EFFECTS OF CHANGES IN PLASMA VOLUME, OSMOLALITY AND SODIUM LEVELS ON CORE TEMPERATURE DURING PROLONGED EXERCISE IN HEAT

by

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(ABSTRACT)

Six adult males of similar body composition and aerobic capacity were tested to study the effects of changes in plasma volume (PV), osmolality (OSM) and sodium (Na^+) on core temperature (Tc) under three exercise-thermoregulatory stress conditions. The protocol consisted of 120 min of upright stationary cycling at 50% $\dot{V}O_2$max under neutral (24° C, 50% RH) - euhydrated (NE), hot (35° C, 50% RH) - euhydrated (HE), and hot-hypohydrated (HH) environmental conditions. Venous blood samples were obtained at -30 min, 0 min and at 15 min intervals through a 30 min recovery and were analyzed for blood hematocrit and hemoglobin, and for plasma osmolality and sodium. Hematocrit and hemoglobin were used to calculate relative changes in plasma volume. Tc showed qualitatively similar linear increases in the first 45 min of each trial. At 60 min, Tc in the NE trial

plateaued at 37.9°C. In the HE trial, Tc continued to show a slight further increase after 45 min while in NE it became significantly ($p<0.05$) lower at 45 min as compared to HE and HH; at 60 min of exercise, the core temperature of all three trials differed significantly ($p<0.05$), with HH being the highest (38.3°C). Percent change in plasma volume was not different between trials, but did show the greatest decrease in all trials from 0 to 15 min of the exercise phase with at least -4.3%. Osmolality was significantly different ($p<0.05$) between the NE (X = 283.3 mOsmol/kg) and the HH (X = 292.5 mOsmol/kg). Plasma sodium was significantly ($p<0.05$) higher for all intervals of HH (X = 137.9 meq/L) as compared to the NE (X = 135.1 meq/L) and HE (X = 134.8 meq/L). These data suggest that core temperature (Tc) increase in moderate intensity endurance exercise is less related to a decreased circulating plasma volume, but is more strongly associated with rising osmolality, specifically the increase in the Na^+ electrolyte, which occur with progressive hypohydration.

Index terms: core temperature; plasma volume; osmolality; sodium; prolonged exercise thermoregulation; euhydration; hypohydration.

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whose love and support gave me the strength to complete this work. She is an inspiration and her example has been a guide throughout my life. I only hope that some day I'll be able to make her as proud of me as I am of her.

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Chapter I

INTRODUCTION

Exertional heat stroke is a clinical problem that results from prolonged physical exercise under various environmental conditions. It is a most prevalent problem when ambient temperature and relative humidity are high. The human body must dissipate heat even while resting in a hot environment, but the additional heat production of exercise compounds the situation. During intensive exercise, such as marathon running, heat is produced in the contracting muscles at rates in excess of 1100 W, a rate which is 15 to 18 times greater than the basal metabolic rate (Nadel, Wenger, Roberts, Stolwijk, & Cafarelli, 1977). Therefore, running a marathon in a hot environment with a high relative humidity will compromise both performance and health, even for the elite athlete, if adjustments in pacing and fluid intake are not made. An increase in environmental humidity reduces the vapor pressure gradient between the skin and air, and thus, diminishes the evaporative process. Even when the external heat load is moderate, strenuous physical effort can contribute to the pathophysiology of exertional heat stroke (Hubbard, 1979).

In 1980 more than 1,000 deaths in the United States alone were attributed to the heat (Abromowicz, 1980).

Everyone is a potential victim of a heat related injury. This includes well trained athletes to novices, young and old, and physically fit to the seriously ill. However, the individuals that are most susceptible to heat injuries are the young and old and the seriously ill or least fit. Relatively high proportions of heat stroke cases are being reported during sporting events, particularly those which last an hour or more, in warm and humid weather. Trained athletes are not exempt; examples include a Tour de France cyclist (Bernheim & Cox, 1960), 3 Danish cyclists at the summer Olympics in Rome 1960 (Shibolet, Coll, Gilat, & Soher, 1967), and marathon runners (Pugh, Corbett, & Johnson, 1967; Wyndham, 1977). The most vulnerable participants in a long distance run are the novices (Hughson, Green, Houston, Thomson, MacLean, & Sutton, 1980).

Among those who compete in road races of 10K or longer distances, a small number of novices will generally be found collapsed at the finish line on warm days due to heat stress. Novice runners have a tendency to push themselves beyond their capacities to attain personal records. Novices overestimate their abilities when the day is warm, the sun is bright (high radiant heat) and they feel great. They can push for that personal record, ignoring their exercise limitations and water replacement. Novice runners will

compromise their training and begin the race at a much faster pace than they can maintain without experiencing heat storage. Then they try to hold a pace seconds, even a minute, faster than they are accustomed to running. Trying to hold this pace, they will not stop to gain water replacement and will not foresee the early warning signs of heat strain.

Other related literature on heat injuries includes cases involving football players (Graber, Reinhold, & Breman, 1971), industrial workers (Wyndham, 1965), and recruits in the armed forces (O'Donnell, 1975). There has been increasing evidence that in healthy young males, an excessive exercise intensity is the essential factor in the development of exertional heat stroke; the individuals are forced to complete a task in a hot environment for a prolonged period of time and usually without proper fluid replacement.

In summary, there are four main factors that can affect the possibility of a heat stroke. They include the environmental aspects such as ambient temperature, humidity, wind velocity, insulation and clothing; water intake; the intensity and duration of the exercise; and the recent acclimatization to heat. It becomes evident, when examining these factors, that even a slight variation in the stress

conditions could result in an unexpected response, leading to widespread functional and tissue damage or even death to the individual.

Statement of the Problem

When man is subjected to moderate-vigorous prolonged physical exercise in a warm environment his core temperature (Tc) rises progressively over time; this rise can possibly lead to exertional heat exhaustion and/or heat stroke. The precise mechanism for this rise in core temperature is still unclear. Thus, the primary problem seems to relate to understanding the stimuli which lead to ineffective control of the temperature regulatory centers. Could this center (hypothalamus) be impaired by declining circulating blood volume, rising body fluid osmolarity or hypernatremia (high Na^+)? Therefore, the objective of this study was to investigate the role of selected hematologic factors in the rise of the core temperature.

Research Hypotheses

1. Ho: There will be no time related changes in core temperature, or in plasma volume, osmolality, and sodium during a prolonged cycling exercise of moderate intensity in three different thermogenic conditions.
2. Ho: Core temperature, plasma volume, osmolality and sodium patterns in prolonged exercise do not

significantly differ between the aforementioned treatment conditions.

3. Ho: If there are any changes in plasma volume, osmolality, or sodium under these different thermal conditions, these do not influence the rate of rise in core temperature.

Delimitations

The following delimitations were imposed:

1. only male subjects with less than 20% body fat, who were moderately active and possessed maximal oxygen uptakes between 40-60 $ml \cdot kg^{-1} \cdot min^{-1}$, were studied;
2. a 2 h cycling bout at 50% $\dot{V}O_2max$ served as the criterion exercise;
3. only two environmental conditions were imposed, i.e., 24° C, 50% RH vs 35°C, 50% RH.

Limitations

Due to the foregoing delimitations, the following major limitations apply to this study:

1. these results may be applicable only to males possessing the same physiological characteristics as the subjects in the study;
2. these results may only be applicable to individuals involved in stationary leg cycling at of moderate intensity (50% $\dot{V}O_2max$);

3. these results may not be applicable to environmental chamber conditions used beyond the temperature and humidity ranges imposed in this study;
4. the sample size was limited to six volunteers.

Definitions

1. Core Temperature (Tc): Deep body temperature obtained by esophageal, rectal or tympanium measurements.
2. Euhydration: The process of replacing body water losses and maintaining a water balance within the body.
3. Hyperhydration: A state of excessive water content in the body due to ingestion of "extra" water.
4. Hypohydration: A state of decreased water content of the body. A greater loss of water than salt.
5. Maximal Oxygen Uptake ($\dot{V}O_2max$): The maximal amount of oxygen that can be consumed in dynamic, large-muscle activity, usually expressed in liters$\cdot$min ^{1} or ml$\cdot$kg $^{-1}\cdot$min $^{-1}$.
6. Osmolality: osmotic concentration, defined as the number of osmols of solute per liter of solution.
7. Plasma Volume (PV): The total non-cellular fluid volume of the circulating blood found in the intravascular space.
10. Rectal Temperature (Tr): The temperature measured 15-17 cm into the rectum. An acceptable means of estimating core temperature.

Chapter II

REVIEW OF LITERATURE

Heat Injuries and Prevention in Prolonged Exercise

When the body is unable to adapt to a heat stress condition, three major clinical disorders might present themselves: heat cramps, heat exhaustion, and heat stroke. Although these conditions are all mediated by a loss of body water and/or electrolytes, coupled with an inability to dissipate heat, the etiologies and clinical significance are somewhat different with each disorder.

Heat Cramps. Heat cramps are the least severe of the heat disorders and are common, even in well-acclimatized athletes who are in good physical condition. The main sign is an acute, intermittent, painful cramping in skeletal muscles, generally in the calf or abdominal area. The exact cause is unknown, but it is speculated to be prolonged, excessive sweating, which results in a loss of body fluids and/or local electrolyte imbalances, mainly sodium chloride, potassium, and magnesium (Cain, 1985). In a case of heat cramps no abnormal signs or symptoms will appear and the cramps will disappear within a few minutes, spontaneously. The treatment for this disorder would be to rest in a cool place and replace lost fluids. To prevent heat cramps, it is important to insure adequate hydration and increase the

consumption of foods rich in these electrolyes, but without the salt tablets due to the electrolyte imbalance produced (Dasler, 1973).

Heat Exhaustion. Heat exhaustion, or heat prostration, is the most common heat disorder. It results from prolonged exposure in high environmental temperature and humidity, which causes an excessive water loss in the body. This disorder occurs mainly in unacclimatized individuals. Fatalities are rare, but when such do occur, a predisposing constitutional disorder has generally been involved. Depending on the severity of the water loss, a wide range of symptoms may be presented including fatigue, excessive thirst, anxiety, weakness, and impaired judgment. These symptoms may lead to central nervous system impairments manifested by hyperventilation, muscle incoordination, agitation and hysteria (Feinstein, 1985). The clinical signs are a rectal temperature of approximately 40°C, dehydration, and a reduced ability to sweat in some cases. The treatment for heat exhaustion is to move the individual to a cooler and less humid surrounding, give fluids if the victim is able to swallow; if voluntary water consumption is not possible, intravenous fluids of isotonic saline should be started, and a blood chemistry and urinalysis performed. If recovery is slow, it is important to evaluate the victim

and treat for any underlying factors or secondary complications (Cain, 1985).

Heat Stroke. Heat stroke is the least common but the most serious of the heat disorders. Heat strokes alone in the last 10 years have killed 50 football players, and in order of prevalence, heat stroke ranks second after spinal injuries among reported cases of death in high school athletes (Knochel, 1975). Heat strokes are preventable and to allow their continuous occurrence in sports events is inexcusable. Obviously, a preventive approach is better than emergency treatment, but for prevention to be effective, there must be a clear understanding of both the circumstances that lead to the disorder and its pathophysiology.

Healthy, highly acclimatized, and physically fit athletes can suffer a fatal heat stroke when the body's heat load exceeds its ability to dissipate heat. This failure is a result of a breakdown of the normal sweating and thermoregulatory mechanisms and leads to mental confusion, anhidrosis, and a rectal temperature in excess of 41°C (108.8°F). The rectal temperature is the most important part of a triage. It helps distinguish between heat stroke and heat exhaustion and indicates the severity of the situation. Heat stroke is invariably fatal unless

recognized early and treated accurately. All untreated victims of heat stroke will die (Cain, 1985).

Even with rapid treatment, the mortality rate can be as high as 50%, especially in the elderly with predisposing factors such as heart disease, obesity, heavy-alcohol ingestion, diabetes mellitus, previous heat injuries, and those taking certain medications (diuretics, tricyclic antidepressants, anticholinergics, phenothiazines, amphetamines, and lithium). The normal circulatory adjustments for the elderly person or individuals with the aforementioned predisposing factors or secondary complications is to compensate for heat stress by decreasing the cardiac output ($\dot{Q}$) and increasing their peripheral resistance. However, when the environmental conditions cause an excess of heat in the body, a tachycardia and decreased peripheral resistance will cause peripheral pooling and a hypovolemic condition will then exist which eventually will lead to circulatory failure (Khogali, 1983).

"Exertional" heat stroke occurs in athletes, military recruits, and industrial workers who perform manual labor in hot, humid environments. The circulatory adjustments that are required to dissipate heat loads in healthy individuals are an increase in heart rate, an increased cardiac output ($\dot{Q}$), and a decrease in peripheral resistance (O'Donnell &

Clowes, 1972). With an exertional heat stroke, there will be, in most cases, profuse sweating, but as the temperature of the body increases rapidly above 41°C sweating will eventually cease due to probable heat damage to the sweat glands causing anhidrosis (Shibolet, Lancaster, & Danon, 1976). Early warning signs of both environmental and "exertional" heat stroke are headache or throbbing sensations in the temples, nausea, vomiting, dizziness, piloerection (gooseflesh) of the upper chest and arms, unsteady gait, and disorientation with gradual impairment of consciousness (Hubbard, 1979).

With the excessive heat load that occurs in heat stroke, eventual widespread cellular damage may begin to occur in the vital organs and skeletal muscles. Central nervous system complications can include a coma, convulsions or even a cerebrovascular accident. Heart failure due to sinus tachycardia, and an accompanying hypotension or myocardial infarction may also occur in the heart (Kew, Tucker, & Bersohn, 1969).

During "exertional" heat stroke, serum glutamicoxaloacetic transaminase (SGOT) and lactate dehydrogenase (LDH) are grossly elevated, and hyperbilirubinemia may occur arising from liver damage (Kew, Bersohn, & Kent, 1970). Impaired coagulation may occur in

severe cases with complications of thrombocytopenia, prolonged bleeding and clotting, lowered plasma fibrinogen and prothrombin levels and increased fibrinolytic enzyme activity (Hart, Egier, Shimizu, Tandon, & Sutton, 1980).

Kidney problems may arise secondary to heat stroke, including conditions ranging from acute renal failure to chronic nephritis (Kew, Abrahams, & Levine, 1967). Myoglobinuria may also be present, suggesting muscle cell damage (rhabdomyolysis) and renal insufficiency. If physical activity continues under conditions of impending or evolving heat stroke, so will the release of certain time enzymes SGOT, LDH, and creatine phosphate kinase (CPK) occur into the circulatory system from vital organs, indicating thermal injury (Hart et al., 1980; Hubbard, 1979). Myoglobinuria, excessively high serum CPK and uric acid levels from the skeletal muscle will complicate renal function leading to possible renal failure, following the acute period (Hart et al., 1980).

The treatment of heat stroke requires trained personnel to immediately recognize imminent warning signs, and if these are present, procedures must be started promptly to effect cooling, rehydrate and correct circulatory collapse.

When the rectal temperature is measured at 41°C or above, cooling strategies must begin immediately. The old

method of cooling was to remove all the clothing of the victim and place him in an ice bath. This method proved to be difficult if the victim was disoriented and combative. It was also dangerous since it was very difficult to control the extent of fall in the rectal temperature. If the body is cooled below 38.9°C (102°F), it is hard to control body temperature and the victim become hypothermic. Thus, circulatory shock and death might subsequently occur during the rewarming phase. Ice baths can also cause peripheral vasoconstriction which interferes with proper heat loss (Abromowicz, 1980).

The most effective method of cooling the body is via ice water sponge baths and application of ice packs to the abdomen, axilla, neck and groin regions. An alternative is to place the victim in a shaded area or an air conditioned room and sprinkle cold water on him while fanning the body. Rectal temperatures should be taken every 5 minutes. During cooling it will take about one hour to reduce the victim's rectal temperature below 30°C. During this time medical attendants should guard against possible circulatory shock and metabolic acidosis. Intravenous fluids should be started to guard against this circulatory shock and sodium bicarbonate should be infused to guard against metabolic acidosis (Feinstein, 1985).

The victim should be moved to a shaded, cool place where cooling and rehydration can begin. No fluid preparation is better than water in replacing sweat and preventing heat injuries. A person with heat stroke should be moved as soon as possible to a hospital to be stabilized and observed at least for 24 to 36 hours (Hart, et al., 1980). Thereafter, an accurate record of intake/output of body fluids must be made and serum electrolytes and enzymes monitored closely during the hospitalization (O'Donnell, 1975).

The medical director, the race director, and their staffs, and all competitors in an event need to be educated about the awareness prevention of heat injuries. The medical director should be knowledgeable in exercise physiology and sports medicine and should coordinate all preventive and therapeutic aspects of the running event (ACSM, 1984). A hospital in close proximity to the run should be notified and available ambulance service should be planned far in advance. These arrangements should be verified periodically before the actual date to insure adequate support for the event. The medical staff should be well trained and equipped to handle any emergency at a centralized location at the race site, and they should have a step by step procedure for triage and treatment of all

emergencies. Medical personnel with transportation should be positioned along the route to give immediate aid when needed.

The race director and his staff should be aware of the likelihood of heat injuries when planning a race. If they sponsor an event in the hot summer months, they should schedule the event before 8 a.m. or after 6 p.m. (ACSM, 1984). The event should be delayed or rescheduled if there is a 5°C increase in air temperature above that for the preceding day and the relative humidity is above 50% (Hughson et al., 1980). The race route should be designed as an "out-and-back" course to make supervision easier and should be located predominantly in shaded areas (Moore, 1982). On the entry form, all precautions for hot conditions should be listed because information at the starting line is usually not heard or heeded. Warning flags for thermal stress should be clearly visible at the starting line, if heat stress conditions prevail. During the race, accurate and frequent split times should be given. Additionally, well-staffed watering stations must be spaced every 2-3 km along the course (ACSM, 1984).

Hyperthermia is still the most common serious problem encountered in North American fun runs and races, and competitors should be made fully aware of this fact (ACSM,

1984). Competitors need to be educated about water intake, the early warning signs of heat stress, proper clothing, prior training and acclimatization. Inadequate fluid intake is a major factor in the occurrence of man's heat injuries. Asking runners to drink "to satiation" is not adequate. Thirst mechanisms are satisfied well before body fluid losses are replenished. Man, when subjected to heat stress, will not drink sufficient fluids to replace evaporative water loss, but will suffer from voluntary dehydration (Shibolet et al., 1976). Therefore, everyone participating in prolonged exercise in heat should be encouraged to drink at least 500 ml of cool water 15 to 20 minutes prior to the event, and to drink 250 ml at every water station. Competitors should avoid solution with high sugar concentration since they retard gastric emptying and water assumption in the intestines (Ribisl & Herbert, 1980).

