National Library
of Canada

Acquisitions and
Bibliographic Services Branch

395 Wellington Street
Ottawa, Ontario
K1A 0N4

Bibliothèque nationale
du Canada

Direction des acquisitions et
des services bibliographiques

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

NOTICE

The quality of this microform is heavily dependent upon the quality of the original thesis submitted for microfilming. Every effort has been made to ensure the highest quality of reproduction possible.

If pages are missing, contact the university which granted the degree.

Some pages may have indistinct print especially if the original pages were typed with a poor typewriter ribbon or if the university sent us an inferior photocopy.

Reproduction in full or in part of this microform is governed by the Canadian Copyright Act, R.S.C. 1970, c. C-30, and subsequent amendments.

AVIS

La qualité de cette microforme dépend grandement de la qualité de la thèse soumise au microfilmage. Nous avons tout fait pour assurer une qualité supérieure de reproduction.

S'il manque des pages, veuillez communiquer avec l'université qui a conféré le grade.

La qualité d'impression de certaines pages peut laisser à désirer, surtout si les pages originales ont été dactylographiées à l'aide d'un ruban usé ou si l'université nous a fait parvenir une photocopie de qualité inférieure.

La reproduction, même partielle, de cette microforme est soumise à la Loi canadienne sur le droit d'auteur, SRC 1970, c. C-30, et ses amendements subséquents.

Canada

The Effect of Water Intake on Post-Exercise Hyperthermia:

Investigating the Implications For Theories of Thermoregulation

By

Jeff Beraznik

In partial fulfillment of the requirements
for the degree of Master of Science
in Human Kinetics

The University of Ottawa
July, 1995

ISBN 0-612-04919-1

Canada

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

ACKNOWLEDGMENTS

The following document was assembled with the help of each of the following individuals in one way or another and I would like to thank them for their contributions:

Dr. J. Thoden - Thank you for sharing your knowledge and expertise and providing constant encouragement. I am sincerely grateful for this opportunity and the tailor-made supervision which few others would have given.

Dr. M. Jette, Dr. G. Robertson, and Dr. F. Reardon - Thank you for acting as my committee members and providing valuable criticisms and suggestions to my work.

Dr. G. Kenny - Thank you for your help in introducing me to all of the details of the thermoregulation laboratory.

Dr. M. Fortier - My struggles with the statistical analyses were made much easier with your help.

Dan MacLennan - Your assistance in the laboratory ensured consistent and smooth data collection which I could not have done on my own.

I appreciate the technical assistance of Mr. Andre Gadouais, Mr. Michael Marcoux, and Mr. Roland Serrat. I would also like to acknowledge the participation of Bill, Chris, Dan, Darren, Duane, Francois, Glen, Leon, and Luc who endured the demanding conditions of the testing protocol.

Lastly, I would like to mention a special thanks to my family for providing continued support throughout my studies and to Laur for not only helping out with the analyses of the results and the editing of the document, but for always being there.

CONTENTS

LIST OF TABLES

LIST OF FIGURES

ARTICLE

The Effect of Water Intake on Post-Exercise Hyperthermia:

Investigating the Implications For Theories of Thermoregulation

by

Jeff Beraznik

School of Human Kinetics
Faculty of Health Sciences
University of Ottawa
125 University
Ottawa, Ontario K1N 6N5

ABSTRACT

The purpose of this investigation was to examine the role of water intake on post-exercise hyperthermia. Ten active, non-competitive male volunteers (mean age 22.8 $\pm$ 2.859) performed twenty minute treadmill exercise at equal relative intensities during two separate trials where (1) gradual hypohydration took place, and (2) euhydration was induced by water ingestion in order to compensate for the body water volume loss demonstrated in the hypohydrated condition. Core temperature, represented by both esophageal and rectal temperatures, was recorded, along with surface temperatures (forearm, finger, chest thigh, and calf) and heart rate during a twenty-one minute adaptation period, throughout exercise and for forty minutes of recovery. Hematocrit samples were taken immediately prior to and following the exercise protocol and subject weights were measured before and after the testing session. Data were analyzed using a two-way repeated measure analysis of variance (ANOVA) and Pearson product-moment correlation coefficient procedures. The interaction between trials and time for esophageal ($p=.3035$) and rectal ($p=.4389$) temperatures and the difference between the hypohydration and euhydration trials for esophageal ($p=.2438$) and rectal ($p=.9114$) temperatures were all non-significant, and demonstrated that core temperature values did not differ between the hypohydrated and euhydrated conditions. However, core temperature was significantly different across time for both esophageal ($p=.0001$) and rectal ($p<.0001$) temperatures, due to a post-exercise elevated plateau from pre-exercise values. This elevation corresponded to the core temperature at which skin vessel dilation occurred during exercise for both trials. The correlation between the difference in the hypohydration and euhydration esophageal elevations and the absolute ($r=.453$) and relative percentage ($r=.334$) weight loss of each subject after hypohydration further demonstrated a weak role for water intake in post-exercise hyperthermia. Several hypotheses are proposed to explain the post-exercise core temperature elevations in both the hypohydrated and euhydrated trials and the lack of significant difference between the two trials. It is possible that blood flow redistribution during exercise recovery may not favor cutaneous vasodilation, because the fall in cardiac output together with dilation in previous active tissue causes blood pressure to drop. This may better explain the post-exercise elevation than hypohydration alone. It was also suggested that changes in skin temperatures (forearm-finger) during exercise may not be sufficient by themselves to identify the core temperature threshold for skin vessel dilation for all subjects under the conditions of the present investigation.

Key words: hypohydration, core and surface temperatures, cutaneous blood flow, sweat output, baroreceptors, hematocrit.

INTRODUCTION

During exercise, competition exists between the demands for blood flow to support both thermoregulation and increased metabolic rate. Exercise requires blood in order to transport oxygen and nutrients to working muscle. On the other hand, proper thermoregulatory homeostasis requires blood flow as a means for transporting heat from the core to the skin in order to match the heat production caused by exercise (Johnson, 1992). During exercise, blood flow to working muscle is of primary importance and cutaneous vasoconstriction produces redistribution of blood that reduces thermoregulatory efficiency and results in core temperature elevation (Fortney et al., 1981; Rowell, 1974). As exercise ends and recovery begins, the competition for blood flow is terminated. According to "set-point" theorists, reducing the metabolic rate should allow core temperature to return to pre-exercise levels because of the large deviation between post-exercise core temperature and "set-point" and the redistribution of blood toward the skin as the competition with muscle is ended (Sawka and Wenger, 1988; Brengelmann et. al., 1977). Most theories of thermoregulation are based on this "set-point" concept and refer to the load-error principle which states that heat dissipating responses will remain active until heat production is matched by heat loss (Sawka and Wenger, 1988).

While popular theory predicts the drop of core temperature to pre-exercising values as exercise ends, more recent investigations have studied the possibility of an inoperative range whereby thermoregulatory processes are shut off (Thoden et al., 1994; Mekjavic et. al., 1991; Jessen & Ludwig, 1971) and post-exercise core temperature plateaus whereby recovery to "set-point" is not demonstrated (Thoden et al., 1994). These authors have all demonstrated that a passive range or "null zone" exists in which no attempt is made to drive core temperature towards pre-exercising "set-point".

The demonstration of a post-exercise inoperative temperature regulating system when the demands of activity are no longer present suggests that "set-point" theory may not fully explain thermoregulatory functioning. It is presumed however, that "set-point" theorists and supporters of the load error theory would suggest that the demonstration of an inactive thermoregulatory band is a result of an exercise related response which causes effector responses to become inefficient, rather than inoperative as suggested above (Sawka and Wenger, 1988).

Perhaps if the exercise related response was controlled for, the core temperature outcome would be predicted by "set-point" theory and core temperature would return to pre-exercise levels.

Sweating is an exercise related response that occurs at the expense of both intracellular and extracelluar fluid volumes (Murray, 1992) and will inevitably result in a state of hypohydration (progressive dehydration) (Noakes, 1993). Furthermore, an exercise induced increase in heat production requires that part of the cardiac output be diverted to the skin at a time when body water volume is decreasing from sweat loss. This results in a decrease in central blood volume, and an associated drop in blood flow to the surface; a condition that is not conducive to producing heat loss.

Perhaps the demonstration of post-exercise hyperthermia is simply a result of excessive sweating resulting in inadequate levels of body water and an associated drop in blood volume necessary for active vasodilation. It has long been recognized that hydration has a significant influence upon the body's ability to regulate core temperature during exercise (Montain and Coyle, 1992; Deschamps, 1989; Fortney et al., 1981; Nadel et. al., 1980). At least that is one explanation that interpretations of load error theory of thermoregulation would suggest. On the other hand, perhaps maintaining body water volume through water intake would have no effect on post-exercise hyperthermia as the extended elevation may be a result of another exercise related response or an indication that temperature regulation is passive within a certain range.

These uncertainties were addressed by the present study which investigated the role of water intake on post-exercise hyperthermia and its implications regarding the "set-point" and "null-zone" theories of temperature regulation.

The hypothesis was based on the rationale that a thermoregulatory "null-zone" exists whereby heat dissipating (sweating) and heat conserving (shivering) responses do not coincide when plotted on the same axis (Mekjavic et al., 1991). Moreover, the level at which core temperature plateaus during post-exercise corresponds to the core temperature at which skin vessel dilation occurs during exercise (Thoden et. al., 1994). These two observations suggest that: thermoregulatory processes are inactive once post-exercise core temperature reaches the level of vessel dilation; and that increasing body water volume would have no effect on thermoregulatory functioning. In other words, it is possible that post-exercise hyperthermia is a result of an inactive thermoregulatory system, as opposed to some exercise related response, such

as hypohydration (progressive dehydration) caused by excessive sweat output. Based upon the results of Thoden et al. (1994) and Mekjavic et al. (1991), it was hypothesized that maintaining body water volume through water intake would not attenuate post-exercise hyperthermia.

In an effort to investigate the mechanisms responsible for an elevated post-exercise core temperature, this study examined the effect of maintaining body water volume and its effect on post exercise core temperature elevation as assessed by the time-course of rectal and esophageal temperatures, various skin temperatures (forearm, finger, chest, thigh, calf), and the cardiopulmonary variables of heart rate and oxygen consumption.

The objectives of the present study were to:

(1) investigate post-exercise esophageal and rectal temperature responses following exercise with and without water ingestion;

(2) investigate body weight loss and hematocrit values both prior to and following exercise with and without water ingestion and the association with the degree of post-exercise core temperature elevation.

The relevance of the study can be demonstrated from two separate domains. First, from a basic research point of view, the present study will contribute to the evolving knowledge of thermoregulation and will help to understand the mechanisms responsible for the maintenance of core temperature. Second, from an applied point of view, the present study will help to identify factors that limit human performance when exercise demands are in competition with thermoregulatory demands. More specifically, the study will help determine how water replacement affects human performance and thermoregulatory functioning during physical stress.

The results of the present study can not be generalized to the general population as the subject population was restricted to male university students who were active, but not training competitively and who were familiar with the exercise protocol. As well, it is not possible to generalize the results of the present investigation to women because of hormonal changes that take place during their menstrual cycle, and which influence changes to core temperature.

METHODOLOGY

The present study consisted of one preliminary and two experimental sessions conducted on three separate days. Post-treadmill exercise core and surface temperatures were examined for two separate trials: (1) hypohydrated condition in which fluid replacement was not employed; and (2) euhydrated condition in which water ingestion was used to compensate for the sweating volume loss demonstrated in the hypohydrated condition.

Subjects

Ten male university students, who were physically active, but not regularly engaging in competitive athletics, acted as the subjects for the present study. All subjects gave appropriate Physical Activity Readiness Questionnaire (Par-Q) responses and read and signed the Informed Consent Form prior to participation in the study (Appendix C). The study received approval from the Human Research Ethics Committee of the University of Ottawa (Appendix D).

Instrumentation

The predicted maximal oxygen consumption test and subsequent testing were performed using a Quinton Uniwork treadmill ergometer model 844. Automated gas analysis was recorded by a Quinton Q-plex model 1 and heart rate was monitored with a cardiotachometer (Polar model PE 3000). A Collins, Hans-Rudolph model 2700 two-way breathing valve, along with a Warren E. Collins rubberized mouth piece and nose clip were used to ensure accurate gas collection.

Core temperature was represented by both esophageal and rectal temperatures. Esophageal temperature was measured at the level of the left atrium with a Baxter model 400 catheter, inserted at the nares to a depth equal to one fourth of the subjects height, while rectal temperature was monitored using a YSI model 401 catheter inserted 10 to 15 centimeters into the anus (Thoden et al., 1994). Skin temperatures were measured with YSI model 44004 thermistors taped to the forearm, finger, calf, thigh, and chest using 3M Transpore adhesive tape. Both core

and surface temperatures were collected and digitized at five second intervals using a HP model PE 3000 and were graphed using Microsoft Excel on a standard 486 IBM compatible computer.

Blood sampling was performed with Monolet sterile lancets attached to a Monojector lancet device. The blood samples were collected in 75mm Fisherbrand microhematocrit capillary tubes and were spun using an Adams Autocrit centrifuge. Middle fingers were prepared prior to and following sampling with Professional Disposables alcohol preparation pads and standard drying tissue. Elastoplast fabric strips were used to cover the area of sampling after blood withdrawal.

Other instrumentation included a Fisherbrand thermometer to record ambient temperature, a Taylor hygrometer to measure relative humidity, an Eberbach barometric pressure device to gauge barometric pressure, a Detecto-Medic scale to determine subject weights and heights, a Wedge Innovations Pro-Smart leveling device to determine the percent incline of the treadmill, and a Cronus stopwatch to keep accurate timing of the testing protocol.

Procedures

Preliminary Session

The preliminary session involved informing all subjects individually as to the nature of the testing and their involvement in the study. The subjects read and signed the Informed Consent Form, completed the Par-Q, and were given a Letter of Information to read at their own convenience (Appendix D). Resting heart rate was then determined after ten minutes of supine rest, while predicted maximum heart rate was calculated by subtracting their age from 220. The subjects' height and weight were then taken followed by the administering of the predicted maximal oxygen consumption protocol (Appendix E). Subjects performed treadmill exercise at a self-determined (comfortable) speed while the grade of the treadmill was elevated by one percent every two minutes until their heart rate matched or exceeded their target heart rate (THR) as determined through a THR formula (Appendix E). The speed and grade at which each subject reached their THR were used during both experimental sessions. Lastly, subjects were introduced

to the process of inserting the core temperature probes in order to avoid complications during the experimental sessions.

Experimental Sessions

The experimental sessions consisted of two trials of testing separated by no more than 72 hours and no less than 48 hours, all performed as early in the subjects' day as possible so that they would enter the protocol without significant cardiovascular or thermal stress. Both sessions or trials were preceded by a non-active twenty-four hour period in which subjects were instructed to sleep approximately eight hours, eat a high carbohydrate meal (i.e. pasta) the night before testing, ingest five hundred milliliters of water between two hours and one hour prior to exercising, and eliminate their wastes prior to the start of the protocol, all in order to ensure stable levels of hydration and core temperatures at the onset of testing. Subjects, wearing only shorts were then weighed and then preceded to insert the core temperature probes. Barometric pressure, relative humidity, and ambient temperature were all calculated while the Quinton Q-plex 1 gas analyzer was calibrated. All surface probes (forearm, finger, calf, thigh, and chest) were attached along with the heart rate monitor and the subjects then began a twenty-one minute or greater habituation period that consisted of sitting/leaning in the first part and standing during the last ten minutes. Blood samples were taken from the non-dominant middle finger in order to determine pre-exercise hematocrit values, during the sitting/leaning phase, while a base-level oxygen consumption sample was collected for three minutes during the standing phase. Subjects then performed twenty minutes of treadmill exercise, wearing shorts, socks, and running shoes at an intensity determined during the preliminary session (Appendix E) in an ambient temperature between 24°C and 29°C. This exercise protocol was used because post-exercise hyperthermia has been previously demonstrated during approximately twenty minutes of treadmill exercise and is further believed to be just about the maximum intensity to ensure complete thermoregulatory responses and exercise completion by all subjects (Thoden et al., 1994). A second oxygen consumption collection was acquired during the last five minutes of exercise, again for a duration of three minutes. At the end of the treadmill exercise subjects were lightly towel dried while beginning their forty minute sitting/leaning recovery. A second blood sample was taken at two

minutes post-exercise, followed by a third oxygen consumption collection at fifteen minutes post-exercise, again for three minutes. At the end of recovery, data collection was terminated and all surface and core temperature probes, along with the heart rate monitor, shoes, and socks were removed and subjects were weighed for weight loss.

The procedures as described above took place during both Trial I, where no water was administered (hypohydration), and the water ingesting Trial II (euhydration). In addition, in order to achieve a state of euhydration, during Trial II only, water was ingested at four equal intervals, five minutes prior to exercise and during the tenth, twelfth, and fourteenth minutes of exercise in the amount equal to the weight lost in Trial I, converted to liters of water (Appendix G). The threshold for skin vessel dilation was determined by plotting the forearm minus finger data; the point at which it began to drop steeply was then identified as the threshold.

Statistical Analysis

Data analyses were conducted from the core temperature data collected prior to, during, and after exercise to determine if post-exercise core temperature was significantly different from pre-exercise values and to determine if the core temperature at which skin vessel dilation occurred, corresponded to the post-exercise core temperature plateau, for both trials of hypohydration and euhydration. This was established by performing separate two-way repeated measure analysis of variance (ANOVA) procedures for esophageal and rectal data. The independent variables were the level of hydration and time of measurement, while the dependent variables were esophageal and rectal temperature. The two trials and times of core temperature measurement served as repeated measures or within factors, as the same subjects were tested in both trials and were measured across time.

Further analyses were conducted from the weight loss and esophageal data collected throughout the testing protocol, to determine the degree of correlation between the individual weight loss in Trial I and the difference between the esophageal temperature elevations between Trial I and Trial II. This was established by performing a Pearson correlation coefficient between these two factors.

The first step in conducting the two-way ANOVA procedures as described above was performing a one-way ANOVA procedure between several post-exercise temperatures for both esophageal and rectal data in order to group data for the two-way procedures. Eight post-exercise values (POST1, POST2, POST3,...POST8) at equal five minute intervals for both the esophageal and rectal data were compared to test if they were statistically different. The independent variable was the time of core temperature measurement, while the dependent variables were esophageal and rectal temperature. Only after establishing the grouping of the post-exercise data through the one-way ANOVA, were five esophageal and rectal temperature measurements compared for both Trial I and Trial II using two separate two-way ANOVA procedures. These five measurements included a pre-exercise value (PRE), a skin vessel dilation value (DIL), and three separate post-exercise values (POST, POST7, and POST8). The post-exercise values were grouped according to five minute post-exercise measurements which were statistically similiar. The first six, five minute post-exercise measurements (30 minute average) made up the group POST, the seventh five minute post-exercise measurement made up the group POST7 and the eighth five minute post-exercise measurement made up the group POST8.

All data were reported as mean values and standard deviations. Because the temperature data were collected at five second intervals with random fluctuation constantly occurring, the data points used in the statistical analyses were actually seven point averages of the specific times used, consisting of three points before the specific time utilized (fifteen seconds) and three points after.

Statistical analyses were performed using the Biomedical Data Processing (BMDP) statistical package (1990) on the mainframe computer at the University of Ottawa. Scheffe's post hoc analysis procedure was performed to localize where the differences existed among the compared means. The level of significance for all analyses was 0.05.

RESULTS

The mean and standard deviation of age and physical characteristics of each subject are shown in Table 1. The mean age for the ten subjects was 22.8 ($\pm$2.859) years; mean height, 177.24 ($\pm$6.109) cm.; mean weight, 172.95 ($\pm$7.835) lbs.

The mean resting heart rate for the subjects was 60.1 (±9.620) bpm; mean target heart rate, 162.9 (±3.078) bpm; mean hematocrit pre-exercise Trial 1, 46.03 (±3.01) %; mean hematocrit post-exercise Trial 1, 47.75 (±2.456) %; mean hematocrit pre-exercise Trial II, 45.53 (±3.313) %; mean hematocrit post-exercise Trial II, 46.6 (±3.438) %; mean weight loss from Trial I (hypohydrated), 1.5 (±.31) lbs.; and mean weight loss from Trial II (euhydrated), 0.0 (±.176) lbs. (Table 2).

Esophageal Temperature

The mean and standard deviation of esophageal temperatures prior to exercise [PRE] (M=36.775, SD=.262), at skin vessel dilation [DIL] (M=37.266, SD=.604), during the first post-exercise measurement [POST] (M=37.158, SD=.244), during the second post-exercise measurement [POST7] (M=37.068, SD=.268), and during the last post-exercise measurement [POST8] (M=37.026, SD=.281), were compared along with the mean esophageal temperature within Trial I (M=37.126, SD=.446) and Trial II (M=36.991, SD=.310), using a two-way repeated measure ANOVA procedure (Table 3).

A plot of the mean esophageal data (Figure 1) demonstrated that in both trials, esophageal temperature immediately fell at the start of exercise, but within approximately two minutes began rising at a rapid pace. The temperature rose at a constant rate until approximately seven minutes into exercise when the rate of rise in Trial I decreased to a lower, but still positive rate. This apparent change in slope occurred approximately two minutes after skin vessel dilation occurred. Although the rate of rise in Trial II is not detectable because of water ingestion interference with temperature recordings, it is assumed that the slope would have followed a pattern similiar to that of Trial I if no interference had existed. This assumption is based on observations that similiar responses exist between trials conducted with drinking earlier and later in the protocol. Esophageal temperature began to fall rapidly at approximately one minute post-exercise for a period of five minutes, when it reached a plateau which was maintained throughout the remainder of recovery. The sharp drops in esophageal temperature, during Trial II, occurring at five minutes pre-exercise and at ten, twelve, and fourteen minutes into exercise represent water ingestion. The mean esophageal data is representative of all subjects except for GK who graphically

demonstrated a virtually complete recovery during Trial II (Figure 2). In addition, the individual esophageal data with corresponding skin temperatures and determination of the onset of vasodilation of a single typical subject is presented in Figure 3.

The ANOVA procedure showed that the interaction between trials and time ($F_{4, 36}$=1.26, p=.3035) and the difference between Trial I, during which no fluid was administered, and the water ingesting Trial II, ($F_{1,9}$=1.56, p=.2438) were both non-significant. However, the main effects showed that esophageal temperature was significantly different across time ($F_{4, 36}$=8.02, p=.0001). The Scheffe procedure indicated the following pairs of means differed significantly: PRE (M=36.775, SD=.262) and DIL (M=37.266, SD=.604), PRE and POST (M=37.158, SD=.244), as well as PRE and POST7 (M=37.068, SD=.268). Furthermore, these results revealed that although an extended elevated post-exercise plateau existed compared to pre-exercise levels, the plateau was without a statistically identifiable difference from the esophageal temperature at which skin vessel dilation occurred during exercise ($F_{3, 27}$=2.38, p=.0915) (Table 4).

