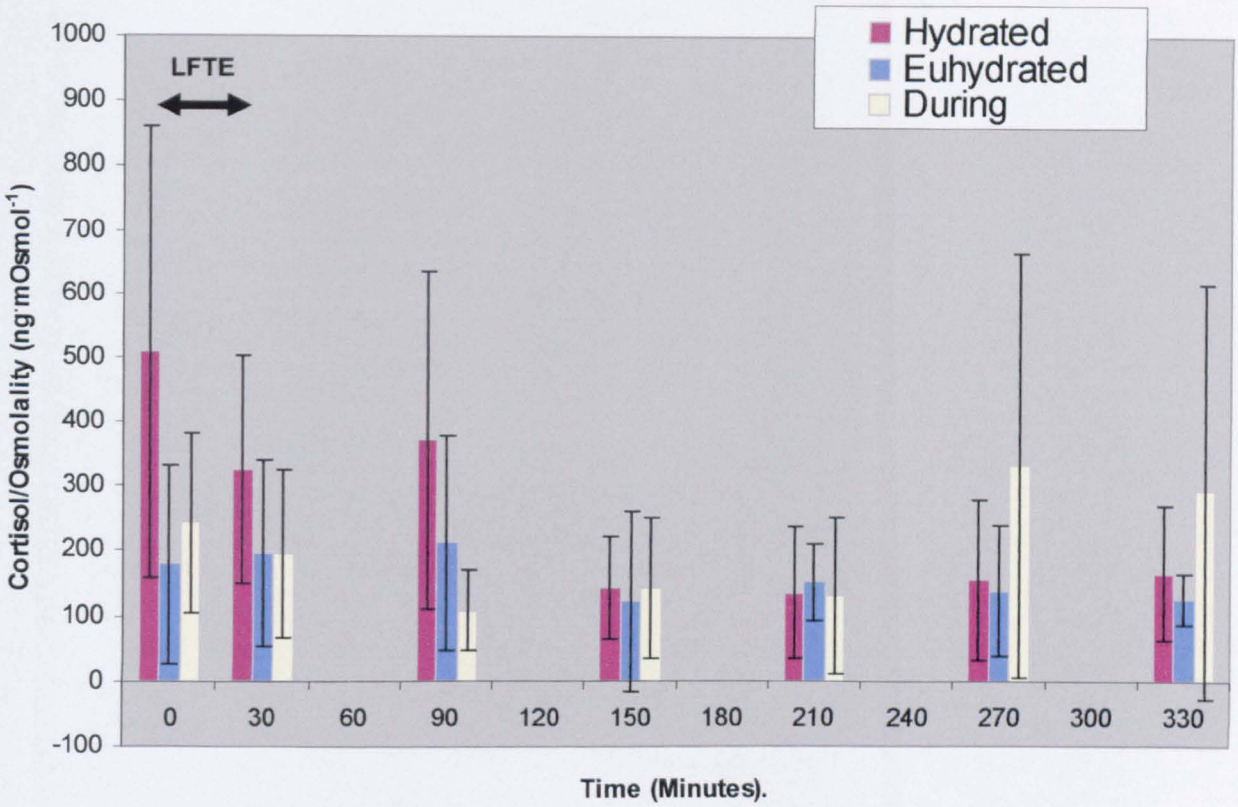


Figure 8.6 – Mean salivary cortisol (normalised to osmolality) produced by BAIs when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocol 3). Saliva samples were obtained immediately pre exposure, immediately post exposure and every hour post exposure to the exercises for five hours. Data is presented as the mean ± SD (n = 6). Significance was tested using a paired t-test.

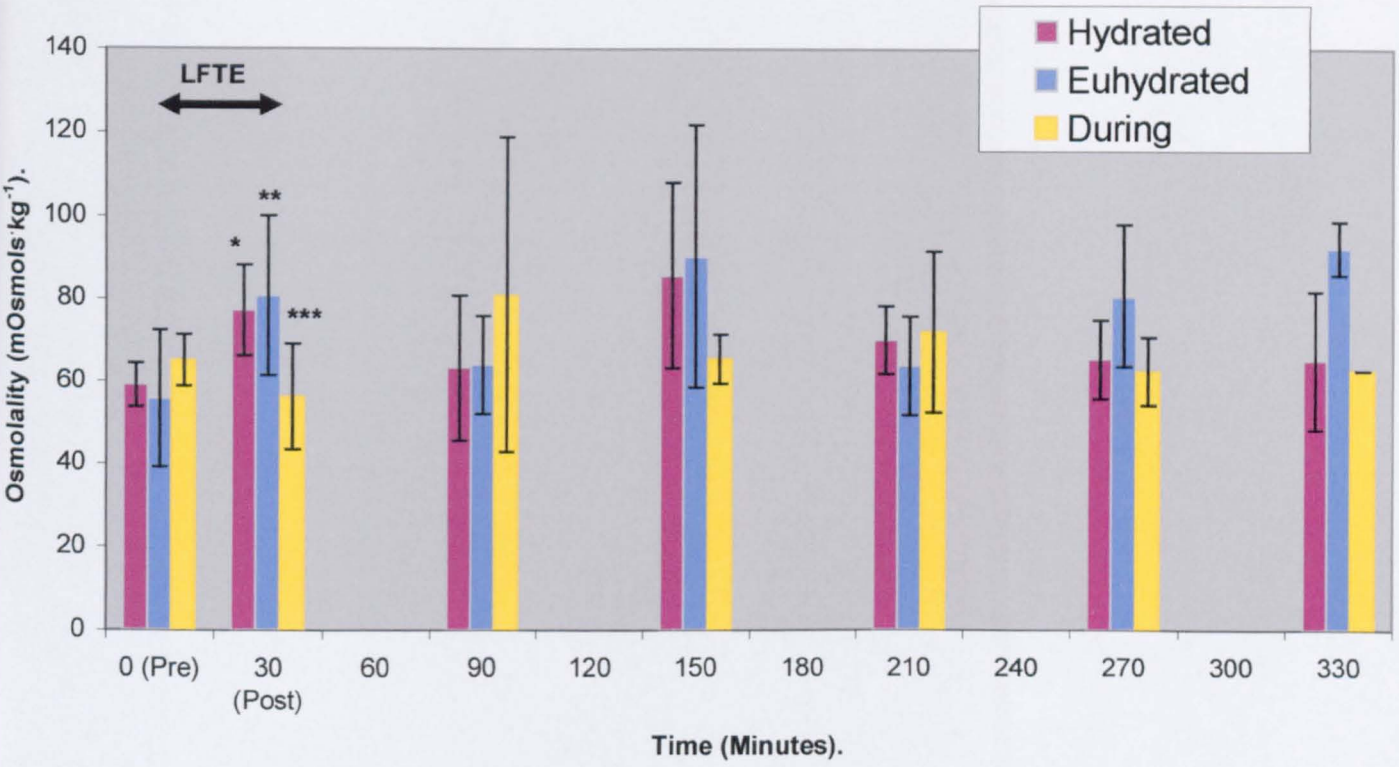


Figure 8.7 – Mean salivary osmolality produced by BAIs when exposed to live fire training exercises (LFTE). BAIs completed the LFTEs without consuming fluid prior to exposure (Euhydrated), consuming 600 ml of fluid 1 hour prior to exposure (Hydrated) or drinking 200 ml every 10 minutes during exposure (During) (Protocol 3). Saliva samples were obtained immediately pre exposure, immediately post exposure and every hour post exposure to the exercises for five hours. Data is presented as the mean and ± SD (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the hydrated pre and immediately post exposure salivary osmolality measurements. ** - significant differences ($p<0.05$) between the euhydrated and hydrated post exposure salivary osmolality measurements. * - significant differences ($p<0.05$) between the hydrated and during post exposure salivary osmolality measurements.**

8.4 Discussion.

During Protocols 1, 2 and 3, the pre exposure salivary cortisol concentrations were all consistent with pre test values reported by others. Cook *et al* (1992) observed resting salivary cortisol concentrations between 8 nmol·litre^{-1} and 45 nmol·litre^{-1} and Kirschbaum and Hellhammer (1992) between 15 nmol·litre^{-1} and 25 nmol·litre^{-1}. Kahn *et al* (1992) investigated the stress responses in students during examinations and observed resting salivary cortisol concentrations between 20 nmol·Litre^{-1} and 50 nmol·Litre^{-1}. However, some subjects produced salivary cortisol concentrations between 70 and 80 nmol·Litre^{-1} which were consistent with the present study's findings (Table 8.1).

Table 8.1 – Mean salivary cortisol concentrations for BAIs prior to exposure to the LFTEs during Protocols 1, 2 and 3. Values are mean (± SD) (n = 6).

	Protocol 1		Protocol 2		Protocol 3		
	Mocks	**LFTEs**	**Euhydrated LFTEs**	**Hydrated LFTEs**	**Euhydrated LFTEs**	**Hydrated LFTEs**	**During LFTEs**
Mean Salivary Cortisol (nmol·Litre^{-1})	29.5 (± 11.3)	21.6 (± 13.4)	35.7 (± 20.1)	46.2 (± 35.3)	30.1 (± 20.5)	75.4 (± 39.9)	37.6 (± 24.0)

During Protocol 1 there were significant increases in the cortisol concentrations post exposure to the LFTEs when compared to the pre exposure concentrations. However, this difference was not seen in Protocol 2 or 3. During mild heat exposure, when the body's core temperature was raised by 0.6-1.0^{0}C, cortisol concentrations did not increase (Brenner *et al*, 1998). Whereas, Collins *et al* (1969) have previously reported a rise of 1.2 ^{0}C in core temperature which was accompanied by a rise in salivary cortisol concentration. It is therefore of interest that during the mock fires, the mean increase in aural temperature was 0.5 (± 0.3) ^{0}C, where the mean increase during the live fires was 2.2 (± 0.5) ^{0}C. Therefore the results of the present study and Collins *et al* (1969) suggest the observed differences in cortisol concentration between the mock and LFTEs may be due to the increase in aural temperature.

Although the significant increase in cortisol was not evident 1 hour after exposure to the LFTEs, the results obtained suggest that the increase in temperature between the mock

and LFTEs significantly amplified the stress response of the BAIs in the short term. The mean cortisol concentrations suggest that the exposure to the LFTEs may have been too low an exercise intensity to elicit a marked cortisol response over a longer period post exposure to the LFTEs. These findings are consistent with the observations of Pollard (1995) and Kirschbaum and Hellhammer (1992). They reported that salivary cortisol is not affected until exercise intensity reaches exercise intensities greater than 70% $VO_{2\ max}$ (mean VO_2 during the LFTEs was 17.4 (± 2.8) ml $O_2 kg^{-1} min^{-1}$ which equated to 47.8 (± 10.7) % of $VO_{2\ max}$).

Significant differences were expected between the mock and LFTEs due to the increased aural temperature produced during the LFTEs. However, the high individual variation (Table 8.2 shows the high standard deviation) and the addition of a small sample size (n = 6) may have contributed to no significant differences between the mock training exercises and LFTEs.

Table 8.2 – Mean salivary cortisol concentrations obtained immediately prior and post exposure to mock training exercises and LFTEs. Values are mean ± SD (n = 6). Significance was tested using a paired t-test with post hoc Bonferroni. * - significant differences ($p<0.05$) between the mean LFTEs pre and post salivary cortisol concentrations.

	Pre Mock	Post Mock	Pre LFTE	Post LFTE
Salivary Cortisol Concentration (nmolLitre^{-1})	10.4 (± 3.9)	11.1 (± 4.4)	7.9 (± 4.8)	15.4 (± 11.9)*

Kenefick *et al* (1998) reported no significant increases between pre and post trial plasma cortisol concentrations when heat acclimated subjects were exposed to a temperate environment (23^{0}C) and exercised at low intensities (36% $VO_{2\ max}$). They also reported that there were no significant increases in cortisol concentrations when different heat intensities were compared (23^{0}C and 38^{0}C). However, intense exercise (in excess of 70% $VO_{2\ max}$) in the heat causes marked increases in cortisol concentrations (Armstrong *et al*, 1989). This suggests that exercise intensity as opposed to heat exposure is the

major stimulus to increased cortisol production. However, considering the only difference between the mock and LFTEs in the present study was the presence of heat and the addition of students, the cortisol concentrations show that as a result of exposure to the LFTEs, there were significant increases between pre and immediately post exposure measurements.

There were no significant increases in cortisol between pre and immediately post exposure to the mock training exercises. This suggests that the addition of very intense heat (107 (± 32.9) ^{0}C in the fire room) and the presence of students did produce an increase in salivary cortisol concentrations. Previous research has not observed the effect of such intense temperatures on cortisol concnetrations. As such, the results presented here, at least immediately post exposure to LFTEs, are in contrast to those of Kenefick *et al* (1998) who showed temperature was not a factor influencing cortisol levels. Following the removal of the stress situation (fire and students), cortisol concentrations returned to pre exposure values by 2 hours post exposure to the LFTEs.

The osmolality results observed when BAIs were exposed to LFTEs, showed significant increases between the pre and immediately post exposure measurements. This demonstrates that the addition of heat and students had an affect on the BAIs saliva flow consistency, and thereby suggests that the BAIs were dehydrated to a greater extent after LFTEs than the mock training exercises. These findings were consistent with the increases in the urine colour and urine osmolality that showed the LFTEs urine colour increased by 58.0 (± 37.6) % and osmolality by 230.0 (± 309.6) % between the pre and post exposure measurements. The mock training exercise urine colour only increased by 8.3 (± 20.4) % and osmolality by 48.6 (± 59.2) % between the pre and post exposure measurements.

During Protocol 2 the salivary cortisol concentrations showed no significant differences between BAIs when either hydrated or euhydrated prior to exposure. However, Kenefick *et al* (1998) reported that dehydrated subjects, when exposed to mild exercise (50% $VO_{2\ max}$) in temperate environments (38^{0}C), have increased cortisol concentrations when compared to subjects who were hydrated. The present study's findings were not

consistent with the study of Kenefick *et al* (1998). In addition, other authors have reported no significant increases in hormone concentrations (ACTH) when subjects were heated passively in a sauna bath for 15 minutes at 100^0C (Leppaluto, 1975).

In the previous chapters the physiological stress parameters (HR, HRV, blood pressure and skin, aural and micro-climate temperatures) were increased by exposure to LFTEs. Drinking during the LFTEs reduced this affect. This study attempted to use salivary cortisol as a biochemical measure of stress. The data from the LFTEs seem to confirm a short term stress response with an increase in cortisol but the effect was small. When the BAIs were hydrated prior to exposure, there was no effect on salivary cortisol concentrations.

However, it is evident in Figure 8.5, when BAIs drank during the LFTEs, mean cortisol concentrations are lowered in comparison to the pre exposure values. In comparison, the immediately post salivary cortisol concentrations increased when the BAIs were hydrated prior to exposure and when they were euhydrated prior to the LFTEs. This demonstrates that it may be possible to reduce salivary cortisol concentration immediately post exposure to LFTEs by BAIs exiting the FFU to drink during the LFTEs. This in turn allowed the heat stored in the protective clothing to escape creating less stress on the BAIs. This was demonstrated by lower skin and micro-climate responses during these LFTEs (refer to Figure 6.7 in Chapter 6).

The results in this chapter confirm two things:

1. the LFTE stress was not acute;
2. BAIs were not suffering from long term stress.

There were no significant increases in osmolality between pre and immediately post exposure measurements when BAIs drank during the LFTEs. These results suggest that drinking during LFTEs is more beneficial than drinking prior to exposure.

Also, there were significantly lower post exposure osmolality results when BAIs left the FFU to drink during the LFTEs when compared to BAIs either in a hydrated or

euhydrated state prior to exposure. This suggests BAIs were not dehydrated when they drank during the LFTEs compared to when they were either hydrated or euhydrated prior to exposure.

8.5 Summary.

During LFTEs there were significantly increased salivary cortisol concentrations between the pre and immediately post exposure measurements. However, during the mock training exercises there were no changes in the salivary cortisol concentrations between the pre and immediately post exposure measurements. This suggests the addition of heat and students encountered during LFTEs increases the stress placed on the BAIs over that of carrying out the mock training exercises in thermoneutral conditions without the presence of students.

In comparing cortisol concentrations when BAIs were euhydrated prior to exposure, to when they were hydrated prior to exposure to LFTEs, it can be concluded that there is no effect of hydration status on the salivary cortisol concentrations to LFTEs. This demonstrates that there were no significant differences or clear trends to suggest hydration did have an effect during either Protocol 2 or 3.