Competitors should be constantly aware of the early warning signs. Runners can and should assume a major role in the prevention of serious heat injuries; these are dizziness, weakness, confusion, unsteady gait, irritability, or disorientation. The ACSM position stand of 1984 suggests "partner running," with each partner being responsible for the other's well-being and watching for the warning signs. A given competitor should refrain from participation if he

or she has had a recent fever or has one at the time of the event. Clothing should be light-weight and consist of only one layer of absorbant (cotton) material; such attire will aid in the evaporative process. Up to 70% of the cooling effect of evaporation can be lost when clothing inhibits air convection, radiation and evaporation (Robertshaw, 1983). Furthermore, a light colored hat is vital to protect the head because it reflects solar radiation and allows proper dissipation of heat.

Prior training and acclimatization are preferred for optimal performance in heat. A competitor should know his exercise capacities and stay well within these while performing in a hot environment. But competitors should also avoid any sudden increases in pace at the end of a race, as this has been shown to cause problems in heat (Hanson & Zimmerman, 1979). Finally, by gradually increasing the amount of exercise per day, acclimatization can occur with 10 to 14 days after exposure to a warm environment and this adaptation will markedly improve the capacity for dissipating heat loads during the race.

Theoretical Considerations of Temperature Regulation During Prolonged Exercise in the Heat

A basic overview of the neurogenic pathways, on which thermoregulatory signals travel, is necessary to understand

before discussing the theories of the thermoregulatory system. The operation of the thermoregulatory system will be divided into two main sections: 1) central mechanism control factor theories, and 2) peripheral factors.

The basic network is centered around a central nervous interface, which lies between the temperature sensors and the thermoregulatory correction effectors. The central nervous interface receives and interprets information transmitted to it from the sensors and issues appropriate signals to the effectors to correct the disturbance. The principal central nervous interface is located in the preoptic anterior hypothalamus, a region of the midbrain. Secondary central nervous interfaces are located in the medulla oblongata and the thoracic spinal cord (Bligh, 1979).

The pathway from warm sensors leads through the hypothalamus directly to heat loss effectors and from cold-sensors to heat production effectors. These sensors are free-nerve endings located superficially, in the body core and in the hypothalamus itself. Cold-sensors are closer to the surface of the skin and much more abundant than warm-sensors, but warm sensors are more abundant around the hypothalamus (McArdle, Katch, & Katch, 1986).

These sensors are stimulated externally by environmental temperature changes, and internally by increased heat production due to exercise or increased energy demands. The venous blood from the tissues active in heat production or heat loss drains back to the heart and lungs, and becomes thermally well mixed. The temperature of the arterial blood pumped out of the left side of the heart acts as a thermal feedback to the sensors (thermoreceptors), thus reducing or negating the disturbance signals (Bligh, 1979). Through a set-point determinant and the hypothalamic thermoreceptors, a comparison is made, and according to the direction and amount of offset, the appropriate effector system is activated to help maintain a constant deep body temperature.

Central Mechanisms

Many theoretical views have been proposed to describe thermoregulatory control. The major differences in these views entail: 1) whether or not there is a set-point determinant, 2) whether or not there is a central control and its location(s), and 3) the mechanism(s) by which thermoregulation operates.

Vendrik (1959) was one of the first to describe a set-point hypothesis of thermoregulation. According to him, the system was a genetically fixed activity/temperature

characteristic of the warm- and cold-sensors. The opposing activity/temperature of these sensors would function as a set-point determinant by adjusting the intensities of thermoregulatory heat production and heat loss effectors in opposing ways. A point of equilibrium would then be reached between heat production and heat loss. Therefore, the specialties of temperature regulation lie in the properties of the sensors and the thermoregulatory effector, rather than in the central nervous interface.

Benzinger, Kitzinger and Pratt (1963) described the thermoregulatory mechanism as a reflex response to loss or gain of heat from the stimuli of temperature at the sensory level. His hypothesis was that the behavioral defense against overheating originates from central hypothermia. The core temperature, not the skin temperature, is the source of the heat complaint. Therefore, the autonomic and behavioral thermoregulation originated from the core, not skin temperature. Central warm-sensors in the pre-optic anterior hypothalamus elicit sweat secretion and cutaneous vasodilation for increased heat loss, and cold-sensors under the surface of the skin elicit metabolic overproduction in response to cold. Skin cold-sensors inhibit sweating and central warm-sensors inhibit overproduction of heat.

They also demonstrated that the core temperature thresholds for heat production and evaporative heat loss vary with variation in the ambient temperature or skin temperature. The power of the responses compared to the small amount of stimulation is unbelievable: A 0.1°C change of central temperature may double the normal rate of heat loss or heat production.

One of the first theoretical views using the hypothalamus as the control center with a set-point determinant, was presented by Feldberg and Myers (1963). They stated that the mechanism was a biochemical gating influence on thermosensors to the thermoregulatory pathways. Their concept was that adrenaline and nor-adrenaline are continuously released in sufficient amounts in the anterior hypothalamus to keep the temperature at a constant level. When pyrogens or 5-hydroxytryptamine (5-HT) were introduced into the body, the temperature would increase, and when adrenaline and nor-adrenaline were injected into the cerebral ventricles, the temperature was brought down to normal or nearly normal depending on the amounts injected. Through biochemical gating, 5-HT and nor-adrenaline released from the anterior hypothalamus are in functional opposition to one another in order to control body temperature around a set-point. A rise in temperature could also be obtained by inhibiting the release of non-adrenaline or adrenaline.

Hammel (1965) described a theoretical neuronal model he called temperature sensitive neurons "high Q10 units" and temperature insensitive neurons "low Q10 units". The interaction between the two populations of neurons determined the set-point. The set point was reached when the activities between the two populations were equal. When the temperature was above the set-point, the activity of the high Q10 neuerons was greater than that of the low Q10 neurons. The thermoregulatory responses when the core temperature was above the set-point increased heat loss effectors and reduced heat production effectors. When the core temperature was below the set-point, the opposite was true. These populations of neurons were interneurons upon which the pathways from peripheral temperature sensors impinged (Hammel, 1965).

The set-point was affected by many physiological factors including changes in skin temperature. This adjustment in the set-point temperature due to changes in skin temperature was attributed by Hammel (1965) to a shift in the activity/temperature characteristics of both the high Q10 and the low Q10 neurons, and to the hypothalamic temperature at which the activities of the neurons were rated in response to a fall in skin temperature. These shifts in set-point temperature could account for an

increase in heat production in response to a fall in skin temperature and an increase in heat loss in response to a rise in skin temperature in the absence of any prior change in hypothalamic temperature.

A possible ionic mechanism used to regulate body temperature was given by Myers and Veale (1970). These researchers proposed that the set-point for body temperature was controlled by osmoreceptors within the posterior hypothalamus, and that the set-point was maintained and determined by the ratio in the concentrations of two essential cations, Na^+ and Ca^{++}. If a normal physiological concentration of both Na^+ and Ca^{++} was perfused at sites in the posterior hypothalamus, the temperature was unchanged. If the Na^+ ratio to the concentration was increased, it produced shivering and immediately, the temperature increased sharply. When Na^+ was omitted and Ca^{++} was normal or in excess, the temperature fell sharply. Myers and Veale (1970) hypothesized that the constancy in the concentration of extracellular ionic constituents maintains the firing rate of the neurons of the posterior hypothalamus.

At a procedural symposium in Paris on temperature regulation and drug action, Myers (1974) integrated the monoamine mechanism and the ionic mechanism of control by the hypothalamus of body temperature. He hypothesized that

the monoamine-containing neurons controlled the anterior hypothalamic set-point mechanism (Feldberg & Myers, 1963), and the ratio of cations determined the posterior hypothalamic set-point mechanism (Myers & Veale, 1970) and added that acetylcholine (ACh) transmitted the impulse signaling thermostasis. The net effect of 5-HT, acetylcoline, Na^{+} ions, and pyrogens was the same: hyperthermia, conversely, nor-adrenaline, adrenaline, and Ca^{++} ions bring hyperthermia back to a set-point determinant.

Houdas, Lecroart, Ledru, Carette and Guieu (1978) doubted whether a set-point existed at all. They suggested a model in which the controlled variable was the heat content of the body, and the load error signal that activates the control actions is a change in heat control. These changes in heat content are measured as changes of temperature at various sites by means of signals originating in all the thermoreceptors of the body. Therefore, what is measured in an average body temperature, as long as the heat capacity of the body does not change, the average body temperature remains parallel with heat content. But this correlation will not work with changing heat capacity, which occurs during growth and during gain or loss of weight in adults.

Bligh (1984) combined his work from 1979 with the models (Figure 1) presented by Hammel (1965), Wyndham and Atkins (1968), and the principles of central neurology (Sherrington, 1906). The resulting model allowed for the "resetting" of the set-point with changes in skin temperature and within other non-thermal environmental and physiological disturbances. The variability of the effective set-point in temperature regulation is a consequence of the synaptic convergence of other influences within the central nervous system. It allowed for species and seasonal variations in the level at which body temperature is held. These variations could depend on: genetically-fixed differences in the activity/temperature profiles of the warm- and cold-sensors (Vendrik, 1959); the concentrations of warm and cold sensors located in various parts of the body; and upon seasonal variations in the intensities of excitatory and inhibitory influences acting on the central neuronal pathways.

The cutaneous tissues, spinal cord, and hypothalamic thermoreceptors send disturbance spinals along afferent pathways to the central nervous interface. Applying the Sherrington principle of the summation of stimuli in the central nervous interface would demand a dominant influence by the core temperature sensors when the core temperature is

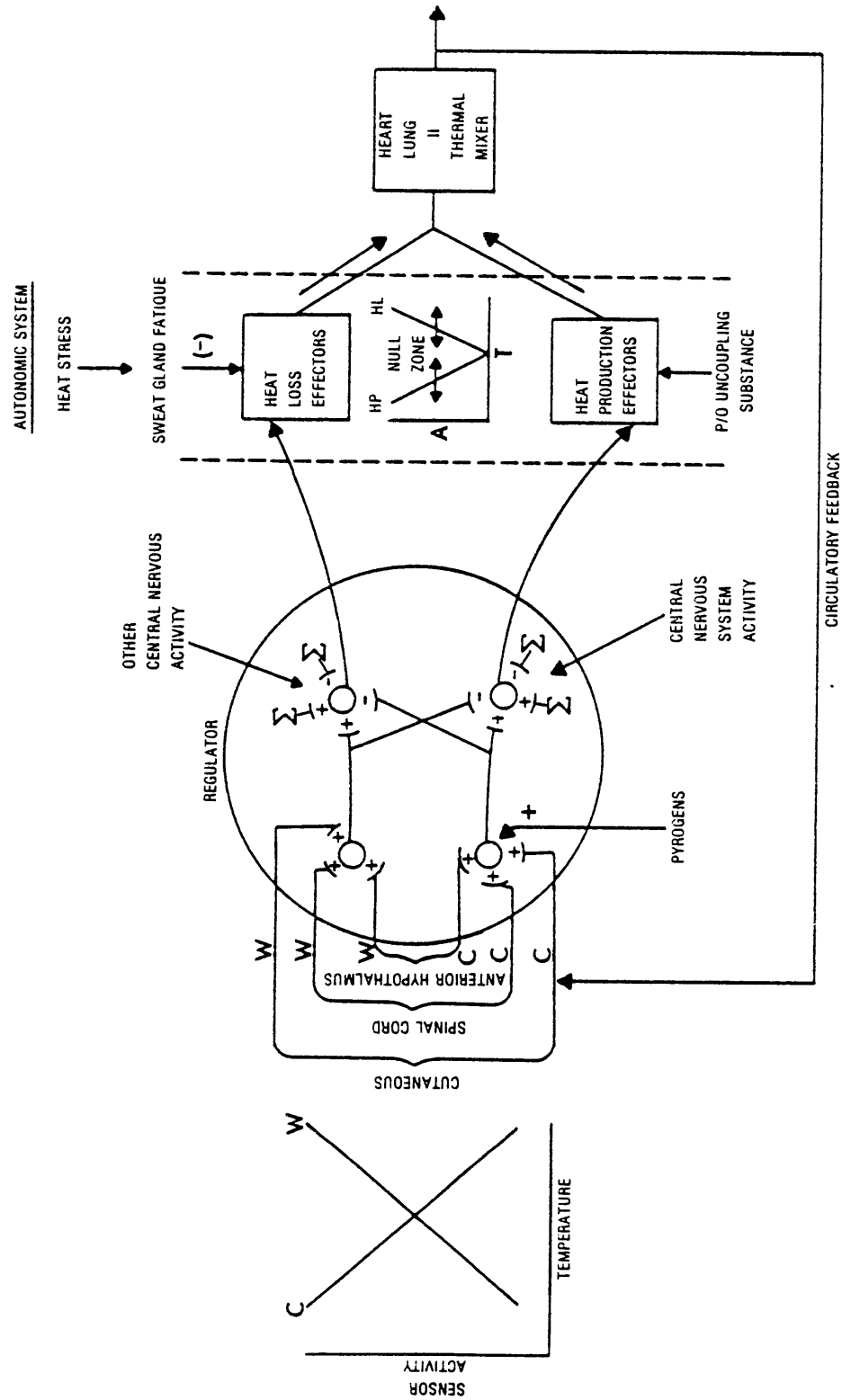

Figure 1. A schematic representation of the thermoregulatory system and factors that influence its control. Adopted from Bligh (1979).

fairly stable, as in man. From the central nervous interface, the stimulus for heat production or heat loss travels to the effectors in the behavioral and autonomic systems. But a behavioral response can modify the relations between man and his environment, and the need for autonomic thermoregulatory response. This implies no need for central nervous system coordination between behavioral and autonomic thermoregulation. The arterial blood, which has been thermally mixed is fed back to the thermoreceptors once the correction is made to stabilize temperature regulation.

In this section the central mechanisms, theoretical views, and neuronal models have been stated chronologically to show the necessity for a central nervous interface (hypothalamus) to control a "set-point." Only (Houdas et al., 1978) disaffirmed this idea, but their theory was not accepted due to other biological factors. Bligh (1984) has given the most complete explanation, considering all the variability involved with thermoregulation, and his model would be controlled primarily by central nervous system factors.

Peripheral factors which play a major role in thermoregulation are the baroreceptors and chemoreceptors, within the circulatory system, sweat glands and their functions, and the skin temperature. All of these factors are discussed in detail in this section.

Baroreceptors

Two types of baroreceptors identified in the human circulatory system function as high pressure and low pressure sensors. Baroreceptive reflexes are activated by changes in cardiac filling pressure and stroke volume. Both of these sensors are compensated by changes in heat rate (HR) and vasoconstriction in the peripheral vascular beds (Nielsen, 1984).

High pressure baroreceptors are located at the bifurcation of the common carotid arteries, the carotid sinus, and throughout the wall of the aortic arch (the aortic bodies). High pressure baroreceptors are stretch receptors that send information to the central nervous system reporting the level of arterial blood pressure. Any fall in arterial blood pressure is detected by high pressure baroreceptors resulting in neural feedback which leads to increased heart rate and a parallel rise in aortic pulse pressure (Rowell, 1977). This decrease in arterial blood pressure activates the sympathetic nerves to increase contractility of the heart and to increase peripheral resistance by vasoconstriction, thus increasing arterial blood pressure (Thew, Mutschler & Vaupel, 1985).

The low pressure (atrial) baroreceptors are located in both atria and the wall of the vena cava. These

baroreceptors respond to a decreased cardiac filling pressure and decreased blood volume. Hypovolemia, i.e., lowered blood volume, stimulates low pressure baroreceptors to modify neural information to the hypothalamus which then causes an upward shift in the threshold for cutaneous vasodilation and controlling sweat rate (Nadel, Fortney, & Wenger, 1980). Thus, a reduced circulating blood volume stimulates a baroreflex to maintain blood pressure by vasoconstricting cutaneous vascular beds and decreasing maximum cutaneous blood flow (Nadel, 1983). Moreover, hypovolemia decreases the cardiac filling pressure, which reduces neural input to the hypothalamus. The response is an increased secretion of the antidiuretic hormone (ADH) that reduces water excretion by the kidney (Best & Taylor, 1985).

Chemoreceptors

Chemoreceptors are located in specialized cells near the carotid sinus and aortic bodies. They are sensitive to changes in blood gases and pH. When chemoreceptors become hypoxic and/or acidic due to decreased ventricular function, they will reflexly stimulate vasoconstriction in the peripheral circulation (Biscoe, 1971).

Sweat Gland Function

Sweat is hypotonic compared to blood and Na^+ and Cl^- are the principal ions lost from the extracellular compartment. Sweat rate is the major factor regulating the concentrations of these ions in perspiration (Costill, 1977). If the sweat rate is slow, the Na^+ content of sweat will be low because the sweat duct is able to reabsorb most of the Na^+, but as the sweat rate increases the loss of Na^+ also increases (Houdas et al., 1978). Exercise sweating results in a total body hypohydration and an increased hemoconcentration (Kozlowski & Saltin, 1964). The loss of plasma with exercise exerts an increased osmolality, and the increased serum osmolality then further leads to a decrease in sweating (Greenleaf, 1972).

Sweat glands are innervated by a sympathetic cholinergic fibers which are controlled by the thermoregulatory center in the hypothalamus (Fortney, Nadel, Wenger, & Bove, 1981), and acetylcholine is the chemical activator. Sweating transfers metabolic heat to the environment by evaporation and compensates for changes in the ambient temperature. Evaporation cools the skin to help maintain a proper core-to-skin thermal gradient for heat loss. The main factor limiting the evaporative process is the difference in water vapor pressure between the skin and

the air. If the water vapor pressure of the air is higher, evaporation at the skin surface cannot occur (Nadel et al., 1977). With a low ambient water vapor pressure, evaporation tends to increase as the sweat rate increases until heat loss equals heat production. Once this occurs, the body is no longer storing heat and the Ts stabilizes.

Ts and Tc act together to control the rate of sweat secretion. If the Ts is above 33°C, it has no effect on the control of sweat rate, but if Ts is below 33°C, it systematically suppresses sweating (Brengelman, Johnson, Hermansen, & Rowell, 1977). During exercise, the metabolic heat load is high, and this drives the Tc up to stimulate a sweat response. The sweating mechanism is primarily responsive to the deep body temperatures (Nadel, 1985).

The final factor which limits sweat gland function is hypovolemia, i.e., a reduced blood volume. Total sweat loss at anytime during exercise is lower when hypovolemia exists (Fortney et al., 1981). This is at least partly due to an impaired ability to maintain optimal cutaneous circulation and that to metabolically active muscle. As a consequence, heat transportation from muscle to body surface is reduced and Tc will increase, even if sweating and evaporation are maintained.

Skin Temperature

Major thermoregulatory effects via the circulatory system are mediated by the skin. When exercising in a hot environment, the circulatory system is under stress to maintain adequate cardiac output ($\dot{Q}$) for the metabolism of active muscles, "resting" muscles and tissues, and heat transport to the skin (Benzinger et al., 1963). The heat production that occurs within the active muscles during exercise can increase the basal metabolic rate as much as 15 to 20 times and is sufficient to raise the core temperature by 1°C every 5 min. if no thermoregulatory mechanism were activated (Nadel et al., 1977). Heat is transported to the skin by the circulating blood, and the temperature of the skin maintains an equilibrium between heat production and heat loss.