In addition, the Pearson correlation coefficient showed that the individual differences between the esophageal temperature of each subject (Appendix F), prior to exercise (PRE) and during the first post-exercise measurement (POST) for both trials, were not highly associated with both the absolute and relative percent weight loss of each subject (Table 5). This result was based on the finding that the correlation coefficients between the difference in esophageal elevation between Trial I and Trial II and the absolute (r = .453) and relative percentage (r = .334) weight loss of each subject during Trial I were not strong (Table 6). This indicates that absolute weight loss accounts for 20.5 % (r^2 = .2052) of the variance in the difference between Trial I and Trial II post-exercise esophageal temperature, while relative weight loss accounts for 11.2 % (r^2 = .1115) of this variance. These results suggest that the amount of water intake during Trial II, representing the amount of weight loss in Trial I, is not highly associated with the degree of post-exercise esophageal temperature depression from Trial I to Trial II.

Rectal Temperature

The mean and standard deviation of rectal temperature prior to exercise [PRE] (M=37.130, SD=.250), at skin vessel dilation [DIL] (M=37.229, SD=.260), during the first post-exercise measurement [POST] (M=37.925, SD=.331), during the second post-exercise measurement [POST7] (M=37.628, SD=.363), and during the last post-exercise measurement [POST8] (M=37.548, SD=.342), were compared along with the mean rectal temperature within trial one (M=37.497, SD= .415) and trial two (M=37.487, SD=.429), using a two-way repeated measure ANOVA procedure (Table 7).

A plot of the mean rectal data (Figure 4) demonstrated a slow progressive increase in temperature at the start of exercise which continued until 1-2 minutes post-exercise, whereby rectal temperature began a steady decline to a value forty minutes post-exercise which remained elevated from pre-exercise levels. Both the slopes of increase and decrease during exercise and recovery respectively appeared to be constant, unlike those of the esophageal profiles. These results were true of both Trial I and Trial II, except during the first few minutes of Trial I where a drop in rectal temperature was seen and associated with one subject's (DB) difficulty with the rectal probe.

The ANOVA procedure showed that the interaction between trials and time (F [4, 36]=.96, p=.4389) and the differences between trials (F [1,9]=.01, p=.9114) were both non-significant. However, the main effects did show that rectal temperature varied over time during the experiment (F [4,36]=55.71, p<.0001). The Scheffe procedure indicated that the following pairs of means differed significantly: PRE (M=37.130, SD=.250) and POST (M=37.925, SD=.331), PRE and POST7 (M=37.628, SD=.363), PRE and POST8 (M=37.548, SD=.342), DIL (M=37.229, SD=.260) and POST, DIL and POST7, DIL and POST8, POST and POST7, as well as POST and POST8. Furthermore, these results revealed that, unlike the esophageal post-exercise plateau which corresponded to the esophageal temperature at skin vessel dilation, post-exercise rectal values differed significantly from the rectal temperature at which skin vessel dilation occurred (Table 8).

Heart Rate and Oxygen Consumption

Heart rate demonstrated an immediate and sharp increase during the first few minutes of Trial I and Trial II exercise. Heart rate then continued to increase gradually until exercise termination where it reached its peak and began an initially rapid fall towards pre-exercise values during the first few minutes of recovery. The remaining heart rate recovery was much slower, demonstrating a slight elevation at forty minutes post-exercise which was greater in Trial I (Figure 5). A plot of the oxygen consumption data revealed that values prior to exercise were at typical resting values and increased approximately ten-fold during exercise and then quickly recovered to resting values within fifteen minutes post-exercise, during Trial I and Trial II (Table 9).

Skin Temperatures

All skin temperatures, led by the finger, fell at the onset of exercise after achieving a relatively stable state prior to exercise. At approximately 6-7 minutes into exercise, skin temperatures began to rise. This time of rise corresponded to the peak of Forearm minus Finger data (Figure 6) and the increase continued until several minutes post-exercise when recovery began. The thigh temperature demonstrated the slowest initial rate of recovery, as it rose sharply during the last few minutes of exercise and maintained a graphically identifiable elevation from pre-exercise values. The Finger demonstrated a post-exercise plateau which remained stable throughout recovery, while the Forearm and to a greater extent the Chest and Calf showed complete recovery at forty minutes post-exercise. This description is true of both Trial I (Figure 7) and Trial II (Figure 8), during which no visible differences can be seen. The individual forearm minus finger data for each subject, which was used to determine the threshold for skin vessel dilation, is presented in Appendix F.

DISCUSSION

The purpose of the present study was to compare the post-exercise core temperature (esophageal and rectal) responses for subjects performing treadmill exercise during hypohydrated

and euhydrated states. A distinctive aspect of this study was its measurement of post-exercise core temperature during two separate conditions of gradual hypohydration (dehydration) and water replacement to achieve euhydration, compared to previous studies which either investigated the role of fluid intake on core temperature during exercise or the profiles of single trial post-exercise measurements. It was hypothesized that ingestion of water sufficient to replace that which was lost in a condition of hypohydration, would not attenuate post-exercise hyperthermia.

The results of this study indicated the following:

(1) There was an extended post-exercise core temperature elevation from pre-exercise values in both trials of hypohydration and euhydration and that these elevations corresponded to the temperatures at which skin vessel dilation occurred during exercise for both trials.

(2) Although graphically there appears to be a difference between conditions, euhydration did not result in a statistically significant attenuation of post-exercise hyperthermia compared to hypohydration.

(3) A weak relationship was demonstrated between the amount of weight lost during hypohydration and the magnitude of elevation in core temperature post-exercise.

(4) Although the core temperature threshold for vasodilation between the hypohydrated and euhydrated trials appeared to differ graphically, no significant difference was demonstrated.

It was further noted that although changes in skin temperature (Forearm - Finger) appear to accurately predict the onset of skin vasodilation, it may not be a sufficient method of identifying the threshold of all subjects. The following discussion analyzes and elaborates on the main findings reported above, further addressing core temperature elevation post-exercise.

Post-Exercise Core Temperature Elevation

The demonstration of an elevated post-exercise core temperature plateau compared to pre-exercise values in Trial I (hypohydration) and its correspondence to the core temperature at which skin vessel dilation occurred (Figure 1) supports the results of Thoden et al. (1994). The core temperature at which skin vessel dilation occurred represents the threshold for active vasodilation in response to an increasing core temperature (Thoden et al., 1994; Johnson and Park, 1981; Roberts and Wenger, 1980) and the prolonged core temperature plateau suggests that the homeothermic defense mechanisms may not be similiar to pre-exercise values. Support for this hypothesis is made by Thoden et al. (1994) and the findings of the present study, which both demonstrated that despite a prolonged post-exercise core temperature plateau skin temperatures fell (Figure 7) and oxygen consumption levels reached pre-exercise values within 15 minutes post-exercise (Table 9).

While a definitive answer has not been found, Thoden et al. (1994) have addressed but not endorsed the idea that the post-exercise core temperature plateau might be a result of an exercise related response which has altered the hypothalamic "set-point", similiar to a situation when a fever is present. Examples of proposed mechanisms include the direct effect of increasing metabolic rate (Jonhnson, 1986) as well as more indirect outcomes resulting from decreases in blood volume (Fortney et al., 1981a) or increases in plasma osmolarity (Noakes, 1993; Horowitz, 1990). Although these explanations remain as possibilities, Johnson and Park (1981) argue that although increasing metabolic rate results in a delay in the onset of skin vasodilation, the delay of sweat rate is not as obvious, suggesting that there is not a shift in the "set-point". So, although these exercise related responses may play a role, it is unlikely that the hyperthermia is because of a shift in "set-point".

Another exercise related response is the gradual and inevitable hypohydration or progressive dehydration. Although it has not been proposed with regard to post-exercise hyperthermia, several investigators have demonstrated that maintaining hydration is a key mechanism in the attenuation of hyperthermia during exercise (Montain and Coyle, 1992a; Montain and Coyle, 1992b; Fortney et al., 1981a; Fortney et al., 1981b; Deschamps et al., 1989), and based on their rationale it would seem likely that maintaining hydration might have the same

effect on post-exercise hyperthermia. In other words, the post-exercise hyperthermia demonstrated by Thoden et al., (1994) and replicated by the first condition of the present study might easily be explained through the suggestion by Sawka (1988) that hypohydration results in hyperthermia by reducing the ability to dissipate heat. The maintenance of hydration in the second trial is discussed in the following section and will clarify the role of water intake on post-exercise hyperthermia and its connection to the body's ability to dissipate heat efficiently.

Comparison of Core Temperature Profiles Between Trials

In addition to replicating the findings of Thoden et al. (1994) as described above, the present study also demonstrated a significant elevated plateau in core temperature during the euhydrated condition (Figure 1). Although graphically there appears to be a difference between the two trials, euhydration did not result in a statistically significant attenuation of post-exercise hyperthermia compared to hypohydration. Therefore, pointing to the exercise related response of hypohydration in describing this finding appears to suggest that such an effect may be identifiable with a larger experimental population or more severe fluid loss and better replenishing. Nevertheless, it is certain that no conclusive explanation to the post-exercise core temperature plateau was related to volume loss in this study. While previous research has not investigated the role of hydration in post-exercise core temperature, the results of the present study indicate that hypohydration is not the main mechanism responsible for the elevated post-exercise core temperature because maintenance of hydration during exercise results in virtually the same extent of elevation as the hypohydrated condition. Therefore, this result does not support "set-point" theory in that the thermoregulatory system remained inoperative or inefficient following maintenance of body water volume and during a post-exercise time when there is more than adequate capacity to rapidly reduce heat content.

The attenuation by water loading of hyperthermia during exercise has been experimentally associated with decreases in serum osmolality and sodium concentrations (Noakes, 1993) as they result in higher cutaneous blood flow and associated with lower core temperatures during exercise (Noakes, 1993; Montain and Coyle, 1992a; Fortney et al., 1981a). Perhaps these mechanisms which are responsible for the hydration mediated attenuation of hyperthermia during exercise are

the same accounting for the graphically attenuated post-exercise hyperthermia demonstrated in the present study. Therefore, while euhydration, through graphical analysis, appears to reduce the post-exercise hyperthermia demonstrated during hypohydration and may provide a partial explanation to post-exercise hyperthermia, a more definitive answer probably lies in one or a combination of the several explanations which follow.

(1) Several investigators have demonstrated that euhydration or hyperhydration reduces core temperature during exercise (Montain and Coyle, 1992a; Fortney et al., 1981b; Moroff and Bass, 1965). As discussed above, the water intake in the present study may compensate for the reduced blood volume during exercise as described by Fortney et al. (1981a) and Sawka et al. (1979) and provide additional blood flow to the peripheral circulation to increase heat dissipation. Furthermore, water intake during exercise may delay or negate the onset of baroreceptor activity which limits skin blood flow during hypohydration (Nielsen et al., 1984), by maintaining blood volume and pulse pressure. Brengelmann et al. (1977) demonstrated that skin blood flow reaches a plateau during exercise despite the continued rise in core temperature. Perhaps this limitation is occurring during exercise recovery, providing an explanation for the post-exercise hyperthermia demonstrated in the present investigation and that of Thoden et al. (1994). It was suggested that this limitation in vasodilation may result because of the baroreceptor reflex, as the recovery period is associated with pooling of blood in the dilated vessels of previously active tissue and the associated veins (Roberts and Wenger, 1980). They noted that pooling of blood to the accommodating veins of the lower limbs when exercise has terminated results in postural hypotension, which induces the baroreceptor reflex and results in cutaneous vasoconstriction which overrides the vasodilation expected with the beginning of exercise recovery. Thus, the pattern of change of skin blood flow during recovery and hence the post-exercise hyperthermia, can possibly be explained by an elevated vasoconstrictor activity with changes in arterial or cardiopulmonary pressures.

(2) The post-exercise core temperature elevations seen in this and other studies (Thoden, 1994) may not be a result of inadequate fluid volume necessary for thermoregulatory homeostasis. Perhaps the redistribution of blood that occurs during and following exercise results in an inevitable compromise of skin blood flow, regardless of hydration state. Conceivably, blood flow towards the skin to meet thermoregulatory demands are of reduced priority during recovery as

cardiac output and blood pressure begins to fall. In the same way that cutaneous blood flow is restricted during exercise (Johnson, 1992) to provide blood flow to other regions (i.e. muscle), perhaps skin blood flow is being compromised again in order to provide blood flow to other, more vital regions of the body. As the demands for skin and muscle blood flow increase during exercise, cardiac output compensates for this need by increasing its output by up to seven times (Savard et al., 1988; Rowell, 1974). During recovery however, cardiac output drops dramatically as heart rate begins to fall and can no longer maintain the necessary blood flow to all regions. In addition, blood pressure falls, adding to the difficulty of maintaining blood flow to regions of need, including peripheral, central, and vital organs (i.e. the brain). Furthermore, as exercise ends, muscle remains dilated through chemical control (see Figure 6 and 7 for indications of a persistent high thigh temperature) adding to the blood pressure strains already present. As a result, perhaps cutaneous vessels do not dilate entirely as a safety mechanism to maintain constant blood flow to vital regions. This could provide a partial or full explanation to the post-exercise elevated core temperature and would conceivably result whether hydration was maintained or not.

Perhaps if the decrements in central circulation and heat dissipation were compensated for by immersing subjects in water, as suggested by Nielsen et al. (1984), adequate peripheral blood flow would take place, resulting in a continued drop in post-exercise core temperature to pre-exercise values. This has been demonstrated in unpublished work by Thoden and Kenny (1993). It has also been suggested that a possible mechanism in maintaining cardiac output and central venous pressure during exercise is the redistribution of blood away from the periphery (Fortney et al., 1981a). When they infused blood to maintain central circulation, redistribution of blood away from the periphery no longer occurred, presumably because the volume of blood was sufficient to adequately supply the peripheral and central circulations as well as the vital organs, such as the brain. They further suggested that the rate of sweating for a given core temperature is reduced when blood volume decreases during exercise as central integrative centers compete with thermoregulatory outflow. It seems likely that a similiar competition may exist post-exercise when cardiac output is falling and demands for blood flow to the brain are harder to maintain. Future research focusing on ways of maintaining central circulation or the role of blood volume infusion on post-exercise hyperthermia may help determine why core temperature remains elevated post-exercise.

(3) A third explanation for the possibility of an elevated post-exercise core temperature lies in the idea that the thermoregulatory system operates within a passive "null-zone" as described by Mekjavic et al. (1991) and that the corresponding temperature at which esophageal temperature plateaus and skin vessel dilation occurs during exercise represents the upper limit for the normal range of temperature control. Support for this idea can be made by the fact that heat dissipating responses appeared to be shut off once post-exercise core temperature reached the threshold for skin vessel dilation seen during exercise. An indication that heat dissipation was no longer functioning was seen by the drop in skin blood flow, reflected by the falling skin temperatures within 15 minutes post-exercise (Figure 7 and 8).

Although it seems that the results demonstrated by Mekjavic et al. (1991) provide an adequate explanation to the post-exercise core temperature elevation, other findings contradict their conclusions and suggest that other mechanisms are involved. Cabanac and Massonnet (1977) argued that investigations demonstrating "null zones" in models of core temperature regulation are a result of threshold responses to either very high or very low ambient temperatures and further demonstrated that the thresholds for sweating and shivering do coincide. This finding suggests that the effector responses seem to be continually active until heat or cold displacement is reversed to steady state "set-point" levels.

(4) Although the present study provides graphical evidence suggesting that hydration may provide a partial reduction of post-exercise hyperthermia, perhaps its role is much greater and is being overlooked by the methodology of the protocol. It is possible that future studies with greater numbers of subjects or administration of water in excess of body loss (hyperhydration) as done by Fortney et al. (1981b), may provide significant results.

In summary, the demonstration of a post-exercise core temperature elevation during conditions of euhydration in addition to the previously established elevation during hypohydration (Thoden et al., 1994) strongly suggests that hydration is not the main mechanism responsible for the apparently inactive thermoregulatory system once recovering to the threshold for skin vessel dilation demonstrated during exercise. Perhaps other exercise related responses which are indirectly affected by hydration level, such as reflexive response to maintain blood pressure in the face of reduced blood volume, play a larger role in controlling post-exercise hyperthermia. It is also possible that the core temperature responses as demonstrated in the present study may be a

result of an inoperative thermoregulatory system as suggested by Mekjavic et al. (1991), rather than a result of hypohydration or any other exercise related response.

Hypohydration and the Magnitude of Post-Exercise Elevation

The present study demonstrated a weak relationship between the degree of hypohydration and magnitude of post-exercise core temperature elevation (Table 6). This indicates that the elevation in post-exercise core temperature demonstrated in the present study and that by Thoden et al. (1994), does not appear to be a result of hypohydration. These findings were made despite previous research which documents the thermoregulatory benefits of body water hydration during exercise.

Because water is the main substance lost in sweat (Guyton, 1981) and because excess body water deficit is associated with hyperthermia (Montain and Coyle, 1992a; Fortney et al., 1981), several investigators have hypothesized and investigated the idea that hypohydration is the mechanism responsible for hyperthermia (Montain and Coyle, 1992b; Sawka, 1988; Fortney et al., 1981). The rationale behind these studies is presumably that hypohydration results in inadequate body water volume necessary to ensure proper heat dissipation through either sweat output, cutaneous blood flow, or both. Montain and Coyle, (1992b) demonstrated support for the hypothesis proposed by Sawka (1988) that reductions in body water volume result in hyperthermia by reducing the ability of the body to dissipate heat. These authors argue that minimizing hypohydration or maintaining hydration throughout exercise will reduce the competition between the central and peripheral circulations so that attenuation of hyperthermia will follow.

In the present study, the ingestion of water in Trial II, in the amount corresponding to that which was lost in Trial I, attempted to clarify the role of hydration in maintaining core temperature. Although core temperature recordings were collected throughout exercise as well as during post-exercise, the relationship between the degree of hypohydration and the magnitude of exercise hyperthermia was not investigated. The ingestion of water during Trial II resulted in disturbances in esophageal recordings at peak temperatures which did not permit accurate comparisons between Trial I and Trail II. Therefore, although the present study implies that

hydration is not the main mechanism in attenuating post-exercise hyperthermia, it is unable to support the findings of the studies investigating hyperthermia during exercise as described above by Montain and Coyle (1992) and others in the review of literature. Future research controlling for the effect of water intake on esophageal recordings will further elucidate the issue of hydration on core temperature.

While it is unanimously agreed that hypohydration can accentuate hyperthermia during exercise, Gisolfi and Copping (1974) investigated the role of sweat loss in predicting hyperthermia and challenged "set-point" theory, supported by many including Sawka (1988) that hypohydration-mediated hyperthermia occurs because of the bodies inability to properly dissipate heat. These authors suggest that although maintaining body water volume attenuates hyperthermia, it is not a result of increased capabilities for heat dissipation. They demonstrated that sweating rates were not decreased following progressive hypohydration and therefore argue that reductions in core temperature were not a result of increased sweat output. Fortney et al. (1988) recognized the findings of Gisolfi and Copping (1974), but further demonstrated that reduced sweat output seen during hypohydration is not necessarily a sign of reduced heat dissipating capabilities, but rather results in a shift in heat dissipation from evaporative cooling to convective cooling, through cutaneous blood flow.

In one subject (GK), the present study demonstrated what seemed like a large association between the magnitude of sweat lost in the hypohydrated trial and the return of post-exercise core temperature to pre-exercise levels in the euhydrated trial (Figure 2). This result presumably supports "set-point" theory that large deviations in load-error will be eliminated if body water volume is sufficient to provide efficient thermoregulatory functioning. This finding prompted the statistical correlation between the magnitude of sweat lost in Trial I and the return of post-exercise core temperature in Trial II in every subject, where the calculated correlations were low. This suggests that while one individual subject demonstrated support for hydration as a main mechanism for post-exercise elevation, an overall effect can not be concluded. Future research, controlling subject variability more closely may possibly determine the association between individual core temperature responses, hydration level, and sweat output.

In summary, although hypohydration appears to be one underlying mechanism resulting in exaggerated elevated core temperatures during exercise, the present study demonstrates that the

specific mechanism controlling post-exercise hyperthermia can not be compensated for simply by replacing the water loss during exercise with equal volumes of ingested water. This observation is supported by the fact the correlation between the level of sweat output or hypohydration and the extent of post-exercise core temperature elevation is weak.

Hydration Level and the Threshold for Heat Dissipation

Although the esophageal temperature value at which skin vessel dilation occurred between the hypohydrated and euhydrated conditions appeared to differ (Figure 6), no statistically significant difference was demonstrated (Table 3). Previous research has investigated the role of hydration on the threshold for sweating (Sawka et al., 1985; Johnson and Park, 1981) and the effect of hydration on circulatory control of attenuating hyperthermia (Montain and Coyle, 1992b; Johnson and Park, 1981). Sawka et al. (1985) demonstrated that a progressive delay in the core temperature onset of sweating occurred as the degree of hypohydration increased, while Montain and Coyle (1992b) showed that fluid intake to maintain hydration weakened hyperthermia by increasing the onset and rate of skin blood. Johnson and Park (1981) further studied the effects of hypohydration on heat dissipating capabilities and demonstrated that while both mechanisms are significantly affected, the threshold for vasodilation is delayed further.

While the present study did not measure sweat output directly except by weight loss, nor the core temperature threshold for the onset of sweating, the threshold for vasodilation was determined. In contradiction to the findings of all of the authors described above (Montain and Coyle 1992b; Sawka et al., 1985; Johnson and Park, 1981), the present study did not demonstrate the thermoregulatory benefits of euhydration during exercise to the same extent, in that the threshold for vasodilation did not differ significantly between the hypohydrated and euhydrated conditions. However, after individually analyzing the difference between forearm and finger temperatures for each subject (Appendix F), it was noted that changes in skin temperature may not be an accurate method for determining the threshold for skin vessel dilation for all subjects. This method was proposed by Rubenstein and Sessler (1990) who demonstrated that changes in skin temperature can be accurately used to identify the vasodilation threshold. When individually locating the point when the difference between forearm and finger temperature rapidly dropped, it

seemed possible that it was not accurately identifying the time for locating the vasodilation threshold. While the statistical results of the present study did not support the findings that hypohydration will delay the onset of vasodilation, compared to euhydration, it could have possibly been a result of the method of threshold determination.

Summary and Conclusions

In summary, the purpose of the present investigation was to compare the post-exercise core temperature profiles of subjects performing exercise under two separate conditions of hypohydration and euhydration. It was hypothesized, based on the theory that the thermoregulatory system operates within an inoperative or passive zone, that euhydration would not attenuate the post-exercise core temperature elevated plateau demonstrated in previous research as well as during the hypohydrated condition of the present study.

It was demonstrated that water ingestion to achieve euhydration showed no significant difference when compared to a condition where hypohydration or progressive dehydration took place. Although the hypothesis for the study was confirmed by the findings of this study, the reasoning behind the establishment or rationale of the hypothesis was not conclusively endorsed based upon the possible explanations for the post-exercise core temperature elevations as provided by the present investigation. It was suggested that post-exercise core temperature elevation may be a result of either one or a combination of:

(1) inadequate body water volume necessary for efficient thermoregulation;

(2) the redistribution of blood flow that takes place during exercise recovery;

(3) inadequate blood volumes necessary for active vasodilation;

(4) the delay in the onset of baroreceptor activity resulting from one or a combination of (1), (2), and (3) above; and

(5) the characteristic of the thermoregulatory system to remain inoperative within a certain "null-zone" of core temperature regulation as provided in the rationale to the hypothesis.

