

A COMPARISON OF TEMPERATURES INSIDE PROTECTIVE HEADGEAR WITH
INDICATORS OF PHYSIOLOGICAL STRAIN AND CORE TEMPERATURES DURING
EXERCISE IN A HOT ENVIRONMENT

By

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A COMPARISON OF TEMPERATURES INSIDE PROTECTIVE HEADGEAR WITH
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CHAPTER ONE

INTRODUCTION

Background

Because humans are homeotherms, proper thermoregulation is critical during physical activity when metabolic heat production or environmental heat gain can lead to substantial disturbances in thermal homeostasis. Even deviations of 3.5° C above or below a typical resting core temperature of approximately 37° C, (6, 20, 46) can lead to cell impairment or death (46). If the body's core temperature exceeds 42° C, damage can occur to the cytoskeleton of the cells, leading to the dysfunction of both the central nervous system and other major organ systems (10, 15, 46). Over the past few decades, there has been growing concern about heat related illnesses. Heat illness is the third cause of death in US high school athletes (23). The National Center for Catastrophic Sport Injury Research reported 127 deaths from heat stroke in football players from 1960-2009. Since 1995 there have been 46 reported deaths in football from heat stroke. This is a conservative number because many heat related injuries go unreported (67). From the year 1980 to 2002 the rate of heat stroke hospitalization for the US Army increased five-fold (15). The reason for this increase remains unclear.

Countless other athletes, (1) physical laborers, (67) and military personnel (3, 14) suffer symptoms of heat illness or heat exhaustion. This risk is further magnified when uniforms, protective clothing or headgear must be worn to prevent head trauma, exposure to harmful chemicals or other environmental hazards (3, 14, 32). Given that the onset of heat-related illness is often associated with a rise in core temperature (T_c) (23), accurately monitoring T_c in clinical or field settings is an essential step in combatting heat illness.

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Methods of Measuring Core Temperature

Core temperature includes temperatures recorded in the abdominal, thoracic or cranial cavities. Temperature is regulated in the hypothalamus (6, 28, 45, 46), and at rest, T_c averages 36.8° C. “True” core temperature is taken from the blood at the intra pulmonary artery (IPA); however, this method is highly invasive and impractical for field or laboratory settings (28, 46). As a result, in these settings, if assessed at all, T_c is most commonly measured as a rectal (T_{re}) or an esophageal (T_{es}) temperature (17). Because these methods are also somewhat invasive, they can be impractical; thus, an accurate and reliable method for monitoring temperature that is easily accessible, instantaneous and non-invasive can be of value to the occupational medicine, sports medicine, and research communities.

The Role of the Head in Thermoregulation

The head weighs approximately 4-5kg which contributes to 7% of total body mass and 7% of the body’s total surface area (14, 67). The surface area to mass ratio of the head is 1 to 6, making it equivalent to that of the torso (14, 67). The large surface area as well as the extensive vasculature of the head allows for rapid heat dissipation. During resting conditions, the head loses heat at a rate that is 8 times faster than the rest of the body (14, 64). Because of the significant contribution to heat dissipation, there is some concern that wearing a helmet can adversely affect thermoregulation to an even greater extent than the impairment encountered by those workers who must have protective clothing covering all, or a significant part of their body (26, 67). There is evidence that helmets can reduce evaporative cooling up to 25-40% (29, 31). Liu & Holmer (47) stated that wearing a helmet in a hot environment causes greater heat discomfort because the lack of air circulating over the head.

Practical Application of Head Temperature Monitoring

An accurate and reliable method for monitoring temperature that is both easily accessible and non-invasive can be of great importance to the occupational medicine, sports medicine, and research communities. Hot Head Technologies, Inc.TM is currently developing a temperature measuring device that will measure forehead temperature continuously over the duration of physical activity while also providing the necessary head protection that is vital to those in high risk environments (athletes, military, fire fighters, construction workers, etc.) The versatility of the product could help to meet the demands of a large group and help to decrease the incidence of heat related illness.

In a previous study, Wickwire (70) found a high correlation, 0.801 ($R^2 = 0.64$, $SEE = 0.25$, $p = 0.00$) between the Hot HeadTM device and core temperature measured using a rectal thermistor. The device was able to predict core temperature more accurately than previous methods (70). Gunga (35) compared rectal temperature, nasopharyngeal temperature and the device under test (Double sensor placed inside a fire fighters helmet) in 3 environmental temperatures. At the highest ambient temperature (40°C), 1660 data points were collected during exercise. With the confidence interval set at $\pm 1.0^\circ\text{C}$, 99.2% of data points were between $\pm 1.0^\circ\text{C}$. With the confidence interval set at $\pm 0.5^\circ\text{C}$, 88.1% of data points were between $\pm 0.5^\circ\text{C}$ (35). This device (double sensor) did not come in direct contact with the subjects skin; instead, it was placed at the vertex of the head on the sagittal suture. Because different individuals have different amounts of hair, having a device that would be in direct contact with the skin may be of greater value and allow for more accurate temperature readings (35).

To avoid serious head trauma, protective headgear must be worn in certain sports and other settings where head trauma is likely. Because the presence of the helmet is mandatory, and

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because of the importance of the head in thermoregulation, a potentially advantageous compromise is to monitor the temperature changes of the head inside the helmet in order to determine when the head has become dangerously hot and simultaneously providing a valid measure of T_c .

Purpose

The primary purpose of this project was to determine the relationship between the in-hardhat temperature readings and core temperature as measured by accepted methodology (rectal and/or esophageal probes). An additional purpose of the proposed project was to establish the relationship between the in-hardhat temperature readings and markers of physiological strain, and between the in-hardhat temperature readings and perceptual responses of the subjects that are indicative of an elevated thermal load.

Hypothesis

As a descriptive study, we hypothesized that the helmet sensors from Hothead Technologies, Inc.Tm would show good agreement with conventional T_c temperature measurements (T_{es} and T_{re}). This would be based on observations of in-hardhat sensor temperatures of less than $\pm 1.0^\circ\text{C}$ different from T_{es} and T_{re} at the termination of the trial.

Significance

Determining the relationship between the protective headgear temperature readings, physiological strain and perceptual responses to physical activity in the heat will enable healthcare professionals to recognize the warning signs of heat illness and potentially prevent

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serious heat related incidences from occurring. Establishing a relationship between core temperature and the temperature detected in protective headgear could will aid in the prevention of heat illness by providing a reliable and accurate method of measuring temperature noninvasively during physical activity.

Brengelmann (9) suggested that a measured index of T_c must: (1) accurately reflect hypothalamic temperature as the hypothalamus is the site of the thermoregulatory controller; (2) respond rapidly and accurately to changes in brain temperature; (3) not be influenced by changes in environmental temperature; and (4) be reliable, easy to use and harmless to the test volunteer (9). The development of a non-invasive device that meets these objectives could be of value in preventing heat injury.

CHAPTER TWO REVIEW OF LITERATURE

Non-invasive temperature monitoring with a sensor mounted inside protective headgear such as a football or military helmet or a construction hardhat may be an effective method of detecting temperatures that are indicative of increased risk of heat illness. The following literature will discuss evidence relevant to this issue and the applicability of previous findings to the current research investigation.

Basic Thermoregulation

The first law of thermodynamics states that energy within a closed system will remain constant. Energy may be converted from one form to another but never created or destroyed. When we exercise, the energy stored as lipids or carbohydrates (potential energy) is converted into kinetic and thermal energy. Most of the energy released from the body's metabolism of nutrients results in heat production. The body is a relatively inefficient system with only 20-25% of energy expended converted to usable work under the best conditions. The remainder of this energy ends up as heat (10, 36). When the rate of heat production is greater than the rate of heat loss, body temperature increases.

Two factors greatly influence heat loss from the human body, the first factor is how rapidly the heat produced in the body can reach the skin and second, how quickly heat can be transferred from the skin to surrounding environment. The 4 methods by which heat is transferred from the skin to the environment are, radiation, conduction, convection and evaporation. In hot environments the dominant method for heat dissipation is the evaporation of sweat (10).

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While external temperatures may vary, core temperature (T_c) must stay within a narrow range to prevent cell damage. In order to maintain resting thermal balance, T_c must fall in the narrow range of $36.7^\circ\text{C} \pm 0.3$. The center responsible for maintaining thermal balance is found in the hypothalamus (9, 10, 28, 36, 38). The posterior hypothalamus receives feedback from receptors in other parts of the body. Thermoreceptors in the skin detect external disturbances, while the preoptic areas of the anterior hypothalamic preoptic areas sense changes in core temperature and enable the posterior hypothalamus (thermoregulatory center) to produce various autonomic control actions to prevent dangerous changes in internal temperature from occurring. The 3 main mechanisms that the thermoregulatory control center uses to decrease body heat are; vasodilation of skin blood vessels, sweating and a decrease in heat production. At the onset of exercise in both thermoneutral and hyperthermic environments, cutaneous vasoconstriction occurs (36). As exercise continues, increases in T_c activate cutaneous vasodilation that continues until it reaches its upper limit at a T_c of approximately 38°C (21). Evaporative cooling, facilitated by sweat production is the primary means of heat dissipation during exercise. Sweat rates ranging from one to three liters an hour have been observed in individuals exercising in the heat. If heat dissipation by evaporative heat loss is limited, but exercise continues, heat production and heat storage also continue, leading to impaired thermoregulatory and physiological function (50). The increasing internal temperature is thought to induce fatigue through both central and peripheral mechanisms (21).

Aerobic training and heat acclimation both enhance heat dissipation by decreasing the core temp threshold for skin vasodilation and sweating. Aerobically trained and heat acclimated individuals have higher sweat rates and higher blood flow to skin. Paradoxically, aerobically trained individuals have greater increases in rectal temperature than untrained ones during

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exercise in the heat (at similar relative intensities) because, the higher the absolute workload produces a higher metabolic heat production (50). During exercise, oxygen consumption in well-trained athletes can increase up to 20 times; thus, the amount of heat produced by the body is directly proportional to the oxygen consumption. If heat dissipation cannot keep pace with metabolic heat production increases, body core temperature continues to rise resulting in a loss of thermal balance and can lead to serious heat illness (36).

Exertional Heat Illness

According to the National Athletic Trainers Association, the traditional categories of exertional heat illness include; heat cramps, heat exhaustion, and heat stroke, however, some important categories are overlooked (6). According to Coris (23) there are five categories of exertional heat illness; heat oedema, heat cramps, heat syncope, heat exhaustion, and heat stroke (14, 23). Regardless of the number of the categories, exertional heat illness can range from slight to severe physiological and cognitive impairment. Minor heat related illness includes heat cramps and heat syncope while serious exertional heat illness includes heat exhaustion and heat stroke (15). Exertional heat illness is most likely to occur in a hot and humid environment; however, it is possible to suffer from exertional heat illness in the absence of a hot or humid environment (6).

Heat oedema occurs when blood pools in the lower extremities, due to peripheral vasodilatation. The oedema can easily be resolved by elevating the lower extremities. Heat oedema is a mild form of exertional heat illness, but if left untreated, the orthostatic pooling can lead to heat syncope (6). During heat syncope, individuals may experience dizziness or a loss of consciousness. As blood pools in the legs, venous return is reduced and cardiac output is

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decreased (6). Lying in the supine position with the feet elevated will help to restore consciousness. Heat cramps can occur during or after an exercise bout and may consist of intense, involuntary muscle contractions.