When different timings of drinking were employed (Protocol 3), there appears to be no statistically significant affect on lowering the cortisol concentrations as a result of exposing the BAIs to LFTEs. However, there were consistent patterns to suggest lower mean cortisol concentrations between pre and immediately post exposure to the LFTEs when BAIs either drank 600 ml of water 1 hour prior to exposure, or drank during exposure.

The high intra-subject variability observed pre and post exposure to the LFTEs, resulted in few significant differences in cortisol concentrations. However, when the mean percentage change between the pre and immediately post cortisol concentrations were observed, there were consistent increases between the LFTEs throughout all protocols. This suggests that the high intra-subject variability may have concealed any significant effects of exposure to the LFTEs which was also reported by Blannin *et al* (1998). Table

8.3 shows the percentage change between the pre and immediately post exposure salivary cortisol concentrations for Protocols 1, 2 and 3.

Table 8.3 – Percentage change in salivary cortisol (observed from the change between the pre and post exposure cortisol concentration expressed as a percentage of the pre result). Values are mean and standard error of the mean (± SEM) (n = 6). Significance was tested using a paired t-test. * - significant differences ($p<0.05$) between the pre and post salivary cortisol measurements when BAIs were euhydrated prior to exposure.

	Protocol 1	Protocol 2		Protocol 3		
	Euhydrated	Euhydrated	Hydrated	Euhydrated	Hydrated	During
Mean % change	110.8 (± 48.6) *	75.2 (± 33.6)	62.0 (± 34.5)	64.3 (± 45.2)	-4.0 (± 18.4)	-15.8 (± 21.1)

In addition, when the pre and immediately post exposure salivary cortisol data obtained during Protocols 1, 2 and 3 were collated (n = 18), there were significant increases in the post exposure salivary cortisol concentrations. This significant increase was not evident when each Protocol was observed individually. This suggests that the small value of n (n = 6) may have affected the significance between the pre and immediately post exposure readings. This shows that when BAIs were exposed to LFTEs this was, in the short term, a stressful situation.

According to Weicker and Werle (1991) the varied cortisol concentrations are due to the training status of the individual subjects (fitter subjects demonstrate lower cortisol levels and the less fit demonstrating higher cortisol levels). This suggests that future research, in order to reduce the inter-subject variability, should ensure the subjects are matched for training status.

Overall Summary of Results and Discussion.

The intention of this chapter is to summarise the findings of the whole study. This is achieved through answering the research questions presented in Chapter 2.

1. Is there additional stress placed on individuals when wearing protective clothing? If so, is it possible to:
    - Quantify what contributes to the HR responses incurred whilst wearing PC+SCBA?
    - Identify what causes the additional stress?

During LFTEs BAIs are exposed to very hot temperatures (average temperatures of 107.0 (± 32.9) ^{0}C in the fire chamber at mid level (1.2 meters from floor level) and maximum temperatures of 209.6 (± 34.1) ^{0}C). BAIs have a number of key factors affecting them during exposure to LFTEs, which include;

- the additional weight of the PC+SCBA, which incurs a metabolic cost (Baker *et al*, 2000);
- the impermeable nature of the PC impedes the process of evaporation. Therefore, heat accumulates inside the PC;
- the workload from carrying out the LFTEs and;

The study was, therefore, designed to attempt to assess the contribution of these factors during exposure to LFTEs. The first part of the study investigated the effect of wearing PC+SCBA, aiming to quantify the combined effects of wearing PC and SCBA together during exercise in thermoneutral conditions. This allowed observation of the components of the HR responses produced during LFTEs in a controlled environment. To investigate the additional effect of the fire on the BAIs during LFTEs, in addition to wearing PC+SCBA and the workload of carrying out the exercises, mock fire training exercises were compared to LFTEs.

The results obtained from the Step Test whilst dressed in PC+SCBA (Chapter 4) showed that stepping at 15 steps·minute^{-1} (19.7 (± 2.0) ml O_2·kg^{-1}·min^{-1}) was equivalent to the workload carried out during LFTEs (17.4 (± 2.8)

ml $O_2 \cdot kg^{-1} \cdot min^{-1}$). When subjects stepped at 15 steps·minute^{-1} dressed in PC+SCBA there was a 17.9 (± 7.6) % increase in HR compared to wearing gym kit at the equivalent step rate.

- This was primarily due to an increase in workload imposed on the BAIs by the additional weight from wearing the PC+SCBA. White *et al* (1989) also showed additional weight from wearing PC+SCBA resulted in an increased HR during light to moderate exercise (30% of $VO_{2\ max}$). Also, Baker *et al* (2000) reported there was an additional metabolic cost through wearing PC+SCBA.
- Exercising in gym kit with a weighted rucksack (equivalent to the weight of the PC+SCBA), compared to stepping in gym kit at 15 steps·minute^{-1} and 35 steps·minute^{-1}, produced significant increases in HR (15.5 (± 4.3) % and 14.3 (± 3.7) % respectively) and oxygen cost (19.4 (± 17.1) % and 9.2 (± 11.7) % respectively). This showed the effect of the additional weight from wearing the PC+SCBA.
- There were significant increases in skin temperature (3.0 (± 1.9) % when exercising at 15 steps·minute^{-1} and 9.1 (± 3.8) % when exercising at 35 steps·minute^{-1}) as a result of wearing PC+SCBA, compared to exercising in gym kit whilst wearing a weighted rucksack. This strongly suggests it was the increase in heat storage that resulted in the increase in skin temperature. This was consistent with increases in micro-climate that increased by 7.1 (± 3.3) % when exercising at 35 steps·minute^{-1}.
- The results from the Step Test and Muir *et al* (2001) show there was a significant increase in HR (17.9 (± 7.6) %) from wearing the PC+SCBA, and an increase in HR through a build up of heat stored in the PC.
- Hence, the difference between the gym kit with weighted rucksack and PC+SCBA trials was the encapsulating nature of the PC.
- Therefore, before the BAIs entered the LFTEs there was a degree of stress placed on them, not only from the additional weight of the PC+SCBA, but also through an increase in the heat stored within the PC. These findings are consistent with the findings reported by Muir

et al (2001), as the ability of the body to loose heat from the process of evaporation is compromised (Havenith, 1999).

The control results were consistent throughout Protocols 1, 2 and 3 for the physiological parameters (HR, oxygen cost, skin, aural and micro-climate temperatures, glucose, lactate, blood pressure, body mass losses and haematocrit). This shows internal validity across the three protocols.

The physiological responses of BAIs were monitored when carrying out mock fire training exercises. This involved the workload necessary to carry out the exercises and the effect of wearing the PC+SCBA. These physiological responses were compared to those produced when carrying out LFTEs, thus observing the additional effects of heat from the fire and the presence of students (Protocol 1).

- As a result of exposure to LFTEs, there were significant increases in HR, oxygen cost, haematocrit, skin, aural and micro-climate temperatures, lactate, systolic blood pressure and RPE compared to the mock training exercises. There were also significant decreases in body mass (lost through sweating) and also plasma volume when compared to the mock training exercises.
- This suggests that there were significant increases in the amount of short term stress placed on the BAIs through the addition of the heat and the presence of students. These findings are consistent with Lusa *et al* (1993) who also observed higher HR responses when exercising dressed in PC+SCBA in the heat, compared to exercising dressed in PC+SCBA in thermoneutral conditions.
- There were significant increases in the oxygen cost, through exposure to the LFTEs when compared to the mock training exercises. This occurred with the inclusion of the fire and students. The increase in oxygen cost was not caused by an increase in the workload, as both the mock and LFTEs followed a set work schedule.
- The mean HR responses during the LFTEs increased by 39.8 (± 20.1) % when compared to the mock training exercises. This showed an additional significant increase in HR with the inclusion of fire and

students. This was in addition to the workload necessary to carry out the LFTEs, the weight of the PC+SCBA and also the heat storage from wearing the PC+SCBA.

- It appears that the amount of heat stored within the PC is exacerbated through an increase in the environmental temperature. The skin, aural and micro-climate temperatures significantly increased during the LFTEs, compared to the mock training exercises. The workload was the same between the mock and LFTEs, showing that the increase in the skin, aural and micro-climate temperatures was indeed due to the increase in environmental temperature and not due to any difference in workload.
- The differences in HR results between the mock and LFTEs, suggest that by reducing the heat storage within the PC+SCBA, may in turn reduce the increase in HR and lessen the short term stress placed on the BAIs during LFTEs.
- The results from the Step Test and Protocol 1 could significantly impact on working practices through the manner in which BAIs lower the micro-climate, skin and aural temperature, in turn lowering the HR responses to LFTEs.

2. Can incorporating hydration strategies prior to, or during LFTEs:

- Reduce the physiological, psychological and biochemical responses of BAIs during LFTEs?
- Result in the BAIs maintaining fluid balance throughout the days of LFTEs?

The BAIs were either

1) euhydrated prior to LFTEs (euhydrated was defined as normal fluid balance where the BAIs were neither hydrated (where urine colour was 1) nor dehydrated (where urine colour was greater than 3) prior to exposure)

2) hydrated prior to LFTEs (the BAIs drank 600 ml of water 1 hour prior to exposure) (Protocol 2), or

3) euhydrated prior to exposure but drank during the LFTEs (Protocol 3).

It was hypothesised that hydrating BAIs prior to LFTEs would reduce the heat stress responses when undertaking fire training exercises. This would be consistent with the findings of Riebe *et al* (1997) who observed significant reductions in HR and RPE readings when their subjects were hydrated prior to exercise (treadmill walking at 50% of $VO_{2\,max}$).

- The results of the present study showed there to be no significant effects of hydrating BAIs prior to exposure, compared to when BAIs were euhydrated prior to LFTEs.
- There were similar findings in temperature responses between Protocols 1, 2 and 3. For example, there were significant increases in skin, aural and micro-climate temperatures when BAIs wore PC+SCBA compared to when they wore gym kit prior to exposure.
- This suggests that hydrating BAIs prior to the LFTEs did not stem the increase in skin, aural and micro-climate temperature when PC+SCBA was worn. In addition, these temperatures were significantly increased further during the LFTEs. This also suggests hydrating BAIs prior to exposure did not lower the heat stress response to LFTEs.

It was hypothesised that, during Protocol 3, by removing the BAIs from the FFU to drink 200 ml of water every 10 minutes would lower the stress responses of the BAIs.

- When BAIs exited the FFU to drink 200 ml of water there was a significant reduction in the micro-climate temperatures (-4.2 (± 4.3) %) and the HR responses (-23.2 (± 13.8) %) compared to the equivalent times during the LFTEs when the BAIs were euhydrated prior to exposure.
- This suggests that when the BAIs drank during the LFTEs, this placed lower levels of stress on the BAIs when compared to the LFTEs when the BAIs were euhydrated prior to exposure.

3. When using heart rate variability (HRV) to detect changes in the sympathetic and parasympathetic balance of HR control:
    - Is the HRV method sensitive enough to detect these changes: when breathing is controlled; during hot environments; when different hydration strategies are implemented during LFTEs; and identify whether BAIs are stressed as a result of exposure to LFTEs?

During HRV measurements, it was considered unnecessary to control the breathing frequency of BAIs due to no significant differences in the HRV variables (SD, pNN50, HF Norm and LF Norm) between the controlled (CB) and spontaneous breathing (SB) trials.

- These results are consistent with the findings of Patwardhan *et al* (1995) and Stark *et al* (2000) who observed no significant differences between the HF Norm and HRV values between spontaneous and controlled breathing trials.
- This suggests there was no significant consequence of the cortical effect of controlling breathing on HRV under these conditions. Thus, when subjects were placed in a sauna at 74.3 (± 5.9) ^{0}C or pre and post exposure to LFTEs, their breathing was not controlled.

According to Brenner *et al* (1998) there is very little research into the effects of heat exposure on HRV. Therefore an initial study investigated whether the method of measuring HRV (using a Polar HR monitor and software, Finland, Oy) was sensitive to the changes produced when subjects were exposed to the controlled conditions of a Finnish sauna.

- As a result of exposure to the sauna there were significant increases in HR through an increase in the sympathetic influence on HR control, indicated by significant increases in the LF Norm and LF:HF ratio. These findings were consistent with Brenner *et al* (1997). There was also a significant decrease in the parasympathetic influence of HR control (indicated by a reduction in the HF Norm, pNN50 and RMSSD).

- There were significant reductions in the overall HRV which was also observed by Parsons *et al* (1992) when subjects were exposed to an environment of 27^0C compared to 17^0C. A reduction in overall HRV is often accompanied by an increase in the sympathetic indicators of HR control (Task Force, 1996), suggesting an increase in stress on the cardiac system.
- The skin, aural and climate temperatures increased to 41.9 (± 1.6) ^{0}C, 38.8 (± 0.5) ^{0}C and 74.3 (± 5.9) ^{0}C respectively during exposure to the sauna, producing:
    - significant increases in the sympathetic indicators of HR control (LF Norm and LF:HF ratio) and also significant decreases in the parasympathetic indicators of HR control (HF Norm, pNN50 and RMSSD).
- This shows that exposure to hot environments produces a sympathetically driven control of HR which suggests a stress response to this type of environment.

To investigate the cardiac stress placed on the BAIs, in addition to observations made on increases or decreases in mean HR when carrying out LFTEs, heart rate variability (HRV) was monitored immediately pre and post exposure to the LFTEs. This allowed greater depth of analysis of the stress placed on the cardiac system.

- During Protocol 1 the skin, aural and micro-climate temperatures increased to 37.5 (± 1.4) ^{0}C, 37.5 (± 0.7) ^{0}C and 36.0 (± 2.5) ^{0}C respectively on exposure to LFTEs which consistently showed;
    - increases in the sympathetic indicators of HR control (LF Norm and LF:HF ratio) and also a significant decrease in the parasympathetic indicators of HR control (HF Norm, pNN50 and RMSSD). This was in agreement with the results produced during exposure to a sauna.
- The sympathetic indicator of HR control (LF Norm) increased by 21.5 (± 32.7) %, whilst the parasympathetic indicator of HR control (pNN50) decreased by -53.6 (± 69.8) %.

- This suggests the stress through exposure to LFTEs was from a reduction in parasympathetic control of HR and an increase in the sympathetic control of HR.
- This suggests there is an increase in cardiac stress when exposed to LFTEs.

The BAIs were either hydrated prior to exposure through drinking 600 ml of water 1 hour prior to exposure, euhydrated prior to exposure (Protocol 2) or euhydrated prior to exposure but drank during the LFTEs (Protocol 3).

- Prior to exposure there were no significant differences between BAIs when they were hydrated or euhydrated prior to exposure, or drank during the LFTE for the indicators of sympathetic HR control (LF Norm and LF:HF ratio). There were also no significant differences for the indicators of parasympathetic control of HR (HF Norm, pNN50 and RMSSD).
- There were significant decreases in the parasympathetic indicators of HR control (pNN50 and RMSSD) post exposure to the LFTEs when the BAIs were hydrated prior to exposure, compared to when they were euhydrated prior to exposure. This suggests an increase in the stress response post exposure to the LFTEs when the BAIs were hydrated prior to exposure. Hence, there was no advantage of hydrating BAIs prior to exposure.
- This was consistent with the findings of McLellan *et al* (1999) who reported that uncompensatable heat stress negates the positive physiological responses of hydration.
- Both the pre exposure and post exposure results were consistent with the other physiological and psychological results from Protocols 2 and 3, suggesting that hydrating the BAIs prior to exposure does not reduce the stress placed on the BAIs during LFTEs.
- Post exposure to the LFTEs when the BAIs drank during the LFTEs, compared to when the BAIs were hydrated prior to exposure, there were significant increases in the parasympathetic indicators of HR control (pNN50 and RMMSD) and total power.

- The increase in pNN50 and RMSSD post exposure suggests a lower stress response from BAIs was produced when they drank during the LFTEs.
- The increase in total power represents an increase in overall HRV, which according to the Task Force (1996) is accompanied by an increase in parasympathetic control of heart rate. This also suggests less stress was placed on BAIs when they drank during the LFTEs.

