11-15-2006

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THE PHYSIOLOGICAL EFFECTS OF HOCKEY PROTECTIVE EQUIPMENT ON

HIGH INTENSITY INTERMITTENT EXERCISE

A Thesis Submitted to the
Yale University School of Medicine
in Partial Fulfillment of the Requirements for the
Degree of Doctor of Medicine

by

Benjamin Carter Noonan

2006

ABSTRACT

Ice hockey is a contact sport played in a cold environment which leads to assumptions that players are not exposed to a thermal challenge. The purpose of this study was to test the hypothesis that the wearing of hockey protective equipment during an exercise protocol designed to simulate a hockey game would induce a thermal challenge and lead to decrements in performance. In order to test this hypothesis and qualify the physiological responses, subjects performed a standardized protocol performed on a stationary cycle ergometer in an environmental chamber set at typical (12ºC) ice hockey ambient conditions. The simulation was performed twice; once while wearing cotton undergarments only (NP), and once while wearing cotton undergarments and the typical protective equipment worn during a hockey game (P). Work intensity during each trial was held constant and was evaluated by examining mean power output, which was similar under both P and NP conditions (348.2 W vs 352.08 W, $P > 0.05$) P vs NP, respectively. Body (37.18 ºC vs 36.58 ºC) and skin temperatures (34.12 ºC vs 28.85 ºC) were elevated in P vs NP, respectively ($P<0.05$). Core temperatures (37.50 ºC vs 37.41ºC) displayed a trend towards being higher in P vs NP particularly during the third period of simulation ($P = 0.053$). Sweat loss as a percent of body mass was greater in P vs NP (2.57% vs 1.18%, respectively $P<0.05$), which led to an increase in plasma osmolality (287 vs 283 mosmol/kg H_2O, respectively $P<0.05$) working heart rate (83.7% vs 78.8% of maximum heart rate), resting heart rate (63.4% and 55.9% of maximum heart rate), and urine specific gravity (1.026 vs 1.017) for P vs NP respectively (each $P<0.05$). The drop-off in power from pre to post simulated game was examined in both conditions by the use of five repeated maximal six second sprints interspersed with 24 seconds of recovery. The drop-off in both peak (12.0% vs 0.2%) and mean power (14.5% vs 2.7%) was greater in P versus NP ($P<0.05$). Plasma lactate concentration was higher following the simulated game in P vs NP (9.64 vs 5.96 mmol/L, $P<0.05$) as was plasma norepinephrine (2274.0 vs 1366.9 pg/ml, $P<0.05$). Rating of Perceived Exertion increased by 30-53% in the P condition ($P<0.05$) even though power outputs were equivalent. The elevated body temperature and increased water loss appeared to increase glycolytic flux, which when coupled with the consequences of thermal stress, reduced power output and led to the perception of elevated work intensities during the simulated game.

ACKNOWLEDGMENTS

INTRODUCTION

PLASMA VOLUME

BODY WATER DISTRIBUTION

The human body is made up largely of water which comprises about 45-70% of the body mass in an average adult (1). Total body water (TBW) is divided into two main compartments, 2/3 of the fluid is inside of the cells, the intracellular fluid (ICF), while 1/3 of the fluid is outside of the cells, the extracellular fluid (ECF) (2). The ECF is further divided into three compartments, the plasma volume (PV) which is intravascular and makes up approximately 20% of the ECF, the interstitial fluid (ISF) which is extravascular and makes up approximately 75% of the ECF, and the transcellular fluid (TCF) which is extravascular and makes up approximately 5% of the ECF. Within the ISF there are two smaller compartments which communicate slowly with the rest of the ISF. These are the dense connective tissue (cartilage and tendons) and the bone matrix. The intravascular compartment consists of the blood volume (BV), further subdivided into two components; the PV, and a portion of the ICF contained within the cellular components of the BV, the hematocrit (Hct) (2). These compartments and a derivation of their volumes are shown graphically in figures #1 and #2.

ICF = 2/3 of TBW

TBW = 45-70% of BM

ECF = 1/3 of TBW

ISF = 75% of ECF

PV = 20% of ECF ⟶ BV = PV / (1-Hct)

TCF = 5% of ECF
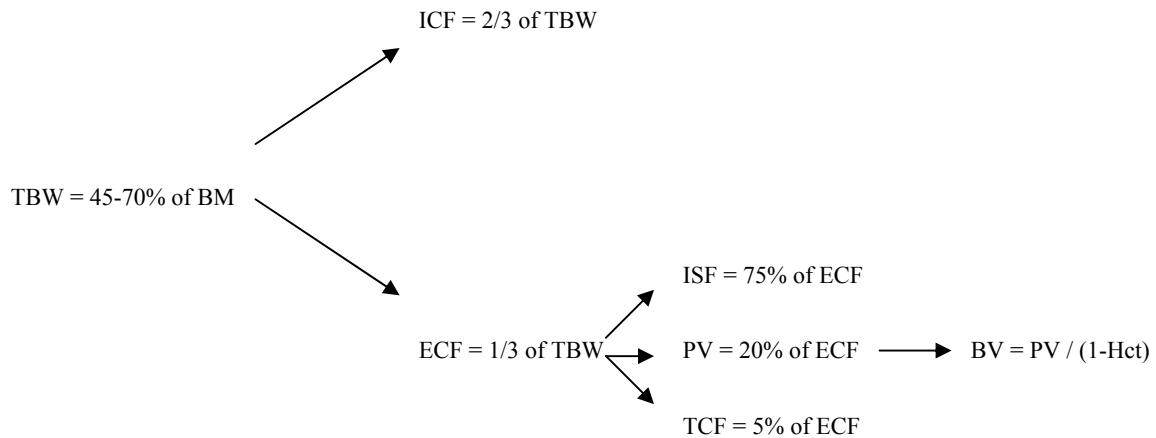
Figure 1: Derivation of compartment volumes

The distribution of water among the various compartments is controlled by two forces within each compartment: the difference in the concentration of water (with surrogate representation by osmolality), and the differences in hydrostatic pressures. Osmolality is the quantity of osmotically active solutes per kg of water within a compartment and is really just an easier way of expressing water concentration, as in dilute solutions the water gradient across a membrane is roughly proportional to the difference in osmolalities.

Capillary
Endothelium

Plasma
Membrane

Blood Plasma (3L)

$[Na^+]$ = 142 mM
$[K^+]$ = 4.4 mM
$[Cl^-]$ = 102 mM
[Protein] = 1 mM
Osmolality = 290 mOsm

Interstitial Fluid (13 L)
Bulk = 8 L
Bone = 2L
Dense Connective Tissue = 3L

$[Na^+]$ = 145 mM
$[K^+]$ = 4.5 mM
$[Cl^-]$ = 116 mM
[Protein] = 0 mM
Osmolality = 290 mOsm

Intracellular Fluid (25L)

$[Na^+]$ = 15 mM
$[K^+]$ = 120 mM
$[Cl^-]$ = 20 mM
[Protein] = 4 mM
Osmolality = 290 mOsm

Transcellular Fluid (1L)
i.e. synovial fluid, CSF
$[Na^+]$ = variable
$[K^+]$ = variable
$[Cl^-]$ = variable
[Protein] = variable
Osmolality = variable

Epithelial
Cells

Figure 2: Graphical representation of the fluid compartments (typical volumes for a 70

kg male) of the body (adapted from Boron and Boulpaep (2))

With changes in either of these driving forces, water is transferred between the

compartments across selectively permeable membranes. Imbalances in the relationship

between these two forces are essential for passive water transport across membranes.

These forces are summarized by the following equation:

$$J_v = L_p [RT(Osm_1 - Osm_2) + (P_2 - P_1)]$$

J_v = Fluid Movement
L_p = Hydraulic Conductance
P_x = Hydrostatic Pressure in compartment "x"
Osm_x = Osmolality in compartment "x"

When looking at water movement across capillaries, the osmolality difference between the intravascular and extravascular compartment is largely determined by proteins, and is referred to as oncotic pressure. The hydrostatic component plays an important role in regulating shifts between the intra- and extravascular compartments as the blood vessel walls are elastic and in concert with elevated cardiac output, are able to generate significant mean arterial pressures in the circulatory system. Cell membranes on the other hand, are easily deformed, which prevents the buildup of a significant hydrostatic gradient between the ICF and ECF, so water shifts between cells and surrounding fluids is largely dependent on osmotic differences. The predominant osmolites are different in each compartment. The ECF contains 75% of the freely exchangeable sodium in the body and its volume is dictated by changes in its sodium content. Sodium and its anion are responsible for 90% of the osmolality in the ECF. Sodium is freely permeable across capillaries and will equalize between the PV and the ISF. Proteins of the intravascular system, which are too large to cross the capillary membrane, are responsible for the osmotic pressure of the vascular system which is referred to as oncotic or colloid osmotic pressure. These forces, together with hydrostatic forces, are known as Starling Forces and control movement across the capillary wall.

Of particular interest for this project were changes in TBW and the distribution of this water, focused primarily on PV changes associated with exercise and dehydration. The following sections will discuss three stimuli (skin and core temperature changes, exercise, and dehydration) which all cause changes in the distribution of fluid compartments.

TECHNIQUES OF MEASURING BODY WATER COMPARTMENTS

Dilution methods in which some type of tracer are introduced into the body are considered the gold standard of estimating fluid compartment volumes (3). Tracers used to assess TBW include stable isotopes of hydrogen or oxygen which delivered in known amounts, allow for a dilution factor to be calculated and volumes to be estimated. The most common tracers utilized include deuterium (^2H), deuterium oxide (^2H_2O, D_2O), and oxygen-18 (^{18}O), which is a constituent of heavy water ($H_2^{18}O$) (3). Extracellular volume changes are commonly measured by using isotopes of sodium and/or chloride, and in recent years, bromide (3). Once the ECV is determined, ICV is typically calculated by subtracting the ECV from TBW. The measurement of the absolute PV is also done by the use of dilutions. Molecules such as labeled albumin (T-1824 or ^{125}I) and labeled red blood cells (^{51}Cr, carbon monoxide, or ^{99}Tn) are used to calculate the PV, but often give variable results (1). By using tagged albumin the investigator is assuming that all of the albumin tagged will remain within the vascular space. If any albumin exits the space, the measured concentration will be more dilute, yielding a greater PV than actually exists. Evans blue dye (T-1824) is a dye which when injected into the vascular system, will bind with albumin and allow for a dilution to be calculated. Problems with this method include the loss of dye from the vascular system prior to binding to albumin (which can be overcome by pre-binding the dye with albumin from a whole blood sample), and again, anything which causes albumin to be lost from the vascular compartment. In an attempt to remedy these shortcomings, some researchers have utilized the dilution of

tagged red blood cells to calculate PV (1). These limitations make the maintenance of a very stable environment mandatory when undertaking the study of PV.

Hemoglobin Hematocrit Method

A commonly used method of calculating relative changes in PV is the hemoglobin (Hb)/hematocrit (Hct) method (4). This method relies on the fact that as the PV is reduced, the red blood cells (RBC's) should represent a greater fraction of the blood volume represented by an increase in Hct. Changes in Hb are used to correct for any changes in the volume of the RBC's as this could lead to an erroneous interpretation. For example, assuming one starts with an initial blood sample of 100 mL with a Hb of 15.1 and a Hct of 43.7, and after some intervention, obtains a second blood sample with a Hb of 16.7 and a Hct of 45.3, the calculations in figure 3 would apply:

Methods of Estimating Hydration Level

There are many methods that are used (sometimes inappropriately) to estimate fluid compartment size and dehydration with exercise. Bioimpedance spectroscopy involves the use of current flow through the body at many different frequencies to estimate resistance and then predict body water compartment sizes. This method requires strict standardization of protocol as it is affected by postural changes, level of hydration, and ingestion of fluid with varying tonicities (3). When controlling for such factors, the TBW prediction error ranges from 3.5% to 6.9% for adults with a 70 kg body mass. This variance makes it unsuitable for detecting small changes in TBW during exercise (3).

STEP 1:
In original sample, how much Hb was actually present?
(100 ml) * (15.1 gHb/100 ml) = 15.1 g Hb

STEP 2:
With new Hb concentration (16.7 g/100 mL), what volume of blood would contain the same 15.1 g of Hb?
(15.1 gHb) / (16.7 gHb/100 mL) = 90.42 mL blood
(it is important to remember that when measuring Hb, the RBC's are lysed, so the volume of interest is BV)

STEP 3:
So what is the reduction in BV?
(100 mL – 90.42 mL) / 100 mL = 9.58% decrease in blood volume.

STEP 4:
Knowing the two blood volumes and two Hct's, calculate the change in actual RBC volume:
Initial: (100 mL) * (43.7%) = 43.7 mL   Final sample: (90.42 mL) * (45.3%) = 40.96 mL

Reduction in RBC Volume: (43.7 mL – 40.96 mL) / (43.7 mL) = 6.3%

STEP 5:
Knowing the two blood volumes and the two RBC volumes, calculate the plasma volumes using the simple equation:
Plasma Volume = Blood Volume – RBC Volume
Initial: PV = 100 mL - 43.7 mL = 56.3 mL   Final sample: PV = 90.42 mL - 40.96 mL = 49.46 mL

Change in PV: (56.3 mL – 49.46 mL) / 56.3 mL = 12.2 % reduction in PV

Figure 3: Derivation of the Hb/Hct method of calculating changes in plasma volume.

(adapted from Dill and Costill (4))

Plasma Osmolality (P_{osm}) is widely regarded as an accurate indicator of hydration status as variations in P_{osm} stimulate important fluid regulating mechanisms such as the release of arginine vasopressin (3). Normal values rarely deviate by more than 1-2% from the base level of 2878 mmol/kg in healthy, well hydrated, adults.

Urine indicators are often examined when assessing hydration status but demonstrate a high variability and are subject to the affects of recent water or drink ingestion. A

healthy person on a normal diet, will need to excrete 600 to 800 mosmol of solute/m^2 of body surface are per day (5). With dilution capabilities of the kidney ranging from 100-1200 mosmol/kg, these solutes can be excreted in as little as 666 mL or greater than 13 L of urine per day. The basal amount of urine that typically must be excreted ranges from 20-50 mL/hr in order to provide adequate waste product excretion, however in most healthy people, the actual amount often exceeds this value (5).

Urine osmolality U_{osm} is a general indicator of fluid status, but it can be affected by many variables, and no universally accepted standard exists for defining dehydration. In order to use U_{osm} as a hydration marker, it is recommended to establish a baseline for a subject and measure changes from their baseline as a large degree of variability exists in U_{osm} measures. In a study by Shirreffs et al (5), two groups of subjects collected their first urine samples of the day over consecutive days. The control group had no restrictions placed on activities, eating, or fluid ingestion. The second group undertook a dehydrating protocol on a cycle ergometer each night, followed by fluid intake which led to a BM reduction of 1.85% For euhydrated subjects, U_{osm} averaged 675 (+/- 232) mosmol/kg over the 5 days of collection. For dehydrated subjects, U_{osm} averaged 924 (+/-99) mosmol/kg over 7 days of collection. These values were significantly different, but both groups demonstrated a large range (429-1014 in the euhydrated and 643-940 in the dehydrated group) of measured mosmol/kg (5). Urine specific gravity is another urinary index which is commonly used and easily measured, which has been shown to demonstrate a high correlation with U_{osm} ($r^2 = 0.96$).

In addition, changes in body mass are a commonly used measure of hydration status. These calculations must be corrected for ingestion, excretion, sweat, respiratory, and substrate oxidation values to remain completely accurate. Some difficulties in using body mass include that a baseline is required which is often difficult to obtain in athletes and interday variations in body mass can be as much as 0.5 kg.

Popowski et al (6) examined the changes in P_{osm}, U_{osm}, and U_{sg} during progressive dehydration (1, 3, and 5% loss of body mass) induced by light exercise in the heat followed by rehydration (ingested water) of body mass water losses. Plasma osmolality (reference values of 280-290) was a very rapid and sensitive marker of dehydration, while U_{sg} (euhydration being less than 1.020) and U_{osm}, were accurate but delayed markers of dehydration (U_{osm} showed no significant change until 5% body mass loss, and U_{sg} showed no change until 3% body mass loss). The use of a U_{sg} of 1.020 for a criteria of euhydration led to a false positive (indicating euhydration when actually dehydrated by both delta body mass and P_{osm}) rate of 100%, 58%, and 33% at dehydration levels of 1%, 3%, and 5% respectively (6). Moreover, similar findings existed during rehydration values with P_{osm} reacting quickly, and urine values lagging.

Urine color is a sensitive method of monitoring hydration status and correlates well with urine specific gravity ($r^2 = 0.77$ to 0.96) (3). The use of onset of thirst can provide a general indicator of hydration status, with onset occurring at approximately 1-2% loss of TBW. However, thirst is affected by many factors including: fluid palatability, time allowed for consumption, gastric distension, age, gender, and heat acclimatization (3).

STIMULI THAT AFFECT BODY WATER DISTRIBUTION

Plasma Volume Shifts With Posture

Fluid shifts begin immediately upon positional changes secondary to changes in

hydrostatic pressures in the vascular space (1). Hagan et al (7) described the timeline for

changes in PV when moving from standing for 10 min, to supine for 35 min, and back to

standing again for 35 min. A "relative stability" had been achieved after the 20th min

following each postural change. However, while changes during this "relative stability"

were not statistically significant, there were still observable corrections occurring. In

follow up studies, they then examined two subjects for one hour intervals alternating

between supine-standing-supine and demonstrated that 40 to 60 min were required for

actual equilibrium (no observable corrections) to be attained (7). In the original

experiment, PV was increased by 11.7% when moving from standing to supine, and

reduced by 16% when returning to standing. The authors also found that there were no

positional changes in P_{osm} and therefore no driving force for changes in RBC volume

(MCHC remained the same throughout) (7). The authors interpreted the changes as being

due to blood redistribution to the dependent areas of the arms and legs as a result of

elevated hydrostatic pressures in the distal capillaries. This elevation is secondary to the

development of long columns of blood developed with standing. The minimal change in

P_{osm} is due to the fact that there has been no change in TBW or total body solutes with

these interventions. As the hydrostatic pressures increase in the capillaries, fluid is

forced out, which would have caused a transient increase in P_{osm}. However there would

have also been an increase in the concentrations of sodium and other freely diffusible PV

constituents in the vascular space. These constituents would move down their concentration gradients to equalize the P_{osm} across the capillary wall. The remaining differences in P_{osm} would be accounted for by the retained plasma proteins. This finding lends support the to the concept discussed in following sections that significant changes in P_{osm} are due to losses of water and electrolytes in sweat and or urine during exercise.

Plasma Volume Shifts With Acute Heat or Cold Stress

Acute heat/cold stress describes the physiological responses which take place when the human body is first exposed to hot/cold conditions - absent any significant changes in TBW. The changes in PV during acute hot/cold exposure may be due to changes in skin circulation (1). Changes in the body's skin temperature, independent of changes in the core temperature, can elicit changes in PV (8). The act of moving from a temperate (22ºC) environment to a hot (36.2ºC) or cool (14.4ºC) environment for 30 min led to PV expansion or contraction respectively (8). These changes were seen with skin temperatures that increased from 31.4ºC (temperate) to 35.8ºC (hot) or decreased to 28.1ºC (cold). Core temperatures as measured by a zero-gradient auditory canal thermistor were paradoxically elevated by 0.6ºC in the cold and decreased by 0.2ºC in the hot conditions. An explanation often given for these acute changes in PV is that with skin temperature increases, there is local **veno**dilation leading to a reduction in hydrostatic pressure in the capillary which promotes intra-vascular fluid resorption (1). With skin temperature decreases, there is local **veno**constriction leading to an increase in hydrostatic pressure which promotes intravascular fluid extravasation (1). In past studies, there has been contrasting information published on hemoconcentration with heat

exposure with some studies demonstrating hemoconcentration, and other studies demonstrating hemodilution (9). Cutaneous venules are much more sensitive to changes in skin temperature than are arterioles especially at normothermic core temperatures (9). Hence, with elevated skin temperatures local **veno**dilation will occur, and with decreased skin temperatures local **veno**constriction will occur. These vessel or capillary changes lead to a decreased or increased hydrostatic pressure respectively in the upstream capillary where fluid exchange takes place and thus promote fluid absorption or excretion. Vasomotor tone of the cutaneous arterioles (**vaso**dilation) is driven, at rest, more by changes in core temperature than skin temperature (9). **Vaso**dilation of the arterioles increases the hydrostatic pressure in the capillary because they are upstream of the capillary. Thus, during dilation the arterioles allow increased blood pressures to be transmitted to the capillary, elevating hydrostatic pressures and promoting fluid excretion.

Another factor which may contribute to acute change in PV with heat/cold stress would be the loss of plasma proteins. Studies are mixed as to whether or not there is actual changes in plasma proteins with heat/cold stress and exercise (1). It is hypothesized that with vascular dilation, the junctions of the endothelial cells become more permeable to the proteins (10). Changes in intravascular protein content would shift the osmotic pressures in the vascular bed leading to fluid shifts out of this compartment.

Plasma Volume Shifts With Exercise

Exercise by itself has been shown to result in an acute reduction in PV, although the extent of the PV loss is dependent upon exercise modality and intensity (9). The changes seen during exercise are attributed to changes in the balance between hydrostatic pressures and osmotic pressures in the vascular system. It is assumed that the hydrostatic contribution to the reduction in PV is due to both elevated mean arterial pressures (MAP), and arteriole vasodilation to the muscular beds. As mentioned before, this will allow a greater blood flow and higher pressures to be transmitted to the capillary, promoting fluid extravasation. With progressively increasing intensity of both upper and lower body ergometry, PV shifts are correlated with % VO_{2max} of the exercise (11). Moreover, elevations in MAP are correlated with exercise intensity, supporting the hypothesis that at least part of the PV shifts from exercise are due to elevations in hydrostatic pressures (11). Components of the osmotic contribution to PV shifts could include loss of plasma proteins or generation of osmotically active byproducts of metabolism in the exercising muscles which would lead to shifts of fluid from the ISF to ICF volumes (12). Understanding the variation in intravascular volumes with exercise is complicated by the fact that there are differences in how blood flow is distributed during exercise as opposed to during rest (9). During exercise, the cardiac output is divided between supplying blood to the working muscles and delivering blood to the skin for heat dissipation. Thus the skin is no longer strictly under thermoregulatory control, even when the exercise is occurring in a hot environment (9). Studies examining PV shifts with high intensity intermittent exercise are sparse. Whittlesey et al (13) examined the change in PV (Hb/Hct method) after the performance of two thirty-sec Wingate anaerobic

tests separated by 10 min of rest. The authors found that PV fell by 17.4% following the first Wingate test, recovered to a net reduction of 12.8% during the 10 min rest interval, and yielded a net 20.1% reduction following the second Wingate test. In addition, there was no relationship between power output of the second Wingate and reduced PV present at the beginning of the test (13). Gaitanos et al (14) also reported a reduction in PV of 12.1% following a series of 10 six sec maximal sprints followed by 30 sec of rest.

Plasma Volume Shifts With Dehydration

Dehydration is common either as exercise continues, or as subjects are exposed to chronic heat; and the body begins sweating in order to maintain its core temperature. In response to this water loss (which originates in the ISF), water is redistributed between compartments. Under resting conditions, the relative sizes of the ICF and ECF is determined by their electrolyte concentrations (1). During sweating, the body loses both water and electrolytes leading to shifts in the electrolyte concentrations in both compartments. The proportion of PV losses versus TBW losses is dependent upon dehydration procedure, with heat stress causing a greater insult to PV (1). For example, PV losses accounted for 22.7% of the TBW loss (18.3% reduction in PV) when exposed to sauna conditions for 2.5 hours (15). However, when similar TBW losses were incurred by light exercise in the heat or heavy exercise in a cool environment, PV losses were reduced to 11.4% and 2.8% of the overall TBW losses (1.5% and 10.6% reduction in PV) respectively (15).

The loss of PV lost during dehydration is thought to be due to the loss of ECF compartment electrolytes during sweating (1). These electrolytes would normally be responsible for an increase in ECF and plasma osmolality during dehydration. This increase in ECF osmolality would defend against ECF and PV losses by raising the osmolality, leading to a shift of fluid from the ICF compartment to the ECF. The reduction in sweat electrolyte loss is one of the mechanisms the body uses to protect the ECF and subsequently the PV spaces with heat acclimatization. Defense of the PV during exercise induced dehydration was demonstrated in a study by Jimenez et al (16). Plasma volume reduction during dehydration by 2.8% of body mass by either sauna or exercise was compared in eight subjects. It was demonstrated that PV was reduced by 11.4% via sauna induced dehydration, and by 4.2% during exercise induced dehydration ($p<0.001$). Possible mechanisms for defense of PV proposed included the release of water complexed to glycogen, the generation of metabolic water, or possible mobilization of proteins to the vascular space (16). Water bound to glycogen lies initially within the muscular compartments (ICF). During glycogen breakdown during exercise, this water is released and will dilute the ICF space lowering the osmolality leading to effusion of water from the muscular compartment into the ECF and vascular space.

In a study by Nose et al (17) subjects exercised at 40% of maximal aerobic power in the heat (36ºC, <30% Relative Humidity (Rh)) for 90-110 min to induce a body mass loss of 2.3% BM. They collected sweat and plasma samples and measured/calculated changes in compartment volumes and electrolytes. Immediately following exercise, PV was reduced by 4.3 ml/kg (9.4%) which recovered to 2.6 ml/kg (5.6%) one hour later.

This recovery was from fluid shifting from the ISF to the PV, likely the result of lower

capillary hydrostatic pressure following cessation of exercise (figure 4).
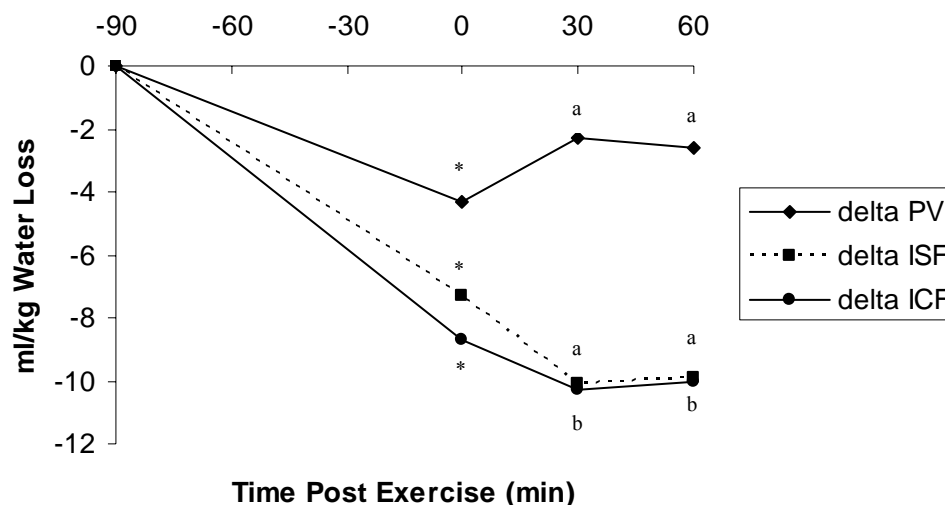
Figure 4: Changes in body water compartments with dehydration induced by exercise in

the heat (adapted from Nose et al (17))

(*) implies significantly different from pre exercise, (a) implies significantly different ($p<0.05$)
from 0 min time point, (b) implies not significantly different from 0 min time point.