Heat cramps often result from a negative sodium balance, or if the individual has not yet acclimated to the environment (16, 17, 29). Heat cramps are often treated with stretching, ice, massage and rehydration. Heat exhaustion is the most common exertional heat illness (23). The individual may experience excessive sweating, sodium loss, dehydration, hyperventilation, inadequate cardiac output and confusion along with impaired muscle coordination and judgment (6). During heat exhaustion, core temperature ranges from 38° C to 40.5° C (23). Heat exhaustion leads to a decline in the ability to do physical work (18-20) and can decrease productivity in a work environment. Hypohydration is an important risk factor and predisposes individuals to exertional heat illness or the most serious condition, heat stroke (6, 16).

When an individual suffers from heat stroke, their total thermoregulatory system fails, leading to central nervous system dysfunction (15). Central nervous system dysfunction consists of the loss of the sudomotor drive and in some cases, vasoconstriction. The individual may experience an elevated core temperature of 40.5° C or greater; however, core temperatures may not be above 40° C, or they may be substantially higher. Core temperatures of 42-43° C have been recorded in collapsed athletes (23). Individuals suffering from heat stroke may experience a cessation of sweating, tachycardia, hypotension, hyperventilation or coma (6).

A variety of health conditions, medications, environmental and individual factors can influence an individual's response or tolerance to the heat (14). It is important to note that not all risk factors are present in all populations. There is no single factor that is solely responsible for exertional heat illness, but a combination of many risk factors. A lack of heat acclimation has

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been identified as an important risk factor for exertional heat illness (14). Hydration status is another important risk factor when examining heat related illnesses. Certain medications, alcohol and caffeine can cause dehydration, which impairs the thermoregulatory response and places strain on the cardiovascular system (15, 16, 23). Other risk factors to consider are: barriers to evaporation, (such as extra clothing or equipment that limits heat dissipation, including helmets) illness, history of exertional heat illness, increased BMI, WBGT on previous day/night (5), excessive dark colored clothing or equipment (increases heat absorption,) overzealousness, medications & drugs and electrolyte imbalance (5, 6, 15, 16, 23).

Heat Induced Fatigue

A number of biological mechanisms such as, cardiovascular strain, dehydration and central fatigue contribute to decreased performance and aerobic capacity in the heat. (21, 55). Fatigue may be defined as an “inability to maintain the required force or power” or an increased difficulty in maintaining the work rate, and may be of peripheral or central origin (55).

Physiological Strain

Cardiovascular Responses

Elevations in T_c are associated with significant increases in physiological strain (19). Because of the role that the cardiovascular system plays in supporting sustained exercise and the associated thermoregulatory responses, changes in cardiovascular function are particularly apparent when exercising in a hot and humid environment. As the body exercises in the heat, the supply of blood increases to the metabolically active tissue and cutaneous vasodilation increases to enable heat dissipation (21). As skin temperature (T_{sk}) rises, the perception of effort during

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the exercise also increases (64). Because of the blood redistribution, the cardiovascular system experiences a decrease in venous return with a decrease in stroke volume. Heart rate increases in a compensatory manner in order to keep up with the blood flow demands of the working muscle and the thermoregulatory demands. With elevated sweat rates, dehydration and the associated decreases in blood volume further exacerbate the cardiovascular strain (1, 14, 16).

Ventilatory responses

In mild environments, respiratory responses during exercise increase linearly with oxygen consumption up until approximately 70-85% of $\text{VO}_{2\text{max}}$ (4). Ventilation (VE) is stimulated by the working muscles, body temperature and both central and peripheral chemoreceptors (36, 37). Studies indicate minute VE increases as T_c rises (37). During resting conditions, a 1-1.5 °C elevation in T_c increases VE. During exercise, there is a distinct T_c threshold where VE will increase at a disproportionate rate. These elevations in T_c generate hyperthermia-induced hyperventilation (4). The increase in VE is possibly a thermoregulatory heat loss response. Any additional control of the ventilatory drive is possibly controlled by the preoptic anterior hypothalamus (4).

Dehydration

When environmental heat stress is present and heat gain ensues, the body relies on cutaneous blood flow for heat dissipation via evaporative cooling to restore thermal balance. In a hot and humid environment evaporative cooling is further reduced (21). If fluids are not replenished, dehydration will occur. Dehydration can challenge the limits of the thermoregulatory system by limiting heat dissipation, compromising cardiovascular function and

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reducing exercise capacity. Hypohydration augments hyperthermia and cardiovascular strain in proportion to the magnitude of body water deficit (51). During exercise in the heat, athletes can lose up to 4.5 liters of sweat/hour while most sustainable sweat rates are 1.5L/hour (10, 21). Studies indicate even a moderate 2% reduction in body mass can lead to decreased performance (21). While training increases plasma volume and can increase aerobic capacity and reduce the risk of dehydration, a reduction in plasma volume can decrease aerobic capacity (57). Total body dehydration of 4% resulted in a 16% decrease in VO_{2max} in the heat (8). This reduction in VO_{2max} makes it difficult to sustain steady state exercise, and increases the perception of effort. Continued sweat loss with inadequate fluid replacement results in a decrease in plasma volume. As plasma volume decreases, end diastolic volume and stroke volume are reduced. In order to maintain adequate cardiac output, peripheral blood flow and sweat rate are reduced resulting in greater thermoregulatory impairment. Dehydration can alter muscle blood flow and alter muscle metabolism. Intense exercise is often not sustained for long periods of time in hyperthermic conditions; however, endurance activities lasting longer than one hour can be greatly effected by dehydration (21, 36). Gonzalez (34) also observed the detrimental effects of dehydration on aerobic capacity. Researchers indicated dehydration lead to impaired systemic and skeletal muscle aerobic capacity. Regardless of T_{sk} , hypohydration has a detrimental effect on exercise capacity. When T_{sk} was held constant at 32°C in both hypohydration and euhydration trials. Kenefick (43) observed that hypohydration by 4% body mass impaired performance by 18%.

Tissue Effects/Peripheral Fatigue

Exercise in a hyperthermic environment causes increased muscle glycogen use which leads to increased carbohydrate oxidation (41). If carbohydrates are not ingested during exercise,

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hepatic glucose production increases leading to hyperglycemia; however, if exercise is sustained in a thermally stressful environment, blood is directed away from skeletal muscles and the hepatic gluconeogenic areas to the skin. If cardiac output is compromised, aerobic capacity is reduced (10, 55). Jentjens (41) demonstrated, muscle glycogen utilization was 25% higher during exercise in the heat than in a cool environment. Even though blood flow to the liver was reduced, hepatic glucose production was increased in the heat. Glucose-6-phosphate is elevated due to increased muscle glycogenolysis during exercise in the heat. Even though the rate of exogenous glucose oxidation is reduced in the heat, (about 10% in heat when compared to cold) total carbohydrate oxidation is higher due to an increase in muscle glycogenolysis (41).

Substrate utilization is accelerated during exercise in the heat; this in turn means that substrate depletion is accelerated. During endurance bouts of exercise in the heat, performance may be hindered due to limited fuel availability; however, during bouts of aerobic activity in the heat that are less than 1 hour, it is unlikely that glycogen depletion is the limiting factor (2, 8).

During exercise in the heat, ATP production is decreased (10). Hydrogen ions are released from the degradation of ATP and glycolysis. The accumulation of hydrogen ions reduces muscle pH and elicits a need for hydrogen ion buffering. The bicarbonate buffering reaction generates increased levels of CO₂ in the body, stimulating VE, and leading to an increase in the respiratory exchange ratio (21, 41). Phosphagen depletion during exercise in the heat results in phosphate accumulation, which interferes with glycolysis and restricts calcium from binding to troponin, ultimately inhibiting muscle contractions (8).

Muscle blood flow during strenuous exercise in thermoneutral conditions can increase by 25% (36). Whether or not muscle blood flow is a limiting factor during exercise in the heat is a controversial topic; however, most researchers agree that muscle blood flow is sustained during

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submaximal exercise in the heat (41). Gonzalez (33) demonstrated dehydration lead to decreased blood flow to the leg but did not impair glucose delivery (33). In another investigation, Gonzalez (33) observed no difference between trials (precooling, control, preheating) in blood lactate or blood glucose at exhaustion. Furthermore, blood lactate was low whereas blood glucose was maintained at euglycemic levels in all conditions. They concluded that hyperthermia, rather than altered circulation and metabolism, was the main factor causing fatigue (30, 33, 34).

CNS Fatigue

Researchers agree that both submaximal and maximal exercise capacity in the heat is impaired (21, 25, 55, 63, 68). Over the years there have been numerous theories as to what causes this impaired performance. Nelson (55) concluded that skeletal muscle blood flow did not reach limiting values during exercise in the heat. Muscle glycogen stores were not depleted, and muscle lactate and potassium levels were not elevated to levels that would explain exercise termination due to fatigue. *“Therefore the cause of fatigue during exercise most likely due to a reduction in neural drive from the CNS which leads to a decrease in sustained force production”* (55).

The hypothalamus is known as the temperature control center of the brain (10, 36, 38). In animal studies, it has been observed that a reduction in muscle spindle activity occurs in response to local heating of the hypothalamus (55). Numerous other studies have been conducted to examine electrical activity in the brain in response to local heating (55, 58, 61). Although researchers have found decreased electrical activity in the brain when brain temperature was increased, the topic is far from understood.

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As stated previously, during exercise in the heat, blood flow is redistributed and increased to the skin to allow for greater heat dissipation, while blood flow to the splenic, renal and hepatic regions are reduced (55). Nybo (57) investigated if this reduction in blood flow could also affect the cerebral region. He found that during exercise in the heat cerebral blood flow is reduced causing decreased removal of heat and increased heat storage in the brain (57). While cerebral metabolic rate was actually increased by 7%, lactate production in the brain was not different between thermoneutral and hyperthermic conditions. The area of central fatigue still has much to be discovered; however, it is possible that the reduced motor activity is in response to hyperthermia and protecting the brain from reaching dangerously high temperatures (55).

Perceptual Responses

The physiological strain in a hyperthermic environment, exhibited by the cardiovascular system, can greatly reduce exercise capacity; however, the physiological function may not be the only limiting factor since a combination of perceptual and physiological responses may act as regulators of exercise intensity (25, 64). The rating of perceived exertion (RPE) scale is often used during incremental as well as steady state exercise to determine subject's motivation or feelings of fatigue. While exercising in a hyperthermic environment, perceptions of thermal discomfort and RPE are higher than perceived responses in a mild environment (19).

Furthermore, during exercise in the heat, RPE increases even though the exercise intensity remains constant. Increases in RPE occur in parallel with increasing HR and T_c (57). Pre-cooling may decrease perception of thermal strain as well as decrease the physiological strain index (PSI) (based on T_c and heart rate) during the beginning stages of exercise (20). Individual factors, such as training status, age and hydration, may also influence overall perceived exertion

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(19). Previous studies demonstrate higher aerobic fitness results in improved exercise tolerance regardless of hydration and heat acclimation (19). On the other hand, highly fit individuals may be more susceptible to heat exhaustion because they are able to sustain an increased T_c for a longer period of time and tend to underestimate their estimated physiological strain (19, 20).

The negative effect of perceptual responses on physical performance may be associated with the influence of the central nervous system on fatigue. Specifically, it has been suggested that when critical levels of heat storage are reached, the central nervous system plays an important role in reducing efferent motor responses, thus producing impaired motor function (19).