- When BAIs were euhydrated prior to exposure, they produced significant increases in the sympathetic indicator of HR control (LF Norm) between pre and post exposure to the LFTEs during Protocol 1 (21.5 (± 32.7) %), Protocol 2 (27.5 (± 33.3) %) and Protocol 3 (12.0 (± 8.1) %). There were also significant reductions in the parasympathetic indicator of HR control (RMSSD) during Protocol 1 (53.2 (± 61.1) %), Protocol 2 (33.6 (± 44.7) %) and Protocol 3 (53.7 (± 26.2) %).
- However, there were no significant changes in either the sympathetic or parasympathetic indicators of HR control when the BAIs drank during the LFTEs.
- Therefore, leaving the FFU to consume 200 ml of water every 10 minutes during the LFTEs lowers the stress placed on the cardiac system compared to when the BAIs were euhydrated prior to exposure.

In addition, exposure to heat appears to produce the same HRV responses that post MI patients exhibit.

- It may be hypothesised that repeated exposure to heat stress over long periods of time may lead to increased prevalence of heart conditions.
- However, underlying biological factors are usually associated with the aetiology of MI. However, there is no reason to suspect that these factors are relevant to the BAIs. However, increased HR over time would suggest that further research may be of benefit in this respect.

- Drinking during the LFTEs appears to alter the HRV values from those that are similar to the responses observed post MI (increased sympathetic indicators of HR control and decreased parasympathetic indicators of HR control) to those that exhibit decreases in the sympathetic indicators of HR control and increased parasympathetic indicators of HR control. This in turn may reduce the likelihood of BAIs having an increased prevalence of heart conditions over the long term period.
- Leaving the FFU to drink 200 ml during LFTEs appears to lower skin temperature. In turn, this reduced the amount of heat stress placed on the BAIs during LFTEs.

4. Is salivary cortisol a suitable measurement of short term interrupted stress as found during LFTEs? If this method is viable, can it be adapted as a useful field-based test in this respect?

Salivary cortisol was used instead of plasma due to the field testing nature of the study.

- The biochemical analysis of salivary cortisol showed when BAIs undertook LFTEs there were significant increases (110 (± 119.0) %) in salivary cortisol between the pre and immediately post exposure readings. This suggests BAIs were stressed in the short term when exposed to LFTEs.
- There has been limited research conducted which has investigated similar environmental temperatures the BAIs were exposed to during LFTEs. However, Collins *et al* (1969) exposed subjects to temperate conditions, which observed significant increases in cortisol levels between the pre and post exposure values.
- No significant increases in salivary cortisol were observed between the pre and immediately post exposure measurements when BAIs undertook mock training exercises. This was consistent with the findings reported by Kenefick *et al* (1998) who also observed no

significant differences between pre and post cortisol concentrations when their subjects exercised at a low intensity of exercise (36.0 (± 5.0) % of $VO_{2\ max}$) in thermoneutral conditions.

- During LFTEs (from personal communication with the BAIs) they felt they were not affected by the inclusion of the students due to the safety precautions that exist (the fire can be shut off at the press of a button) and their considerable experience in live fire training. Also, during LFTEs (refer to Chapter 5) the BAIs HR response did not increase to the same degree as the students. This may further suggest the stress placed on the BAIs from the addition of students during LFTEs was negligible.
- Therefore, the inclusion of the fire during LFTEs produces a significant short term stress response from the BAIs, evidenced from the increased cortisol production between the pre and immediately post exposure concentrations.
    - This stress response was not only observed from the biochemical analysis, but was also supported by the significant increases in the mean HR, oxygen cost and skin, aural and micro-climate temperatures.
    - In addition, a stress response was also observed from the HRV data from the significant increases in the sympathetic and decreases in the parasympathetic indicators of HR control.

There were no significant differences in the cortisol concentration between the LFTEs when the BAIs were euhydrated prior to exposure, hydrated prior to exposure (Protocol 2), or drank during the LFTEs (Protocol 3).

- This suggests there to be no positive effects of hydrating BAIs prior to exposure to LFTEs. This was not consistent with the research by Kenefick *et al* (1998). They showed that hydrated subjects produced lower cortisol concentrations, compared to euhydrated subjects after mild exercise in a temperate environment.
- The salivary cortisol concentrations from the present study support the findings of the HRV data and also the parameters such as HR, oxygen

cost and haematocrit. These parameters also show the usefulness of hydration as a heat stress reliever was negated by exposure to extreme heat during LFTEs.

- However, when the BAIs drank 200 ml of water every 10 minutes during the LFTEs, the salivary cortisol concentrations showed patterns to suggest they were lower immediately post exposure to the LFTEs, compared to when the BAIs were hydrated, or euhydrated prior to exposure to the LFTEs.

Therefore, the present study demonstrated that ingestion of fluid did not affect salivary cortisol concentration. However, when BAIs drank during the LFTEs, there were non significant trends suggesting lower salivary cortisol responses immediately post exposure (26.7 (± 16.5) $nmol{\cdot}litre^{-1}$) compared to when BAIs were either hydrated (68.0 (± 31.5) $nmol{\cdot}litre^{-1}$) or euhydrated prior to the LFTEs (45.5 (± 40.0) $nmol{\cdot}litre^{-1}$).

Summary.

- The study has shown the component parts of the short term stress response to LFTEs. The increase in HR during LFTEs is from these contributory factors:-
  - the necessary workload that is carried out during LFTEs by the BAIs (24.4 % increase compared to resting HR);
  - weight from wearing the PC+SCBA (further increase of 6.9 %)
  - heat storage from wearing the PC (additional increase in HR of 8.9 %)
  - heat radiated from the fire (HR increased by 3.5 %, although peak HR responses suggest a further increase of 25.8%).

These results suggest there is an additional short term stress placed on the BAIs from wearing PC+SCBA.

- The study has also shown that hydrating the BAIs prior to exposure does not lower the stress responses produced during the LFTEs. However, the

physiological, psychological and biochemical parameters have consistently shown patterns that suggest leaving the FFU to drink during the LFTEs may lower the stress responses during LFTEs. In addition, the skin, aural and micro-climate temperatures and HR responses when BAIs exited the FFU to drink 200 ml of water, resulted in significantly lower responses compared to the equivalent time during LFTEs when BAIs were euhydrated prior to exposure.

- The study has shown that the Polar HR monitor, in conjunction with the Polar software (Polar, Finland, Oy), is sensitive enough to detect changes in HRV parameters with changes in environmental temperature.
    - The HRV measurements have shown that exposure to LFTEs does place a significant stress on the BAIs, evidenced from the significant increases in the sympathetic and decreases in the parasympathetic indicators of HR control.
    - There was no beneficial effect on the HRV measurements when the BAIs were hydrated prior to exposure. However, when the BAIs drank during the LFTEs there was less stress placed on them. This was evidenced from no significant changes in the sympathetic or parasympathetic indicators of HR control between the pre and post exposure measurements. In comparison, BAIs that were either hydrated or euhydrated prior to exposure produced significant increases in sympathetic and significant decreases in parasympathetic indications of HR control between the pre and post exposure measurements.
    - Also, when the BAIs drank during the LFTEs, post exposure to the LFTEs they produced significantly increased parasympathetic indictors of HR control and total power, compared to when they were either hydrated or euhydrated prior to exposure.
    - In addition, exposure to heat appears to produce the same HRV responses that post MI patients exhibit. It could be hypothesised that repeated exposure to severe heat stress over long periods of time may lead to increased prevalence of heart conditions. Underlying biological factors are usually associated with the aetiology of MI. However, there is no reason to suspect that these factors are relevant

to the BAIs. However, increased HR over time would suggest that further research may be of benefit in this respect.

- However, drinking during the LFTEs appears to produce a reduction in stress and a movement away from the increased sympathetic and decreased parasympathetic indicators of HR control produced by post MI patients

- The biochemical analysis of salivary cortisol has shown that:
  - the fire from the LFTEs does increase the stress placed on the BAIs in the short term. This was observed from the significant increase in cortisol concentration between the immediately pre and post exposure readings. There was no evidence from the salivary cortisol concentrations that exposure to the LFTEs causes long term stress.
  - hydrating the BAIs prior to exposure does not lower this stress response.
  - drinking during the LFTEs appears to lower the cortisol concentration between the pre and immediately post exposure readings, suggesting a lower stress response.

Conclusions And Recommendations.

Applications of the study's findings on the practices of the Fire Service:

- It is recognised that the subject number in the present study was low thus questioning the representation of the whole BAI population. Therefore, additional subject numbers in future studies would be required. However, the power equation presented in the Method chapter shows a consistent trend throughout all studies for power to be in excess of 0.8. On account of the measured effect size being large (> 0.9), the power of the analysis was found to be greater than 0.8. According to Welkowitz *et al* (2000), Howell (2002) and Thomas and Nelson (1996) power should be 0.8 when testing the difference between two matched samples. This suggests the subject number was acceptable.
- The mean HR results obtained during the LFTEs may not suggest that the BAIs were stressed. However, the psychological data showed significant increases in time to complete the Stroop test (between the measurements obtained pre exposure compared to the post exposure readings). This suggests that the BAIs concentration may have been affected through exposure to LFTEs. A loss of concentration by BAIs during LFTEs may lead to mistakes being made, or, may compromise the BAIs ability to recognise when the LFTE should be terminated.
- In addition, the HRV data does suggest that the BAIs are stressed when exposed to LFTEs in the short term. This stress is reduced through leaving the FFU to consumed 200 ml of water every 10 minutes during LFTEs.
- During exposure to LFTEs there are significant increases in the heat stored within the PC+SCBA. Therefore, to alleviate this heat storage the study recommends that BAIs exit the FFU every 10 minutes in order to drink 200 ml of water during LFTEs. However, alternative methods that may also reduce the heat stored in the PC+SCBA which require further investigation include the wearing of cool-vests and wicking garments (to increase evaporative heat loss).
- Drinking 200 ml of water every 10 minutes during LFTEs should not be limited to the fire training ground only. Although the present study monitored BAIs only, it can also be used further a field and embraced by fire fighters responding to emergencies.
- Due to the increase in short term stress responses produced by BAIs during the present study, it follows that they should have a good level of physical fitness. This would allow the BAIs cardiovascular system to cope with the increased

stress placed on them. This increased stress comes from the heat creating a diversion of blood to both the skin and muscle, thus increasing HR.

- Tea, coffee and alcohol have diuretic properties and have a limited use within this situation. Therefore, it may be advised that educating the BAIs to consume rehydration drinks rather than caffeinated drinks would be potentially advantageous in this respect.
- HRV should be monitored regularly (under standardised conditions) to observe increases in the amount of cardiac stress placed on the BAIs, as indicated by increases in the sympathetic indicators and simultaneous decreases in the parasympathetic indicators of HR control.

Future directions.

Further investigation is required to examine the effect of the temperature outside the FFU on the physiological responses of the BAIs. The present study showed small but significant increases in the temperature outside the FFU prior to the LFTEs. Through personal experience and verbal communication with the BAIs, the temperature outside the FFU affects the performance and comfort of the BAIs when conducting LFTEs. Therefore, further studies should investigate the implications of undertaking LFTEs during the summer months and what temperatures outside the FFU constitute safe working environments.

According to the office for national statistics (2001) heart attacks were the largest cause of death in males between the ages of 45 and 54 years of age. Heart attacks claimed 23.5 % of the total number of deaths reported in 2001. Washburn and Harlow (1982) also report that heart attacks are primarily responsible for claiming the lives of fire fighters in the USA. Therefore, further research is required to investigate the usefulness of HRV to identify the key predictors of MI, in order for HRV to be used as an indicator of potential cardiac illness. This would ideally protect the BAIs, and also fire fighters responding to emergencies, from as many further fatalities as possible.

Hydrating the BAIs prior to exposure only, has shown no beneficial effects of lowering the stress responses to LFTEs. Therefore, other strategies to decrease the stress response should be investigated. For example, the usefulness of wearing cool-vests (that incorporate the use of ice packs in a lightweight vest), or wicking garments (to increase evaporative heat loss) that potentially could lower the skin and micro-climate temperatures should be investigated.

HRV could also be used to assess the effectiveness of interventions (such as the cool-vests) that endeavour to lower the stress placed on BAIs during LFTEs.

The present study has concluded that part of the BAI HR response during exposure to LFTEs, is due to the weight of wearing the PC+SCBA. Therefore, future research should investigate the possibility of designing light weight protective clothing, ensuring that thermal protection is not lost in favour of reducing the weight.

The present study has shown that it is beneficial to exit the FFU and drink 200 ml of water every 10 minutes during exposure. However, future studies should investigate whether it is more beneficial to exit the FFU during the LFTEs at 10 minute intervals without drinking, or exit the FFU during the LFTEs at 10 minute intervals with drinking. This would ascertain the most effective method of relieving stress.

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APPENDICES

Appendix A

A1 – RPE Scale

Rate of Perceived Exertion Scale (Borg, 1982)

6	No exertion at all
7	Extremely light
8	
9	Very light
10	
11	Light
12	
13	Somewhat hard
14	
15	Hard (heavy)
16	
17	Very Hard
18	
19	Extremely Hard
20	Maximal Exertion

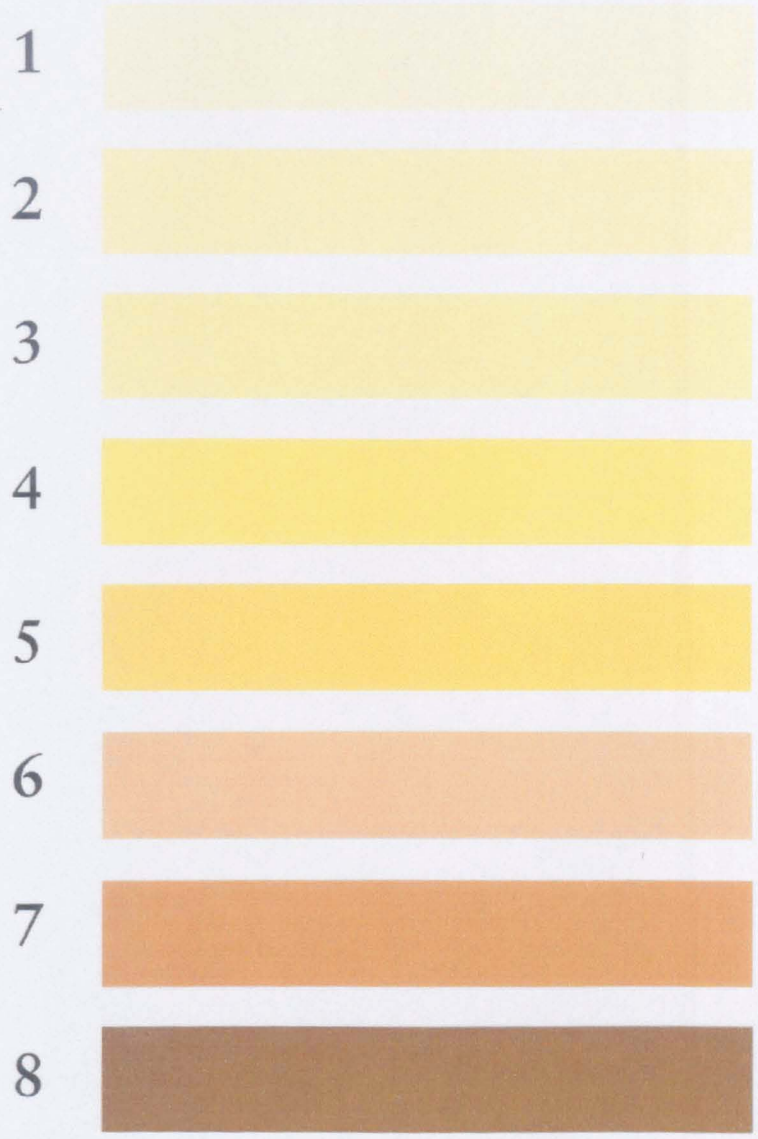
A2 – Discomfort Scale

Scale of Thermal Comfort (Adapted from the RPE scale (Borg, 1982))

1	Thermoneutral
2	Comfortable
3	
4	Very slightly uncomfortable
5	
6	Slightly uncomfortable
7	
8	Somewhat uncomfortable
9	
10	Uncomfortable
11	
12	Very uncomfortable
13	
14	Extremely uncomfortable
15	Maximum tolerance

A3 – Urine colour chart

Urine colour chart (Armstrong *et al*, 1994).



A4 – Consent Forms

INFORMED CONSENT FORM.