Plasma osmolality also increased in proportion ($r = 0.79$) to free water lost from the body

in the forms of sweat and urine with both sweat and urine osmolalities less than that of

plasma. These changes in P_{osm} were also significantly correlated ($r = -0.738$) with water

shifts out of the ICF (17). Moreover, as the Na concentration in sweat increased, the

percentage of TBW lost from the ECF increased ($r = 0.804$), and ECF losses were well

correlated with PV losses ($r = 0.766$). In summary, as sweat Na concentrations increased,

less free water and more electrolytes were lost from the ECF, leading to greater

reductions in ECF volume, which was directly associated with a loss in PV. In contrast,

as more free water was lost, the increases in P_{osm} were accentuated, leading to a greater shift from the ICF into the ECF and ultimately, PV (17). Thus, a dilute sweat is of vital importance in maintaining PV and ECF volume during exercise in the heat.

Many exercise scenarios are combinations of both exercise and an environmental stress. Maw et al investigated the different effects of hot (36.2ºC, 44% Rh), temperate (22ºC, 52% Rh), and cool (14.4ºC, 74% Rh) environments on compartmental water losses during exercise (18). The results of this study are shown below graphically in figure 5.

Subjects exercised on a cycle ergometer at 50% of their maximal aerobic work rate for 50 min in a climate controlled chamber. Blood samples were taken every 10 min during exercise and immediately following exercise, to evaluate changes in each of the body's water compartments. After the first 10 min of cycling, the BV decreased in all environments with the decrease being greater in both the hot and cool versus temperate environment. These changes were largely due to changes in PV. Red blood cell volume (RCV) made only minor contributions to changes in BV in the first 10 min; in fact there were no significant changes in RCV until 20 min of exercise. These changes occurred without any significant changes in TBW.

Hot (36.2ºC / 44% Rh)	Temperate (22ºC / 52% Rh)	Cool (14.4ºC / 74% Rh)

$\downarrow$ $\downarrow$ $\downarrow$

50 min of cycling at 50% VO$_{2max}$
Blood samples obtained every 10 min, analyzed for blood volume (BV), red cell volume (RCV), plasma volume (PV)
Results shown as changes from baseline (BL)

$\downarrow$ $\downarrow$ $\downarrow$

	Hot	Temperate	Cool
10:	BV -470 mL[ab]	BV -114 mL[a]	BV -287 mL[ab]
	PV -356 mL[ab]	PV -110 mL	PV -243 mL[ab]
20:	RCV -114 mL[a]	RCV -76 mL[a]	RCV -75 mL[a]
30:	BV -500 mL[a]	BV return to BL	BV return to BL
		PV return to BL	PV return to BL
50:	BV -635 mL[abc]	BV 0	BV 0
	PV -532 mL[abc]	PV 0	PV 0
	TBW$_{loss}$ 943 mL [c]	TBW$_{loss}$ 673 mL	TBW$_{loss}$ 199 mL
	Sweat 861 mL[c]	Sweat 563 mL	Sweat 61 mL
	Urine 13 mL[c]	Urine 51 mL	Urine 83 mL

Figure 5: Plasma volume losses during exercise in hot, temperate, or cool

conditions.

a: $p<0.05$ from baseline, b: $p<0.05$ compared to temperate value, c: $p<0.05$ compared to
cool value.

After 30 min of exercise in the temperate and cool conditions, BV had recovered to pre-
exercise values, but did not do so in the hot condition where it progressively decreased
with time, eventually reaching a 635 mL deficit at the completion of the exercise.
Following the trend in BV recovery, PV losses had been recovered in the temperate and
cool conditions by the 30 min mark, but slowly continued to decrease in the hot
conditions. In all conditions, the bulk of the water lost was accounted for by reductions
in PV and/or ISF volumes, with PV making up the bulk of losses in the hot condition
(18). Initial PV reductions were attributed to elevated hydrostatic pressures seen with

exercise and elevated intramuscular osmotic forces (presumably from build up of metabolic byproducts) (18).

The PV reduction in the hot environment was contrary to the initial PV expansion experienced when resting in a hot environment secondary to cutaneous venodilation. Assuming all groups experienced equal vasodilation at the onset of exercise, the cool environment would experience the largest loss in PV initially followed by the temperate group, and finally the hot group, due to skin temperature driven venoconstriction/dilation respectively. The authors hypothesized that more powerful and extensive vasodilation, which is experienced during exercise in the heat, elevated hydrostatic pressures enough to offset the effects of temperature induced venodilation (18). Recovering PV's (even in the face of a 1% decrease in TBW) in the cool and temperate environments were attributed to greater ISF hydrostatic pressures and capillary osmotic pressures, both the result of hydrostatically induced shifts of water from the PV to the ISF. These changes would not have been evident in the hot condition due to ongoing water and electrolyte losses, preventing the buildup of ISF pressures and P_{osm} respectively (18).

Plasma Volume Expansion after Exercise

A well documented response to both acute and chronic exercise is the expansion of PV (19, 20) although the precise mechanism by which PV increases is not well understood. Possible mechanisms could include one or more of the following: modifications to the renin-angiotensin-aldosterone axis such as increased aldosterone release, or enhanced renal sensitivity to aldosterone which would promote sodium retention and ECF

expansion; an increase in plasma protein content which would drive PV expansion due to oncotic pressures; and finally, a decrease in hydrostatic pressure secondary to peripheral vasodilation could contribute to PV expansion (19). The time course of PV expansion following high intensity exercise can be very short. Gillen et al (19) demonstrated a recovery of PV following repeated bouts of cycle ergometry at 85% of VO_{2max} (eight, four min bouts with five min recovery periods in between each one). Plasma volume fell by 15% during exercise, but after one hour of seated recovery with no fluid replacement, PV had recovered to baseline, despite an overall body mass loss of 820g. Moreover, plasma albumin and total protein content increased and appeared to account for the entire PV restoration (19). Studies of the mechanisms for the post-exercise protein expansion have suggested a redistribution of ISF (lymphatic) albumin, a reduced transcapillary effusion of albumin, or increased albumin synthesis as possibilities. Due the rapid nature of the PV expansion, it was assumed that albumin synthesis was not a major contributor to the increase in the acute phase, more likely was the movement of ISF albumin into the vascular space. This movement of albumin would be enhanced by the muscular contractions providing extra driving force for lymphatic albumin to be returned to the vascular space (19). Similar studies have demonstrated an increase in albumin synthesis within 24 hours of exercise, and that this synthesis was inhibited by exercise in a supine position (21),(22).

THERMOREGULATION AND EXERCISE

METABOLIC ORIGIN OF HEAT

A majority of the heat which must be dissipated during exercise has its origins in the active skeletal muscle. The heat of skeletal muscle originates in primarily two sources: First, the inefficient conversion of chemical energy to mechanical energy during cross-bridge cycling (and driving of ion pumps), and second, during the regeneration of consumed adenosine triphosphate (ATP). The energetics of converting stored energy into mechanical energy can best be understood by a simple analysis using the first law of thermodynamics which states that energy cannot be created nor destroyed, only changed in form. For a contracting skeletal muscle viewed as a closed system (with no heat transfer into or out of the system) first law balance implies that when ATP hydrolysis occurs, driving cross-bridge cycling and subsequent shortening (assuming the contraction is concentric), the energy released by ATP hydrolysis must equal the mechanical work done plus the heat liberated. The work performed expressed as a percentage of the energy of ATP hydrolysis is referred to as the efficiency of muscular contraction. It can be seen that as muscular efficiency decreases, heat production increases. Barclay and Weber (23) recently published a study in which they examined what they termed "initial and net efficiency" of fibers from the mouse soleus (slow twitch) and extensor digitorum longus (EDL – fast twitch). They defined initial efficiency as the ratio of work output to the sum of work output and heat production over the first sec of muscular contractions which they felt would represent largely ATP and phosphocreatine (PCr) hydrolysis of

contraction. Net efficiency was defined as the same ratio but measured over an entire series of 10 contractions as well as the time interval of elevated metabolism following the series. Barclay and Weber felt that this "net efficiency" would incorporate the recovery process of regenerating spent ATP molecules as well. They described the relationship of net to initial efficiency as follows:

$$\text{Efficiency}_{net} = \text{Efficiency}_{initial} * \text{Efficiency}_{recovery}$$

They found that initial efficiencies were greater in the soleus versus the EDL (30% vs. 23%), but that these differences disappeared when looking at net efficiencies (12.6% vs. 11.7%). These results suggested that the oxidative recovery of slow twitch muscles is actually less efficient that those of fast twitch muscles (23). Citing prior research which had demonstrated that slow twitch muscle fibers consumed less ATP per unit of work compared to fast twitch muscle fibers, Barclay and Weber explained the finding of increased initial efficiency as being indicative of this reduced ATP consumption (23). The findings of reduced ATP consumption per unit of work in slow versus fast twitch muscles were corroborated by Reggiani et al (24), but these authors also commented that the efficiency of contraction differences were abolished as the contraction velocities increased.

Muscular contraction requires hydrolysis of ATP, which can be regenerated by three different metabolic pathways in the human body. In order to regenerate ATP, a coupled reaction must occur which liberates at least the same amount of free energy as

that which is consumed to resynthesize ATP (25). Any free energy release in the associated reaction which exceeds the energy required to resynthesize ATP is generally released as heat and the greater the heat release per mole of ATP resynthesized, the less efficient the process. The first reaction/pathway which is immediately active is the breakdown of PCr to free creatine (Cr) and inorganic phosphate (P_i). This pathway is capable of providing a relatively small total amount of energy as resting concentrations of PCr in skeletal muscle are small, but it is able to provide much of this energy in an "immediate" fashion. This reaction is as follows:

$$PCr + MgADP^- + H^+ \longleftrightarrow MgATP^{2-} + Cr$$
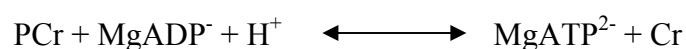
Woledge and Reilly (26) studied this reaction and found that thirty-five kJ of heat is generated per mole of ATP utilized.

The second pathway which can regenerate ATP in muscle is glycolysis. This pathway involves the breakdown of glucose and/or glycogen into two moles of pyruvate (which can continue on to form lactate or enter the Krebs cycle). This pathway can generate a large amount of energy very quickly but is limited in that a byproduct of its turnover is the generation of hydrogen ions which are implicated at least in part in the fatigue of skeletal muscle. The overall equation for glycolysis (carrying pyruvate to lactate) is as follows, and generates sixty-five kJ of heat is generated per mol of ATP utilized (27):

$$Glucose + 2\ P_i + 2\ ADP \longrightarrow 2\ lactate + 2\ ATP + 2\ H_2O$$
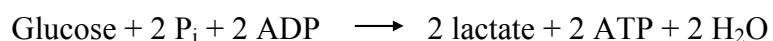
The final pathway of ATP generation is that of the Krebs cycle and oxidative phosphorylation:

Krebs Cycle:

$$\text{Acetyl CoA} + 3\ NAD^+ + FAD + GDP + P_i + 2\ H_2O \longrightarrow 2\ CO_2 + 3\ NADH + FADH_2$$
$$+ GTP + 2\ H^+ + CoA$$
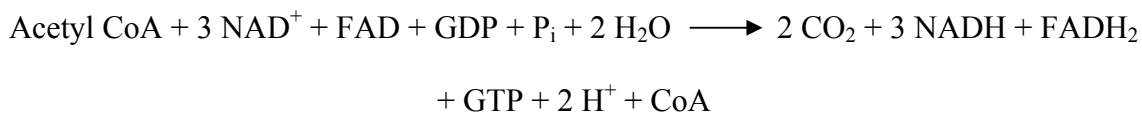
The reduced components are then passed on to the oxidative phosphorylation pathway where they are oxidized and drive the pumping of protons into the mitochondrial matrix. These protons establish an electrochemical gradient which is subsequently used to produce ATP.

Oxidative Phosphorylation:

$$3\ NADH + FADH_2 + 3\ H^+ + 2\ O_2 + 9\ ADP + 9\ P_i \longrightarrow 3\ NAD^+ + FAD^+ + 4\ H_2O +$$
$$9\ ATP$$
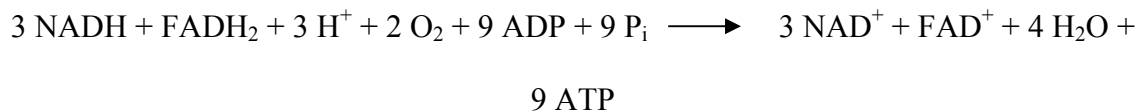
Seventy-two kJ of heat is generated per mole of ATP utilized, making them the least efficient (but capable of producing the most overall energy) of the three (27).

In support of these findings, Gonzalez-Alonso (28) et al evaluated the heat production in skeletal muscle at the onset of intense exercise. Their experiment consisted of 180 sec of intense dynamic knee extensor exercises (80 W) while measuring heat production in the quadriceps and estimating relative energy system contributions. The rate of heat production and ATP turnover increased significantly throughout exercise (with a concomitant reduction in efficiency), and was 107% higher at 180s compared with the initial 5s, during this constant power output exercise. The net contribution of oxidative

processes was estimated to be 32% during the first 30s of exercise, increasing to 86% during the last 30s, while the combined energy contribution from ATP, PCr, and lactate turnover declined from 37% to 3% over the same time intervals (28). Based on the above changes during exercise, mechanical efficiency was calculated to drop from an initial value of 53% to 36% at the end of exercise.

Krustrup et al (29) repeated the above protocol (65 W) but added two subsequent bouts of work (with 6 min rest periods) to evaluate the heat production during repeated intense dynamic exercise. The rate of heat production again increased within each bout (in accordance with increasing oxidative energy contributions), but was not increased between exercise bouts. Oxygen uptake by the active muscles was elevated during the first 120s of bout two and throughout bout three when compared to bout one, indicating a greater oxidative involvement as exercise continued. The anaerobic energy production during the first 105s of bout two and 150s of bout three was lower than bout one. These findings contradict earlier studies in that one would have expected to see elevated heat production with higher reliance upon oxidative ATP resynthesis in the latter bouts. This apparent contradiction may be explained by lower ATP utilization in the repeated bouts due to greater efficiencies even with a higher reliance upon oxidative means, but this requires future research to provide validation (29).

HEAT FLUX DURING EXERCISE

Heat Balance

Under sedentary conditions, heat production in the human body is approximately

80 kcal/hr (90 watts) (2). But during even moderate exercise, this value increases to 400-

600 kcal/hr. This type of heat production would cause an elevation of the body's core

temperature of one degree Celsius every eight to ten min if the extra heat could not be

dissipated into the environment. This heat loss occurs by four means: Conduction,

convection, radiation (collectively known as dry or sensible heat transfer), and

evaporative (insensible) heat transfer (1). The heat balance equation which describes the

thermal exchange between an individual and the environment is as follows (1):

$$\pm S = M \pm W \pm (R+C) - E$$

S = rate of storage (+ indicating energy stored in body)
M = metabolic energy production (always +)
W = work done by body (+)
R = radiant heat transfer (+ for transfer to environment)
C = convective heat transfer from skin and respiratory tract (+ for transfer to environment)
E = evaporative heat transfer from skin and respiratory tract (always -)
All units in W/m^2

Metabolic energy production can be calculated for longer steady state exercise when

aerobic components make up the majority of energy expenditure by the following

equation (1):

$$M \ (W/m^2) = (0.23[R] + 0.77) * (5.873)(VO_2) * (60/A_D)$$

M = metabolic energy production
R = respiratory exchange ratio
VO_2 = oxygen uptake (L/min) STPD
5.873 = caloric equivalent of oxygen in W/hr/L
A_D = DuBois Body Surface Area = $0.202(m)^{0.425}(H)^{0.725}$
m = mass (kg)
H = height (m)

However, this calculation is not accurate when dealing with intermittent high intensity exercise. Two commonly used methods to evaluate energy expenditure during high-intensity exercise include the calculation of an O_2 deficit and the use of a muscle biopsy (30). Oxygen deficit is simply the difference in predicted oxygen consumption at a given supra-maximal workload (calculated from a regression equation generated by multiple sub-maximal points) versus the actual oxygen consumed at this workload. The O_2 deficit method has limitations, and seems to be accurate in its estimation of anaerobic energy contribution when examining single muscle groups, using steady workload periods which are close to $VO_{2\ max}$. Moreover, some investigators have reported decreasing efficiencies of work with increasing power levels which would lead to underestimation of anaerobic contribution to exercise (30). Use of the muscle biopsy technique also has limitations. Questions include; how representative a single muscle biopsy is of whole muscle metabolism, how do delays in obtaining biopsy samples affect results, and accounting for released lactate into the blood (30). Briefly, muscle biopsies are obtained before and after intense exercise from an active muscle(s). These biopsies are analyzed for anaerobic metabolites which then allow for calculation of ATP turnover based on the following equation:

$$ATP = 1.5(\Delta\, La^-) + (\Delta PCr) + 2(\Delta ATP - \Delta ADP)$$

These changes in concentration are then multiplied by an estimate of active muscle mass

(often 25% in cycling). Even with these techniques, there is much work yet to be done in

the area of accurately assessing the energy expenditure during whole body high intensity

anaerobic work (30).

Heat that is not lost is stored in the body. The rate of heat storage can be calculated by

the following equation (1):

$$S = (0.965 * m/A_D) * \Delta T_b/\Delta t \quad (W/m^2)$$

$$S = \text{stored energy}$$
$$m = \text{mass (kg)}$$
$$\Delta T_b = \text{mean body temperature (C)}$$
$$\Delta t = \text{time (hours)}$$

Mean body temperature in a heat challenged subject can be calculated by using core

temperature and mean skin temperature in the following equation (1):

$$T_B = 0.9(T_c) + 0.1(\overline{T}_{sk})$$

$$T_B = \text{Body Temperature}$$
$$T_C = \text{Core Temperature}$$
$$\overline{T}_{sk} = \text{Mean Skin Temperature}$$

Mean skin temperatures are calculated using many different equations, the following is a

common equation used with skin temperatures from eight different sites, weighted for

skin surface area and thermal sensitivity (31):

$$T_M = .115T_1 + .170T_2 + .205T_3 + .090T_4 + .080T_5 + .053T_6 + .190T_7 + .097T_8$$

T_M = mean skin temperature
T_1 = Chest
T_2 = Low Back
T_3 = Forehead
T_4 = Abdomen
T_5 = Deltoid
T_6 = Forearm
T_7 = Thigh
T_8 = Calf

Sensible Heat Transfer

The exchange of sensible heat from the skin involves conduction from the skin to overlying clothing. Conduction is the heat transfer through bodies in contact or through still fluids. Convection is heat transfer through moving fluids, and radiation is heat transfer between two objects by electromagnetic waves. Without a temperature differential between the two surfaces of interest, there can be no sensible heat transfer.

Heat transfer by conduction occurs between skin and overlying clothing and can be represented by Fourier's law (looking at heat transfer between skin and the **exterior** surface of the clothing worn):

$$q_{cond} = (k)(A)(\delta T/\delta x)$$

q_{cond} = conductive heat transfer (W)
k = thermal conductivity (property of the clothing) (W/m$^{\circ}$C)
A = area of contact (m^2)
$\delta T/\delta x$ = temperature gradient through the clothing in direction of heat flow ($^{\circ}$C/m)

If one assumes a constant temperature distribution through the entire thickness of clothing, this equation can be expressed as follows:

$$q_{cond} = (k)(A/d)(T_{Cl} - T_{Sk})$$

d = thickness of clothing layer
T_{Cl} = temperature at the surface of the clothing
T_{Sk} = temperature of the skin

This equation examines the flow of heat from the skin through clothing of a given temperature and describes only one path of heat flow (there can be many parallel paths depending upon layers and composition of garments). Once the sensible heat is transferred from the skin to the garment, convection and radiation are responsible for transferring the heat to the environment.

The basic equation governing convection is Newton's law of convection which is:

$$q_{conv} = (h_c)(A)(\Delta T)$$

$h_{c/rad}$ = convective heat transfer coefficient ($W/m^2 {}^oC$)
A = effective surface area available for heat transfer
ΔT = temperature difference between skin and ambient air temperature

The heat transfer coefficient (h_c) for convection is modified based on surrounding fluid (water or air) velocities. As the surface temperature of the skin is typically warmer than ambient air, there is an air boundary layer that is established at a higher temperature than ambient which reduces heat transfer to the environment. When air velocity increases, cooler air is continuously circulated against the skin which attenuates this affect, greatly increasing the convective heat transfer from skin to the environment (32). The convective heat exchange coefficient increases approximately in proportion to the increase in the square root of the velocity (33) and increases in magnitude up to a wind velocity of 24 km/hr (32). The convective coefficient has been experimentally

determined for stationary cycle ergometry at 50 rpm as being equal to $5.5 + 1.96(V_{air})^{0.86}$

(1).

Convective heat losses also occur in the respiratory tract as follows:

$$C_{res} = 0.0014(M)(34 - T_a)(P_B/760)$$

C_{res} = heat loss via respiratory tract convection (W/m²)
M = metabolic rate (W/m²)
T_a = ambient dry bulb temperature (°C)
P_B = barometric pressure (mmHg)

Because heat transfer due to radiation is based on electromagnetic waves, it is

independent of ambient air temperature or the presence of air. Radiation directed at a

body can be absorbed, reflected, or transmitted through the body, with the total of the

three energy streams equaling the energy delivered. A commonly used term "black

body" implies a body which absorbs all radiant energy directed at it, and also will emit

the maximum possible energy when acting as a source. Objects are not true black bodies

and are described by emissivity (ε) which is a ratio of actual radiation to that of black

body radiation. The emissivity of human skin has been measured to be approximately

0.98 and that of clothing is 0.95 (1).

The energy radiated between two bodies at different temperatures can be expressed by

the following equation:

$$q_{rad} = (\sigma)(A)(F)(T_1^4 - T_2^4)$$

σ = Stefan-Boltzman constant (5.670 E^{-8} W/m²K⁴)
A = Area
F = configuration factor which is dependent upon shapes, emmissivities, and orientations
of the two surfaces
T = surface temperatures (K)

This equation is expressed in the following manner when used to express radiant heat transfer between a subject and the environment. Of note, the ambient temperature has been replaced by a derived temperature known as the "mean radiant temperature" (MRT) which is the temperature of a theoretical black box which, if surrounded the subject, would provide the same radiant heat transfer as the actual environment (1):

$$q_{rad} = (\varepsilon)(\sigma)(A_r/A_d)([T_r + 273.15]^4 - [T_{cl} + 273.15]^4)$$

q_r = radiant heat transfer (W/m^2) between environment and a surface
A_r = "open" radiating area of the body surface
A_d = Dubois surface body area
A_r/A_d = 0.65 sitting, 0.72 standing
T_{cl} = temperature of clothing surface
T_r = mean radiant temperature

This equation has been often simplified to the following, which replaces much of the above equation with a linear radiation exchange coefficient. This coefficient, by the nature of the variables it is replacing, is dependent upon geometry, configuration, and emissivity of the bodies:

$$q_r = h_r(T_{cl} - T_r)$$

h_r = linear radiation exchange coefficient = $(\varepsilon)(4\sigma)(A_r/A_D)(f_{acl}) * [(T_{cl} + T_r)/2 + 273.15]^3$

Exercise outdoors under full solar radiation can impart a heat load which would be the equivalent of increasing the ambient dry bulb temperature by 8-10°C, while exercise in a room whose structure and contacts are kept at the same temperature of the skin will eliminate heat transfer by radiation (33).

The heat transfer coefficients for convection and radiation are often combined and used in an equation which makes use of a derived temperature known as the "operative

temperature". This is the temperature of a black enclosure which, if surrounded the subject, would provide the exact same amount of convection and radiation (1). This combined equation is often expressed as DRY indicating sensible heat transfer (assuming no contact between surfaces, and at least minimal air movement, thus eliminating conduction):

$$DRY = h(T_o - T_{sk})$$

DRY = combined convection and radiation between environment and skin
h = weighted average of h_{conv} and h_{rad} = ([h_{conv}][T_a]+[h_r][T_r]) / (h_c + h_r)
T_o = operative temperature

Latent Heat Transfer

The fourth mechanism of heat exchange with the environment is by evaporative cooling of secreted sweat, lowering skin temperature which allows for conductive heat transfer between the warm blood and the cool skin. A major benefit of evaporative cooling is that it allows for cooling to occur even with ambient temperatures which are greater than the skin surface. A drawback to evaporative cooling is that the water which is excreted by sweat glands reduces the overall body water and electrolyte content which can provide a significant PV reduction and cardiovascular challenge to the dehydrated individual. The amount of heat lost through sweat evaporation can be approximated by measuring the change in weight during exercise (assuming all excreted sweat has been evaporated with no dripping losses). This weight loss must be corrected for water lost due to respiration and from the mass difference between oxygen uptake and carbon dioxide rejection (34):

$$m_e = 0.019(VO_2)(44 - P_a)$$

m_e = mass of water lost due to evaporation of respiratory tract water (g/min)
VO_2 = oxygen consumption in L/min STPD
P_a = ambient water vapor pressure (mm Hg)

$$m_r = VO_2 * [R(\rho_{CO2}) - (\rho_{O2})]$$

m_r = mass difference of oxygen and carbon dioxide (g/min)
R = respiratory quotient
VO_2 = oxygen consumption L/min STPD
ρ_{CO2} = density of carbon dioxide (1.96 g/L STPD)
ρ_{O2} = density of oxygen (1.43 g/L STPD)

$$E_{sweat} = m(\lambda/A_D)$$

E_{sweat} = Heat rejected by sweat evaporation (W/m^2)
m = continuous measured change in body mass (g/min)
λ = latent heat of sweat evaporation (40.8 W hr/g or 2.45 J/g)

Typical weight losses due to respiratory mechanisms amounted to 1-2 g/min each for

evaporation and for loss of mass due to $O_2 - CO_2$ differences (34). The respiratory water

losses are much more difficult to assess for supramaximal exercise as the equations use

VO_2 as a surrogate for respiratory rate which is actually the determining factor. There is

a linear relationship between oxygen consumption and respiratory rate up to 70% of

VO_{2max} but above this threshold, the linearity is not maintained (34). Thus estimating

respiratory water loss is much more difficult at high workloads and will lead to

underestimation using the above equations. There is also a steady diffusion of water

vapor through the skin which is not under thermoregulatory control (1):

$$E_d = [(\lambda)(m)(A_D)(P_s - P_a)]/A_D$$

E_d = heat rejected by evaporation of water diffusion through skin surface (W/m^2)
λ = latent heat of evaporation of sweat (40.8 W hr/g or 2.45 J/g)
m = permeance coefficient of the skin (1.694 x 10^{-4} g/s m^2 mmHg)
P_s = partial water vapor pressure at the skin surface (mmHg)
P_a= partial water vapor pressure in ambient air (mmHg)
A_D = body surface area (m^2)

As well as a component of evaporative cooling provided by respiratory tract water

evaporation (1):

$$E_{res} = 0.0023(M)(44-P_a)$$

E_{res} = Heat rejected by vaporization of respiratory tract water (W/m^2)
M = metabolic rate (W)
P_a = ambient water vapor pressure (mm Hg)

Sweat rates in resting humans without active thermoregulatory sweating have been

estimated to be 0.5 g/min and have been observed as high as 25-30 g/min for short bursts

of activity (1). The maximal evaporative capacity is limited by environmental conditions

and can be expressed by the following:

$$E_{max} = (w)(h_e)(P_s - P_a)$$

E_{max} = maximal evaporative heat transfer (W/m^2)
w = fraction of body surface wet with perspiration = 0.06 + 0.94(E_{skin}/E_{max})
h_e = evaporative heat transfer coefficient (W/m^2 kPa) at sea level = 16.5(h_c)
P_s = partial water vapor pressure at the skin (kPa)
P_a = partial water vapor pressure of the environment (kPa)

Traditionally, the evaporative heat transfer coefficient referenced above for nude subjects

is calculated from the Lewis Relation and the convective heat transfer coefficient (33).