It was further demonstrated that one subject (GK) produced results which presumably support "set-point" theory, in that post-exercise core temperature elevation may be a result of hypohydration, causing the temperature regulating system to become inefficient. However, the remaining subjects showed no similiar pattern to GK, and demonstrated results that heavily discount hypohydration as a main mechanism in post-exercise hyperthermia. Lastly, while the present study demonstrated no difference between the timing of the vasodilation threshold for the hypohydrated and euhydrated condition, it was suggested that the method for threshold determination may have been invalid in this study.

In summary, the results of this investigation support the following conclusions:

(1) In the present study, water intake to establish euhydration did not have an effect on returning post-exercise core temperature elevation to pre-exercise, "set-point" values.
(2) In the present study, the magnitude of body water loss during the hypohydrated trial was not associated with the degree of post-exercise core temperature elevation.

Limitations and Recommendations for Future Research

The limitations of the present study include the small sample size, the method for determining changes in blood volume, and the lack of control for inter-subject variability. Because of the relatively intense and uncomfortable testing protocol, subject recruitment was difficult. This resulted in a small sample size, limiting the power of the statistical analyses. Thus, if the sample size was larger, significant differences may have been demonstrated between the two trials. Future research should include a greater number of subjects in order to increase the power of the analyses.

Hematocrit sampling was used in the present study to measure changes in blood volume, or more specifically, relative changes in plasma volume. Although changes in hematocrit presumably reflect changes in plasma volume, it can not be accurately used as a measure for determining absolute blood volume changes. Future research, employing more direct measurements of blood volume changes as a result of hypohydration may further clarify the phenomena of post-exercise hyperthermia.

In addition, the method of determining the time of skin vessel dilation by monitoring skin temperatures and the forearm minus finger differences proved difficult to use in that the temperature change associated with vasodilation did not always show a definitive inflection. It is therefore recommended that studies which place a principle focus on the time of vasodilation should employ a more direct measurement of vascular conduction, such as that offered by Laser Flowmetry.

The potential for confounding inter-subject variability was not controlled for in the present study. More specifically, factors such as subjects' fitness and heat acclimation levels, which are important determinants of core temperature responses to exercise, were not considered when designing the protocol of the present study. Heat acclimatized subjects will experience different heat dissipating responses than non-acclimatized individuals the same way physically fit individuals will respond differently compared to non-fit subjects. A more comprehensive protocol which considers these factors would better predict post-exercise core temperature responses to hypohydrated and euhydrated individuals.

TABLE 1. Individual Subject Age and Physical Characteristics

Subject	Age (yrs.)	Height (cm.)	Weight (kg.)
CM	20	175	179.25
DB	20	164.5	159.125
DM	23	173.4	173.5
GK	30	178	186
LA	23	175	162.375
JB	24	175	178.635
DJ	23	180	170.625
LM	22	185	172.75
BH	22	182	174.125
FS	21	184.5	173.125
M	**22.8**	**177.24**	**172.951**
SD	**2.859**	**6.109**	**7.835**

TABLE 2. Individual Subject Heart Rates, Body Weights, and Hematocrit Values

Subject	HR (bpm)	THR (bpm)	HCT1 (%)	HCT2 (%)	HCT3 (%)	HCT4 (%)	WL1 (kg)	WL2 (kg)
CM	74	168.5	50	51.5	48	50	.568	-0.171
DB	62	165.5	47	49	45.5	50.5	.625	.114
DM	67	164.5	52	51.5	53	51.5	.568	0
GK	68	159.5	42	44.25	41.5	44	.909	0
LA	58	162.25	45.25	49	44	46	.739	0
JB	53	160.25	45	46	45	41	.516	0
DJ	49	160	46	48	47.25	47	.682	0
LM	69	165	45	46.5	44	46.5	.568	0
BH	44	159.5	43	46	42	42.5	.909	.114
FS	57	164	45	46	45	47	.739	-0.057
M	**60.1**	**162.9**	**46.025**	**47.775**	**45.525**	**46.6**	**.682**	**0**
SD	**9.620**	**3.078**	**3.010**	**2.456**	**3.313**	**3.438**	**.141**	**.08**

Note: HR = Resting heart rate
THR = Target heart rate (Predicted Maximum HR [220-age] -HR * .75 + HR)
HCT1 = Hematocrit value pre-exercise, Trial I.
HCT2 = Hematocrit value post-exercise, Trial I.
HCT3 = Hematocrit value pre-exercise, Trial II.
HCT4 = Hematocrit value post-exercise, Trial II.
WL1 = Weight loss from Trial I.
WL2 = Weight loss from Trial II.

TABLE 3. Mean Esophageal Temperatures Across Time and Trials

	PRE	DIL	POST	POST7	POST8	M	SD
TRIAL I	36.76	37.41	37.24	37.13	37.08	37.13	.45
TRIAL II	36.78	37.11	37.07	37.01	36.98	36.99	.31
M	**36.77**	**37.26 ***	**37.16 ***	**37.07 ***	**37.03**		
SD	.26	.60	.24	.27	.28		

Note: N = 10

(* - indicates a significant difference from PRE, p=.0001)

Reported temperatures are averages of seven points taken directly around that point.

PRE = pre-exercise measurement taken 150 seconds before the start of exercise.

DIL = exercise measurement taken at the peak of forearm - finger data (dilation).

POST = post-exercise measurement representing an average of the first 30 minutes of recovery.

POST7 = post-exercise measurement taken during the 35th minute of recovery.

POST8 = post-exercise measurement taken during the 40th minute of recovery.

TABLE 4. Absolute Value of Differences Among Esophageal Means (MSerror=0.0672 [df=76], p=5, n=20)

		PRE	DIL	POST	POST7	POST8
PRE	36.78	x	0.490*	0.383*	0.292*	0.250
DIL	37.27	x	x	0.108	0.199	0.241
POST	37.16	x	x	x	0.090	0.133
POST7	37.07	x	x	x	x	0.042
POST8	37.03	x	x	x	x	x

Note: N = 20 cases

(* - Scheffe procedure indicates these pair of means differ significantly)

Scheffe's critical difference = 0.2598

TABLE 5. Difference Between Trial I and Trial II Esophageal Temperature Elevations and Weight Loss

Subjects	POST - PRE Difference		Trial Difference	Weight Loss (kg)	Weight Loss
	Trial I	Trial II	Trial II - Trial I	Absolute	Relative
BH	.33	.35	-.01	.909	1.149 %
CM	.23	.12	.11	.568	.697 %
DB	.30	.12	.18	.625	.864 %
DJ	.36	.12	.24	.686	.879 %
DM	.61	.36	.26	.568	.720 %
FS	.10	.19	-.09	.739	.939 %
GK	1.10	-.01	1.11	.909	1.075 %
JB	.38	.47	-.09	.516	.635 %
LA	.54	.47	.07	.739	1.001 %
LM	.81	.71	.10	.568	.724 %

Note: -Weight Loss (absolute) = The absolute weight loss in Trial I, corresponding to the amount of water ingested in Trial II.
-Weight Loss (relative) = Weight loss in Trial I expressed as a percentage of body weight.

TABLE 6. Correlation Coefficients Between Trial Differences and Weight Loss

	Trial Difference	Absolute Weight Loss	Relative Weight Loss
Trial Difference	x	x	x
Absolute Weight Loss	.453	x	x
Relative Weight Loss	.334	x	x

TABLE 7. Mean Rectal Temperatures Across Time and Trials

	PRE	DIL	POST	POST7	POST8	M	SD
TRIAL I	37.11	37.21	37.95	37.66	37.56	37.50	.42
TRIAL II	37.15	37.25	37.90	37.60	37.54	37.49	.43
M	**37.13**	**37.23**	**37.93****	**37.63****	**37.55****		
SD	.25	.26	.33	.36	.34		

Note: N = 10
(** - indicates a significant difference from PRE and DIL, $p<.0001$)
Reported temperatures are averages of seven points taken directly around that point.
PRE = pre-exercise measurement taken 150 seconds before the start of exercise.
DIL = exercise measurement taken at the peak of forearm - finger data (dilation).
POST = post-exercise measurement representing an average of the first 30 minutes of recovery.
POST7 = post-exercise measurement taken during the 35th minute of recovery.
POST8 = post-exercise measurement taken during the 40th minute of recovery.

TABLE 8. Absolute Value of Differences Among Rectal Means
(MSerror=0.0233 [df=76], p=5, n=20)

		PRE	DIL	POST	POST7	POST8
PRE	37.10	x	0.10	0.80*	0.50*	0.42*
DIL	37.23	x	x	0.70*	0.40*	0.32*
POST	37.93	x	x	x	0.30*	0.38*
POST7	37.63	x	x	x	x	0.08
POST8	37.55	x	x	x	x	x

Note: N = 20 cases
(* - Scheffe procedure indicates these pair of means differ significantly)
Scheffe's critical difference = 0.1531

TABLE 9. Oxygen Consumption Values (ml/kg/min)

	Trial I	Trial II
Pre-exercise	4.9	4.7
Exercise	48.4	48.9
Post-exercise	4.96	5.3

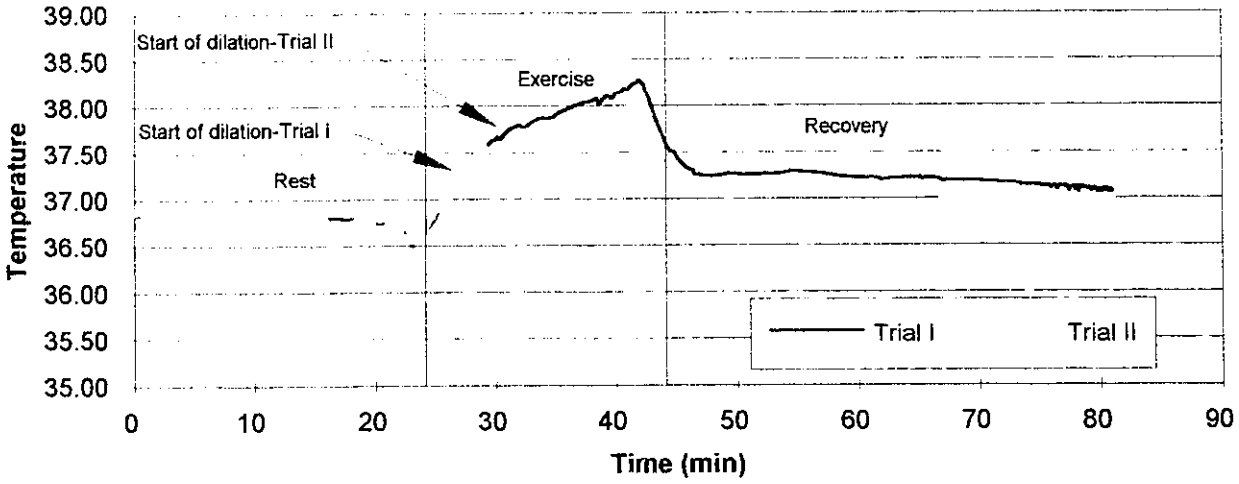


Fig. 1. Mean esophageal profiles during 21 minute rest, 20 minute exercise, and 40 minute leaning recovery for Trial I where no water was ingested and for Trial II where hydration was maintained. Skin vasodilation occurred at 26 minutes in Trial I and 29.5 minutes. in Trial II. (Ambient temperature, 24-29°C; relative humidity, 30-50%; n=10). Sharp dropes during Trial II represent water ingestion, not error.

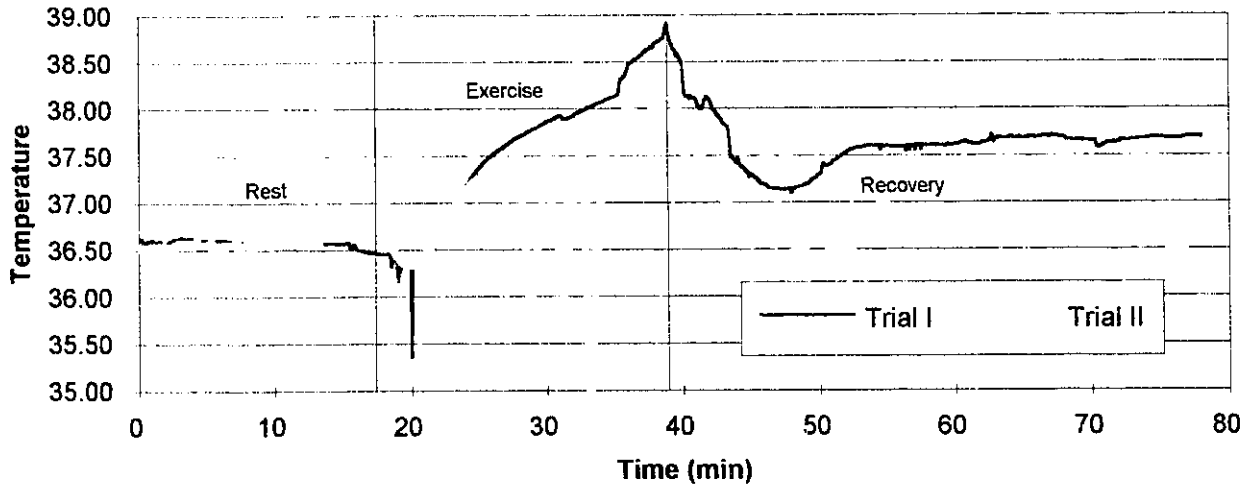


Fig. 2. Esophageal profiles for subject GK during 21 minute rest, 20 minute exercise, and 40 minute leaning recovery for Trial I where no water was ingested and for Trial II where hydration was maintained. These profiles differ from the other subjects in that a virtual complete recovery is shown in Trial II (Ambient temperature, 24°C; relative humidity, 41%).

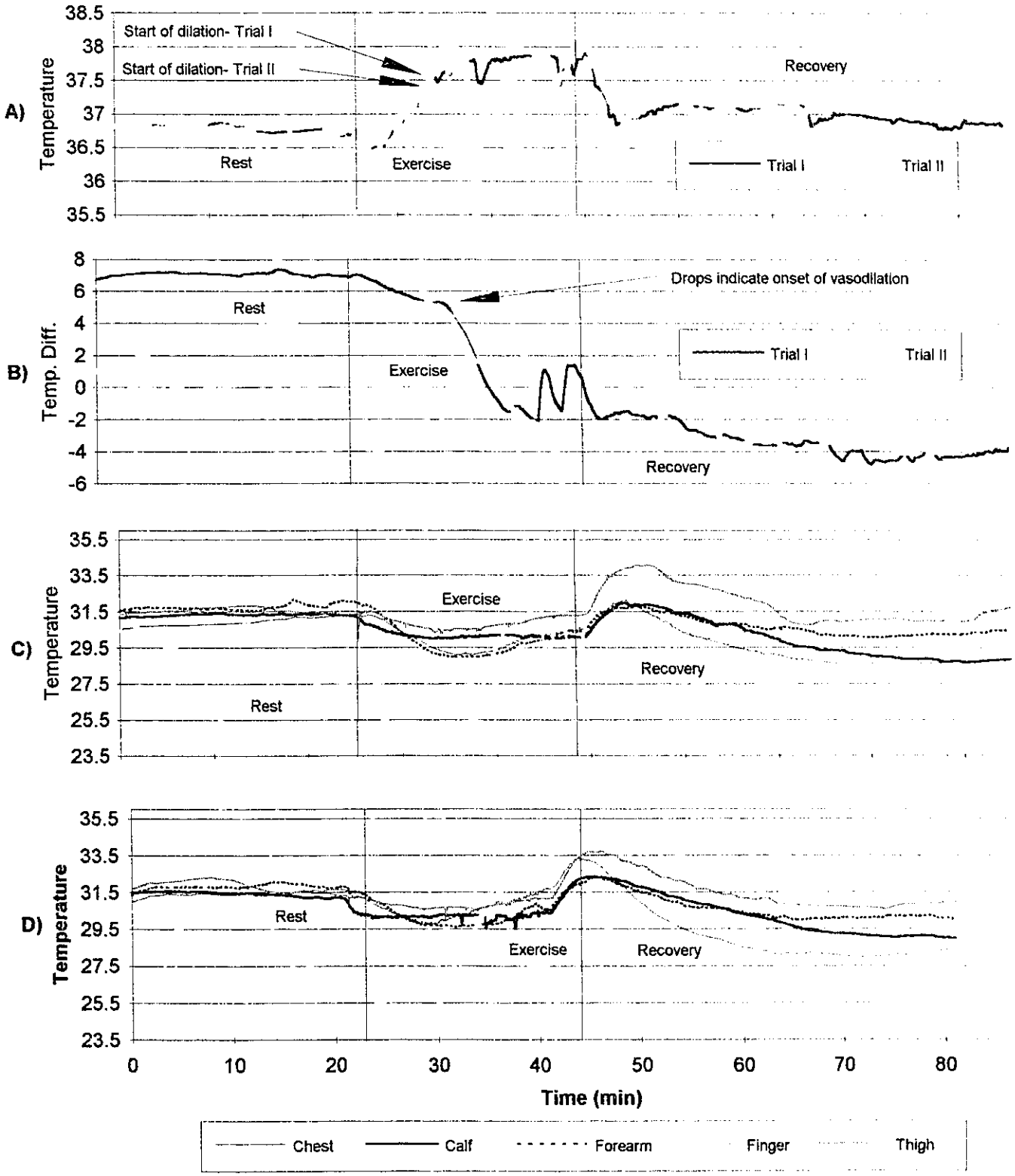


Fig. 3. Individual data of a typical subject. (a) Esophageal profiles with time of onset of vasodilation as determined through (b) for both Trial I (Ambient temperature, 24°C; relative humidity, 37%) and Trial II (Ambient temperature, 24°C; relative humidity, 33%); (b) Forearm minus finger data used to determine onset of vasodilation; (c) Skin temperature profiles for Trial I; (d) Skin temperature profiles for Trial II.

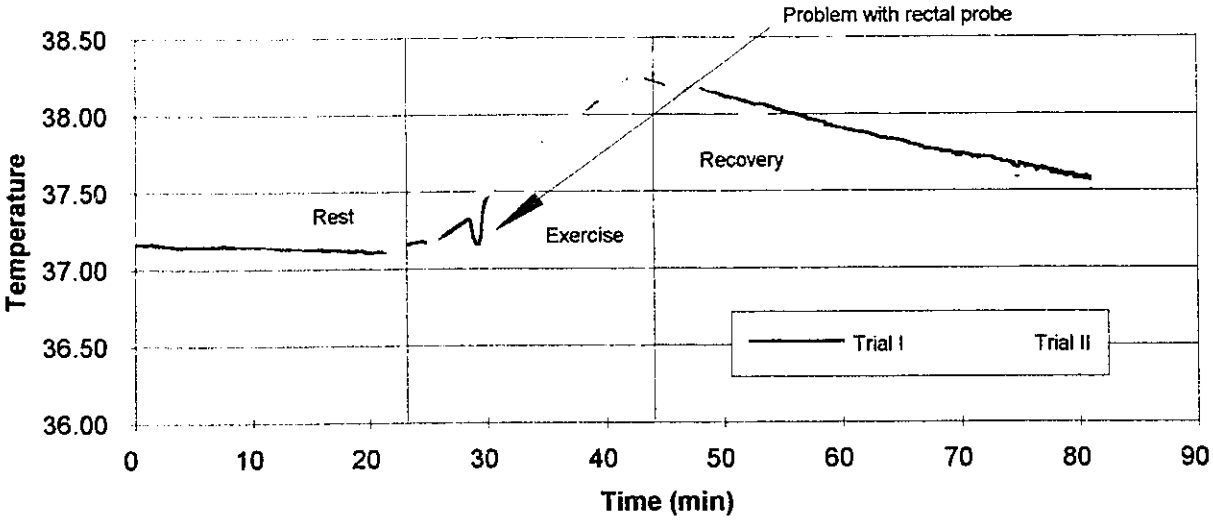


Fig. 4. Mean rectal profiles during 21 minute rest, 20 minute exercise, and 40 minute leaning recovery for Trial I where no water was ingested and for Trial II where hydration was maintained. Skin vasodilation occurred at 26 minutes in Trial I and 29.5 minutes in Trial II. (Ambient temperature, 24-29°C; relative humidity, 30-50%; n=10).

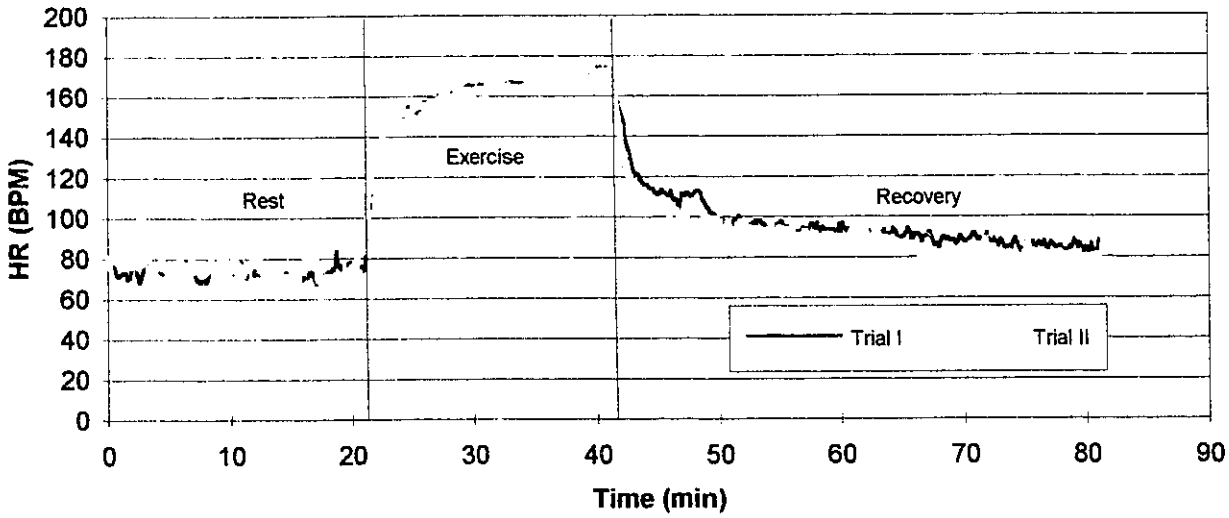


Fig. 5. Mean heart rate profiles during rest, exercise, and recovery for both Trial I and Trial II.