Section A – For the subjects;

1. *Stewart Bruce-Low who is a researcher from Chester College, has requested my participation in a research study to be carried out at Chester College. The title of the research proposal is 'Physiological responses to graded exercise whilst wearing protective clothing and self contained breathing apparatus (PC+SCBA)'.*
2. *I have been informed that the purpose of the study is to observe the effects of conducting a Step Test (ST) on heart rate and the oxygen cost of carrying out the activity.*
3. *My participation will involve being tested over a 4 day period. Each of the 4 days will involve carrying out the ST once on each of the days.*

(i) *The initial test will involve carrying out the ST dressed in gym kit whilst having the amount of air you are breathing being monitored via a face mask. A heart rate monitor will be worn in order to obtain resting heart rates and subsequent heart rate readings during the ST.*

(ii) *The second will involve the same protocol as day 1, but you will be dressed in gym kit and wear a rucksack weighted to the equivalent of the PC+SCBA whilst wearing a facemask. A heart rate monitor will be worn in order to obtain resting heart rates and subsequent heart rate readings during the ST.*

(iii) *The third test will involve carrying out the ST in PC+SCBA whilst wearing a facemask. A heart rate monitor will be worn in order to obtain resting heart rates and subsequent heart rate readings during the ST.*

(iv) *The fourth test will involve carrying out the ST in PC+SCBA whilst under air (therefore not with the facemask on). A heart monitor will be worn in order to obtain resting heart rates, and subsequent heart rate readings during the ST.*

(v) *Due to the random nature of the trials, this may not be the order in which you carry out the trials.*

4. *I have also been made aware that I will be required to be weighed in shorts only, as well as in PC+SCBA in order to obtain realistic weighing results.*
5. *I understand that there are foreseeable discomforts and risks that I will be subjecting myself to if I participate in this study. These may include headaches and lethargy.*
6. *There are no alternative procedures to this study that are feasible.*
7. *I understand that the possible benefits of my participation in this research involves obtaining information about the different physiological responses whilst carrying out the ST when dressed in gym kit and PC+SCBA.*
8. *I understand that the study may be published, however, my identity will remain confidential and will only be known to the researcher named above.*
9. *I have been informed that I will not be compensated for my participation from the researcher identified above.*
10. *I have read the above information and the risks, demands and benefits of the study have been explained to me. I knowingly assume the risks involved. I also understand that my consent may be withdrawn and participation terminated at any time, without penalty or loss of benefit to myself. I am not waiving any legal claims, rights or remedies, by signing this consent form. There will be a copy of this consent form given to me.*

Signature of Subject _____ **Date** _____

Section B – For the researcher;

11. *I certify that I have explained to the above individual the purpose and nature, possible risks and potential benefits of this study.*
12. *Furthermore, I certify that I have answered any questions that have been raised, and have witnessed the above signature.*
13. *I have provided the subject with a copy of this consent form.*

Signature of researcher_____ **Date**_____

INFORMED CONSENT FORM.

Section A – For the subjects;

1. *Stewart Bruce-Low who is a researcher from Chester College, has requested my participation in a research study to be carried out at Chester College of Higher Education. The title of the research proposal is 'responses of Heart rate variability to different breathing rates'.*
2. *I have been informed that the purpose of the study is to observe the effects of the different rates of breathing on heart rate variability.*
3. *My participation will involve being tested over a period of 30 minutes. Initially, your body mass will be measured on the Seca scales, and height will be obtained using a free-standing stadiometer. A heart monitor will be worn in order to obtain resting heart rate and HRV.*
4. *There are no alternative procedures to this study that are feasible.*
5. *I understand that the possible benefits of my participation in this research involves obtaining information about the physiological responses of heart rate variability when my breathing is controlled, compared to when I breathe naturally.*
6. *I understand that the study may be published, however my identity will remain confidential and will only be known to the researcher named above.*
7. *I have been informed that I will not be compensated for my participation from the researcher identified above.*
8. *I have read the above information and the risks, demands and benefits of the study have been explained to me. I knowingly assume the risks involved. I also understand that my consent may be withdrawn and participation terminated at any time, without penalty or loss of benefit to myself. I am not waiving any legal claims, rights or remedies, by signing this consent form. There will be a copy of this consent form given to me.*

Signature of Subject _____ Date 6-12-00

Section B – For the researcher;

9. *I certify that I have explained to the above individual the purpose and nature, possible risks and potential benefits of this study.*
10. *Furthermore, I certify that I have answered any questions that have been raised, and have witnessed the above signature.*
11. *I have provided the subject with a copy of this consent form.*

Signature of Researcher _____ Date 06/12/00.

INFORMED CONSENT FORM.

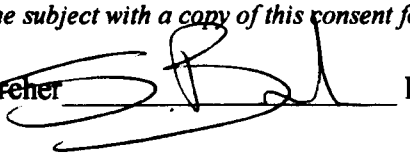
Section A – For the subjects;

1. *Stewart Bruce-Low who is a researcher from Chester College, has requested my participation in a research study to be carried out at Chester College of Higher Education. The title of the research proposal is 'responses of body mass, body temperature, heart rate, heart rate variability, hematocrit volumes, and psychological parameters such as the RPE scale and Stroop test during exposure to intense heat in the Finnish sauna'.*
2. *I have been informed that the purpose of the study is to observe the effects of the sauna on body mass, heart rate, HRV, hematocrit volumes, and psychological parameters such as the RPE scale and the Stroop test.*
3. *My participation will involve being tested over a period of two mornings. The first morning will involve obtaining a maximal aerobic capacity test following the BASES protocol. Body mass will be measured on the Seca scales, and height will be obtained using a free-standing stadiometer. A heart monitor will be worn in order to obtain resting heart rate and HRV. A 2 ml blood sample will be collected via vene-puncture in order for hematocrit volumes to be ascertained. During the single days exposure to the sauna these measurements will be collected immediately pre and post exposure. Some parameters will be measured during exposure. For details refer to table 1 or ask the researcher for clarification.*
4. *I have also been made aware that I will be required to be weighed naked as well as in gym gear in order to obtain realistic weighing results.*
5. *I understand that there are foreseeable discomforts and risks that I will be subjecting myself to if I participate in this study. These may include headaches and lethargy.*
6. *There are no alternative procedures to this study that are feasible.*
7. *I understand that the possible benefits of my participation in this research involves obtaining information about the physiological and psychological responses to the sauna elicited by students, when exposed to a sauna.*
8. *I understand that the study may be published, however my identity will remain confidential and will only be known to the researcher named above.*
9. *I have been informed that I will not be compensated for my participation from the researcher identified above.*
10. *I have read the above information and the risks, demands and benefits of the study have been explained to me. I knowingly assume the risks involved. I also understand that my consent may be withdrawn and participation terminated at any time, without penalty or loss of benefit to myself. I am not waiving any legal claims, rights or remedies, by signing this consent form. There will be a copy of this consent form given to me.*

Signature of Subject _____ Date 04/09/00.

Section B – For the researcher;

11. *I certify that I have explained to the above individual the purpose and nature, possible risks and potential benefits of this study.*
12. *Furthermore, I certify that I have answered any questions that have been raised, and have witnessed the above signature.*
13. *I have provided the subject with a copy of this consent form.*

Signature of Researcher  Date 04/09/00.

INFORMED CONSENT FORM.

Section A – For the instructors;

1. *Stewart Bruce-Low who is a researcher from Chester College, has requested my participation in a research study to be carried out at Greater Manchester Fire Service Headquarters. The title of the research proposal is 'responses of body mass, body temperature, blood pressure, blood glucose blood lactate, heart rate, heart rate variability (HRV), haematocrit volumes, salivary cortisol and psychological parameters such as the RPE scale and adapted Stroop test when exposed to intense heat in the Fire Flashover Unit'.*
2. *I have been informed that the purpose of the study is to observe the effects of the FFU on body mass, body temperature, blood pressure, blood glucose blood lactate, heart rate, heart rate variability (HRV), haematocrit volumes, salivary cortisol and psychological parameters such as the RPE scale and adapted Stroop test.*
3. *My participation will involve being tested over a period of two weeks and a day. The first day will involve obtaining aerobic capacity (by carrying out a Step Test), body mass will be measured on the weighing scales, and height will be obtained using a free-standing stadiometer. A heart monitor will be worn in order to obtain resting heart rate and HRV and a 5 ml blood sample will be collected via vene-puncture in order for haematocrit volumes, glucose and lactate to be ascertained. During the 4 days of exposure to the Fire flashover unit (2 days without the fire and students and 2 days with the fire and students) these measurements will be collected immediately pre and post exposure.*
4. *I have also been made aware that I will be required to be weighed naked as well as in PC+SCBA in order to obtain realistic weighing results.*
5. *I understand that there are foreseeable discomforts and risks that I will be subjecting myself to if I participate in this study. These may include headaches and lethargy.*
6. *There are no alternative procedures to this study that are feasible.*
7. *I understand that the possible benefits of my participation in this research involves obtaining information about the physiological and psychological responses to the FFU elicited by the BAIs, when exposed to the live fire training exercises.*
8. *I understand that the study may be published, however my identity will remain confidential and will only be known to the researcher named above.*
9. *I have been informed that I will not be compensated for my participation from the researcher identified above.*
10. *I have read the above information and the risks, demands and benefits of the study have been explained to me. I knowingly assume the risks involved. I also understand that my consent may be withdrawn and participation terminated at any time, without penalty or loss of benefit to myself. I am not waiving any legal claims, rights or remedies, by signing this consent form. There will be a copy of this consent form given to me.*

Signature of Instructor _____ Date _____

Section B – For the researcher;

11. *I certify that I have explained to the above individual the purpose and nature, possible risks and potential benefits of this study.*
12. *Furthermore, I certify that I have answered any questions that have been raised, and have witnessed the above signature.*
13. *I have provided the subject with a copy of this consent form.*

Signature of Researcher_____ Date_____

INFORMED CONSENT FORM.

Section 1 – For the instructors

1. *Stewart Bruce-Low who is a researcher from Chester College, has requested my participation in a research study to be carried out at Greater Manchester Fire Service Headquarters. The title of the research proposal is 'responses of body mass, body temperature, blood pressure, blood glucose, blood lactate, heart rate, HRV, haematocrit volumes, salivary cortisol and psychological parameters such as the RPE scale and adapted Stroop test during exposure to intense heat in the Fire Flashover unit' when hydrated prior to exposure (consuming 600 ml of water 1 hour prior to exposure), euhydrated prior to exposure (not actively encouraged to drink 2 hours prior to exposure) or drinking during the exposure (consuming 200 ml of water every 10 minutes during the live fire training exercises).*
2. *I have been informed that the purpose of the study is to observe the effects of hydration and the timing of fluid ingestion when exposed to the FFU on body mass, body temperature, blood pressure, blood glucose, blood lactate, heart rate, HRV, haematocrit volumes, salivary cortisol and psychological parameters such as the RPE scale and the adapted Stroop test when exposed to the FFU.*
3. *My participation will initially involve obtaining body water levels, aerobic capacity (by carrying out a Step Test), body mass and height will be obtained using a free-standing stadiometer. A heart monitor will be worn in order to obtain resting heart rate, HRV, and a 5 ml blood sample will be collected via vene-puncture in order for haematocrit volumes, lactate and glucose levels to be ascertained. These measurements will be collected immediately pre and post exposure. During the days of exposure to the Fire flashover unit each instructor will be randomly assigned to each of the four timings of fluid ingestion.*
4. *I have also been made aware that I will be required to be weighed naked as well as in full BA turnout gear in order to obtain realistic weighing results.*
5. *I understand that there are foreseeable discomforts and risks that I will be subjecting myself to if I participate in this study. These may include headaches and lethargy.*
6. *There are no alternative procedures to this study that are feasible.*
7. *I understand that the possible benefits of my participation in this research involves obtaining information about the effect of hydration on the physiological and psychological responses to the FFU produced by the BAIs, when exposed to the live fire training exercises.*
8. *I understand that the study may be published, however my identity will remain confidential and will only be known to the researcher named above.*
9. *I have been informed that I will not be compensated for my participation from the researcher identified above.*
10. *I have read the above information and the risks, demands and benefits of the study have been explained to me. I knowingly assume the risks involved. I also understand that my consent may be withdrawn and participation terminated at any time, without penalty or loss of benefit to myself. I am not waiving any legal claims, rights or remedies, by signing this consent form. There will be a copy of this consent form given to me.*

Signature of Instructor _____ Date _____

Section 2 – For the researcher

11. *I certify that I have explained to the above individual the purpose and nature, possible risks and potential benefits of this study.*
12. *Furthermore, I certify that I have answered any questions that have been raised, and have witnessed the above signature.*
13. *I have provided the subject with a copy of this consent form.*

Signature of researcher_____ Date_____

A5 – Pre-test fitness questionnaire.

Pre-Test Fitness Questionnaire.

Name _____ **D.O.B.** _____ **Max HR** _____

1. Are you at present taking any form of medication? Yes/No?
   If YES please give brief details

   _____

2. Have you had to consult your doctor during the last six months? Yes/No?
   If YES please give brief details

   _____

3. Do you, or have you ever suffered from Diabetes? Yes/No?

4. Do you, or have you ever suffered from Asthma? Yes/No?

5. Do you, or have you ever suffered from Bronchitis? Yes/No?

6. Do you, or have you ever suffered from any other heart complaint, circulatory problems or high or low blood pressure? Yes/No?

7. Is there any history of heart disease in your family? Yes/No?

8. Have you any cause to suspend your normal working day for the past two weeks prior to this testing? Yes/No?

9. Do you currently have any form of joint injury that may be aggravated by exposure to heat? Yes/No?

10. As far as you are aware, are you pregnant? Yes/No?

11. Are you suffering from any infectious skin diseases, sores, wounds, or blood infections i.e., Hepatitis B, HIV, etc.? Yes/No?
    Please give brief details:

    _____

12. Are you suffering from a disease that inhibits the sweating process? Yes/No?

13. Alcohol consumption:
    Abstain Yes/No?
    Occasional Yes/No?
    One per day Yes/No?
    More than one per day Yes/No?

14. Do you use a sauna:
    Never Yes/No?
    Occasional Yes/No?
    Once per day Yes/No?
    Once a week Yes/No?
    More than once a week Yes/No?

15. Do you participate in aerobic exercise? Yes/No?
    If YES, how many times a week?

    _____
    And what is the type of activity?

    _____

16. Finally, is there anything to your knowledge that may prevent you from participating in the testing that have been outlined to you? Yes/No?

Signed (instructor) _____ **Date** _____

Signed(researcher) _____ **Date** _____

A6 – Total body water result sheet

Body Water Result Sheets.

Date.....	**Time.....**
Subject Number.....	**Subject Name.....**

Weight.....Kgs

Height.....cms

ECW.....%
Norm.....%
ECW.....Ltrs
ICW.....%
Norm.....%
ICW.....Ltrs
TBW.....%
Norm.....%
TBW.....Ltrs

Impedance @ 5 Hz.....

Impedance @ 200 Hz.....

A7 – Hydration diary

Hydration Diaries. Date.....

Time	Type of Drink	Quantity

Appendix B

An Adaptation Of The Stroop Test For Time Constrained Situations.

Introduction.

There have been isolated incidences of Breathing Apparatus Instructors (BAIs) collapsing during exposure to the extreme heat of the training environments in which they are required to monitor recruits. This work involves the recruits (students) carrying out the various duties required in a real fire situation and is known as a live fire training exercise (LFTE). The problem exists because the students are being monitored by the BAIs, but no one is monitoring the BAIs. During extreme heat, dehydration and general heat stress can occur. This may have the resultant effect of a loss of concentration and would endanger not only the students but also the BAIs. According to Brooks, Fahey and White (1996) as dehydration levels of even as little as 1% occur, concentration of the individual may be compromised. In a LFTE this is unacceptable.

For this reason, a quick and efficient psychological test was required that did not interfere with the BAIs roles within the LFTEs, yet was still be able to monitor the BAIs concentration within the LFTEs. To conduct the full Stroop test (approximately 2 minutes) with in this type of volatile environment would be very unsuitable. However, by reducing the size of the test (to about 6-10 seconds) the BAIs would not be unnecessarily distracted from their role within the LFTE.

The original Stroop test, designed by Ridley Stroop in 1935, was designed to investigate the effect of interference or inhibition. It was based on the rationale that individuals require substantially longer to name colours when printed in ink of a incongruent colour, than when these colours are presented in non-sensical format, i.e., when in colour patches as an alternative to coloured words (Nealis, 1973). Within fire fighter research the Stroop test (1935) will be used as an indication of concentration. This will be shown by an increased time to complete the test by the BAIs as they lose concentration through heat stress.

Very early reports such as Muller and Schumann (1894) discovered that after original recall, it was necessary to relearn a series of nonsense syllables, especially if these syllables were associated with others simultaneously. This was concluded as the law of associative inhibition (Muller and Schumann, 1894). This is where a visual stimulus such as bus is associated with being red, it becomes very difficult then to associate the bus with blue as red gets in the way.