Researchers concluded that metabolic changes in the muscles are not the primary factor for increased RPE ratings during steady state exercise in the heat; however, high T_c is most likely the major influencing factor (25). Many animal and human studies have found that exercise is voluntarily stopped when reaching a certain internal or brain temperature regardless of the starting internal temperature. If the brain gets too hot it is unable to recruit sufficient number of motor units to sustain power outputs. Other studies continue to illustrate the fact that increased T_{sk} leads to an earlier time to fatigue, or a longer time for a time trial (43). Time to exhaustion was 55% shorter with increased T_{sk} (36-38C); however HR and T_c were similar between low and high T_{sk} (34). During these trials, physical exhaustion occurred in 50% of the subjects at relatively low T_c (<38.5°C), but with high heart rates relative to the exercise intensity. Researchers concluded that hot skin will impair performance and induce exhaustion well below levels associated with a critical T_c (34).

Cognitive Function and Postural Control

The impact of elevated T_c on cognitive function has not been examined extensively; however, clinical and anecdotal observations indicate impaired function in conjunction with exertional heat illness (19). Recent studies tend to focus on physiological responses to heat; however, cognitive performance may be more sensitive to thermal stress than physiological markers (19). Impaired cognitive function could have serious health and safety consequences in the occupational setting. It is difficult to determine the relationship between cognitive function and thermal strain due to the wide variety of methods used to evaluate cognitive function (14, 19). It is important to establish a method that is both scientifically and extrinsically valid. Sharma (65) observed consistent and steady decreases in subjects' psychological scores as dehydration continued. At total body dehydration of 2% and higher, mental function decreased dramatically (65). Caldwell (14) examined the effect of military combat armor on physiological strain and cognitive function. Subjects performed six cognitive function tests during each trial (vigilance, three-term reasoning, filtering, verbal, divided attention and perceptual reaction time). They concluded that the helmet increased thermal and cardiovascular strain but did not impair cognitive function. In addition, they concluded that heat did not impair cognitive function (14). Cognitive deterioration may have been more evident if subjects were suffering from exertional heat illness but it is unethical to induce a state of exertional heat illness on the subject.

Many factors may influence balance including, hyperthermia, hypohydration and fatigue (54); however, few studies have been able to isolate these variables. Hypohydration may impair neuromuscular function, which may lead to postural instability; however, there is a limited amount of literature that examines the effect of T_c on balance (27). The balance error scoring system (BESS) is a reliable method used to measure postural control that includes six positions

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on two different surfaces (27, 60, 62). Distefano (27) observed post-exercise BESS scores were worse than pre BESS scores regardless of condition. Researchers detected diminished postural control (evaluated using BESS) after exercise in a hot environment in both euhydrated and hypohydrated subjects; however, hypohydration alone did not impair balance ability. BESS scores were worse after exercise in a hot environment than after exercise in a thermoneutral environment, leading to the conclusion that hyperthermia can impair balance. Fatigue, regardless of hydration status or hyperthermia impairs balance and postural control immediately after exercise (27).

Head Temperature

Various regions of the body have different levels of sensitivity to temperature sensation. The face displays a greater thermosensitivity than other regions of the body (24). Previous works have repeatedly found that, per unit area of skin, facial temperature exerts the largest peripheral influence on autonomic thermoregulation (42). Nakamura (53) found the face tended to show stronger discomfort during heat exposure than the other sites on the body (chest abdomen thigh).. In hot environments, a warm face and head are associated with thermal discomfort (11, 53). Cooling the head leads to a significant reduction in thermal strain (56). Cotter JD (24) determined cooling the face resulted in 2-5 fold suppression of sweating and thermal discomfort than cooling other sites of the skin. Nunneley (56) demonstrated a significant reduction in thermal strain when the head was cooled. Taylor (67) also demonstrated a suppression of sweat as well as a decreased rating of thermal discomfort from subjects when the face was cooled.

In conditions where helmets are not worn, sweat from the forehead evaporates and increases heat loss; however, according to Hsu (39) protective helmets limit airflow to the head, impair

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evaporation of sweat and can increase the air temperature around the head 2-3°C. Caldwell (14) observed a significant increase in thermal load when wearing a combat military helmet (1.3kg).

The increase in thermal load was not evident when wearing a cloth hat (14).

Methods of Measuring Core Temperature

As previously stated, T_c includes temperatures recorded in the abdominal, thoracic or cranial cavities. The gold standard for “true” T_c is taken from the blood at the intra pulmonary artery (IPA); however, the only way to obtain this measurement is highly invasive and is impractical for the field or laboratory settings (44, 46). Several non invasive methods often used in the clinical setting are used to determine temperature, including; oral sublingual, axilla and tympanic membrane (46).

Sublingual Sublingual temperature fluctuates approximately -0.4 below the IPA temperature and is relatively easy to administer to the general population. This method has the capability to detect changes in body temperature quickly making the sublingual measurement the most popular method in the clinical setting (51). This method is highly influenced by food or beverage intake and temperature and altered greatly by breathing, making this method impractical during or after exercise (51). Axillary measurements take longer to equilibrate compared to oral measurements, cannot be measured continuously and is often much lower than T_c in athletes. Studies reveal axillary temperature and less accurate than rectal, esophageal or tympanic (44, 51).

Tympanic The tympanic method is a non-invasive approach for measuring temperature and has one of the strongest associations with core and brain temperature. The reason for the strong association is because the tympanic membrane receives blood from the internal carotid artery.

Detection of thermal strain

The carotid artery also travels to the hypothalamus, which is the region of the brain that regulates temperature (32); however, the accuracy of the tympanic reading depends on the skill of the technician (44) and infrared scanners used for measuring tympanic temperature may not meet healthcare standards. The tympanic method is unable to measure temperature continuously, making this approach unsuitable to use during exercise (46). There are rising concerns that the tympanic measurements have not been tested on athletes exercising in extreme heat, and are an inaccurate and ineffective method for measuring T_c (17, 44). When compared to IPA, only about 50.9% of tympanic readings reflected true T_c , which could lead to a delay in treatment or unnecessary interventions (46). Casa (17) demonstrated that as rectal temperature increased during exercise, tympanic temperature remained significantly lower at every time point.

Rectal In order to monitor temperature continuously during exercise, more invasive methods such as; rectal, esophageal and gastrointestinal temperatures are most commonly used. The preferred and most common method of measuring T_c in clinical and laboratory settings is by a rectal thermistor (6, 31) inserted to a depth of approximately 12 cm (31). This method is common because it provides a reliable and valid measurement for T_c and is relatively low cost. Rectal temperature can be measured constantly during physical activity. It also remains stable throughout the exercise and is not influenced by ambient temperatures (46). Rectal thermistors are common and helpful in single-subject testing; however, this method would be highly impractical for a large number of people in a work or sports environment. In addition, because of the location of the probe, participants are hesitant to assent to this method of measurement because of psychological factors and the obvious invasion of privacy that is required. There is also a reported lag in response time when compared to measurements such as the esophageal, making recovery temperatures during interval training impossible to track (49).

Detection of thermal strain

Esophageal The esophageal thermistor is inserted through the nasal cavity and drops down to a depth of approximately 25% of the individual's height. The rapid response as well as the location of the thermistor allows the sensor to be placed approximately 0.1-0.2 cm from the IPA making the esophageal measurement the most accurate representation of the true T_c . The esophageal thermistor is the preferred method of assessing temperature if the subjects are willing and able to tolerate the probe (51). If subjects have structural or nasal abnormalities or an excessive gag reflex, the esophageal thermistor can be difficult or impossible to insert (32)

Gastrointestinal Gastrointestinal temperatures have becoming increasingly popular over recent years. A telemetric sensor is ingested 4-8 hours prior to measurement. This device transmits the temperature of the GI tract wirelessly, to an external logger. The GI capsule responds slower to changes than the esophageal method, most notably at the beginning and end of exercise or if the intensity changes, however, the GI method has a faster response time than the rectal thermistor (46). The GI capsule causes no discomfort to the subject and allows T_c to be measured continuously in a mass-participation race in environmental conditions (13). It is difficult to standardize the location of capsule due to the range of gastrointestinal tract motility among individuals. If the capsule remains in the stomach, it will be influenced by food and water intake (46).

Skin Temperature Although T_{sk} is important when calculating heat exchange (conduction, convection, radiation), it is not an accurate representation of T_c . Skin temperature is most often measured using sensors that come in direct contact with the skin; however, the methods used to attach the sensors lack consistency (12). During exercise in a thermally stressful environment, blood flow to the skin increases, causing sensations of warmth, skin wetness, and general thermal discomfort. Schlader (64) demonstrated that T_{sk} is an important determinant for self-

Detection of thermal strain

selected exercise intensity. Elevated T_{sk} increase the perception of heat stress independent of T_c (20, 64). Previous studies suggest that T_{sk} and heart rate are influential in perceived exertion (14, 66).

In a previous study, Wickwire (70) tested a device (Hothead Technologies- the same device tested in the current investigation) that continuously measured forehead temperature during exercise (70). The device consisted of a thermocouple (a proprietary encapsulated temperature sensor) with a wireless radio frequency identification transponder that was small enough to be placed inside a football helmet. The sensor was able to estimate T_c by detecting forehead T_{sk} and using a proprietary algorithm (v1.0.2.) to calculate T_c (70). Researchers found a high correlation, 0.801 ($R^2 = 0.64$, $SEE = 0.25$, $p = 0.00$) between the Hothead device and T_c (measured using rectal thermistor). The device was able to predict T_c more accurately than previous methods (chest temperature, ear canal temperature) in the hyperthermic lab environment during continuous aerobic activity. Future studies should examine different modes of exercise, such as high intensity intervals with rest periods. This may mimic real situations found in football or construction environments. Field studies are also needed to determine if the device can accurately measure T_c when external environmental factors are present.

Gunga (35) compared rectal temperature, nasopharyngeal temperature and a double sensor, a device placed at the vertex of the head on the sagittal suture on the surface of the hair. The device was tested in three environmental temperatures (10°C, 25°C and 40°C). At the highest ambient temperature (40°C) a total of 20 male subjects completed 60 minutes of a work/rest protocol. The exercise intensity corresponded to 25%, 35% & 45% of their VO_{2max} . In order to be considered valid, researchers assumed the difference between the double sensor and rectal temperature should be less than $\pm 1.0^\circ\text{C}$ for ± 2.0 S.D. at all temperatures. At 40°C, 1660 data

Detection of thermal strain

points were collected during exercise. When the confidence interval was set at $\pm 1.0^{\circ}\text{C}$, 99.2% of data points were between these intervals. When the confidence interval was set at $\pm 0.5^{\circ}\text{C}$, 88.1% of data points were between these intervals. Because this device did not come in direct contact with the subject's skin, and because individuals have different amounts of hair, having a device that would be in direct contact with the skin will likely be of value and allow for more accurate temperature readings (35).

CHAPTER THREE METHODS

Subjects

Thirty males with a minimum fitness classification of moderately fit/recreationally active between the ages of 18 and 44 years were selected to participate. Subject characteristics are shown in Table 1. They must have engaged in a minimum of three, 40 to 60-min aerobic exercise sessions per week for at least the preceding three months. Their activity level was verified using an activity questionnaire, and their maximal aerobic capacity was measured for descriptive purposes. The activity status of the subjects ensured that they were fit enough to complete a rigorous exercise session in a thermally stressful environment. A medical history questionnaire was administered to exclude individuals who had contraindications for vigorous exercise. Given the time of year of the testing (Oct-Nov), some of the subjects may have had some residual heat acclimation from the preceding summer months, but none were specifically heat acclimated. Subjects were recruited via word-of-mouth, posted flyers on campus and in the TCU area, and e-mail postings on campus. All subjects signed an informed consent form that had been approved by the Institutional Review Board.