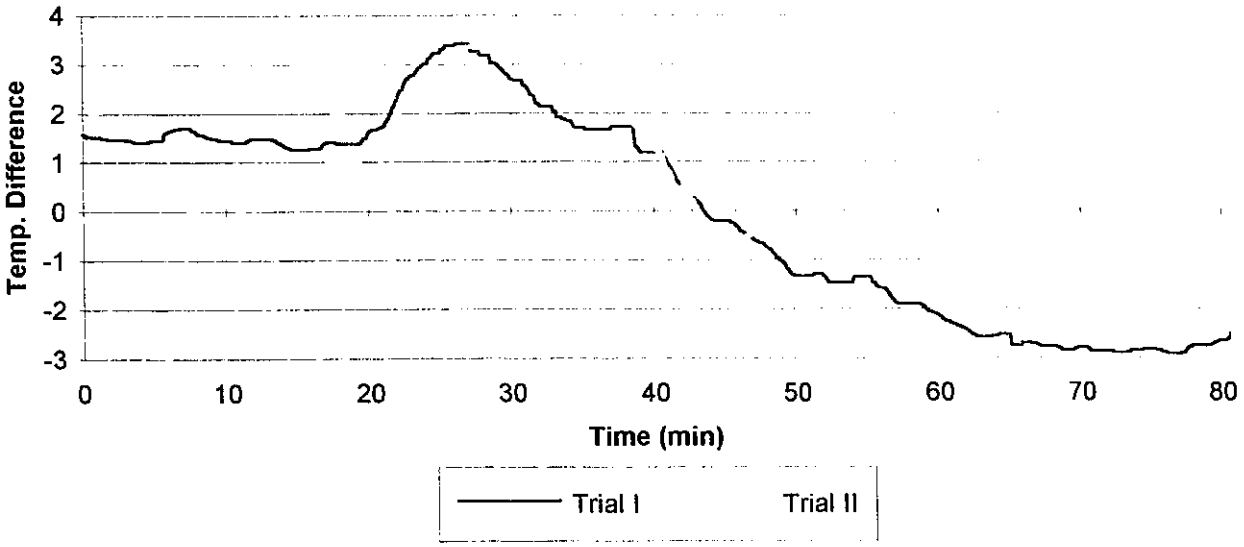


Fig. 6. Mean Forearm Minus Finger data calculated throughout rest, exercise, and recovery for Trial I and Trial II. Peak values correspond to time of skin vessel dilation as described by Rubenstein and Sessler (1990).

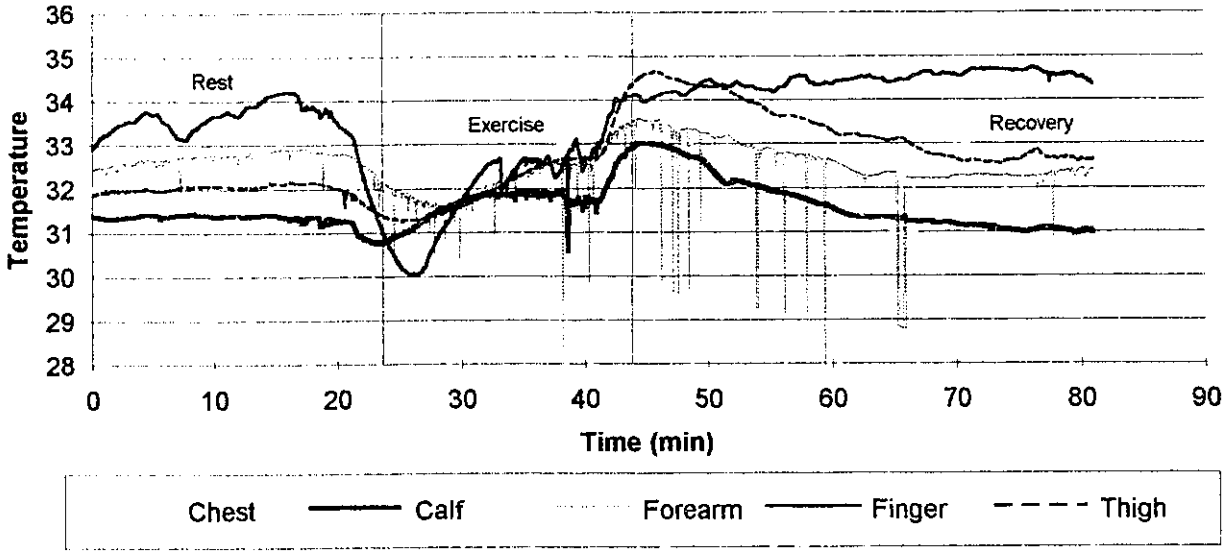


Fig. 7. Skin temperature profiles during 21 minute rest, 20 minute exercise, and 40 minute leaning recovery during Trial I where water ingestion was not administered. (Ambient temperature, 24-29°C; relative humidity, 30-50%; n=10).

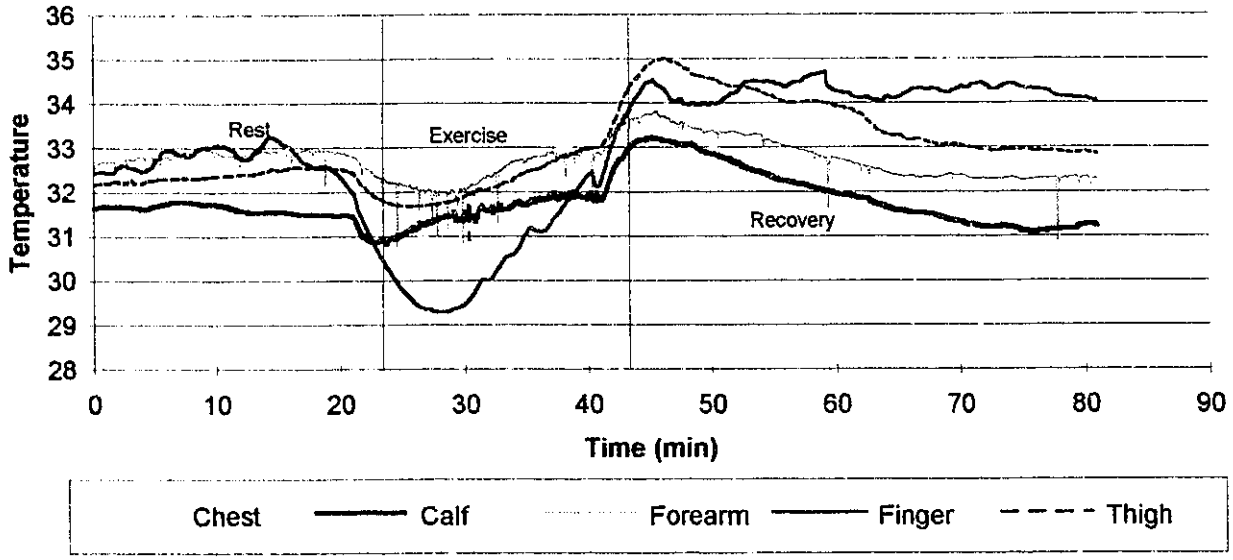


Fig. 8. Skin temperature profiles during 21 minute rest, 20 minute exercise, and 40 minute leaning recovery during Trial II where water ingestion was administered. (Ambient temperature, 24-29°C; relative humidity, 30-50%; n=10).

REFERENCES

Brengelmann, G.L., Johnson, J.M., Hermansen, L., and Rowell, L.B. (1977). Altered control of skin blood flow during exercise at high internal temperatures. Journal of Applied Physiology. 43(5): 790-794.

Brown, W.K., and Sargent, F. (1965). Hidromeeiosis. Archives of Environmental Health. 11: 442-452.

Cabanac, M., and Massonnet, B. (1977). Thermoregulatory responses as a function of core temperature in humans. Journal of Physiology. 265:587-596.

Deschamps, A., R.D. Levy, M.G. Cosio, E.B. Marliss, S. Magder (1989). Effect of saline infusion on body temperature and endurance during heavy exercise. Journal of Applied Physiology. 66(6): 2799-2804.

Fortney, S.M., Nadel, E.R., Wenger, C.B., and Bove, J.R. (1981a). Effect of acute alterations of blood volume on circulatory performance in humans. Journal of Applied Physiology: Respiration, Environmental, and Exercise Physiology. 50(2): 292-298

Fortney, S.M., Nadel, E.R., Wenger, C.B., and Bove, J.R. (1981b). Effect of blood volume on sweat rate and body fluids in exercising humans. Journal of Applied Physiology: Respiration, Environmental, and Exercise Physiology. 51:1594-1600.

Gisolfi, C.V., and Copping, J.R. (1974). Thermal effects of prolonged treadmill exercise in the heat. Medicine and Science in Sports. 6(2): 108-113.

Guyton, A.C. Metabolism and Temperature Regulation; The body fluids and the Kidneys. In: Textbook of Medical Physiology. W.B. Saunders Company. Philadelphia, 1981. 358-402, 838-847.

Horowitz, M. (1990). Thermoregulation under conditions of impaired body fluid/ osmotic balance in mammals. Journal of Basic Clinical Physiology and Pharmacology. 1:267-276.

Johnson, J.M., and Park, M.K. (1981). Effect of upright exercise on threshold for cutaneous vasodilation and sweating. Journal of Applied Physiology. 50: 814-818.

Johnson, J. M. (1986). Nonthermoregulatory control of human skin blood flow. Journal of Applied Physiology. 61(5): 1613-1622.

Johnson, J.M. (1992). Exercise and the cutaneous circulation. Exercise and Sports Sciences reviews. 20: 59-97.

Mekjavic, I.B., Sundberg, C.J., Linnarsson, D. (1991). Core temperature "null zone". Journal of Applied Physiology. 71:1289-1295.

Mekjavic, I.B., and Bligh, J. (1989). Core threshold temperatures for sweating. Canadian Journal of Physiology and Pharmacology. 67:1038-1044.

Montain, S.J., E.F. Coyle (1992a). Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. Journal of Applied Physiology. 73(4): 1340-1350.

Montain, S.J., E.F. Coyle (1992b). Fluid ingestion during exercise increases skin blood flow independent of increases in blood volume. Journal of Applied Physiology. 73(3): 903-910.

Moroff, S.V., and Bass, D.E. (1965). Effects of overhydration on man's physiological responses to work in the heat. Journal of Applied Physiology. 20: 267-270.

Murray, R. (1992). Nutrition for the marathon and other endurance sports: environmental stress and dehydration. Medicine and Science in Sports and Exercise. 24(9): S319-S323.

Nadel, E.R., Fortney, S.M., and Wenger, C.B. (1980). Effect of hydration state on circulatory and thermal regulations. Journal of Applied Physiology. 49(4): 715-721.

Nielsen, B., Rowell, L.B., and Bonde-Petersen, F. (1984). Cardiovascular responses to heat stress and blood volume displacements during exercise in man. European Journal of Applied Physiology. 52: 370-374.

Noakes, T.D. (1993). Fluid replacement during exercise. Exercise and Sports Science Reviews. 21: 297-330.

Roberts, M.F. and Wenger, C.B. (1980). Control of skin blood flow during exercise by thermal reflexes and baroreflexes. Journal of Applied Physiology. 48(4): 717-723.

Rowell, L.B. (1974). Human cardiovascular adjustments to exercise and thermal stress. Physiology Reviews. 54: 75-159.

Rubenstein, E.H., and Sessler, D.I. (1990). Skin surface temperature gradients correlate with fingertip blood flow in humans. Anaesthesiology. 73: 541-545.

Savard,G.K., Nielsen, B., Laszczynska., Larsen, B.E., and Saltin, B. (1988). Muscle blood flow is not reduced in humans during moderate exercise heat stress. Journal of Applied Physiology. 64(2):649-657.

Sawka, M.N., Knowlton, R.G., and Critz, J.B. (1979). Thermal and circulatory responses to repeated bouts of prolonged running. Medicine and Science in Sports. 11: 177-180.

Sawka, M.N., Young, A.J., Francesconi, R.P, Muza, S.R., and Pandolf, K.B. (1985). Thermoregulatory and blood responses during exercise at graded hypohydration levels. Journal of Applied Physiology. 59: 1394-1401.

Sawka, M.N., and Wenger, C.B. Physiological responses to acute exercise-heat stress. In: Human Performance Physiology and Environmental Medicine at Terrestrial Extremes (ed: K.B. Pandolf, M.N. Sawka, R.R. Gonzalez), Benchmark press, Inc. Indianapolis, 1988.

Sawka, M.N. Body fluid responses and hypohydration during exercise-heat stress. In: Human Performance Physiology and Environmental Medicine at Terrestrial Extremes (ed: K.B. Pandolf, M.N. Sawka, R.R. Gonzalez), Benchmark press, Inc. Indianapolis, 1988.

Thoden, J., Kenny, G., Reardon, F., Jette, M., and Livingston, S. (1994). Disturbance of thermal homeostasis during post-exercise hyperthermia. European Journal of Applied Physiology. 68(2):170-176.

APPENDIX A:
Proposal for the study of
"The Effect of Water Intake on Post-Exercise Hyperthermia"

CHAPTER I: INTRODUCTION

CHAPTER I

1.0 Introduction

Exercise increases heat production and results in core temperatures significantly greater than pre-exercising values that are presumed to approximate the "set-point" for temperature established in the hypothalamus (Cabanac and Massonet, 1977). Various autonomic effector responses such as sweating and blood flow to the skin are unable to match heat production with heat loss in order to prevent core temperature elevation. An exercise induced vasoconstriction in non-active sites and a weakened homeothermic defense occurs, which results in this temporary state of hyperthermia (Sawka and Wenger, 1988).

During exercise, competition exists between thermoregulatory and exercise demands for blood flow. Exercise requires blood in order to transport oxygen and nutrients to working muscle. On the other hand, proper thermoregulatory homeostasis requires blood flow as a means for transporting heat from the core to the skin in order to match the heat production caused by exercise. During exercise blood flow to working muscle is of primary importance and the cutaneous vasoconstriction which produces this redistribution of blood reduces thermoregulatory efficiency and results in core temperature elevation (Rowell, 1974). As exercise ends and recovery begins, the competition for blood flow is terminated. Blood flow is redirected from the working muscle to the skin, in order to satisfy thermoregulatory demands and bring core temperature back towards its normal operating range or "set-point" (Brengelmann et. al., 1977).

Popular theory suggests that core temperature will continue to decline following an exercise induced hyperthermia until "set-point" is reached. The argument states that heat dissipating responses will remain active until heat production is matched by heat loss. This is known as the load-error principle (Sawka and Wenger, 1988). Recently, some authors have investigated the possibility of an inactive range whereby thermoregulatory processes are shut off (Thoden et al., 1994; Mekjavic et. al., 1991; Jessen & Ludwig, 1971). These authors have all demonstrated that either a passive range or null zone exists in which no attempt is made to drive core temperature towards "set-point".

Supporters of the load error theory suggest that the demonstration of an inactive thermoregulatory band is a result of an exercise related response which causes effector responses to become inefficient, rather than inactive (Sawka and Wenger, 1988).

Sweating is an exercise related response which occurs at the expense of intra- and extracelluar fluid volumes (Murray, 1992). Exercise causing increases in heat production requires that part of the cardiac output be diverted to the skin at a time when body fluid volume is decreasing from sweat loss. This results in a decrease in central blood volume, and an associated drop in blood flow to the surface; a condition which is inefficient for producing heat loss.

Perhaps the demonstration of an inactive system is simply a result of excessive sweating resulting in inadequate levels of cardiac output to engage in active vasodilation. After all, it has long been recognized that fluid hydration has a significant influence upon the body's ability to regulate core temperature (Nadel et. al., 1980). At least that is one explanation that the proponents of the load error theory of thermoregulation offer. On the other hand, perhaps increasing plasma volume and\or cardiac output would have no effect on post-exercise hyperthermia as the extended elevation may not be a result of plasma volume related response, but an indication that temperature regulation is passive within a certain range.

The following study has been designed to investigate the role of water intake on post-exercise hyperthermia and its implications regarding the load error theory of thermoregulation and the controversy which seems to exist.

1.1 Rationale

The rationale for the hypothesis is that the level at which core temperature plateaus post-exercise corresponds to the core temperature at which skin vessel dilation occurs during exercise (Thoden et. al., 1994). This would suggest that thermoregulatory processes are inactive once core temperature reaches the level of vessel dilation post-exercise and that increasing plasma volume would have no effect on thermoregulatory functioning. In other words, post-exercise hyperthermia is a result of inactive thermoregulation, as opposed to some exercise related response, such as inadequate levels of plasma volume.

1.2 Hypothesis

Based upon the results of Thoden et. al., 1994 and Mekjavic et. al., 1991 that the resting system operates within a zone of passive response to temperature change, it is hypothesized that maintaining plasma volume will not attenuate post-exercise hyperthermia.

1.3 Statement of the Problem

In an effort to investigate the mechanisms responsible for an elevated post-exercise core temperature, this study will examine the effect of maintaining water volume and its effect on post exercise core temperature elevation as assessed by the time-course of rectal and esophageal temperature and various skin temperatures, and the cardiopulmonary variables of heart rate and oxygen consumption.

1.4 Objectives

The objectives of the present study are to:

1) investigate post-exercise esophageal and rectal temperature responses following exercise without water ingestion,
2) investigate post-exercise esophageal and rectal temperature responses following exercise with water ingestion,
3) investigate hematocrit values both prior to and following exercise without water ingestion,
4) investigate hematocrit values both prior to and following exercise with water ingestion.

1.5 Relevance

The relevance of the study can be demonstrated from two separate domains. Firstly, from a basic research point of view, the present study will contribute to the evolving knowledge of thermoregulation and will help understand the mechanisms responsible for the maintenance of

core temperature. Secondly, from an applied point of view, the present study will help understand factors that limit human performance when exercise demands are in competition with thermoregulatory demands. More specifically, the study will help determine how fluid replacement affects our performance and thermoregulatory functioning during physical stress.

1.6 Delimitations

The results of the present study will not be generalized to the general population as the subject population will be restricted to male university students who are active, but not training and familiar with the exercise protocol. As well, it will not be possible to generalize these results to women because of hormonal changes that take place during their menstrual cycle, causing changes to core temperature.

1.7 Limitations

The present study requires subjects to exercise at moderate to high levels (75-85% of Vo2 Max.) for a moderate period of time (20 minutes) with surface and core temperature probes attached. This may cause some subjects to lose their motivation and to become uncompelled to tolerate or complete the series of testing. Furthermore, the necessity to measure core temperature at the esophageal site may cause some potential subjects to become disinterested.

1.8 Definitions

Autonomic effector responses - Is defined as mechanisms which act to maintain or defend against changes in core temperature from "set-point".

Blood volume expansion - Is the result of increasing central blood volume.

Baroreceptors - Sensory nerve terminals that respond to changes in pressure. Also called pressoreceptors.

Cardiac output - Is defined as the amount of blood pumped by the heart in one minute.

Core Temperature - Core temperature represents the temperature at the hypothalamus. It is also described as the integration of temperatures monitored throughout the body and integrated at the hypothalamus.

Dehydration - Diminution of water content of the body tissues and fluid compartments.

Euhydration - Is defined as normal levels of fluid in the body.

Hematocrit - The volume percentage of red blood cells in whole blood; in normal males it constitutes about 45 to 50% of the whole blood volume; in the normal female it constitutes approximately 40-45%.

Hemoconcentration - Is defined as an increase in concentration or proportion of formed elements in the circulatory blood, usually resulting from the loss of plasma from the blood stream.

Hemodilution - Is defined as an increase in the plasma content of the blood with a resulting decrease in the concentration of red blood cells.

Hyperthermia - Is defined as a core temperature significantly above the normal operating range or "set-point".

Hyperhydration - Is defined as an excess of fluids in the body. Also called overhydration.

Hypohydrtaion - Diminution of water content of the body or tissues. More specifically, in the present study hypohydration refers to decreases in body water volume resulting from either

inadequate water intake or excess water output lost through sweating. Hypohydration, in the present study, is used interchangeably with dehydration.

"Load error" - Is defined as the difference between heat production and heat loss. Normally expressed as the deviation of core temperature from the normal resting average temperature or the temperature preceding a thermal challenge.

Plasma - Is defined as the yellow fluid portion of blood devoid of blood cells.

Post-Exercise Hyperthermia - An elevated core temperature from pre-exercising or "set-point" levels which occurs during exercise recovery and which has been statistically demonstrated to persist without change for extended periods.

"Set-point" - Is defined as a specific temperature at which the thermoregulatory system attempts to maintain the core temperature during heat loss or gain; a central reference temperature. It is usually expressed with the assumption that core temperature represents the temperature at the hypothalamus, but is also described as the integration of temperatures monitored throughout the body and integrated at the hypothalamus.

Thermoregulation - Is defined as the regulation of body temperature; the main control center is found within the hypothalamus.

Vasoconstriction - Is defined as the narrowing of the lumen of blood vessels (especially of arterioles) leading to decreased blood flow to the area of vasoconstriction.

Vasodilation - Is defined as the widening of the lumen of blood vessels (especially the lumen of arterioles) leading to increased blood flow to the area of vasodilation.

Vasodilation Threshold - Is the core temperature at which active skin vessel dilation occurs; resulting from an elevation in core temperature from pre-exercising or "set-point" levels. Specifically, in this study it is expressed as esophageal temperature very close to the right atria and the ascending aorta.

APPENDIX B

CHAPTER II: REVIEW OF LITERATURE

CHAPTER II

2.0 Review of Literature

2.1 Introduction

The aim of the present study was to investigate the role of water intake on post-exercise hyperthermia by comparing the post-exercise core temperature profiles of exercise-induced hypohydrated subjects and the profiles of the same euhydrated subjects. Therefore, the following review will utilize the historical and current literature to develop a framework for understanding the mechanisms and factors that control the thermoregulatory and cardiovascular responses to exercise and the effect of hydration on these variables.

2.2 Thermoregulatory Equilibrium

Factors constantly acting upon the human body require the hypothalamus to maintain core temperature at a specific operating value ("set-point") or within a certain range ("null zone") (Mekjavic et al., 1991; Sawka and Wenger, 1988). This maintenance is achieved through a dynamic system that is capable of heat generation and dissipation. If not for this system, the human body would produce heat with no avenue for exchange, resulting in a rise of core temperature by approximately 1°C per hour in a resting individual and to life threatening values within minutes in an exercising human (Murray, 1992). Fortunately, a very sophisticated system is in place whereby we lose heat to the environment, as a constant temperature gradient exists, in most cases, between our inner core and outer shell. This results in a transfer of heat from the cells of our body to the outer surface where it is dissipated to the environment either by convection, conduction, radiation or by the evaporation of sweat (Guyton, 1982).

2.3 Heat Dissipation

The two main mechanisms for the dissipation of heat, namely skin blood flow and sweat evaporation can alter heat transfer between the surface of one's body and the environment, providing for the maintenance of core temperature within safe limits. Before discussing these mechanisms in detail, it is necessary to review the physical processes which aid in the dissipation of heat. It is through these processes or pathways in which the mechanisms for heat dissipation function efficiently.

2.3.1 Pathways for Heat Exchange

The pathways for heat exchange can be divided into sensible (dry) and insensible (evaporative) heat transfer. Convection, conduction, and radiation are all forms of sensible heat exchange whereas the evaporation or condensation of water is the sole mechanism for insensible heat exchange. The specific avenue chosen by the body depends upon environmental, and behavioral conditions which will be discussed below.