According to Schack, Chen, Mescha and Witte (1990), there appears to be 3 major explanations of the Stroop effect. Firstly, there is the 'distraction effect'. This is where the colour-word disrupts the identification of the colour that the word is 'typed' in by diverting attention away from the colour of the print. This is also known as perceptual encoding hypothesis (Schack *et al*, 1999). However, it appears that this hypothesis is limited as it can not explain why similar stimuli do not cause interference even when the meaning similarity between word and colour is at its greatest.

The second explanation, known as the response-competition hypothesis (Keele, 1972, Warren, 1972), states that until the motor programmes are activated, colour and words are processed in a very similar manner. According to Schack *et al* (1999) there appear to be two forms of stimulus dimension, a relevant and irrelevant dimension. When there are similar stimuli (congruent) to be named or read, there is no conflict as both these stimuli components activate the same response programme. Reading incongruent material also does not appear to elicit any conflict for responses as the

irrelevant motor programme does not reach the execution stage before the relevant programme. However, when the stimulus is incongruent and more importantly has to be *named*, the irrelevant codes arrive before the relevant codes do. Therefore, the delay in reaction time can be explained by two articulatory codes. The colour the word is printed in and the printed colour word, arrive at the execution stage in quick succession and hence delay occurrs as the subject attempts to suppress the first and execute the second articulatory code (Schack *et al,* 1999). Therefore, according to Ashcraft (1998) an automatic process occurs whether we actually consciously want it to or not.

Thirdly, there is a contradictory hypothesis that believes that the conflict occurs earlier when the stimuli meet the 'meaningful' or semantic memory post perceptual encoding. Therefore, the delay in processing is caused by the incoming stimuli acting as ambiguous information and hence need resolving before further processing (Schack *et al,* 1999).

Cohen, Dunbar and McClelland (1990) state that the simplest explanation for the Stroop effect is the speed of processing. That is to say, individuals are consistently quicker at reading words as opposed to naming colours. For this reason, it is believed that the word that has been read arrives at the stage of processing known as the response stage, which is before the arrival of colour naming. Furthermore, if the colour that the word is printed in is the same as the colour the word describes then this process is facilitated. However, if the colour of the ink and the colour that the word describes is different, this creates an influence on the decision making processes. This means a response may be generated by overcoming the influence of the colour the word describes for the correct response of the colour it is written in. This process will therefore result in a delay and hence longer response time (Cohen *et al*, 1990). Conversely, because colour information arrives after words at the response stage, there is no effect on the word-reading process (Cohen *et al*, 1990).

According to Stroop (1935), one of the factors contributing to interference is the use of colours and their names from previous experience and therefore the strength of the association between the words and the colours that already exists.

Peterson (1925) augmented this by stating a response habit has been formed with each word through experience. However, with the colours themselves there have been a number of responses learned. Stroop (1935) developed this argument further by stating that literate adults have so much practice reading that the Stroop test requires little or no attention and is effortlessly and rapidly completed. In fact, literate adults appear to read so quickly and effortlessly that *not* reading becomes very hard. Therefore, when confronted with a task entailing reading, they can not help but read them. An automatic response is one where little attention and concentration is required (Galotti, 1994).

Ashcraft (1998) agrees with the ideas of Galotti (1994) by stating that it is due to the years of practice, on seeing a printed word its meaning is automatically processed from the memory banks, thus highlighting the differences between automatic and conscious decisions (refer to table 1.1).

Table 1 - Table to show criteria for automatic and conscious processing.

Automatic	Conscious
1. The process occurs *without* intention, without conscious decision.	1. The process only occurs *with* intention, with a deliberate decision.
2. (Informal) The process operates very rapidly, usually within 1 second.	2. (Informal) The process is relatively slow, taking more than a couple of seconds for completion.
3. The process consumes few if any conscious resources, that is, it consumes little if any conscious attention.	3. The process is open to awareness and introspection
4. The mental process is not open to conscious awareness or introspection.	4. The process uses conscious resources; that is, it drains the pool of conscious attention capacity.

Taken from Ashcraft (1998).

Medin and Ross (1997) used the processing model of Cohen, Dunbar and McClelland (1990) (Figure 1) to better understand the mechanisms of the Stroop test. They believe that there is competition of control when reading the colours of the words aloud, between the activation of the colour and word information occurring simultaneously.

The model highlights this further by considering the left hand side input units which are activated by ink colour. On sight of the ink colour, a signal is sent to an intermediate input. Depending on the ink colour, for example if it is red, then an excitatory signal is sent to the intermediate unit straight above it, simultaneously sending an inhibitory signal to the adjacent intermediate unit. Once activated the intermediate unit on the far left will send an excitatory signal to the red response unit and the inside left intermediate (2) will send an inhibitory signal to 'green'. Therefore, when red ink is the visual stimulus there is a process of inhibitory signals to stop a 'green' response being given but excitatory signals to ensure the 'red' response is executed (Medin and Ross, 1997).

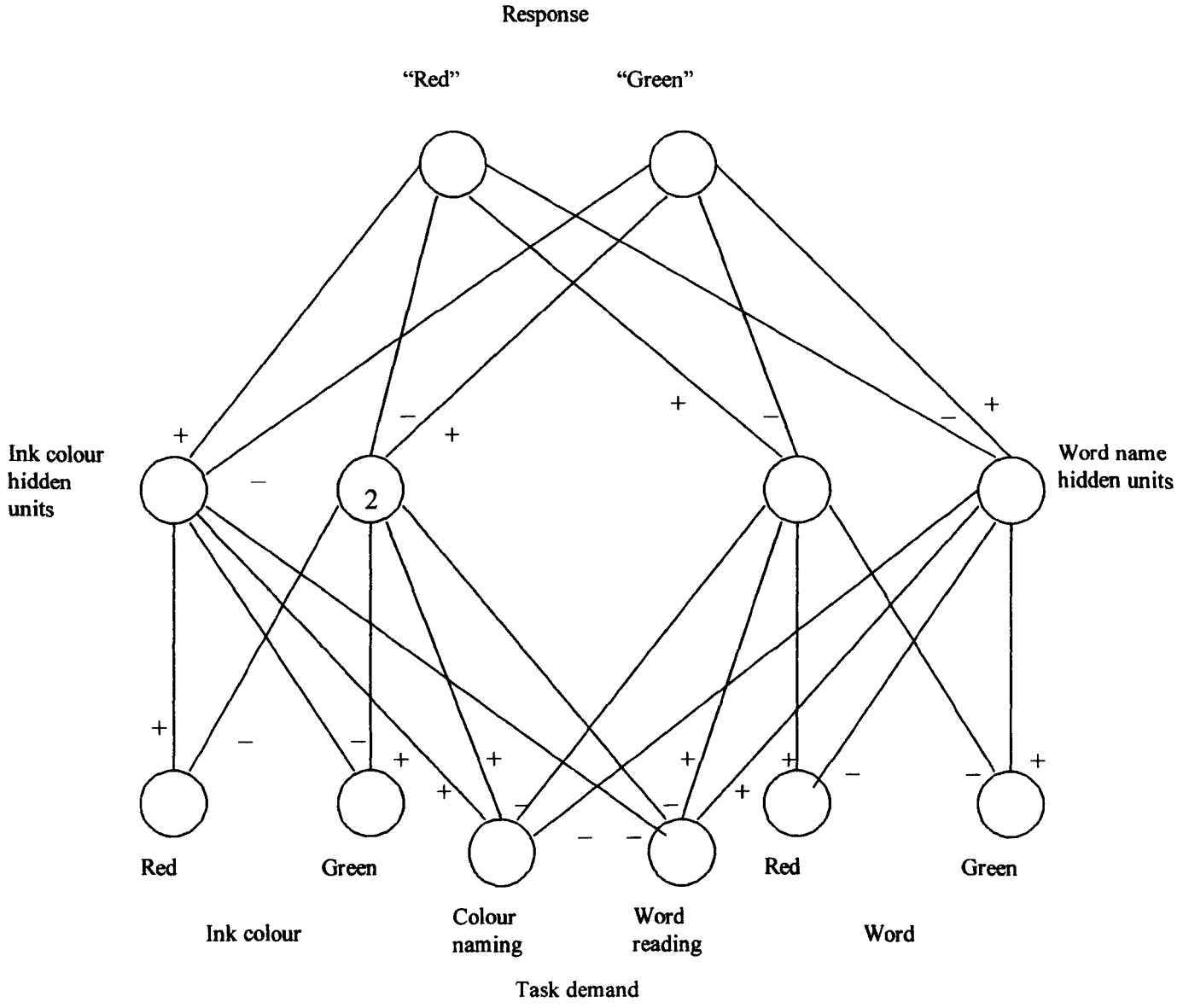
The units on the bottom right hand side represent the colour the word describes rather than the colour it is printed in. As with the ink colour, the visual stimulus of the colour 'red' sends excitatory signals to the response 'red' and inhibitory signals to the response of 'green' and vice versa (Medin and Ross, 1997).

This is a simple concept to follow until the ink colour and word shape are added together. For example, if the word 'green' is printed in 'red' and a subject was required to name the colour that the word was printed in, then there will be excitatory signals from the ink colour units towards a 'red' response. Simultaneously, the exact opposite will occur on the word colour. The confusion is over-ridden by the bottom middle units, the Task demand units. The task given in this example was print colour naming, the bottom-middle-left unit representing colour naming as a task, will activate an inhibitory stimulus towards the green word naming but sends excitatory signals to the intermediate unit to facilitate the ink colour response towards a 'red' response. At the same time sending an inhibitory signal to the ink colour 'green' response (Cohen *et al*, 1990, Medin and Ross, 1997). Meanwhile, the word naming units will be sending excitatory signals to the response 'green' but will be inhibited by the colour naming task demand unit to stop this occurring. Therefore, the result is a slight delay whilst these processes are correlated - this delay became to be known as the Stroop effect.

The adapted Stroop will also follow this model exactly as the components that make up the adapted version do not differ in any way. That is to say, the adaptations still assume there is the same competition for control between reading the colour the

words are printed in and the colour the written word describes as highlighted by Cohen *et al* (1990).

Fig. 1 - Shows the Stroop test according to Cohen *et al* (1990).



The network architecture applied to the Stroop test by Cohen, Dunbar and McClelland (1990).

According to Milliken, Lupianez, Debner and Abello (1999), this may also be highlighted in a slightly different manner by looking at priming. This process has widely been used to look at memory and learning processes (Milliken *et al*, 1999). This theory states that the response to a target stimulus is affected by previous exposure to either the same or similar stimulus. Similarly, time taken to name a word can be effected by how it has been presented previously. These are known as semantic and repetition priming respectively (Milliken *et al*, 1999).

There are many different methods of comprehending the Stroop effect. The above approach utilised by Cohen *et al* (1990) is one such method.

Methods.

Twelve subjects were recruited to the study, mean age 23 (± 7.53) years. Prior to the study, subjects had never seen the Stroop test. Subjects were sat in a well-lit room and were randomly assigned to carryout either the adapted or original Stroop test first.

Each subject was instructed that the test involved naming the colour that the words were printed in and not the colour that the word described. Furthermore, subjects were given one of four pieces of paper in turn, each with a sample of one of the colours that they would encounter during the tests, therefore eliminating any possible confusion of the printed colour. The subjects were also given 5 minutes to practice the Stroop test prior to the actual test session. This accounted for the practice effect on the results. After the practice session, subjects were allowed to converse in quiet conversation with the researcher for a further 5 minutes. After the 5 minutes had elapsed the researcher would ask for a signal from the subject to the researcher to signify that s/he was ready to begin the task.

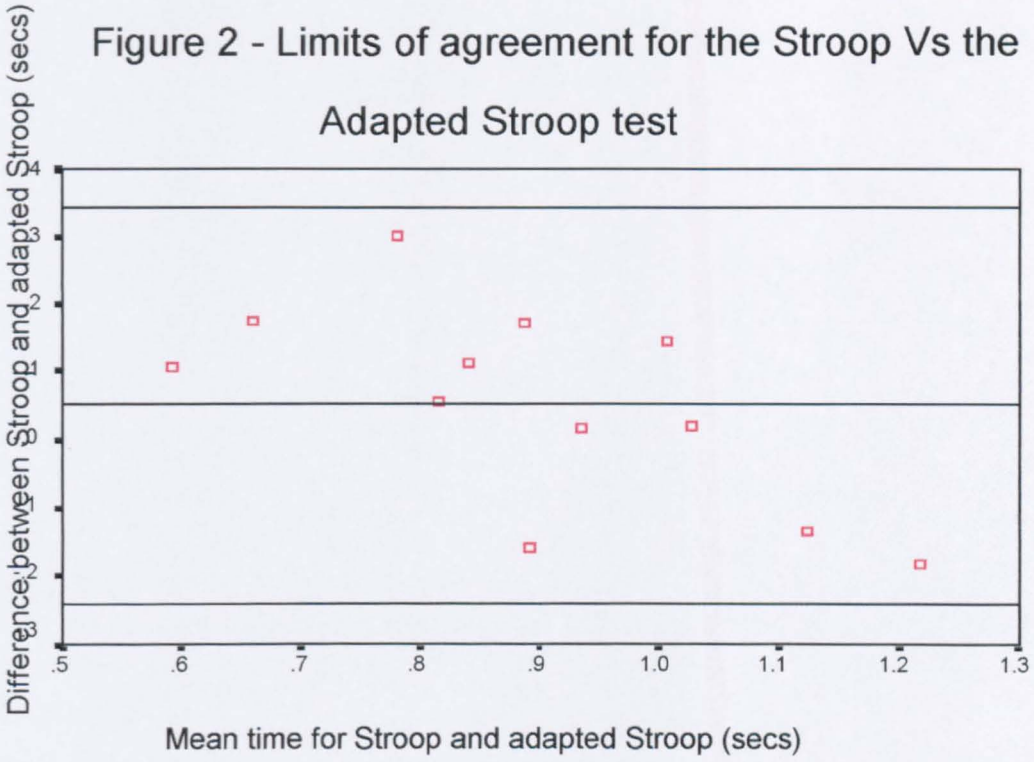
Once this had been observed, the researcher gave the subjects a 3 second countdown before turning over the paper with the Stroop test (or the adapted Stroop test) on it. As the tester turned the paper, the stopwatch was started. The stopwatch was stopped as soon as the subject had said the final colour. After the first trial had been carried out the subjects were required to have a 5 minute break and carry out an alternative cognitive task: either, 'hangman', 'boxes' or 'noughts and crosses'. The subjects were then required to carry out the second test following an identical protocol, this time using the version of the Stroop test they did not use in the initial trial. All subjects were randomly assigned to either carrying out the Original Stroop test first (ST) or the adapted Stroop test (AS).

Results.

The results show that there was a significant increase ($p>0.05$) in total time taken to carry out the ST and AS with a mean time of 92.4 (± 14.3) seconds for the original Stroop and 7.8 (± 2.1) seconds for the adapted Stroop. However, there was a significant correlation ($p<0.05$, $r = 0.8$) between the time taken to complete the ST and AS test per word. The paired t-test that was conducted also highlighted that the mean time for each individual word for both the Stroop and adapted Stroop test was not significantly different with times of 0.9 (± 0.1) seconds and 0.8 (± 0.2) seconds for the original and adapted Stroop respectively.

The Bland and Altman (1986) plot was used in the study as it has been shown to be a very useful indicator of two separate mechanisms closeness using 'limits of agreement' analysis. The Bland and Altman (1986) plot appears to be of more use than a correlation as it is possible to have a significant correlation and not have similar values. The Bland and Altman (1986) plot appears to also suggest that there was a relationship between the two Stroop tests. The mean bias is an acceptable distance from zero showing that there is only a + 0.05 second bias for the time taken to read each word on the adapted test when compared to the original Stroop test.

Figure 2 - Limits of agreement for the Stroop Vs the Adapted Stroop test



The number of mistakes made and in turn the number of corrections made during the Stroop test were significantly increased ($p<0.05$) compared to the adapted Stroop test (the Stroop test produced 58.3 (± 51.5) % more mistakes and 50.0 (± 52.2) % more corrections than the adapted Stroop test). The number of mistakes and corrections made during the two tests were not correlated.

Discussion.

The focus of this discussion is to establish whether the adapted Stroop test is a valid replacement for the original Stroop test. This will be examined through observing statistical analysis of the results of the present study and other investigations that have adapted the Stroop test.