The Lewis Relation expresses the relationship between mass and thermal diffusivity and

is equal to (h_e/h_c) where h_c is the average convective heat transfer coefficient over an

unclothed skin surface (33). This number is constant for still air (18.15ºK/kPa) and for turbulent air flow is 16.5ºK/kPa (33). It can be calculated as follows:

$$LR = 1.65/P_{atm}$$

P_{atm} = atmospheric pressure in atm

CLOTHING AND HEAT TRANSFER

Clothing can affect all modes of heat transfer from the body to the environment. Critical properties of clothing include the amount of trapped air, ventilation allowed, and resistance to vapor transfer (33).

Dry Heat Transfer

Both the clothing and the air boundary surrounding the clothing provide resistance to heat transfer from the skin to the environment. The intrinsic clothing resistance, I_{cl} (m^{2}ºC/W) can be visualized as the resistance the clothing provides to the movement of sensible heat from the skin to the surface of the clothing. The resistance to sensible heat transfer from the clothing surface through the air boundary layer to the environment is represented by I_{a}. Adding clothing increases the effective surface area available for heat transfer, this requires making corrections to I_{cl} and I_{a} by dividing by f_{cl}. For instance, the resistance of an air boundary layer of a nude subject is I_{a}, for the same person wearing clothing, their surface area is increased, which increases the surface are of the boundary layer ($I_{a\ clothed} = I_{a\ nude}/f_{cl}$). For indoor ensembles, f_{cl} can be approximated

by 1.0+0.31(I_{cl}) (35). This allows us to now express the sensible heat transfer from the skin to the environment as:

$$DRY_{sk-envir} = (1/I_T)(T_{sk} - T_o)$$

$$I_T = \text{total insulation of clothing and environment} = I_{cl} + I_a$$

The heat transfer from the skin to the environment can also be represented by using combined heat transfer coefficient and a thermal efficiency factor (1):

$$DRY_{sk-envir} = (h)(f_{cl})(F_{cl})(T_{sk} - T_o)$$

h = combined convective and radiation heat transfer coefficient of the **environment** =

$$1/I_a$$

$F_{cl} = h_{cl} / (h_{cl} + h_{envir})$ (clothing thermal efficiency factor for dry heat exchange – for unclothed subjects it is by definition = 1)

F_{cl} can also be expressed as $= (T_o - T_{cl}) / (T_o - T_{sk}) = (I_a)/(I_a + I_{cl})$

The combined convective and radiation heat transfer coefficient is again dependent upon air velocities, geometry, configuration, and emissivity of the clothing surface and the environment. This coefficient describes the resistance to dry heat transfer from the clothing surface to the environment (this is why it is equal to the inverse of I_a which is the thermal resistance of the air boundary) and says nothing about the intrinsic clothing properties. To examine the heat transfer from the skin to the clothing surface, the thermal efficiency factor (Burton thermal efficiency factor) is used. The Burton thermal efficiency factor describes the magnitude of resistance to dry heat transfer provided by

the air boundary layer in relation to the amount of resistance provided by the clothing.

As the boundary resistance decreases in relation to the clothing, the surface temperature

of the clothing will more closely approximate the ambient temperature:
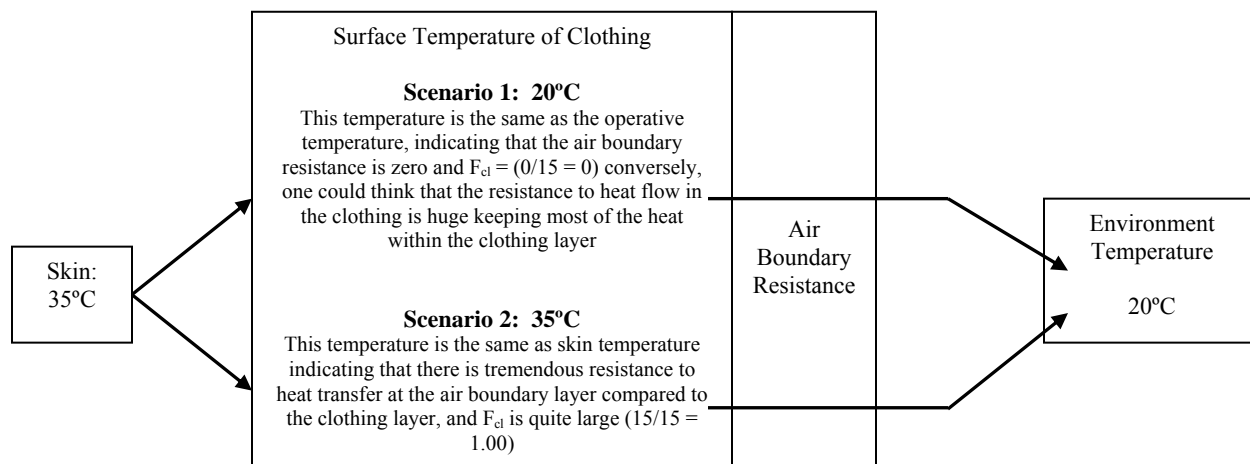
Figure 6: Example calculation of F_{cl}

A commonly cited measure of clothing insulation is the clo. One clo of insulation was

defined as the insulation necessary to maintain comfort and mean skin temperature of

33°C in a room at 21°C with air movement of less than 10 cm/sec, humidity less than

50%, and metabolism of 50 kcal/hr m² (32). It can also be thought of as the equivalent of

furnishing a resistance to sensible heat loss of 0.155 m²°C/W (32). Given I_T in Clo units:

$$(DRY_{skin-envir}) = 6.45 \ (T_{sk} - T_o)/ \ I_T$$

I_T = total insulation from skin to environment expressed as Clo units
T_{sk} = mean skin temperature
T_o = operative temperature
6.45 = 1 clo/(0.155 m² K / W)

Evaporative Heat Transfer

Evaporative cooling is also significantly impacted by clothing and its properties. There are correlates between factors of evaporative efficiency to those used in thermal efficiency. The intrinsic resistance of clothing to vapor transfer is known as I_{ecl} (m^2kPa/W). The environment or air boundary layer also provides a resistance to evaporation; this is expressed as I_{ea} and is equal to $1/h_e$. A total resistance to evaporation can be expressed by $I_t = (I_{ea}/f_{cl} + I_{ecl})$ which again corrects for the increased surface area of the air boundary layer due to clothing. Evaporative heat transfer can now be expressed as:

$$E = (w/I_t)(P_{sk} - P_a)$$

$$w = \text{skin wettedness}$$

The permeation efficiency factor F_{pcl} is analogous to F_{cl} for dry heat loss; it is a measure of the impedance of water vapor transmission through a garment and is unitless. As the resistance increases, F_{pcl} is reduced and by definition for a nude subject it is equal to one. For most normal porous clothing, it ranges between 0.5 and 0.9 and can be represented by the following equation:

$$F_{pcl} = I_{ea} / (I_{ea} + I_{ecl})$$

$$I_{ea} = \text{resistance of the air to the transfer of water vapor}$$
$$I_{ecl} = \text{resistance of clothing to the transfer of water vapor}$$

Which allows the expression of evaporative heat exchange using the following equation:

$$E_{sk} = (w)(h_e)(F_{pcl})(P_{sk} - P_a)$$

w = fraction of body surface wet with perspiration
h_e = evaporative heat transfer coefficient (= 16.5*h_c)
F_{pcl} = permeation efficiency factor (unitless)
P_{sk} = saturated vapor pressure at temperature T_{sk} (vapor pressure at skin)
P_a = ambient vapor pressure

Another factor commonly cited in evaporative transfer is the moisture permeability index

(i_m). It is a dimensionless factor and represents the ratio of the actual evaporative heat

flow capability between the skin and the environment to the sensible heat flow capability

as compared to this same property in air, which is by definition, the Lewis ratio (35). It

can be expressed as follows:

$$i_m = [(1/I_{et})/(1/I_t)] / [(1/I_{ea})/(1/I_a)] = I_t / (LR)(I_{et})$$

It can be seen that i_m can range from 0 for a material impermeable to water vapor (I_{et} =

infinity) to 1 for air ($I_t = I_a$ and $I_{et} = I_{ea}$).

This allows us to express the evaporative heat transfer as:

$$E_{sk} = [(16.5)(i_m)/(I_t)][P_{sk} - P_a]$$

Properties of Different Materials

When considering a clothing ensemble for sport, one must understand certain

basic properties of the materials used to construct these garments. The literature on

textiles abounds with definitions which must be understood to appreciate the discussions

on certain materials. The terms hygroscopic, hydrophilic, hydrophobic, and moisture regain are commonly used to describe a fabric. Hygroscopic describes the property of the material that absorbs or retains water in a reversible fashion. Water is contained within capillaries of the fiber and as such expresses a lower vapor pressure than would water merely resting on the surface of the fiber. Thus, the surrounding vapor pressure must drop before this water will be released. Hydrophilic/hydrophobic are terms used to describe the affinity of fabrics for moisture. These terms do not describe the ability to retain this moisture, only to attract or repel it. Moisture regain in its strict definition is the amount of moisture found in a given fabric at a standard temperature of 70ºF dry bulb and 65% relative humidity, expressed as a percentage of its moisture free weight (wool = 15%, cotton = 7%, nylon = 4%, polyester = 0.4%). By its definition, moisture regain is intimately linked to the hygroscopic properties of a material. New materials used in exercise garments combine a hydrophilic surface characteristic with a low hygroscopic rating to provide a garment which "wicks" the sweat away from the body and does not become saturated with sweat (36).

Polyester is a synthetic fiber which is hollow and possesses limited absorbency (non-hygroscopic) and resiliency, and in its basic form has poor wicking properties. New surface treatments have been developed that make the inner surface hydrophobic and the outer surface hydrophilic which greatly increases the "wicking" capacity of the fibers (33). A highly touted fabric for athletic wear is polypropylene, which is a synthetic fiber with an organic base. It has low thermal conductivity, low moisture regain, high wicking properties, but tends to trap skin oils (33).

Smaller diameter fibers allow for increased contact area with the skin which allows for greater conductive heat transfer between skin and clothing when wet clothing is cooled by evaporation, but also leads to a feeling of "clamminess" under the same conditions (33).

Effects of Different Fabrics on Thermoregulation and Exercise

Kwon et al (36) examined the physiological significance of hydrophilic and hydrophobic materials during intermittent exercise in the heat. Subjects wore clothing consisting of a long sleeve shirt and pants made out of a wool/cotton blend, cotton alone, or polyester. All three ensembles had similar moisture transfer and air permeability characteristics, but a large range of moisture regain (see table 1).

Condition	Material	Moisture regain (%)	Moisture transfer (g/m² hr)	Air permeability (m/kPa s)	Water absorbency (cm/10 min)
A	Wool / cotton	8.7	518.4	4.12	8.6
B	Cotton	6.8	525.6	4.20	8.9
C	Polyester	0.4	518.4	3.68	4.9

Table 1: Garment conditions for Kwon et al (1998).

Moisture regain was defined as water content of fibers at 65% relative humidity per 100g dry fiber.

Subjects exercised intermittently in an environment of 30ºC and 50% relative humidity. The exercise protocol consisted of six bouts of 10 min of cycle ergometry at 40% VO_{2max} followed by five min of rest. After the first three bouts were completed, a fan was utilized to provide a wind velocity of 1.5 m/s over the final three work/rest intervals. Heat strain was reduced in the wool/cotton ensemble once the fan was turned on (table 2).

Fan	Core Temp(ºC)	Skin Temp(ºC)	Body Temp(ºC)	Micro-H Chest(kPa)	Micro-H Back(kPa)	Skin-T Chest(ºC)	Micro-T Chest(ºC)	Surf-T Chest(ºC)	HR
Off	NSD	NSD	NSD	C>A,B	C>A,B	C>A	NSD	A>B B>C	C>B
On	B,C>A	C>A,B	C>A,B	C>B B>A	C>B B>A	C>B B>A	C>B B>A	NSD	C>A,B

Table 2: Comparison of thermal data obtained by different clothing ensembles worn

during exercise in the heat from Kwon et al (36).

A: wool/cotton blend, B: cotton, C: polyester
Micro-H: humidity of microclimate under clothing (at either chest or back site), Skin-T:
temperature of skin at chest site, Micro-T: temperature of microclimate under clothing at chest
site, Sur-T: clothing surface temperature. NSD: not significantly different.
Data in original paper was presented in graphical form only, therefore data is expressed in
relation to each other in lieu of absolute values

It was inferred that the moisture absorbed by the wool/cotton combination provided a

greater evaporative cooling once the fan was activated; resulting in lower skin

temperatures for ensemble A (this is assuming equal air movement and cooling by

convection – based upon similar air permeability's of the fabrics). The authors also

reported the weight gains of each ensemble as: 8.06 g (A), 5.98 g (B), and 1.23 g (C).

This weight gain demonstrated the increased regain/absorption of ensemble A even

though sweat rates were higher in C. Thus depending upon where absorbed water lies in

a garment; when evaporated, the heat of vaporization can come from the environment,

the garment, the microclimate within the garment, or the skin itself. The origin of the

heat of vaporization would be an important point to consider when examining the thick,

layered, construction of most contact sport protective equipment.

Weave structure also plays a role in the heat transfer properties of materials. Nielsen et al

(37) examined the differences in five different knit structures of polypropylene

undergarments (1-by-1 rib, fleece, fishnet, interlock, and double layer rib), all of which

had similar thermal resistances as measured with a manikin. Subjects completed a bout

of 40 min of cycle ergometry at 52% VO_{2max} followed by 20 min of rest, repeated twice,

at environmental conditions of 5ºC and 54% relative humidity.

The authors found that the weave type did not affect core temperatures but did

have a significant effect upon mean skin temperature with the fishnet design recording

the lowest skin temperatures. The fishnet design also yielded the lowest skin wettedness,

and a low total sweat loss, despite the similar thermal resistances among the three fabrics.

The authors attributed the results to the open design of the fishnet underwear which,

when coupled with the pumping action of the arms and legs during exercise, greatly

increased the airflow across the open skin. This benefit was presumably reduced with the

tighter fitting undergarment designs (37). These findings were supported by Nielsen et al

(38), who reported that a tight inner layer resulted in warmer skin temperatures than a

loose fitting inner layer. Moreover, sweating began earlier and at a larger skin

wettedness area in tight versus loose fitting undergarments (38).

There are many new designs of undergarments available to keep athletes cool and

dry. Most of these are some type of polyester which has been modified physically

(grooving) or treated to improve its wicking properties. However, with extremely high

sweat rates or humidity, the moisture remains on the inside layer of the fabric adding to

skin and clothing wettedness (39). Some of these garments are very form fitting which

would reduce airflow and convective cooling next to the skin. Unfortunately, there has

been very little research into the efficacy of these wicking undergarments on

thermoregulation. Gavin et al (40) examined the effects of a commercially available

evaporative polyester fiber in comparison with cotton and semi-nude conditions.

Subjects completed an exercise protocol of 15 min of seated rest : 30 min of running at

70% VO_{2max} : 15 min of walking at 40% VO_{2max} : 15 min of seated rest (simulated wind

speed of 3 km/hr during rest intervals, 11 km/hr during running, and 6 km/hr during

walking). This protocol was performed in an environment of 30ºC and 35% relative

humidity on three occasions, wearing different garments each time. The semi-nude (SN)

condition consisted of wearing a lycra swimming suit, polyester socks, and running

shoes. The cotton (COT) and synthetic (SYN) conditions consisted of wearing a

crewneck, short sleeve T-shirt, cycling shorts, and anklet socks made out of the

respective materials. The only significant results reported were that the COT garments

retained three times as much sweat as did the SN or SYN (approximately 30 g versus 10

g), and as a result, sweating efficiency was significantly lower in the COT group

(approximately 95% versus 98%). Changes in body mass and actual amounts of sweat

evaporated were not different between groups. The authors found no differences in mean

body temperature, rectal temperature, mean skin temperature, VO_2, heart rate, or

subjective differences in comfort (40). It is counterintuitive to find that even with a

decreased sweating efficiency in the cotton ensemble, that core temperature was not

different between groups. This could have been secondary to an increased efficiency of

cooling from convection due to properties of the saturated cotton garment. Pugh (41)

demonstrated that when outergarments were saturated with water during exercise in cold conditions, their insulative properties were reduced to 15% or original values.

Studies of Contact Sport Protective Equipment and Thermoregulatory Challenges

There have been no studies of thermoregulation with hockey protective equipment. However, there have been multiple studies published examining the effects of American football uniforms. Kulka et al (42) examined "critical temperature" and "critical water vapor pressure" for subjects wearing either practice (helmet, undershirt, shoulder pads, jersey, and shorts) or full football (practice gear plus game pants with thigh and knee pads) gear. Critical temperature was defined as being the point of inflection in core temperature elevation while being subjected to gradual increases in ambient dry bulb temperature. Critical water vapor pressure was defined as the point of inflection in core temperature elevation while being subjected to gradual increases in ambient water vapor pressure. Subjects exercised at a constant workload of 35% VO_{2max} on a treadmill without any wind. To find a critical temperature the authors held the ambient water vapor pressure constant at 16 mmHg and increased the temperature by 1ºC every five min. To find the critical water vapor pressure, they held the temperature constant at 36ºC and raised the ambient water vapor pressure by 1 mmHg every five min. The authors compared these critical points with those obtained in another study in which subjects with similar attributes exercised with only cotton shorts, t-shirt, and running socks at the same intensity and environmental conditions (43). The comparison of the three ensembles is shown in table 3.

Ensemble	T_{crit} (ºC) at P = 16 mmHg	P_{crit} (mmHg) at T = 36ºC
Cotton	42.1	25.4
Practice	36.23	17.5
Game	35.73	13.3

Table 3: Critical temperatures and vapor pressures for three levels of clothing.

Cotton: cotton shorts, t-shirt, and running socks, Practice: football practice outfit, Game: football game outfit. Critical temperature indicates the dry bulb temperature under constant water vapor pressure (16 mmHg) at which an inflection in core temperature was obtained. Critical Pressure indicates the water vapor pressure under constant dry bulb temperature (36ºC) at which an inflection in core temperature was obtained.

Mccullough et al (44) examined the thermal and evaporative resistance of football uniforms by the use of an electrically heated manikin. They reported these resistances for five different levels of game and practice attire and compared them to a reference ensemble of shorts and t-shirt. These resistances are presented in table 4.

The football uniforms provided a significant resistance to both sensible and insensible heat transfer, with the temperate uniform providing roughly three times as much resistance to both modes as did the t-shirt and shorts combination.

Ensemble	Clothing Thermal Resistance R_{cl} (m²ºC/W)	Clothing Evaporative Resistance R_{ecl} (m² kPa/W)
G1: Game uniform warm weather	0.124	0.022
G2: game uniform temperate	0.153	0.028
G3: game uniform cold weather	0.158	0.029
P1: practice uniform w/lower pads	0.112	0.020
P2: practice uniform with shorts	0.100	0.017
T-shirt and shorts	0.055	0.009

Table 4: Thermal and Evaporative Resistances of Different Football Uniforms

G1:   Upper Body: Helmet, shoulder pads, sleeveless cut-off t-shirt, short sleeved mesh jersey (tucked into pants).
Lower Body: jock strap, hip girdle with hip, thigh, and tail bone pads, football pants with knee pads and belt, ankle length socks, turf shoes
G2:   G1 but long sleeved knit shirt instead of t-shirt, short sleeved knit jersey instead of mesh (again, tucked into pants), knee length socks, and gloves
G3:   G2 but thick long sleeved shirt, and knit long underwear
P1:   G1 but mesh jersey cut off at waist and hanging loose, and mesh shorts instead of football pants
P2:   P1 but no hip girdle or lower pads

Mathews et al (45) examined the physiological responses during exercise and recovery while wearing a football uniform. Subjects exercised on a treadmill (9.6 km/hr) for 30 min followed by 30 min of recovery while dressed in: 1) shorts only; 2) a football uniform; and 3) shorts plus a backpack weighing the same as the uniform, in environmental conditions of 25ºC dry bulb and 16ºC wet bulb. Rectal temperatures increased by 1.68 C with the uniform during exercise as opposed to an increase of 1.08ºC with the shorts ensemble and 1.37ºC with the backpack. After the 30 min rest interval, rectal temperature remained elevated by 1.20ºC in the pad condition vs 0.42ºC in the shorts condition and 0.65ºC with the backpack. Skin temperatures were elevated by 3-4ºC in the pad versus both no pad conditions and weight loss (as a % of body mass) was

1.8%, 1.1%, and 0.9% in the pad, pack, and short condition respectively (45). These

results demonstrated that the pads imparted a physiological burden during exercise over

and above just the weight of the pads, which was even more magnified during recovery.

Hockey protective equipment would be at least as inhibitive as the temperate game

uniform, perhaps even more so as the lower body equipment is even more extensive and

layered than that of football.

THERMOREGULATORY RESPONSE TO EXERCISE

The body has multiple effectors used to control core body temperature which

include: the rate of metabolic heat production, heat flow via the blood from the core to

the skin, and sweating (1). The central regulation area of body core temperature setpoint

and thermoregulatory effectors is the hypothalamus. The hypothalamus receives inputs

from core temperature sensors located throughout the body (heart, blood vessels, spinal

cord), as well as from temperature sensors from within the hypothalamus itself. The

hypothalamus also receives input from temperature sensors at the surface of the skin.

Effector signals originating in the hypothalamus are based upon integration of peripheral

and central signals in comparison to a desired setpoint. As exercise begins, core

temperature begins to rise. It has been shown that the elevation in core temperature is

largely dependent upon exercise intensity (expressed as a percentage of maximal VO_2),

across a wide range of environmental conditions known as the prescriptive zone (1).

Once ambient conditions exceed this prescriptive zone (lower thresholds with higher

intensity exercise), core temperature begins to rise at a constant workload. It is this change in core temperature which provides most of the stimulus for effector signals to the peripheral vasculature and sweat glands. The contribution or core temperature can be visualized by examining the equation used to calculate body temperature in a subject undergoing vasodilation of the cutaneous beds:

$$T_B = 0.9(T_c) + 0.1(T_m)$$

T_B = Body Temperature
T_C = Core Temperature
T_M = Mean Skin Temperature

In addition, skin temperature tends to exhibit a much larger range of temperatures and as such, even 10 percent of its change makes a large contribution to calculated body temperature (1).

Wyss et al (46) evaluated the effects of skin and core temperatures on the control of skin blood flow (Skbf), sweat rate (SR), and heart rate (HR). They expressed their results as coefficients of multiple regressions for each variable in the following format:

$$\text{Variable} = \alpha(T_{core} - T_{core0}) + \beta(T_{skin} - 33°C) - \delta(-\Delta T_{skin}) + \gamma$$

Core temperature when expressed as right atrial temperature had 22 times the effect on skin blood flow (α=22.1) than did skin temperature, although at the onset of skin heating, skin blood flow increased by 50-100%. When expressing core temperature as esophageal temperature, this was reduced to α=6.5 (all following coefficients are expressed using

core temperatures expressed as atrial temperatures first followed by esophageal temperatures second i.e. $\alpha=22.1/6.5$). These changes demonstrate the lag in changes in esophageal versus atrial temperatures which were shown to be in the range of 1-3 min (46). Skin temperature also had a minor influence on SR ($\beta=15/5.2$) but only once core temperature had reached a threshold high enough to initiate sweating (approximately 37ºC) as well as a minor influence on HR ($\beta=14.3/4.6$). The rate of change in skin cooling had a minor impact on skin blood flow ($\delta=7.6/7.9$) but a significant effect on sweat rate ($\delta=5.0/4.6$) and HR ($\delta=6.8/4.3$).

Thermoregulatory sweating can begin within a few sec to min after starting muscular exercise, and increases in sweating parallel increases in core temperature (1). With increasing sweat rates, there is an initial increase in the number of sweat glands recruited, followed by an increase in the actual sweat rate of each gland. The back and chest have the greatest sweat rates in the body while the limbs have relatively low sweat rates. The sweat glands are innervated by the sympathetic nervous system, but are one of the few effector organs of this system whose ligand is acetylcholine as opposed to norepinephrine. Sweat glands do, however, have receptors for both epinephrine and norepinephrine, and respond in particular to circulating epinephrine (1).

Skin temperature plays a key role in modulating the effector signals of the hypothalamus to the sweat glands. As mean skin temperature rises, the response of the sweat gland for a given core temperature is magnified. Locally elevated skin temperatures will produce increased sweat rates at their site, independent of an elevated mean skin temperature for the entire body, indicating that at least part of sweating is under local control. Another

role that skin temperature plays in latent heat transfer is by its affect on the saturated vapor pressure of the skin. It is generally assumed that the vapor pressure at the skin interface is saturated. If this is the case, as skin temperature is elevated, the saturated vapor pressure is elevated. This provides more of a driving force for latent heat transfer from the skin to the environment (recall the equation for E_{max} contains a component ($T_{sk} - T_a$). Wetting of the skin can serve to gradually reduce sweat secretion, known as hidromeiosis. As the skin becomes saturated, the stratum corneum swells and provides a mechanical resistance to the secretion of sweat (1).