Convection is heat transfer between a body and a fluid, normally air or water (Morehouse and Miller, 1971). Heat will flow from a body to the surrounding air or water if the body's surface is warmer than its environment. The reverse is also possible if the environmental temperature is greater than the body's. This will result in a positive heat gain for the body. An example of this was demonstrated by Mekjavic and Bligh (1989), who studied two different methods of establishing the core thresholds for sweating. In the first procedure, subjects exercised in room temperature (20-22°C) for twenty minutes and were then transferred to a bath of water maintained at 28°C. In this condition heat dissipation through convection was very high at the end of exercise. In the second procedure, in place of exercise, subjects were immersed in a water bath maintained at 40°C. In this situation, the thermal gradient was reversed and the direction of heat flux was from the water bath to the cutaneous tissue. If the surface of the body and the environment are equal in temperature, with no gradient, heat exchange will not occur through convection (Guyton, 1982).

Two types of convection are possible. "Free" convection is a function of fluid density and occurs if the air movement is due only to the local heating of the body. "Forced" convection is a function of fluid velocity and is important when wind movement is increased by external forces. Examples of convective heat loss include swimming in a pool or exercising outdoors on a cool day. Cold air from the surrounding environment is heated by the warmer body and then rises as it is replaced by additional colder air. Therefore, there is a constant loss of heat from the surface of the body to the environment (Santee and Gonzalez, 1988).

Conduction is heat transfer between two solid objects. The rate of conduction is dependent not only on the temperature gradient between the two bodies, but the thermal conductivity of the objects as well. For example, standing bare-footed on a skating rink would result in a greater rate of conduction than standing on a platform of wood with the same temperature as the ice. Conductive heat loss in humans is not common considering that most passive or exercising individuals have adequate insulation, minimizing the chance of direct body contact with colder objects (Santee and Gonzalez, 1988).

Radiation is heat transfer between two bodies that are not in direct contact with one another. This process occurs as infrared rays are emitted from one object to another without contact. For example, as an individual stands outside on a sunny day, heat in the form of radiation is emitted directly from the sun, indirectly as objects close by are directly heated, and indirectly through reflective solar radiation as the sun reflects off the ground. Radiation is a major contributor to thermal balance in non-exercising individuals in comfortable environments (20-22°C) as skin temperature is greater than the temperature of surrounding objects. Lastly, the positioning of the objects will determine the rate of heat exchange. As the cross-sectional area of surface exposure increases, so does the intensity of heat transfer (Santee and Gonzalez, 1988).

The three modes of heat exchange described above are all sensible or dry forms of heat transfer that involve a two-way heat exchange between two bodies. Insensible or evaporative heat transfer is the result of condensation or evaporation of water on the body's surface and is a one way heat flow from an organism to its environment. The single factor that differentiates insensible heat transfer is the need for water, which the sensible avenues do not require. Evaporation occurs through both visible sweating and non-visible water loss. Non-visible water loss, at a rate of 600 ml. per day (Guyton, 1981) occurs through the skin and lungs and accounts

for practically the entire amount of water loss when skin temperature is low (this is a result of very low sweat rates at low skin temperature). Both cutaneous and respiratory evaporation increase tremendously from rest to exercise, but water loss through perspiration (sweating) accounts for the majority of the increase in heat loss required to maintain heat balance (Fortney and Vroman, 1985).

2.4 Description of Blood Flow Patterns During Thermal Stress

The first mechanism of heat dissipation as described here is cutaneous blood flow. The following description provides not only a discussion focusing on skin blood flow, but its interaction with blood circulation to other areas within the body. This section will specifically depict the circulatory outcomes that arise from the competition between different regions for blood flow at both the onset and extended state of exercise, as well as during exercise recovery.

2.4.1 Heat Stress at Rest and Introduction of Exercise

When a resting human is exposed to a thermal stress, the need for heat dissipation arises and thermoregulatory reflexes call upon cutaneous blood flow to rid the body of heat, potentially reaching levels of 8 L/min when the skin vessels are fully dilated (Rowell, 1974). A maximum response signifies an eight times increase from 3-4 ml/100mil•min in a resting normothermic human to 24 ml/100 mil•min which represents 60% of total vascular conductance (blood flow/ arterial pressure). The increase in skin blood flow is made possible by an increased cardiac output (by 3-7 L/min), which accounts for two-thirds of the skin blood flow increase and by redistribution of blood from other regions (splachnic and renal), which accounts for one-third of the increase. This activation of skin vasodilation will maintain core temperature within its operating range under normal environmental conditions at rest, but the presence of exercise creates a more complex situation (Mekjavic and Bligh, 1989).

Although skin blood flow rises in response to an increased core temperature from both exogenous and endogenous heat loads (Johnson and Park, 1981), the introduction of exercise (endogenous) to the overall control of heat dissipation raises the question as to whether the skin

blood flow demonstrated during exogenous heat loading will remain the same or decrease. If it remains the same, thermoregulatory processes will be satisfied, but overall cardiovascular homeostasis will suffer significantly. On the other hand, if exercise competes with thermoregulatory control for blood flow, then temperature regulation will be compromised.

This challenge creates a major problem for homeostasis because the challenge from exercise to meet muscle blood flow demands are met by increased cardiac output and blood redistribution; i.e.: the same mechanisms used to satisfy increased skin blood flow. Thermoregulatory homeostasis requires blood flow as a means for transporting heat from the core to the skin, while exercise requires blood in order to transport oxygen and nutrients to working muscle (Rowell, 1974). This results in blood circulation being subjected to conflicting demands and compromise of either or both cutaneous or muscle blood flow is inevitable.

2.4.2 Muscle Blood Flow

It seems that the competition between exercising demands and thermoregulatory demands does not compromise muscle blood flow. As muscle is exercised, it utilizes oxygen very quickly, resulting in vasodilation to increase muscle blood flow by 20 times (Guyton, 1982). Muscle blood flow is maintained by an increase in overall cardiac output (Savard et al., 1988) by 5-7 times resting values (Guyton, 1982)); the activation of arterial and cardiopulmonary baroreflexes which will be discussed later (Savard et al., 1988); or as a result of redistribution of blood away from non-active areas such as renal and splachnic regions (Radigan and Robinson, 1949). The role of blood redistribution in maintaining muscle blood flow during exercise is demonstrated by the findings of Rowell (1974) who showed that redistribution of blood from renal and splanchnic areas can total 800 ml/min during moderate activity.

2.4.3 Cutaneous Blood Flow at the Start of Exercise

That cutaneous circulation is compromised as blood flow to working muscle takes precedence at the onset of exercise has been well demonstrated by studies which have shown that hand (Barger et al., 1949) and finger (Johnson and Park, 1982) temperatures fall as a result of

reductions in skin blood flow as exercise is initiated (Nakayama, 1977). Although forearm temperature falls at the onset of exercise and the absolute reduction in forearm blood becomes greater as the pre-exercise blood flow level is higher, venous occlusion plethysmography demonstrates that responses are more profound in the finger and hand (Johnson and Park, 1982).

Other factors such as exercise intensity (Johnson, 1992; Wenger, 1986) and ambient temperature (O'Leary, 1991) have an effect on the degree of vasoconstriction and hence skin blood flow at the onset of exercise. Although not linear, there is an inverse relationship between skin blood flow at the onset of exercise and exercise intensity (Johnson, 1992). It also seems that an intensity of exercise equivalent to 40% of ones maximal oxygen consumption is needed to elicit a vasoconstriction response (Wenger, 1986). If ambient temperature is low and therefore initial blood flow to the skin is low, redistribution will result in a small amount of blood being rerouted to working muscle. On the other hand, if ambient temperature is higher, with a greater initial skin blood flow then exercise will result in a greater absolute loss of blood flow to the skin (O'Leary, 1991).

2.4.4 Cutaneous Blood Flow and the Vasodilation Threshold

When exercise is extended to several minutes, thermoregulatory reflexes begin competing with exercising demands as cutaneous vasodilation results and skin blood flow is elevated (Johnson and Park, 1981). An exception to this response exists when initial skin temperature is greater than 44°C (Bevegard and Shepherd, 1966). Although vasodilation occurs when ambient temperature is below 44°C, there is still a greater degree of skin vasoconstriction influenced by exercise that does not occur during the same thermal stress at rest. This result and the skin blood flow-core temperature relationship along with the threshold for skin vessel dilation have been investigated by several authors (Taylor, 1988; Johnson, 1986; Johnson and Park, 1981; Roberts and Wenger, 1980).

The competition between thermoregulatory reflexes and exercise-associated nonthermoregulatory reflex modifies the thermoregulatory response and results in the delay of skin vessel dilation activation, contributing to a less than efficient thermoregulatory system compared to resting conditions (Johnson, 1986; Johnson and Park, 1981). Furthermore, a graded

response exists during supine exercise, wherein the threshold for vasodilation is delayed by 0.8°C as exercise intensity increases from moderate to heavy (Taylor, 1988). Supine exercise was utilized by Taylor (1988) to minimize baroreceptor-mediated sympathetic activity to the skin, but in other investigations, the threshold for vasodilation is increased further as the gravitational effects of upright posture are increased (Johnson and Park, 1981; Roberts and Wenger, 1980). In other words, forearm blood flow is lower at any given core temperature during supine or upright exercise as compared to rest for the same thermal stress.

2.4.5 Cutaneous Blood Flow With Extended Exercise

When exercise is extended beyond 5-10 minutes, core temperature continues to increase, as does the level of cutaneous vasodilation. The large majority of vasodilation occurs within 5-10 minutes of the start of skin vasodilation. Skin blood flow, thereafter rises at a continued, but slower rate (Johnson and Rowell, 1975). As exercise intensity increases, core temperature rises and the requirement for heat elimination is increased in order to achieve a thermoregulatory steady-state (Nielsen, 1966). When exercise intensity is very high, achieving a steady-state is unlikely (Shaffrath and Adams, 1984).

There exists a point where the rate of vasodilation reaches a plateau, failing to keep up with the continued rise in core temperature (Brengelmann et al., 1977). Nielsen et al., (1984) showed that forearm blood flow is limited beyond a core temperature of 38°C when subjects are exercising upright in a 45°C environment. However, they further demonstrated that forearm blood flow will continue to rise above a core temperature of 38°C if subjects are immersed in 35°C water to the level of the xiphoid process. The authors argue that peripheral blood flow is maintained by limiting the gravitational shift of blood towards the legs that takes place when subjects are not immersed. It has been further suggested that the level at which skin blood flow plateaus is below its maximum capacity and a modification in the control of skin blood flow, which will be described later, is occurring as a result of a baroreceptor-mediated response to exercise (Johnson, 1986).

2.4.6 Cutaneous Blood Flow During Exercise Recovery

As exercise is terminated and recovery begins, competition for blood flow between cutaneous vessels and muscle is no longer present. Despite this, there is no change in forearm blood flow following the first few minutes post-exercise (Johnson and Parker, 1982). Although there is no initial increase in forearm blood flow, over the succeeding minutes, there is a rise to values greater than those seen during exercise. Again, this recovery process lengthens as exercise intensifies (Taylor, 1988) and as the gravitational effects of upright posture becomes greater (Roberts and Wenger, 1980). Thoden et al., (1994) evaluated post-exercise esophageal profiles and demonstrated that there was an elevation in post-exercise core temperature. Although it was not suggested, perhaps this elevation was partly a result of vasodilation reaching a plateau as demonstrated by Brengelmann et al. (1977) during exercise.

In summary, evidently as exercise begins, competition between thermoregulatory and exercising demands result in cutaneous vasoconstriction as muscle requirements are met prior to thermoregulatory requirements. This is signified by not only the decrease in skin blood flow at the onset of exercise, but by the delay in the core temperature threshold for cutaneous vasodilation and the upper limit of skin vasodilation demonstrated as exercise is prolonged. The delay in increased blood flow during recovery and the post-exercise core temperature elevation demonstrated by Thoden et al. (1994) further supports the idea that thermoregulatory functioning is of secondary priority.

2.5 Mechanisms Controlling Blood Flow Patterns

The description of blood flow during exercise, as provided above, occurs as a result of several mechanisms acting upon both muscle and skin. Although several questions remain unanswered concerning the control of muscle and skin blood flow, it is understood that the control of muscle blood flow is achieved mainly through local regulation whereas skin blood flow is controlled almost entirely by nervous mechanisms (Guyton, 1982). The mechanisms involved in blood flow control and their respective roles have been investigated by several authors (Kellog and Johnson, 1991; Kellog et al., 1990; Nose, 1990; Kenney et al, 1991; Johnson, 1986; Nielsen

et al., 1984; Roberts and Wenger, 1980; Rowell, 1974; Bevegard and Shepherd, 1967; Blair et al., 1960) and their findings are presented below.

2.5.1 Characteristics of the Vasoconstrictor and Vasodilator Systems

Before describing how the hypothalamus controls blood flow in the skin and muscle in response to changes in core temperature, it is necessary to review characteristics of the vasoconstrictor and vasodilator systems; mechanisms responsible for the control of blood flow during exercise. Skin of non-acral (feet, nose, hands, lips, and ears), acral (trunk, head, and limbs), and skeletal muscle regions are all supplied with sympathetic adrenergic vasoconstrictor fibers which release norepinephrine (Johnson, 1986). Only skin arterioles in non-acral regions are also controlled by a sympathetic active vasodilator system. Although the mechanism responsible for the functioning of the active vasodilator remains unknown, several investigators believe it is related to sudomotor control (Johnson, 1986; Brengelmann et al., 1981). In other words, arterioles in acral skin and skeletal muscle of humans are innervated solely by vasoconstrictor nerves, while arterioles in non-acral skin are innervated by both vasoconstrictor and vasodilator nerves.

2.5.2 Increased Muscle Blood Flow

When exercising results in muscle utilizing oxygen very quickly, the mechanisms responsible for the increased muscle blood flow are mainly the release of vasodilator substances and the secondary importance of reduced sympathetic vasoconstrictor nerve presence (Bevegard and Shepherd, 1967). When oxygen content in muscle becomes deficient, vasodilator substances, controlled by local regulation, such as adenosine, lactic acid, and acetylcholine are released which increase muscle blood flow following oxygen depression (Guyton, 1982). Although the secretion of norepinephrine represents a small degree of vasoconstriction in muscle compared to other areas of the body, stimulation or lack thereof can play a secondary role in varying the amount of muscle blood flow (Guyton, 1981).

2.5.3 Vasoconstriction at the Start of Exercise

The cutaneous vasoconstriction, seen at the onset of exercise is mediated by the sympathetic nervous system and is a result of the activation of noradrenergic vasoconstrictor fibers (Bevegard and Shepherd, 1966). Administration of substances such as bretylium (Blair et al., 1960), which block norepinephrine release, and surgical sympathectomy (Bevegard and Shepherd, 1966) eliminated the vasoconstrictor response at the onset of exercise, demonstrating vasoconstrictor fiber control. In addition, Kellog and Johnson (1991) demonstrated that regardless of the internal state prior to exercise (normothermia vs. hyperthermia), the mechanism for reduced skin blood flow is sympathetic nervous system.

2.5.4 Vasodilation During Exercise and its Threshold

The mechanism underlying the eventual rise in skin blood flow during exercise is not universally agreed upon. Some authors believe that the cutaneous vasoconstrictor response is temporary and the following rise reflects an increase in the vasodilator response to a rising core temperature and a reduction in the vasoconstrictor response to exercise (Johnson, 1986). As suggested by Johnson (1992), this explanation implies that the level of skin blood flow at any core temperature would be the same for the same given thermal stress whether the subject was at rest or exercising. The demonstration that the vasoconstriction response by other regional circulations is sustained during exercise argues against this explanation (Rowell, 1974). On the other hand, the level of cutaneous blood flow throughout exercise is possibly dictated by the competition between the vasoconstrictor reaction during exercise and the vasodilator response to heat dissipation. According to this explanation, the skin blood flow response to rising core temperature would be greater during rest than during exercise (Johnson, 1986). Support for this explanation can be made by the fact that the level of skin blood flow at any core temperature is higher at rest than during exercise, indicating that a sustained vasoconstrictor response to exercise exists.

According to Kellog et al. (1991), the neural mechanism which is modulated to result in an upward shift in the threshold for vasodilation during exercise is probably the cutaneous active

vasodilator system. In their experiments, certain sites were pretreated with or without bretylium, in order to eliminate and maintain vasoconstrictor function respectively. This demonstrated that the internal temperature at which vasodilation began was identical between the treated and non-treated sites during rest and exercise and that the shift is a result of an exercise-induced delay of the vasodilator system, while the vasoconstrictor system does not play a role. These findings are in contradiction to those of Roberts and Wenger (1980), who suggest that the most likely cause of reduced forearm blood flow during upright exercise is an increase in cutaneous sympathetic vasoconstrictor activity, secondary only to reduced cardiac filling pressure as baroreceptor reflexes are activated. Regardless of whether sympathetic vasoconstrictor activity is present, the involvement of the active vasodilator system in shifting the threshold during exercise, as described here, contrasts the sole involvement of the vasoconstrictor system in controlling blood flow away from skin at the onset of exercise, as described above.

2.5.5 Limitation of Skin Blood Flow During Exercise

The neural mechanisms that seem to be responsible for the limitation of skin blood flow during prolonged exercise appears to be the restraint on increasing active vasodilator activity and baroreceptor reflex activity. The role of the vasodilator system was demonstrated when blockade of active vasoconstriction did not affect the establishment of the plateau in skin blood flow during exercise in warm settings (Kenney et al, 1991).

2.5.6 The Role of Baroreceptors in Limiting Vasodilation

Although the restriction on increasing active vasodilator activity appears to be responsible in limiting cutaneous blood flow, the role of baroreceptors as the main barrier for continued skin blood flow has been hypothesized by several investigators (Johnson, 1992; Kellog et al., 1990; Bevegard and Shepherd, 1966). Baroreceptors are nerve endings located in the walls of the aortic arch & the carotid arteries and they transmit impulses to the medulla. They plays a key role in short-term adjustements of blood pressure when abrupt changes in blood volume, cardiac ouput, or peripheral resistance occur, as in exercise (Guyton, 1982). Any rise in pressure stimulates the

baroreceptor reflex and causes a transmition of signals to the central nervous system, which inhibits the vasomotor center, causing a decrease in the number of sympathetic impulses transmitted to the heart and to the blood vessels. This has three main effects in lowering pressure back to normal which include a decrease in the pumping activity of the heart, a increase in the ease of blood flow, as vasodilation occurs throughout the peripheral circulatory system, and lastly a decrease in the strength of contraction of the heart. A drop in pressure has the opposite effect, raising pressure back to normal (Rowell and O'Leary, 1990).

As core temperature rises, so does skin blood flow and venous volume, influencing cardiopulmonary filling, stroke volume, pulse pressure, and inevitably baroreceptor activity such that any increase in skin blood flow resulting from thermoregulatory drives will be reversed by the baroreceptor reflex (Nielsen et al., 1984). Johnson (1992) argues that blood volume distribution induced by barorecptors is the main factor in the upper limit to skin blood flow during exercise. He points to studies which demonstrate the elimination of the skin blood flow plateau by immersing exercising subjects in water where distending pressure is maintained (Nielsen et al., 1984), and by infusing subjects with saline solution (Nose, 1990). The action of the baroreceptor reflex is most likely to be through the active vasodilator system, demonstrated by surgical slicing of the baroreceptor filled carotid sinus, which results in a larger forearm vasodilation during exercise as opposed to rest (Bevegard and Shepherd, 1966).

Regardless of the sensory mechanism responsible for the skin blood flow upper limit, it is the constraint on skin vasodilation during exercise which maintains the demand for skin blood flow within the capability of the heart to raise cardiac output and visceral circulations constrict respectively.

2.5.7 Limitation of Skin Blood Flow During Exercise Recovery

One mechanism which seems to be responsible for the delayed and depressed skin blood flow during recovery is the baroreceptor reflex. The recovery period is associated with pooling of blood in dependent veins and a substantial reduction in arterial and cardiopulmonary pressures immediately following exercise. Holtzhausen and Noakes (1992) investigated the mechanism responsible for a condition known as Exercise-Associated Collapse (EAC) whereby long-distance

runners collapse when exercise has ended and recovery has begun. They noted that pooling of blood to the accommodating veins of the lower limbs when exercise has terminated results in postural hypotension. This pooling induces the baroreceptor reflex (Roberts and Wenger, 1980) and results in cutaneous vasoconstriction that overrides the vasodilation expected with the beginning of exercise recovery. This process is similiar to the description provided in the previous section on the limitation of skin blood flow during exercise, where vasodilation plateaus as well. Thus, the pattern of change of skin blood flow during recovery seems to be a combination of a declining response to the previous period of exercise and possibly an elevated vasoconstrictor activity with changes in arterial or cardiopulmonary pressures.

2.6 Description of Sweat Output Patterns During Thermal Stress

The second main mechanism for heat dissipation as described here is sweat evaporation. Although blood flow to the skin provides effective dissipation during heat stress, evaporation of sweat plays a significant role in addition to skin blood flow and in situations where skin blood flow is limited, such as when exercise intensity (Sawka and Wenger, 1988) or ambient temperatures (Fortney and Vroman, 1985) are very high. The characteristics and factors controlling sweat output have been studied by several investigators (Murray, 1992; Nadel et al., 1980; Fortney et al., 1981; Cabanac and Massonnet, 1977), and a review of their findings and others are presented below.

2.6.1 Factors Affecting Sweat Output

Sweat output is a vital thermoregulatory response that is triggered by an increase in core temperature. During exercise, sweating is initiated at the expense of intracellular and extracellular fluid volumes (Murray, 1992), with the percentage loss from each of the fluid compartments varying depending upon the volume of sweat output (Sawka, 1988). As the intensity of exercise and hence the amount of body water loss increases, the proportion of water loss from extracellular and intracellular spaces becomes smaller and greater respectively (Durkot et al., 1986). In other words, when exercise intensity is high, fluid loss is mainly from extracellular

space, whereas when intensity is high, fluid loss occurs mainly from intracellular space. Furthermore, sweating contributes to the depletion of vascular volume and cardiac filling pressure (Fortney et al., 1981a) and is dependent on several factors including environmental conditions, clothing characteristics, and individual physical activity levels (Sawka, 1988). Heat dissipation through sweating is an important mechanism for individuals at rest as it accounts for 20 to 25% of total heat loss. Moreover, perspiration becomes increasingly more important as exercise intensity (Sawka and Wenger, 1988) or ambient temperature (Fortney and Vroman, 1985) increases.

Although sweating creates the greatest potential for heat loss during high intensity activity or when high ambient temperatures exist, in order to function as an effective mechanism, sweat must evaporate. Effective evaporation is limited if humidity (Sawka, 1988) is high or if insufficient air movement exists (Pandolf, 1979), as surrounding air becomes saturated with water vapor. Another factor that limits sweat output is a phenomenon known as hidromeiosis. Hidromeiosis occurs as the epidermal cells enlarge because of a mechanical blockage of the sweat ducts (Brown and Sargent, 1965). Initially it was believed that decreases in subjects sweat rate over time was a result of tiring sweat glands, caused by a lack of neurotransmitters or fuel needed for sweating (Fortney and Vroman, 1985).