The human body must maintain a proper thermal gradient where the core temperature (Tc) is greater than the skin temperature (Ts) to make heat transport possible (Houdas et al., 1978). As the Tc increases with exercise, the sympathetic vasomotor tone decreases allowing vasodilation of cutaneous vessels and skin blood flow increases (Mitchell, 1977).

The most important factor determining heat transfer is Ts. The temperature of the skin is constantly changing to

adjust to both external and internal factors. The major external factors are the ambient temperature (Ta), and to a lesser extent, air velocity and relative humidity. When the Ta increases to 35°C the range of the Ts is minimal, but as Ta decreases, the range will increase. The temperature of the skin will rarely exceed 37°C. Cutaneous vessels regulate cardiovascular mechanisms and are triggered by changes in the environmental temperature.

The internal factors are initiated by increases in the core temperature (Tc). The Tc can effect Ts directly through tissue conductivity from underlying organs and tissues, especially subcutaneous tissues, or indirectly, by blood convection. Blood convection is dependent on the loss of sympathetic vasomotor control to the skin allowing for increased skin blood flow (Houdas et al., 1978).

Constitutional Factors Modifying Temperature Regulation

Numerous factors effect the body itself, and how the body adapts to these natural occurring phenomena, is the major concern in this section. The constitutional factors which are discussed in this section are age, sex, anthropometry, exercise capacity, physical training and heat acclimatization. All of these have internal and external effects on the body's thermal adaptations to exercise in a hot environment.

Age

Thermal injuries are of major concern throughout one's life, but the most susceptible groups in the population are children and the elderly. In newborns, due to the thinness of their skin, involuntary heat loss is much greater than an adult's at the same water vapor pressure level (Houdas et al., 1978). The sweat gland functioning of newborns' for maximal secretion is only one-third to one-half that of an adult, but by the postnatal phase of growth the child reaches maximal secretion levels (Houdas et al., 1978). But children still perspire less than adults and have a lower heat-exercise tolerance (Bar-Or, 1980). When children become dehydrated, they will have a greater increase in core temperature (Tc) than an adult at the same level of dehydration.

The susceptibility to thermal injuries in the elderly was confirmed by Austin and Barry (1956) and Levine (1969). A reduced basal metabolic rate due to loss of lean body mass and lack of physical activity are two reasons why the elderly prefer a higher ambient temperature than younger people (Langkilde, 1979). But in a hot environment, the elderly have a reduced ability to discriminate (perceive) between temperatures.

Under thermal stress conditions, preexisting medical conditions can add to the susceptibility of the elderly. During prolonged exposures in hot environments, the elderly are reported to have a decrease in sweat production and this factor places a strain on the cardiovascular system (Schwartz & Itoh, 1956). Foster, Ellis, Dore, Exton-Smith, and Weiner (1976) reported this reduction was manifested in the quantity of sweat secreted by each gland rather than the number of active glands, as age increased. Furthermore, older subjects have a significantly lower body conductance and are much less able to dissipate heat by radiation and convention. Therefore, there is a relatively greater dependence on peripheral blood flow for heat dissipation, and this puts an increased strain on the aging cardiovascular system (Irion, Wailgum, Stevens, Kendrick, & Padlone, 1984). Exacerbating this problem is the fact that the elderly have a reduced capacity for increasing the cardiac output to allow for the necessary increase in peripheral blood flow to dissipate heat from the core of the body (Hellon & Lind, 1958). It has been reported that the lower skin temperatures observed in the elderly are at least partly due to a lower skin blood flow than in younger subjects (Wenger, 1984).

Fleisch (1980) found evidence of a decreased sensitivity to vasoactive substances in the smooth muscles of blood vessels in older animals. A decrease in vascular distensibility is characterized by aging, and the stretch receptors in the blood vessels are less effective (Wenger, 1984).

Sex

The most common thermoregulatory difference observed between males and females is in the sweating response. The Tc during steady-state exercise in males is initially higher, and males are more sensitive to increases in Ts (Hardy, 1981). However, females start sweating at lower Ts and Tc (Leithead & Lind, 1964). Females produce less sweat for the same exercise heat load (Dill, Soholt, Drost, & Loughran, 1977). Even with this lower sweat output, females are able to maintain a similar heat-exercise tolerance as compared to males at the same percentage of the exercise capacity (Bar-Or, Lundegren, & Buskirk, 1969).

Drinkwater, Denton, Kupprat, Talag, and Horvath (1976) explained the sweating differences between males and females in that females rely more on peripheral blood flow (specifically vasomotor control) whereas males depend more on their ability to cool the body through evaporative means.

Exercise Capacity

The core temperature rises to a level which is proportional to the oxygen intake ($\dot{V}O_2$) and is independent of a wide range of ambient temperatures. The core temperature and the temperature of the working muscle is proportional to the relative work load and not to the absolute work load of the individual (Nielsen & Nielsen, 1962). Astrand (1970), Davies, Brotherhood and Zeidifard (1976), and Saltin and Hermansen (1966) all found that what first appears to be large intra-subject variability of core temperature is reduced if it is related, not to the absolute but to the relative metabolic rate. Therefore, at any fixed level of work, individuals with low $\dot{V}O_2$ max develop higher body temperature while exercising in a hot environment than individuals with higher $\dot{V}O_2$max (Wyndham, Strydom, Van Rensburg, Benade, & Heyns, 1970). A fit individual at the same %$\dot{V}O_2$max produces more energy in exercise and still has the same core temperature as the unfit individual (Herbert, 1979). A fit individual at the same workload as an unfit individual will exercise at a lower core temperature and will be at an advantage at any given absolute exercise intensity (Senay, 1979).

Saltin and Hermansen (1966) found the core temperature to %VO max relationship to be linear, but their study is limited to relative workloads at or below 75% of $\dot{V}O_2$max. A

follow-up study by Davies et al. (1976) at higher relative workloads shows that the core temperature becomes a curvilinear function of $\%\dot{V}O_2$max.

Acclimatization and Training

The major time course adaptations which take place during acclimatization and training are related to the cardiovascular system. When man is initially exposed to heat, an acute decrease in blood volume (BV) occurs during an exercise bout due to a decrease in plasma volume (PV) (Houdas, 1978). Another initial problem is peripheral pooling of blood, due to the dilation of peripheral blood vessels, and causing a decreased venous return to the heart (Roberts & Wenger, 1979). Therefore, stroke volume (SV) decreases and cardiac output ($\dot{Q}$) can only be maintained by an increase in heart rate (HR) to near maximal levels; this circulatory inadequacy may contribute to heat syndromes (Rowell, Kraning, Kennedy, & Evans, 1967).

The instability of the cardiovascular system is affected by volume receptors that stimulate the secretion of the antidiuretic hormone (ADH) and aldosterone (Wyndham, Benade, Williams, Strydom, Goldin, & Heyns, 1968). These hormones function to help the body retain sodium chloride and water retention which helps maintain BV, regulate osmolarity of extracellular fluids and protect against dehydration (Sawka, Toner, Francesconi, & Pandolf, 1983).

During the first few days of acclimatization and training, PV expands to account for an increase in BV (Greenleaf, Sciaraffa, Shvartz, Keil & Brock, 1981). Venous return to the heart increases and augments an increase in SV. Stabilization of the cardiovascular system occurs with this increase in SV, and $\dot{Q}$ can be maintained at a lower HR for a given intensity, thus providing a wider safety margin in emergency situations, i.e., peripheral pooling.

The improvement of central circulation increases the capacity for heat conductance to the skin and facilitates the onset of sweating at a lower Tc; this provides for a lower steady-state Ts and Tc, respectively. Through training and acclimatization, the sweating mechanism becomes more responsive and heat dissipation occurs more rapidly (Nadel et al., 1977). The number of active sweat glands increases and according to Sato and Sato (1985), the sweat glands actually hypertrophy with training. The amount of sweat secreted increases and becomes more dilute, thus minimizing loss of electrolytes (Nadel, Pandolf, Roberts, & Stolwijk, 1974).

Anthropometry (Mass/Body Surface Area Ratio)

Individuals with excess adipose tissue have an increased heat insulative capacity. Subcutaneous body fat has a fixed resistance that insulates the core from the

skin. The thermal conductance of fat is less than 50% that of muscle tissue and about 35% that of blood. Therefore, those with endomorphic body types are at a definite disadvantage in the heat due to the increase in insulation of fat tissue (Nadel, 1977).

Smaller individuals weighing 50 kg or less, are shown to be at a disadvantage when performing strenuous physical tasks (i.e., external loads, % watts) in the heat. The maximal oxygen intake of a heavier individual is higher than that of a lighter person. Therefore when performing at the same workload, the heavier individual will be working at a lower % $\dot{V}O$ max than the lighter individual. Secondly, the small individual has a smaller surface area through which to evaporate sweat and to dissipate heat fast enough to offset heat production (Strydom, Benade, Heyns, & Hons, 1971).

According to Robinson (1942), if a large individual's total increase in heat production is the same as the small individual for a given workload, the larger man can distribute this extra heat through the tissues more effectively. But when both men walked at the same rate and heat production was proportional to weight, the larger man produced, in relation to surface area, about 20% more heat than the smaller man. However, in a hot-humid environment a large man's heat dissipation is restricted because

evaporative cooling is impeded, and he is forced to store heat.

Environmental Factors Modifying Temperature Control

Environmental factors, which modify temperature control in the human body are ambient temperature, relative humidity, wind velocity, and the heat loss modes of radiation, conduction, convection and evaporation. All of these factors can increase the heat load upon the thermoregulatory system.

Ambient Temperature

The ambient temperature has the greatest effect on which heat loss mode will be used to dissipate the body's heat load (Mitchell, 1977). At low ambient temperatures, convection is the major mode of heat loss, and radiation, evaporation, and conduction play minor roles. As long as the temperature of the skin is considerably higher than the ambient temperature, the heat loss by convection and radiation are high (Saltin et al., 1966). However, when the ambient temperature is greater than the internal body temperature, the thermal gradient is reversed. Instead of losing heat by radiation, convection and conduction, the body will gain heat by these modes. Hence, leaving evaporation as the only potential mode for effective heat dissipation (Costill, 1977).

The core temperature of the body is independent of ambient temperatures when the latter is between -5° to 30°C and is determined mainly by metabolic rate (Snellen, 1969). Wyndham (1973) stated that the higher the metabolic rate, the lower the critical air temperature had to be for acclimitization to occur with hard work. Ambient temperature has the greatest effect on skin temperature, but skin temperature is independent of the workload placed on the body (Saltin et al., 1966). The rate of sweat production increases directly with increasing ambient temperature and, at 35°C, 85% of the heat loss must be achieved by evaporative sweating (Mitchell, 1977).

Relative Humidity

The heat loss mode affected the most by relative humidity is the evaporative process. In a hot dry environment sweat evaporates freely and cools the skin. Therefore, the level of evaporative heat loss loss required to dissipate the metabolic heat load is reached at a lower internal body temperature (Roberts & Wenger, 1979). As the relative humidity increases, the evaporative process decreases, and the sweat rate is suppressed. This causes the body temperature to rise and a thermal equilibrium cannot occur until a higher core temperature is attained (Nadel, 1977).

The sweat rate is suppressed by a hidromeiotic effect (skin wettedness) (Fox, Lofstedt, Woodward, Eriksson, & Werkstrom, 1969). Different ambient humidities require different amounts of skin wettedness to achieve the same evaporative heat loss, but with a high relative humidity, the evaporative process is ineffective; this causes hypohydration and leads to a steady-state body temperature that is much higher than would occur under conditions of lower humidity (Mitchell, 1977). The amount of heat released by the evaporative process is dependent on the water vapor pressure differential between the skin and air. If the water vapor pressure of the skin is lower than that of the air, the evaporative process is reduced. But even at a relative humidity of 100%, the evaporative process can occur as long as the temperature of the skin is greater than the ambient temperature (Thew et al., 1985).

Wind Velocity

When cool air blows across the surface of the skin, heat produced by the body can be lost through convection. The velocity and temperature of the air are the major factors determining the amount of heat loss (Fox & Matthews, 1981). Increasing the air velocity on an exercising subject allows a lower steady-state core temperature to be reached with less evaporative sweating (Nadel, 1977). Accordingly,

an increase in convective heat loss reduces the need for evaporation, but even large changes in air velocity produce only small changes in the amount of heat loss (Mitchell, 1977). Clothing or any barrier between the skin and the environment reduce the wind velocity at the surface of the skin and, in addition, decrease the evaporative capacity (Nadel, 1977).

Heat Loss Modes

The major mode of heat loss under "normal" conditions is radiation. According to research by Fox and Matthews (1981), who collected data from a nude person resting quietly in a room temperature of 21°C, sixty percent of the heat loss was due to radiation. However, when the ambient temperature exceeds the temperature of the skin at 35°C or above, other evidence suggests that radiant heat is gained from the environment (McArdle, et al., 1986).

In order for conduction to occur two solid objects of different temperatures must be in contact. As long as the skin temperature is greater than that of an external solid in contact with the skin, body heat loss occurs, but when the external object's temperature exceeds the skin temperature, heat will be gained. Two barriers, which resist conductive heat transfer from the body, are adipose tissue and clothing (Houdas, 1982).

So long as the ambient temperature does not exceed skin temperature, convection is a major mode of heat loss. Internally, blood convection is the ability of the blood to pick up excess metabolic heat from active tissue and transfer this "waste" heat to the skin, where it is than transferred to the ambient environment. Externally, warmed air molecules are removed from the skin by air movement, and cooler molecules replace them. The temperature of blood arriving at different tissues dictates the thermal state of that tissue (Houdas, 1982). Thus, heat gained by blood leaving thermogenic active muscle, is transferred to the cooler tissues of the skin.

Evaporation is constantly occurring at the body surface. Evaporation serves a minor mechanism for heat dissipation compared with other modes when the ambient temperature and relative humidity are low, i.e., "insensible" perspiration. However, when the ambient temperature increases above that of the skin, evaporation becomes the only mode of heat loss. All other modes at this point reverse their gradients and cause heat to be gained.

Individual Factors Modifying Temperature Control

The exercise conditions, such as the intensity and duration of the activity, can be major factors which affect the body's ability to regulate heat. The mode of exercise

(running vs biking) also must be considered. Furthermore, the internal individual physiologic conditions that affect thermoregulation must be addressed, e.g., fluid balance and electrolye, osmolality and plasma protein status in body fluids. Fluctuations in these factors can be detrimental to temperature regulation in exercise.

Exercise Conditions

Intensity and Duration

The steady-state core temperature is proportional to the relative intensity of the exercise, i.e., % $\dot{V}O_2$max. When a subject increases the intensity of exercise, the heat production with the active muscles increases, and the rise in core temperature is proportional (Nadel, et al., 1977).

Water losses from the blood plasma are also proportional to the relative intensity of exercise (Nadel et al., 1980). At the onset of exercise the decrease in plasma volume is attributed to a rise in the mean arterial pressure (Myhre, Hartung, Nunneley, & Tucker, 1985). According to Miles, Sawka, Glaser and Petrofsky (1983), blood plasma water losses in progressive arm and leg exercises are independent of the muscle mass but are directly related to the intensity of the exercise. Subsequently, as the plasma volume decreases during exercise of increased intensity, the plasma osmolality increases along with the concentrations of

sodium, potassium and chloride (Lundvall, Mellander, Westling & White, 1972).

Increasing the intensity of exercise also elicits certain cardiovascular changes such as increased heart rate, stroke volume, cardiac output, and oxygen extraction rate (Best & Taylor, 1985). The degree of vasoconstriction at the onset of exercise is directly related to exercise intensity, and this is associated with a decrease in skin blood flow (Hirata, Nagasaka, Hirai, Hirashita, & Takahata, 1984). In exercises of short duration, these peripheral vasoconstrictive effects related to intensity have virtually no influence on thermal equanimity, but in prolonged exercise situations, the effects of rising intensity are detrimental to performance (Rowell, 1974).

Mode of Exercise

Cycling exercise has been observed to elicit lower oxygen uptakes ($\dot{V}O_2$) and higher blood lactate values than does running on a treadmill (Koyal, Whipp, Huntsman, Bray, & Wasserman, 1976; Hermansen & Saltin, 1969). Wasserman, Whipp, Koyal, and Beaver (1973) attributed these findings to a smaller active muscle mass involved in cycling at any given exercise intensity. Another explanation was given by Matsui, Kitamura and Mizamura (1978) that the leg blood flow during maximal cycling exercise was significantly lower than leg blood flow during maximal treadmill exercise.

Senay (1979) investigated body fluid shifts during exercise and found that the mode of exercise (bicycle, ergometer, treadmill, etc.) significantly influenced the shifts. A reduction in the plasma volume occurs during cycling exercise (Harrison, Edwards & Leitch, 1975; Lundvall, Mellander, Westling, & White, 1972). Body fluid shifts are also affected by the posture of the cyclist (Diaz, Bransford, Kobahashi, Horvath, & McMurray, 1979). During short-term treadmill exercise, a reduction of plasma volume and a hemoconcentration occurred, but with 20 min of treadmill exercise at 76% $\dot{V}O_2$ max no hemoconcentration occurred (Galbo, Holst & Christensen, 1975). Wilkerson, Guten, and Horvath (1977) using a similar protocol found that after 20 min of exercise levels in excess of 60% $\dot{V}O_2$max a hemoconcentration was obtained after prolonged exercise (>2 h), despite considerable water loss, little hemoconcentration was found to occur in subjects studied by Maron, Horvath and Wilkerson (1977). These findings reflect inconclusive evidence concerning the conditions that may induce significant circulatory fluid loss in treadmill exercise.

Hydration State

Under most exercise conditions, sweat losses are greater than the amount of water a subject can take in

during the activity, and a hypohydration state arises. This imposes multiple problems on the exercising human. An increase in the steady-state core temperature is brought about by increased heat storage due to an inability to dissipate the thermal load (Nadel, 1977). Peripheral blood flow decreases due to hypohydration, which in turn decreases the amount of convective heat transfer from active muscle as well as transfer to the sweat glands (Nadel et al., 1980). Therefore, a decrease in evaporation results in further impairment of other modes of heat transfer. During a marathon, Wyndham (1977) gathered data which provided a basis for predicting that a 5% water loss would elicit a 41° C core temperature upon a runner's completion. This is attributable to a decreased sweat rate which is attributable to the body's need to conserve water and prevent excessive losses of fluids.

Hypohydration impairs both exercise performance and physiological function (Harrison, Edwards, & Fennessy, 1978). Development of hypohydration while exercising in a hot environment neutralizes the advantages of aerobic fitness and acclimatization. Aerobic capacity dissiminates due to an inability to attain the normal maximal cardiac output (and stroke volume) due to loss of water from the vascular compartment. At submaximal exercise intensities,

the heart rate increases to compensate for the impaired stroke volume, but this is inadequate as intensity approaches near maximal levels. The reduced cardiac output cannot leads to insufficient oxygenation of the active muscles and thus, performance decreases (Sawka, Francesconi, Young, & Pandolf, 1984).