Shibasaki et el (47), recently published a review of the possible roles of non-thermoregulatory modulation of sweating in humans. In it, the authors commented on several areas of on-going research providing evidence for alternative modulating factors on the sweat rate. These factors include: CNS input and local metaboreceptor input secondary to onset of exercise, baroreceptor input, and both osmotic and volume changes. Of these areas, the first two are still areas of many unresolved questions, while the area of osmotic and volumetric changes have many articles in support of a significant effect (47).

Fortney et al (48) examined the effect of hypo/hyper-volemia with normal P_{osm} on sweating responses. Diuretics were used to reduce BV by 8.7% and an isotonic serum albumin infusion was used to raise BV by 7.9% while maintaining normal P_{osm} under both conditions. Hypovolemia led to increased core temperatures during exercise, reduced whole body sweat rates, and reduced the slope of the T_{core} to sweat rate

relationship, but did not increase the core temperature threshold for onset of sweating (48).

Fortney et al (49) attempted to differentiate the effects of hypovolemia and hyperosmolality on skin blood flow and sweating responses. Subjects restricted water intake and performed mild exercise in the heat to dehydrate by 3% after which they either rested (D) in the dehydrated condition or received a PV restoring bolus of NaCl in sufficient concentration to raise P_{osm} by 5% over pre-exercise levels (I). Subjects then exercised in the heat to examine the sweat and skin blood flow responses in these two conditions as compared to a euvolemic, normal osmolality, control condition. Plasma volume was reduced by 5% in the (D) group and by 0% in the (I) group. Plasma osmolality was not significantly different between (D) and (I), which allowed the authors to examine the effect of hypovolemia independent of osmolality changes. They reported the results shown below in table 5.

Condition	PVD Threshold (ºC)	Slope SBF – T_{es} (ml/minºC)	Max SBF (ml/min)	Sweat Threshold$_{chest}$ (ºC core)	Slope Sweat$_{chest}$ – T_{es} (mg/min cm^{2}ºC)	Rise in core temp during exercise (ºC)	HR during exercise (BPM)
Control	37.1	27.1	25.15	36.87	1.59	0.9	158
Dehydrated	37.3 [a]	16.6 [a]	17.1 [a]	37.25 [a]	1.53	1.3 [a]	176 [a]
Hyperosmotic	37.5 [a]	27.6 [b]	22.2 [b]	37.45 [a]	1.69	1.3 [a]	166 [a]

Table 5: Skin blood flow and sweating responses from Fortney et al (49).

Control: euvolemic, euosmolar, Dehydrated: hypovolemic, hyperosmolar, Hyperosmotic: euvolemic, hyperosmolar.

PVD Threshold: esophageal temperature on onset of peripheral vasodilation. Slope SBF/T_{es}: relationship of changes in skin blood flow to core temperature (higher number indicates greater blood flow for given core temperature). Max SBF: maximum skin blood flow obtained. Slope Sweat$_{chest}$ – T_{es}: relationship of sweat rate measured at the chest site to core temperature (higher number indicates greater sweat response for given core temperature) , [a] p<0.05 between control condition. [b] p<0.05 between dehydrated condition.

Increases in P_{osm} independent of decreases in PV can lead to delayed peripheral vasodilation, reduced maximal skin blood flow, and delayed onset of sweating; all resulting in an increased core temperature during exercise (49). Takamata et al (50) measured the effects of plasma osmolality and water ingestion on sweating rates in women volunteers. Their protocol involved administering 1.2 ml/kg of either 3% or 0.9% saline solution to induce an increase in P_{osm} and provide a control group with normal P_{osm}. Plasma osmolality increased due to the infusion ($\Delta16.8$ mosm/kg H_2O), as did arginine vasopressin (AVP) ($\Delta3.3$ pg/ml) and that these increases were associated with an increased core temperature at the onset of sweating (subjects were passively heated by a lower body water bath and required an elevation in core temperature of 0.91ºC and 0.40ºC in the 3% and 0.9% groups respectively to induce sweating). This delayed sweat response was immediately removed when the subjects drank 4.3 ml/kg of 38ºC deionized water. These results supported prior findings that elevated P_{osm} causes a delayed onset of sweating, and that this delay was a central effect as the ingested water caused an immediate response with no changes in P_{osm} evident. The authors attributed this response to afferent input from the oropharynx region or perhaps from osmoreceptors in the hepatoportal region (50).

<u>INTERMITTENT EXERCISE</u>

High intensity intermittent exercise has received much less attention relative to steady

state aerobic exercise, most likely in part due to the inherent difficulties of assessing

energy contributions as mentioned in section II. However, high power bursts interspersed

with short periods of recovery are major components of many sports. Often the

successful athlete is not just the one who can jump the highest or run the fastest, but also

the one who can recover the quickest in order to repeat these actions multiple times.

When examining the published literature, the term "high-intensity" can be taken to mean

many different things. For the purposes of hockey and other fast paced team sports, it

needs to incorporate short (1-10 sec) bouts of maximal or near maximal (far exceeding

the power output at VO_{2max}) power output, interspersed with varying lengths of recovery.

METABOLISM DURING HIGH INTENSITY INTERMITTENT EXERCISE

Gaitanos et al (14) examined the muscular metabolism during a series of 10

maximal cycle sprints. The sprints were six sec in duration with 30 sec of rest in between

sprints (10x6 test). The authors measured peak power (highest power over 1 sec) and

mean power (averaged power over entire six sec sprint). The highest peak power was

attained in sprint number one, and was 1253 W (approximately five times the power

produced at VO_{2max}). This peak power was maintained until sprint five, when it

decreased by 15.9% and fell by 33.4% by sprint ten. The highest mean power was also

obtained in sprint one (870 W), and was maintained until sprint four. The mean power

was decreased by 12.6% during sprint five and by 26.6% during sprint ten. Blood

lactates and pH results are shown below in figures 7 and 8, plasma catecholamines are

shown in figure 9.
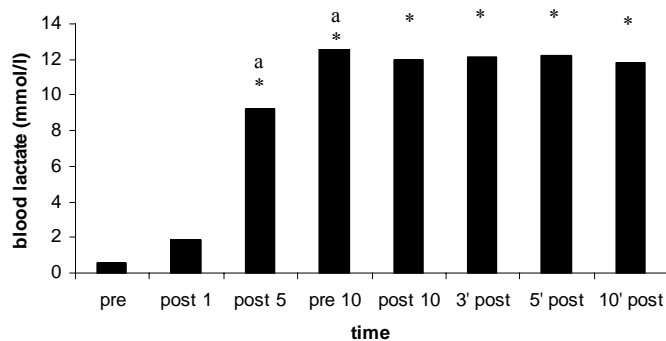
Figure 7: Blood lactates during (10) six sec maximal cycle sprints with 30 sec rest.

Pre: pre-exercise, post 1: immediately following sprint #1, post 5: immediately following sprint #5, pre 10: prior to sprint #10, post 10: immediately following sprint #10, 3'/5'/10' post: 3,5, or 10 min after sprint #10. a: $p<0.01$ from previous sample, *: $p<0.01$ from pre-exercise.
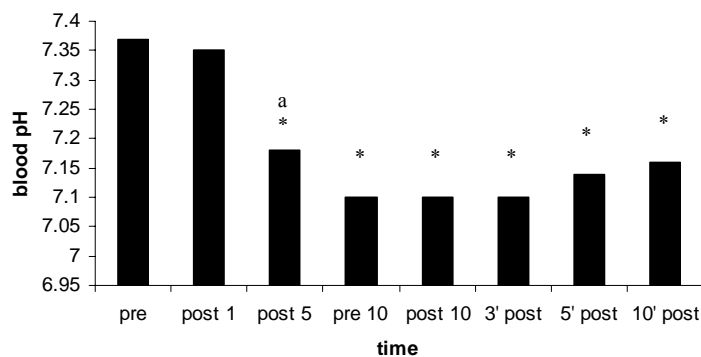
Figure 8: Blood pH during (10) six sec maximal cycle sprints with 30 sec rest.

a: $p<0.01$ from previous sample, *: $p<0.01$ from pre-exercise.

The authors obtained muscle biopsies at rest, after the first sprint, immediately prior to

the tenth sprint, and following the tenth sprint. The results of these biopsies are shown in

table 6. From these muscle biopsy results, the authors were able to estimate the ATP

production from anaerobic sources during the first and final sprint of the test. These

estimates are shown in table 7.
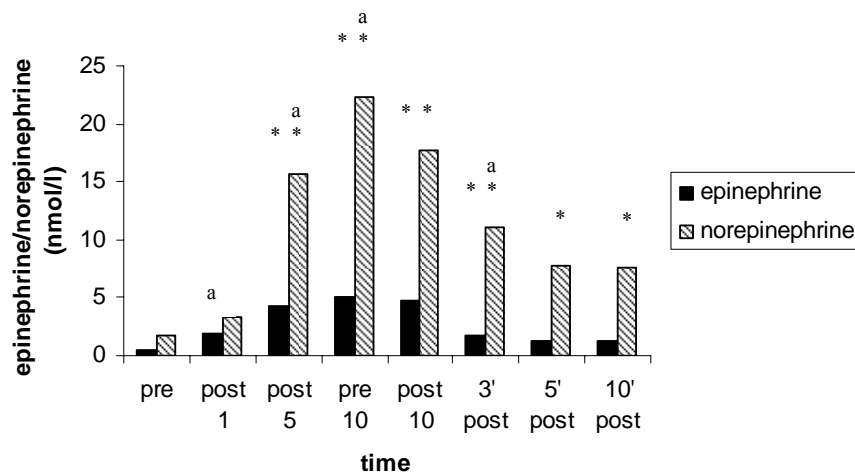
Figure 9: Blood epinephrine/norepinephrine during (10) six sec maximal cycle sprints

with 30 sec rest.

a: p<0.01 from previous sample, *: p<0.01 from pre-exercise.

Metabolite	Pre sprint 1	Post sprint 1	Pre sprint 10	Post sprint 10
Glycogen	316.8	273.3 [a]	221.0 [a,b]	201.4 [a]
ATP	24.0	20.9 [a]	16.4 [a,b]	16.4 [a]
PCr	76.5	32.9 [a]	37.5 [a]	12.2 [a,c]
Glucose	1.4	2.5 [a]	7.9 [a,b]	8.2 [a]
Lactate	3.8	28.6 [a]	116.2 [a,b]	112.3 [a,c]
Pyruvate	0.6	2.0 [a]	1.6 [a]	1.8 [a]

Table 6: Muscle metabolites following ten repeated six sec maximal sprints.

Glycogen: muscle glycogen (mmol/glucosyl units/kg dry wt), ATP: adenosine triphosphate (mmol/kg dry wt), PCr: phosphocreatine (mmol/kg dry wt), Glucose: muscle glucose (mmol/kg dry wt), Lactate: muscle lactate (mmol/kg dry wt), Pyruvate: muscle pyruvate (mmol/kg dry wt). a: significantly different from resting values, b: significantly different from previous sample, c: significantly different from post sprint 1.

System	ATP production mmol/kg dry weight during sprint #1	ATP production mmol/kg dry weight during sprint #10
PCr	44.3	25.3 [a]
Glycolysis	39.4	5.1 [a]
Total (including ATP loss)	89.3	31.6

Table 7: Anaerobic energy system ATP production during ten repeated six sec maximal

sprints

PCr: ATP production from breakdown of muscle phosphocreatine (ΔPcr), Glycolysis: ATP production from flux through glycolysis ($1.5*\Delta$lactate + $1.5*\Delta$pyruvate), Total: total anaerobic ATP production (ΔPcr + ΔATP + $1.5*\Delta$lactate + $1.5*\Delta$pyruvate)
a: significantly different from sprint 1.

In summary, despite the brief duration of each sprint, there was a reduction in power over

the series of sprints, with mean power maintained until sprint 4 and peak power until

sprint 5. These changes were associated with a greatly reduced anaerobic regeneration of

ATP, which could be driven by inhibition of the pathways, or a reduced demand due to

inhibition of the contractile apparatus. This reduction in ATP turnover was associated

with a large reduction in blood and muscle pH which has been reported to at least

partially inhibit both glycolysis and cross bridge cycling. The authors estimated muscle

pH to be approximately 6.59 at their measured blood pH of 7.10, indicating that a fall in

pH was at least partially responsible for the decrease in flux through the anaerobic

pathways (14). The PCr levels were not restored well prior to the final sprint (49% of

resting value). If we assume that PCr levels were approximately the same following

sprint 9 as they were following sprint 10, it is possible to calculate a PCr restoration rate

of 1.3 mmol/kg DM (14). This value is in agreement with previously reported PCr

resynthesis rates, and indicates that it was not the restoration rate of PCr which was the

limiting factor, rather the duration of the recovery interval was insufficient to allow for

adequate PCr resynthesis. It was interesting to see that while anaerobic sources of ATP

provision fell by 65% from sprint #1 to sprint #10, the mean power output was only

reduced by 27%. This disparity suggests either the contractile process became more

efficient (unlikely), or that the aerobic energy system became more prominent in its

energy providing role in the latter sprints (14).

The rate of PCr resynthesis following a single or repeated sprints and its relationship with

intramuscular pH, was investigated by Dawson et al (51). Subjects performed either one

or a series of five six sec sprints, each followed by 24 sec of rest (5x6). Muscle biopsies

were obtained immediately prior to and following (at 10 sec, 30 sec, and 3 min) the single

sprint, and the last sprint in the repeated sprint condition. Even though muscle lactate

concentrations were very high (and by association, pH's very low), there was no

reduction in the rate of PCr resynthesis for sprint five versus sprint one. The biopsy data

are shown in table 8 and a graphical representation of PCr restoration in figure 10:

Metabolite	Rest	10s post-1	30s post-1	3min Post-1	Rest	10s post-5	30s post-5	3min Post-5
ATP	24.3	20.5[a]	21.0[a]	22.5	22.8	15.1[a,d]	16.7[a,d]	19.8[a,b,c]
ADP	3.1	3.4[a,c,d]	3.2[b]	3.0[b]	3.0	4.2[a,c,d]	3.4[a,b,d]	3.1[b,c]
PCr	81.0	44.9[a,c,d]	55.6[a,b,d]	73.1[b,c]	77.1	21.5[a,c,d]	34.5[a,b,d]	64.5[a,b,c]
Lactate	6.8	42.5[a,c,d]	36.5[a,b,d]	20.9[a,b,c]	7.7	103.6[a,d]	88.0[a,d]	62.5[a,b,c]

Table 8: Muscle biopsy data from five repeated six sec sprints.

Muscle biopsy taken at 0, 10, 30, and 180 seconds following a series of five – six second
maximal sprints.
ATP: adenosine triphosphate (mmol/kg dry wt), ADP: adenosine dipohosphate (mmol/kg dry wt),
PCr: phosphocreatine (mmol/kg dry wt), Lactate: muscle lactate (mmol/kg dry wt).
a: P<0.01 from pre, b: P<0.01 from 10s post, c: P<0.01 from 30s post, d: P<0.01 from 3 min post.
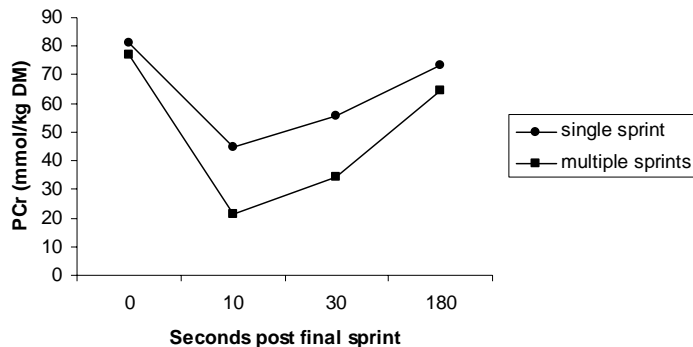
Figure 10: Time course of Phosphocreatine (PCr) resynthesis following single or

multiple intermittent sprints.

Muscle biopsy taken at 0, 10, 30, and 180 seconds following a series of five – six second
maximal sprints.

A decreased regeneration rate of PCr has been linked to a decrease in intracellular pH by

some, but was not supported by either of these experiments. It is intuitive to predict that

an increase in intramuscular $[H^+]$ would reduce the rate of PCr resynthesis as hydrogen

ions are liberated during PCr resynthesis and would inhibit the reaction due to mass

action.

$$PCr + MgADP^- + H^+ \longleftrightarrow MgATP^{2-} + Cr$$
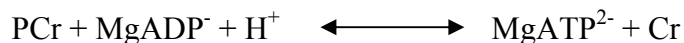
The resynthesis of PCr has been shown to be biphasic with a fast ($t_{1/2}$ of 21s) and a slow

($t_{1/2} = 170s$) component (52). A reduction in intramuscular pH (as well as elevations in

ADP) does have an inhibitory affect upon the slow component which is often

undetectable in short recovery durations such as the ones seen in the above studies.

Moreover, the ATP needed for the resynthesis of PCr is predominantly oxygen dependent

as glycolysis ceases upon the termination of exercise. Thus, perhaps individuals with

higher aerobic capacities would have faster PCr resynthesis times, but due to the large

inter-individual and inter-protocol variations found in PCr resynthesis, this has proven
extremely difficult to validate (52).

Bogdanis et al (53) investigated the contribution of PCr and aerobic metabolism to the
energy supply during repeated sprints. Subjects completed a 30s maximal sprint
followed by four min of recovery at which time they completed either a 10s or 30s sprint.
Muscle biopsies were obtained and respiratory gasses collected to evaluate the aerobic
and anaerobic contributions to energy supply. Anaerobic energy contributions from
sprint 1 to sprint 2 were reduced by 41% even though mean power was reduced by only
18%. This reduction in anaerobic contribution was compensated in large part by an
increase in VO_2 consumption from 2.68 L/min to 3.17 L/min. There was a significant
relation between the percentage of PCr resynthesis and the percentage of restoration of
mean power (r=0.84) and pedaling speed (r=0.91) during the first 10s of sprint 2.
However, no correlation between power recovery and muscle pH was found. The
restoration of PCr at 4 min post sprint #1 was correlated with the % VO_{2max} exhibited at 4
mmol/L blood lactate (a commonly used indicator of aerobic training status) (r=0.94
P<0.01) (53). These results suggest the increased importance of the aerobic system in
both regeneration of PCr and recovery of power between maximal sprints, as well as a
direct contribution to power output during such sprints.
It appears that the aerobic systems contribution to maximal exercise is elevated when
performing repeated sprints, perhaps due in part to a slow initial onset of oxidative
metabolism in working muscles. Bangsbo (54) demonstrated that although oxygen
consumption in working muscles is elevated within sec of intense exercise, it requires 45

sec to reach maximal levels. This restriction was not related to activity of pyruvate dehydrogenase; rather it seemed to be linked to local mismatches in blood distribution. Krustrup et al (55) found that a seven week high intensity (one leg intermittent knee extensor training at 150% peak thigh VO_2 3-5 times per week) training program elevated muscle oxygen uptake at the onset of high intensity exercise and was due to elevations in blood flow and vascular conductance.

FATIGUE IN HIGH INTENSITY INTERMITTENT EXERCISE

Fatigue in high intensity intermittent exercise can be defined as either the failure to maintain a high power output for a given interval, or the lack of ability to maintain high power outputs over repeated short bursts. The mechanisms responsible for such fatigue have remained elusive, but some tested hypotheses do exist. The reduction of power output could originate in a reduced central drive to recruit motor units, or from a peripheral inhibition of either cross bridge cycling and/or ATP regeneration. At the onset of high intensity exercise, there is a rapid depletion of PCr and a generation of H^+ secondary to the reactions of glycolysis. A number of studies have reported the restoration of power to be significantly correlated with restoration of PCr, implicating the ability to restore PCr is as a determinant of the ability to maintain high power outputs over repeated bouts (52, 56) while others have not found such a relationship (57, 58). The reduction in intramuscular pH has been associated with multiple mechanisms which may be associated with a decrease in power output. The disturbance may play a role in the hydrolysis of ATP due to a mass action effect:

$$MgATP^{2-} + H_2O \longrightarrow MgADP^- + P_i + H^+$$
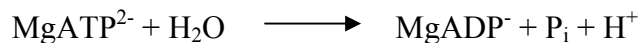
A decreased pH has also been shown to inhibit key enzymes in the glycolytic pathway, in particular phosphfructokinase (52). There may also be inhibition of the contractile apparatus as well. A reduced pH has been associated with disturbances in the release of Ca^{+2} ions from the sarcoplasmic reticulum, as well as resulting in contractions requiring an elevated sarcoplasmic concentration of Ca^{+2} to obtain the same strength (52). Another possible mechanism of fatigue which is receiving a great deal of attention recently is the accumulation of interstitial K^+. This leads to disturbances in the membrane potentials of the muscle fibers, and K^+ concentration is the primary determinant of the resting membrane potential. Nielsen et al (59) studied the effects of high intensity unilateral intermittent training on K^+ kinetics and fatigue. The authors found that a seven week training program was successful in reducing the accumulation of extracellular K^+ probably through the enhanced uptake of K^+ by Na-K-ATPase pumps. Interestingly, the lower accumulation of extracellular K^+ was associated with a delayed fatigue in the trained versus untrained leg, but the absolute levels of K^+ were identical at fatigue in both legs (59).

Glycogen availability has also been cited as a possible contributor to fatigue in intermittent exercise and has been reported to decline by 14% in the Vastus Lateralis following a single six sec all cycle sprint (14). Balsom et al examined the effects lowered intramuscular glycogen on intermittent exercise performance by having subjects perform repeated sprints in either low or high glycogen conditions (60). Subjects performed two

sprint tests, the first consisting of 15 six sec sprints interspersed by 30s of rest while attempting to maintain 140 rpm (at a normalized power output of 958 W); the second performed 24 hours later, consisting of the same sprints performed until volitional fatigue. Glycogen levels were manipulated by performing a glycogen depleting cycle protocol followed by dietary restriction in order to reduce muscle glycogen. This protocol was followed for one day prior to the short sprint and maintained throughout the long sprint condition performed 24 hrs later. Subjects were better able to maintain power output during the short sprints in the high glycogen condition (starting glycogen 180 and 397 mmol/kg dry mass in the low and high glycogen conditions respectively) even though the change in muscle glycogen was not significantly different between groups. Subjects were also able to complete almost three times (111 vs 294) as many sprints in the high glycogen versus low glycogen condition when exercising to failure (starting glycogen 181 and 540 mmol/kg dry mass in the low and high glycogen conditions respectively) (60). The authors pointed out that at the completion of the initial 15 sprints, glycogen levels were 128 and 319 mmol/kg dry mass respectively in the low and high glycogen groups, indicating that total muscle glycogen was not depleted. There was, however, no measurement at the single fiber level, which may be important in high intensity intermittent exercise (60). Gollnick et al has demonstrated that glycogen depletion occurred initially in the type II fibers during high intensity exercise (61).

During more prolonged bouts of exercise (such as an entire game) the mechanism of fatigue is more complex, involving significant components of the CNS such as motivation and drive (62). Mohr et al (63) studied the ability of soccer players to

generate high power outputs following an "intense" period of play during a game.

Players performed three 30 m sprints with 25s of active recovery prior to the game, after

an intense period in the first half, following an intense period in the second half, and at

the end of the game. They found that the ability to generate power was impacted by the

preceding "intense" activity, but the slope of the decline in power was similar at all time

points. A weak but significant correlation was found between muscle lactate and

decreased sprinting performance after an "intense" period (63). However, muscle lactate

concentrations during the game were quite low in comparison to those found at

exhaustion in intermittent exercise protocols (63).

DETERMINANTS OF POWER MAINTENANCE AND RECOVERY IN INTERMITTENT

SPRINT ACTIVITY

It is likely that the determinants of repeated sprint ability are complex and multi-

factorial as it presents a unique blend of anaerobic and aerobic demands. Bishop et al

(64) attempted to quantify the importance on muscle buffer capacity and aerobic fitness

in repeated sprint ability. Blood and muscle samples were collected prior to and

following a 5x6 test in 34 untrained women. The best predictor of the ability to prevent

fatigue was a combination of in vitro muscle buffering capacity and lactate threshold

(LT) (% decrement = $24.60 - 0.02 \beta m_{in\ vivo} - 0.06 LT$, $R^2 = 0.75$ SE=1.8%). Moreover, there

was a significant correlation between total work and VO_{2max} (r= -0.60) and LT (r= -0.56).

These relationships appear stronger in these untrained women compared to trained field

hockey players, indicating that training likely impacts this relationship. The authors

anticipated a stronger correlation for LT than VO_{2max} because LT is a better indicator of

the peripheral adaptations to aerobic training, while VO_{2max} is an indicator of the central

ability to supply oxygen, and based on the results of an earlier study showing a strong

correlation by Gaitanos et al (14). This lack of a stronger relationship may have been due

to their selection of untrained women for the study who may not have developed a large

differentiation between VO_{2max} and LT (64).

In an attempt to address this inter-subject variability, Tomlin and Wenger divided women

recreational soccer players into either a low or moderate VO_{2max} group (34.4 and 47.6

ml/kg/min respectively) (65). They studied the relationship of aerobic variables and

power indices during a 10x6 test. They found that the low and moderate groups

generated the same peak power but that the low group had a significantly larger

decrement in power over the 10 sprints (18% vs 8.8%). The authors also found that

VO_{2max} was a strong negative predictor of power decrement ($r = -0.65$, $p = 0.02$) and that

oxygen consumption in the recovery periods was higher in the moderate versus low

VO_{2max} group (65). Dawson et al (66) examined the relationship between a 6x6 cycle test

or a series of repeated 40m sprints departing every 30s; and other commonly used indices

of aerobic and anaerobic capacities. The authors reported multiple correlations between

tests which are shown in table 9:

Repeat Sprint Test Variable	VO$_{2max}$ cycle L/min	VO$_{2max}$ cycle ml/kg/min	10s Sprint kJ	Peak Power W	30s Work kJ	VO$_{2max}$ run L/min	VO$_{2max}$ run ml/kg/min	VJ	10m Sprint	40m sprint	400m
Total kJ or time	0.664	NSR	0.937	0.700	0.913	NSR	0.487	0.813	0.878	0.912	0.910
% decrement	NSR	0.555	0.596	0.680	0.699	0.617	NSR	NSR	NSR	NSR	NSR

Table 9: Correlations between indices of repeat sprint tests and other aerobic and

anaerobic indices.

Repeat sprint test variable indicates total work/sprint time, or % decrement of either cycling or running test. Last six columns are correlated against repeat sprint running test while first five columns are correlated with repeat cycle sprint test. All results $p<0.05$.

These results demonstrate the reliance upon both aerobic and anaerobic energy systems

with a bias towards anaerobic involvement (66).