2.6.2 Stages and Composition of Sweat Output

Eccrine sweat glands are mainly responsible for sweat production. These tubular structures contain a secretory coil that contain clear cells. The clear cells play a significant part in sweat production as they are involved in the secretion of the precursor fluid to sweat, which is provided by the plasma (Sawka et al., 1988). The release of sweat from the duct portion of the eccrine sweat gland is a process involving two stages. First, sodium ions are actively transported to the secretory coil, where water follows. This results in an isotonic precursor fluid (plasma) in the coil portion of the gland. The fluid is then transported into the duct where sodium is reabsorbed in an excess of water (Sato, 1977). Although water is the main substance lost in sweat, sodium chloride, potassium ions, urea, and lactic acid are all lost as well. At low rates of sweat secretion the concentration of these substances can be extremely high, but at high rates of

secretion the concentration of urea for example is only two times that of the plasma (Guyton, 1981).

2.7 Mechanisms Controlling Sweat Output Patterns

The control of sweat output is dependent upon not only mechanical factors as described above, but on neural and hormonal ones as well. Cholinergic sympathetic nerve fibers originating from the anterior hypothalamus elicit the secretion of the plasma precursor fluid which stimulates sweat production (Guyton, 1981). Afferent input, proportional to core temperature is responsible for this neural output. The role of adrenergic substances such as epinephrine in the alteration of sweat output is somewhat controversial (Fortney and Vroman, 1985) as is the functioning of other agents such as antidiuretic hormone (Fortney et al., 1981a), and Bradykinin (Brengelmann et al., 1981). Although sweat glands in most parts of the body do not have adrenergic innervation, it is thought that they can still be stimulated by adrenergic substances circulating in the blood. Furthermore, it seems that it might be possible that the sweat glands of the hands and feet have some adrenergic innervation. It is also known that both α– and B-adrenergic nerve fibers are present in the eccrine sweat glands (Sato, 1977). The alteration of sweat production through manipulation of the sodium to calcium ratio has been studied by Nielsen (1974) and by Feldberg et al. (1970). While Feldberg et al. (1970) suggested that the regulation of core temperature in unananthetized cats was altered by a shift in the sodium to calcium ratio, Nielsen (1974) demonstrated that by exercising subjects after ingesting hypertonic sodium chloride or calcium chloride solutions, that only ingestion of sodium decreases sweating. On the other hand, Doris and Baker (1982) demonstrated by injecting water into the lateral cerebral ventricle of cats with elevated core temperatures, that evaporative cooling increased. These results were obtained despite no adjustment in the sodium to calcium ratio and the authors suggest that these results indicate that central osmoreceptors may play a role in evaporative cooling.

2.8 Hydration and Its Effect on Heat Exchange

The effect of hydration state on thermoregulation and cardiovascular functioning has been investigated extensively (Noakes, 1993; Montain and Coyle, 1992; Fortney et al., 1981; Nadel et al., 1980). Although the majority of studies have focused on hydration and sweat output, implications regarding skin blood flow have also been considered. Furthermore, the relationship between hydration state and level of blood volume have also been investigated and implications for thermoregulatory functioning have been hypothesized. Although several controversies seem to exist in the literature, there are some basic findings that have been established that clarify consequences of hydration on heat exchange.

2.8.1 Hydration and Sweat Output

Although sweating is required to maintain a safe core temperature, it represents a loss of fluid that often reaches values as high as 2 liters per hour in acclimatized individuals (Bonner et al., 1976). When an individual's sweat output exceeds his or her water intake, a body water deficit results, causing a state known as dehydration or hypohydration (Nadel et al., 1980). For consistency, the remaining review will use the term hypohydration to mean any decrease in body water volume. The effect of hydration level on sweat output appears to be inconsistent. Several investigators have demonstrated that sweating rate decreases with hypohydration (Greenleaf and Castle, 1971; Ekblom et al., 1970), whereas others have failed to show this effect (Montain and Coyle, 1992a; Montain and Coyle, 1992b; Gisolfi and Copping, 1974). An earlier investigation, demonstrated that sweating rate is unaffected by hydration level, except in cases where extreme fluid deficit exists (Ladell, 1955).

Predicted sweating rates of athletes differing in size and running speed were investigated by Barr and Costill, (1989) and they demonstrated that the size of an individual is an important determinant of sweating rate. Apparently, heavy individuals running at slow speeds can generate higher sweat rates than smaller runners who are exercising much quicker. Sweating rates are also increased during high ambient temperatures or during indoor activity when adequate convective cooling is limited (Brown and Banister, 1985). Noakes (1993) suggests that the discrepancy

between hydration levels and sweat output may be explained by large individual differences and environmental factors.

Although the physiological mechanisms controlling the possibly reduced sweating output during hypohydration have not been isolated, the effect of baroreceptor activity (Fortney et al., 1981a) and plasma volume changes (Senay, 1979) have been investigated as explanations. It has been suggested that by modifying the activity of the atrial baroreceptors, hypohydration reduces atrial filling pressure and alters information to the hypothalamus, resulting in reduced sweat output (Fortney et al., 1981a). On the other hand, it has been reported that during hypohydration a strong inverse relationship exists between sweat rate and plasma osmolality so that states of hyperosmolality are associated with reduced sweat output. The mechanism mediating this relationship is argued to be a direct central nervous system effect on the hypothalamic thermoregulatory centers (Senay, 1979).

2.8.2 Hydration and Blood Volume

The connection between fluid intake on blood volume responses to exercise is an important consideration in determining the underlying mechanism to thermoregulatory and cardiovascular consequences. Are these adjustments a result of reductions in hydration levels per se or are they outcomes of reduced blood volumes demonstrated during exercise as well? Water intake in the amount necessary to replace sweat output has been shown to have an effect on this exercise-induced blood volume decrease (Harrison, 1978). Perhaps this will partially explain the fluid intake attenuation of core temperature as reviewed below.

The methods used to investigate body water and blood volume deficits have varied considerably and one must be careful in interpreting results based on these differences. Firstly, fluid intake can be administered in several different ways including, direct injection in to the blood stream, injection beneath the skin, and oral ingestion, where fluid is absorbed from the gastrointestinal tract into the blood (Guyton, 1981). Secondly, the composition of fluid intake can vary considerably. Most studies during the past thirty years have focused on the evaluation of carbohydrate ingestion (Noakes, 1993), while several studies have compared the cardiovascular and thermoregulatory performance of water deprived and compensated subjects (Montain and

Coyle, 1992a, Fortney et al., 1981; Deschamps et al., 1980). Furthermore, other studies have infused saline solution intravenously to reverse the hypohydrated-induced fall in plasma volume associated with exercise and to differentiate between the effects of reduced plasma volume and changes in serum osmolality (Montain and Coyle, 1992b). These differences can all result in inconsistent findings concerning the mechanism responsible for cardiovascular and thermoregulatory consequences of exercise.

2.8.3 Thermoregulatory Responses to Hypohydration and Hypovolemia

Studies investigating the thermoregulatory response to exercise in hypohydrated subjects demonstrated that body water deficits will result in greater heat storage and increased core temperatures compared to that of euhydrated or hyperhydrated subjects (Montain and Coyle, 1992; Sawka, 1988; Greenleaf and Castle, 1971; Gisolfi and Copping, 1974; Strydom and Holdsworth, 1968). After reanalyzing the data of Adolph et al. (1947), Sawka (1988) demonstrated that core temperature increases linearly with the percentage decrease in body water loss. These results were supported by Greenleaf and Castle (1971), who reported that body temperature increased by 0.1° C for each one percent decrease in body weight during submaximal exercise. The core temperature threshold for the onset of sweating is also affected by the level of hydration (Sawka et al., 1985). Mean sweating rates and core temperatures of euhydrated, low hypohydrated, moderate hypohydrated, and high hypohydrated subjects were measured throughout exercise and it was demonstrated that there is a progressive delay in the core temperature onset of sweating as the degree of hypohydration increased (Sawka et al., 1985). Johnson and Park (1981) further argue that although hypohydration lengthens the onset of sweating, it has a more profound effect on the threshold for vasodilation.

Montain and Coyle (1992a) investigated the effects of varying levels of hypohydration on core temperature during exercise, where subjects received varying amounts of fluid that were sufficient to replace 0, 20, 48, and 81% of sweat loss during two hours of moderate intensity exercise. They demonstrated that core temperature increases are directly related to the degree of hypohydration and that as the quantity of fluid ingested became greater, so did the attenuation of

hyperthermia. Although the authors were unable to isolate the underlying mechanism, these results suggest that fluid replacement has a significant effect on the attenuation of hyperthermia.

In an apparent attempt to isolate the underlying mechanism, Montain and Coyle (1992b) followed up on their previous study by investigated the role of blood volume on hypohydration. Because hypohydration results in reduced blood volumes (Sawka et al., 1979), and because Johnson and Park (1981) argue that this may delay the onset of skin blood flow, the possibility of hypovolemia acting to elevate core temperature during exercise seemed likely. Although Montain and Coyle (1992b) had already determined that minimizing hypohydration reduces the extent of hyperthermia, they investigated whether fluid replacement would attenuate hyperthermia as a result of increases in blood volume. Exercising subjects received either fluid replacement or blood volume expander. They demonstrated that the rise in core temperature is reduced during prolonged exercise (60-80 minutes) in proportion to the amount of fluid intake and is least when the rate of fluid ingestion approaches the sweating rate. They further showed that blood volume expansion increased blood volume levels to the same degree as fluid replacement, however, core temperature was significantly lower for the fluid replacement group. Based on this present study and their previous study (Montain and Coyle, 1992a), Montain and Coyle (1992b) concluded that fluid replacement attenuates hyperthermia, but not because this results in an elevation of blood volume.

The findings of Montain and Coyle (1992b) seem to somewhat contradict those of Fortney et. al, (1981a) who demonstrated that blood volume withdrawal results in reduced forearm blood flow as esophageal temperature rises, while increased blood volume results in increased skin blood flow at a given core temperature. While it was demonstrated that hypovolemia results in greater heat storage and an elevated core temperature, hypervolemia did not result in the attenuation of hyperthermia. Although this study did not investigate the impact of maintaining blood volume, it seems that hypovolemia resulting from exercise may help explain the underlying mechanism responsible for exercise-induced hyperthermia and this reduction in blood volume may result from hypohydration.

In addition to their findings above, Fortney et al. (1981b) demonstrated that during 20 minute cycle ergometer exercise at approximately 65% of ones maximum oxygen consumption, a state of hypovolemia occurs as blood volume can decrease by up to 425 milliliters. They further

showed that this results in a delay in the onset of sweating and a lower sweat rate at any given core temperature compared to conditions where blood volume is maintained (Fortney et al., 1981b). Harrison (1978) suggests that this blood-volume dependent ability to sweat efficiently seems to be dependent on changes in plasma osmotic pressure. Although this reduction in sweat rate has only been demonstrated in non-active sites such as the arm or chest, when blood volume is reduced, skin blood flood falls even during moderate level activity. On the other hand, as demonstrated by Fortney et al. (1981a), increasing blood volume artificially, will result in increased skin blood flow. Therefore, it seems possible that heat dissipation during exercise may be partially mediated by reflexes responsive to the level of blood volume and that the findings by Montain and Coyle (1992b) are in contradiction to this hypothesis.

2.8.4 Factors Controlling Thermoregulatory Consequences

The factor controlling the hypohydration mediated increase in core temperature is not specifically known, but Sawka (1988) and Noakes (1993) offer several possibilities. Sawka (1988) compares the arguments between an increase in metabolic heat production or a decrease in heat loss in accounting for the hypohydrated-mediated hyperthermia. An investigation on the effect of hydration on heat production demonstrated that hypohydration does not affect the rate of aerobic or anaerobic metabolism during exercise (Saltin, 1964) and therefore can not be the factor causing increased core temperature. On the other hand, Sawka (1988) argues that because hypohydration affects both sensible and insensible avenues of heat exchange, decreased heat dissipation capabilities must be the controlling factor in the hypohydration mediated core temperature increase. Support for this hypothesis has been provided by Sawka et al. (1984) who showed that during states of body water deprivation, sweating volume is lower at any given body temperature and therefore the potential for heat dissipation through sweating is reduced.

Fortney et al. (1988) interpreted similiar findings in a different way. The authors showed that infusing saline solution to maintain plasma volume resulted in a reduction in sweating rate as well. However, they concluded that this was not a sign of reduced heat dissipation capabilities but rather a shift in heat dissipation from evaporative cooling, through sweating, to convective cooling, through skin blood flow. The overall result was a lower core temperature compared to a

control condition whereby no fluid was administered. On the other hand, reductions in serum osmolality and sodium concentrations, which resulted from hypohydration caused a delay in the onset of sweating and a shift to the right in the threshold for skin vessel dilation.

Montain and Coyle (1992b) support the arguments of Sawka (1988) and Sawka et al. (1984) above by suggesting that the reductions in body water volume result in hyperthermia by reducing the ability of the body to dissipate heat. In their study described above, fluid ingestion weakened hyperthermia by increasing skin blood flow during the latter stage of exercise. These results suggest that if hypohydration is minimized, the competition for fluid volume between the central and peripheral circulations will be decreased. This will allow for proper cardiovascular functioning and maintenance of core temperature within relatively safe limits. Another factor that supports the idea that decreased heat dissipation capabilities must be the controlling factor in the hypohydration mediated core temperature increase is that fluid ingestion results in a higher fraction of forearm blood flow during exercise (Montain and Coyle, 1992a). This may act to weaken the elevated core temperature seen during exercise as greater degrees of hypohydration are associated with greater reductions and increases in skin blood flow and core temperature respectively (Montain and Coyle, 1992b).

On the other hand, Gisolfi and Copping (1974) investigated the role of sweat loss, expressed as a percentage of body weight, in predicting core temperature elevation during prolonged treadmill running. In contradiction to the previous findings above, but in support of Fortney et al. (1988), the authors determined that the reductions in core temperature from fluid ingestion were not a result of increased sweat output. This was demonstrated by additional findings where sweat rates were not reduced following progressive hypohydration. Although sweat output was increased during experiments where core temperature declined, the authors considered this a "wasteful" increase. However, they do not offer a definitive answer to the decline in body temperature, suggesting that plasma osmolarity may play a role.

Noakes (1993) further investigated the specific mechanism which regulates the physiological responses to an exercise-induced hypohydration. He suggests that the two leading possibilities are a change in serum osmolality or a change in plasma volume. Studies utilizing isotonic fluid infusion to maintain fluid volumes, have clarified the issue by increasing plasma volume without altering serum osmolality (Montain and Coyle, 1992a; Montain and Coyle,

1992b). By comparing the results of studies investigating the different responses between fluid ingestion and plasma volume expansion, Montain and Coyle (1992b) suggest that the lower core temperatures seen when fluid is ingested is not occurring because of maintained plasma volume levels, but rather as a result of reduced serum osmolality and serum sodium concentrations. They infer that the maintenance of serum osmolality and sodium concentration results in higher cutaneous blood flow and an associated lower core temperature during exercise. As pointed out by Noakes (1993), the results of Montain and Coyle (1992b) support the early proposal of Dill (1938) who suggested that the intent of fluid ingestion should be to maintain serum osmolality and sodium concentrations. So it seems that although maintaining plasma volume through fluid ingestion is a reasonable goal, the main mechanism that regulates the physiological responses to an exercise-induced hypohydration may be changes in serum osmolality and sodium concentrations.

2.8.5 Hormonal Changes

The effect of maintaining serum osmolality and sodium concentrations may possibly have an attenuating result on hypohydration by reducing certain hormonal secretions. Exercise leading to hypohydration will result in the secretion of antidiuretic hormone, resulting from increases in serum osmolality (Thrasher et al., 1981). Water intake during exercise, on the other hand, causes serum osmolality to remain stable or decrease, resulting in a reduction in antidiuertic hormone secretion. Therefore, hormonal factors may be an additional factor which regulates the physiological responses to an exercise-induced hypohydration.

2.8.6 Hyperhydration and Hypervolemia

While documentation of the effects of hypohydration on core temperature have been established, the effects of hyperhydration are not as clear. Several investigators have looked into the possibility of hyperhydration reducing core temperature and improving performance (Fortney et al., 1981a; Fortney et al., 1981b; Greenleaf and Castle, 1971; Moroff and Bass, 1965), and although the findings are not uniform, Sawka et al. (1988) states that it does seem to delay the

onset of hypohydration. Fortney et al. (1981a) demonstrated that although skin blood flow was higher during hyperhydration and hypervolemia for a given core temperature compared to euhydrated levels, they state that increased blood volume does not reduce core temperature. On the other hand, in a second study where plasma volume was artificially increased, core temperature decreased, with no change in sweat output however (Fortney et al., 1981b). While Moroff and Bass (1965) demonstrated similiar results to Fortney et al. (1981b), Greenleaf and Castle (1971) contradicted their findings and resembled the results of Fortney et al. (1981a). These differences indicate that any possible physiological advantages of hyperhydration are small and can possibly be explained by differences in protocols as suggested by Sawka et al. (1988).

2.8.7 Cardiovascular Responses to Hypohydration and Hypovolemia

As described above, exercise dramatically increases metabolic rate and requires adequate blood volume to provide muscle with essential nutrients as well as to rid the body of excess heat. The dissipation of heat that takes place as exercise is extended beyond several minutes occurs as evaporative, convective, and radiative heat loss is facilitated as central blood volume is lost to the periphery (Nielsen et al., 1984; Rowell, 1974). However, The competition for blood flow during exercise, between skin and muscle, eventually not only results in a less than efficient heat loss system, but a reduction in cardiac output as well. Moreover, sweat secretion can further reduce the blood volume as plasma provides the precursor fluid needed for secreted sweat. This further increases the difficulty of the cardiovascular system to provide adequate blood flow to muscle and skin (Sawka et al., 1985).

Montain and Coyle (1992a) investigated the effects of varying levels of hypohydration, as described above, on heart rate and stroke volume. They demonstrated that cardiovascular drift is directly related to the degree of hypohydration and that the degree to which heart rate increases, and cardiac output and stroke volume decrease, is directly related to the amount of body weight loss. On the other hand, when hydration is maintained, cardiac output and stroke volume are also maintained, while heart rate still increases (Hamilton et al., 1991). Therefore, the mechanism controlling the rise in heart during exercise must be, at least in part, something other than hypohydration.

Even during moderate level exercise with a moderate thermal strain, the presence of hypohydration will result in a decreased blood volume, which results in reduced stroke and end-diastolic ventricular volumes compared to similiar euhydrated strains (Sawka et al., 1979). In order to compensate for these decreases, heart rate rises, but is unable to compensate for the decreased stroke volume and, inevitably, results in a decreased cardiac output. The overall decrease in cardiac output occurs because of the previously discussed competition between central and peripheral circulation for the greatly reduced blood volume. As exercise is prolonged, skin vasodilation occurs, decreasing venous resistance, pressure, and return so that cardiac output is decreased from euhydrated levels (Nadel et al., 1979). Furthermore, an earlier investigation argued that ingestion of fluid during exercise, to weaken body water deficit is associated with a maintained blood pressure when exercise is terminated and recovery commences (Eichna, 1945). This is an important factor considering heart rate, which acts to maintain cardiac output during exercise, begins to fall rapidly as exercise is terminated. If blood pressure is not maintained through some other means, blood circulation will have a very hard time meeting the requirements of muscle, skin, and vital organs. This may possibly explain the post-exercise elevated core temperatures reported by Thoden et al. (1994).

2.8.8 Hydration and Rates of Absorption

The argument as to whether fluid intake improves thermoregulatory and cardiovascular homeostasis and how is further complicated by the possibility that not all of the fluid ingested is necessarily utilized. It has been reported that the maximum rate of water absorption from an isotonic with a sodium concentration of 110 mmol/liter was only 800 ml/hr (Davis et al., 1980). Noakes (1992), showed that fluid replacement during and after exercise equalling fluid loss during exercise was not entirely accounted for in the intracellular and extracellular fluid pools. This author suggests that this probably indicates that the fluid ingested was not entirely absorbed, possibly remaining in the gastrointestinal tract. This could further help explain contradictory findings demonstrated above based on individual differences alone.

2.9 Sensors for Core Temperature Regulation

The maintenance of core temperature within its specific operating point or range is dependent upon temperature sensors located in the skin and the body's core (Jessen and Mayer, 1971). The role of the skin sensors in the regulation of core temperature is secondary to deeper, internal detectors and is more sensitive to falls in core temperature. Normal responses to heat production, as in exercise, are for the most part attributable to drives from internal temperature sensitive mechanisms (Johnson et al., 1986; Proppe et. al., 1976). Although certain areas are well documented as temperature sensitive, there still seems to exist some uncertainty as to the location of others. This section will review the current understanding of how core temperature changes are detected and adjusted for so that maintenance of core temperature homeostasis is realized.

2.9.1 Hypothalamus

The hypothalamus is considered the main center for temperature regulation and accounts for the bodies capability of maintaining core temperature between 36.3°C and 37.4°C when ambient temperature varies from 14.5°C to 60°C during resting conditions. Core temperature is regulated by nervous feedback mechanisms that operate through temperature regulating centers located in the hypothalamus and are able to function effectively as temperature detectors determine fluctuations in body temperature. The thermosensitive sites involved in temperature regulation are the pre-optic and anterior hypothalamic thermosensitive neurons. The preoptic area of the hypothalamus is the main control center for temperature regulation, while the anterior hypothalamus plays a smaller role. Adjustments for detection in core temperature elevations occur as neurons located in these areas increase their firing rate, while stimulation of the preoptic area results in sweating to reduce body temperatures. Early studies such as Barbour (1912) identified the hypothalamus as a temperature sensitive area by showing that cooling the corpus striatum of rabbits resulted in a rise in core temperature, while heating the same area resulted in the opposite effect. More recently studies such as Jessen and Clough (1973) investigated the role of the hypothalamus in controlling core temperature. They stimulated the hypothalamus' of four

goats with warm and cold water while monitoring core temperature responses and demonstrated as well that hypothalamic thermal stimulation generates the opposite effect on core temperature.

2.9.2 Spinal Cord

In addition to the hypothalamus, other core temperature areas have been investigated as temperature sensitive areas. Jessen and Mayer (1971) studied the effect of altering the temperatures of the hypothalamus and the spinal cord of conscious dogs in order to investigate their roles in the regulation of core temperature. Thermodes were artificially implanted in dogs prior to testing. The thermodes were then perfused with water of varying temperatures while rectal and hypothalamus temperatures were measured. Heat production and loss were measured by shivering and respiratory evaporative heat loss respectively. The authors demonstrated that cooling or heating these areas (via the thermodes) resulted in effector responses acting to maintain internal temperature within its unaltered range. For example, when the thermodes were perfused with cold water (20°C), heat production mechanisms, in the form of shivering and panting would result in an elevated core temperature. Furthermore, although the thresholds for shivering and evaporative heat loss differed somewhat for the hypothalamus and the spinal cord, the authors concluded that the hypothalamus and the spinal cord appear to be equivalent temperature sensors within the body core of the dog.