In contrast, when an exercising subject is hyperhydrated the plasma concentration of electrolytes are diluted and osmolality is reduced slightly. Vascular water volume is adequately maintained within the body and cardiac output can reach maximum capacity. Accordingly, the exercise heart rate and steady-state core temperature decrease (Moroff & Bass, 1965; Nadel et al., 1980).

Intracellular Fluid Compartment and Extracellular Fluid Compartment

The two main fluid compartments in the body are the intracellular fluid compartment (ICF) and the extracellular fluid compartment (ECF), the latter includes the interstitial space and the vascular compartment. A water permeable membrane separates these compartments and the three main factors governing fluid shifts among compartments are: 1) gradient of colloid pressure; 2) tissue fluid pressure; and 3) hydrostatic pressure (of the blood).

Costill and Fink (1974) studied the effects of hypohydration on the fluid compartments and found that water is lost proportionally from each. At lower degrees of hypohydration (2% body weight loss) the ECF is the space of primary losses, but as hypohydration increases the distribution of water losses is equal from each compartment. During thermal hypohydration, a greater loss of Na^+ and Cl^- and a smaller loss of K^+ occurs in the ECF than during exercise hypohydration. Harrison, Edwards, Graveney, Cochrane and Davies (1981) suggested that if extracellular sodium chloride concentrations are increased, a redistribution of body water would have occurred at the expense of the ICF. Also an increase in total body fluid would have to occur since more fluid was retained as a result of extracellular osmotic pressure, but since no change occurred to the total body water and the intracellular water, the water and osmolality of the intravascular space could only have increased at the expense of the interstitial space.

At the beginning of exercise, the changes in plasma volume occur in the ECF with a temporary shift of water from the vascular space to the interstitial space without an actual loss of fluid from the body (Beaumont, Strand, Petrofsky, Hipskind, & Greenleaf, 1973). If exercise is

maintained beyond 20 min, lymphatic circulation effects return of some of this fluid to the vascular compartment.

Electrolytes

The major electrolytes involved in thermoregulation are sodium (Na+), chloride (Cl-) and potassium (K+). Sodium chloride is found in abundance in the extracellular fluid compartment and is the ion having most influence on maintenance of the vascular volume. Water loss during sweating reduces the plasma volume which increases the NaCl concentrations in the plasma. The principle ions lost in sweat are Na+ and Cl-. Heat exposure, exercise, and/or hypohydration (if fluid is not replaced) results in an arterial hypovolemia and a renal ischemia, which stimulates an increased release of aldosterone. Aldosterone, a mineralocorticoid, leads to the retention of sodium by increased renal reabsorption (Costill, 1977). Myhre and Robinson (1977) stated that as long as fluids were replaced, plasma NaCl was not affected by prolonged heat exposure of 4 hs, but when hypohydration occurred, NaCl was responsible for 90% of the osmotic activity within the fluid compartment (Myhre & Robinson, 1977).

Potassium is found in large abundance in the intracellular fluid compartment. Marathon running and even repeated days of exercise dehydration only produce slight

decreases in K+ (Costill, 1977). With prolonged exertion the intramuscular and plasma K+ ratio is significantly altered and this finding suggests a possible modification in the muscle cell membrane (Beaumont et al., 1973). However, sweating has little effect on K+ concentrations in plasma or muscle tissue (Costill, 1977).

Osmolality

The hydration state of the body is the major factor dictating the effects of osmolality. If the body is normally hydrated, an osmolar equanimity exists and water balance is maintained among the fluid compartments. In a hypohydration state, hyperosmolality occurs due to the decrease of water in the vascular fluid volume. Small changes in osmolality serve as a more potent stimulus for body water conservation than small changes in the blood volume (Dunn, Brennan, Nelson & Robertson, 1970). Any changes in osmolality correlate with changes in Na+, which is the primary electrolyte determining the plasma osmolality, and both of these changes correlate strongly with decreased rates of evaporation (Greenleaf, van Beaumont, Brock, Morse, & Mangseth, 1979). It is believed that the increased plasma osmotic pressure accompanying hypohydration and the reduced peripheral hydrostatic pressure stimulate the retention of electrolytes,

maintenance of central blood volume, and decreased sweat rate (Greenleaf, Convertino, Stremmel, Bernauer, Adams, Vignau & Brock, 1977). The increase in osmolality stimulates the osmoreceptors in the hypothalamus to increase vasopressin secretion, which increases the water reabsorption in the kidneys and the eccrine sweat glands; this also stimulates the thirst sensation (Nielsen, 1974).

Protein

Plasma proteins are the principle circulatory factors determining colloid osmotic pressure and which resist the loss of fluid from the vascular space (Myhre et al., (1985). The movement of plasma proteins among fluid compartments is due to three factors: environmental temperature; the state of acclimatization; and the proportion of the cardiac output distributed to cutaneous and muscle capillaries (Senay, 1978). During heat exposure, shifts in plasma protein content are results of increased capillary permeability (Myhre & Robinson, 1977). Costill and Sparks (1973) observed an increase in plasma protein equal to the decrease in plasma volume. Harrison (1974) found no significant shifts of albumin from the vascular compartment during 2 hs of passive heat exposure with 10% decrease in plasma volume.

Regarding the effects of acclimatization on vascular protein response to heat exercise stress, acclimatization

either alters the rate at which protein escapes from the cutaneous capillaries or modifies the available protein population residing within the cutaneous interstitial spaces (Senay, 1975). Therefore, the influx of proteins into the vascular volume occurs more rapidly after acclimatization. Rapid dilation and constriction of cutaneous vascular beds by the massaging action of muscles causes an increased perfusion and an increased protein influx to the vascular volume (Senay, 1978).

Summary

The incidence of a heat-related injury occurring during prolonged physical exercise is evident especially on hot humid days. The preceding sections have included important literature in several research areas. The initial section described the clinical factors related to thermal injuries. The second section included the theoretical controls of thermoregulation and related peripheral control factors. The third section presented the constitutional factors which could possibly modify temperature regulation. The next section initialed environmental factors which exert an influence on body temperature, and the final section dealt with the individual factors modifying temperature control. Thus presenting an overview of the thermoregulatory system along with any factor which could possibly modify that system.

EFFECTS OF CHANGES IN PLASMA VOLUME, OSMOLALITY AND SODIUM LEVELS ON CORE TEMPERATURE DURING PROLONGED EXERCISE IN HEAT

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Running Head: Effects of Hematologic Changes on Core Temperature

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ABSTRACT

Six adult males of similar body composition and aerobic capacity were tested to study the effects of changes in plasma volume (PV), osmolality (OSM) and sodium (Na+) on core temperature (Tc) under three exercise-thermoregulatory stress conditions. The protocol consisted of 120 min of upright stationary cycling at 50% $\dot{V}O_2$max under neutral (24° C, 50% RH) - euhydrated (NE), hot (35°C, 50% RH) - euhydrated (HE), and hot-hypohydrated (HH) environmental conditions. Venous blood samples were obtained at -30 min, 0 min and at 15 min intervals through a 30 min recovery and were analyzed for blood hematocrit and hemoglobin, and for plasma osmolality and sodium. Hematocrit and hemoglobin were used to calculate relative changes in plasma volume. Tc showed qualitatively similar linear increases in the first 45 min of each trial. At 60 min, Tc in the NE trial plateaued at 37.9°C. In the HE trial, Tc continued to show a slight further increase after 45 min while in NE it became significantly ($p<0.05$) lower at 45 min as compared to HE and HH; at 60 min of exercise, the core temperature of all three trials differed significantly ($p<0.05$), with HH being the highest (38.3°C). Percent change in plasma volume was not different between trials, but did show the greatest decrease in all trials from 0 to 15 min of the exercise phase with at

least -4.3%. Osmolality was significantly different ($p<0.05$) between the NE (x = 283.3 mOsmol/kg) and the HH (x = 292.5 mOsmol/kg). Plasma sodium was significantly ($p<0.05$) higher for all intervals of HH (x = 137.9 meq/L) as compared to the NE (x = 135.1 meq/L) and HE (x = 134.8 meq/L). These data suggest that core temperature (Tc) increase in moderate intensity endurance exercise is less related to a decreased circulating plasma volume, but is more strongly associated with rising osmolality, specifically the increase in the Na^+ electrolyte, which occur with progressive hypohydration.

Index terms: core temperature; plasma volume; osmolality; sodium; prolonged exercise thermoregulation; euhydration; hypohydration.

Introduction

Heat injuries are a major problem when individuals engage in physical work or prolonged exercise in a hot environment. Environmental conditions, especially ambient temperature and relative humidity, play a major role in the body's ability to regulate internal body temperature. As the ambient temperature rises evaporation becomes the main heat loss mode.

Another factor, which contributes to the rise in core temperature, is the state of hydration. Early studies by (22,23) demonstrated that fluid restriction in a hot environment was associated with a continuous rise in body temperature under resting and exercising conditions, but when fluids were replaced, the internal temperature remained constant.

The hypothalamus is considered the control center for thermoregulation, interpreting neural and humoral inputs from various stimuli in the body (1) and adjusting effector mechanisms to keep dry body temperature constant. Disturbances due to external environmental conditions and internal factors such as changes in body fluid volumes and solute composition which directly relate to the state of hydration may potentially have detrimental effects on thermal control mechanisms. Three clinical problems that

can be presented when exercising in heat are heat cramps, heat exhaustion and heat stroke. Heat stroke, the most severe heat injury, can cause widespread cellular damage in vital organs and skeletal muscles (10,11).

In the present experiment, the rise in body temperature was determined in different environmental conditions and hydration states. The purpose was to assess the effects of time-related changes in plasma volume and changes in the composition of blood, specifically osmolality and sodium concentrations on core temperature during prolonged exercise in heat.

METHODS

Subjects

Six young active adult males volunteered for this study. Each was thoroughly familiarized with the procedures and signed a written informed consent. Preliminary measurements, taken to determine the presence of cardiovascular exclusion criteria included a resting 12-lead electrocardiogram, heart rate and blood pressure. Subsequent pre-experimental testing included the determination of maximal oxygen consumption ($\dot{V}O_2max$) for stationary cycling and body composition; only those with VO max between 40 to 60 $ml \cdot kg^{-1} \cdot min^{-1}$ and hydrostatic estimations of percent body fat between 8 to 20% were

accepted. An habitual physical training level equivalent to running 15,000 to 20,000 m per week also was required. Additionally, the cycling workload vs oxygen consumption ($\dot{V}O_2$) relation for a range or submaximal intensities was determined for each subject. This allowed calculation of the power output necessary for a 50% $\dot{V}O_2$max, the desired exercise intensity for the experimental endurance trials.

Test Protocol

The experimental trials consisted of three different 120 min bouts of stationary cycling with a 30 min preliminary resting period included to establish baseline values and a 30 min recovery to establish return to baseline. The environmental and hydration conditions of each trial were: exercise plus neutral/euhydrated (NE) environmental condition; exercise plus hot/euhydrated (HE) condition, and; a hot/hypohydrated (HH) condition. The NE condition imposed an average ambient temperature of 24 C and a 50% relative humidity. The average ambient temperature for the HE and HH bouts was 35°C with a 50% relative humidity. Euhydration for bouts was achieved by having the subjects drink a volume of water (room temperature) equivalent to 1.0% of his body weight, immediately prior to the preliminary rest period plus an equal amount during the rest period and exercise phase, i.e., a maximum amount of

2.0% of body weight. The HH was with no fluids. At 15 min intervals, environmental conditions and subject's exercise $\dot{V}O_2$ values were monitored and adjustments were made in the heat, humidity and/or workrate to keep these variables constant.

Measurements

Deep body temperature was monitored by a rectal thermocouple inserted 10 to 15 cm into the rectum. Body temperature changes were measured at 15 min intervals by a telethermometer (Yellow Springs, Model TUC 46). Blood samples of approximately 7.0 ml were collected without stasis at 15 min intervals from a 20 gauge teflon-catheter inserted into an antecubital vein. The hematocrit (Hct) was measured in quadruplicate and corrected by a factor of 0.96 for trapped plasma and by 0.93 to adjust for venous to whole body Hct ratio (2). Blood hemoglobin was measured in triplicate with the cyanomethemoglobin method. Relative changes in plasma volume were calculated from the Hct and hemoglobin (Hb) values using the formula of Costill and Fink (2). From each plasma sample, the osmolality was measured by a vapor pressure osmometer, and sodium concentrations were measured by flame photometry.

Statistical Analysis

The data were analyzed to determine the within-trial and between-trial differences in core temperature (Tc), and the hematologic variables of plasma volume (PV), osmolality (OSM) and sodium [Na+] for different environmental conditions and hydration states. Analysis of variance (Table 1) was utilized to determine if significant differences existed across time or between conditions for each of the dependent variables. A Duncan's Multiple Range Test was utilized in order to determine where differences existed within each trial for the dependent variables.

Results

The physical characteristics for each subject are in Table 2. Only one subject was judged to be trained (30 $mi \cdot wk^{-1}$), but all had reported having exercised on a regular basis for several years.

Rectal Temperature (Tr)

During exercise Tr exhibited a gradual rise to equilibrium levels (Figure 1). The overall treatment mean differences for NE, HE, and HH were 37.7, 37.9, and 38.1°C, respectively. During both euhydration conditions (NE and HE), this thermal equilibrium was reached within the first 60 min, however the NE equilibrium was reached at a lower temperature (37.9°C). In the HE condition, the thermal

equilibrium was attained at 90 min ($x=38.2\pm.1^{\circ}C$) and only a mean increase of $0.2^{\circ}C$ occurred over the last 60 min. In the HH condition, no early equilibrium occurred and Tr showed a progressive rise to $38.7^{\circ}C$ the final 30 min. of exercise.

There was a gradual linear increase in Tr during the first 30 min of each trial, but at 45 min the Duncan's Multiple Range Test determined in the NE trial that Tr was significantly different ($p<0.01$) from the HE and HH trials. At 60 min Tr for all three experimental conditions was significantly different ($p<0.01$) and remained significant throughout the 120 min of exercise.

Plasma Volume (PV)

Figure 1 shows that the greatest losses of PV in all experimental conditions occurred during the first 15 min of exercise with at least a (-4.2%) decrease. In the NE trial, PV decreased continuously through the first 60 min with a -7.8% decrement observed at 60 min of (-7.8%). Thereafter PV showed no further systematic change. PV was not affected by treatment ($p>0.05$). Both the HE and HH conditions resulted in a progressive decreases in PV similar to that for NE.

Plasma Osmolality

Plasma osmolality did not vary significantly ($p>0.05$) as a function of time (Figure 2). Plasma osmolality was affected by treatment ($p<0.01$). Mean osmolality was 283.3, 287.8 and 292.5 mOsmol/kg for NE, HE, and HH, respectively.

Sodium Concentration [Na]

Plasma [Na+] during the NE trial exhibited a slow progressive rise throughout the trial until stabilizing in the final two intervals (Figure 2). Mean sodium levels were 135.1, 135.7, and 137.9 meq/l for NE, HE and HH, respectively. The greatest increase occurred during the first 15 min. Plasma [Na+] was significantly different ($p<0.01$) over time. This basic pattern was observed in HE, but from 15 min until the conclusion of the exercise, only small fluctuations were noted. For the HH condition the largest increase in [Na+] again occurred within the first 15 min. The overall treatment mean differences for Na+ were 135.1, 134.8, and 137.9 mEq/L for NE, HE, and HH, respectively. As with plasma osmolality significant differences ($p<0.01$) were found between the two euhydrated conditions (NE and HE) and HH. Through application of a Duncan's Multiple Range test it was determined that this variation was due to treatment differences between the HH and the two euhydration trials (NE and HE). This difference was exhibited throughout the entire 120 min of exercise.

Discussion

Nielsen (21) was the first to find that as deep body temperature increased during exercise to reach a steady plateau which was maintained for the duration of the exercise. That Tc never stabilized at a level equilibrium in the HH condition was attributed to the state of hydration, since the HE trial did stabilize. Pitts et al. (22) was the first to demonstrate that the extent of the Tc increase was affected by the water balance of the subject. The greater the subject's level of dehydration, the greater the increase in body temperature would be; alternately, if the subject was hyperhydrated body temperature was reduced. Such findings also were substantiated by Moroff and Bass (14), Ekblom et al. (4) and Greenleaf and Castle (7) who attributed rising Tc in exercise after hypohydration to reduced sweat rates. Thus, heat loss by evaporation was reduced, and core temperature increased. But when fluids were replaced, the sweat rate was maintained and the evaporative process cooled the body.

The percent decrease in PV during the first 15 min of this study is in total agreement with the findings of Harrison (9) and Greenleaf et al. (8). The PV decrease results from a transcapillary fluid flux into the working musculature, and during prolonged exercise the flux of

fluids is transient and followed by a gradual redistributing of body water (12). During moderate submaximal exercise in the upright position, the loss of PV is influenced by increased hydrostatic pressure and probably systemic blood pressure (5). Miles et al. (13) noted a linear relationship between the amount of PV lost from the vasculature and the exercise intensity (% Peak $\dot{V}O_2$) for both arm and leg exercises. Plasma volume decreased over time, but no differences between experimental condition were noted. The decrease in PV is due to postural changes and exercise, and heat has little effect, if any at all (3).

In the present study, an OSM response to the three different experimental conditions was observed initially and was maintained throughout the 120 min of exercise. Senay and Christensen (26) and Senay (24) reported correlations between the rate of evaporative losses during dehydration and both the plasma OSM and [Na+]. Senay and Christensen (26) also suggested that osmotic pressure could not act as an indicator of changes in body fluid volume. Such a non-thermal indicator along with thermoreceptor inputs might prove a basis for heat regulation via effects at the hypothalamus. Nielsen et al. (20) gave a hypertonic saline solution orally to prevent dehydration and imposed a thermoregulation circumstance comparable to that produced by

dehydration alone. They showed that the effect of the state of hydration on body temperature during work was related to the plasma OSM rather than PV. Snellen and Mitchell (27) gave a hypertonic saline solution and observed a marked depression of sweating. They reported that drinking affected thermoregulatory sweating in three ways: by the temperature of the drink; by a volume effect; and by a salinity effect. Nielsen (18) confirmed these earlier findings that plasma osmolality had a direct effect on temperature regulation by acting peripherally on the sweat glands or centrally on the hypothalamus to shift the "set point."

Nielsen (19) reported that the [Na+] was the most important factor in relationship to OSM. Sodium concentrations in the HH trial were significantly different from the NE and HE experimental condition. Large increases in both OSM and [Na+] of 4.1 mOsmol/kg and .8 mEq/L, respectively, were noted at 45 min. A central effect that [Na+] have on the hypothalamus is that increased [Na+] in the blood determines the firing patterns of neurons within the hypothalamus (15). This [Na+] increase also stimulates the production of the antidiuretic hormone (ADH) and conserves body water at the kidney tubules (Senay & Beaumont, 25). The elevated concentrations effect another

peripheral factor, the sweat glands, causing a decrease in the amount of cutaneous blood bringing water to these glands and helping to conserve body fluids.