VALIDITY AND RELIABILITY OF TESTS USED TO EVALUATE REPEAT

SPRINT POWER

Glaister et al evaluated the reliability of power output during maximal intensity

intermittent cycling (67). The authors had two groups of subjects complete trials of 20

five-sec maximal cycle sprints with either 10 or 30 sec of rest. The subjects completed

eight trials during a seven week period and were evaluated for changes in their maximum

and mean power output over all 20 sprints. The authors found that two familiarization

trials were necessary to eliminate a learning affect, after which, all calculated power

indices other than maximum power in the short rest interval protocol, demonstrated

coefficients of variability of 2.4-3.7% (67). In addition, reliability data from these tests

demonstrate that use of the percent decrement score shown below provided the most

reliable method of expressing fatigue within a series of sprints (intraclass correlation coefficients of 0.81 and 0.83 for the 10s and 30s rest intervals respectively) (68):

$$Fatigue = 100 - [(total\ power\ output)/(ideal\ power\ output)\ x\ 100]$$

Total power output = sum of mean power values from all sprints

Ideal power output = (number of sprints)(highest mean power obtained within a sprint)

EXERCISE IN THE HEAT

Exercise in the heat provides many challenges to an athlete; they must balance their cooling requirements and sweat losses while attempting to maintain an adequate plasma volume as not to impede oxygen delivery to the active muscles. It has been demonstrated that exercise in the heat can bring about fatigue earlier than exercise in thermoneutral conditions. This section will address the mechanisms of this fatigue, as well as potential mechanisms for the generation of increased isolated power outputs at elevated temperatures.

ELEVATED MUSCLE TEMPERATURE AND INCREASED POWER OUTPUT

Rall et al (69) described the thermal dependence of muscle function in many different aspects. The authors point out that the isometric contraction force for many different muscle groups increases with physiological increases in temperature. The

determination of isometric force generation depends upon the number of attached cross bridges and the average force generated by each cross bridge. The number of attached cross bridges can be estimated looking at instantaneous stiffness of a contracting muscle; and as stiffness has not been shown to be drastically altered by elevations in temperature, it is assumed that the mean forces generated per cross bridge are increased in elevated temperatures. It has also been demonstrated that the concentration of Ca^{+2} to sustain 50% of maximal force is inversely correlated with temperatures. Other characteristics of muscle contraction shown to be sensitive to temperature elevations include: rate of isometric force development, maximum shortening velocity, relaxation, and maximal power output (69)

De Ruiter et al (70) studied the effects of temperature on the rates of isometric force development and relaxation in the adductor pollicis (AP) muscle in humans. They found that maximal isometric force was reduced by 16.8% at a muscle temperature of 22°C but was unchanged at temperatures ranging from 25-37°C. Results were presented in the form of temperature coefficient or Q_{10}:

$$Q_{10} = (R_2/R_1)^{(10/(T2-T1))}$$

R_x = rate process of interest
T = temperature $(T_2 > T_1)$

The Q_{10} values for rate of maximal force generation and relaxation were about 2.0 in the temperatures ranging from 37 to 25°C, and increased to 3.8 (more temperature dependent) over the range from 25 to 22°C. These results support previous findings that

colder temperatures require longer times for maximal force generation and relaxation, particularly when examining large physiological differences in temperature (22 vs 37ºC) (70). Interestingly, after fatiguing the muscle, many of the previous findings (reduced maximal force generation, reduced rate of force development, and prolonged relaxation rate) were attenuated with lower temperatures (70).

A regression equation was generated which allowed estimation of muscle temperature from measured skin temperatures. This equation was based on muscle electrodes placed 20-30 mm into the AP muscle, compared with the temperature readings obtained by a sealed skin electrode overlying the muscle belly while the hand was immersed in 45ºC water:

$$\text{Muscle Temperature} = 1.02(\text{Skin Temperature}) + 0.89$$

$$R^2 = 0.98$$

It is unknown how this equation will extrapolate to other muscle groups that are not so superficial to the skin or have been heated/cooled by other means (70).

In order to examine the effects of temperature on the force velocity relationship of concentric muscular contractions; De Ruiter et al (71) performed the same experiments on actively shortening AP muscles. Maximal isometric force, maximal rate of force development, maximal shortening velocity, and power output were all reduced with decreasing muscle temperatures (22 vs 37ºC) (71). These differences were again reduced when evaluating smaller thermal differences such as muscle temperatures of 31 versus

37ºC. Maximal shortening velocity was reduced by 24% in the fresh and by 8% in the fatigued muscle when temperatures were dropped from 37 to 31ºC, while maximal rate of force development was reduced by 22% in the fresh but unaffected in the fatigued muscle during the same conditions (71). It has also been demonstrated that muscle force begins to decline with muscle temperatures above 37ºC (72).

Sargeant (73) examined the effects of muscle temperature on brief power output during cycling in men. Subjects perform a maximal isokinetic (95 rpm) cycle test with muscle temperatures of 29.0, 31.9, 36.6 (room temperature), and 39.3ºC (muscle temperatures modified by 45 min immersion in hot or cold water). Elevated muscle temperature of 39.3ºC, resulted in a higher peak (110%) power output than did a muscle temperature of 36.6ºC. Cooler muscle temperatures (31.9ºC and 29.0ºC) resulted in lower peak power outputs (88% and 79%) than room temperature. There was also an associated increase in rate of fatigue in the elevated muscle temperature condition (33W/s versus 20 W/s in the 29.0ºC condition) (73).

Gray et al (74) examined the effects of elevated muscular temperature on ATP turnover and conduction velocity during a maximal six sec cycle sprint (74). Leg temperatures were elevated by immersion in a 43ºC water bath to increase vastus lateralis temperatures by 3.3ºC (37.5 vs 34.2ºC) (of note, rectal temperatures were elevated to 37.2 and 37.1ºC in the heated and non-heated conditions respectively). This was followed by completion of a single six-sec maximal sprint during which time EMG signals were

recorded and muscle biopsies were obtained. The elevation in muscle temperature led to an increase in both maximal and mean power output (21 and 15% respectively) (74).

ALTERATIONS IN METABOLISM DURING EXERCISE IN THE HEAT

Shifts in Aerobic and Anaerobic Metabolism Due to Cardiopulmonary Factors

Exercise in the heat does not necessarily entail exercise while dehydrated, which will be addressed in the following section. It often does imply exercise under a condition of elevated core temperature. Many of the published articles contain some combination of elevated core and or skin temperatures mixed in with some level of dehydration. In order to separate the effects of hyperthermia and dehydration, they must be controlled for in the study of interest.

Febbraio (75) published a review article on how heat stress alters muscular metabolism during exercise. Based on the evidence, it appeared that if exercise in the heat is submaximal in nature and a marked (0.5ºC) increase in body core temperature was observed, intramuscular carbohydrate utilization was increased through both aerobic and anaerobic means (75). Many, but not all, studies have documented increased muscular levels of lactate following exercise under hyperthermic conditions as well as an increased respiratory exchange ratio, both indicative of respective increases in carbohydrate usage by anaerobic and aerobic energy systems. This increased carbohydrate usage appears to come from intramuscular glycogen and not from uptake of glucose. Elevated blood lactate levels could also be due to decreased clearance of lactate by the liver, a fact which may be supported by the findings of Rowell et al (76), who measured hepatic lactate

clearance at 58% of normal during heat stress and exercise, but commented that lactate levels would have been elevated even with normal hepatic clearance. The effects of heat stress and exercise on protein metabolism have not been well studied; but some studies do find possible indirect evidence of increased protein use in the fact that blood ammonia is often elevated during exercise and heat stress. This elevated ammonia may come from either protein catabolism or from degradation products of ATP. There are a number of possible mechanisms which may contribute to altered metabolism in hyperthermic subjects. One may be a reduction in skeletal muscle blood flow during such exercise, secondary to elevated cutaneous skin blood flow. This contention has met with mixed results with some papers supporting and some refuting this finding (75). A second mechanism may include the effect of a compromised cardiovascular system as a result of hyperthermia.

Gonzalez-Alonso et al (77) designed an experiment in an attempt to identify the limiting factors of aerobic exercise in the heat. Cardiopulmonary as well as peripheral vascular factors were measured during exercise in a heated condition which elevated core temperatures by 1ºC and skin temperatures by 10ºC over neutral conditions. Subjects exercised at maximal aerobic power to volitional failure (5.45 min) in the heat followed by a one hour recovery/hydration period. After this recovery, subjects repeated the exercise in a fan cooled euthermic environment and stopped at the same time point as the volitional fatigue in the hot condition (timepoint #1). This was again followed by a rest period and finally, exercise in thermoneutral environment until volitional fatigue (timepoint #2). The authors reported the following results shown in Tables 10, 11, and 12.

Variable	Timepoint #1	Timepoint #2
Cardiac Output	E	E
Heart Rate (HR)	D	E
Stroke Volume (SV)	I	E
Mean Arterial Pressure (MAP)	I	E
VO$_2$	I	I
PO$_2$	E	E
O$_2$ saturation	E	E
Systemic O$_2$ delivery	E	E
Femoral Blood Temperature	D	E

Table 10: Central cardiopulmonary values during exercise in thermoneutral conditions

compared to prior exercise in heat.

Timepoint #1: Thermoneutral condition at the same timepoint as volitional fatigue occurred in the hot condition (variable expressed as relative to volitional fatigue in hot condition D: indicating decreased at same timepoint in thermoneutral vs volitional fatigue in the heat, E: equivalent, I: increased).
Timepoint #2: Thermoneutral condition at timepoint of volitional fatigue (variables expressed as relative to volitional fatigue in hot condition).

Variable	Timepoint #1	Timepoint #2
2 leg blood-flow	I	I
2 leg O$_2$ Uptake	I	I
2 leg O$_2$ Delivery	I	I
Leg A-V O$_2$ Difference	E	E
Vascular Conductance	E	E

Table 11: Peripheral cardiopulmonary values during exercise in thermoneutral conditions

compared to prior exercise in heat.

Timepoint #1: Thermoneutral condition at the same timepoint as volitional fatigue occurred in the hot condition (variable expressed as relative to volitional fatigue in hot condition D: indicating decreased at same timepoint in thermoneutral vs volitional fatigue in the heat, E: equivalent, I: increased).
Timepoint #2: Thermoneutral condition at timepoint of volitional fatigue (variables expressed as relative to volitional fatigue in hot condition).

Variable	Timepoint #1	Timepoint #2
ATP	E	E
PCr remaining	I	E
Lactate accumulation	D	E
Lactate Efflux	E	E
Glycogen	E	E
Norepinephrine	E	I
Epinephrine	E	I

Table 12: Muscular and plasma metabolites during exercise in thermoneutral conditions

compared to prior exercise in heat.

Timepoint #1: Thermoneutral condition at the same timepoint as volitional fatigue occurred in the hot condition (variable expressed as relative to volitional fatigue in hot condition D: indicating decreased at same timepoint in thermoneutral vs volitional fatigue in the heat, E: equivalent, I: increased).
Timepoint #2: Thermoneutral condition at timepoint of volitional fatigue (variables expressed as relative to volitional fatigue in hot condition).
Lactate efflux exhibited a mild trend toward being significantly elevated in heat (p=0.015)
ATP: adenosine triphosphate, PCr: phosphocreatine

Maximal oxygen consumption was blunted in the heat versus the thermoneutral condition

(4.28 L/min versus 4.72 L/min respectively) and a reduced time to fatigue was exhibited

in the hot condition (5.45 versus 7.63 min) (77). In both hot and neutral conditions, a

drastic reduction in cardiac output and leg blood flow occurred just prior to volitional

fatigue. The addition of thermal stress appeared to shift this threshold to an earlier

timepoint. These results indicate that in maximal aerobic exercise in the heat, VO_{2max} is

reduced secondary to rapidly declining cardiac output and mean arterial pressure which

leads to a reduced blood and oxygen flow to the exercising muscle. Moreover, the

decline in cardiac output can be attributed to a reduced stroke volume (SV) as heart rate

was increasing as cardiac output fell, leading to reduced oxygen delivery and a greater

reliance upon anaerobic metabolism even in the face of equivalent catecholamine levels.

These results occurred in the face of an unmeasured but expected 3-5 times increase in skin blood flow which in some, but not all, studies has shown a reduction in central venous volume and cardiac filling (77). When examining reduced stroke volumes, possible contributing mechanisms would include reduced preload, reduced contractility, reduced filling time, and/or increased afterload. In this study, the MAP was falling along with cardiac output which would remove elevated afterload as a contributing factor. One would not expect reduced cardiac contractility especially in the face of similarly elevated catecholamines, but other driving factors such as centrally mediated control signals, perhaps modified by elevated temperatures, cannot be ruled out. As heart rate was increasing as cardiac output was falling, it is possible that the reduced filling time was responsible for the fall in stroke volume. Reductions in VO_{2max} during exercise in the heat can have important ramifications when using percentages of power output at VO_{2max} in thermoneutral conditions as exercise will be at elevated percentages of VO_{2max} when performed in the heat.

Rowell et al (78) demonstrated that during mild exercise (27-39% VO_{2max}), HR and CO rapidly increased, while SV, aortic mean pressure (A_o) and central blood volume (CBV) fell with spontaneous increases in skin temperature (38.3ºC) induced by liquid perfused suits. These changes were reversed with spontaneous skin cooling to 26.9 ºC and occurred with only mild disturbances to core temperature (78). When performing the same interventions on subjects exercising at a higher intensity (51-64% VO_{2max}), they found the same responses in CO, HR, and SV; but saw counterintuitive increases in A_o with heating and reductions with cooling. The authors attempted to explain the A_o

changes seen during skin heating with the possibility that with the intense exercise, cutaneous vasodilation may have been suppressed or was compensated for by vasoconstriction elsewhere (such as splanchnic organs (76)); and the decrease in A_o during skin cooling being due to sudden removal of this compensatory vasoconstriction (78). These changes in SV, CBV, and CO occurred very rapidly at the early periods of skin temperature elevation before derangements in core temperatures were evident.

Rowell et al (79) examined the reductions in cardiac output, CBV, and SV in thermal stress during exercise in neutral (25.6ºC) and hot (43.3ºC) environments. Subjects exercised at progressively increasing intensities on a treadmill in both conditions. Body mass was maintained by the use of saline infusions and oral hydration. The authors found that cardiac output in the heat was maintained at the lowest two workloads (49 and 58% of VO_{2max}) but fell off (6%) at the higher workloads (67 and 76% VO_{2max}) respectively. This maintenance was due to elevated HR and occurred even in the face of reduced CBV and SV, all of which occurred early on at relatively low rectal temperatures (37.9ºC versus 37.6ºC). Skin temperatures were not reported but would have been expected to be elevated in the hot conditions and would have been a factor in driving peripheral vasodilation and CBV redistribution. Other thermal and cardiovascular variables are shown below in table 13.

Condition	Workload %VO$_{2max}$	Cardiac Output (L/min)	Heart Rate (BPM)	Stroke Volume (ml)	Central Blood Volume (L)	VO$_2$ (ml/kg/min)	Rectal Temperature (°C)
	49%	15.0	136	110	1.21	24.5	37.6
	58%	16.4	151	109	1.24	28.6	38.0
25.6°C	67%	18.1	163	111	1.29	32.1	38.0
	76%	19.8	174	114	1.34	36.4	38.2
	49%	14.8	159 [a]	93 [a]	1.03 [a]	24.8	37.9 [a]
	58%	16.0	177 [a]	91 [a]	1.04 [a]	28.0	38.6 [a]
43.4°C	67%	17.0 [a]	192 [a]	88 [a]	1.09 [a]	32.7	39.1 [a]
	76%	18.6 [a]	195 [a]	95 [a]	1.21 [a]	36.0	39.4 [a]

Table 13: Thermal and cardiovascular variables during exercise in hot (43.4°C) versus

warm (25.6°C) conditions.

Subjects exercised on treadmill and progressively increasing intensities for 15 min each. These were performed in both environmental conditions. Body mass was preserved by saline infusions and oral hydration. [a] indicates significant difference between two thermal conditions ($p<0.05$).

Gonzalez-Alonso et al (80) investigated the role of cutaneous blood flow and elevated

heart rate in the reduction of SV seen during exercise in the heat. Subjects exercised at

72% of VO$_{2max}$ in either hot (35°C) or cold (8°C) conditions at different levels of exercise

induced dehydration. When euhydrated, SV and core temperature (38.2°C) were

equivalent in the hot and cold environment, even with a skin blood flow that was 365%

higher in the hot condition, indicating that skin blood flow is not solely responsible for

reductions in SV during exercise in the heat (80).

Shifts in Metabolism and Efficiency Due to Local Factors

Separate from blood supply or catecholamine induced alterations in metabolism,

there may be a direct temperature effect on skeletal metabolism. This effect is postulated

to be the result of greater enzyme efficiency at elevated muscular temperatures.

Febbraio et al (72) examined the effects of elevated muscular temperature on metabolism. The quadriceps muscle was heated while maintaining euthermic core temperatures and normal catecholamine levels. During 2 min of exercise at 115% VO_{2max} there was an elevation in glycogen consumption (31% increase) and lactate accumulation (25% increase). Pre-exercise muscle temperatures were 37.3 and 35.4°C (thermistor placed 4cm deep into the vastus lateralis) in the heated and normal groups respectively, and at the end of exercise were 37.8 and 37.2°C respectively (significantly different at both timepoints).

Starkie et al (81) also examined the role of muscle temperature by heating and cooling (T_{diff} = 6.9°C pre exercise and 0.4°C post exercise) separate legs during exercise for 20 min at 70% VO_{2max}. The legs were heated and cooled by circulating water through cuffs at either 50-55°C or 0°C respectively both prior to and during exercise. Glycogen consumption was increased (76% increase) in the heated leg with no difference in muscle ATP levels or plasma catecholamines found during exercise. Even with 0°C water circulating around the cooled limb, muscle temperatures at the end of exercise were almost identical in both legs (39°C) along with a core temperature increase from 37 to 38°C. These results indicate that the effects of increased glycogenolysis seem to be a direct effect upon glycolysis and are not secondary to increased energy demands leading to decreased levels of ATP (81).

Ferguson et al (82) examined muscular efficiency and the rate of cross bridge cycling under elevated muscular temperatures. Subjects cycled for 6 min at 85% of VO_{2max} with

either normal (N) or elevated (E) (2.4ºC) quadriceps muscle temperatures; at either 60

rpm or 120 rpm, in order to examine the efficiency of contraction at different speeds and

temperatures. With elevated temperatures, VO_2 consumption was elevated at 60 rpm but

reduced at 120 rpm (+5% and -4% respectively). There was also a relative 5% increase

in efficiency (reduced energy consumption – same amount of work) at 120 rpm with

increased temperatures when comparing N to E conditions. This trend reversed itself at

60 rpm with efficiency being reduced by 5% when heating the muscle. The authors

explained these findings by a shift in the efficiency – velocity curve during heating. They

predicted that at 60 rpm a large portion of the muscular work would be performed by

Type I muscle fibers, which have demonstrated a peak cycling efficiency at 60 rpm. By

heating the muscle, cross bridge cycling is increased (in this case unnecessarily) due to

the increase in temperature dependent ATPase activity. This leads to a reduction in

efficiency as more ATP is being consumed due to the elevated cycling rate, but no extra

work is performed. At 120 rpm, the rate of cross bridge in these same type I fibers would

be on a descending portion of the efficiency-velocity curve. After heating, this curve

would be shifted so that it more optimally matches the elevated cycling rate thereby

increasing efficiencies (82).

These results were supported by the finding of Gray et al (74) who found that during a

single six-sec maximal sprint, elevation of muscle temperature led to no change in the

efficiency of work performed as subjects increased both ATP turnover and power output.

This rpm increase supports the theory that efficiency of cycling at higher rpm's is higher

with elevated temperatures. In effect the subjects may have been self selecting the most

economical pedaling speed to sprint at. The authors also observed that the temperature dependent elevation in power output was significantly correlated with the % of Type IIA muscle fibers (R = 0.82 and 0.85 for max and mean power respectively). It has been demonstrated that type I fibers have a maximum shortening velocity equivalent to approximately 165 rpm, and an optimal shortening velocity of approximately 60 rpm. Therefore during a maximal sprint when rpm's routinely exceed 160 rpm the contribution of type I muscle fibers may be minimal. Type IIA fibers have an optimal shortening velocity around 130-140 rpm and hence would contribute more to maximal sprint outputs. Therefore in this study with the increase in pedal speed and maintenance of cycling efficiency, it appeared that type II fibers increased their efficiency during the high speed contractions when muscle temperatures were elevated (74).

Sargeant et al (73) also found that after heating the quadriceps muscle, peak power was expressed at higher pedaling rates (88, 95, 109, and 125 rpm respectively at muscle temperatures of 29.0, 31.6, 36.6, and 39.3). The authors also found that as rpm increased, the effects of elevated muscle temperatures increased (at 54 rpm, peak power increased by 2% per ºC elevation of muscle temperature, while at 140 rpm, this increase was 10% per ºC).

CENTRAL MECHANISMS OF FATIGUE IN THE HEAT

The fatigue experienced during intermittent exercise in the heat may be secondary to a reduced neural drive, but the mechanisms of central inhibition of muscle contraction

are not well understood. Two possible sites of regulation include an impaired central arousal at the level of the cortex, or an inhibition occurring downstream from the cortex due to some type of signal raising the activation threshold of the motor neurons (83). It has been suggested that there exists a "critical internal" which is approximated by a rectal temperature (T_{re}) of 38.6 to 40.3°C at which exercise will be voluntarily terminated (83). This temperature appears to be slightly elevated (0.7°C) and reached more slowly in more aerobically fit individuals (VO_{2max}>55 ml/kg/min) (84).

Nielsen et al examined the role of decreased brain arousal during exercise in the heat by monitoring alpha (low frequency) and beta (high frequency) brainwaves (85). Subjects cycled at 60% of VO_{2max} under hot (42°C) and cool (19°C) conditions while EEG activity of the brain was recorded. In the hot environment, subjects fatigued after 34.4 min with an esophageal temperature of 39.8°C, a forehead temperature of 37.8°C, and a body mass reduction of 1.7% In the cool environment subjects cycled for an equivalent period of time (did not reach fatigue), exhibited reduced esophageal temperatures (37.8°C), reduced forehead temperatures (35.1°C), and a body mass reduction of 1.0% The ratio of alpha to beta waves was significantly elevated (188% of the value obtained two min after exercise began) at fatigue in the hot condition, whereas the value was not elevated in the cool condition (59% of 2 min value). The increases in the α/β ratio were correlated with the rise in esophageal temperature (R^2 = 0.94-0.98) as well as ratings of perceived exertion, with perceived exertion ratings being higher in the hot condition. This increase in the ratio of alpha to beta waves experienced in the heat is similar to patterns displayed during sleep and may reflect a state of reduced arousal (85).

Hyperthermia has also been demonstrated to affect cerebral blood flow and metabolism during exercise (86). Nybo et al (86) measured cerebral blood flow using the Kety-Schmidt technique during exercise in the heat. Subjects cycled at 170 W in a thermoneutral (20ºC) environment in either shorts and t-shirt (cool) or a plastic waterproof outfit (hot). Global cerebral blood flow and cerebral metabolic consumption of oxygen and glucose was measured at rest, 15 min into exercise, and at the end of exercise (65 min). Core temperatures at 15 min were similar between groups, but at 65 min, core temperature in the hot condition was greater than in the cool (37.9 and 39.5ºC respectively). Cerebral blood flow was similar at 15 min but 18% lower in the hot condition at the end of exercise. This reduced blood flow was associated with a reduced arterial carbon dioxide tension and proportionally larger arterial-venous difference of oxygen and glucose. Moreover, even with the reduced blood flow, the elevated oxygen and glucose extraction demonstrated an elevated cerebral metabolic rate in the heat, and there was no elevation in released lactate from the cortex indicative of minimal metabolic derangement even with the reduced blood flow (86). The authors commented that the reduction in cerebral blood flow seemed to be intimately related to the reduction in arterial carbon dioxide which appeared to be secondary to hyperthermia induced hyperventilation during exercise, but could also be partially attributed to reduced cardiac output also experienced during exercise in the heat (86).

It has been demonstrated that a "pacing" mechanism may exist during exercise in the heat. Tucker et al (87) showed that pacing was reduced during a 20 km cycling trial performed in the heat (35ºC) versus when done in cool (15ºC) conditions. Subjects

completed a 20 km time trial at self-selected pace under both conditions and appeared to modulate power output to maintain an equivalent RPE in both the hot and cool conditions. This was visualized in a reduced integrated electromyographic activity (EMG) to the Vastus Lateralis in the heat when compared to the cool trial at the 10 and 20 km markers, and a reduced power output from 80-100% of the time trial. Average power outputs for the hot and cool trials were 255 and 272 W (P<0.001) respectively. Completion times were 29.6 and 28.8 min (P<0.01) in the hot and cool conditions respectively. These changes were evident without any differences in core temperature, indicating that the body was somehow aware of the elevated thermal challenge in the hot condition and prevented excessive heat buildup by limiting exercise capacity. The authors speculated that it may have been the difference in skin temperatures which led to the anticipation of heat stress and subsequent reduction in power output (4-10°C higher on average in hot condition) (87).

Thomas et al (88) attempted to delineate the different responses to elevated core, skin, and muscle temperatures. Neuromuscular function was evaluated at baseline (BL), after being passively heated to a core temperature (T_c)of 39.5°C (H), at the onset of cooling (C_1), and after being passively cooled back down the T_c of 37.9°C (C_2). Neuromuscular tests included maximal plantar flexor torque and percent voluntary activation (%VA). The %VA was calculated as the torque generated from a maximal voluntary activation divided by the torque of a maximal voluntary effort superimposed with an electrical stimulus to maximally activate the muscle (voluntary torque / voluntary + electrically augmented torque). All measurements were completed in both legs, with

the right leg being kept cool by the use of ice packs, while the left leg was allowed to

track changes in core temperature. Subjects maintained a constant body mass by

ingesting 1.45 L of Gatorade during the test. The thermal indices and neuromuscular test

results are shown below in table 14.

Limb	Skin Temp (°C)				Muscle Temp (°C)				MVC (Nm)				%VA			
	BL	H	C_1	C_2	BL	H	C_1	C_2	BL	H	C_1	C_2	BL	H	C_1	C_2
Right	30.6[c]	37.8[a,c]	32.5[a,b]	32.5[a]	34.5	34.6[c]	34.5	34.5	177.8	165.4[a]	161.3[a]	171.6[b]	97.0	94.0[a]	93.8[a]	95.2[b]
Left	31.5[c]	41.1[a,c]	32.5[a,b]	32.5[a]	34.5	38.7[a,c]	n/a	34.5	177.2	163.3[a]	160.5[a]	170.6[b]	95.8	91.2[a]	92.0[a]	95.2[b]

Table 14: Thermal indices and performance measures during passive heating and cooling

Subjects were passively heated and cooled using a water perfused outfit.