Table 1. Subject Characteristics (mean + SD, n = 30)

Age (yrs)	Height (cm)	Weight (kg)	% Fat	VO ₂ max (mL•Kg ⁻¹ •min ⁻¹)
24.57 ±4.32	180.51 ±7.06	81.06 ±9.35	13.76 ±5.11	46.84 ±7.1

Experimental design

This descriptive study was conducted using a repeated-measures, balanced cross-over design in which all the subjects completed two experimental trials. One was a continuous submaximal exercise (CSE) condition and the second was a series of high intensity 30-second sprints (HIE) with a one-minute rest period between each sprint. Exercise in both conditions was carried out in a hot environment (36° C, 40% RH) and continued until one of the following two criteria was met: the subject voluntarily terminated the session, or the subject's rectal (T_{re}) or esophageal (T_{es}) temperature reached 39.5°C. Temperatures and cardiorespiratory measures were taken throughout and a battery of tests to assess psychomotor responses were conducted before and after exercise in both conditions.

Preliminary Testing

Following completion of the necessary paperwork (medical history, activity questionnaire, and informed consent), each subject completed a test for maximal oxygen uptake (VO_{2max}) on the treadmill. This test involved a series of two and three-minute stages in which the load was systematically increased until the subject reached a point of voluntary termination. During this test, heart rate and respiratory exchange data were collected using an open circuit automated gas analysis system (Parvo Medics, True One, Sandy UT). Test validation criteria included achieving an age-predicted maximal heart rate, a respiratory exchange ratio of 1.1 or greater, and/or a leveling off of VO_2 with increased workload. A seven-site skin-fold assessment was also conducted to determine body composition for descriptive purposes (Jackson and Pollock (40)).

Detection of thermal strain

Subjects performed learning trials on the balance error scoring system (BESS) (62), and a mental function test (MFT) modified from the manual form Letter Memory Test; working memory – forward and reverse) (59) both pre and post VO_2 testing. Introduction to the BESS and MFT was necessary to ensure that learning effects were no longer present on the day of experimental testing. During the BESS, participants balanced for 20 seconds in 6 different positions, which differed based on stance and surface. Participants were instructed to keep their hands on their hips and eyes shut for all 6 positions. Errors were counted if subjects performed any of the following movements; lifted hands from iliac crest, opened eyes, stepped down, stumbled, moved hip more than 30 degrees of flexion or abduction during a single leg stance, lifted the toes or heels off the ground or failed to return to testing position within 5 seconds. The MFT consisted of repeating a list of words of increasing length forward then in reverse while simultaneously pointing to the corresponding picture. Each incorrect response was recorded and when the subject reached a total of three incorrect responses the test was terminated and the number of correct responses was tallied as the final score (with a maximum achievable score of 30 and a minimum score of 0).

The results of the VO_{2max} test were used to establish the general fitness status of the subjects, and to determine the running speeds for the submaximal and supramaximal runs to be used in the experimental testing. During this session, each subject was tested for their tolerance to the esophageal probe. Those who exhibited excessive gagging, had anatomical obstructions that made it difficult to insert the probe, or had other problems with the device were excluded from participation.

Detection of thermal strain

Experimental Testing

Each subject completed the two experimental treatments in a randomly assigned order, separated by a minimum of one week. Both tests were conducted in a 36° C environment with a relative humidity of 40-60%. At least one week after completing the preliminary testing, the subject reported to the laboratory after a minimum of a four-hour fast. Hydration status was standardized by having the subjects consume 500 mL of water either the evening prior to (for early morning trials), or the morning of their trial (for mid-day trials). An initial nude body mass was obtained prior to the placement of temperature probes. A probe for T_{re} was inserted to a depth of 12 cm, and a probe for T_{es} was placed via the nasal passage to a depth corresponding to 25 percent of the subject's height. Skin thermistors (T_{sk}) were placed on the chest, upper arm, thigh, and calf. The temperature probes were monitored using a digital telethermometer (Cole Parmer, City, State). The in-hardhat (T_{ih}) monitoring device (Hothead Technologies, Inc.[™]) was placed inside a standard issue construction site hardhat according to the manufacturer's specifications (ANSI/ISEA Z89.1-2009, type 1, class E). The 2 forehead sensors were mounted on a supportive headband and were in direct contact with the skin. Two Sensors were directly superior to each corrugator supercilii. An additional sensor was placed in the cavity of the helmet, directly superior to the bregma of the skull. Participants wore a Polar heart rate monitor (Polar Electro E600, Finland) around their chest. Subjects wore typical exercise clothing—shorts, t-shirt, and running shoes.

After completing all trial preparations, subjects put on the hardhat with the temperature-sensing device and entered the environmental chamber where they rested for five minutes. After baseline measurements for heart rate, T_{re} , T_{es} , T_{ih} , T_{sk} , BESS, and MFT and perceptual responses (ratings of perceived exertion – RPE using the Borg 20-point scale) (7) and ratings of thermal

Detection of thermal strain

comfort- RTC (The RTC scale spans the numbers 1-9 with 1 corresponding to “very hot, 5 corresponding to “neutral”, and 9 corresponding to “very cold”) were recorded; the subject began the exercise for that day. For the CSE condition, the subjects began the exercise with five minutes of running on the treadmill at 55 percent of VO_{2max} , after which the intensity increased to 65 percent of VO_{2max} for the remainder of the trial. During the exercise, heart rate, temperature, and perceptual responses were recorded every five minutes. In addition, respiratory gas exchange responses were measured in 3-minute segments, every 10 minutes (min 0-3, 10-13, etc.). These were used to calculate relative exercise intensity and energy expenditure, and to assess ventilatory responses. The CSE trial continued until the subject voluntarily terminated the trial, or until the T_c reached $39.5^\circ C$.

For the HIE trial, the subjects also completed a 5-minute warm-up at 55% of VO_{2max} , followed by a series of 30-second sprints at a speed corresponding to 120% of VO_{2max} . After each sprint, the subjects were allowed a one-minute recovery at a walking pace (4.0 mph). Temperature, heart rate, and perceptual responses were recorded at the end of every third sprint, and respiratory exchange responses were measured during sprints one through three, six through nine, etc. The HIE trial also continued until the subject voluntarily terminated the trial, or until the T_c reached $39.5^\circ C$. Upon completion of both trials, the BESS test and the MFT test were administered inside the heat chamber, and a second nude body mass was obtained.

Data Reduction and Statistical Analysis

A physiological strain index was computed using the following formula: $PSI = 5(T_{re\ t} - T_{re0}) / (39.5 - T_{re0})^{-1} + 5(HR_t - HR_0) / (180 - HR_0)^{-1}$ (52). Energy expenditure was determined using standard metabolic calculations based on the respiratory exchange data. Ventilatory (VE)

Detection of thermal strain

responses were expressed as absolute values in $L \cdot \text{min}^{-1}$, and as a $\text{VE}:\text{VO}_2$ ratio. Dehydration, expressed in absolute terms and as a percentage was calculated from pre- and post-exercise body mass.

All data were expressed as the mean and standard deviations. Pearson correlations were conducted to determine possible relationships between temperatures measured using the different detection methods. In addition, a coefficient of variation was conducted to determine the variability of the T_{ih} responses among the 30 subjects at the termination point. Statistical analyses also included a two-factor analysis of variance (ANOVA) with repeated measures to detect differences between the various temperature assessments during the trials (3 x 7- method by time). For the first factor, the three levels consisted of the three detection methods: T_{ih} , T_{re} , and T_{es} . For the second factor, time, the 7 levels corresponded to the number of observations within a trial. A 2 x 2 ANOVA (condition- CSE and HIE x time- pre and post exercise) was used to detect differences for the balance and memory tests. A Newman Keuls *post hoc* analysis was used to isolate the location of differences detected by the ANOVA. An alpha of $p < 0.05$ was accepted for all analyses.

CHAPTER FOUR RESULTS

Thirty males completed all experimental trials. Descriptive data from both the CSE and HIE trial are displayed in Table 2. Based on the methodology employed for termination, the safety cut-offs of 39.5° C T_c using either the T_{es} or T_{re} measurement, or the subject's inability to continue, the number of subjects terminating for each method is varied as indicated.

Table 2. Trial Characteristics (mean + SD)

Trial	Speed (mph)	Duration (min)	Reason for Termination
CSE	5.73 ± 0.82	27.69 ± 4.86	Fatigue - 7 T_{es} (39.5° C) - 20 T_{re} (39.5° C) - 3
HIE	9.85 ± 1.16	26.70 ± 5.94	Fatigue - 17 T_{es} (39.5° C) - 10 T_{re} (39.5° C) - 3

Table 3 contains the pre and post temperature data collected using the T_{ih} , T_{re} , and T_{es} devices along with peak T_{sk} responses. The coefficient of variation (CV) calculated for the final temperatures using each measurement method are also displayed. Also shown in Table 3 are the differences between the T_{ih} compared to the T_{re} and T_{es} responses at the point of termination.

Table 3. Temperature Responses (°C) (mean + SD)

Trial	Pre T_{ih}	Post T_{ih}	Pre T_{es}	Post T_{es}	Pre T_{re}	Post T_{re}	Post T_{sk}	T_{ih} diff from T_{es}	T_{ih} diff from T_{re}
CSE	34.58 ±0.98	38.77 ±0.41	36.70 ±0.29	39.29 ±0.58	37.05 ±0.27	38.90 ±0.49	37.42 ±0.72	-0.56 (n = 27)	-0.15 (n = 29)
CV		1.07%		1.48%		1.25%	1.92%		
HIE	34.71 ±0.80	38.76 ±0.37	36.65 ±0.37	39.19 ±0.57	37.00 ±0.31	38.91 ±0.47	37.30 ±0.78	-0.41 (n = 27)	-0.15 (n=30)
CV		0.96%		1.45%		1.21%	2.10%		

CV = Coefficient of Variation (= SD/Mean x 100)

Detection of thermal strain

Table 4 shows a variety of responses related to the psychophysiological strain encountered in the two trials. These values include the peak perceptual ratings (RPE = rating of perceived exertion- Borg 20-pt scale, and RTC = ratings of thermal comfort), and the physiological strain index (PSI) values calculated based on both the T_{es} and T_{re} (Note: a 10 is considered a maximal PSI response). In addition, the HR drift calculation is displayed, and is based on the peak HR compared to the initial steady state HR measured during the first five minutes of exercise.

Table 4. Perceptual Ratings, PSI, HR and Dehydration Responses (mean \pm SD)

Condition	Peak RPE	Peak RTC	PSI - T_{es}	PSI - T_{re}	Peak HR (bt/min)	HR Drift (bt/min)	% Dehyd.
CSE	17.48	1.15	10.04	9.18	186.53	28.63	-1.58
	± 1.73	± 0.37	± 1.05	± 1.11	± 9.96	± 7.45	± 0.71
HIE	17.53	1.18	9.86	9.29	184.70	18.83	-1.31
	± 1.78	± 0.38	± 1.02	± 0.99	± 19.92	± 11.27	± 0.63

The mean time-course data for T_{ih} , T_{re} , T_{es} , and T_{sk} in the CSE condition and HIE conditions are shown in Figure 1 and 2, respectively. Note that for these figures the number of subjects varies for both conditions after the 20-minute time point due to variations in the duration of the exercise bouts among the 30 subjects. The insert included as part of each figure shows the number of subjects at each time point. For the CSE trial, correlation coefficients of $r = 0.43$ and $r = 0.52$ were determined for the relationship between T_{ih} and T_{es} and between T_{ih} and T_{re} , respectively ($p > 0.05$). For the HIE trial, correlation coefficients of $r = 0.48$ and $r = 0.46$ were determined for the relationship between T_{ih} and T_{es} and between T_{ih} and T_{re} , respectively ($p > 0.05$).