There were no significant differences between the Stroop test or adapted Stroop test in the time to complete the tests per word. In addition, the time to complete the Stroop test and adapted Stroop test were strongly correlated. This suggests there was no significant affect of adapting the Stroop test from the original test design and subjects responded similarly to both tests. This was supported by the Bland and Altman (1986) plot that showed the mean bias was an acceptable distance from zero suggesting that the adapted version is a valid adaptation of the original.

The number of mistakes and corrections made during the Stroop test were significantly greater when compared to the adapted Stroop test. This may suggest that the adapted Stroop test was too short to allow subjects time to make mistakes. Therefore, it appears that time to complete the test should be used when using the adapted Stroop test where the number of mistakes and subsequent corrections made, should be avoided.

The study investigated the validity of an adaptation of the original Stroop to a shorter version for use in the field. The Stroop test has to be taken over a very small time period as the BAIs have to constantly monitor the students through out the LFTEs. In these circumstances it could be argued that the original Stroop test lacked 'ecological validity' (Medin and Ross, 1997). According to Medin and Ross (1997), this means, developing theories that describe cognitive processes that work in everyday situations. The Stroop test works in everyday situations, although this is not the case during LFTEs whilst monitoring the attention/concentration of the BAIs. There was a time limit imposed on the researcher not to impose on the BAIs duties whilst they were conducting LFTEs.

It was not possible to compare the present studies results with other studies as to the best of the authors' knowledge this type of adaptation to the Stroop test has not been done before. This study will therefore attempt to show how other investigations have successfully adapted the Stroop test to their individual needs.

Many investigators have manipulated the Stroop test over the last century (MacLeod, 1992). It has been used as a well-known attentional phenomenon, but is also a research phenomenon in its own right. According to MacLeod (1992) psychologists still do not adequately explain the Stroop effect. This is highlighted by the findings of Schack *et al* (1999) who reported on the effect of the Stroop test and the resultant EEG responses. They stated that there were 3 classes of explanations of the Stroop (previously discussed) demonstrating that the mechanism of the Stroop is still not clearly comprehended.

Studies tended to move away from the original mundane tasks such as opening doors and writing, as carried out by the earlier researchers of the interference effect (not known as the Stroop effect at this time), towards the use of colours. Culler (1912) and Garrett and Lemmon (1924) appear to be among the instigators of colour usage when investigating the effects of interference. Culler (1912) investigated the effect of associating training one hand to react to a certain colour and the other hand of the subject was trained to react to a different colour. The ensuing trial swapped the colour stimuli and found minor interference effects.

Macleod and Dunbar (1988) used an adapted version of the Stroop word colour interference test. They investigated the effects of training on the time delay through word and colour interference. They used the standard version but adapted it through rotating the colour words, i.e., they turned them back to front and upside down. They were still able to show that there were dramatic differences between reading the colour words over that of the 'normal' words ($p<0.001$) (Macleod and Dunbar, 1988).

It appears that the Stroop test is not solely applicable to test interference, but also to depression. Research carried out by Gotlib and McCann (1984) highlighted the diverse uses of the Stroop test. They looked at the effect of carrying out the Stroop test and how the subjects reacted to differing visual stimuli. The authors were able to calculate which were and which were not eliciting symptoms of depression. Their findings showed that individuals who were depressed were slower to name the 'colour' of words that were associated with depression, for example, inadequate and worthless) over that of words that were neutral in nature. Individuals that were not depressed did not exhibit this effect.

Research carried out by Abramczyk, Jordan, and Hegel (1983), investigated the Stroop test when reversed. For example, the subjects were asked to read colour-words, therefore, obtaining a 'reversed' interference effect. Their results showed there were no correlations between the Stroop test when compared to the reversed Stroop. Even though direct comparisons between the present study and other investigations are not appropriate, the results produced by the present study did elicit significant ($p<0.05$) correlations ($r = 0.8$). Abramczyk *et al* (1983), describe the Stroop as a versatile test and that this versatility should be taken advantage of by designing adaptive methods to the Stroop test to use it as an instrument for psychological research.

One such example of this is the investigation conducted by Dalrymple-Alford and Budayr (1966), who successfully adapted the Stroop test through using bilingual students, who were fluent in both English and Arabic. The procedure involved each of the subjects being presented with either an English or Arabic colour name or a nonsense 'squiggle' (the 'squiggle was comprised of a mixture of both English and Arabic letters). The cards were then displayed and the subjects were required to read aloud the colour of the words. The language in which the response was given was not enforced and subjects were encouraged purely to give a response to the visual stimuli as quickly as possible. It was, however, not clear from the investigation whether the students had either of the languages as a first language or second language, and whether this had any effect on the time of the responses given. It is unclear as to the effect that this may have had on the investigation. The study however, clearly showed how the Stroop can also be adapted through using not only colour words and colour print but also the effect the Stroop test has on language and visual concentration and/or interference.

Dalrymple-Alford and Budayr (1966) conclude that by exposing their subjects to several different tasks all based on the Stroop test, the structure of the list does have an effect on reading the test. However, it should be noted that their investigation had moved away from the original structure of the Stroop test and had not followed the basic word structure, i.e., simply name the colour that the words were printed in. As mentioned previously, it was not clear whether it was colour interference or a language interference that was observed in their investigation, as the content of their study used words in both English and Arabic and a third type which was nonsensical through incorporating letters from both languages.

Therefore, it appears that if the basic structure of the original Stroop test is maintained, then any adapted version of the Stroop test remains a valid test and is not affected by changing it e.g., through shortening the test. The adapted version used in the present study still used English words, the same colours and adhered to the same structure as the original Stroop apart from length.

Summary.

The rationale behind adapting the original Stroop test (Stroop, 1935) was to enable it to be used in a field testing environment (fire flashover unit) where BAIs have very little time to carry out a psychological test that can last over 2 minutes in length. The experiment was made up of conducting the Stroop test following the criteria set by Stroop (1935) and an adapted version that used the same criteria for its creation, however, was much shorter in length (100 words and 9 words respectively). As expected, the overall time taken to complete the tests were significantly different ($p<0.01$). However, the difference between the time it took to complete each of the different experiments per word was not significantly different ($p>0.05$) with a significant ($p<0.05$) correlation ($r = 0.8$). The number of mistakes made and correction made between the Stroop test and adapted Stroop test were significantly different, suggesting that time to complete the adapted Stroop test is the most appropriate measure to be used. These results suggest that the adapted Stroop test is a viable adaptation to the original and is a valid replacement for use in a field environment where test time is limited.

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Appendix C

Study Investigating The Effects Of A 30 Minute exposure To A Finnish Sauna On Body Water Levels: Is the BIA method of predicting body water applicable in heated environments?

Introduction.

The purpose of this study was to observe the acute physiological responses to 30 minutes of exposure to the sauna, once a day for five consecutive days and to assess the validity of using the Dual Scan 2005 body water analyser (BodyStat, Isle Of Man, England) after exposure to hot conditions.

The original single bioelectrical impedance analysers have been further developed to measure at various different frequencies. The other methods are known as dual or multi-frequency bioelectrical impedance. According to NIHTACS (1994), it is possible to differentiate between extracellular and intracellular water. They continue that the biophysical significance of reactance appears to be poorly understood. However, it appears to be related to the extracellular/intracellular water ratio. According to Baumgartner (1996), this can be explained through resistance increasing proportionally to reactance. Hence, at low frequencies of a MFBIA current, cell membranes completely block the currents' flow through the intracellular pathway. The measured impedance is therefore representative of the extracellular compartment only. However, as the frequency is increased and there is an increased transmission of the electrical charge across the cell membranes, results in an increase in reactance proportionally to resistance. This reflects high frequencies and represents the combination of both the intra and extracellular compartments (Bauumgartner, 1996).

Therefore, at the lower frequencies used by MFBIA, such as 1 kHz, current flow through the intracellular space is impeded by cell membranes. However, at higher frequencies, i.e., 100 kHz, the electrical current penetrates the cell membranes and passes through all body fluid compartments. Therefore, in theory it should be possible to determine an accurate estimation of intracellular (ICW), extracellular (ECW) and total body water (TBW).

Hannan, Cowan, Plester, Fearon, and Debeau (1995) investigated how the multifrequency BIA (MFBIA) compared to the bioimpedance spectroscopy for the assessment of TBW and ECW. Resistance and reactance were measured at 50 different frequencies ranging from 5 kHz to 1 MHz. Their results exhibited that MFBIA compared favourably to the spectroscopy method (0.9% and 3.0% and 0.9% and 0.6% for coefficients for spectroscopy and MFBIA respectively). The frequencies of 5 kHz and 200 kHz, correlated with the extracellular and total body water readings observed from the bio-impedance spectroscopy most favourably. The Dual Scan 2005 body water analyser (BodyStat, Isle Of Man, England) used with in the present study utilised these frequencies.

Therefore, the present study, in consistency with the findings of Hannan *et al* (1995), utilised the Dual Scan 2005 body water analyser (BodyStat, Isle Of Man, England) that used the dual frequency bands of 5 kHz and 200 kHz that accurately measure body water.

Method.

Seven subjects were used in the study. All were male with a mean age and height of 21.7 (± 2.0) yrs and 178.5 (± 7.1) cm respectively. The South Cheshire Ethics Committee at the Countess of Chester Hospital approved the study.

Each subject gave his informed consent to participate with in the study. They were requested to not eat or drink for up to 4 hours and not to exercise for at least 12 hours prior to testing. On arrival to the sauna room subjects were requested to change into appropriate clothing for the sauna. They were then required to urinate and then to rest for five minutes in a supine position. This was followed by a mean resting heart rate being obtained. This was conducted over a period of two minutes at 15 second intervals.

Measurements for total body water content using the Dual Scan 2005, a dual frequency bioelectrical impedance analyser (Bodystat, Isle of Man, England) were obtained following the guidelines from Bodystat. This was followed by the attainment of the subject's nude weight nude using the Tanita scales (TBF-305).

Exposure to the sauna was for exactly 30 minutes where subjects were required to lie in the supine position. After the 30 minutes had elapsed, the subjects exited from the sauna, undressed and towelled down. They were then required to stand on the Tanita scales and be weighed. Dry shorts were then worn and body water measurements were taken in the same manner as previously described.

The subjects were not exposed again to the sauna that day. However, measurements were taken not only immediately pre and post exposure to the sauna, but also 30 minutes and an hour post exposure following the protocol outlined above for each measurement.

Results.

The mean body mass and body water measurements obtained pre, immediately post exposure, 30 minutes exposure and 1 hour post exposure to the sauna are shown in Table AP1.

Table AP1 – Mean body mass and body water results. Values are mean and standard deviation (± SD) (n = 7). * - Denotes significance ($p<0.05$) between pre exposure and post exposure measurements. ** - Denotes significance ($p<0.05$) between pre exposure and 30 minutes post exposure measurements. *** - Denotes significance between pre exposure and 1 hour post exposure measurements.

	Pre	Post	30 mins post	1 hour post
Body Mass (kg)	78.9 (± 11.0)*	78.3 (± 10.8)	78.3 (± 10.8)**	78.4 (± 10.7)***
ECW (Litre)	18.8 (± 1.8)*	19.0 (± 1.8)	18.8 (± 1.8)	18.6 (± 1.8)***
ICW (Litre)	22.6 (± 2.3)	22.7 (± 2.4)	22.5 (± 2.3)	22.4 (± 2.2)
TBW (Litre)	41.3 (± 4.1)*	41.7 (± 4.2)	41.3 (± 4.1)	41.0 (± 4.0)

There were significant decreases ($p<0.05$) between the pre exposure and immediately post exposure body mass readings, which was further reduced by 30 minutes ($p<0.05$) and was reduced again by the 1 hour post exposure measurements ($p<0.05$). The ECW results showed significant increases in body water between the pre exposure and post exposure which was also true of the TBW measurements. There were no significant changes in the ICW measurements when the 30 minutes post exposure and 1 hour post exposure measurements were compared to the pre exposure measurements.

Table AP 2 shows the mean change and percent change between the pre exposure and immediately post exposure values for body mass and TBW. There was a significant difference ($p<0.05$) between the body mass readings and body water readings when the pre exposure and post exposure changes were compared. There were no differences between the body mass and TBW readings when the pre and 1 hour post changes were compared. The changes between pre and 1 hour post for body mass and TBW were not correlated.

Table AP2 - Mean change and percent change in body mass and TBW. Values are mean and standard deviation (± SD). Changes are between pre exposure and immediately post exposure to the sauna, and also between pre exposure and 1 hour post exposure to the sauna (n = 7). * - Denotes significance ($p<0.05$) between body mass and TBW.

	Change		% Change	
	Body Mass	TBW	Body Mass	TBW
Between Pre and Post	-0.6 (± 0.3)*	0.4 (± 0.6)	-0.7 (± 0.3)*	1.0 (± 1.4)
Between Pre and 1 Hour Post	-0.5 (± 0.6)	-0.3 (± 0.5)	-0.6 (± 0.7)	-0.7 (± 1.1)

Heart rate responses produced by the subjects prior to exposure to the sauna were 71.0 (± 9.7) beats·minute^{-1}. Immediately post exposure to the sauna the average heart rates were 82.0 (± 18.2) beats·minute^{-1} which were significantly increased ($p<0.01$) when compared to the pre exposure values.

Discussion.

The Dual Scan 2005 (Bodystat, Isle of Man, England) appears to be sensitive enough to differentiate between ICW and ECW where both volumes summated to the TBW value. However, when the TBW results were compared to the body mass measurements there was a significant difference. The mean TBW produced an increase of 0.4 (± 0.6) litres, whereas the mean body mass measurements produced losses of 0.6 (± 0.3) kg. This suggests that the Dual Scan 2005 (Bodystat, Isle of Man, England) was not sensitive enough to register water losses when subjects were exposed to a heated environment. However, this was not consistent with Scheltinga, Jacobs and Kimbrough (1991), who believe that the method of BIA is a simple technique that is useful in monitoring minimal alterations in total body water.

However, Pullicino *et al* (1992) observed that measuring patients with altered body water volumes (e.g., oedema) may pose problems when attempting to obtain accurate body water levels. Capillary ultrafiltration is removed by the lymphatic system. When the ultrafiltration becomes excessive, this results in an increase in the interstitial fluid. When this becomes medically detectable then it is said to be

oedema. The increase in fluid will alter the resistivity of the current, thus affect the accuracy of the final body water reading, thus, questioning the usefulness of the method of BIA in individuals with altered body water volumes, such as those with oedema or have recently been exposed to hot conditions as the subjects in the present study.

The present study compared with Caton, Mole, Adams and Heustis (1988), investigated the effects of bioelectrical impedance measurements when their subjects were exposed to heated (35^0C) ambient conditions. Their findings indicated that by altering ambient temperature therefore, altering skin temperature, significantly changes resistance measurements and the estimation of body water measurements (body water increased by 2 litres where the present study increased by 1.0 (± 0.8) litre). Caton *et al* (1988) stipulate that the temperature-induced changes in resistance were due to alterations in cutaneous blood flow and/or compartmental distribution of body water. Caton *et al* (1988), conclude that BIA measurements should only be carried out under well-standardised thermoneutral environments.

However, when Liang and Norris (1993) investigated the effects of skin blood flow and BIA, they did not expose their subjects to heated ambient temperatures. Instead, their subjects exercised at approximately 83% of maximal heart rate for 30 minutes. Their results show that skin temperature and skin blood flow were both significantly increased ($p<0.05$) when their subjects exercised. The BIA resistance and reactance were both unaffected. They conclude that exercise induced skin temperature and blood flow increases do not affect the BIA measurements. However, it is unclear as to whether the skin temperature and skin blood flow increases elicited by the exercise are different to those exhibited by exposure to the heat. If they are indeed different then this may offer an explanation of the differences in results elicited by the different studies.

Table AP2 shows the changes between the pre exposure and 1 hour post exposure measurements for body mass and TBW were not significantly different, which initially suggesting that the body mass losses were reflected in the measurements obtained using the Dual Scan 2005 (Bodystat, Isle of Man, England). However, the changes between the pre exposure and 1 hour post exposure for the losses for body mass and TBW (Table AP2) were not correlated suggesting that although the mean changes in body mass were the same, individually the body mass losses were not reflected when the body water measurements were obtained using the Dual Scan (Bodystat, Isle of Man, England). This signifies that the Dual Scan 2005 (Bodystat, Isle of Man, England) does not accurately reflect body water in subjects who have been exposed to heated environments 60 minutes preceding the body water measurements.