Variables are measured at different timepoints of temperature intervention and core
temperatures: Core temperatures: baseline (BL) = 37.2°C, Hot (H) = 39.5°C, onset of cooling
(C_1) = 39.5, completion of cooling (C_2) = 37.9°C
MVC = maximal isometric plantar flexor torque
%VA = voluntary activation (max voluntary torque / max voluntary + superimposed electrically
evoked torque)
Right calf was kept cool using ice wraps while left calf was allowed to fluctuate with core
temperature
a: different from BL value, b: different from previous value, c: different between R and L leg
($p < 0.05$)

Subjects were not able to maintain MVC or %VA with core temperature elevations

induced by circulating hot water around the entire body. Even with reduced muscle and

skin temperatures seen in the right leg, at elevated core temperatures, MVC and %VA

were reduced similarly. At the onset of cooling, even with dramatic reductions in skin

temperatures, neuromuscular values did not return to baseline until core temperature was

restored to baseline. These results point to the importance of core temperature,

independent of skin and/or muscle temperatures in the reduction of maximal isometric

force and muscle activation (87).

Many studies have demonstrated the negative effects of elevated core temperatures during high intensity intermittent exercise (89-94) these studies are presented in tabular form in appendix A1.

Morris et al (94) had subjects perform a repeated exercise protocol under hot (30ºC 66% Rh) or moderate (20ºC 70% Rh) conditions. The protocol consisted of repeated walking, sprinting, and jogging in the following format:

Part A: [Walk (3x20m) : Sprint (1x15m) : walk (1x3m) : Cruise (3x20m) : Jog (3x20m)] = subset x 11 = 1 set. Three min rest after each set, total of 6 sets. (total exercise time of 90 min)

Followed by **Part B:** which consisted of repeated 60s run : 60s rest at 100% VO_2 max until fatigue.

Walk = 1.54 m/s
Sprint = maximal
Walk = 4s
Cruise = 93% VO_{2max}
Jog = 49% VO_{2max}

Some subjects were unable to complete part A when exercising in the heat, and exercise time to fatigue was shortened in the hot conditions. This decrement occurred even though there was no difference in the levels of dehydration, rating of perceived exertion (RPE), or blood lactates between the environmental conditions when evaluated at similar timepoints. There was however, a significantly elevated rectal temperature in the hot versus the moderate group (39.4ºC vs 38.0ºC) and moreover, the rate of core temperature elevation was correlated with distance completed in the hot condition.

Morris et al (92) repeated the above protocol using intermittent sport athletes (VO_{2max} =

50.8 ml/kg/min) and endurance athletes (VO_{2max} = 56.3 ml/kg/min) to examine the

responses of different types of athletes. Once again, a number of subjects were unable to

complete all of the intervals of part A, and distance to fatigue was reduced in part B.

Sprint times also took significantly longer to complete in the heat and sprint performance

declined in the hot but not moderate conditions. Subjects did demonstrate an elevated

RPE when exercising in the heat as well as elevated heart rates, however no difference in

blood lactates, plasma volume, or body mass changes were evident between the

environmental conditions. Once again the rate of rise in core temperature was correlated

with the distance completed in part B (r=-0.93) The only difference evident between

game athletes and endurance athletes was a higher blood lactate overall in the game

athletes, with no difference in the response to the heat between groups (92).

EXERCISE AND DEHYDRATION

Exercise while dehydrated has been shown to be deleterious to the performance of

intermittent high intensity exercise, muscular endurance, and maximal aerobic power (95,

96). But the same levels of hypohydration have not been shown to effect isometric

muscle strength or single bouts of anaerobic exercise (96-98). There are many postulated

contributing factors to altered exercise performance while dehydrated, some of which

include: a reduced blood flow to the muscle, alterations in muscle metabolism, altered

central nervous system function and derangements in the cardiovascular system. Even

though much is discussed about the negative affects of dehydration on performance,

many of the studies examining hypohydration are confounded by the added effects of hyperthermia. This is most likely due to the fact that hypohydration negatively affects the body's ability to maintain core temperature which results in elevated core temperatures during exercise (80, 99).

CARDIOVASCULAR AND THERMOREGULATORY EFFECTS OF DEHYDRATION

The negative effects of dehydration on cardiovascular function are likely related to the drop in cardiac output associated with a drop in plasma volume. This reduction in plasma volume is likely compounded by the fact that blood is shunted to the skin in conditions requiring heat rejection. In support of this theory, multiple studies have shown that the impact of dehydration on exercise performance is more consistently present when exercising in the heat or at conditions of elevated core temperature (80, 100). This accentuation of effect when combined with hyperthermia is made even more relevant by the fact that hypohydration leads to a reduced ability to maintain stable core temperatures.

Montain et al (101) examined the effects of graded dehydration on hyperthermia and cardiovascular drift during exercise by examining the effects of varying levels of dehydration on exercise performance. During separate visits, subjects were exposed to cycling exercise and water restriction in order to achieve progressive levels of dehydration (4.2%, 3.4%, 2.3%, and 1.1% body mass reduction) over two hours of exercise. At the end of the exercise bout (62-67% VO_{2max}) the authors obtained measures

of esophageal temperature, HR, and stroke volume (SR), all of which were significantly different between trials. The magnitude of dehydration was related to the increase in core temperature ($r = 0.98$), to the increase in HR ($r = 0.99$), and the decline in SV ($r = 0.99$). The elevation in core temperature was also correlated with an increase in P_{osm} ($r = 0.81$-0.98). It appeared as though the fluid intake which kept body mass losses at 1.1% prevented hyperthermia by maintaining skin blood flow which was 21% higher than in the more dehydrated conditions (101). Even with the large variation in body mass reduction, there was no difference in sweat rate among the four trials.

Buono et al (99) examined the effects of dehydration by 5% on core temperature during exercise in hot (33ºC) and temperate (23ºC) environments. The authors found that hypohydration led to elevated core temperatures during exercise in the hot (0.16ºC per % of body mass reduction) and in temperate conditions (0.08ºC per % of body mass reduction). This elevation in core temperature in the heat was secondary to a reduced sweat rate (-211 g/hr and -149 g/hr in hot and temperate respectively) and reduced skin blood flow (-60 ml/min and -15 ml/min in hot and temperate respectively) in the dehydrated condition (99).

Gonzalez-Alonso et al (100) examined the effects of dehydration independent of hyperthermia on cardiovascular function by having subjects exercise in the cold after undergoing a dehydrating protocol. Subjects exercised in the heat for 120 min to induce a 4% body mass reduction, then rested in ambient conditions, and then performed 30 min of exercise in 2ºC conditions. This was followed by a 45 min rest in ambient conditions

during which an IV infusion of a dextran solution was performed to restore BV while

maintaining a reduced body mass of 4% to allow for examination of BV reduction versus

intracellular and interstitial hypohydration effects. Subjects then again exercised at 70%

of VO_{2max} for 30 min in this BV restored state. Multiple thermal and cardiovascular

variables were compared during exercise in these conditions and are shown in table 15.

Variable	Control	Dehydrated	Dehydrated with BV restoration
Esophageal temperature(ºC)	38.1	38.2	38.1
Mean skin temperature (ºC)	20.9	20.4	20.9
% Body mass lost	0.0	4.1[a]	4.1[a]
Blood volume (ml)	5035	4840[a]	5106
Plasma volume (ml)	3035	2884[a]	3124
VO_2 l/min	3.22	3.20	3.22
Cardiac output (l/min)	21.4	20.7	22.1
Heart rate (beats/min)	147	154[a]	153
Stroke volume (ml/beat)	146	136[a]	145
Mean arterial pressure (mmHg)	112	110	112
Perceived exertion	13.1	14.1[a]	14.6[a]
Blood lactate (mmol/l)	2.3	2.5	2.6

Table 15: Cardiovascular and thermoregulatory variables during exercise in control,

dehydrated, or dehydrated with blood volume restored conditions.

Subjects exercised for 30 min at 70% of VO_{2max} in 2ºC conditions in order to keep core
temperature stable.
[a] significantly different than control condition

Dehydration reduced stroke volume by 7% and increased heart rate by 5% without

affecting other cardiovascular variables. These values were restored with restoration of

BV. However, the restoration of BV did not restore the elevated RPE experienced during

exercise in the hypohydrated condition. A logistical issue with this study is that the

lowered skin temperatures may have impacted skin blood flow in that the cutaneous

vasculature would be more vasoconstricted than typical. This may remove some of the

cardiovascular strain seen in typical exercise conditions, but was necessary in order to maintain a reduced core temperature.

McConell et al (102) examined the effects of fluid ingestion during prolonged exercise on thermal, cardiovascular, and physiological responses. Subjects cycled for 2 hours at 69% VO_{2max} while receiving either no fluid replacement (NF), a volume estimated to completely match water losses (FR-100) or 50% of water losses (FR-50). The 2 hour exercise period was followed by a ride to exhaustion at 90% VO_{2max}. Body mass lost was 3.2%, 1.8% and 0.1% in the NF, FR-50, and FR-100 conditions respectively. Ride time to fatigue was reduced from 328 sec in FR-100 to 248 sec and 171 sec in FR-50 and NF conditions, with no differences between the latter two. Heart rates and rectal temperatures were not different between the groups up to the 60 min mark at which point HR's progressively increased to a larger extent in NF and FR-50 with HR increases in NF being greater than those in FR-100 at 80-120 min and HR's in the FR-50 condition being greater than those in the FR-100 condition at 100 and 120 min. Heart rates were also elevated in NF versus FR-50 at the 100 and 120 min mark. Of interest, HR's were greater in NF versus FR-50 even though blood and plasma volumes were no different (15.1% and 15.5% reductions) between the two conditions, indicating that HR is dependent upon more than just these volumes. The difference in HR between these two conditions could have been due to the differences in core and/or skin temperatures which have been shown to independently elevate HR (77, 78).

Core temperatures followed a similar pattern with final core temperatures being greater in the NF vs FR-50 and FR-100 and FR-50 being greater than FR-100 (39.1, 38.8, 38.5ºC

respectively). Plasma arginine vasopressin (AVP) also increased at the 120 min mark with levels in NF being greater than those in FR-100. Plasma renin, aldosterone, and atrial natriuretic peptide (ANP) were elevated after 60 min with no differences between conditions. These findings demonstrated the importance of hydration in curtailing the increase in HR, core temperature, and AVP during exercise (102)

CENTRAL MECHANISMS OF FATIGUE AND DEHYDRATION

Many of the proposed mechanisms of reduced central drive in hyperthermia are also postulated to be possible contributors to reduced central drive in hypohydrated subjects.

Several studies have demonstrated in increase in RPE with dehydration (102-104). Gonzalez-Alonso (100) demonstrated this elevation in RPE during exercise while hypohydrated by 4.1% of body mass. This elevated RPE was not due to elevated blood lactates, which were found to be similar. Of particular interest was the fact that restoration of BV and PV by saline infusion restored the cardiovascular derangements experienced due to the hypohydration but did not restore the elevated RPE (100). As mentioned earlier, often studies which are attempting to examine effects of hyperthermia and/or dehydration are confounded by derangements in both systems. As cited previously, Nielsen et al (85) demonstrated an increased brain α/β ratio during exercise in the heat during which core temperature was elevated to 39.8ºC and body mass was reduced by 1.0% which makes separation of the contributions of hyperthermia and

hypohydration difficult. Nybo et al (86) also reported reduced cerebral blood flow during exercise induced hyperthermia, but did not report changes in body mass which could have been significant.

DEHYDRATION AND MUSCLE METABOLISM

Montain et al (95) examined the effects of hypohydration on skeletal muscle performance by having subjects perform single leg knee extensions to fatigue under euhydrated or hypohydrated (4% body mass reduction) conditions. Subjects underwent a dehydrating protocol which consisted of moderate intensity cycling and treadmill exercise in hot and humid conditions and water restriction to obtain a 4% body mass reduction, if this target was not achieved, sauna exposure was used to reach target body mass reduction. When performing the euhydrated condition, subjects were allowed to drink water ad libitum which prevented body mass reductions. Following this dehydrating protocol, subjects were given a standardized meal and then rested for 3-8 hours before completing the exercise protocol.

To examine muscle performance, subjects performed single leg knee extensions to exhaustion while lying supine in a whole body magnetic resonance (MR) system with a resistance set to elicit fatigue in 4-5 min (average power output 19 W). Endurance time was defined as the time required for power output to fall by 20%. The MR system allowed instantaneous measurement of the ratios of PCr and ATP to inorganic phosphate (P_i) as well as pH. Muscular strength was assessed by having subjects perform a maximal voluntary isometric contraction (MVC) for five sec. This was performed at 100

and 50 sec prior to exercise, at every 30 sec of the first two min of recovery and every

min thereafter through five min of recovery. Both legs were tested in each hydration

condition and the results were treated as independent observations.

Muscle endurance was reduced from 251 to 213 sec in the hypohydrated condition. Four

of ten subjects had reduced endurance times in both legs when hypohydrated, whereas in

three others only one leg was affected (occurred in the second leg tested). Muscle

strengths were not different at rest in the two conditions and hypohydrated subjects

actually displayed an elevated MVC 30 sec after exhaustion; which disappeared at the

sixty sec mark (this increase was not correlated with the reduction in endurance time).

The pH and ratios of ATP and PCr to P_i were not altered at rest or during exercise by

hypohydration indicating that the premature fatigue was not due to the accumulation of

hydrogen ions or inorganic phosphate (95). The authors proposed that a central

mechanism may have been responsible for the premature fatigue as they could not, with

the metabolites measured, demonstrate a peripheral factor that may have been responsible

for the fatigue (95).

Hargreaves et al (105) examined the effects of hypohydration on muscle metabolism by

having subjects exercise at 67% VO_{2max} for 120 min while either allowing or restricting

fluid intake. While cycling in 20-22C ambient conditions, subjects experienced either a

2.9% reduction or 0.2% increase in body mass respectively, with a corresponding

increase in HR (155 versus 144 respectively). Both conditions experienced a reduction in

PV (24% and 14%) and BV (17% and 10%) with greater reductions being present in the

dehydrated condition. These changes were associated with an increase in rectal

temperature, occurring at the 120 min mark of exercise and not prior (38.6 and 38.0ºC respectively). Muscle temperatures were also significantly elevated at this timepoint (39.1 versus 38.6ºC respectively). There were no measured differences in VO_2 consumption, but an elevated respiratory exchange ratio (RER) was evident in the dehydrated condition at the 60 and 120 min mark (2-3% increase).

Muscle metabolites were examined by the use of a muscle biopsy prior to and at the completion of the 120 min of exercise. These biopsies demonstrated no difference in ATP, PCr, or creatine at exercise completion; but did demonstrate elevated lactate (141%) and decreased glycogen concentrations (70%) in the dehydrated versus euhydrated conditions. Plasma lactates were also elevated in the dehydrated condition at the 30 and 120 min timepoint. Plasma epinephrine values were not different between the two conditions at any timepoint, but plasma norepinephrine was elevated in the dehydrated condition at the completion of exercise (12.27 versus 7.13 nmol/l respectively). Possible explanations presented by the authors included increases in muscle temperature and catecholamines secondary to elevated core temperatures, or a better maintenance of muscle blood flow. Although in this study, plasma epinephrine levels did not reach statistically significant values, when coupled with preliminary data which showed increased levels using the same protocol (106) there was an elevated epinephrine level at the end of exercise (120%) in the dehydrated condition (105).

Plasma epinephrine (E) and norepinephrine (NE) have multiple roles in the body. Both catecholamines stimulate adrenergic receptors with α receptors exhibiting a higher affinity for NE and β receptors exhibit a higher affinity for E (2). The α_1 receptors are predominantly found on blood vessels throughout the body and promote vasoconstriction

while α_2 receptors are found on pre-synaptic terminals and serve to moderate the amount of neurotransmitter release. β_1 receptors are found in the heart, β_2 in the bronchial muscle of the lungs, and β_3 receptors are found in adipose cells. All of the β receptors are activated by both NE and E with a greater affinity for E (2). It has previously been demonstrated that exercise in dehydrated conditions can lead to elevated plasma epinephrine levels (106), and during exercise, elevated levels of E have been found to increase glycogenolytic rate by stimulating the conversion of phosphorylase to its active form (107, 108).

INTERMITTENT EXERCISE PERFORMANCE AND DEHYDRATION

While the evidence for reductions in endurance exercise while hypohydrated are numerous (102, 109-111) the evidence for intermittent exercise is not as abundant. The majority of published evidence indicate that dehydration of up to 7% of body mass can be tolerated without a reduction in maximal isometric strength (112).

Griewe et al (97) were not able to replicate the finding of Montain et al (95) when examining MVC of the knee and elbow after dehydration. Subjects were dehydrated by sauna exposure until a body mass reduction of 4% was obtained (water loss was replaced in the control condition). The 4% body mass reduction did not have an impact on maximal isometric knee extension or elbow flexion nor time to fatigue. Watson et al (98) also found no impact of acute diuretic induced dehydration by 2.2% of body mass on single high power events. Following dehydration, subjects performed a vertical jump or

completed a 50, 200, or 400 meter sprint. The 2.2% level of hypohydration had no negative impact on the single bouts with the only noted difference being an elevated blood lactate following the 50 m sprint in the hypohydrated condition (98).

McGregor et al (113) examined the impact of fluid restriction versus drinking during intermittent exercise which led to dehydration levels of 2.4% versus 1.4% respectively. Subjects completed an intermittent exercise protocol shown below while ingesting no fluid or while ingesting 5 ml/kg prior to exercise and 2 ml/kg every 15 min during exercise.

[Walk (3x20m) : Sprint (1x15m) : walk (1x3m) : Cruise (3x20m) : Jog (3x20m)] = subset x 11 = 1 set. Three min rest after each set, total of 6 sets. (total exercise time of 90 min)

$$\text{Walk} = 1.54 \text{ m/s}$$
$$\text{Sprint} = \text{maximal}$$
$$\text{Walk} = 4\text{s}$$
$$\text{Cruise} = 95\% \text{ VO}_{2max}$$
$$\text{Jog} = 55\% \text{ VO}_{2max}$$

They found that mean heart rates (170 versus 164 BPM), perceived exertion (30% higher during last two sets), serum aldosterone, osmolality, and cortisol (all elevated during final two sets) were all higher in the no-fluid trial. There was no difference in blood lactate, glucose, free fatty acids (FFA), glycerol, or insulin. When examining sprint times during the sprint contained within each subset, times increased during the final set in the no-fluid trial. The authors conceded that as they had not measured core or skin temperatures, some of the differences seen could have been due to changes in these variables as well (113).

EXERCISE AND COMBINED HYPERTHERMIA AND DEHYDRATION

Most real world situations involving either hyperthermia and/or dehydration actually display components of both as they are intimately related. Most of the studies which attempt to examine the effects of one have difficulty in controlling for the other. Exercise in the heat typically leads to elevated skin and core temperatures and often leads to some degree of hypohydration which results in an impaired ability to maintain core temperature. Most of the mechanisms for premature fatigue during exercise in hyperthermic and hypohydrated conditions have been discussed. This section will focus on those that are particularly germaine to muscle metabolism and intermittent exercise.

CARDIOVASCULAR EFFECTS OF HYPERTHERMIA AND DEHYDRATION

Gonzalez-Alonso et al (100) examined the effects of dehydration and hyperthermia on endurance athletes during exercise. Subjects exercised in the heat for 100-120 min in 35ºC conditions and either became dehydrated by 4% of body mass or remained euhydrated by drinking fluids. Subjects then exercised at 71% VO_{2max} for 30 min under one of six different conditions which were comprised of two different environmental conditions dictating hyperthermia levels, two levels of hydration, and a euhydrated normothermic control in each condition. In the two hyperthermic conditions, subjects were not allowed to thermally equilibrate after the 100-120 min of exercise and began subsequent exercise in 35ºC with elevated core and skin temperatures. The control condition in this ambient was performed following the euhydrated/hyperthermic

condition by allowing euhydrated subjects a 45 min cool down period in 23ºC ambient prior to repeat exercise in 35ºC. To prevent hyperthermia during exercise, subjects in the normothermic conditions exercised in ambient conditions of 2ºC and therefore had a separate euhydrated control under these conditions. The authors also examined the effect of restoring blood volume on the dehydrated/normothermic condition by a dextran infusion and repeat exercise (discussed above in dehydration section). Cardiovascular and thermal variables for all groups are shown below in table 16.

Variable	35ºC Control	Hyperthermic Euhydrated	Hyperthermic Dehydrated	2ºC Control	Euthermic Dehydrated	Euthermic Dehydrated with BV restoration
Esophageal temperature (ºC)	38.3	39.3 [a]	39.3 [a]	38.1	38.2	38.1
Mean skin temperature (ºC)	34.0	34.6	34.6	20.9	20.4	20.9
% Body mass lost	0.0	0.1	4.4 [a]	0.0	4.1 [a]	4.1 [a]
Blood volume (ml)	4902	4858	4689 [a]	5035	4840 [a]	5106
Plasma volume (ml)	2946	2913	2756 [a]	3035	2884 [a]	3124
VO_2 l/min	3.15	3.16	3.14	3.22	3.20	3.22
Cardiac output (l/min)	21.1	20.4	18.4 [a,b]	21.4	20.7	22.1
Heart rate (beats/min)	164	172 [a]	178 [a,b]	147	154 [a]	153
Stroke volume (ml/beat)	130	119 [a]	104 [a,b]	146	136 [a]	145
Mean arterial pressure (mmHg)	101	99	96 [a,b]	112	110	112
Perceived exertion	14.7	17.0 [a]	17.6 [a]	13.1	14.1 [a]	14.6 [a]
Blood lactate (mmol/l)	2.9	2.9	3.0	2.3	2.5	2.6

Table 16: Cardiovascular and thermoregulatory variables during exercise in control, dehydrated, or dehydrated with blood volume restored conditions.

Following hypohydration inducing exercise at 35ºC during which subjects either became dehydrated by 4% or maintained euhydration, subjects then exercised for 30 min at 70% of VO_{2max} in either 35ºC or 2ºC conditions in order induce or prevent hyperthermia. [a] significantly different than respective control condition [b] significantly different than hyperthermia alone

The authors found that hyperthermia and dehydration each separately lowered SV 7-8%

and increased HR enough to prevent significant declines in cardiac output. However,

when dehydration was superimposed on hyperthermic conditions, the reductions in SV

became even greater (20%) and subjects were not able to maintain cardiac output which

fell by 13%. Moreover, mean arterial pressure, which was maintained under dehydrated

conditions when normothermic with cool skin temperatures was not maintained when

dehydrated and hyperthermic (100). A limitation of this study is that the lowered skin

temperatures used to prevent hyperthermia may have impacted skin blood flow by

inducing cutaneous vasoconstriction. This may have removed some of the cardiovascular

strain seen in typical hypohydrated exercise conditions. In support of this contention, it is

of interest to note that although no statistical comparisons were made, there was an

apparent reduction in SV and increase in HR between the two control conditions where

the only noticeable difference were elevated skin temperatures.

To further investigate the effects of hyperthermia and hydration on stroke volume,

Gonzalez-Alonso (80) examined euhydrated/dehydrated subjects in both a hot (35ºC) and

cold (8ºC) environment at varying levels of dehydration. They found that when

euhydrated, subjects maintained equivalent core temperatures in both environments, but

as levels of dehydration increased, core temperatures diverged with core temperatures

being greater in dehydrated conditions. They also found that SV was maintained while

euhydrated even with large variations in skin blood flow, but with each 1% of body mass

reduction, SV declined by 5% in the heat and by 2.5% in the cold. Moreover, these

reductions were not associated with increased skin blood flow but rather were highly

associated with increased heart rate and reduced blood volume ($r = 0.96$ for both). This increased HR would reduce filling time in the left ventricle and the reduced blood volume would reduce filling pressure, both contributing to a reduced left ventricle end diastolic volume and reduced SV. The increase in HR was most likely driven by elevations in NE which were measured at all hydration levels in the heat and at 3 and 4.2% dehydration levels in the cold. This elevation in NE could also drive greater contractility and enhance ejection fraction in the heart which would offset some of the reduced SV.

The results of these studies point to a multi-factorial cause of reduced SV with exercise during hot and dehydrated conditions. It is likely that reduced BV and left ventricle filling pressure due to dehydration, a reduction in CBV due to cutaneous pooling, and reduced filling time due to elevated HR all play a role in the reduction in SV during exercise under heat stress conditions.

MUSCLE BLOOD FLOW WITH HYPERTHERMIA AND DEHYDRATION

The possibility of reduced muscular blood flow during exercise and heat stress has met with mixed results (114-118). A possible explanation for this disagreement could lie in the need for a systemic blood flow disturbance such as reduced CO or mean arterial pressure to be present in order for muscle blood flow derangements to be present (117).

Gonzalez et al (117) examined muscular blood flow during dehydration and hyperthermia in subjects exercising in the heat under either euhydrated or dehydrated (3.9% body mass

reduction) conditions. When dehydrated, subjects became hyperthermic to a core temperature of 39.7ºC while euhydrated, core temperature was maintained at 38.2ºC. During the dehydration protocol (DE), subject's fluid intake was restricted and they experienced progressive dehydration as they cycled to failure. The euhydration (EU) protocol involved matching fluid losses with oral and IV hydration and ended at the same timepoint as volitional failure in the dehydrated condition. By following this protocol, hydration levels became significant only after the first hour of exercise.

During the final 20 min (120 min mark) in DE; CO, leg blood flow (LBF), mean arterial pressure, and systemic vascular conductance declined significantly (15%, 13%, 5.5%, 8% respectively) compared to EU, without any changes in muscle vascular conductance. Arterial catecholamines were also elevated in DE versus EU, with NE demonstrating a much more rapid increase (50 min mark) than did epinephrine (120 min mark). This was explained by the authors as most likely being secondary to NE spillover from the non-active leg vasculature which was presumably undergoing vasoconstriction to preferentially shift blood flow to the active muscles (117). Interestingly, the reductions in LBF in DE occurred after a reduction in CO was already evident (90 min mark), but at the same time (120 min mark) as the fall in mean arterial pressure. At the completion of DE, the reduction in leg blood flow accounted for 2/3 of the reduced CO while at least a portion of the remaining decline in CO was accounted for by a 39% reduction in skin blood flow. Whole body VO_2 and leg oxygen extraction increased in parallel in both conditions, with leg VO_2 being maintained by an increased oxygen extraction in the DE condition. During the final min of exercise in DE, leg oxygen delivery fell below levels

of EU, and even though no statistical significance was obtained, it appeared as though leg VO_2 consumption was falling as well. Levels of potassium and lactate also increased in DE vs EU conditions which could contribute to early fatigue during exercise under thermal strain (117).

These results support the notion that in hyperthermia and dehydration, leg blood flow is reduced secondary to a reduced mean arterial pressure and systemic blood flow. It also appeared that oxygen delivery to the muscle is maintained, albeit less robustly so, by an increased oxygen extraction.

LOCAL METABOLIC CHANGES EXPERIENCED WITH DEHYDRATION AND HYPERTHERMIA

The reduction of blood flow to exercising muscles brings forth the question of whether or not this impairment leads to a shift in metabolic pathways in the muscle which could have an impact on fatigue.