Therefore, in the present study plasma [Na+] was the major factor in the increase in core temperature. Our results suggest that hypernatremia exerted a specific effect on the sweating mechanism to decrease the activity of the sweat glands and stimulated the hypothalamus to increase body temperature. Findings by Greenleaf et al. (6) support this hypothesis in that the rise in Tr was related to a 5 mEq/l increase in plasma [Na+]. In the present study during the HH trial, plasma [Na+] increased 4.5 mEq/l compared to 3.1 mEq/l for NE and 2.9 mEq/l for HE. Thus, plasma [Na+] was probably the major factor involved with the increase in Tc. The large increase in plasma [Na+] is sufficient to have a definite effect on the sweat mechanism (16). In the present experiment the decrease in the sensitivity of the sweat gland was not studied, but Neilsen and Greenleaf (17) assumed it was related to decreased activity in the thermoregulatory center in the brain or to a decreased responsiveness in the sweat gland.

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Figure Legends

FIG 1'. Mean core temperature (°C) changes before, during and after 120 min of cycle ergometry at normal (24 C, 50% RH)/euhydration, NE; Hot (35°C, 50% RH)/euhydration, HE; and hot/hypohydration, HH conditions. ◆ denotes time when NE was statistically different ($p < 0.01$) from HE and HH. ◇ denotes times when NE, HE, and HH are all statistically different ($p < 0.01$). SEM = 0.38 for all time intervals in each condition.

Mean change of plasma volume (%) changes before, during and after 120 min of cycle ergometer at normal (24°C, 50% RH)/euhydration, NE; hot (35°C, 50% RH)/euhydration, HE; and hot/hypohydration, HH, conditions. SEM = 1.03 for all time intervals in each condition.

FIG 2. Mean osmolality (mOsmol/kg) changes before, during and after 120 min of cycle ergometry at normal (24° C, 50% RH)/euhydration, NE; hot (35°C, 50% RH)/euhydration, HE; and hot/hypohydration, HH, conditions. SEM = 1.49 at all time intervals in each condition.

Mean Na^+ (meq/L) changes before, during and after 120 min of cycle ergometry at normal (24°C, 50%

RH)/euhydration, NE; hot (35°C, 50% RH)/euhydration, HE; and hot/hypohydration, HH, conditions. ◆ denotes time when HH was statistically different ($p < 0.01$) from NE and HE. SEM = 0.38 for all time intervals in each condition.

Table 1. Analysis of Variance for Core Temperature, Percent Change in Plasma Volume (PV), Osmolality (OSM) and Sodium (Na+) in Subjects Exposed to Heat Stress During Exercise

Source	df	Core Temperature MS	% Change PV MS	OSM MS	NA+ MS
Treatment a	2	2.235	0.82	1119.29	153.18
Time a	8	2.52	116.166	14.235	20.861
Subject b	5	2.722	23.98	367.846	51.27
Subject x Treatment	10	0.505	39.828	113.096	9.954
Treatment x Time c	16	0.156	1.761	18.081	1.224
Subject x Treatment x Time	40	0.036	5.397	12.206	1.507

a tested by treatment x subject

b Subject tested by treatment x subject x time

c treatment x time tested by treatment x subject x time

* = $p < 0.05$

** = $p < 0.01$

Table 2. Subject Characteristics

Subject	Height cm	Weight kg	Body Fat %	$\dot{V}O_2$ max $ml \cdot kg^{-1} \cdot min^{-1}$
1	176.5	82.7	17.8	46.0
2	177.8	70.8	10.4	39.0
3	185.4	89.5	9.1	52.9
4	182.9	56.3	10.5	51.4
5	185.4	86.7	15.5	41.0
6	193.0	85.2	13.0	60.4
$\bar{X}$±SD	183.5±6.0	80.1±13.5	12.8±3.4	48.5±8.0

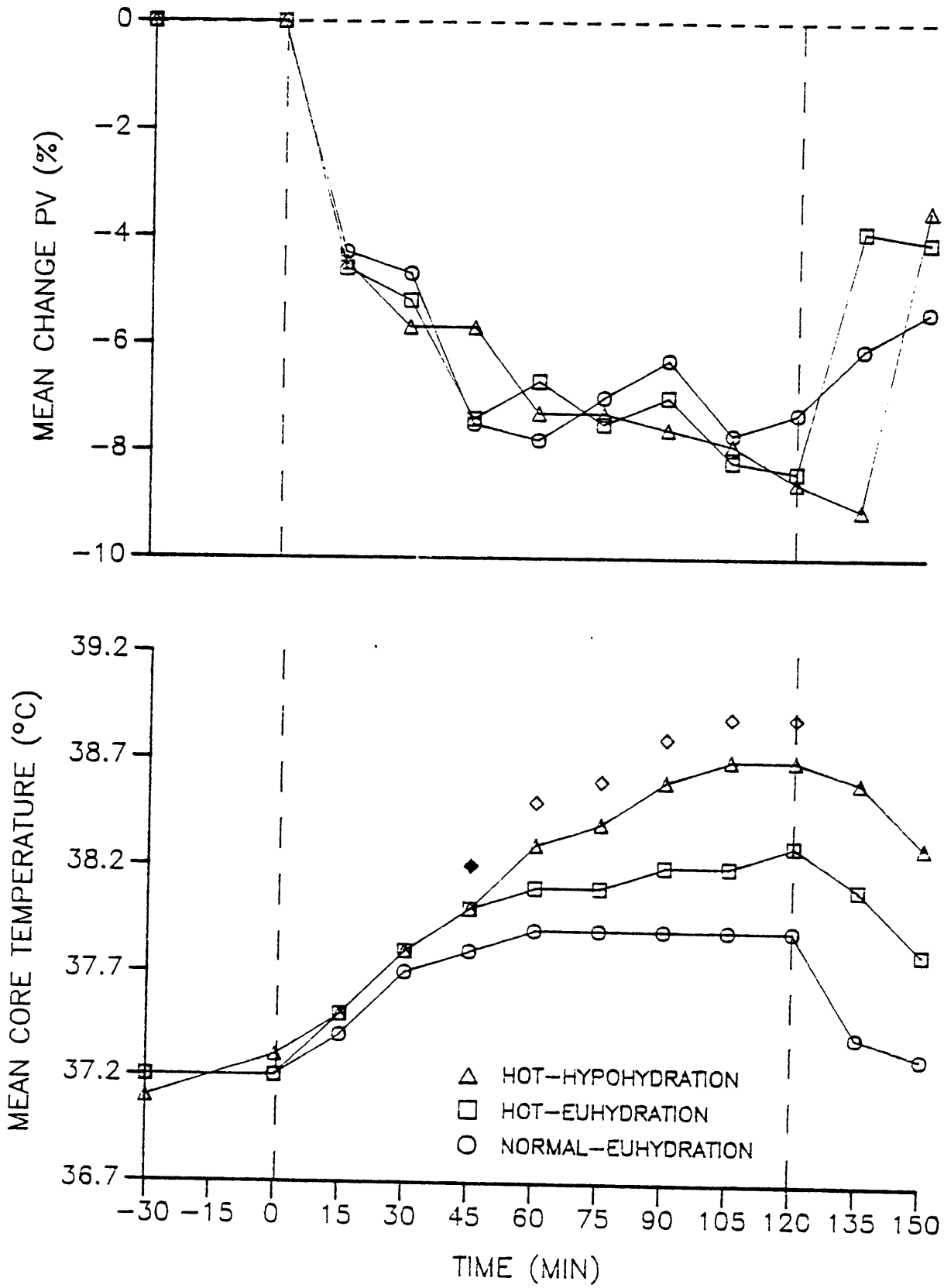
0
-2
-4
-6
-8
-10
MEAN CHANGE PV (%)
39.2
38.7
38.2
37.7
37.2
36.7
MEAN CORE TEMPERATURE (°C)
HOT-HYPOHYDRATION
HOT-EUHYDRATION
NORMAL-EUHYDRATION
-30 -15 0 15 30 45 60 75 90 105 120 135 150
TIME (MIN)

Figure 1'.

Table 3. Core Temperature Responses (°C) a

Trial	-30	0	15	30	45	60	75	90	105	120	$\bar{x}$ e	135	150
NE	37.2	37.2	37.4	37.7	37.8 •	37.9 •	37.9 •	37.9 •	37.9 •	37.9 •	37.7	37.4	37.3
HE	37.2	37.2	37.5	37.8	38.0	38.1 ••	38.1 ••	38.2 ••	38.2 ••	38.3 ••	37.9	38.1	37.8
HH	37.1	37.3	37.5	37.8	38.0	38.3	38.4	38.6	38.7	38.7	38.1	38.6	38.3

a Values represent the means. S.E. = .05

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Means of Treatment Period (0 to 120 min)

Temperatures with different superscripts are statistically different ($p < 0.05$) from temperature with the same time interval.

Table 4. Plasma Volume Responses (%) a

Trial	-30	0	15	30	45	60	75	90	105	120	$\bar{x}$ e	135	150
NE b	0	0	-4.3	-4.7	-7.5	-7.8	-7.0	-6.3	-7.7	-7.3	-5.8	-6.1	-5.4
HE c	0	0	-4.6	-5.2	-7.4	-6.7	-7.5	-7.0	-8.2	-8.4	-6.1	-3.9	-4.1
HH d	0	0	-4.5	-5.7	-5.7	-7.3	-7.3	-7.6	-7.9	-8.6	-6.1	-9.1	-3.5

a Values represent the means. S.E. = 1.03

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Mean of Treatment Period (0 to 120 min)

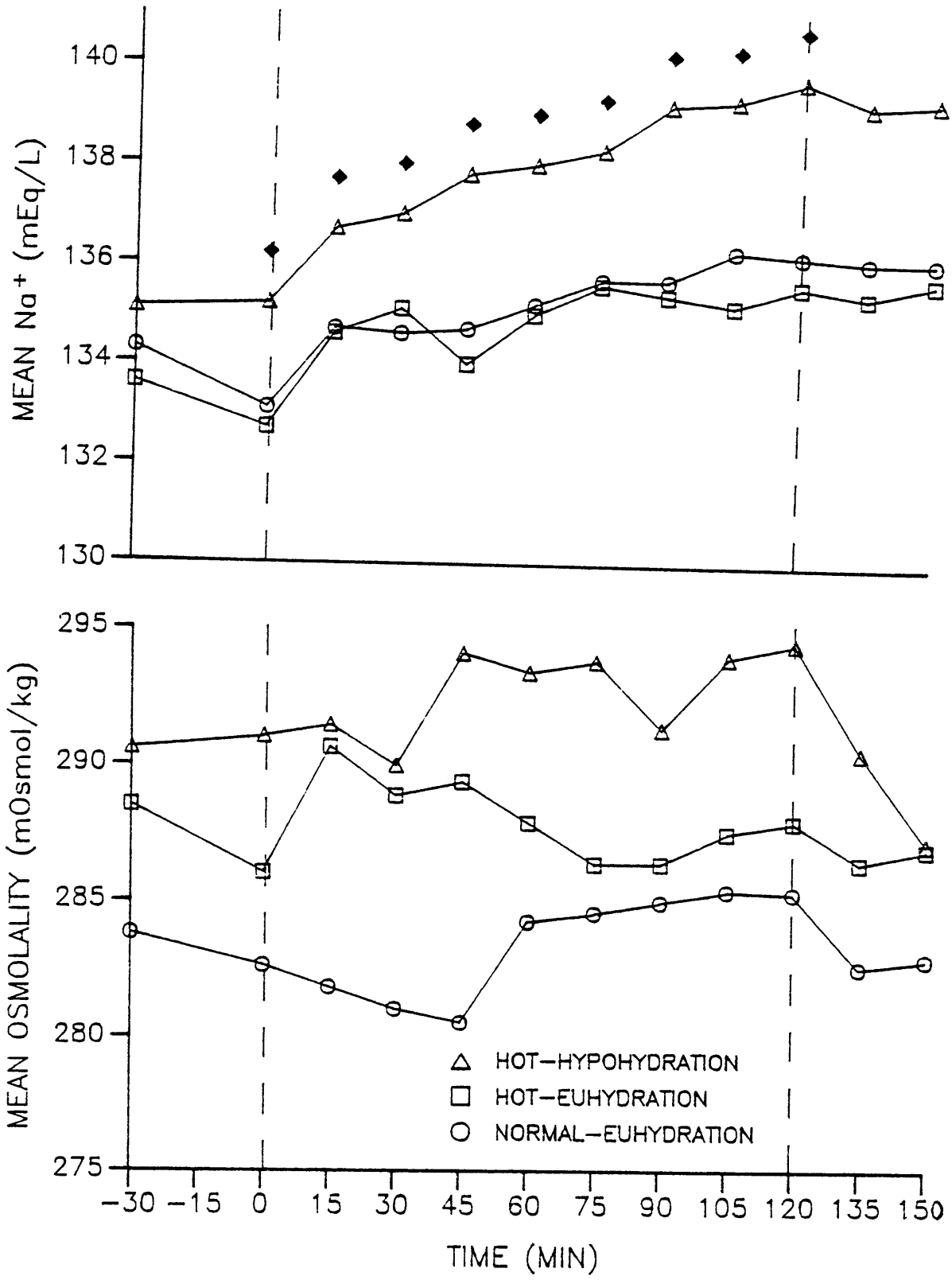
MEAN Na+ (mEq/L)
140
138
136
134
132
130
MEAN OSMOLALITY (mOsmol/kg)
295
290
285
280
275
HOT—HYPOHYDRATION
HOT—EUHYDRATION
NORMAL—EUHYDRATION
−30 −15 0 15 30 45 60 75 90 105 120 135 150
TIME (MIN)

Figure 2.

Table 5. Osmolality Responses (mosmol/kg) a

Trial	-30	0	15	30	45	60	75	90	105	120	$\bar{x}$ e	135	150
NE b	283.8	282.6	281.8	281.0	280.5	284.2	284.5	284.9	285.3	285.2	283.3	282.5	282.8
HE c	288.5	286.0	290.6	288.8	289.3	287.8	286.3	286.3	287.4	287.8	287.8	286.3	286.8
HH d	290.6	291.0	291.4	289.9	294.0	293.3	293.7	291.2	293.8	294.3	292.5	290.3	287.0

a Values represent the means. S.E. = 1.49

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Mean of Treatment Period (0 to 120 min)

Table 6. Sodium Responses (meq/L)

Trial	-30	0	15	30	45	60	75	90	105	120	$\bar{x}$ e	135	150
NE b	134.3	133.1	134.7	134.6	134.7	135.2	135.5	135.7	136.3	136.2	135.1	136.1	136.1
HE c	133.6	132.7	134.6	135.1	134.0	135.0	135.6	135.4	135.2	135.6	134.8	135.4	134.8
HH d	135.1	135.2 *	136.7 *	137.0 *	137.8 *	138.0 *	138.3 *	139.2 *	139.3 *	139.7 *	137.9	139.2	137.9

a Values represent the means. S.E. = 0.38

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Mean of Treatment Period (0 to 120)

* Sodium values with different superscripts are statistically different ($p < 0.05$) from temperature with the same time interval.

Chapter IV

SUMMARY AND RECOMMENDATIONS FOR FUTURE RESEARCH

Prolonged physical exercise in a hot, humid environment can lead to exertional heat stroke. Exertional heat stroke is the most severe form of heat injury. Heat cramps and heat exhaustion are other conditions brought on by heat stress. All of the injuries involve the loss of adequate body fluids and/or electrolytes that produce an inability to dissipate heat, and depending on the severity of the water loss, a wide range of clinical manifestations may occur.

When exertional heat stroke occurs core temperature is continuing to rise without ever reaching an equilibrium level. The factors responsible for the excessive increase in core temperature are varied and even slight variations in the stress placed upon the body can result in unexpected responses, leading to widespread functional and tissue damage or even death to the participant. The problem is understanding the factors which lead to the ineffective control in the hypothalamus, the thermoregulatory center. Speculation has been given that a decline in circulating blood volume, a rise in osmolality, or a large increase in sodium concentration are possible causes for the lack of equilibrium. The role of selected hematological factors (plasma volume, osmolality, and sodium level) is

investigated in an effort to understand the time course rise of core temperature.

The hypothalamus integrates neural stimuli which are received from peripheral receptors in various locations throughout the body. The receptors are controlled by internal and external factors. Most internal receptors are effected by the state of hydration of the body. The level of fluids in the vascular compartment and cell and the concentrations of electrolytes are controlled by the hydration state, and the sweat gland function and skin temperature range also are indirectly involved with the hydration state of the body. The intensity and duration of the exercise is another internal factor which involves the metabolic heat gain within the body and complicates the mode of heat loss. Other factors which can reduce the body's ability to dissipate heat are constitutional factors, including age, sex, body type, exercise capacity, physical training and acclimatization.

External factors which affect peripheral receptors are the environmental conditions including ambient temperature, relative humidity and wind velocity. These factors effect the actual modes of heat loss and can reduce proper function of the mechanism involved such as sweat gland function and skin temperature.

This study was undertaken to observe changes in the hematologic factors and the core temperature responses to prolonged exercise in various conditions of thermal stress and hydration. The changes in PV, osmolality, and [Na+] are related to environmental stress and give a greater understanding of the importance of hydration in a hot environment. Six adult male subjects of similar body composition were selected to participate in this study. All subjects volunteered and signed an informed consent prior to the study. All subjects received a preliminary trial 60 min at 50% $\dot{V}O_2$ max to acquaint them with the test procedures. The study consisted of three experimental conditions with the same protocol for every trial. The control trial consisted of 120 min of cycle ergometry (50% $\dot{V}O_2$ max) under neutral (24°C, 50% RH)/euhydration conditions (NE). The other two conditions were administered in a hot environment (35°C, 50% RH). Fluids were given in the hot/euhydration (HE), and no fluids were given in the hot/hypohydration (HH) trial.

The results of this study showed that Tc was significantly different ($p<0.01$) in the HH trial from the NE and HE trials at 45 min. At 60 min all three trials became significantly different ($p<0.01$) and remained this way throughout the exercise period. Plasma volume in all three

experimental conditions decreased over time but no treatment differences were noted. Osmolality exhibited a treatment difference where the mean osmolality values at the beginning caused an initial difference and did not diverge over time. The results from sodium concentrations [Na+] exhibited a time and treatment effect. The time effect was that over time [Na+] showed an increase and the treatment effect was that in the HH trial [Na+] values were significantly different ($p<0.01$) from the HE and NE trials throughout.

Conclusions

Low correlations did not allow the prediction of a relationship between the dependent variables, but regression analysis gave the ability to determine changes over time for each dependent variable under the three conditions. The effects of hypohydration can be substantial and vary considerably between individuals. Subject variability was the major factor limiting any definite patterns of change within the present study.