Conclusions.

The Dual Scan 2005 (Bodystat, Isle of Man, England) did not reflect body water as was indicated by the body mass losses immediately post-exposure to the sauna. This was possibly due to an increased skin temperature and skin blood flow, caused by the heat from exposure to the sauna. This was highlighted by the TBW results producing an increase of 0.4 (± 0.6) litres between pre exposure and immediately post exposure

measurements, and the losses of body mass between pre exposure and post exposure, through presumably the sweating response, was 0.6 (± 0.3) kg.

When changes between the pre exposure and 1 hour post exposure measurements for body mass and TBW were compared, although they produced the same mean losses, they were not correlated suggesting the Dual Scan 2005 (Bodystat, Isle of Man, England) was not consistently sensitive enough to detect changes in body water in subjects who had been exposed to a heated environment 60 minutes previously.

The results from the present study suggest using the Dual Scan 2005 (Bodystat, Isle of Man, England) to estimate body water immediately after being exposed to heated environments does not accurately reflect body water. Therefore, the Dual Scan 2005 (Bodystat, Isle of Man, England) should only be utilised when subjects, who have been exposed to heat, are cooled and not still hot from the exposure. Therefore, when measuring BAIs it would seem advisable to monitor their body water (using the Dual Scan 2005 (Bodystat, Isle of Man, England)) at the beginning of the shift and then again at the termination of the shift, thus allowing time for the BAIs to cool down from exposure to the LFTEs.

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Appendix D

HEAT STRESS RESPONSES TO FIRE FIGHTER BREATHING APPARATUS INSTRUCTORS (BAIs) TO LIVE FIRE TRAINING EXERCISES (LFTEs).

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The increase in heart rate (HR) in response to heat stress is well documented (1). However, the increase in HR elicited during LFTEs may be caused by the intensity of work, the weight of wearing protective clothing and self-contained breathing apparatus (PC+SCBA) (20 kg), heat storage from the PC, the heat from the fire (typical shoulder temperatures are 200^{0}C), and psychological stress elicited from supervising the fire fighter students

To observe the effects of wearing PC+SCBA on the heart rate responses and oxygen cost, six fire fighters (mean age 40.6 (± 2.9 S.D) years, weight 77.1 (± 5.8) kg and height 173.5 (±2.6) cm) carried out the Chester Step Test (CST) (3) with either gym kit (thermoneutral conditions) or PC+SCBA (Table 1).

Table 1 –Differences in HR and O_2 cost responses to the CST dressed in gym kit or PC+SCBA.(*P<0.05, paired t-test)

CST Level	HR Gym	HR (PC+SCBA)	O_2 cost (gym)		O_2 cost (PC+SCBA)	
	(bpm)	(bpm)	ml/kg/min	% max	ml/kg/min	% max
1 (Low)	93.8 (± 10.3)	113.5(± 10.8)*	17.3	37.1	23.1*	49.6
2	101.7(± 11.3)	122.5(± 11.8)*	19.2	41.2	27.2*	58.4
3	115.0(± 13.5)	141.0(± 9.6)*	23.6	50.6	32.8*	70.4
4	133.4(± 12.7)	156.6(± 8.1)*	29.8	63.9	39.5*	84.8
5 (High)	152.7(± 13.6)	170.0(± 7.2)*	36.4	78.0	46.6*	100.0

Thus the cardiovascular stress elicited from the workload and weight of the PC+SCBA can be quantified (Table 1).

HR and workload were then measured during the LFTE. The oxygen cost was indirectly measured (2). Exercise at approximately 16mls $O_2/kg^{-1}/min^{-1}$ elicited peak heart rates of 150 bpm. In comparison, CST in gym kit, an oxygen cost of 17 ml O_2/kg/min elicited a HR of 93 bpm and with PC+SCBA, 113 bpm (Table 1). In order to investigate this additional 37 bpm in HR in LFTE, aural, skin and microclimate temperatures were recorded during the LFTE.

A steady increase in aural temperature from 35.0 to 36.5 C, microclimate from 30.5 to 35.9 °C and skin from 31.2 to 36.8 °C suggests that a rise in temperature within the PC may also be responsible for part of the increase in HR. This suggests that heat dissipation from the PC may be important in reducing heat stress responses during LFTEs.

1.Cochrane D. J., Sleivert G. G. (1999) Do changing patterns of heat and humidity influence thermoregulation and endurance performance? J.Sci.Med. Sport. 2 (4): 322-332.
2. Louhevaara V., Tuomi T., Korhonen O., Jaakkola J. (1984). Cardiorespiratory effects of respiratory protective devices during exercise in well-trained men. Eur. J. Applied Physiol. 52:340-345.
3. Sykes K (1995). Aerobic capacity assessment in the workplace: A new step test. *J. Occ. Health.* :20-22.

The Chester College Ethics Committee and Fire Safety Officer from the Greater Manchester Training headquarters approved the study.

Appendix E

There is a high scatter of results, as represented by the high standard deviation of the absolute values in the Tables listed in these Appendices.

For example the SD expressed as a percent of the mean HR (in ms^{-1}) (Table 7.4a) shows the intra-subject variability. These results show that the standard deviation of the individual subject means are low, showing it is the large inter-subject variability that causes the large SD of the mean values. It is possible to observe that when the mean HR taken from a single subject is observed, the mean HR (msec) has a low standard deviation (Subject 7 mean HR prior to sauna exposure was 934.5 (± 103.3) msec). However, the mean absolute HF values observed prior to the sauna for ten subjects was 1835.8 (± 1440.7) $msec^2$.

However, the high scatter has been taken into account through either using a paired t-test, or the data has not been analysed in the result sections.

Table 7.1a – Means (M), standard deviations (SD) mean changes (change) and mean % change (%) for heart rate variability when subjects breathing frequency was either controlled at 12 breaths·minute^{-1} (0.2 Hz) (CB) or breathing frequency was spontaneous (SB) (n = 18).

Variable	Units	CB		SB			
		M	SD	M	SD	Change	%
Breathing Frequency	Cycles · min^{-1}	12	00.0	12.3	0.9	0.3	2.4 NS
Heart Rate	Bpm	74.0	16.2	73.0	15.3	-1	-1.3 NS
Interbeat intervals SD	msec.	70.2	29.6	72.6	27.3	2.4	3.3 NS
SD as a % of mean HR	%	7.7	2.4	8.0	2.4	0.3	3.8 NS
PNN50	%	12.7	9.8	14.1	9.5	1.4	9.9 NS
RMSSD		47.7	27.9	49.0	26.6	1.3	2.7 NS
Power	msec^2.						
Total		7104.9	5675.2	7910.4	5910.3	805.5	10.2 NS
Very Low Frequency		3617.2	3066.5	4687.5	4535.7	1070.3	22.8 NS
Low Frequency		2049.2	1090.5	1993.3	1413.9	-55.9	-2.7 NS
High Frequency		1438.6	1392.2	1229.6	1128.2	-209	-14.5 NS
Normalised Low Frequency power	Normalised units	65.3	22.4	64.5	22.3	-0.8	-1.2 NS
Normalised high frequency	Normalised units	34.6	22.4	35.5	22.3	0.9	2.5 NS
Low frequency to high frequency ratio	%	4.6	5.6	4.2	5.6	-0.4	-8.7 NS

* - Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.4a – Means (M), standard deviations (SD), change (change), % change (%), and p values for all heart rate variability variables prior to sauna exposure and during sauna exposure (n = 10).

Variable	Units	Pre		Sauna			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	66.5	8.1	106.0	11.7	39.5	60.7*
Interbeat intervals SD	msec.	84.7	25.9	30.6	9.0	-54.1	63.9*
SD as a % of the Mean HR	%	9.3	3.1	5.4	1.6	-3.9	41.9*
PNN50	%	15.9	7.7	0.2	0.2	-15.7	98.7*
RMSSD	msec.	57.4	23.2	8.8	4.0	-48.6	84.7*
Power	msec^2.						
Total		9682.8	5849.2	860.2	663.1	-8822.6	91.1*
Very Low Frequency		5626.8	5003.6	582.8	501.2	-5044	89.6*
Low Frequency		2220.1	1272.7	257.4	177.6	-1962.7	88.4*
High Frequency		1835.8	1440.7	20	31.2	-1815.8	98.9*
Normalised Low Frequency power	Normalised units	58.4	21.8	93.3	6.2	34.9	59.8*
Normalised high frequency	Normalised units	41.6	21.8	6.7	6.2	-34.9	83.9*
Low frequency to high frequency ratio	%	1.9	1.8	24.7	16.7	22.8	8.2*
Discomfort scale		6.0	2.5	11.6	0.4	5.4	87.1*

* - Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Figure 7.6a - Subject 1 Pre Sauna Exposure

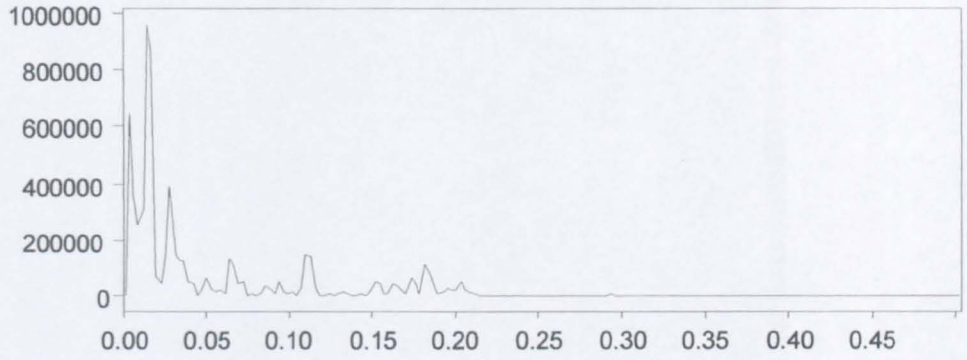

Figure 7.6b - Subject 1 During Sauna Exposure

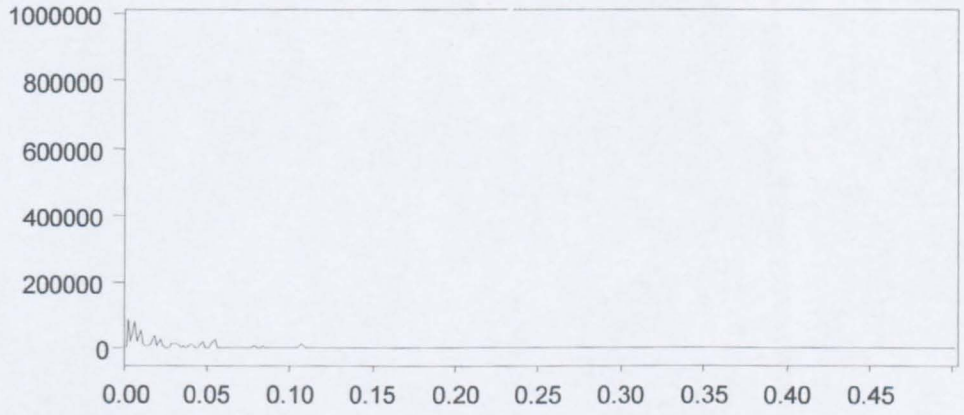

Figure 7.6c - Subject 2 Pre Sauna Exposure

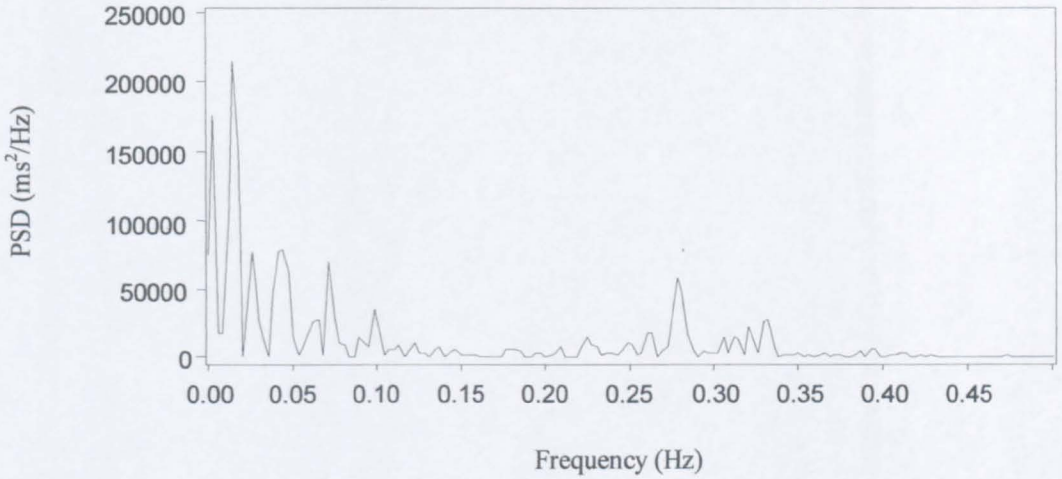

Figure 7.6d - Subject 2 During Sauna Exposure

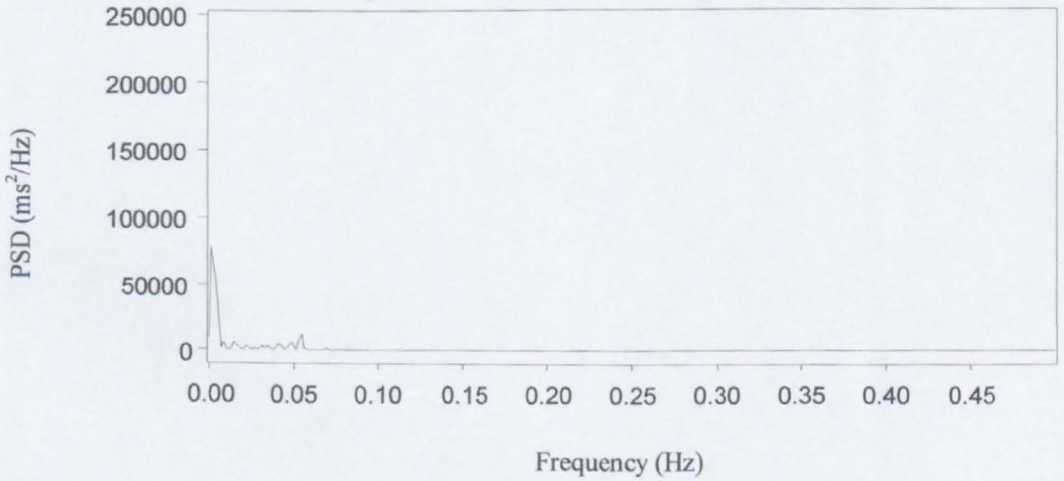

Table 7.5 – Means (M), standard deviations (SD), change (Pre Mock means subtracted from Pre LFTE means), % change (percentage change between the pre mock mean and Pre LFTEs mean), and p values for all HRV variables between pre exposure to the Mock and LFTEs whilst dressed in PC+SCBA (n = 6).

Variable	Units	Pre Mock Fires		Pre LFTEs			
		M	SD	M	SD	Change	% Diff †
Heart Rate	Bpm	71.4	11.7	75.8	16.9	4.4	6.2 NS
Interbeat intervals SD	msec.	49.9	16.0	45.0	19.5	-4.9	-9.8 NS
SD as a % of the Mean HR	%	5.7	1.3	5.3	1.9	-0.4	-7.0 NS
PNN50	%	4.0	4.8	4.0	5.5	0.0	0.0 NS
RMSSD	msec.	26.5	13.1	23.3	14.2	-3.2	-12.1 NS
Power	$msec^2$.						
Total Power (TP)		3149.4	1806.0	2773.5	1883.3	-375.9	-11.9 NS
Very Low Frequency		2126.1	1258.7	1870.8	1493.6	-255.3	-12.0 NS
Low Frequency		776.8	418.8	696.7	520.3	-80.1	-10.3 NS
High Frequency		246.4	229.4	206.0	275.2	-40.4	-16.4 NS
Normalised Low Frequency power	Normalised units	80.5	10.1	80.6	18.2	0.1	0.1 NS
Normalised high frequency	Normalised units	19.5	10.1	19.3	18.2	-0.2	-1.0 NS
Low frequency to high frequency ratio	%	5.5	3.3	8.8	7.4	3.3	60 NS

* - Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.6 – Means (M), standard deviations (SD), change (Post Mock means subtracted from Post LFTE means), % change (percentage change between the Post mock mean and Post LFTEs mean), and p values for all HRV variables between Post exposure to the Mock and LFTEs whilst dressed in PC+SCBA (n = 6).