Gonzalez-Alonso et al (119) examined this possibility using the same protocol utilized to examine reduced leg blood flows (117). They measured femoral arterial and venous blood samples to determine leg exchange of glucose, lactate, glycerol, free fatty acids (FFA), and convective heat exchange, and obtained muscle biopsies to determine glycogen and lactate content. They found that DE led to a reduced uptake of FFA, higher muscle glycogen usage, muscle lactate accumulation, and net lactate release than in EU.

As in the prior experiment, many of these changes did not appear until later in exercise (100-120 min) and were apparently the result of dehydration/hyperthermia or both. As core temperatures were significantly elevated in DE (39.7ºC versus 38.1ºC) separating the effects of hyperthermia from dehydration was impossible. Possible mechanisms for the elevated glycogen consumption included an increased muscle temperature, or elevated circulating epinephrine, both of which were demonstrated in DE. Muscle temperature tracked core temperature very closely with peak temperatures reaching 40.4ºC and 39.7ºC respectively in DE and 38.1ºC and 38.65ºC in EU.

Subjects rated their perceived exertion as more intense (19.4 versus 13.6) in DE versus EU. Even with the metabolic shifts, it did not appear as though fatigue was due to low muscle glycogen or critically high muscle metabolites, but rather appeared to be associated with an elevated core temperature (39.7ºC). When comparing data from this study with that of an earlier study which examined fatigue and core temperatures in the same subjects while euhydrated (120) the authors found the core temperature at fatigue was reduced by 0.5ºC in the dehydrated condition, implying perhaps that dehydration impairs the ability to tolerate higher core temperatures (119).

INTERMITTENT EXERCISE PERFORMANCE WHILE DEHYDRATED AND HYPERTHERMIC

Drust et al (121) examined the effects of elevated muscle and core temperature and mild dehydration in repeated sprint performance by having subjects perform a series of work in either hot (40ºC/17% Rh) or neutral (20ºC/23.6%Rh) conditions. Subjects

completed 40 min of intermittent cycling (15 sec sprint : 15 sec rest) at work intensities

which when averaged over the entire work/rest period equaled 60% VO_{2max}. This was

followed by a series of five 15 sec sprints for maximal power interspersed with 15 sec of

rest between each. Data was collected after the completion of the 40 min of work as well

as following the series of five maximal sprints; these results are shown in table 17.

Variable	Hot Condition	Neutral Condition
T_m (ºC)	40.2 [a]	38.9
T_c (ºC)	39.5 [a]	38.2
% BM	1.2 [a]	0.7
HR (bpm)	178 [a]	143
RPE	18 [a]	12
Grip Strength (N)	474 [a]	515
BLa (mmol/l)	5.6	2.8
NE (µmol/l)	38.9 [a]	27.0
Epi (µmol/l)	6.1	5.8
Muscle La (mmol/kg DM)	23.2	16.8 [b]

Table 17: Comparison of physiological variables following 15s sprint : 15s rest x 40 min

when performed in hot or neutral conditions.

T_m: muscle temperature, T_c: core temperature, %BM: % body mass reduction, BLa: blood lactate,
NE: norepinephrine, Epi: epinephrine, Muscle La: accumulated muscle lactate.
[a] significantly different than Neutral condition [b] trend towards difference between conditions
(p=0.06)

After completion of this repeated sprint/rest protocol, subjects performed the repeated

maximal sprint protocol. Peak power was maintained whereas mean power was reduced

in the hot versus neutral condition (10%) with this decrease in mean power coming in

sprints 2-5 (mean power was preserved in sprint 1). Subjects in the hot conditions also

consumed less oxygen during the repeat maximal sprints (7789 ml versus 8695 ml) but

consumed the same amount of glycogen and generated equivalent amounts of lactate.

The authors attributed the fatigue in the hot condition to elevated core and muscle

temperatures as it did not seem as though any metabolic byproducts were altered between groups, although the glycogen consumption between conditions exhibited a trend towards significance (p=0.06). This central mechanism also seemed to be supported by a reduced central drive as measured by the reduced handgrip force generated following the 40 min protocol, however this did not correlate well with the maintenance of peak power in the maximal sprints. Even though levels of dehydration were relatively small, they were significantly different making the dehydration contribution difficult to estimate.

Morris et al (122) examined the effects of intermittent high intensity running in hot (33°C/28% Rh) or moderate (17°C/63% Rh) environments. Subjects completed an intermittent running protocol shown below. Subjects first performed this protocol in the heat to failure, then completed the same amount of work in neutral conditions to allow comparison, and finally exercised to failure in moderate conditions.

[Walk (3x20m) : Sprint (1x15m) : walk (1x3m) : Cruise (3x20m) : Jog (3x20m)] = subset x 11 = 1 set. Three min rest after each set, total of 6 sets. (total exercise time of 90 min)

Walk = 1.54 m/s
Sprint = maximal
Walk = 4s
Cruise = 85% VO_{2max}
Jog = 45% VO_{2max}

The distance covered in the hot condition was significantly less than that covered in the moderate condition (11,216m versus 21,644m). When looking at sprint times as they changed from baseline (subjects sprinted faster initially in the heat than in neutral

conditions) it was apparent that sprint times fell off in the heat more than in the moderate

conditions. Other physiological variables are shown in table 18.

Variable	Hot [failure]	Moderate [same timepoint as failure in the heat]	Moderate [failure]
T_m (°C)	40.2	39.3 [a]	39.3 [a]
T_c (°C)	39.6	38.75 [a]	38.85 [a]
RPE	19	14 [a]	19
BLa (mmol/l)	5	2.7 [a]	2.8 [a]
Muscle La (mmol/kg DM)	7.2	3.9 [a]	6.0
NE (µmol/l)	21.5	9.0 [a]	15
Epi (µmol/l)	1.95	1.0	6.3

Table 18: Comparison of physiological variables following repeated running protocol

performed in hot or neutral conditions.

T_m: muscle temperature, T_c: core temperature, BLa: blood lactate, NE: norepinephrine, Epi:
epinephrine, Muscle La: accumulated muscle lactate.
[a] significantly different than failure in heat

The authors also measured peak knee extensor and flexor torques which were unchanged

in the heat, suggesting that isolated muscle function was not compromised. Body mass

reduction was equivalent in both conditions (2%) as water intake was nearly double in the

heat. Accompanying the increases in blood and muscle lactate was a non-significant

trend (p=0.055) in increased glycogen use (34%) in the heat. These results corroborate

previous findings that glycogen utilization can be elevated during exercise in the heat

(106, 123, 124) although not all studies have demonstrated such results (125-128). The

authors suggested that such varying results may be due to the acclimation status or the

degree of thermal strain put on subjects (122). Results from other intermittent exercise

protocols performed in the heat under thermal strain are shown in appendix B.

<u>PSYCHOLOGICAL IMPACT OF EXERCISE, DEHYDRATION, AND</u>

<u>HYPERTHERMIA</u>

RATINGS OF PERCEIVED EXERTION AND ENVIRONMENTAL INTERACTIONS

The Borg perceived exertion scale (RPE) is widely used in exercise science to monitor levels of exercise intensity. Borg originally created the ratings based on a correlation of heart rates during exercise with every increase of 10 bpm representing a unitary increase in the rating (129). But the integration of perceived exertion takes into account many more factors than just heart rate, one such modifier has been show to thermal stress (130-132). Maw et al (130) investigated the role of hot and cool environments on ratings of perceived exertion. Subjects completed 30 min of constant intensity (determined individually by eliciting a "somewhat hard" rating of RPE in neutral environment) exercise in hot (40ºC), neutral (24ºC), or cool (8ºC) conditions. Several thermal and exercise variables were measured during each exercise and are shown in table 19.

Variable	Hot (40ºC)	Neutral (24ºC)	Cool (8ºC)
T_{sk} (ºC)	37.2 [a]	33.3	27.9
T_{re} (ºC)	38.1	37.9	37.9
HR (bpm)	163.5 [a]	139.5 [b]	135.1
Affect	0.7 [a]	2.3	2.9
Thermal Sensation	6.9 [a]	6.0 [b]	4.0
RPE	12.9 [a]	12.6	11.7

Table 19: Comparison of thermal and psychological variables during exercise in hot,

neutral, or cool environments.

Subjects exercised for 30 min at equivalent work intensities in each environment
T_{sk} = mean skin temperatures, T_{re} = rectal temperature, HR = heart rate, Affect = 11 point bipolar
scale (-5 very bad - +5 very good, Thermal Sensation = 7 point scale (1 cold – 7 hot), RPE =
rating of perceived exertion (6 no exertion – 20 maximal all out effort)
[a] significantly different than neutral and cool, [b] significantly different than cool

The authors found that RPE was significantly elevated in the heat even though core

temperatures were not different. They attributed this to a combination of elevated heart

rates, increased vasodilation, increased thermal sensation, and decreased affect during

exercise in the heat (130). It has been demonstrated that skin temperatures contribute an

equivalent or greater amount than core temperature to thermal comfort (133, 134) and

thermal discomfort has been associated with cutaneous vasodilation (135). Blood lactate

and ventilation rates have also been shown to correlate the RPE values, particularly at

high intensities (136). Not all studies have demonstrated this influence of skin

temperatures on RPE however, Glass et al (137) found no differences in RPE when

cycling at 80% VO_{2max} in high, moderate, or low wet bulb temperatures.

The correlations with ratings of exertion and blood lactates led Borg et al to develop a

new scale which would allow for the non-linear increase in lactate experienced during

intense exercise (138). This scale consisted of a ranking from 0 to maximum with 10

being almost maximal. This scale was also implemented to evaluate exertion in three

different areas; a rating of leg effort, a rating of cardiorespiratory effort, and leg pain

(138). These ratings were found to correlate well with both muscle and blood lactate

accumulation found during progressive maximal exercise.

Further supporting the role of lactate in RPE values, Swank et al (139) demonstrated that

RPE values were reduced during intermittent high intensity exercise following ingestion

of sodium bicarbonate which increased the pH of the blood. Subjects exercised for three

five min bouts at 90% VO_{2max} separated by 10 min rest intervals. These tests were

repeated after ingestion of bicarbonate which elevated both resting and exercise blood pH

values. Subjects rated their exertion using three 0-10 scales, rating legs, chest, and

overall body fatigue. The authors found that all three RPE values were negatively

correlated with blood bicarbonate concentration (139).

EXERCISE AND ENVIRONMENTAL EFFECTS ON REACTION TIME

Exercise has been shown to have an inverted U effect on the performance of a

cognitive task (140) with increased levels of arousal being linked to an increase in heart

rate and/or perceived exertion. During aerobic exercise, increased arousal has been

described by measuring increased levels of β activity and decreased levels of α activity.

Some of these changes have been linked with catecholamine levels which increase during exercise.

It is difficult to delineate the effects of exercise and environmental stress on reaction time as many of the experiments have yielded conflicting results (141). Some of the variability in results can be attributed to whether or not the perceptual task is being completed during or after the exercise bout. Lemmink et al (142) found no effect of prior intermittent exercise on multiple choice reaction time performance in soccer players; while Davrance et al (143) found that subjects reaction time was decreased when completing the task during moderate exercise. Some results have demonstrated an improvement in the speed of calculations with an increase in core temperature while others have found a reduction (140). As in studies evaluating performance, many of the thermal studies have by their nature, the confounding effects of dehydration. Dehydration, independent of hyperthermia has been shown to result in depressed cognitive performances (140). Cian et al (144) demonstrated that dehydration impaired cognitive abilities such as perceptive discrimination, and short term memory, but did not impair reaction time.

THE PHYSIOLOGY OF ICE HOCKEY

Ice hockey is a complex game involving substantial contributions from all of the energy systems. It also involves a great deal of cognitive processing ranging from the reading of game situations to quick decisions on where to move or pass the puck. All of

these occur while skating and being constantly aware of the positions of all other players on the ice.

TIME MOTION DATA

Green et al (145, 146) performed time-motion analysis of Canadian varsity hockey players during actual game situations. Data from these two studies are presented in Table 20.

Criteria	Green et al (146)		Green et al (145)		
	Forwards n=3	*Defense n=3*	*Centers n=2*	*Wings n=5*	*Defense n=3*
Actual playing time (s)	1152 +/- 54	1723 +/- 97	1247 +/- 93	1403 +/- 283	1682 +/- 261
Shifts	20.2 +/- 0.6	24.3 +/- 0.7	14.5 +/- 2.1	16.0 +/- 2.2	20.7 +/- 2.3
Play time per shift (s)	57.9 +/- 2.5	73.1 +/- 4.7	87.6 +/- 19.4	88.1 +/- 10.6	81.4 +/- 5.2
Play stops per shift	2.0 +/- 0.1	2.6 +/- 0.2	2.3 +/- 0.7	2.2 +/- 0.2	2.3 +/- 0.4
Play time between stops (s)	29.5 +/- 0.8	28.5 +/- 0.3	42.5 +/- 2.7	41.0 +/- 4.4	37.5 +/- 4.4
Time for play stop (s)	29.1 +/- 3.3	30.5 +/- 4.1	25.7 +/- 6.5	25.8 +/- 3.0	28.7 +/- 7.7
Recovery time between shifts (s)	293 +/- 16	189 +/- 18	291 +/- 24	248 +/- 93	159 +/-39

Table 20: Time motion analysis of Canadian varsity hockey players.

Data taken during actual ice hockey games.

It can be seen from this data that hockey is very intermittent in nature and is subject to many stops and starts during the course of a game. Green measured the average skating velocity during the game and found it to be 227 m/min (145). Bracko et al (147) examined the skating characteristics of professional hockey players and found that 56.2%

of on ice time was spent in some form of skating or struggling for position, while 43.8%

of the time was spent gliding or standing.

METABOLIC DEMANDS OF ICE HOCKEY

Since ice hockey is an extremely variable sport, on ice heart rates are extremely variable

depending upon what level of activity has just preceded measurement. Paterson (148),

Green (145), and Spiering et al (149) have all reported heart rates during ice hockey

games, which are presented in table 21.

Criteria	Competitive League Paterson et al (148)	House League Paterson et al (148)	Spiering et al (149)	Green et al (145)
Peak on ice heart rate (bpm / % max)	190.7 / 96.5	198.1 / 99.9	-	-
Mean on ice heart rate (bpm / % max)	182.4 / 92.3	187.7 / 94.7	- / 90.0	173 / 89.0
Mean off ice heart rate (bpm / % max)	135.3 / 68.4	141.0 / 71.1	- / 59.0	125 / 64.4

Table 21: Comparison of heart rates during actual hockey games.

Subjects in Paterson et al were adolescent, Green et al college age males and in Spiering et al adult women hockey players. Off ice heart rates implies time spent sitting on bench between shifts.

Peak on ice heart rates have been estimated to be in excess of 90% of maximum with

average heart rates approximating 85% of maximum (150). The average on ice intensity

is estimated at 70-90% of VO_{2max} (151).

Muscle biopsy studies have been performed to determine the fuel usage during a typical

hockey game. Green et al (146) have demonstrated glycogen reduction in all fiber types

with the greatest reduction occurring in type I muscle fibers, but also found a widely

divergent pattern between players of the same position indicative of the highly variable nature of ice hockey (146). The authors also recorded post period blood lactates of 6.16, 4.65, and 5.63 mmol/L in forwards following the first, second, and third periods respectively (blood samples taken 4-6 min following last shift). The authors also found significant elevations in free fatty acids and glucose over rest. Other reported blood lactates have ranged from as high as 11 mmol/L to as low as 2.9 mmol/L again, representative of the highly variable nature of hockey (151).

Maximal oxygen consumption as measured by cycle ergometry also demonstrates a wide range of values. When expressed as relative to body mass, values range from 44.1 ml/kg/min to 62.4 ml/kg/min with an average of 53.9 ml/kg/min (151). Peak anaerobic power outputs in forwards, as measured by a 45 sec maximal cycle sprint were found to be 12.2 W/kg with a standard deviation of 1.02 W/kg (151).

THERMOREGULATION IN ICE HOCKEY

Published ambient temperatures in ice hockey range from 4 to 15ºC with a range of relative humidity from 50-71% (152-154). Even with the cool temperatures, hockey players experience a thermal challenge based on the high intensity and intermittent nature of their sport along with the restriction to convective and evaporative cooling provided by the layers of protective padding they wear. The consequence of this thermal challenge and ensuing levels of sweat loss are significant levels of voluntary dehydration which are reported to average 3% (146) with self reports ranging as high as 10% of body mass lost

(155).   These levels of dehydration could impose significant reductions in work capacity as discussed in prior sections. Beyond the physiological impact, the high rate of water loss could add a significant mass to the hockey protective equipment as much of it appears to be absorbed. Montgomery et al (150) investigated the role of added mass on skating performance using a repeated skating task. They found that 5% added mass caused a reduction in sprint capacity by 4%. Leger et al (154) examined the effects of the extra mass of wearing hockey protective equipment on skating VO_2 (pad weight = 7.3 kg) and found a reduction of 4.8% in mechanical efficiency ratios when wearing the pads. There have been no actual studies of the thermoregulatory implications of hockey protective equipment, but MacDougall wrote about the theoretical implications wearing such gear (156). The author made several suggestions to improve cooling which included the removal of the helmet and gloves between shifts, and encouraging frequent ad libitum water ingestion (156).

METHODS

OVERVIEW OF DESIGN

The study was designed to examine changes in physical, cognitive, and physiological markers of performance and fatigue during and after a simulated hockey game performed on a cycle ergometer. Two test conditions (order was randomized) were examined one week apart. The independent variable was exercise attire. In one condition, subjects wore full hockey protective equipment to simulate hockey conditions plus cotton undergarments (P), while in the other condition subjects wore only the undergarments (NP). Room temperature and relative humidity was kept constant in both testing conditions (12°C dry bulb temp and 10.5°C wet bulb temperature).

SUBJECTS

Subjects included 8 recreationally active males with a mean age of 26.8 (range 22-34), average body mass of 75.0 (62.6-83.1) kg, and an average maximal oxygen uptake of 55.3 (45.9-61.4) ml/kg/min. A minimum maximal oxygen consumption of 45 ml/kg/min was established, to match the lower end of the published range for elite hockey players. All participants were fully informed of experimental procedures and possible discomforts associated with the study before giving their written consent to participate. The study was approved by the Human Investigational Committee of the Yale University School of Medicine.

Before the two experimental trials, subjects reported to the laboratory for two

visits, during which physiological information was be obtained and subjects were allowed

to practice the various tests of physical and mental function to eliminate potential

learning affects during the experimental sessions. The two visits consisted of the

following test items:

Visit	SRT/CRT	5 x 6 Test	VO$_2$ max Test
1	x	x	X
2	x	x	

Table 22: Tests performed during familiarization visits

SRT: simple reaction time, CRT: choice reaction time

Subjects were required to abstain from strenuous exercise for 24 hours prior to

testing days. Subjects were also required to refrain from ingesting alcohol or caffeine for

12 hours prior to testing.

Cognitive Testing

Cognitive testing consisted of a simple reaction time (SRT) test and a three choice

reaction time test (CRT). The SRT consisted of the subject pressing a mouse key as

quickly as possible after a 2" by 2" white box appeared on a computer screen placed three

feet directly in front the subject. The program provided 30 stimuli, each with a 1 to 2

second delay (order was randomized) and measured their response time. The CRT

program presented one of three color boxes (red, yellow, or green) and required subjects

to press the right mouse key for a green box, the left mouse key for a yellow box, and

make no response for a red box. The color remained on the screen for 1 sec, followed by

a random 1 to 2 sec and then the next box (30 in total, 10 of each color). The program

again recorded reaction times and number of incorrect responses.

Cycle Ergometer Testing

Maximal oxygen consumption was measured from continuous readings of the

fractions of expired O_2 and CO_2 via electronic analyzers. The electronic analyzers were

calibrated against standard gases and the flow meter was calibrated with a calibrated

syringe. Values were appropriately corrected to STPD (standard temperature and

pressure, dry air) or BTPS (body temperature and pressure, saturated air). Maximal

oxygen consumption was determined by monitoring oxygen uptake during an incremental

exercise test using a cycle ergometer (Monark 839E). The protocol began with a three-

min warm-up at 50 Watts, at which time the power output ramped up at a linear

progression of 30 W per min. Criteria for maximal oxygen consumption include a

plateau of oxygen consumption in concert with a reduction in pedaling frequency, and an

RER (respiratory exchange ratio) > 1.10. Completion of the graded exercise test

typically required between 10 and 15 min.

Repeated sprint ability was also determined (5 x 6) twice; 10 min prior to the start of the first simulated shift, and 250 sec following the final shift of period 3. This test consisted of five 6-sec maximal sprints performed every 30 sec and was performed on a separate cycle ergometer (Monark 894E). Subjects performed a standard warm-up of sub-maximal cycling at 80 rpm for 5 min with a resistance of 1.5 kg, with a five sec sprint at min 3 and 4. This was followed by 3 min of pedaling at 80 rpm against 1 kg resistance. Upon completion of the warm-up, subjects had a 3-min cool down period. The ergometer resistance was then set at 7.5% of body mass and subjects accelerated the unloaded flywheel to 80 rpm at which time the full resistance was applied instantaneously. Subjects then accelerated to a maximal velocity for 6 sec at which time the resistance was removed and they were allowed to pedal at 80 rpm against no resistance for 24 sec. This sequence was repeated for a total of five sprints. Power was calculated at 1-sec intervals throughout each sprint, with corrections being made to account for inertial properties of the flywheel (157). Peak power (PPO) and mean power (MPO) (W/kg) were calculated for each set of sprints with MPO representing the average power over all five sprints and PPO representing the highest one sec power over all five sprints.

EXPERIMENTAL SESSIONS

Subjects were instructed to maintain normal activity patterns and to attempt to replicate their food intake for 1d before the first experimental session. Exercise, alcohol, and caffeine instructions were the same as in the familiarization tests. Subjects were reminded of these guidelines before each experimental session. In addition, the day of

the week and time of day were kept constant to minimize any possible affects these

variables may have had on performance. The entire protocol is shown in both graphical

and tabular form in appendix C and D respectively.

Subjects were given 500 ml of water to drink the night before and 500 ml to drink 90 min

before arrival to ensure adequate hydration. Subjects emptied their bladders upon arrival

on the morning of the visit and urine specific gravity (SG) was measured to ensure

adequate hydration (SG<1.020). If this requirement was not met, subjects ingested

another 1L of water over a 30 min time period with urinary SG verification before the

trial was allowed to continue. An indwelling catheter was then inserted in an arm vein,

and subjects then changed into standardized, pre-weighed undergarments (cotton

underwear, shorts, t-shirt, and socks, and were weighed on a digital scale (accuracy +/- 10

g). Subjects then rested in a seated position for 45 min to allow for plasma volume

stabilization. During this rest period, skin surface and esophageal temperature

thermocouples were attached and resting measurements were recorded. After the 45-min

period, a resting blood sample was taken. Subjects then donned the protective equipment

if the trial dictated, collected urine, and then entered the environmental chamber which

was maintained at 12ºC dry bulb and 10.5ºC wet bulb for the entire study. Subjects then

completed a pre-trial SRT/CRT test and began the warm-up for the initial 5x6 test. After

completion of the rested 5x6 test, a second blood sample was taken, followed by a 10 min

cool-down period after which the simulated game was started.

Simulated Game Protocol (See appendix C): The simulated game consisted of three periods with six shifts per period. Each period was followed by a 15 min period break, with the last period break extended to 45 min to allow for the final resting blood sample to be taken. The post-game fatigued 5x 6 test was performed four min and 10 sec following shift six of the third period in order to simulate a standard recovery time. Each period consisted of 6 standardized shifts with each shift composed of alternating periods of sprinting and coasting and was performed on a Monark 894E which was modified to allow for instantaneous changing of pedaling resistance. A fan was used simulate whole body wind due to skating and was calibrated based on published average skating speeds of 227 m/min (145). This fan was activated any time the subject was performing work during the simulated shifts. Blood samples were taken at the end of the sixth shift of each period, following period breaks one and two, following the fatigued 5x6 test, and after 45 min of seated rest at the end of the protocol. Urine was collected during each period break and at the end of the protocol.

Shifts: Each shift consisted of three 25-sec work intervals separated by two 25-sec passive seated rest intervals. Each shift was followed by a 250 sec passive seated rest interval to simulate two other lines skating (shown in appendix C).

Work interval within a shift: Each 25-sec work interval consisted of five alternating 5-sec periods of varying work intensity (high:low:high:low:high). High intensity power output was defined as the power output that would be predicted to elicit 155% of the subjects measured maximal oxygen consumption based on a linear regression equation

generated during the graded exercise test with oxygen consumption as the independent and power output as the dependent variables respectively. Low intensity power output was defined as 50% of the power output at maximal oxygen consumption. All work done during the simulated game was performed at 100 rpm. Subjects started each work interval with the flywheel at target rpm before the load was applied. Total power output for the entire simulated game was kept constant between the two conditions.

SRT / CRT / RPE / HR: The SRT and CRT were performed at rest, immediately following the sixth shift of period 1 and 2, and following the fatigued 5x6 test at the end of period 3. Rating of Perceived Exertion (RPE) on a three variable (breathing effort, leg effort, overall body fatigue) Borg scale (0-10) were recorded immediately following shift 6 for each period (129, 138, 139). Subjects were provided with a visual analog scale for the RPE test which was used to compare subject's perception of work intensities between test conditions. Heart rate was recorded via a Polar 810i heart rate monitor in five-sec intervals for the entire simulated game and was expressed as a percent of maximal heart rate established during the maximal oxygen consumption tests. Heart rate was expressed for both working and resting intervals. Work intervals included the 125 seconds of working and rest during a simulated shift, while rest intervals included only the 250 seconds between shifts.

Blood and Urine Analysis: All blood sampling was done via an 18-gauge catheter placed in an arm vein. Sampling was done from free-flowing blood and the catheter was filled with heparinized saline (20 units/ml) after each sample. Blood samples were taken at the

times shown in appendix C. Blood samples were separated into aliquots. One aliquot

was immediately analyzed for microhematocrit and hemoglobin by cyanomethemoglobin

which was then used to calculate changes in plasma volume based on the methods of

Gillen (19).

The remaining blood was placed into tubes containing EDTA, the tubes were centrifuged

at -4ºC and plasma was taken off. A 500 μL plasma sample was used for the analysis of

blood lactate and glucose in triplicate using a YSI 2300 lactate analyzer (Yellow Spring

Instruments, Yellow Spring OH). A one mL sample was analyzed for P_{osm} by freezing

point depression (model 3DII, Advanced Instruments). The remaining plasma was frozen

at $-70°$ C for future analysis of catecholamines and arginine vasopressin (AVP).

Catecholamines were analyzed using high performance liquid chromatography (HPLC

with electrochemical detection, Colorchem detector ESA Corp. Acton MA) performed by

the research laboratory in the General Clinical Research Center at Yale New Haven

Hospital. Arginine vasopressin was analyzed using the methods described by Freund and

colleagues (158, 159) on octadecylsilane cartridges (SEP-PAK C_{18}; Waters Associates)

(this assay was performed by a lab technician at the John B. Pierce Laboratory).