Recommendations for Future Research

The results of this investigation suggest certain changes and additional procedures should be included for future research. If a follow up study is to be conducted, I offer several recommendations: a) use a larger sample size; b) add a neutral/hypohydration trial; c) insure the

measurements of all electrolytes in the blood which have been shown to relate to thermal stress.

Due to the small number of subjects, individual variability produced a wide range of responses in each trial. Increasing the number of subjects would give more meaningful physiological responses at each time interval and increase the ability to interpret data in more precise detail. Thus, a larger sample size would provide increased stability and give more specific results. The initial values for plasma osmolality and plasma [Na+] in the HH were significantly different from the NE Trial, thus the inclusion of another trial, the neutral/hypohydration, would allow researchers a greater understanding as to which factor, environmental temperature or hydration, was producing the lack of an equilibrium level in Tc during execise.

Plasma sodium is the greatest contributor to the electrolyte pool involved in the osmolality of blood, but the evaluation of plasma chloride, potassium and total plasma protein would increase the understandings of electrolytes and other solutes in plasma osmolality. The procedural changes in blood collection would be to insure the equipment and supplies the researcher uses the freedom to measure all electrolytes involved in thermal stressful

situations. This would allow a detailed description of changes occurring within the osmolality measurement of the blood during fluctuations, and might demonstrate increases in certain electrolyte values while others are decreasing to prevent changes in osmolality.

Recommendations for Race Sponsors and Runners

The recommendations for individuals that participate in prolonged exercise in a thermal stressful environment are in four areas: 1) training, 2) acclimatization, 3) environmental conditions, and 4) state of hydration. The training an individual is involved in during cold months should gradually be incorporated into a summer program when an individual's exercise capacity for training is 75% $\dot{V}O_2$ max. The first weeks of a hot environment the individual should reduce the strain of high muscle metabolism combined with increased ambient temperature by decreasing the percent $\dot{V}O_2$max. Individuals should allow time for their body's to acclimate, which is accomplished within the first 2 weeks of exposure, and reduce the time involved in the activity to a minimum after acclimatization has occurred. The individuals should never try to push above the training level they have set especially toward the end of an event. Thus, placing excessive strain upon the thermoregulatory system.

In environmental conditions of a high ambient temperature and high relative humidity, the individual should avoid activity and try to train or race during the cooler times of day. Even if ambient temperature or the relative humidity are high, individuals should again try to train before 8 am or after 6 pm.

When discussing heat injuries, probably the most important aspect is the state of hydration. Prior to activity an individual should insure adequate hydration especially of a prolonged nature. During a prolonged event even in a training run, the individual should place water in strategic points along the route. After the exercise, hydration should continue, and a good way to know if proper hydration has occurred is urinating within 15 to 30 min after the exercise. Cool water should be used for this process of replacing fluids but not cold because the gastric absorption level decreases with cold liquids. Proper nutrition and added salt at the table is adequate to replace lost electrolytes. The use of salt tablets should be avoided.

Other populations which should be made aware of the problems of thermal stress include people who are young or over 60, overweight people, and people who are not fit. Through further research, a greater understanding of the

mechanisms and factors involved in thermoregulation will be able to assist in educating the public to the problems involved during thermal stress.

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APPENDIX A

DETAILED METHODOLOGY

METHODOLOGY

Introduction

The data needed to fulfill the objectives for this investigation required three trials per subject. The control for this study was in a thermoneutral environmental, and the other two trials were in a hyperthermic exercise condition. The exercise was performed on a stationary cycle ergometer at a workload equivalent to 50% of the individual's maximum $\dot{V}O_2$ capacity ($\dot{V}O_2$max), and lasted a maximum of 120 min. Data were collected during the pretest period at -30 min and at 0 min. Thereafter data were collected every 15 min even during a 30 min recovery period.

Subject Selection and Screening

From a pool of 15 adult male prospects, six subjects were selected to participate in the study. Prior to participation in the study each subject was required to read and sign an informed consent which explained in detail the procedures necessary for data collection during the testing and explained all the risk possibly involved. Approval from the University Board for the Protection of Human Subjects was obtained prior to any testing. Each subject was given a medical examination consisting of a resting 12 lead electrocardiogram; this was done to screen out any individuals with potential cardiovascular problems. A

functional capacity test ($\dot{V}O_2$ max) to insure an adequate fitness level for the study. A $\dot{V}O_2$ max between 40 to 60 $ml \cdot kg^{-1} \cdot min^{-1}$ was required along with an habitual training level of 15,000 to 30,000 m per week and a percent body fat of 10 to 16%. These criteria insured a homogeneous subject sample.

Preliminary Functional Testing Procedures

Since stationary cycling was the exercise mode of choice for the experimental trials, a Monarch cycle ergometer (Model 868) was used for all $\dot{V}O_2$ max testing. Each subject wore gym shorts, socks, and tennis shoes. Prior to the test, the subjects adjusted the height of the seat to a position where their leg would be slightly bent when the pedal was at the vertical position. After a few minutes of quiet, resting heart rate and blood pressure were taken by auscultation. A careful explanation of the test protocol was given to each subject; each was asked to continue exercise until exhaustion. Each subject was then fitted to a mouthpiece and a Daniel's two-way valve, which was coupled to a 3.81 cm, ID, flexible breathing hose of less than 1 meter in length. The other end of the hose was connected to a Hewlett-Packard Pneumotach (Model A 7303A) which was used to determine the ventilation rate volume ($\dot{V}_e$) in $l \cdot min^{-1}$). The mouthpiece was placed in the mouth and a nose clip was placed on the nose to occlude the nasal passage.

Prior to the first exercise stage, the subject free wheeled the cycle ergometer for 1 min at a rate of 50 rpm. This pedal cadence was maintained by a metronome calibrated to 50 beats•min^{-1}. Immediately after the free wheeling period, 1 kg of resistance, which is the equivalent to a workrate of 300 kpm•min^{-1}, was applied to the wheel of the ergometer. After 8 min the resistance was increased by an increment of 0.5 kg load at 2 min intervals until exhaustion.

At the end of each 2 min interval the oxygen content of the dry expired air was determined by a rapid response Applied Electrochemistry Oxygen Analyzer (Model 5-3A). The carbon dioxide content was also determined by a rapid response Applied Electrochemistry Carbon Dioxide Analyzer (Model CD-3A). At the same time the volume of expired air ($\dot{V}_e$) was measured. The oxygen uptake was calculated for each workload and the highest oxygen uptake was assumed to be the $\dot{V}O_2$max. A work rate equivalent to 50% of the VO_2max was assessed by a regression equation from the linear portion of the oxygen uptake vs work rate, and this workrate was then used as the steady-state exercise requirement for each experimental trial. During the $\dot{V}O_2$max test, a heart rate was taken by auscultation in the last 10 sec of each 2 min interval to insure an accurate maximum heart rate at exhaustion.

Hydrostatic Weighing Procedure

Subjects were asked to refrain from food and drink for 10 hours before the weighing. Upon arrival at the lab subjects were asked to shower with soap to eliminate any body oils which might hold air and eliminate any bodily waste since both could lead to underestimation of body density. Then each subject was weighed nude with a medical balance to determine the body mass. The weighing tank was filled half way with warm water (30-33°C) to minimize subject discomfort. Prior to each test, a routine calibration with a known amount of weight was conducted. With assistance each subject would carefully lower themselves to a sitting position on a rectangular stainless steel basket. At each corner of the basket were load cells which transferred signals to a single channel Speedomax H Azar Recorder for a graphic readout. Each subject then placed a set of scuba weights around his ankles and laid another set in his lap. These "lap" weights were to be moved by the subject to the thoracic area as he reclined and forcibly (maximally) expelled air from their lungs. The subject stayed in the supine position motionless, as to prevent any artifacts on the recorded tracing, for approximately 10 sec. This procedure was repeated a minimum of seven times to allow for learning the technique. An

average of the final two attempts was computed as the actual underwater weight.

Estimation of Percent Body Fat

Approximately the same percent body fat and body surface area were necessary in the subject set in order to minimize differences in heat storage tendencies. Measuring each subject's height (cm) and body weight (kg) enabled investigators to calculate body surface area. Hydrostatic weighing was the most precise technique for estimation of body density. After the body density was calculated, the percent body fat was estimated from the equations below.

Body Density Equation:

$$BD = \frac{Ma}{\left[\frac{Ma-Mw}{Dw}\right] - (RV+VGI)}$$

where BD = body density

Ma = mass in air (grams)

Mw = mass in water (grams)

Dw = density of water due to temperature

RV = residual volume; assumed to be a constant 1300 ml.

VGI= volume of gas in the gastrointestinal tract assumed to be a constant 100 ml.

Equation for Percent (%) Body Fat Using Body Density:

$$\% \text{ BF} = \frac{4.570 - 4.142}{\text{BD}} \times 100$$

where BF = body fat

BD = body density.

Experimental Trial Conditions and Protocol

Prior to the actual study, a practice trial was administered to each subject to relieve anxiety, to minimize any learning effect, and to acquaint the subject with all experimental procedures. The practice trial was conducted over 60 min in a thermo-neutral environment with 50% relative humidity. Blood samples were not collected during the practice trial.

All experimental trials were conducted in a controlled environmental chamber between 0930 and 1300 hs to avoid any diurnal effects or physiological responses, such as upon heart rate. Each subject's trials were randomly ordered and separated by 1 wk periods to avoid any training effect. Also, the testing was conducted in late April and early May to avoid any acclimatization effect. Subjects were required to exercise during the cool part of the day ($<22^{\circ}$C) and limit activity to the criteria for the study. Subjects wore gym shorts, socks and tennis shoes for each trial.

The experimental trials consisted of four phases: 1) preparatory phase, 2) pretest phase, 3) exercise phase, and 4) recovery phase. All subjects in the study were required to be in the Human Performance Lab at least 60 min prior to testing. The first 30 min was the preparatory phase, where subjects were fitted with equipment necessary for measurement of dependent variables. Also during this phase, each subject consumed the equivalent of 1% of his body weight in water of room temperature for the euhydration trials. The next 30 min was considered the pretest phase in which all baseline measurements of dependent variables were obtained, i.e., 30 min and at time 0. During this phase each subject rested in a sitting position on the ergometer within the environmental chamber. At 15 min intervals all experimental measurements were taken, including cardiac output ($\dot{Q}$), heart rate (HR), blood pressure, skin temperatures (Ts; from thermisters), core temperature (Tc; via rectal probe), blood samples and thermograms. At the end of the exercise period each subject was weighed and the recovery phase began. At +15 and +30 min post-exercise, experimental measurements were obtained while the subjects rested on the ergometer.

Experimental Trial Conditions

Each of the experimental trials were conducted in the controlled environmental chamber. The conditions of environment and hydration were as follows:

1. Neutral euhydration. The ambient temperature within the chamber was set at 24°C with a relative humidity 50%. Euhydration was accomplished by having each subject consume 1% his body weight in room temperature water during the preparatory phase and 1% during the exercise phase, for a total of 2% of his body weight.
2. Hot euhydration. The ambient temperature was set at 35°C within the chamber and the relative humidity was 50%. Euhydration was accomplished in the same manner as in neutral euhydration.
3. Hot hypohydration. Ambient temperature again was 35°C and a relative humidity of 50%, but no water was given during any phase of the trial.

Maintaining a Constant Environment and Workload

The environmental conditions and exercise workload were constantly maintained and if fluctuations occurred the proper adjustment was made. Every 15 min the environmental factors of ambient temperature and relative humidity within the chamber were checked.

To insure the proper exercise workload (50% $\dot{V}O_2max$) was maintained, the heart rate (HR) and oxygen uptake measures were checked every 15 min. The HR was monitored by a V configuration of electrodes and displayed on a Quinton Telemetry system. The oxygen uptake was monitored by gas analyzers, which were calibrated before each measurement. If the workload varied, the ergometer was adjusted.

Measurement of Dependent Variables

The core temperature was obtained by a rectal thermocouple self-inserted to a depth of 10 to 16 cm, and all changes were monitored on a Yellow Springs Telethermometer (Model 46 TUC) (Saltin & Hermannsen, 1966).

All blood samples were collected from a 20 guage 3.18 cm teflon catheter inserted in the antecubital vein by a registered nurse. A pharmaseal K52 novex 3-way stopcock with extension tube (2.3 ml) capacity was attached to the catheter. To maintain patency in the catheter, a 0.01 heparinized saline solution was used (every 15 min interval) before the blood sample (7 ml) was drawn. The heparinized solution was discarded and after the blood was taken a new (2.3 ml) amount of heparinized solution was infused into the extension tube and catheter.

Blood samples were obtained by using a 10 ml sterile syringe. From the syringe a small amount was placed on a

clean slide and microcapillary tubes for Hct (4) and Hb (3) were filled from the slide. After the small amount was placed on the slide, the remainder of the blood was slowly ejected from the syringe into a vacutainer tube (10 ml) treated with crystaline potassium-ethylediaminetetraacetate (K+-EDTA) and placed on ice. After each trial the samples were centrifuged in a refrigerator 4 C Sorvall Preparatory centrifuge at 10,000 RPM for 20 min. The plasma was collected for each sample and stored in polypropylene tubes at -20 C for subsequent analysis for osmolality and electrolyte (Na^+ and Cl^-) values.

Hematocrit (Hct) microcapillary tubes were centrifuged and values read (Appendix B), and hemoglobin (Hb) values were obtained (Appendix B). From these values, changes in plasma volume were calculated with formulas by Costill and Fink (1974). The hematocrit was corrected by a factor of 0.96 for trapped plasma and a factor of 0.93 to adjust for venous to whole body Hct ratio.

Osmolality and the electrolyte of Na^+ analyzed for each time interval (Methodology Appendix B). The mean results for all the analyses are in Appendix C.

Statistical Analysis

The data were analyzed to determine the changes in dependent variables of core temperature (Tc), plasma volume

(PV), osmolality (OSM) and sodium concentration [Na^+] in the blood for different environmental conditions and hydration states. An analysis of variance was utilized to determine if significant differences existed between the dependent variables during all three experimental conditions. Instead of computing a separate variance for each trial-treatment combination based on six observations, a combined estimate of variability was computed. An equal variance for the analysis of variance was assumed, thus allowing for a more precise estimate of variance and more degrees of freedom for the tests. A Duncan's Multiple Range Test was utilized in order to determine where differences existed.

APPENDIX B

DETAILED METHODOLOGY OF BLOOD PROCEDURES

PROCEDURES FOR CONTINUOUS VENOUS BLOOD SAMPLING (CATHETERIZATION)

(Adapted from Goss, 1983)

1. Have the subject extend his/her forearm with the palm up. The elbow and the back of the hand should rest on a flat surface.
2. Place a tourniquet securely around the upper arm.
3. Locate antecubetal vein. NOTE: If vein is difficult to locate have the subject alternately clench and relax his/her hand.
4. Cleanse skin around selected vein with Beta-Dyne or alcohol.
5. Insert sterile 20 gauge (5.08 cm length) teflon catheter into antecubital vein.
6. Place a 2.54 cm x 2.54 cm sterile gauze pad under that portion of the catheter extending out from the skin.
7. Remove catheter needle and place sterile catheter extension tubing (25.4 cm) on the distal end of the catheter.
8. Place a sterile 3-way stopcock on the distal end of the catheter extension. NOTE: The catheter extension may be shortened or omitted and the 3-way valve connected directly to the catheter if the subject is to be engaged in stationary cycling or light to moderate treadmill walking.

9. Release tourniquet.
10. Secure catheter by taping the extending portion (distal end) to the skin. NOTE: For maximum stability narrow strips of tape (0.64 cm) should first be carefully wrapped clockwise and then counterclockwise around the catheter projection and then in a similar fashion wrapped around the circumference of the forearm.
11. Secure catheter system by taping the junction of the catheter and extension tubing to the skin. For additional stability 5.08 cm to 7.62 cm of the tubing situated along the longitudinal axis of the forearm should be taped to the skin. For best results tape should reach around circumference of the forearm.
12. Turn stopcock valve to position 2 and attach sterile disposable syringe. Carefully draw blood out of vein through the tubing and valve into the syringe. Switch valve to position 1. Remove and cap syringe.
13. Attach a sterile disposable syringe containing 2 to 3 ml of heparinized saline (2 drops heparin/5 cc saline) and switch valve to position 1. Draw any remaining blood from the blood sampling port. NOTE: If this step is omitted clotting of blood could interfere with subsequent sampling.

14. Without removing syringe switch valve to position 3 and carefully draw a small (less than 1 ml) sample of blood into the syringe. Next, carefully inject heparinized saline solution into the system (i.e., valve tubing and catheter) in order to maintain the potency of the catheter. Switch valve to position 1. Remove and cap syringe.
15. Attach sterile syringe and switch stopcock to position 3. Draw off the heparinized solution plus 1 ml of blood to insure that the subsequent sample is not contaminated by the saline/heparin infusion. Switch stopcock to position 1. Remove and cap syringe.
16. Attach sterile disposable syringe and adjust stopcock to position 2. Draw off the desired volume of blood. Switch valve to position 1 and cap syringe.
17. Rinse system with heparinized saline (see steps 13 and 14).
18. Repeat steps 15 and 16 and then 13 and 14 for each subsequent blood sample.
19. After the final blood sample has been taken, carefully remove the catheter and immediately apply pressure (with a sterile gauze pad) over the puncture area.
20. Place a bandaid on the catheter insertion site. Watch carefully for the development of a subdermal hematoma before dismissing the subject.

Supplies for Blood Sampling Procedure

Item	No. Required	Supplies	Cat. No.	Cost
3-way sterile stopcocks	50	American Sci Products	58965	$.64 ea. 50 for $31.48
catheters	50	B-D		
Sterile extension tubing	50	B-D		
Sterile syringes	2110	American Sci Products		5cc $71.46/500 10cc $76.60/500
Tape	2 bx	Whittaker Med.		
Sterile heparin				
Sterile saline				
Bandaids	50	Whittaker Med.		
Sterile gauze pads	50	Whittaker Med.		
Beta Dyne/alcohol		Whittaker Med.		
Tourniquets	3	Whittaker Med.		
2 needles	1 bx			2/$.57/50

Faye List Equipment

- catheters - 20 gauge teflon

75 - IV set up trays

4 box - alcohol wipes

60 - 3 way stopcock with extension tubing

60 - 50 cc bag with .5% NaCl

- heparin

60 - minidrip chamber set ups

- 5 cc syringes or 3 cc syringes

- 10 cc syringes

18 boxes - heparinized test tubes 10 ml

100 /vial - 20 - 20 ul hemoglobin pipette capillary tubes

500 vial - 4 - hematocrit tubes

- disposable test tubes and caps

DETAILED PROCEDURE FOR HEMATOCRIT (Hct) DETERMINATION

Collect Specimen

1. Put small amount of blood sample on a clean slide (use new slide for each interval).
2. Place the end of a microcapillary tube in the sample. Blood will be drawn up into the tube. Allow the tube to fill at 2/3 full and remove from the sample. (Repeat 3 more times for quadruplicate readings per sample).
3. Wipe off the blood on the external surface of the microcapillary tube with a kleenex being careful not to come in contact with the tip of the tube.
4. After the blood is in the tube, seal the opposite end from where the blood was taken in with a flame.