2.10 Reflexive Versus Local Influences of Heat Dissipation

Locally heating skin results in increased blood flow only in the heated area. Core temperature and skin blood flow in other areas does not increase when local heating is less than 42.5°C. However, as core temperature increases, skin blood flow increases regardless of whether local heating is used or not. Therefore, reflexive drives for heat dissipation is not only stronger than local drives, but local drives do not seem to weaken reflexive ones (Johnson et al., 1976).

2.11 Theories of Temperature Regulation

It is well understood that that the body loses heat by vasodilation and sweating and produces or conserves heat through vasoconstriction and shivering as the pre-optic and anterior hypothalamic thermosensitive neurons act as the main integrators of body temperature. On the other hand, a universal model for temperature regulation does not exist, especially in the presence of certain factors such as exercise. While several investigators have demonstrated support for the idea that core temperature is maintained at a certain point, other observations argue that core temperature is maintained within a larger range.

2.11.1 "Set-point" Hypothesis

Sawka and Wenger (1988), refer to the elevation in core temperature from its pre-exercise set-point, as a load error. They demonstrated that any deviation that drives core temperature away from the set-point will result in the initiation of effector responses. Moreover, the authors predict that the effector responses will be active until heat displacement is reversed to the original "set-point". This theory infers that set-point is a central reference temperature whereby heat dissipating responses function to maintain it.

Cabanac and Massonnet (1977) determined the patterns of sweating (heat dissipating) and shivering (heat activating) responses by immersing subjects in water at 38, and then 28 degrees. They monitored both sweat rates and oxygen consumption (as a measure of shivering), and then demonstrated that the thresholds for sweating and shivering coincide. This finding supports the load error theory as later described by Sawka and Wenger (1988) in that the effector responses seem to be continually active until heat or cold displacement is reversed to "set point".

2.11.2 "Null-zone" Hypothesis

Jessen and Ludwig (1971) challenged the idea of an existence of a thermoregulatory "set-point". They demonstrated that when data for evaporative heat loss and heat production in dogs

are plotted on the same core temperature axis, a dead-band or null zone exists whereby thermoregulatory mechanisms are shut off.

Mekjavic et al. (1991), designed an experimental protocol which demonstrated that the core temperature thresholds for sweating and shivering do not coincide. Rather, the authors argued that a null zone exists whereby neither thermoregulatory processes are working. Subjects exercised and then recovered in water at 28 degrees. Core and skin temperature were measured along with sweating and shivering rates. By conducting the exercise in water at 28 degrees, cooling was simply induced by termination of exercise. This procedure minimized the changes in skin temperature and its effect on esophageal temperature that occurred in studies by Cabanac and Massonnet (1977), whereby subjects went from heated to cooled baths. Therefore threshold temperatures in this study were determined without fluctuations in skin temperature.

Kellogg et al. (1991) investigated the thresholds for vasomotor and sudomotor control. They demonstrated that exercise elevates the core temperature at which the cutaneous active vasodilator system is initiated. However, they further argued that the threshold for the activation of the sudomotor system is unchanged. In other words, they believe that there is a possible upward shift in the vasomotor set-point, while the sudomotor set-point remains unchanged. The authors suggest that their data support the conclusion that set-point is not affected by exercise, but that it may seem to increase because of the variation in the thresholds of the two effector responses.

Thoden et al. (1994), investigated the pattern of core temperature during recovery and its relation to exercising core temperature values. They demonstrated that while a sharp drop in core temperature is experienced following the termination of exercise, esophageal temperature remains significantly elevated from pre-exercise levels up to 65 minutes post-exercise. Moreover, they demonstrated that the plateau at which post-exercise esophageal temperature lies corresponds to the temperature at which skin vessel dilation occurs during exercise. The authors suggested that this represents a non-active range of thermoregulatory mechanisms. They further argued that the corresponding temperature at which skin vessel dilation occurs and esophageal temperature plateaus represents the upper limit for the normal range of resting temperatures.

This finding by Thoden et. al, (1994) suggests that heat dissipating responses may be inactive although heat production has not been matched by heat loss. This finding challenges the

load error theory and the idea that a single "set-point" for temperature established in the hypothalamus exists. On the other hand, although it was not suggested by the authors, these results could be interpreted as supporting the idea that the thermoregulatory system is inactive within a certain range of core temperatures.

REFERENCES

Adolph, E.F. (1947). Physiology of Man in the Desert. New York: Interscuence Publishers.

Barger, A.C., Greenwood, W.F., DiPalma, J.R., Stokes III, J., and Smith, L.H. (1949). Venous pressure and cutaneous active hyperemia in exhausting exercise and certain other circulatory stresses. Journal of Applied Physiology. 2: 81-96.

Barr, S.I., and Costill, D.L. (1989). Water: can the endurance athlete get too much of a good thing? Journal of the American Dietitian Association. 89: 1629-1632.

Bevegard, B.S., and Shepherd, J.T. (1966). Circulatory effects of stimulating the carotid arterial stretch receptors in man at rest and during exercise. Journal of Clinical Investigations. 45: 132-142.

Blair, D.A., Glover, W.E., and Roddie, I.C. (1960). Vasomotor responses in the human arm during leg exercise. Circulation and Respiration. 9: 264-274.

Brengelmann, G.L., Johnson, J.M., Hermansen, L., and Rowell, L.B. (1977). Altered control of skin blood flow during exercise at high internal temperatures. Journal of Applied Physiology. 43(5): 790-794.

Brengelmann, G.L., Freund, P.R., Rowell, L.B., Olerud, J.E., and Kraning, K.K. (1981). Absence of active vasodilation associated with congenital absence of sweat glands in humans. American Journal of Physiology. 240 (Heart and Circulation Physiology 9): H571-H575.

Brown, W.K., and Sargent, F. (1965). Hidromeeiosis. Archives of Environmental Health. 11: 442-452.

Brown, S.L., and Banister, E.W. (1985). Thermoregulation during prolonged actual and laboratory-simulated bicycling. European Journal of Applied Physiology. 54: 125-130.

Cabanac, M., and Massonnet, B. (1977). Thermoregulatory responses as a function of core temperature in humans. Journal of Physiology. 265:587-596.

Colt, E.W.D, Wang, J., and Pierson Jr., R.N. (1978). Effect on body water of running 10 miles. Journal of Applied Physiology: Respiration, Environmental, and Exercise Physiology. 45(6): 999-1001.

Davis, G.R., Santa Ana, C.A., Morawski, S.G., Fordtran, J.S. (1980). Development of a large solution associated with minimal water and electrolyte absorption or secretion. Gastroenterology. 78: 991-995.

Dengel, D.R., Weyand, P.G., Black, D.M., and Cureton, K.J. (1992). Effect of varying levels of hypohydration on responses during submaximal cycling. Medical Science in Sports and Exercise. 24(10): 1096-1101.

Deschamps, A., R.D. Levy, M.G. Cosio, E.B. Marliss, S. Magder (1989). Effect of saline infusion on body temperature and endurance during heavy exercise. Journal of Applied Physiology. 66(6): 2799-2804.

Dill, D.B. (1938). Physiological effects of hot climates and great heights. In: Life, Heat, and Altitude. Cambridge: Harvard University Press.

Dill, D.B., Hall, F.G., and Van Beaumont, W. (1966). Sweat chloride concentration: sweat rate, metabolic rate, skin temperature, and age. Journal of Applied Physiology. 21(1): 99-106.

Doris, P.A., and Baker, M.A. (1982). Intracranial osmoreceptors control evaporation in heat stressed cat. Brain Research. 239: 644-648.

Durkot, M.J., Martinez, O., McQuade, D., and Francesconi. (1986). Simultaneous determination of fluid shifts during thermal stress in a small-animal model. Journal of Applied Physiology. 61: 1031-1034.

Eichana, L.W., Bean., W.B., Ashe, W.F., and Nelson, N. (1945). Performance in relation to environmental temperature. Reactions of normal young men to hot, humid (simulated jungle) environment. Bull Johns Hopkins Hospital. 76: 25-58.

Ekblom, B., Greenleaf, C.J., Greenleaf, J.E., and Hermansen, L. (1970). Temperature regulation during exercise dehydration in man. Scandinavian Archives of Physiology. 79: 475-483.

Feldberg, W., Myers, R.O., and Veale, W.L. (1970). Perfusion from cerebral ventricle to cisterna magna in the unanaesthetized cat. Journal of Physiology (London). 207: 405-416.

Fogelhom, G.M., Koskinen, R., Laakso, J., Rankinen, T., and Ruokonen, I. (1993). Gradual and rapid weight loss: effects on nutrition and performance in male athletes. Medical Science in Sports and Exercise. 25(3): 371-377.

Fortney, S.M., Nadel, E.R., Wenger, C.B., and Bove, J.R. (1981a). Effect of acute alterations of blood volume on circulatory performance in humans. Journal of Applied Physiology: Respiration, Environmental, and Exercise Physiology. 50(2): 292-298

Fortney, S.M., Nadel, E.R., Wenger, C.B., and Bove, J.R. (1981b). Effect of blood volume on sweat rate and body fluids in exercising humans. Journal of Applied Physiology: Respiration, Environmental, and Exercise Physiology. 51:1594-1600.

Fortney, S.M., and Vroman, N.B. (1985). Exercise, performance, and temperature control: Temperature regulation during exercise and implications for sports performance and training. Sports Medicine. 2: 8-20.

Fortney, S.M., Vroman, N.B., Beckett, W.S., Permutt, S., and LaFrance, N.D. (1988). Effect of exercise hemoconcentration and hyperosmolality on exercise responses. Journal of Applied Physiology. 65: 519-524.

Gisolfi, C.V., and Copping, J.R. (1974). Thermal effects of prolonged treadmill exercise in the heat. Medicine and Science in Sports. 6(2): 108-113.

Gonzalez-Alonso, J., Heaps, C.L., and Coyle, E.F. (1992). Rehydration after exercise with common beverages and water. International Journal of Sports Medicine. 13: 399-406.

Greenhaff, P.L., and Clough, P.J. (1989). Predictors of sweat loss in man during prolonged exercise. European Journal of Applied Physiology. 58: 348-352.

Greenleaf, J.E., Averkin, E.G., and Sargent II, F. (1966). Water consumption by man in a warm environment: a statistical analysis. Journal of Applied Physiology. 21(1): 93-98.

Greenleaf, J.E., and Castle. (1971). Exercise temperature regulation in man during hypohydration and hyperhydration. Journal of Applied Physiology. 30: 847-853.

Guyton, A.C. Metabolism and Temperature Regulation; The body fluids and the Kidneys. In: Textbook of Medical Physiology. W.B. Saunders Company. Philadelphia, 1981. 358-402, 838-847.

Guyton, A.C. The body fluids and the Kidneys. In: Human Physiology and Mechanisms of Disease. W.B. Saunders Company. Philadelphia, 1982. 525-569.

Hamilton, M.C., Gonzalez-Alonso, J., Montain, S., and Coyle, E.F. (1991). Fluid replacement and glucose infusion during exercise prevent cardiovascular drift. Journal of Applied Physiology. 71: 871-877.

Harrison, M.H., Edwards, R.J., and Fennessy, P.A. (1978). Intravascular volume and tonicity as factors in the regulation of body temperature. Journal of Applied Physiology. 44: 69-75.

Harrison, M.H. (1985). Effects of thermal stress and exercise on blood volume in humans. Physiology Reviews. 65(1): 149-207.

Holtzhausen, L.M., and Noakes, T.D. (1992). Postural hypotension after ultra marathon running is not related to levels of dehydration (Abstract). Medicine and Science in Sports and Exercise. 24: S44.

Horowitz, M. (1990). Thermoregulation under conditions of impaired body fluid/ osmotic balance in mammals. Journal of Basic Clinical Physiology and Pharmacology. 1:267-276.

Jessen, C., and Clough, D.P. (1973). Evaluation of hypothalamic thermosensitivity by feedback signals. Pflugers Archives. 345:43-59.

Jessen, C., and Mayer, E.T. (1971). Spinal cord and hypothalamus as core sensors of temperature in the conscious dog. Pflugers Archives. 324:189-204.

Johnson, J. M., and Rowell, L.B. (1975). Forearm skin and muscle vascular responses to prolonged leg exercise in man. Journal of Applied Physiology. 39: 916-924.

Johnson, J. M., Brengelmann, G.L., and Rowell, L.B. (1976). Interactions between local and reflex influences on human forearm skin blood flow. Journal of Applied Physiology. 41(6): 826-831.

Johnson, J.M., and Park, M.K. (1981). Effect of upright exercise on threshold for cutaneous vasodilation and sweating. Journal of Applied Physiology. 50: 814-818.

Johnson, M., and Park, M.K. (1982). Effect of heat stress on cutaneous vascular responses to the initiation to exercise. Journal of Applied Physiology. 53(3): 744-749.

Johnson, J. M. (1986). Nonthermoregulatory control of human skin blood flow. Journal of Applied Physiology. 61(5): 1613-1622.

Johnson, J.M. (1992). Exercise and the cutaneous circulation. Exercise and Sports Sciences reviews. 20: 59-97.

Kaciuba-Uscilko, H., Kruk, B., Szczypaczewska, M., Opaszowski, B., Stupnicka, E., Bicz, B., and Nazar, K. (1992). Metabolic, body temperature, and hormonal responses to repeated periods of prolonged cycle ergometer exercise in men. European Journal of Applied Physiology. 64(1): 26-31.

Kellog, D.J. Jr., Johnson, J.M, and Kosiba, W.A. (1990). Baroreflex control of the cutaneous active vasodilator system in humans. Circulation and Respiration. 66: 1420-1426.

Kellog, D.J. Jr., Johnson, J.M, and Kosiba, W.A. (1991). Control of internal temperature threshold for active cutaneous dilation by dynamic exercise. Journal of Applied Physiology. 71(6): 2476-2482.

Kenney, W.L., Tankersley, C.G., Newswanger, D.L., and Puhl, S.M. (1991). Alpha 1-adrenergic blockade does not alter control of skin blood flow during exercise. American Journal of Physiology 260 (Heart Circulation Physiology 29): H855-H861.

Kraning II, K.K., and Gonzalez, R.R. (1991). Physiological consequences of intermittent exercise during compensable and uncompensable heat stress. Journal of Applied Physiology. 71(6): 2138-2145.

Kraning II, K.K., Belding, H.S., and Hertig, B.A. (1966). Use of sweating rate to predict other physiological responses to heat. Journal of Applied Physiology. 21(1): 111-117.

Ladell, W.S.S. The effects of water and salt intake upon the performance of men working in hot and humid environments. Journal of Physiology. 127: 11-46.

Mekjavic, I.B., Sundberg, C.J., Linnarsson, D. (1991). Core temperature "null zone". Journal of Applied Physiology. 71:1289-1295.

Mekjavic, I.B., and Bligh, J. (1989). Core threshold temperatures for sweating. Canadian Journal of Physiology and Pharmacolagy. 67:1038-1044.

Montain, S.J., E.F. Coyle (1992a). Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. Journal of Applied Physiology. 73(4): 1340-1350.

Montain, S.J., E.F. Coyle (1992b). Fluid ingestion during exercise increases skin blood flow independent of increases in blood volume. Journal of Applied Physiology. 73(3): 903-910.

Morehouse, L.E., and Miller, A.T. Metabolic and environmental aspects of exercise: In Physiology of Exercise. The C.V. Mosby Company. Saint Louis, 1971. 214-221.

Moroff, S.V., and Bass, D.E. (1965). Effects of overhydration on man's physiological responses to work in the heat. Journal of Applied Physiology. 20: 267-270.

Murray, R.H. (1966). Cardiopulmonary effects of brief, intense thermal exposures. Journal of Applied Physiology. 21: 1717-1724.

Murray, R. (1992). Nutrition for the marathon and other endurance sports: environmental stress and dehydration. Medicine and Science in Sports and Exercise. 24(9): S319-S323.

Nadel, E.R., Cafarelli, E., Roberts, M.F., and Wenger, C.B. (1979). Circulatory regulations during exercise in different ambient temperatures. Journal of Applied Physiology. 46(3): 430-437.

Nadel, E.R., Fortney, S.M., and Wenger, C.B. (1980). Effect of hydration state on circulatory and thermal regulations. Journal of Applied Physiology. 49(4): 715-721.

Nadel, E.R. (1988). New ideas for rehydration during and after exercise in hot weather. Sports Science Exchange. 1(3).

Nakayama, T., Ohnuki, Y., and Niwa, K. (1977). Fall in skin temperature during exercise. Japanese Journal of Physiology. 27: 423-437.

Nielsen, B. (1966). Regulation of body temperature and heat dissipation at different levels of energy and heat production in man. Scandinavian Archives of Physiology. 68: 215-227.

Nielsen, B. (1974). Effect of changes in plasma volume and osmolality on thermoregulation during exercise. Scandinavian Archives of Physiology. 91: 123-129.

Nielsen, B. (1984). The effect of dehydration on circulation and temperature regulation during exercise. Journal of Thermal Biology. 9(1): 107-112.

Nielsen, B., Rowell, L.B., and Bonde-Petersen, F. (1984). Cardiovascular responses to heat stress and blood volume displacements during exercise in man. European Journal of Applied Physiology. 52: 370-374.

Noakes, T.D. (1992). The hyponatremia of exercise. International Journal of Sports Nutrition. 2: 205-228.

Noakes, T.D. (1993). Fluid replacement during exercise. Exercise and Sports Science Reviews. 21: 297-330.

Nose, H., G.W. Mack, X. Shi, K. Morimoto, E.R. Nadel. (1990). Effect of saline infusion during exercise on thermal and circulatory regulations. Journal of Applied Physiology. 69(2): 609-616.

O'Leary, D.S. (1991). Regional vascular resistance vs. conductance: which index for baroreflex responses? American Journal of Physiology. 260 (Heart Circulation Physiology. 29): H632-H637.

Pandolf, K.B. Effects of physical training and cardiorespiratory physical fitness on exercise-heat tolerance: recent observations. (1979). Medicine and Science in Sports. 11: 60-65.

Proppe, D.W., Brengelmann, G.L., and Rowell, L.B. (1976). Control of baboon limb blood flow and heart rate - role of skin vs. core temperature. American Journal of Physiology. 231(5): 1457-1465.

Radigan, L.R., and Robinsom, S. (1949). Effects of environmental heat stress and exercise on renal blood flow and filtration rate. Journal of Applied Physiology. 2: 185-191.

Roberts, M.F. and Wenger, C.B. (1980). Control of skin blood flow during exercise by thermal reflexes and baroreflexes. Journal of Applied Physiology. 48(4): 717-723.

Rowell, L.B. (1974). Human cardiovascular adjustments to exercise and thermal stress. Physiology Reviews. 54: 75-159.

Rowell, L.B., and O'Leary, D.S. (1990). Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. Journal of Applied Physiology. 69(2): 407-418.

Rubenstein, E.H., and Sessler, D.I. (1990). Skin surface temperature gradients correlate with fingertip blood flow in humans. Anaesthesiology. 73: 541-545.

Saltin, B. (1964). Circulatory response to submaximal and maximal exercise after thermal dehydration. Journal of Applied Physiology. 19: 1125-1132.

Saltin, B., Gagge, A.P., Bergh, U., and Stolwijk, J.A.J. (1972). Body temperatures and sweating during exhaustive exercise. Journal of Applied Physiology. 32(5): 635-643.

Santee, W.R., and Gonzalez, R.R. Characteristics of the thermal environment. In: Human Performance Physiology and Environmental Medicine at Terrestrial Extremes. (ed: Kent B. Pandolf, Michael N. Sawka, and Richard R. Gonzalez. Benchmark Press, Inc. Indianapolis, 1988. 1-43.

Sato, K. (1977). The physiology, pharmacology and biochemistry of the eccrine sweat gland. Reviews of Physiology Biochemistry and Pharmacology. 79: 51-131.

Savard,G.K., Nielsen, B., Laszczynska., Larsen, B.E., and Saltin, B. (1988). Muscle blood flow is not reduced in humans during moderate exercise heat stress. Journal of Applied Physiology. 64(2):649-657.

Sawka, M.N., Knowlton, R.G., and Critz, J.B. (1979). Thermal and circulatory responses to repeated bouts of prolonged running. Medicine and Science in Sports. 11: 177-180.

Sawka, M.N., Francesconi, R.P, Young, A.J., and Pandolf, K.B. (1984). Influence of hydration level and body fluids on exercise performance in the heat. Journal of AMA. 252: 1165-1169.

Sawka, M.N., Young, A.J., Francesconi, R.P, Muza, S.R., and Pandolf, K.B. (1985). Thermoregulatory and blood responses during exercise at graded hypohydration levels. Journal of Applied Physiology. 59: 1394-1401.

Sawka, M.N., and Wenger, C.B. Physiological responses to acute exercise-heat stress. In: Human Performance Physiology and Environmental Medicine at Terrestrial Extremes (ed: K.B. Pandolf, M.N. Sawka, R.R. Gonzalez), Benchmark press, Inc. Indianapolis, 1988.

Sawka, M.N. Body fluid responses and hypohydration during exercise-heat stress. In: Human Performance Physiology and Environmental Medicine at Terrestrial Extremes (ed: K.B. Pandolf, M.N. Sawka, R.R. Gonzalez), Benchmark press, Inc. Indianapolis, 1988.

Sawka, M.N., and Young, A.J. (1989). Acute polycythemia and human performance during exercise and exposure to extreme environments. Exercise and Sports Science Reviews. 17: 265-293.

Senay, L.C. (1979). Temperature regulation and hypohydration: A singular view. Journal of Applied Physiology: Respiratory, Environmental, and Exercise Physiology. 47: 1-7.

Shaffrath, J.D., and Adams, W.C. (1984). Effects of airflow and work load on cardiovascular drift and skin blood flow. Journal of Applied Physiology: Respiration, Environmental, and Exercise Physiology. 56(5): 1411-1417.

Smolander, J., Ilmarinen, R., Korhonen, O., Pyykko, I. (1987). Circulatory and thermal responses of men with different training status to prolonged physical work in dry and humid heat. Scandinavian Journal of Work Environmental Health. 13: 37-46.

Strydom, N.B., and Holdsworth, L.D. (1968). The effects of different levels of water deficit on physiological responses during heat stress. Int. Z. angew Physiology. 26: 95-102.

Taylor, W.F., Johnson, J.M., Kosiba, W.A., and Kwan, C.M. (1988). Graded cutaneous vascular responses to dynamic leg exercise. Journal of Applied Physiology. 64:1803-1809.

Thoden, J., Kenny, G., Reardon, F., Jette, M., and Livingston, S. (1994). Disturbance of thermal homeostasis during post-exercise hyperthermia. European Journal of Applied Physiology. 68(2):170-176.

Thornton, R.M., and Proppe, D.W. (1988). Influence of vasoconstrictor systems on leg vasodilation during heating of dehydrated baboons. American Journal of Applied Physiology. 254(Heart Circulation Physiology 23): H11-H19.