Variable	Units	Post Mock Fires		Post LFTEs			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	69.3	8.9	90.8	17.3	21.5	31.0*
Interbeat intervals SD	msec.	60.6	28.1	32.7	14.3	-27.9	-46.0*
SD as a % of the Mean HR	%	7.0	3.7	4.7	1.6	-2.3	-32.9*
PNN50	%	3.9	3.9	0.5	1.0	-3.4	-87.2*
RMSSD	msec.	30.2	17.0	12.1	8.1	-18.1	-59.9*
Power	$msec^2$.						
Total Power (TP)		4741.5	3625.0	1107.8	1193.0	-3633.7	-76.6*
Very Low Frequency		3215.8	2700.6	705.3	815.1	-2510.5	-78.1*
Low Frequency		1199.0	580.2	347.4	341.1	-851.6	-71.0*
High Frequency		326.7	444.3	55.2	89.5	-271.5	-83.1*
Normalised Low Frequency power	Normalised units	82.9	9.6	88.8	6.6	5.9	7.1*
Normalised high frequency	Normalised units	17.1	9.6	11.2	6.6	-5.9	-34.5*
LF:HF ratio	%	6.1	2.5	10.8	6.4	4.7	77.0*

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.8 – Means (M), standard deviations (SD), change (Change) and percentage change (%), and p values for all HRV variables between Pre exposure to LFTEs when in hydrated (Hyd) and euhydrated (Euh) states prior to exposure, whilst dressed in PC+SCBA during Protocol 2. (n = 6).

Variable	Units	Euh		Hyd			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	72.3	12.2	74.7	16.4	2.4	3.3 NS
Interbeat intervals SD	msec.	64.1	36.2	49.5	20.5	-14.6	-22.8 NS
SD as a % of the Mean HR	%	7.3	3.6	5.8	1.8	-1.5	-20.5 NS
PNN50	%	5.5	6.7	3.9	4.6	-1.6	-29.1 NS
RMSSD	msec.	31.0	17.8	24.9	13.9	-6.1	-19.7 NS
Power	$msec^2$.						
Total		7586.3	8962.4	3537.7	3323.8	-4048.6	-53.4 NS
Very Low Frequency		5239.9	7618.8	2418.7	1908.6	-2821.2	-53.8 NS
Low Frequency		1297.5	1263.4	1122.3	1345.2	-175.2	-13.5 NS
High Frequency		355.9	346.0	194.7	165.5	-161.2	-45.3 NS
Normalised Low Frequency power	Normalised units	79.4	13.6	81.8	15.7	2.4	3.0 NS
Normalised high frequency	Normalised units	20.6	13.6	18.2	15.7	-2.4	-11.6 NS
Low frequency to high frequency ratio	%	5.9	4.1	8.7	5.0	2.8	47.5 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.9 – Means (M), standard deviations (SD), change (Change) and percentage change (%), and p values for all HRV variables between Post exposure to LFTEs when in hydrated (Hyd) and euhydrated (Euh) states prior to exposure, whilst dressed in PC+SCBA during Protocol 2. (n = 6).

Variable	Units	Euh		Hyd			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	91.3	24.3	105.6	16.5	14.3	15.7 NS
Interbeat intervals SD	msec.	39.4	18.3	22.3	8.8	-17.1	-43.4*
SD as a % of the Mean HR	%	5.4	1.6	3.8	1.1	-1.6	-29.6*
PNN50	%	2.6	3.7	0.0	0.0	-2.6	-100*
RMSSD	msec.	21.0	15.2	5.4	2.8	-15.6	-74.3*
Power	$msec^2$.						
Total		2678.7	2039.3	242.9	172.3	-2435.8	-90.9*
Very Low Frequency		1851.1	1603.3	178.3	119.4	-1672.8	-90.4*
Low Frequency		656.9	693.6	57.1	46.6	-599.8	-91.3*
High Frequency		170.7	184.5	7.5	7.2	-163.2	-95.6*
Normalised Low Frequency power	Normalised units	80.2	18.1	88.8	3.9	8.6	10.7 NS
Normalised high frequency	Normalised units	19.9	18.1	11.2	3.7	-8.7	-43.7 NS
Low frequency to high frequency ratio	%	10.2	11	9.1	3.8	-1.1	-10.8 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.10 – Means (M), standard deviations (SD), change (Change) and percentage change (%), and p values for all HRV variables between Pre exposure to LFTEs when in hydrated (Hyd) and euhydrated (Euh) states prior to exposure, whilst dressed in PC+SCBA during Protocol 3. (n = 6).

Variable	Units	Euhydrated		Hydrated			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	70.3	15.9	71.7	12.9	1.4	2.0 NS
Interbeat intervals SD	msec.	61.8	30.8	71.5	69.9	9.7	15.7 NS
SD as a % of the Mean HR	%	6.8	2.6	7.8	6.2	1.0	14.7 NS
PNN50	%	6.0	6.6	4.0	5.6	-2.0	33.3 NS
RMSSD	msec.	27.6	17.5	25.2	17.0	-2.4	8.7 NS
Power	$msec^2$.						
Total		4952.1	5108.5	5793.3	8811.6	841.2	17.0 NS
Very Low Frequency		3737.7	4136.2	4422.9	6923.8	685.2	18.3 NS
Low Frequency		933.0	809.3	1118.7	1606.0	185.7	19.9 NS
High Frequency		281.4	294.5	251.7	313.3	-29.7	10.6 NS
Normalised Low Frequency power	Normalised units	79.9	15.6	81.7	12.0	1.8	2.3 NS
Normalised high frequency	Normalised units	20.1	15.6	18.3	12.0	-1.8	9.0 NS
Low frequency to high frequency ratio	%	8.3	6.0	7.3	6.4	-1.0	12.0 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.11 – Means (M), standard deviations (SD), change (Change) and percentage change (%), and p values for all HRV variables between Post exposure to LFTEs when in hydrated (Hyd) and euhydrated (Euh) states prior to exposure, whilst dressed in PC+SCBA during Protocol 3. (n = 6).

Variable	Units	Euhydrated		Hydrated			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	87.7	16.1	90.8	15.0	3.1	3.5 NS
Interbeat intervals SD	msec.	34.6	17.9	30.2	13.9	-4.4	12.7 NS
SD as a % of the Mean HR	%	4.7	1.8	4.3	1.2	-0.4	8.5 NS
PNN50	%	0.9	1.9	0.4	0.9	-0.5	55.6 NS
RMSSD	msec.	12.1	10.8	9.6	8.2	-2.5	207 NS
Power	$msec^2$.						
Total		1425.1	1981.2	1246.3	2077.2	-178.8	12.5 NS
Very Low Frequency		1076.9	1667.0	904.7	1524.0	-172.2	16.0 NS
Low Frequency		279.9	254.3	306.7	491.1	26.8	9.6 NS
High Frequency		68.3	136.6	35.0	63.6	-33.3	48.8 NS
Normalised Low Frequency power	Normalised units	88.5	12.8	91.1	3.1	2.6	2.9 NS
Normalised high frequency	Normalised units	11.5	12.8	9.0	3.1	-2.5	21.7 NS
Low frequency to high frequency ratio	%	16.4	13.1	12.1	6.8	-4.3	26.2 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.12 – Means (M), standard deviations (SD), change (Change), percentage change (%) and p values for all HRV variables pre exposure to LFTEs when in either a hydrated state prior to the LFTEs (Hyd) or euhydrated state pre exposure but drinking during the LFTEs (During), whilst dressed in PC+SCBA during Protocol 3 (n = 6).

Variable	Units	Hyd		During			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	71.7	12.9	68.7	13.4	-3.0	4.2 NS
Interbeat intervals SD	msec.	71.5	69.9	53.4	16.1	-18.1	25.3 NS
SD as a % of the Mean HR	%	7.8	6.2	6.0	2.2	-1.8	23.1 NS
PNN50	%	4.0	5.6	4.2	4.7	0.2	5.0 NS
RMSSD	msec.	25.2	17.0	25.1	12.2	-0.1	0.4 NS
Power	$msec^2$.						
Total		5793.3	8811.6	2954.6	2121.2	-2838.7	49.0 NS
Very Low Frequency		4422.9	6923.8	2018.9	1445.2	-2404.0	54.4 NS
Low Frequency		1118.7	1606.0	721.9	597.4	-396.8	35.5 NS
High Frequency		251.7	313.3	213.8	185.6	-37.9	15.1 NS
Normalised Low Frequency power	Normalised units	81.7	12.0	78.0	15.2	-3.7	4.5 NS
Normalised high frequency	Normalised units	18.3	12.0	22.0	15.2	3.7	20.2 NS
Low frequency to high frequency ratio	%	7.3	6.4	7.0	4.9	-0.3	4.1 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.13 – Means (M), standard deviations (SD), change (Change), percentage change (%) and p values for all HRV variables post exposure to LFTEs when in either a hydrated state prior to the LFTEs (Hyd) or euhydrated state pre exposure but drinking during the LFTEs (During), whilst dressed in PC+SCBA during Protocol 3 (n = 6).

Variable	Units	Hydrated		During			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	90.8	15.0	79.8	21.8	-11	12.1 NS
Interbeat intervals SD	msec.	30.2	13.9	50.8	17.9	20.6	68.2*
SD as a % of the Mean HR	%	4.3	1.2	6.4	1.3	2.1	48.8*
PNN50	%	0.4	0.9	2.2	1.1	1.8	450*
RMSSD	msec.	9.6	8.2	20.1	11.6	10.5	109.3*
Power	$msec^2$.						
Total		1246.3	2077.2	2256.6	2397.5	1010.3	81.1*
Very Low Frequency		904.7	1524.0	1325.0	1822.8	420.3	46.5*
Low Frequency		306.7	491.1	802.9	576.6	496.2	161.8*
High Frequency		35.0	63.6	128.6	154.0	93.6	267.4 NS
Normalised Low Frequency power	Normalised units	91.1	3.1	88.7	6.3	-2.4	2.6 NS
Normalised high frequency	Normalised units	9.0	3.1	11.3	6.3	2.3	25.6 NS
Low frequency to high frequency ratio	%	12.1	6.8	11.1	7.9	-1.0	8.3 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.14 – Means (M), standard deviations (SD), change (Change), percentage change (%) and p values for all HRV variables pre exposure to LFTEs when in either a euhydrated state prior to LFTEs (Euh) or euhydrated state pre exposure but drinking during the LFTEs (During), whilst dressed in PC+SCBA during Protocol 3 (n = 6).

Variable	Units	Euh		During			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	70.3	15.9	68.7	13.4	-1.6	2.3 NS
Interbeat intervals SD	msec.	61.8	30.8	53.4	16.1	-8.4	13.6 NS
SD as a % of the Mean HR	%	6.8	2.6	6.0	2.2	-0.8	11.8 NS
PNN50	%	6.0	6.6	4.2	4.7	-1.8	30 NS
RMSSD	msec.	27.6	17.5	25.1	12.2	-2.5	9.1 NS
Power	$msec^2$.						
Total		4952.1	5108.5	2954.6	2121.2	-1997.5	40.3 NS
Very Low Frequency		3737.7	4136.2	2018.9	1445.2	-1718.8	46.0 NS
Low Frequency		933.0	809.3	721.9	597.4	-211.1	22.6 NS
High Frequency		281.4	294.5	213.8	185.6	-67.6	24.0 NS
Normalised Low Frequency power	Normalised units	79.9	15.6	78.0	15.2	-1.9	2.4 NS
Normalised high frequency	Normalised units	20.1	15.6	22.0	15.2	1.9	9.5 NS
Low frequency to high frequency ratio	%	8.3	6.0	7.0	4.9	-1.3	15.7 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Table 7.15 – Means (M), standard deviations (SD), change (Change), percentage change (%) and p values for all HRV variables post exposure to LFTEs when in either a euhydrated state prior to LFTEs (Euh) or euhydrated state pre exposure but drinking during the LFTEs (During), whilst dressed in PC+SCBA during Protocol 3 (n = 6).

Variable	Units	Euhydrated		During			
		M	SD	M	SD	Change	%
Heart Rate	Bpm	87.7	16.1	79.8	21.8	-7.9	9.0 NS
Interbeat intervals SD	msec.	34.6	17.9	50.8	17.9	16.2	46.8 NS
SD as a % of the Mean HR	%	4.7	1.8	6.4	1.3	1.7	26.6 NS
PNN50	%	0.9	1.9	2.2	3.1	1.3	144.4 NS
RMSSD	msec.	12.1	10.8	20.1	11.6	8.0	66.1 NS
Power	$msec^2$.						
Total		1425.1	1981.2	2256.6	2397.5	831.5	58.3 NS
Very Low Frequency		1076.9	1667.0	1325.0	1822.8	248.1	23.0 NS
Low Frequency		279.9	254.3	802.9	576.6	523.0	186.9*
High Frequency		68.3	136.6	128.6	154.0	60.3	88.3 NS
Normalised Low Frequency power	Normalised units	88.5	12..8	86.7	6.3	-1.8	2.0 NS
Normalised high frequency	Normalised units	11.5	12.8	13.3	6.3	1.8	15.7 NS
Low frequency to high frequency ratio	%	16.4	13.1	11.1	7.9	-5.3	32.3 NS

*- Denotes significance ($p<0.05$) / NS – Denotes no significant difference ($p>0.05$), using a paired samples t-test.

Appendix F

Figure App 1 – shows a BAI carrying out the adapted Stroop test prior to exposure.

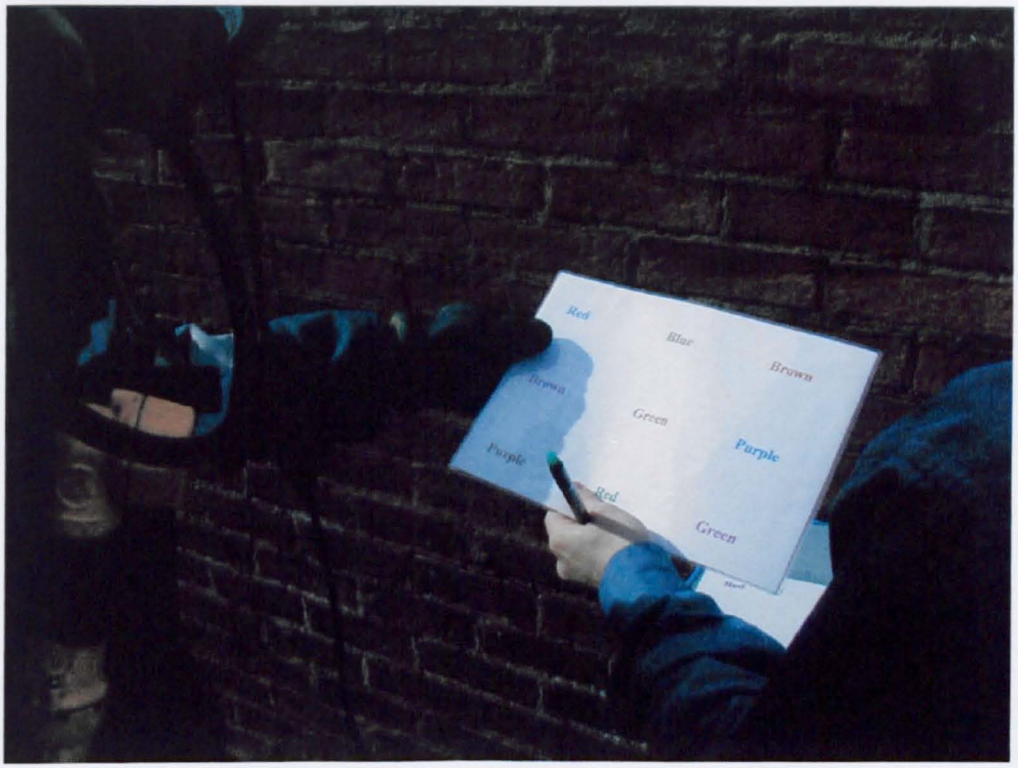

Figure App 2 – shows the BAI on the right is dressed to go into the FFU to carry out a LFTE and the BAI on the left is dressed to go into the control room.

Figures App 3, 4 and 5 – show how fluid was administered to the BAIs when they drank during the LFTEs. By adapting the oral-nasal mask it was possible to insert a straw that allowed the BAIs to drink without removing the SCBA.

Figure App 3

Figure App 4

Figure App 5

Figures App 6 and App 7 show the 'in the field' laboratory.

Figure App 6 – Field Laboratory.

App 7 – Field laboratory.