Detection of thermal strain

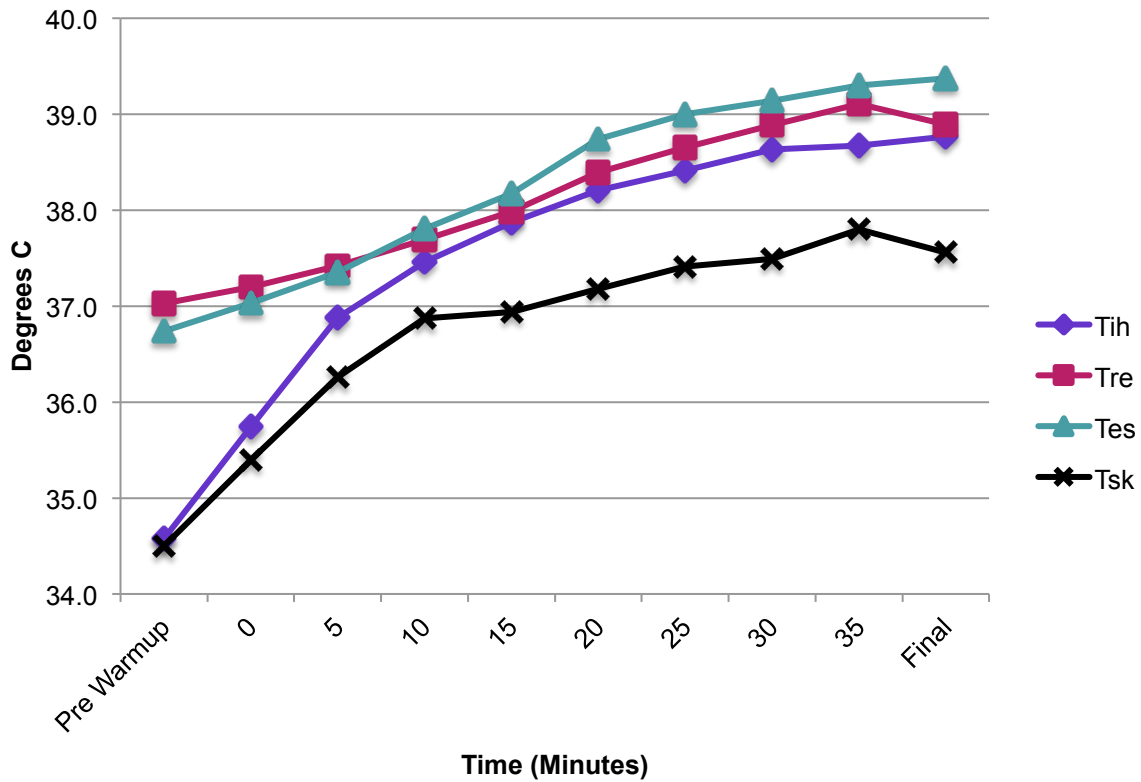


Figure 1. Temperatures recorded in the CSE condition (n = 30).

Number of subjects per time point (min) CSE Trial

Pre Warm-up (Wu)	0	5	10	15	20	25	30	35	Final
30	30	30	30	30	30	20	8	2	30

Detection of thermal strain

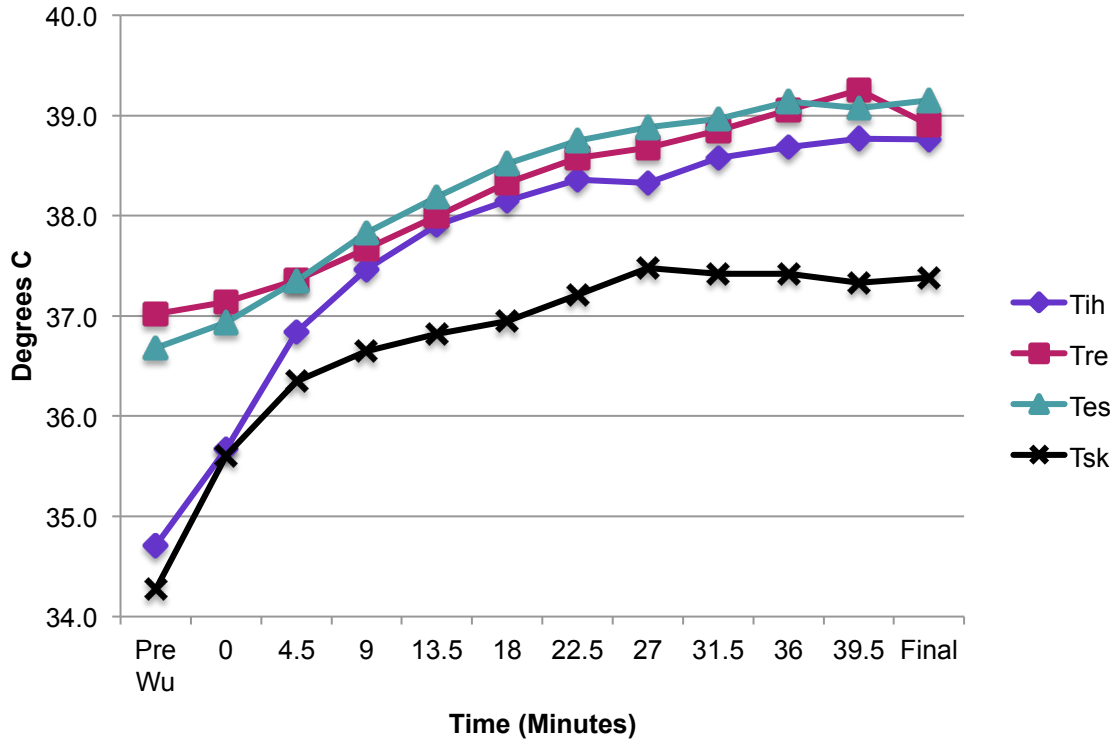


Figure 2. Temperatures recorded in the HIE condition (n = 30)

Number of subjects per time point (min) HIE Trial

Pre Warm-up (Wu)	0	4.5	9	13.5	18	22.5	27	31.5	35	39.5	Final	
30	30	30	30	30	30	30	25	14	6	3	2	30

Detection of thermal strain

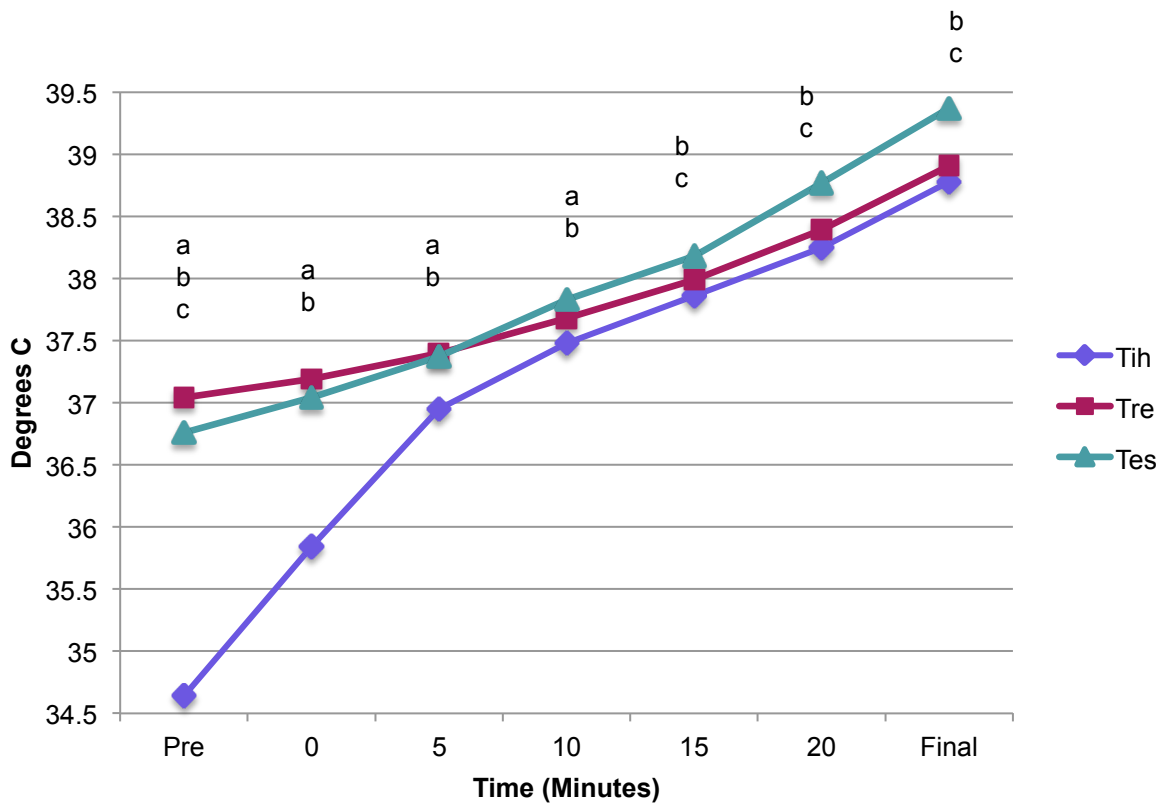


Figure 3. Mean temperatures displaying the results of the ANOVA comparing methods (T_{ih} , T_{es} , T_{re}) across 7 time points ($n = 26$). The “a” indicates a significant difference between T_{ih} and T_{re} , the “b” indicates a significant difference between T_{ih} and T_{es} , and the “c” indicates a significant difference between T_{re} and T_{es}

The results of the ANOVA indicated a significant method by time interaction ($p = 0.001$) for the CSE condition (Figure 3). Post-hoc analyses indicated significant differences at all time points among the various methods as indicated in the figure. Relative to the primary comparison of T_{ih} versus the other methods, at the pre warm-up time point, all methods were significantly different from each other, from 0 to 10 min, T_{ih} was significantly lower than T_{re} and T_{es} , and from 15 min to final, T_{ih} was not significantly lower than T_{es} only.