Centrifuge

5. Place an even number of sealed microcapillary tubes (2 intervals, 4 interval) into a microhematocrit centrifuge (Model MB) for 10 min.
6. Remove microcapillary tubes after centrifuging and place each tube on the Hct stand in the proper interval slot.

Reader

7. While the next 2 intervals of tubes are in the centrifuge, read the microcapillary tube just removed from the centrifuge on a micro-capillary reader (Model CR)

8. Slide the microcapillary tube into the slot underneath the magnifier with the sealed end down and in contact with the edge of the holder.
9. Turn the adjust screw at the base of the holder until the lower edge of the red cells is directly at the edge of the scale.
10. Slide the sample to the right, until the scale arm is at the 100% mark (the plasma meniscus) and tighten the scale arm at this point.
11. Slide the sample back to the left until the scale line intersects with the red cell meniscus. Where the scale line intersects read the Hct in %.

PROCEDURE FOR THE QUANTITATIVE COLORIMETRIC DETERMINATION OF TOTAL HEMOGLOBIN IN WHOLE BLOOD

(from Sigma Technical Bulletin No. 525, 1980)

I. Reagents (for in vitro diagnostic use)

A. Drabkin's Reagent, Stock No. 525-2

1. A dry mixture, consisting of sodium bicarbonate, 100 parts, potassium ferricyanide, 20 parts, and potassium cyanide, 5 parts. Store in dark at room temperature.

B. Drabkin's Solution

1. Reconstitute Drabkin's Reagent, stock No. 525-2 with 100 ml water.
2. Add 0.5 ml 30% Brij-35 solution, stock no. 430 AG-6, and mix well.
3. Filter if insoluble particles remain.
4. Store in amber bottles at room temperature.
5. Stable for at least 6 months.

*Caution: Drabkin's reagent and Drabkin's solution contains cyanide.

C. Brij-35 Solution, Stock No. 430 AG-6

1. Contains Brij-35, 30 gm/100 ml
2. Store at room temperature.

D. Hemoglobin standard, Stock No. 525-18

1. Lyophilized human methemoglobin

2. Equivalent to 18 gm (±1%) hemoglobin per 100 ml whole blood when reconstituted and used according to this procedure.
3. Store in Refrigerator.

E. Cyanmethemoglobin Standard Solution

1. Reconstitute vial of hemoglobin standard, stock no. 525-18 with 50.0 ml Drabkin's solution (Reagent A')
2. Mix well and allow to stand for at least 30 minutes.
3. Stable for at least 6 months when stored in refrigerator at 0-5°C.

II. Instrument and Materials Required

A. Instrument: Any filter photometer or spectrophotometer that transmits light at 540 nm is suitable.

B. Materials

1. Test tubes: 10 ml.
2. Pipets: 20 ml, sahli type or micro, 10 ml, serologic.

III. Calibration.

A. Reconstituting the hemoglobin standard as directed yields the cyanmethemoglobin standard solution (Reagent E). This solution will yield an

absorbance equivalent to that of a whole blood sample containing 18 gm (±1%) hemoglobin per 100 ml which has been diluted 1:251 with Drabkin's solution (Reagent B). Dilutions of the cyanmethemoglobin standard solution with Drabkin's solution are used to prepare a calibration as follows:

1. Prepare working standards by pipeting and mixing thoroughly the solutions indicated below.

1	2	3	4	5
Tube No.	Cyanmethemoglobin Standard Solution Reagent E (ml)	Drabkin's Solution, Reagent B (ml)	Absorbance (A)	Blood Hemoglobin (gm/100 ml)
1	0.0	6.0		0.0
2	2.0	4.0		6.0
3	4.0	2.0		12.0
4	6.0	0.0		18.0

NOTE: Diluted Standards are stable for as long as 6 months when stored tightly capped and refrigerated at 0-5°C in the dark.

2. Read absorbance of tubes 2-4 vs. tube 1 as reference at a wavelength between 530-550 nm.
3. Record absorbance valves in column 4 on chart.
4. Plot a calibration curve of absorbance value (column 4) vs. blood hymoglobin (gm/100 ml) in column 5. The curve is linear, passing through the origin.

NOTE: A curve should be constructed prior to each assay session.

IV. PROCEDURE

A. Label two or more test tubes, blank, test 1, test 2, etc.

B. To all tubes add:

1. 5.0 ml Drabkin's solution (Reagent B)

C. To Test add:

1. 20 ul whole blood, rinsing pipet 3-4 times with reagent.
2. Mix well.
3. Allow to stand at least 15 minutes at room temperature.

D. Place solution from blank, test 1, test 2, etc. in to (4) cuvettes.

E. Place cuvette tray into spectrophotometer.

F. Read and record absorbance (A) of test vs. blank as reference at the same wavelength and in the same instrument as used in the preparation of your calibration curve.

DETAILED PROCEDURE FOR OSMOLALITY DETERMINATION

Steps for the use of a vapor pressure osmometer (Wescor, Inc., Model 5100).

Calibration

1. Process a 290 mmol/kg standard. Set the meter to 290 using the calibrate 290 control.
2. Process a 1000 mmol/kg standard. Locate the instrument reading of the left hand side of the calibration nomograph. Then using the calibration 1000 control adjust the meter to read the corresponding set value on the right hand side of the calibration nomograph.
3. Repeat step 1. Then calibration is complete.

Operational Procedure

4. Pipet an 8 microliter sample of plasma.
5. Place sample on a solute-free paper disc.
6. The sample slide is inserted into the sample chamber and sealed.
7. The next four steps are electronically controlled and the measurement proceeds automatically until final display on the panel meter 90 sec later. Steps electronically controlled are:

a. equilibration and zero set (50 sec)

b. maximum cooling-peltier effect (13 sec)

c. dewpoint convergence (27 sec)

d. end of sequence and readout (total 90 sec)

8. This procedure should be repeated in triplicate per sample taken.

9. The readout is calibrated in units of osmolality, mOsmol/kg.

DETERMINATION OF PERCENT CHANGE IN PLASMA VOLUME

Equations Adopted from Costill and Fink, 1974

$PV_B = 100 - Hct_B$

$PV_A = 100\ (Hb_B/Hb_A) - 100(Hb_B/Hb_A) \times Hct_A$

$\%\ PV = 100\ (PV_A - PV_B)/PV_B$

PV_B = The volume of plasma in 100 ml of blood before exercise

Hct_B = % Hct before exercise

PV_A = The volume of plasma in 100 ml of blood after exercise

Hb_B = Hemoglobin concentration in $g \cdot 100\ ml^{-1}$ before exercise

Hb_A = Hemoglobin concentration in $g \cdot 100\ ml^{-1}$ after exercise

Hct_A = % Hct after exercise

% PV = % change in plasma volume.

DETAILED PROCEDURE FOR SODIUM DETERMINATION

Sodium concentrations were measured on a flame photometer (Model 510).

I. Preparations:

A. Lithium Diluent

1) Dilute 510-022 lithium stock concentrate, 1500 meq Li/l, 100 times with distilled water (final concentration 15 meq/l). Store lithium diluent in plastic container.

B. Calibration Standards

1) Dilute all calibration standards 1:200 with the lithium diluent.

2) Dilute 510-020 flame standard - serium 140 meq Na/l one part to two hundred parts of total solution with the 15 meq Li/l lithium diluent.

C. Samples

1) Dilute 1 part sample to 200 parts of total solution with the 15 meq Li/l lithium diluent.

2) Normal 0.5 ml sample is diluted to 100 ml with lithium diluent.

a) normal concentration = 136-145 meq Na/l.

*Aspirate sample long enough for readings to assume their final value. 30 sec recommended between start and actual reading.

II. Detailed Sodium (Na+) Operation:

A. Fill drain tube.

B. Gas on - power on, get flame on display.

C. Warm-up.

1) Aspiration lithium zero solution for 15 min.

2) Set needle tip 0.16 cm clear of the bottom of the sample cup.

D. Calibrate.

1) Aspirate zero lithium solution. With direct and range Li depressed, adjust lithium zero to 0.00.

2) Aspirate calibrating standard. Adjust fuel flow for a maximum reading. Set Li display to the concentration of the lithium stock standard (1.50 meq/l).

3) Touch up. Zero on zero lithium solution. Calibrate on calibrating standard, at 30 sec.

E. Read unknowns.

1) Aspirate and read at 30 sec.

2) Initially touch up zero and calibration every 4 to 5 samples. After 15 min of operation need for zero and calibration occurs a lot less.

F. Turn off.

1) Aspirate distilled water for a few minutes.

2) Turn off gas. When flame on display goes out turn power off.

APPENDIX C

MEAN DATA TABLES

Table 7. Core Temperature Responses (°C) a

Trial	-30	0	15	30	45	60	75	90	105	120	$\bar{x}$ e	135	150
NE	37.2	37.2	37.4	37.7	37.8 •	37.9 •	37.9 •	37.9 •	37.9 •	37.9 •	37.7	37.4	37.3
HE	37.2	37.2	37.5	37.8	38.0	38.1 ••	38.1 ••	38.2 ••	38.2 ••	38.3 ••	37.9	38.1	37.8
HH	37.1	37.3	37.5	37.8	38.0	38.3	38.4	38.6	38.7	38.7	38.1	38.6	38.3

a Values represent the means. S.E. = .05

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Means of Treatment Period (0 to 120 min)

Temperatures with different superscripts are statistically different ($p < 0.05$) from temperature with the same time interval.

Table 8. Plasma Volume Responses (%) a

Trial	-30	0	15	30	45	60	75	90	105	120	$\tilde{x}$ e	135	150
NE b	0	0	-4.3	-4.7	-7.5	-7.8	-7.0	-6.3	-7.7	-7.3	-5.8	-6.1	-5.4
HE c	0	0	-4.6	-5.2	-7.4	-6.7	-7.5	-7.0	-8.2	-8.4	-6.1	-3.9	-4.1
HH d	0	0	-4.5	-5.7	-5.7	-7.3	-7.3	-7.6	-7.9	-8.6	-6.1	-9.1	-3.5

a Values represent the means. S.E. = 1.03

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Mean of Treatment Period (0 to 120 min)

Table 9. Osmolality Responses (mosmol/kg) a

Trial	-30	0	15	30	45	60	75	90	105	120	$\bar{x}$ e	135	150
NE b	283.8	282.6	281.8	281.0	280.5	284.2	284.5	284.9	285.3	285.2	283.3	282.5	282.8
HE c	288.5	286.0	290.6	288.8	289.3	287.8	286.3	286.3	287.4	287.8	287.8	286.3	286.8
HH d	290.6	291.0	291.4	289.9	294.0	293.3	293.7	291.2	293.8	294.3	292.5	290.3	287.0

a Values represent the means. S.E. = 1.49

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Mean of Treatment Period (0 to 120 min)

Table 10. Sodium Responses (meq/L)

Trial	-30	0	15	30	45	60	75	90	105	120	x e	135	150
NE b	134.3	133.1	134.7	134.6	134.7	135.2	135.5	135.7	136.3	136.2	135.1	136.1	136.1
HE c	133.6	132.7	134.6	135.1	134.0	135.0	135.6	135.4	135.2	135.6	134.8	135.4	134.8
HH d	135.1	135.2 *	136.7 *	137.0 *	137.8 *	138.0 *	138.3 *	139.2 *	139.3 *	139.7 *	137.9	139.2	137.9

a Values represent the means. S.E. = 0.38

b NE = Neutral Euhydration

c HE = Hot Euhydration

d HH = Hot Hypohydration

e Mean of Treatment Period (0 to 120)

* Sodium values with different superscripts are statistically different ($p < 0.05$) from temperature with the same time interval.

APPENDIX D

DATA TABLES

Table 11. Individual Weight Loss (%)

	Trial		
Subject	NE	HE	HH
1	0.0	0.9	2.1
2	0.0	2.1	2.7
3	0.7	1.0	2.7
4	0.8	0.0	2.2
5	0.9	2.9	1.8
6	0.7	1.9	3.8
$\bar{X}$±SD	0.5±0.2	1.5±0.4	2.6±0.3

NE = Neutral Euhydration, HE = Hot Euhydration,
HH = Hot Hypohydration

Table 12. Environmental Conditions of Neutral Euhydration Trials

Subject	Ambient Dry Temperature (°C)	Relative Humidity (%)
1	21.5	49.0
2	27.1	45.4
3	25.1	55.6
4	22.5	56.1
5	23.9	49.0
6	25.0	47.0
$\bar{X}$±SD	24.2±1.9	50.35±4.5

Values represent a mean of 12 observations taken per subject trial.

Table 13. Environmental Conditions of Hot Euhydration Trials

Subject	Ambient Dry Temperature (°C)	Relative Humidity (%)
1	35.6	61.4
2	35.5	46.5
3	33.1	49.0
4	35.5	46.5
5	35.5	49.9
6	33.4	49.2
$\bar{X}$±SD	34.8±0.2	50.5±5.4

Values represent a mean of 12 observations taken per subject trial.

Table 14. Environmental Conditions of Hot Hypohydration Trials

Subject	Ambient Dry Temperature (°C)	Relative Humidity (%)
1	35.9	48.6
2	35.2	51.6
3	34.7	51.0
4	35.0	49.0
5	36.3	50.7
6	35.1	50.3
$\bar{X}$±SD	35.4±0.6	50.2±1.2

Values represent a mean of 12 observations taken per subject trial.

APPENDIX E

RAW DATA TABLES FOR EACH SUBJECT

Table 11. Raw Data for Subject #1 (DG)

Trial	Variable	Time (min) -30	0	15	30	45	60	75	90	105	120	135	150
NE	T_c	37.2	37.2	37.5	37.8	38.0	38.1	38.2	38.2	38.15	38.1	37.6	37.4
	Hct	39.0	39.0	41.1	40.9	42.3	44.1	45.1	45.2	45.0	44.7	44.8	45.1
	Hb	15.1	15.3	15.4	15.4	15.6	15.9	15.8	15.7	16.1	16.3	16.0	15.7
	PV%Δ	-	-	-3.5	-3.2	-6.3	-10.0	-10.7	-10.3	-12.5	-13.0	-11.7	-10.2
	Na+	136.7	134.3	134.9	135.3	136.0	134.6	136.7	134.8	136.5	135.9	136.0	135.9
	Cl-	-	105	-	-	-	102	-	-	-	103	-	-
	OSMO	286.0	283.0	281.0	289.0	286.0	289.0	283.5	284.5	287.0	288.0	283.5	288.5
HE	T_c	37.6	37.7	37.9	38.1	38.3	38.5	38.6	38.7	38.7	38.8	38.2	38.0
	Hct	41.3	41.4	42.0	42.0	42.4	42.4	42.3	42.4	42.4	42.5	42.5	42.0
	Hb	15.3	15.1	15.4	15.5	15.8	15.6	15.5	15.8	15.9	15.5	15.4	15.3
	PV%Δ	-	-	-2.9	-3.4	-6.1	-4.5	-4.0	-6.1	-6.5	-4.2	-3.6	-2.4
	Na+	135.4	135.4	135.2	135.7	134.5	135.9	135.7	135.0	135.3	134.3	135.1	135.7
	Cl-	-	105	-	-	-	105	-	-	-	104	-	-
	OSMO	294.5	291.0	294.0	289.0	292.5	290.5	291.0	294.5	288.5	288.5	292.0	288.0
HH	T_c	37.4	37.5	37.7	37.9	38.2	38.3	38.5	38.6	38.7	38.7	38.4	38.2
	Hct	39.8	41.0	42.8	43.7	43.8	44.2	44.4	44.5	45.1	45.8	43.8	43.1
	Hb	15.1	15.5	15.5	15.9	15.4	15.7	15.7	15.8	15.9	15.7	15.8	15.1
	PV%Δ	-	-	-2.5	-6.1	-3.5	-5.7	-6.2	-6.6	-7.9	-8.0	-5.7	-0.2
	Na+	137.4	134.7	139.1	138.3	138.3	138.5	137.2	138.3	138.8	138.0	139.7	141.2
	Cl-	-	108	-	-	-	110	-	-	-	109	-	-
	OSMO	293.5	304.5	293.0	293.0	296.0	295.5	295.5	290.0	293.5	301.0	292.5	293.0

T_c = core temperature (°C), Hct = hematocrit (%), without correction for trapped plasma, Hb = hemoglobin (gm/100 ml), PV(%Δ) = plasma volume (% change), Na+ = sodium (mEq/l), Cl- = chloride (mEq/l), OSMO = osmolarity (mOsm/l), NE = (normal) euhydration, HE = hot-euhydration, HH = hot-hypohydration

Table 12. Raw Data for Subject #2 (BD)

Trial	Variable	Time (min) -30	0	15	30	45	60	75	90	105	120	135	150
NE	T_c	36.6	36.6	37.05	37.5	37.65	37.8	37.8	37.7	37.6	37.6	37.2	36.9
	Hct	45.6	45.6	45.7	45.4	44.0	45.5	45.1	44.7	44.4	45.3	44.5	45.7
	Hb	14.6	14.6	15.1	14.9	14.9	16.2	15.7	15.9	15.1	15.4	14.9	14.7
	PV%Δ	-	-	-3.7	-2.1	-8.0	-9.7	-6.6	-7.3	-2.0	-4.8	-0.5	-0.8
	Na+	134.7	134.7	134.8	136.3	134.6	134.7	136.4	136.7	137.7	137.3	138.3	139.7
	Cl-	-	107	-	-	-	105	-	-	-	104	-	-
	OSMO	277.0	277.0	274.0	290.0	274.5	282.0	286.0	289.0	285.0	284.0	283.0	289.0
HE	T_c	37.2	37.2	37.5	37.9	38.2	38.3	38.4	38.6	38.7	38.7	38.4	37.9
	Hct	41.3	42.2	41.8	42.7	43.9	45.5	44.8	45.2	45.7	45.3	44.3	45.1
	Hb	14.1	15.0	15.4	14.4	15.0	15.6	15.9	15.3	15.7	15.8	14.7	15.3
	PV%Δ	-	-	-1.8	3.5	-2.4	-8.0	-9.1	-5.7	-8.8	-9.1	-0.9	-5.6
	Na+	130.0	129.9	134.1	134.2	133.1	135.2	134.2	135.9	135.5	134.0	133.1	133.4
	Cl-	-	105	-	-	-	105	-	-	-	103	-	-
	OSMO	285.0	283.0	293.0	292.0	289.0	287.0	287.5	293.0	287.5	293.0	287.5	288.0
HH	T_c	37.1	37.2	37.4	37.8	38.2	38.6	38.8	39.1	39.3	39.4	39.0	38.5
	Hct	44.2	44.0	45.5	45.6	45.4	46.1	46.7	46.4	46.6*	46.5	46.0	45.1
	Hb	16.1	15.7	16.6	16.7	16.1	17.5	16.6	16.6	16.5	16.4	16.2	16.4
	PV%Δ	-	-	-7.5	-8.1	-9.8	-13.0	-9.3	-8.7	-8.3	-8.1	-6.1	-6.0
	Na+	130.8	134.7	136.0	135.5	137.3	136.7	137.8	138.0	138.2	138.5	138.3	138.4
	Cl-	-	104	-	-	-	105	-	-	-	106	-	-
	OSMO	286.5	294.5	290.5	288.0	287.0	286.5	289.0	282.5	288.0	284.5	278.0	282.0