Thrasher, T.N., Nistal-Herrera, J.F., Keil, L.C., and Ramsay, D.J. (1981). Satiety and inhibition of vasopressin secretion after drinking in dehydrated dogs. American Journal of Physiology. 240: E394-E401.

Wenger, C.B. (1986). Non-thermal factors are important in the control of skin blood flow during exercise only under high physical strain. Yale Journal of Biological Medicine. 59: 307-319.

APPENDIX C

C.1 Physical Activity Readiness Questionnaire (Par-Q)

C.2 Informed Consent Form

C.3 Letter of Information

C.1
Physical Activity Readiness Questionnaire (PAR-Q)*

NAME OF PARTICIPANT _____

DATE _____

PAR Q & YOU

PAR-Q is designed to help you help yourself. Many health benefits are associated with regular exercise, and the completion of PAR-Q is a sensible first step to take if you are planning to increase the amount of physical activity in your life.

For most people physical activity should not pose any problem or hazard. PAR-Q has been designed to identify the small number of adults for whom physical activity might be inappropriate or those who should have medical advice concerning the type of activity most suitable for them.

Common sense is your best guide in answering these few questions. Please read them carefully and check (√) the ☐ YES or ☐ NO opposite the question if it applies to you.

YES	NO	
☐	☐	1. Has your doctor ever said you have heart trouble?
☐	☐	2. Do you frequently have pains in your heart and chest?
☐	☐	3. Do you often feel faint or have spells of severe dizziness?
☐	☐	4. Has a doctor ever said your blood pressure was too high?
☐	☐	5. Has your doctor ever told you that you have a bone or joint problem such as arthritis that has been aggravated by exercise, or might be made worse with exercise?
☐	☐	6. Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
☐	☐	7. Are you over age 65 and not accustomed to vigorous exercise?

If You Answered

YES to one or more questions

If you have not recently done so, consult with your personal physician by telephone or in person BEFORE increasing your physical activity and/or taking a fitness appraisal. Tell your physician what questions you answered YES to on PAR-Q or present your PAR-Q copy.

programs

After medical evaluation, seek advice from your physician as to your suitability for:

- unrestricted physical activity starting off easily and progressing gradually;
- restricted or supervised activity to meet your specific needs, at least on an initial basis. Check in your community for special programs or services.

NO to all questions

If you answered PAR-Q accurately, you have reasonable assurance of your present suitability for:

- A GRADUATED EXERCISE PROGRAM – a gradual increase in proper exercise promotes good fitness development while minimizing or eliminating discomfort;
- A FITNESS APPRAISAL – the Canadian Standardized Test of Fitness (CSTF).

postpone

If you have a temporary minor illness, such as a common cold.

* Developed by the British Columbia Ministry of Health. Conceptualized and critiqued by the Multidisciplinary Advisory Board on Exercise (MABE). Translation, reproduction and use in its entirety is encouraged. Modifications by written permission only. Not to be used for commercial advertising in order to solicit business from the public.
Reference: PAR-Q Validation Report, British Columbia Ministry of Health, 1978.
Produced by the British Columbia Ministry of Health and the Department of National Health & Welfare.

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

C.2 CONSENT FORM FOR THE INVESTIGATION OF WATER INTAKE ON POST-EXERCISE HYPERTHERMIA

INVESTIGATOR: Jeffrey Beraznik
Faculty of Health Sciences
School of Human Kinetics
125 University
Ottawa, Ontario K1N 6N5
Tel: 237-4256 (H) 564-9136 (lab)

ADVISOR: Dr. Jim Thoden
Faculty of Health Science
School of Human Kinetics
125 University
Ottawa, Ontario K1N 6N5
Tel: 564-5912

CHAIR OF FHS-HREC: Dr. Marie-des-Anges Loyer
Faculty of Health Sciences
Human Research Ethics Committee
Room 2009, 451 Smyth Road
Ottawa, Ontario K1H 8M5
Tel: 787-6550

Date:_____ Name of Volunteer:_____

The purpose of this study is to determine the effect of varying water volume on body temperature.

I have read and understand the information provided in the letter of information. I understand that I will be asked to participate in both a preliminary and two experimental sessions requiring treadmill running for up to twenty minutes in a thermal chamber set at 24-29^{0}c. Furthermore, I understand that the procedure requires blood sampling. My participation in this study will contribute to the knowledge of thermal control mechanisms and its relationship to fluid volume intake. I understand that the risks associated with this protocol consists of infection from blood sampling, irritation from inserting an esophageal and rectal probe, and temporary general discomfort.

I understand that the confidentiality of my data will be maintained at all times. Only the principal investigator and supervisor will have access to the data. My name will not appear on any sheet, but will be identified by a coded number.

ÉCOLE DES SCIENCES DE L'ACTIVITÉ PHYSIQUE
125 UNIVERSITÉ CP 450 SUCC A
OTTAWA ON K1N 6N5 CANADA

SCHOOL OF HUMAN KINETICS
125 UNIVERSITY PO BOX 450 STN A
OTTAWA ON K1N 6N5 CANADA

(613) 564-5920 • TÉLÉC./FAX: (613) 564-7689

I understand that I am free to withdraw this consent and to discontinue my participation at any time without penalty or discrimination of any sort.

Signature of volunteer: _____

Date:_____ _____Signature of witness: _____

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

C.3 LETTER OF INFORMATION

INVESTIGATOR: Jeffrey Beraznik
Faculty of Health Sciences
School of Human kinetics
125 University
Ottawa, Ontario K1N 6N5
Tel: 237-4256 (H) 564-9136 (lab)

ADVISOR: Dr. Jim Thoden
Faculty of Health Science
School of Human Kinetics
125 University
Ottawa, Ontario K1N 6N5
Tel: 564-5912

CHAIR OF FHS-HREC: Dr. Marie-des-Anges Loyer
Faculty of Health Sciences
Human Research Ethics Committee
Room 2009, 451 Smyth Road
Ottawa, Ontario K1H 8M5
Tel: 787-6550

This project involves research and is submitted in partial fulfillment of the degree of masters of science in movement studies.

The purpose of the present study is to determine the effect of water intake on body temperature.

The study consists of one preliminary and two experimental sessions conducted on three separate days.

The preliminary session involves detailed briefing as to the nature of the study and your involvement as a subject. You will be made familiar with all of the equipment being used (treadmill, temperature probes, heart rate monitor, and blood analyzer) and you will then be asked to complete the Physical Activity Readiness Questionnaire (Par-Q). Furthermore, you will be asked to complete a predicted maximal oxygen consumption test which will be assessed during a jog on a treadmill which increases in angle every three minutes. The duration of the preliminary session is approximately one hour.

The experimental conditions consist of treadmill running in an ambient temperature of 24-29 degrees Celsius at an intensity equal to 75% of your maximal oxygen consumption (moderate intensity). Thus, the absolute workload will be different for each subject, but the intensity of exercise relative to an

ÉCOLE DES SCIENCES DE L'ACTIVITÉ PHYSIQUE
125 UNIVERSITÉ CP 450 SUCC A
OTTAWA ON K1N 6N5 CANADA

SCHOOL OF HUMAN KINETICS
125 UNIVERSITY PO BOX 450 STN A
OTTAWA ON K1N 6N5 CANADA

(613) 564-5920 • TÉLÉC./FAX: (613) 564-7689

individual's maximum ability will be the same for everyone. The running lasts for 20 minutes at which time you are connected to skin (chest, forearm, finger, thigh and back) and core (rectal & esophageal) temperature probes. Finger blood samples and body weight measurements are taken prior to and following the treadmill running. The only difference between the two experimental conditions is that one condition requires water consumption at set intervals during the running, while the other does not. Including an adaptation and recovery period, the experimental sessions each last approximately 2 hours and will require you to avoid exercise and other stresses 24 hours prior to experimentation. You are also asked to get a full 8 hours sleep the night before experimentation and eat breakfast, lunch and dinner at regular times. As part of the experimental conditions, you are required to consume 500 ml. of fluid between two hours prior to exercise and one hour prior to exercise. The exercise is conducted in shorts and running shoes.

The present study has certain risks associated with it that you should be aware of. The risk of exercising to maximum capacity includes the very minor possibility of experiencing a stroke or heart attack or even the possibility of death. However, subjects in this study will not exceed an exercise intensity equal to 75% of their maximum capacity and the risks associated with this intensity are essentially zero in young, healthy adults. The risk of infection is present during blood sampling, however is minimized when done under proper hygienic conditions. Every effort is made to conduct the tests in such a way as to minimize risk.

The present study has certain discomforts associated to it that you should be aware of. There exists the possibility of minor nasal and throat discomfort both during the experimental protocol and for a few days following the experiments as a result of the esophageal probe. Moreover, you may experience minor rectal soreness as a result of the rectal probe. Furthermore, exercise always carries the risk of minor muscle soreness (e.g. charley horse) in the two days following exercise. Every effort is made to conduct the testing in such a way as to minimize these sources of discomfort.

This study is designed to enhance knowledge with respect to the science community's understanding of thermal control mechanisms and its relationship with fluid consumption.

Your anonymity is maintained in this study as no data is ever associated with your name, but rather by code. Moreover, no data is ever viewed in any form whatsoever outside the laboratory or

apart from the participating personnel. All data is kept in secure files and you are welcome to request and discuss the results at any time with myself or my advisor.

It is understood that you may withdraw/refuse participation in any session.

Further information related to ethics in human research can be requested from the chair of the Faculty of Health Sciences Human Research Ethics Committee, M. Loyer at the Faculty of Health Sciences, University of Ottawa, (613) 787-6550.

APPENDIX D

Certification of Institutional Human Research Ethics Committee

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

October 12, 1994

Jeffrey Berzanik
c/o Professor James Thoden
Faculty of Health Sciences
School of Human Kinetics
Montpetit Hall
University of Ottawa
INTRA

SUBJECT: Your project entitled "Investigation of water intake on post-exercise hyperthermia"

Dear Student

It is my pleasure to inform you that the Faculty of Health Sciences, Human Research Ethics Committee, after study of the documentation provided, concluded that your project met the appropriate standards of ethical acceptability and falls within Category 1A.

I hereby attach a copy of the certificate of clearance granted by the University Human Research Ethics Committee.

This certificate is valid for a period of one year from the time of issuance. I would also like to remind you that, in accordance with the policies of the UHREC, it is your responsibility to notify the Committee of any major changes in this project.

On behalf of the Committee, I wish you success in your project.

Sincerely,

Francis D. Reardon, Ph.D
Chair
Human Research Ethics Committee

CABINET DE LA DOYENNE/OFFICE OF THE DEAN
451 SMYTH, OTTAWA, ONTARIO, CANADA K1H 8M5
(613) 787-6705 FAX: 787-6725 TÉLÉCOPIEUR

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

FACULTÉ DES SCIENCES DE LA SANTÉ
FACULTY OF HEALTH SCIENCES

**CERTIFICATION OF INSTITUTIONAL HUMAN RESEARCH ETHICS COMMITTEE
FACULTY OF HEALTH SCIENCES**

This is to certify that the Institutional Human Research Ethics Review Committee of the Faculty of Health Sciences has examined the research proposal by **Jeffrey Beraznik,** from the School of Human Kinetics for the project entitled : **"Investigation of water intake on post-exercise hyperthermia "** and concludes that, in all respects, in the proposed research protocol meets the appropriate standards of ethical acceptability, at a Category 1A level.

MEMBERS OF THE COMMITTEE

Name (Optional)	**Position held**	**Department of discipline**
Victor Boucher	Professor	Programme of Audiologie/Speech Pathologie
Claire-Jehanne Dubouloz	Professor	Programme of Occupational Therapy
Fabienne Fortin	Professor	School of Nursing
Nadia Lebreux	Student	Human Kinetics
Roch Paquin	Member-at-Large	
Daniel Proulx	Professor	Faculty of Law
Francis Reardon	Chair	Human Research Ethics Committee School of Human Kinetics

SIGNATURE

10.10.94.

Date **Committee Chairperson - Francis D. Reardon, Ph.D**

CABINET DE LA DOYENNE/OFFICE OF THE DEAN
451 SMYTH, OTTAWA, ONTARIO, CANADA K1H 8M5
(613) 787-6705 FAX: 787-6725 TÉLÉCOPIEUR

APPENDIX E

E.1 Testing Procedure for the Predicted Maximal Oxygen Consumption Test

E.2 Individual Subjects' Exercising Intensity

E.3 Calculation for Converting Weight Loss Into Liters of Water

E.1 Testing Procedure for the Predicted Maximal Oxygen Consumption Test

1. The purpose and procedures of the test were fully explained to the subject and any questions which remained were answered.
2. Subjects, wearing heart rate monitors began running on the treadmill at a comfortable pace. They were then instructed to indicate at what speed they felt they could run at least fifteen minutes continuously.
3. After the appropriate speed was chosen, the subject exercised on the treadmill until his target heart rate was reached as determined through a pre-determined formula[**] .
4. In order to reach the subjects' target heart rates, the treadmill was inclined by two percent every two minutes.
5. When the subjects' met or exceeded their target heart rates, the treadmill speed and percentage grade were noted and used as the exercising intensity during the experimental sessions.
6. Subjects' were then encouraged to continue running at a comfortable speed with no incline in order to "warm down".

[**] Target Heart Rate (THR) = [(220-age)-(Resting Heart Rate) * .75 + Resting Heart Rate]

E.2 Individual Subjects' Exercising Intensity

Subject	Speed (Km/ h)	Percent Incline
BH	8.83	4
CM	8.83	1
DB	8.83	2
DJ	10	3
DM	8	3
FS	11.66	2
GK	10	5
JB	9	3
LA	11	0.9
LM	N/A	2

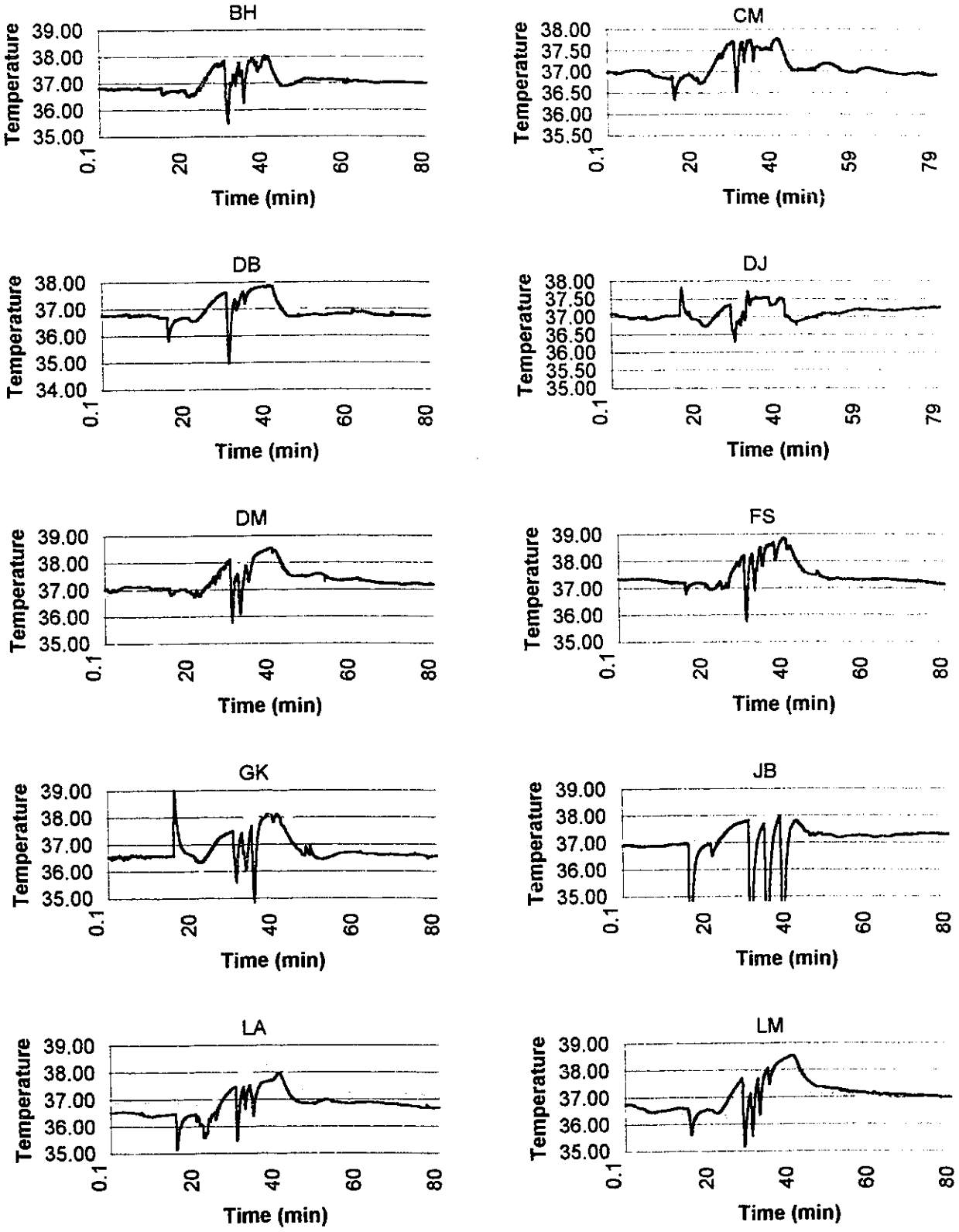
E.3 Calculation for Converting Weight Loss Into Liters of Water

In order to calculate the necessary amount of water ingestion needed to compensate for the body water volume lost during the hypohydrated condition, subjects were weighed prior to and following the hypohydrated testing session. Considering the formula that one liter of water equals 2.205 pounds (Morehouse and Miller, 1971), the weight loss was converted from pounds to liters, and ingested at four equal intervals in order to maintain hydration during the euhydrated condition.

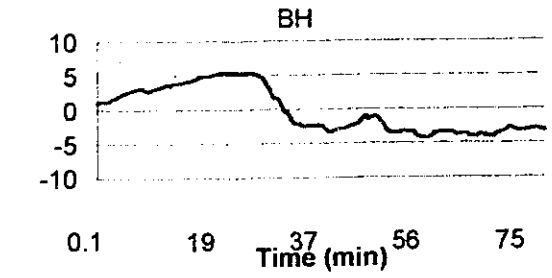
APPENDIX F

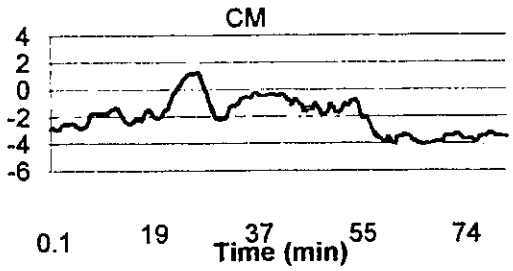
F.1 Individual Esophageal Profiles for Each of the Ten Subjects

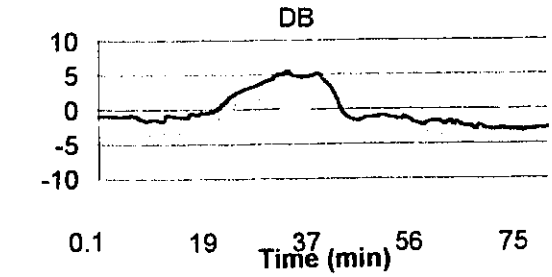
F.2 Individual Forearm - Finger Profiles (Vasodilation Threshold Identification)

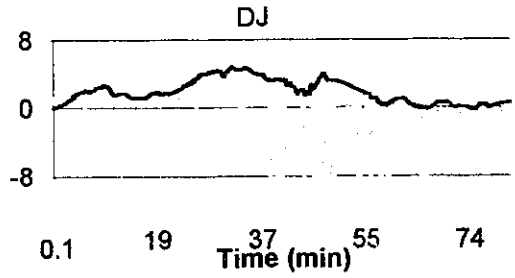


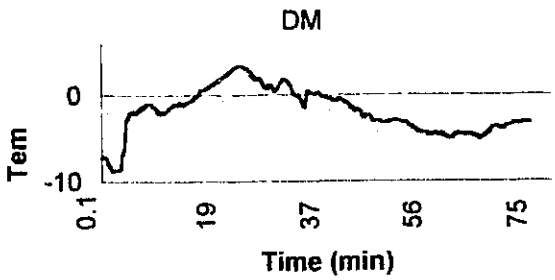
F.1 Individual Esophaheal Profiles for Each of the Ten Subjects

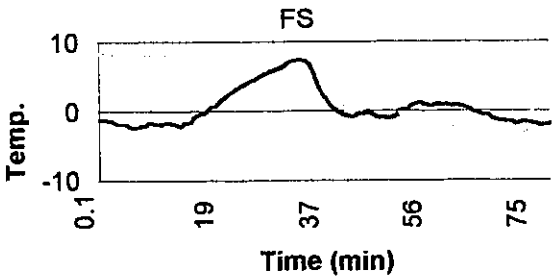


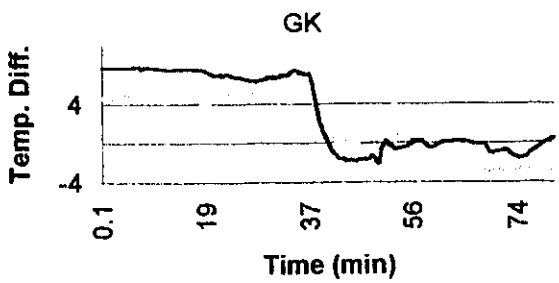


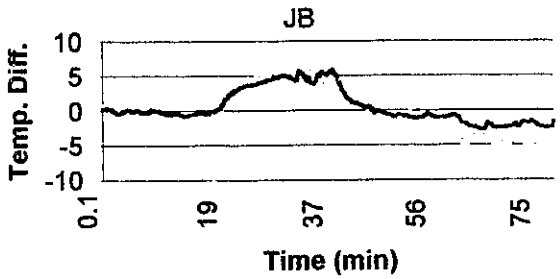


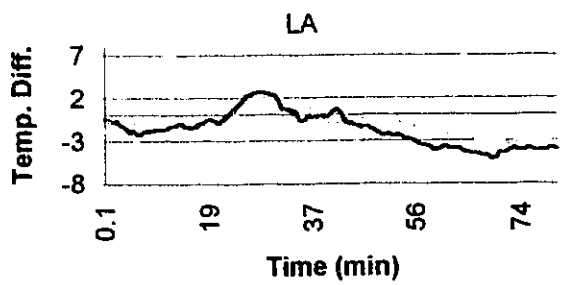


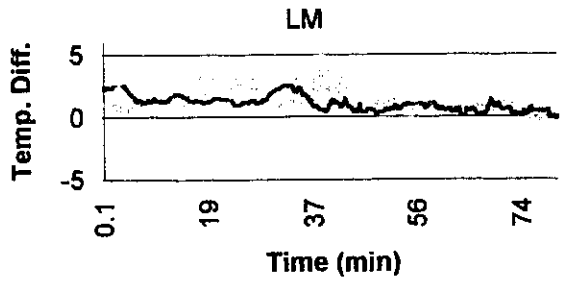












F.2 Individual Forearm - Finger Profiles