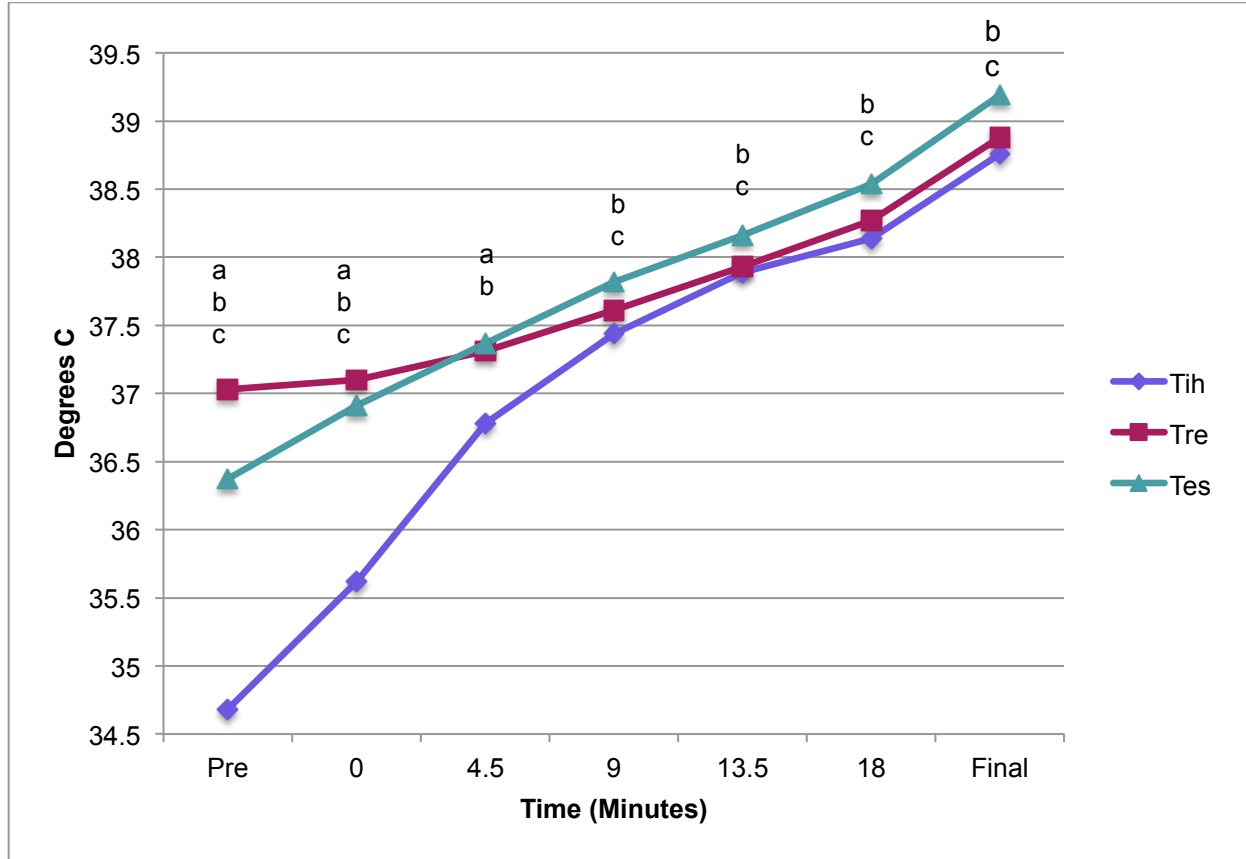


Figure 4. Mean temperatures displaying the results of the ANOVA comparing methods (T_{ih} , T_{es} , T_{re}) across 7 time points ($n = 24$). The “a” indicates a significant difference between T_{ih} and T_{re} , the “b” indicates a significant difference between T_{ih} and T_{es} , and the “c” indicates a significant difference between T_{re} and T_{es}

The results of the ANOVA indicated a significant method by time interaction ($p = 0.001$) within methods for the HIE condition (Figure 4). Post-hoc analyses indicated significant differences at all time points among the various methods as indicated in the figure. Relative to the primary comparison of T_{ih} versus the other methods, from the pre warm-up time point to 0 min, all methods were significantly different from each other, at 4.5 min, T_{ih} was significantly

Detection of thermal strain

lower than T_{re} and T_{es} , from 0 to 10 min, and from 9 min to final, T_{ih} was not significantly lower than T_{es} only.

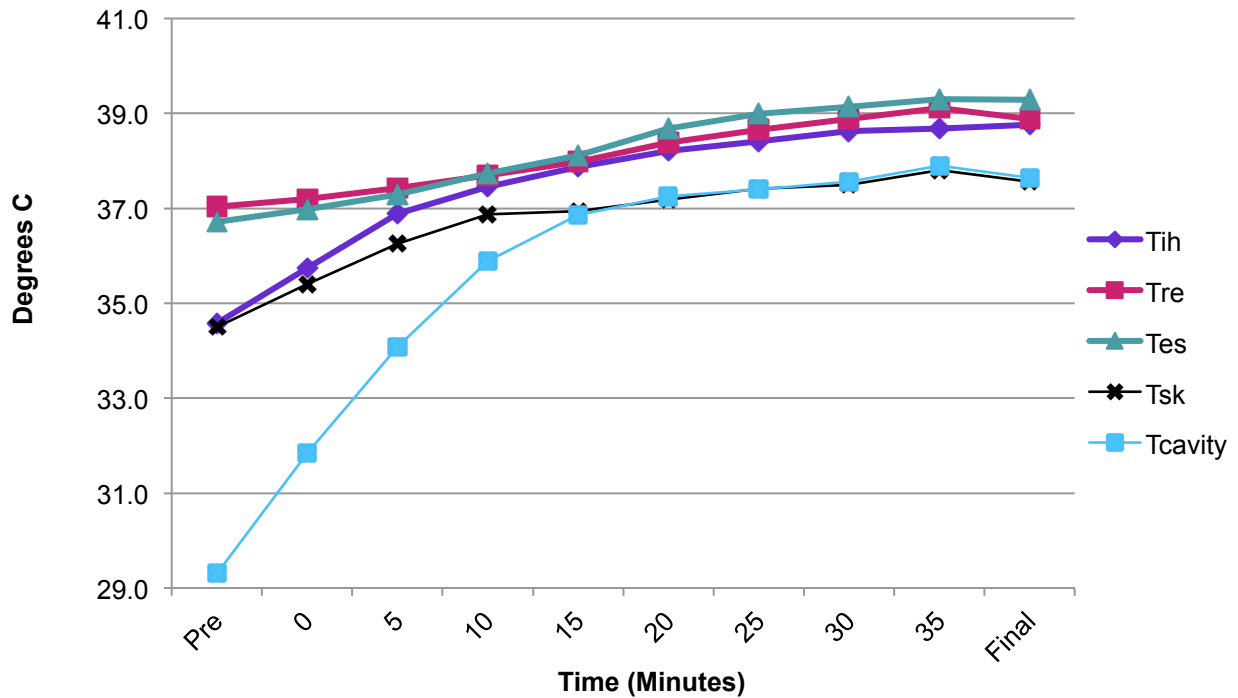


Figure 5. Mean temperature responses for all methods: (T_{ih} , T_{re} , T_{es} , T_{sk} & T_{cavity}).

The mean time-course data for the hardhat temperatures for the CSE and HIE conditions with the cavity temperature added are shown in Figure 5 and 6, respectively.

Detection of thermal strain

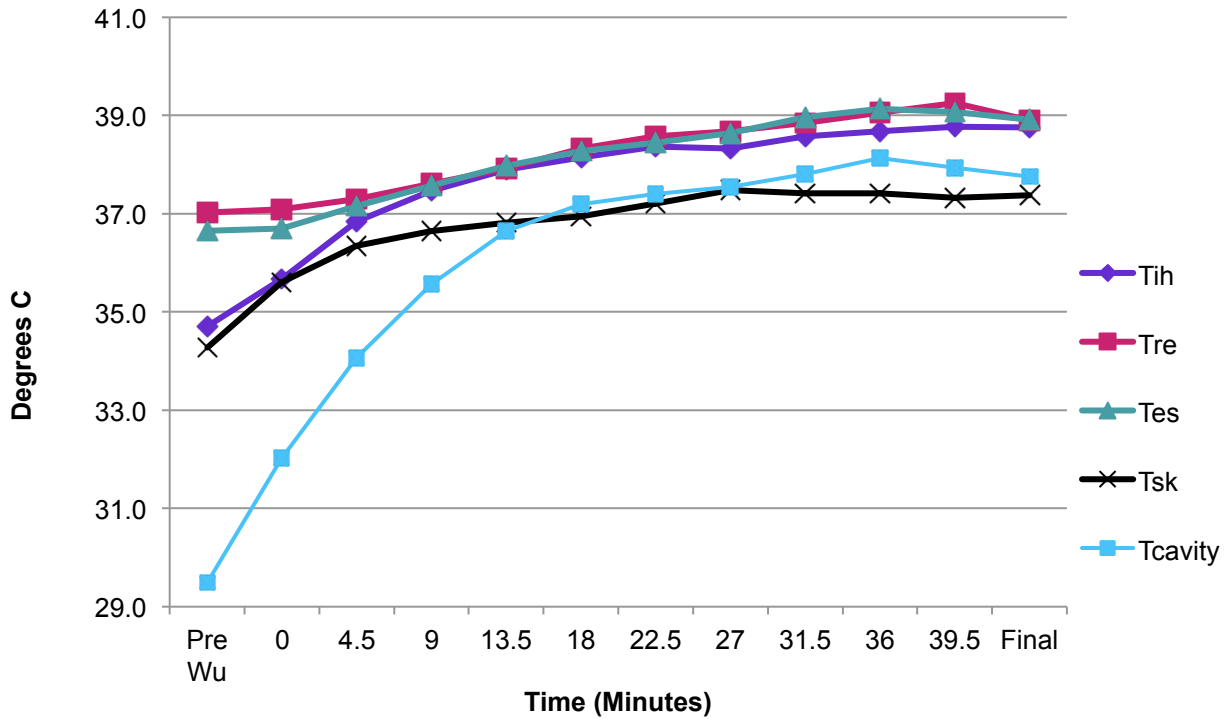


Figure 6. Mean temperature responses for all methods: (T_{ih} , T_{re} , T_{es} , T_{sk} & T_{cavity}).

Balanced Error Scoring System

The results of the postural stability test (BESS = Balanced Error Scoring System) are displayed in Table 5. A 2x2 ANOVA on BESS revealed a significant main effect of time. For both the firm and foam tests, in both the CIE and HIE, the post-exercise responses were higher than pre-exercise.

Table 5. BESS Results (mean + SD)

Condition	Firm Pre	Firm Post	Foam Pre	Foam Post
CIE	2.73 ± 2.41	7.17 ± 3.81*	11.33 ± 4.38	15.5 ± 4.78*
HIE	3.67 ± 2.59	7.37 ± 4.1*	10.8 ± 3.46	15.53 ± 5.55*

Detection of thermal strain

Values represent the number of balance errors during the 20-sec test. The * indicates a significant difference from the pre-exercise value ($p < 0.05$).

Mental Function Test

The results of the mental function test, forward and backward memory, are displayed in Table 6. A 2x2 ANOVA on Mental Function results revealed no significant differences.

Table 6. Mental Function Results (mean + SD)

Condition	Forward Pre	Forward Post	Reverse Pre	Reverse Post
CSE	18.57 ± 7.21	18.57 ± 6.66	14.97 ± 7.74	13.50 ± 7.10
HIE	18.67 ± 7.73	17.33 ± 6.88	13.97 ± 7.85	12.60 ± 7.12

CHAPTER FIVE DISCUSSION

The purpose of the present study was to determine the relationship between the temperature readings obtained inside protective headgear (a construction hard hat) and T_c as measured by the accepted methodology (rectal- T_{re} and/or esophageal - T_{es} probes). An additional purpose of the proposed project was to establish the relationship between the in-hardhat temperature (T_{ih}) readings and markers of physiological strain, and between the T_{ih} readings and perceptual responses of the subjects, all of which are indicative of an elevated thermal load.

The primary finding in this study was that T_{ih} readings maintained a general agreement with both the T_{es} and T_{re} measures during the trials, especially during the latter stages of the trials when temperatures were elevated to the greatest extent. The agreement is quantified by final T_{ih} temperatures that were within approximately 0.50°C of the established methods. In addition, T_{ih} demonstrated consistent readings among the 30 subjects at the point of termination for both the CSE and the HIE conditions, as indicated by the low coefficient of variation in the recordings of the T_{ih} sensor. This consistency suggests that this device may have application as a warning system for impending heat-related problems. Additionally, high T_{ih} temperatures were recorded in the presence of markers of heat-induced physiological stress and indicators of general motor dysfunction, responses that are typically associated with hyperthermia.