T_c = core temperature (°C), Hct = hematocrit (%), without correction for trapped plasma, Hb = hemoglobin (gm/100 ml), PV(%Δ) = plasma volume (% change), Na+ = sodium (mEq/l), Cl- = chloride (mEq/l), OSMO = osmolarity (mOsm/l), NE = (normal) euhydration, HE = hot-euhydration, HH = hot-hypohydration

* subject stopped at 110 min

Table 13. Raw Data for Subject #3 (TK)

Trial	Variable	Time (min) -30	0	15	30	45	60	75	90	105	120	135	150
NE	T_c	37.4	37.4	37.7	37.8	37.9	37.8	37.8	37.8	37.7	37.6	37.4	37.3
	Hct	42.1	42.5	42.8	44.0	44.6	44.0	43.3	43.8	44.3	44.4	44.7	43.7
	Hb	15.0	15.7	15.7	16.4	16.5	15.9	16.1	16.1	16.2	16.4	16.5	16.0
	PV%Δ	-	-	-0.7	-6.6	-7.8	-3.5	-3.8	-4.5	-5.6	-6.7	-8.6	-3.7
	Na+	133.0	131	135	135	135	137	137	137	137	138	136.4	136.0
	Cl-	-	101	-	-	-	107	-	-	-	104	-	-
	OSMO	282.0	267.0	278.0	274.0	275.0	279.0	282.0	281.0	285.0	282.0	283.5	282.5
HE	T_c	37.2	37.2	37.6	38.0	38.2	38.2	38.3	38.3	38.4	38.2	38.0	37.8
	Hct	41.4	41.6	43.0	43.0	43.2	42.8	42.8	43.4	43.2	43.4	44.2	43.4
	Hb	15.7	15.7	16.5	16.8	16.5	16.0	16.1	15.3	15.9	15.8	16.4	15.5
	PV%Δ	-	-	-6.4	-8.3	-6.9	-3.5	-4.1	-0.3	-3.3	-2.9	-7.4	-1.2
	Na+	135.4	135	136	136	136	136	137	136	135	138	138.0	138.6
	Cl-	-	107	-	-	-	107	-	-	-	109	-	-
	OSMO	285.5	286.0	290.0	284.0	299.0	286.0	284.0	277.0	283.0	284.0	284.0	282.5
HH	T_c	37.2	37.3	37.6	37.8	38.0	38.2	38.4	38.5	38.6	38.6	38.0	37.6
	Hct	42.7	42.8	44.1	44.3	45.7	45.3	45.8	45.9	46.6	45.0	45.0	45.0
	Hb	15.9	16.2	16.7	16.7	17.0	17.0	17.4	16.9	17.3	17.3	16.4	16.4
	PV%Δ	-	-	-4.5	-4.8	-10.6	-8.6	-8.1	-10.5	-8.4	-11.4	-5.1	-4.3
	Na+	137.7	137	137	138	140	138	140	141	141	142	140.8	140.0
	Cl-	-	105	-	-	-	106	-	-	-	106	-	-
	OSMO	291.5	290.0	294.0	294.0	306.0	301.0	300.0	299.0	304.0	302.0	301.0	299.0

T_c = core temperature (°C), Hct = hematocrit (%), without correction for trapped plasma, Hb = hemoglobin (gm/100 ml), PV(%Δ) = plasma volume (% change), Na+ = sodium (mEq/l), Cl- = chloride (mEq/l), OSMO = osmolarity (mOsm/l), NE = (normal) euhydration, HE = hot-euhydration, HH = hot-hypohydration

Table 14. Raw Data for Subject #4 (JW)

Trial	Variable	Time (min) -30	0	15	30	45	60	75	90	105	120	135	150
NE	T_c	37.2	37.2	37.4	37.6	37.7	37.7	37.6	37.6	37.6	37.7	37.4	37.4
	Hct	42.0	42.5	45.0	45.2	44.6	45.0	45.0	44.0	45.3	45.0	42.9	43.6
	Hb	15.8	15.9	17.4	16.8	17.5	16.0	16.8	16.0	16.3	16.5	15.3	15.9
	PV%Δ	--	--	-11.7	-8.9	-12.0	-4.4	-8.8	-2.2	-6.2	-7.3	-2.9	-1.8
	Na+	134.3	135	136	134	136	136	136	136	136	136	135.9	134.4
	Cl-	-	107	-	-	-	105	-	-	-	103	-	-
	OSMO	282.0	288.0	288.0	277.0	277.0	281.0	284.0	288.0	282.0	281.0	283.0	278.0
HE	T_c	36.9	36.9	37.3	37.1	37.4	37.6	37.6	37.7	37.7	37.8	37.6	37.4
	Hct	42.2	42.0	43.0	43.6	44.3	45.2	45.1	45.3	45.4	45.7	43.3	43.3
	Hb	15.2	15.5	15.9	16.0	17.3	16.4	16.9	16.9	16.6	16.6	15.7	16.2
	PV%Δ	-	-	-5.2	-3.9	-13.0	-9.6	-11.9	-12.5	-11.0	-11.1	-3.1	-5.8
	Na+	133.1	131	133	133	131	133	133	134	133	133	133.1	132.9
	Cl-	-	102	-	-	-	101	-	-	-	102	-	-
	OSMO	289.0	288.0	285.0	287.0	287.0	286.0	283.0	281.0	287.0	283.0	279.0	288.5
HH	T_c	36.9	37.1	37.4	37.6	37.8	38.0	38.0	38.0	38.1	38.3	38.1	38.0
	Hct	41.3	42.9	43.6	43.9	44.4	43.7	44.3	44.4	45.0	44.9	42.4	42.4
	Hb	15.5	15.9	16.2	16.4	16.5	16.3	16.6	16.6	16.8	17.1	16.2	16.6
	PV%Δ	-	-	-7.0	-8.8	-10.0	-7.9	-10.2	-10.6	-12.4	-13.5	-6.8	-7.7
	Na+	137.2	135	136	137	138	138	139	138	139	139	140.5	139.9
	Cl-	-	103	-	-	-	108	-	-	-	106	-	-
	OSMO	288.0	286.0	290.0	285.0	287.0	290.0	288.0	289.0	290.0	290.0	287.0	286.0

T_c = core temperature (°C), Hct = hematocrit (%), without correction for trapped plasma, Hb = hemoglobin (gm/100 ml), PV(%Δ) = plasma volume (% change), Na+ = sodium (mEq/l), Cl- = chloride (mEq/l), OSMO = osmolarity (mOsm/l), NE = (normal) euhydration, HE = hot-euhydration, HH = hot-hypohydration

Table 15. Raw Data for Subject #5 (GJ)

Trial	Variable	Time (min) -30	0	15	30	45	60	75	90	105	120	135	150
NE	T_c	37.1	37.1	37.2	37.5	37.9	37.8	37.8	37.9	37.9	37.9	37.4	37.2
	Hct	45.6	45.3	46.2	46.4	46.8	47.1	47.4	47.8	48.2	47.6	47.3	46.8
	Hb	16.1	16.1	17.0	16.9	17.0	17.6	17.0	16.9	17.8	16.9	17.1	16.9
	PV%Δ	-	-	-6.9	-6.7	-7.8	-11.2	-8.8	-8.7	-13.9	-8.5	-9.1	-7.2
	Na+	131.1	132	132	132	132	133	133	133	133	132	131.9	131.8
	Cl-	-	96	-	-	-	98	-	-	-	98	-	-
	OSMO	289.0	280	285	283	280	285	285	283	284	282	291.0	288.0
HE	T_c	36.7	36.6	37.0	37.2	37.3	37.4	37.4	37.5	35.7	37.7	37.6	37.4
	Hct	44.5	44.6	45.9	46.7	46.9	47.3	47.6	47.8	48.4	48.7	45.8	45.7
	Hb	15.8	15.7	16.7	17.1	16.8	16.7	16.8	17.3	17.2	17.3	16.3	16.4
	PV%Δ	-	-	-7.2	-10.7	-10.3	-9.8	-10.3	-13.2	-13.5	-14.3	-5.3	-5.8
	Na+	132.5	131	134	136	134	134	135	134	135	135	135.1	135.0
	Cl-	-	98	-	-	-	102	-	-	-	101	-	-
	OSMO	279.5	281	289	286	288	285	282	278	281	285	232.0	282.0
HH	T_c	33.0	36.8	37.0	37.2	37.4	37.6	37.8	37.9	38.0	38.0	38.0	37.7
	Hct	43.1	42.7	42.8	44.1	44.3	45.7	45.3	45.8	45.9	46.6	46.6	44.7
	Hb	16.5	16.3	16.9	17.1	16.5	16.8	17.0	16.7	16.7	16.6	16.8	16.4
	PV%Δ	-	-	-4.8	-5.8	-3.4	-5.3	-6.3	-7.6	-5.5	-5.5	-5.4	-0.4
	Na+	130.5	134	135	134	134	135	134	137	135	137	135.9	136.1
	Cl-	-	103	-	-	-	101	-	-	-	103	-	-
	OSMO	294.0	282	290	284	287	280	284	287	286	284	285.0	285.5

T_c = core temperature (°C), Hct = hematocrit (%), without correction for trapped plasma, Hb = hemoglobin (gm/100 ml), PV(%Δ) = plasma volume (% change), Na+ = sodium (mEq/l), Cl- = chloride (mEq/l), OSMO = osmolarity (mOsm/l), NE = (normal) euhydration, HE = hot-euhydration, HH = hot-hypohydration

Table 16. Raw Data for Subject #6 (BL)

Trial	Variable	Time (min) -30	0	15	30	45	60	75	90	105	120	135	150
NE	T_c		37.7	37.5	37.8	38.0	38.2	38.2	38.2	38.2	38.2		
	Hct	43.5	43.4	43.7	44.3	44.8	46.0	44.8	44.7	45.0	44.5	43.1	42.3
	Hb	14.6	15.0	14.8	14.9	15.2	15.7	15.3	15.5	15.6	15.3	14.5	13.9
	PV%Δ	-	-	-0.7	-1.1	-3.3	-8.2	-4.2	-4.9	-6.0	-3.8	+3.4	+8.8
	Na+	136.1	132	136	136	134	136	137	137	138	138	137.8	138.3
	Cl-	-	100	-	-	-	103	-	-	-	104	-	-
	OSMO	289	291	285	284	291	289	286	289	280	285	293.0	285.0
HE	T_c	37.8	37.8	37.9	38.2	38.4	38.4	38.4	38.4	38.4	38.4	38.6	38.4
	Hct	41.4	42.4	43.3	43.6	43.6	42.6	43.1	43.2	43.5	43.9	43.0	43.0
	Hb	14.6	14.6	15.3	15.5	15.4	15.3	15.3	15.2	15.4	15.7	15.0	15.1
	PV%Δ	-	-	-5.6	-7.3	-6.6	-4.6	-5.3	-4.8	-6.2	-8.8	-2.9	-3.9
	Na+	135	134	136	135	135	136	139	138	138	138	138.0	138.4
	Cl-	-	102	-	-	-	104	-	-	-	104	-	-
	OSMO	291	288	292	297	290	294	292	297	298	294	293.0	292.0
HH	T_c	37.4	37.6	37.9	38.2	38.6	39.0	39.1	39.2	39.5	-	39.6	39.1
	Hct	40.8	42.8	43.5	43.8	44.2	44.3	44.1	46.6	44.6	-	43.3	43.4
	Hb	14.1	14.4	15.0	14.9	15.1	15.2	15.3	15.4	15.4	-	14.9	14.7
	PV%Δ	-	-	-7.5	-8.1	-4.8	-13.0	-9.3	-8.7	-8.3	-	-4.2	-3.3
	Na+	137	136	137	139	140	140	142	142	144	-	139.9	140.3
	Cl-	-	106	-	-	-	108	-	-	112	-	-	-
	OSMO	290	289	292	296	302	305	305	303	301	-	298.5	290.5

T_c = core temperature (°C), Hct = hematocrit (%), without correction for trapped plasma, Hb = hemoglobin (gm/100 ml), PV(%Δ) = plasma volume (% change), Na+ = sodium (mEq/l), Cl- = chloride (mEq/l), OSMO = osmolarity (mOsm/l), NE = (normal) euhydration, HE = hot-euhydration, HH = hot-hypohydration

APPENDIX F

INFORMED CONSENT

LABORATORY FOR EXERCISE AND WORK PHYSIOLOGY

Division of Health, Physical Education and Recreation
College of Education
Virginia Polytechnic Institute and State University
Blacksburg, VA 24061

INFORMED CONSENT

I, _____, do hereby voluntarily agree and consent to participate in a testing program conducted by the personnel of the Human Performance Laboratory of the Division of Health, Physical Education and Recreation of Virginia Polytechnic Institute and State University.

Title of Study: Effects of Endogenous B-endorphin and Hydration State on Thermoregulatory Mechanisms During Prolonged Exercise in the Heat.

The purposes of this experiment include: 1) to establish a comprehensive model of thermoregulation in man during prolonged work in the heat, integrating the time course changes in body fluids, hemodynamics and heat dissipation, 2) to determine the possible role of the hormone b-endorphin on exercise thermoregulation, 3) to investigate the possible effects of B-endorphin on body fluid balance during prolonged exercise in the heat.

I voluntarily agree to participate in this testing program. It is my understanding that my participation will include:

1. medical screening to determine by suitability for participation in this study,

2. a brief interview concerning by habitual activity,

3. maximal oxygen uptake test performed on a bicycle ergometer,

4. anthropometric measures including percent fat, (underwater weighing) height and weight,

5. a maximum of four trials of prolonged cycling at a workload equivalent to 50% of my maximal oxygen uptake. These exercise sessions will continue for a maximum of 120 minutes. I understand that I am encouraged to

exercise to the end of each trial or to the point of exhaustion.

a. the first trial will be an orientation trial in order to acquaint me with the experimental protocol. It will take place in a cool environment (25°C, 50% relative humidity) and last 60 minutes.

b. A trial (120 min duration) will also take place in a cool environment (25°C, 50% relative humidity). Sweat losses will be replaced with water at 15 minute intervals.

c. Another trial (120 min duration) will take place in a hot environment (35°C, 50% relative humidity). Sweat losses will be replaced with water at 15 minute intervals.

d. The last type of trial (120 min duration) will take place in a hot environment (35°C, 50% relative humidity). Sweat losses will not be replaced.

Note: The order of trials will be randomized.

6. During each of the aforementioned trials the following measures will be taken at periodic intervals.

a. cardiac output: This will involve the rebreathing of a gas mixture containing a small amount of carbon dioxide in oxygen for a period not to exceed 15 seconds.

b. Blood pressure will be measured indirectly via the placement of a cuff on the upper arm.

c. heart rate: Electrocardiographically determined from a series of three electrodes placed upon the chest.

d. core temperature: Determined by a rectal thermocouple self inserted to a depth of approximately 10 cm.

e. perceived exertion: Subject reporting of the perception of effort based upon the Borg Relative Perceived Exertion Scale.

f. blood samples: obtained from a catheter placed in the forearm by a registered nurse.

g. skin temperature patterns: obtained from video camera recordings of infra red radiation emitted from the body, and eight skin thermocouples taped on the body at various anatomical locations.

I understand that participation in this experiment may produce certain discomforts and risks. I understand that with any extended aerobic activity I will probably experience a general feeling of fatigue. In addition, due to the exercise mode (cycling, leg fatigue and leg cramps may occur. Residual muscular soreness may be present following the trials and may persist for several days. During the exercise tests, especially the exhaustive bouts, I understand that there is a remote chance of dizziness and nausea. It is also understood that the region surrounding the catheter insertion site may develop a sub-dermal hematoma (slight swelling due to fluid moving out of the vein into the surrounding area).

Certain personal benefits may be expected from participation in this experiment. As a participant in the subject selection process, I will be informed of the results of the medical screening. If cleared to continue in this endeavor, I will be informed of my body composition and physical work capacity, i.e., fitness level. Additionally, at the completion of the study I will have gained knowledge relative to my capacity to perform extended work in normal and heat stressful surroudnings.

I understand that any data of a personal nature will be held confidential and will be used for research purposes only. I also understand that these data may only be used when not identifiable with me.

I understand that I may abstain from participation in any part of the experiment or withdraw from the experiment should I feel the activities might be injurious to my health. The experimenter may also terminate my participation should he feel the activities might be injurious to my health.

I understand that it is my personal responsibility to advise the researchers of any preexisting medical problem that may affect my participation or of any medical problems that might arise in the course of this experiment and that no medical treatment or compensation is available if injury is suffered as a result of this research. A telephone is available which would be used to call the

community rescue squad for emergency service. A trained team of exercise technologists will administer each test.

I have read the above statements and have had the opportunity to ask questions. I understand that the researchers will, at any time, answer my inquiries concerning the procedures used in this experiment.

Scientific inquiry is indispensable to the advancement of knowledge. Your participation in this experiment provides the investigator the opportunity to conduct meaningful scientific observations designed to improve the safety of exercise and physical work in the heat.

If you would like to receive a copy of the results of this investigation, please indicate this choice by marking in the appropriate space provided below. A copy will then be distributed to you as soon as the results are made available by the investigator. Thank you for making this important contribution.

_____ I request a copy of the results of this study.

Date _____ Time _____a.m./p.m.

Participant Signature _____

Witness _____
HPL Personnel

Project Director Dr. William Herbert Telephone 961-6565

HPER Human Subjects Chairman Dr. Don Sebolt
Telephone 961-5104

Dr. Charles Waring, Chairman, International Review Board for Research Involving Human Subjects. Phone 961-5283.

APPENDIX G

CORRELATION MATRICES

Table 21. Correlation Matrices Between Core Temperature (Tc), Plasma Volume (PV), Osmolality (OSM), and Sodium Na+ in Different Environments and States of Hydration

Normal/Euhydration			
	PV	Na+	Tc
OSM	0.15	0.22	0.56*
PV		0.52*	-0.24
Na+			-0.05
Hot/Euhydration			
	PV	Na+	Tc
OSM	0.29	0.37	0.58*
PV		0.56*	0.68*
Na+			0.53*
Hot/Hypohydration (HH)			
	PV	Na+	Tc
OSM	0.39	0.75*	0.37*
PV		-.53*	-0.55*
Na+			0.63*

Statistically significant correlation coefficient
* ($p < 0.01$)
df = 34

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