T_{ih} Sensor Responses

The data displayed in Figures 1 and 2 demonstrate close tracking of the T_{ih} sensor data with the T_{es} and T_{re} responses, especially after the 5-min time point. The average deviation of the T_{ih}

Detection of thermal strain

sensors from the T_{es} and T_{re} recording ranging from -0.15 to -0.56°C at peak also indicate good agreement between the external and internal sensors. All 30 subjects demonstrated low variability in the peak T_{ih} temperature, as indicated by the low CV calculated for both the CSE and HIE conditions. In fact, the CV for T_{ih} was lower than all other methods (T_{re} , T_{es} , and T_{sk}) at peak. (CV for CSE condition - $T_{ih} = 1.07\%$, $T_{es} = 1.48\%$, $T_{re} = 1.25\%$ and $T_{sk} = 1.92\%$; HIE condition - $T_{ih} = 0.96\%$, $T_{es} = 1.45\%$, $T_{re} = 1.21\%$ and $T_{sk} = 2.10\%$).

Although helpful in suggesting that the T_{ih} sensor was reliable, the CV analysis itself does not provide definitive comparative analysis as to the agreement with T_{es} or T_{re} . Previous researchers have used correlational analyses to show agreement between various T_c detection methods; however, in the current investigation this statistical approach did not provide information that could be used to determine the accuracy of the T_{ih} sensor. The determination of mean differences via the ANOVA provides additional insight into the validity of the T_{ih} methodology worthy of discussion. In examining the differences between T_{ih} , T_{es} and T_{re} , it is first important to address the question of which method should be used as the standard against which other methods can be compared.

It is accepted that T_{es} is a valid representation of T_c , and the rapid response of T_{es} thermistors makes it a preferred method in research settings where rapid heat gain is expected (49). When inserted correctly, the esophageal thermistor has a difference of $\pm 0.1^{\circ}\text{C}$ to 0.2°C of the intrapulmonic artery (IPA) temperature (46). Based on the concept that IPA temperature is the most valid representation of T_c , it could be argued that T_{es} is as close to that temperature as possible in a laboratory setting. During exercise, increased cardiac output and blood flow to the muscles produces a large volume of warm blood returning to the thoracic cavity where the esophageal thermistor is positioned near the heart. Due to the difficulty of insertion, however,

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and the sensitivity to external factors such as air temperature or fluid intake, T_{es} is not a common method in the clinical setting (51). Because of these issues, T_{re} is typically considered the “gold standard” for T_c ; however, even T_{re} has some limitations. Clearly, it is not influenced by the external environment, but the reported lag in response time due to the reduced rate of blood flow to the rectal region and the mass of organs in the abdominal cavity, make it less sensitive to rapid changes in T_c (17, 45, 51). For long duration submaximal exercise, T_{re} is adequate since the rate of heat gain is more gradual; however, for maximal or supramaximal exercise, T_{es} is likely the better choice due to the ability of the probe to detect rapid heat gain. For conditions of rapid heat gain, T_{es} responses have been reported to be higher in comparison to T_{re} (49).

Based on the preceding comparison, it can be seen that the determination of whether T_{es} or T_{re} is the best indicator of T_c is not definitive, and is situational. Because of this, it is reasonable to make comparisons of T_{ih} with either, or both of these conventional methods. Returning to the ANOVA analysis, therefore, the fact that T_{ih} had a rapid rate of response and met T_{re} and was not significantly different from 15 min to the final measurement in the CSE condition, and from 9 min to the final measurement in the HIE condition, suggests that based on T_{re} , T_{ih} can be a valid measure/estimate of T_c . The interpretation based on a comparison with T_{es} is somewhat different since T_{es} and T_{re} themselves were significantly different in both CSE and HIE. As a result, the T_{ih} responses were likewise different from T_{es} and in much closer agreement to T_{re} . We would expect to see a more rapid temperature increase with the T_{es} compared to T_{re} especially in the HIE condition where multiple bouts of high intensity intervals produced brief periods of rapid heat gain. For this condition, it could be concluded that T_{es} is the most accurate representation of true T_c . The presence of statistically different responses among all three temperature detection devices raises the question of which of these devices should be relied upon

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from a temperature detection standpoint. Relative to the application in a clinical or field setting, this has yet to be determined.

In the present study, using the final temperatures for all subjects, in the CSE condition, T_{ih} was different from T_{es} by -0.56°C and from T_{re} by -0.15°C . In the HIE condition, peak T_{ih} was different from T_{es} by -0.41°C and from T_{re} by -0.15°C . In addition to the differences in T_{es} and T_{re} already discussed, from a methodological standpoint, the nature of the T_{ih} measuring device should be considered. Core temperature is heavily influenced by the metabolic heat production that is directly related to the intensity and duration of exercise. Although T_{sk} is also influenced by metabolic heat production, it is also influenced a great deal by both environmental temperature and relative humidity (50). While T_{ih} essentially measured forehead T_{sk} , because the plastic band where the forehead sensors were mounted insulated the sensors against the ambient environment, these sensors did not respond like a skin thermistor. At the onset of exercise, T_{ih} temperatures were lower than the initial T_c and T_{es} readings and were similar to T_{sk} . This response would be expected as these temperature data were recorded prior to the commencement of exercise. As exercise continued, the T_{ih} recordings increased rapidly due to the combination of exposure to the warm environment and the subcutaneous vasodilation in response to heat gain; the latter delivering heat to the skin via blood convection. The fact that the sensors were placed on the forehead, a location with the highest reported T_{sk} (69), helps explain why the T_{ih} sensors provided information that more closely paralleled T_c than T_{sk} . The fact that T_{sk} data were collected from the conventional locations on the trunk and extremities where greater evaporative cooling and air movement generated from the subject while running also helps to explain why T_{ih} responses were higher than T_{sk} . (9).

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As mentioned previously, Brengelmann (9) suggested that a measured index of T_c must: (1) accurately reflect hypothalamic temperature as the hypothalamus is the site of the thermoregulatory controller; (2) must respond rapidly and accurately to changes in brain temperature; (3) must not be influenced by changes in environmental temperature; and (4) should be reliable, easy to use and harmless to the test volunteer (9). In response to these suggestion for acceptable T_c methodology, (9) the current research indicates T_{ih} may have reflected hypothalamic temperate as 2 of the 3 sensors were in direct contact with the forehead and near the superficial temporal artery (44). Although this interpretation must be made with caution, the position of the forehead sensors may have allowed them to detect the temperature of the blood that was also circulating in the hypothalamic region. Relative to the second criterion above, T_{ih} responded rapidly at the onset of exercise (in both conditions). Because of the positioning and insulation effect of the forehead band, the T_{ih} readings appeared to be influenced more by metabolic heat gain, thus remaining relatively unaffected by the external environment throughout the trial compared to the T_{sk} measurements. Although we did not conduct test-retest reliability measures, the two conditions produced similar responses, and the low coefficient of variability suggests good reproducibility. The T_{ih} sensors were easy to use, as they were already positioned on the helmet. T_{ih} measurements updated continuously every 15 seconds and no additional connecting wires were needed as signals were transmitted wirelessly. The fact that T_{ih} had a rapid rate of response and met T_{re} and was not significantly different from 15 min to the final measurement in the CSE condition, and from 9 min to the final measurement in the HIE condition, and caused no additional harm to the test volunteer, suggests that based on T_{re} , T_{ih} can be a valid measure/estimate of T_c .

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Physiological Strain

Physiological strain can be described as the overall demands on an organism imposed by a stressful event. Exercise in combination with a thermally stressful environment can produce maximal strain. Core temperature (T_c & T_{es}), cardiorespiratory responses (heart rate and ventilation), the physiological strain index (PSI), perceptual markers of exertion (RPE), and thermal comfort were used to determine the extent of the exercise and thermal load. An examination of the data in Table 3 and Table 4, and Figures 1 and 2 suggest that many of these responses were near maximal relative to tolerable laboratory limits. Core temperature, specifically in the CSE condition averaged above 39°C. The PSI calculation, an index that takes into account changes in both T_c and heart rate, reached levels that are at the top end of the range the index is designed to handle (an index of approximately 10).

There was a substantial amount of cardiovascular drift in both the CSE and HIE conditions as indicated in table 4. Although the participants exercised at 65% of their VO_{2max} (in the CSE trial), this submaximal exercise elicited a near maximal heart rate. In order to maintain adequate cardiac output, the heart rate must increase in response to the redistribution of blood flow to the skin for heat dissipation, which reduces venous return. The cardiovascular drift seen in the present study was likely due to the redistribution of blood flow to the skin for the purpose of cooling the body along with mild dehydration.

Although the total dehydration was not severe, the percent dehydration recorded was fairly high for total exercise time. As shown in Table 4, the duration of the trials in both conditions was relatively short (25-27 min) compared to many exercise/heat studies. For the population selected and their aerobic capacity, one would think that the subjects would be able to maintain exercise for a longer period of time (34). Gonzalez (34) demonstrated that when

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subject's temperature was slightly above 40°C they reached fatigue regardless of their starting T_c , mean T_{sk} or their rate of heat storage. The reason for termination in the present study varied between trials. While most subjects in the CSE trial terminated due to reaching the T_c cutoff (23 out of 30), most subjects in the HIE trial terminated due to fatigue (20 out of 30). (T_{ih} of 39.08 \pm 0.42 CSE and 39.07 \pm 0.37 HIE; and T_{es} 39.38 \pm 0.37 CSE; 39.15 \pm 0.49 HIE; T_{re} 38.90 \pm 0.49, 38.90 \pm 0.49.) Heat exposure elicited an exaggerated respiratory response. The hyperventilation observed was due to excessive CO_2 driven by elevated temperatures and metabolic acidosis. Metabolic acidosis occurs when muscle pH decreases, therefore, there is a build up of hydrogen ions generating the demand for hydrogen ion buffering. Bicarbonate buffering reactions remove the hydrogen ions. As a result of the bicarbonate buffering reaction, CO_2 accumulates in the body and is expelled via expiration, which is the driving force for inspiration. The build up of hydrogen ion concentration was most likely accelerated during the anaerobic demands of the HIE condition. The T_{ih} measurements increased as physiological responses neared maximal limits, regardless of the condition or reason for termination. Thus, we can determine that subjects with T_{ih} readings comparable to those recorded at peak temperatures will be unable to continue their specified physical activity much longer without nearing exhaustion or reaching dangerous T_c that lead to the increased risk of exertional heat illness.

Helmets and Thermoregulation

Given that the effectiveness of the T_{ih} device is based on its application within protective headgear, from an applied standpoint it is necessary to consider the thermoregulatory implications of helmets in general. Previous work has indicated that the presence of a helmet can interfere with thermoregulation and adversely impact performance (39, 47). In this study,

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the termination times, based on either achieving a critical T_c , or based on voluntary termination were relatively short compared to previous investigations. Although we did not conduct a non-hardhat control condition, it is likely that the heat retention in the head region exacerbated the thermal strain encountered by the subjects, thus causing early termination.

Previous research indicates that ventilation in the helmet itself reduces air temperature around the head and eases thermal discomfort (47). The high hardhat cavity temperatures suggest the retention of considerable heat around the head, a result that is likely due to the lack of ventilation in the hardhat. Previous studies indicate relative humidity has an adverse effect on heat loss via evaporative cooling (47). Liu (47) demonstrated with increased relative humidity there was a reduction in heat loss regardless of the type of helmet (ventilated or non-ventilated). In the present study, relative humidity averaged 40% but reached recorded peaks of 80% during trials. It is probable that the ability to thermoregulate was impeded by the hot microenvironment created within the hardhat, and the lack of ventilation in the helmet along with an increasing relative humidity likely exacerbated thermoregulatory impairment, thus contributing to a rapid rise in T_c and early termination.

Liu and Holmer (47) found solar radiation increased the surface temperature of a plastic helmet shell up to 47°C, while “wind” reduced solar gain and increased heat loss in all helmets tested. While this increase in surface temperature may not increase the forehead temperature sensors to the same extent, higher temperature readings from the cavity helmet sensor may be observed (47). Field studies in which participants suffering from clinically significant heat-related illness while monitored with this device should be conducted.

Another consideration relative to the presence of protective headgear is the added weight

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of the helmet. The protective headgear was a standard lightweight construction helmet with a chinstrap attached to minimize movement. The total weight of the helmet and chinstrap was only 582.9 g; therefore, it is unlikely that the added weight of the helmet contributed to early fatigue/termination of the trial. Regardless of the reason for termination, physiological (T_c , HR, PSI) and perceptual responses indicated that the subjects were experiencing a substantial amount of exercise and heat-induced physiological stress. Regardless of the physiological and thermoregulatory problems associated with wearing a helmet, the benefits of a helmet in protecting against head trauma will always be given greater weight than the other limitations.

Balance and Memory Responses

Although there was no difference in the mental function test, the decreased performance in the BESS responses indicated that the subjects had experienced exercise and heat-induced impairment of motor function. A higher value for the BESS score indicates a greater number of errors indicating poor balance or postural control. Post BESS scores were significantly higher than pre scores. The post BESS scores suggested that the exercise and heat stress were associated with the deterioration of balance/postural control. It is, however, difficult to determine if fatigue or hyperthermia were the primary cause of this weakened postural control. Regardless of the primary factor of causation, with peak T_{ih} temperatures, subjects experienced significant balance/motor deterioration that could warn supervisors that exertional heat illness is in the subsequent future.

Memory

Because of the diversity of methods used to test mental function and the variety of variables that could potentially influence cognitive function such as the degree of hyperthermia,

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percent dehydration, sleep disturbances, hypoglycemic etc., it is difficult to generalize results.

The relationship between the degree of hyperthermia as well as hydration on cognitive performance is an emerging area in which further studies need to be carried out.

The values for the mental function test correspond to the number of correct responses given before a total of three errors were made within one test session (forward or reverse).

Exercise, especially in hyperthermic conditions can cause mild to severe dehydration. Previous studies indicate total body dehydration can lead to decreased mental function (65). Cian (22) observed a 2.8% reduction in body mass impaired cognitive abilities (perceptive discrimination and short-term memory). Although subjects in the present study were under a great amount of physiological stress, the exercise duration, as previously stated, was relatively short and total body dehydration was below 2%. Sharma (65) observed a steady decline of mental function when total body dehydration reached 2% and above. While subjects were not allowed to consume water during the exercise session, as this would affect the esophageal thermistor readings, subjects were instructed to consume 500 ml of water 4 hours prior to the exercise session to ensure adequate hydration. Mean percent dehydration was only -1.58 ± 0.71 and -1.31 ± 0.63 for the CSE and HIE studies, respectively. Thus, percent dehydration was likely not great enough to elicit cognitive deterioration. McMorris (48) determined heat stress had no effect on verbal and spatial recall, and choice reaction time tasks. Because of the safety T_c cut offs in place and ethical limitations, we could not induce heat illness in our subjects. Certainly in the sport or occupational setting, T_c may reach levels much greater than those tested in the lab. If an individual was, in fact, suffering from heat exhaustion or heat stroke, they would likely have impaired mental function. Caldwell (14) examined the effect of military combat armor on physiological strain and cognitive function. They concluded that the helmet increased thermal

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and cardiovascular strain, but did not decrease cognitive function, a finding that is in agreement with the current results. It is possible that with increased thermal load and or increased metabolic strain, cognitive function may be altered to a greater extent.

Perceptual Responses

The participants experienced a substantial amount of perceptual discomfort as indicated by the thermal comfort ratings of near 1.0 and the RPE of over 17 (based on the Borg scale). Facial skin temperature and perceptions of facial temperature have the largest peripheral influence on autonomic thermoregulation (42, 43, 56). Because the skin generates a great deal of perceptual information, it has a substantial influence on thermal comfort even in the absence of T_c fluctuations (53). In the present study, ratings of thermal comfort were near maximal limits at peak temperatures. Studies demonstrate whole-body thermal discomfort, in the head, corresponds with the perceived comfort in the head region (42, 43). The addition of the helmet likely exacerbated feelings of thermal discomfort by inhibiting heat dissipation and evaporation, accelerating heat gain and creating an even hotter microenvironment around the head.

Practical Implications and Conclusion

In conclusion, due to the necessity of the hardhat in specific occupational settings, it would be advantageous to monitor temperature in a protective device that is always going to be mandatory. Because protective headgear of all kinds impairs thermoregulation and may lead to a more rapid gain in T_c , thus contributing to the earlier onset of exertional heat illness, the ability to continually monitor temperature changes of the head, while inside the helmet, by a non-invasive method is even more essential in determining when the head has become dangerously

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hot and may induce severe heat related illnesses. The fact that the T_{ih} reading closely tracked acceptable and valid measurements of T_c (T_{re} and T_{es}) and that high T_{ih} data were recorded in the presence of significant postural deterioration as well as physiological strain indicates that the T_{ih} device may be a pragmatic approach to combatting exertional heat illness in the occupational setting.

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ABSTRACT

A Comparison of Temperatures Inside Protective Headgear With Indicators of Physiological Strain and Core Temperatures During Exercise in a Hot Environment

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Introduction: Non-invasive temperature monitoring with a sensor mounted inside protective headgear such as a football or military helmet or a construction hardhat may be an effective method of detecting temperatures that are indicative of increased risk of heat illness. Hothead Technologies has developed a system for providing temperature information to medical personnel responsible for the health of the individual. **Purpose:** The purposes of this study were to establish the relationship between in-hardhat temperature (T_{ih}) readings, markers of physiological strain and perceptual responses, and to determine the differences between the in-helmet temperature readings and core temperature (T_c) as measured by rectal (T_{re}) and esophageal (T_{es}) probes. **Methods:** Thirty males (age, 24.57 ± 4.32 years; height 180.51 ± 7.06 cm; body mass, 81.06 ± 9.35 kg; percent body fat, 13.76 ± 5.11 ; VO_{2max} , 46.84 ± 7.10 mL/Kg/min) completed two experimental trials separated by a minimum of one week: a continuous submaximal exercise (CSE) condition and a series of high intensity 30-second sprints (HIE) with a one-minute rest period between each sprint. Exercise in both conditions was carried out in a 36° C environment with a relative humidity of 40% while wearing a standard construction hardhat with a sensor mounted in the forehead area of the head strap, and continued until one of the following two criteria was met: the subject voluntarily terminated the session, or the subject's T_c reached 39.5° C. Temperatures, heart rate, cardiorespiratory, and perceptual responses were monitored throughout, and balance error scoring system (BESS) and mental function tests were conducted before and after exercise. A physiological strain index (PSI) was calculated from T_c and HR.

Results:

Table 1a. Temperature Responses ($^{\circ}$ C), BESS results and PSI (mean + SD)

Condition	Post T_{ih}	Post T_{re}	Post T_{es}	BESS Firm Pre	BESS Firm Post	BESS Foam Pre	BESS Foam Post	PSI T_{re}	PSI T_{es}
CSE	38.77 ± 0.41	38.90 ± 0.49	39.29 ± 0.58	2.73 ± 2.41	7.17 ± 3.81	11.33 ± 4.38	15.50 ± 4.78	9.18 ± 1.11	10.04 ± 1.05
CV	1.07%	1.25%	1.48%						
HIE	38.76 ± 0.37	38.91 ± 0.47	39.19 ± 0.57	3.67 ± 2.59	7.37 ± 4.10	10.80 ± 3.46	15.53 ± 5.55	9.29 ± 0.99	9.86 ± 1.02
CV	0.96%	1.21%	1.45%						

CV = Coefficient of Variation (= SD/Mean x 100)

Conclusion: The general agreement between the T_{ih} and other temperature measures along with the consistency as indicated by the low coefficient of variation in the recordings of the T_{ih} sensors at the point of termination suggest that this device may have application as a warning system for impending heat-related problems.

Detection of thermal strain

Kelly James

Objective Statement: To gain experience in the field of exercise physiology via career in cardiopulmonary rehabilitation.

Education

Texas Christian University, Fort Worth, TX

Master of Science in Kinesiology - Exercise Physiology, May 2013

Baylor University, Waco, TX

Bachelor of Science in Exercise Physiology, May 2011, Overall GPA: 3.5

- Dean's list: Fall 2007, Spring 2010, Fall 2010

High School – Southwest Christian Academy, Houston, TX, Diploma Spring 2007

- Salutatorian

Internship Experience

Gettnerman Wellness Center, Waco, TX, - Summer 2010

Intern for cardiopulmonary rehabilitation clinic

- Aided with risk assessment, exercise testing, education classes, chart work

Work Experience

Baylor All Saints Medical Center, Fort Worth, TX, March 2013 – Present

Exercise Physiologist for cardiopulmonary rehabilitation

- Monitor cardiac and pulmonary patient's responses to exercise, exercise prescription, group classes, medical chart work

TCU Graduate Assistant, Fort Worth, TX, August 2011-May 2013

Instructor, Research Assistant and Teaching Assistant

- Weight training instructor for undergraduate classes
- Assisted with projects in the physiology lab
- Teaching Assistant: Exercise physiology lab instructor and personal fitness TA

Cindy Hauss Fitness, Fort Worth, TX, Summer 2012

Personal assistant

- Updated web page, scheduled CPR classes, business promotion

LA Fitness, Dallas, TX, Summer 2011

Front desk

- Membership sign in, taking personal training appointments, billing information

Biron Elite Cheer, Houston, TX, Summer 2009

Cheerleading Coach

- Coached competitive and school age teams, private lessons

Perry's Steakhouse and Grille, Houston, TX, Summer 2008

Hostess

- Planning Private Parties, taking reservations, escorting guests, clearing tables

Detection of thermal strain

Biron Gymnastics, Houston, TX, Summer 2006

Gymnastics Coach

- Coached school age children

Volunteer Experience

Baylor Buddies - 2009-2010

- Mentoring 2nd grade student once a week

Camp Periwinkle - Summer 2008

- Summer camp counselor for children with cancer

Texas Children's Hospital - Summers 2005, 2008 & 2009

- Making A Mark art show
- Volunteer on children's cancer floor

Ronald McDonald House - 1999-2007

- Performed and taught dance/gymnastics routines

Activities

Baylor Club Gymnastics - 2007-2011

- President - 2010-2011
- Vice President - 2009-2010

Kappa Alpha Theta - 2008-2011

- Social Committee, Education Committee, CASA Philanthropy

Biron Gymnastics Show Team -1998-2007

- World Gymnaestrada participant: 1999, 2003 and 2007, Team Gymn Champions, GAT Pride of Texas award - 2006

National Champion CCA - 2005

- 1st place for individual
- 1st place team

Certifications

ACLS

AHA - CPR/AED

American Council on Exercise - Personal Trainer