Extracted samples were assayed using a disequilibrium assay with the extracts incubated

with the anti-serum at 4ºC for 72 hours, followed by the addition of ^{125}I labeled AVP

(New England Nuclear, Boston MA). Bovine albumin-coated charcoal was used for

separation of free and antibody bound labeled AVP. This assay is highly specific for

AVP, with the antiserum prepared against a lysine vasopressin-thyroglobin conjugate,

and has a sensitivity of 0.6 pg/ml. Extraction recovery of AVP was determined using

plasma spiked with a known concentration of AVP (Peninsula Laboratories, Belmont

CA). The recovery sample was extracted and analyzed along with the subject's samples.

The extraction recovery was 90%.

Urine was analyzed for volume and osmolality by freezing point depression (model 3DII,

Advanced Instruments).

Fluid Intake/Body Mass: Subjects ingested 1.5 ml/kg of water after shift 3 and shift 6 of

each period. These volumes were based on pilot data in order to obtain a 2-3% reduction

in body mass in the pad condition and were held constant in both test conditions. In an

attempt to minimize esophageal probe temperature disturbances, water was kept at 23ºC.

No other water ingestion was allowed for the duration of the experiment until the final

resting blood sample was taken. Sweat loss was calculated by subtracting final from

initial body mass with corrections for water ingestion and urine output. Sweating

efficiency was calculated as the sweat evaporated (sweat loss minus water retained in

pads and/or clothing) divided by sweat loss, and assumed no dripping which was not

observed. No corrections were made for respiratory water losses which were assumed to

be equal between test conditions, and because oxygen consumption was not recorded

during the simulated game (34).

Skin and Esophageal Temperature: Internal core temperature (T_c) was measured every

five seconds by an esophageal thermocouple at a depth determined by passing the

thermocouple through the nose for a distance of one-fourth the subject's height as

described previously (160). Core temperature measurements during the 250 seconds

following the ingestion of water after shift three of each period was excluded to avoid the

impact of water temperature on recorded esophageal temperature. Core temperature was

expressed in three manners, first as an absolute number, second as a change from resting

values, and third, as an accumulated area under the curve. The area under the curve

analysis was used to represent the accumulated thermal challenge to subjects and was

derived by summation of each core temperature by the sampling interval of five seconds.

The resulting area for each period was added to each subsequent period in order to

evaluate the additive thermal strain undergone by subjects. Skin temperature was

measured with thermocouples mounted across acrylic rings, which were attached to the

skin so that the outer surface of the thermocouples was freely exposed to the air. Mean

skin temperature ($\overline{T}_{sk}$) was calculated every five sec from temperatures at eight skin sites

according to the equation:

$$\overline{T}_{sk} = .115T_1 + .170T_2 + .205T_3 + .090T_4 + .080T_5 + .053T_6 + .190T_7 + .097T_8$$

Where T_1 = Chest, T_2 = Low Back, T_3 = Forehead, T_4 = Abdomen, T_5 = Deltoid, T_6 =

Forearm, T_7 = Thigh, and T_8 = Calf temperatures. This weighting is based on the product

of regional area (161) and relative thermal sensitivity (162). Overall mean body

temperature ($\overline{T}_b$) was calculated as:

$$\overline{T}_b = 0.9(T_c) + 0.1(\overline{T}_{sk})$$

Where T_c represents esophageal core temperature and $\overline{T}_{sk}$, represents mean skin

temperature (1).

DATA ANALYSIS

Comparisons were made of all dependent variables between the two test conditions of

protective equipment versus no protective equipment.

Statistics: To evaluate relative changes in body and equipment masses, skin, core, and

mean body temperature, dependent group t-tests were used. All other variables were

analyzed with an ANOVA for repeated measures (within group variables: pad condition

and time). When significant differences were found, orthogonal contrasts tested

differences between specific means related to the hypothesis of interest. To generate the

area under the curve analysis for core temperature, if significance was determined by

ANOVA, a minimum significant difference was calculated using the pad condition * time

mean square error term. This minimum significant difference was then used in a post-

hoc t-test analysis to look for differences in the cumulative areas for each period. Data

are expressed as means +/- SE. Differences were considered statistically significant

when $P < 0.05$ (SPSS, SPSS, Chicago IL).

Sample Size Calculation: As power drop-off is of particular interest to those in the

hockey community, it was selected as the criterion variable to calculate statistical power.

Published values of peak anaerobic power outputs in hockey forwards, as measured by a

45 sec maximal cycle sprint have been demonstrated to be 12.2 W/kg with a standard deviation of 1.02 W/kg (151). Using the following equation with an effect size of 15%, a sample size of 5 was calculated in order to achieve a power of 0.8 with a two-sided test at an α level of 0.05.

$$N = 2*(s/d)^2 * \langle Z_\alpha + Z_\beta \rangle^2$$

Where s = standard deviation, d = effect size, Z_α = 1.96 for a two sided test, and Z_β = 0.84 for a power of 80%.

RESULTS

THERMOREGULATORY INDICES

Skin and Core Temperatures: Mean skin temperatures averaged over the entire game were elevated in P versus NP (34.1ºC versus 28.8ºC, $P < 0.05$) and are shown by period in figure 10. Individual skin sites are shown by period in appendix E. Mean core temperature was not different between pad conditions when comparing absolute values, but when expressed as a change from resting values exhibited a trend ($P = 0.053$) towards elevations in the pad condition, particularly in the third period. When analyzing the area under the curve for core temperature (Table 23) it was apparent that the accumulated area was greater ($P < 0.05$) during both the second and third periods. Absolute and relative core temperatures are shown in figures 11 and 12 and core temperature by period is shown in appendix F. Data from three subjects were omitted in the core temperature calculation ($n = 5$); two due to technical problems with collection, and the third due to the inability to tolerate the core temperature sensor.

Period	Accumulated Area Under Curve (ºC min)	
	No Pads	*Pads*
1	18.05 (1.17)	23.18 (3.03)
2	34.27 (2.56)	46.27 (5.53) [A]
3	48.21 (3.05)	70.85 (8.52) [A]

Table 23: Accumulated area under the curve for esophageal core temperature.

Area calculated by multiplying core temperature by sampling interval (5 seconds). Data is accumulated (period 2 data = period one area + period 2 area)
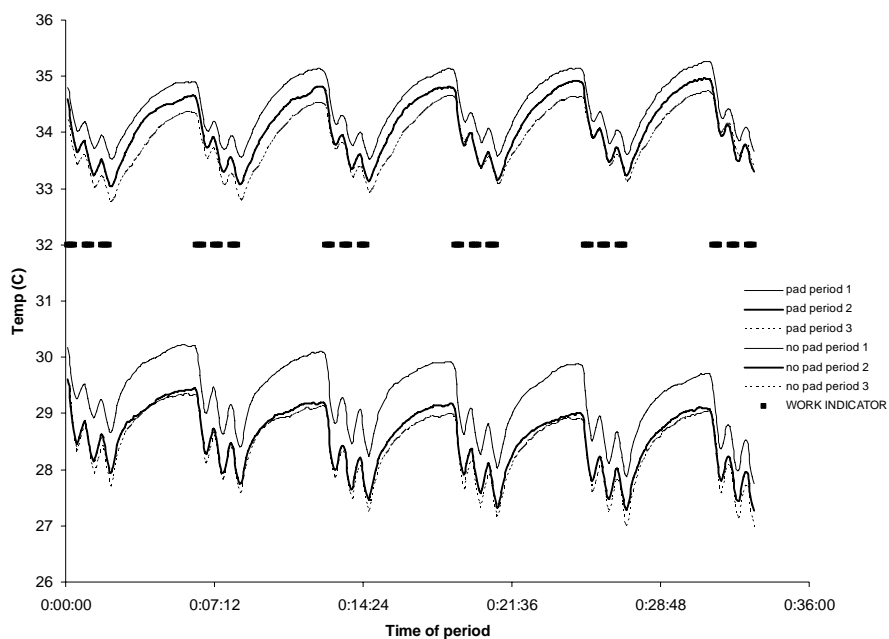[A] significantly greater than no pad condition ($P < 0.05$)

Figure 10: Mean skin temperatures by period in pad versus no pad condition.

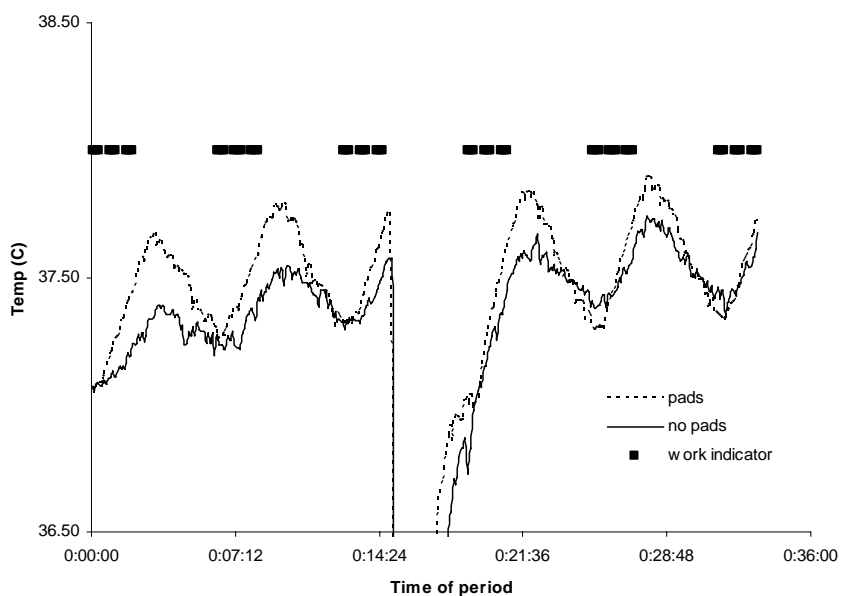Data sampled every five sec. Pad condition > No pad (P < 0.05)

Figure 11: Esophageal core temperature for entire game in pad versus no pad condition.

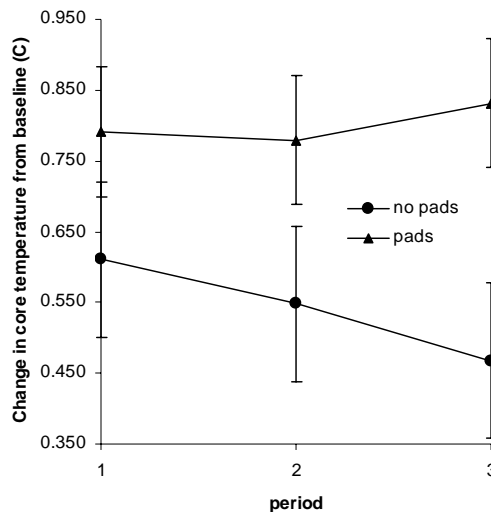Data sampled every five sec. P vs NP not significantly different

Figure 12: Mean relative changes in esophageal core temperature from baseline in pad

versus no pad condition by period.

Data averaged over entire period both work and rest intervals. Values are means +/- SE. Effect of pads: P = 0.053

The elevated skin temperatures contributed to an elevated mean body temperature in P

versus NP over the entire game (37.18ºC versus 36.58ºC, $P < 0.05$).

Water Losses: Sweat losses as expressed as percentage of body mass reduction were

elevated in P versus NP (2.57% versus 1.18%, $P < 0.05$). The majority (96%) of the

sweat secreted was evaporated (see methods for determination) in NP, while only 66.7%

was evaporated in P ($P < 0.05$). This led to an increase of 0.7 kg in the mass of the

clothing worn during P versus 0.04 kg in NP.

Plasma: Plasma indices include data from seven subjects as blood was not able to be

collected from one subject due to peripheral vasoconstriction during exercise.   Plasma

osmolality (Figure 13) was similar at rest in both conditions, but while wearing pads,

displayed an increase during exercise (P < 0.05).
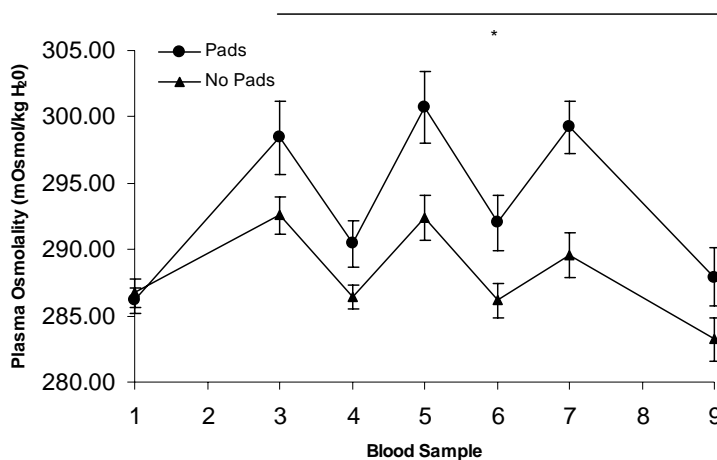

Figure 13: Plasma osmolality during experimental protocol in pad versus no pad

condition.

Values are means +/- SE. * P < 0.05 pad greater than no pad condition.

Changes in Hb and Hct led to PV decreases (P < 0.05) during blood samples 4 and 6

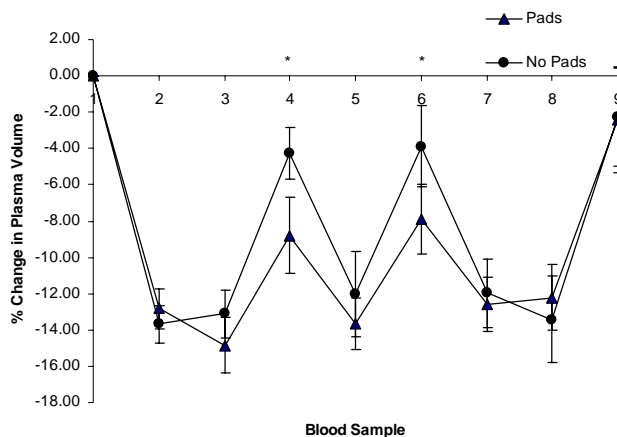(Figure 14). By the end of the final 45 min rest period plasma volume had recovered in

both groups.

Figure 14: Changes in plasma volume during experimental protocol in pad versus no pad

condition.

Values are means +/- SE. * P < 0.05 pad greater than no pad condition.

Plasma [AVP] (Figure 15) displayed a time effect, (P < 0.05), but displayed no effect (P
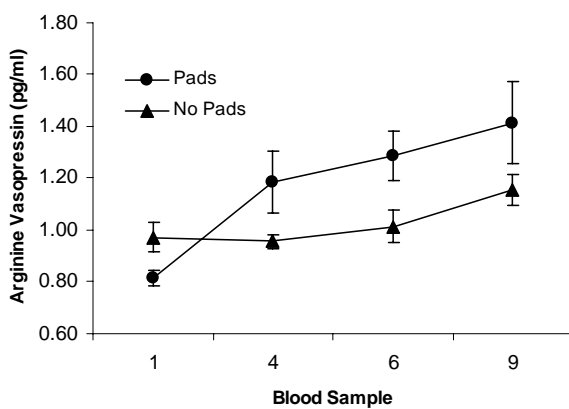
= 0.115) of garment condition between groups.

Figure 15: Changes in AVP during simulated game protocol in pad versus no pad

condition.

Values are means +/- SE. significant effect of time (P < 0.05), no effect of pads.

Urine: Urine osmolality (Table 24) was elevated over the duration of the simulated game

($P < 0.05$), but was not different between groups. Urine specific gravity (Table 24)

displayed an effect of both time and pads, with P > NP for urine samples 4 and 5 (P <

0.05).

Variable	Condition	Sample 1	Sample 2	Sample 3	Sample 4	Sample 5
Urine Specific Gravity	Pad	1.005 (0.0011)	1.006 (0.0017)	1.008 (0.0013)	1.025 (0.0013) [a]	1.026 (0.0015) [a]
	No Pad	1.005 (0.0010)	1.004 (0.0010)	1.007 (0.0013)	1.018 (0.0017)	1.017 (0.0027)
Urine Osmolality (mOsm/L)	Pad	236.44 (43.57)	189.63 (35.1)	255.0 (42.9)	744.0 (43.2)	772.94 (50.14)
	No Pad	235.75 (73.72)	280.81 (58.29)	283.94 (39.07)	628.31 (55.4)	560.71 (78.41)

Table 24: Urinary indices of hydration collected during period breaks.

Values are means (SE). [a] P < 0.05 pad greater than no pad condition.

PERFORMANCE INDICES

5 x 6 Test Sprint Power: Both mean and peak powers (Table 25, Figure 16) were reduced

during the post game sprints in the pad condition ($P < 0.05$). Mean power was reduced

by 2.7% and 14.5% in NP and P, while peak power was reduced by 0.2% and 12.0%

respectively in NP and P.

Power Output (W)	Familiarization Visit #2	No Pad		Pad	
		Rested	*Fatigued*	*Rested*	*Fatigued*
Peak Power	971.94 (23.4)	923.4 (31.1)	921.3 (29.5)	941.64 (19.8)	829.0 (40.6) [a]
Mean Power	884.9 (19.8)	852.7 (19.0)	829.3 (23.8)	860.9 (14.2)	736.3 (37.1) [b]

Table 25: Power outputs during 5 x 6 tests

Tests performed during final familiarization visit, pre and post simulated game in both pad and no pad conditions.
Values are means +/- SE
Mean power = average power output across all five sprints. Peak power = highest one sec power output during series. [a] different than all other peak power values ($P < 0.05$). [b] different than all other mean power values ($P < 0.05$)
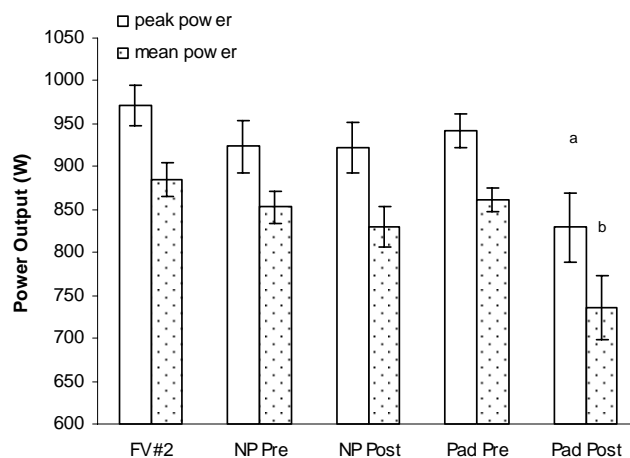

Figure 16: Peak and mean power outputs during 5 x 6 tests.

FV#2: familiarization visit #2, NP Pre: no pad rested condition, NP Post: no pad fatigued condition, Pad Pre: pad rested condition, Pad Post: pad fatigued condition.
Values are means +/- SE
[a] different from all other peak power outputs ($P < 0.05$), [b] different from all other mean power outputs ($P < 0.05$)

Power Output During Simulated Game: Mean power output during the simulated game

was not different from pad to no pad condition (348.2 versus 352.08 W pad versus no pad

condition respectively) (P > 0.05). A correlation was used to compare individual power

outputs between conditions which demonstrated an r = 0.99 (P<0.05).

PHYSIOLOGICAL INDICES

Heart Rate: Heart rate (Figure 17) was elevated both during work (83.7 and 78.8% of

maximal HR) and rest (63.4 and 55.9% of maximal HR) in the pad versus no pad

condition. Peak heart rates experienced during the simulated game ranged from 93-98%
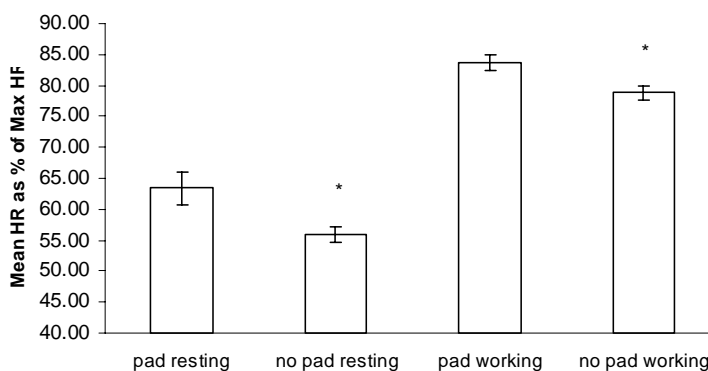
of MHR in the pad condition.

Figure 17: Working and resting heart rates as a percentage of maximal heart rate.

Maximal heart rate established during incremental maximal test in familiarization visit.
* Different from pad condition (P < 0.05)
Values are means +/- SE

Plasma: Immediately following periods one, two, and three, plasma lactate (Figure 18)

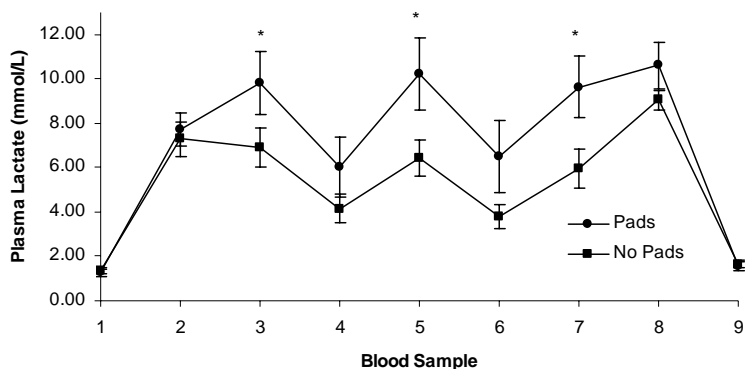was elevated (42%, 60%, and 64% respectively) in the pad condition (P < 0.05).

Figure 18: Plasma lactates during experimental protocol.

* Different between pad conditions (P < 0.05)
Blood samples 2 and 8 were obtained immediately following 5x6 sprint tests.
Values are means +/- SE

Plasma glucose (Figure 19) was elevated in the pad condition (P < 0.05) following blood

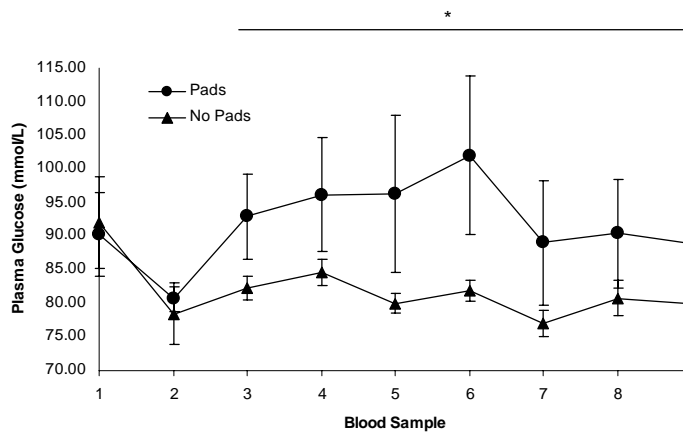sample 3 and remained so for the remainder of the protocol.

Figure 19: Plasma glucose during experimental protocol.

* Different between pad condition (P < 0.05)
Values are means +/- SE

Plasma norepinephrine (Figure 20) demonstrated an increase in pad versus no pad condition following blood samples 5 and 7, while plasma epinephrine (Figure 21) revealed no such differences.
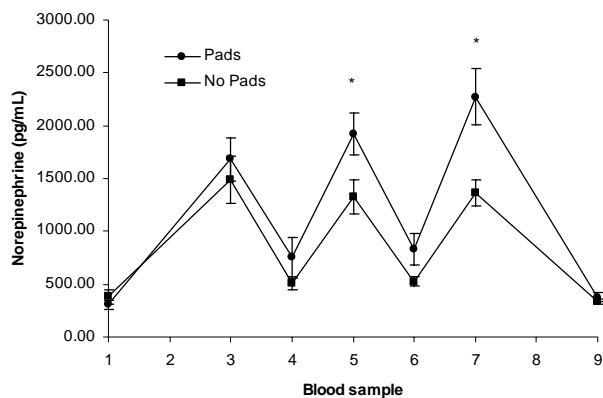

Figure 20: Plasma norepinephrine values for experimental protocol.

* Different between pad condition (P < 0.05)
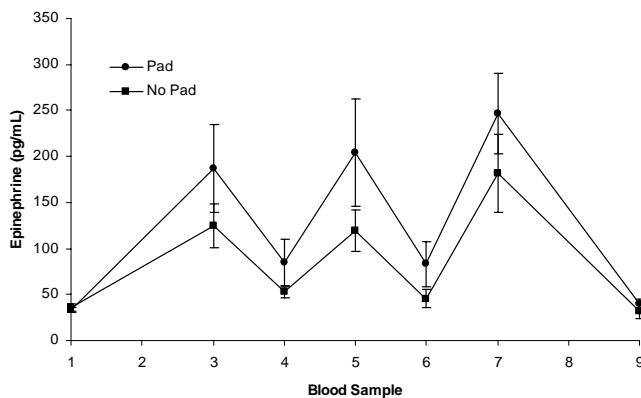Values are means +/- SE

Figure 21: Plasma epinephrine values during experimental protocol.

No significant differences between conditions.
Values are means +/- SE

PSYCHOLOGICAL INDICES

Ratings of Perceived Exertion: Subjects consistently rated the work performed during the simulated game as being 30-53% more intense ($P < 0.05$) in the pad condition (Table 26).

Condition	Period 1			Period 2			Period 3		
	Leg	*Chest*	*Body*	*Leg*	*Chest*	*Body*	*Leg*	*Chest*	*Body*
Pad	5.88 (0.04)	6.00 (0.38)	6.13 (0.40)	7.13 (0.40)	7.25 (0.31)	7.63 (0.26)	8.13 (0.03)	8.00 (0.38)	8.25 (0.25)
No Pad	4.50 (0.33) [a]	4.25 (0.45) [a]	4.25 (0.37) [a]	5.50 (0.27) [a]	4.88 (0.40) [a]	5.00 (0.27) [a]	6.00 (0.19) [a]	5.63 (0.26) [a]	5.63 (0.18) [a]

Table 26: Ratings of perceived exertions during simulated game.

Subjects rated exertion based on 0-10 scale at the end of shift 6 of each period. Ratings were given for leg fatigue, breathing difficulty, and overall feeling of body. Values are means +/- SE
[a] indicates less than pad condition ($P < 0.05$)

CRT, SRT: There was no effect of time or pad conditions on either SRT or CRT results (Table 27).

Test	Familiarization visit 2	Pads				No Pads			
		rest	*period 1*	*period 2*	*period 3*	*rest*	*period 1*	*period 2*	*period 3*
SRT	0.237 (0.007)	0.231 (0.008)	0.221 (0.010)	0.225 (0.008)	0.230 (0.011)	0.240 (0.013)	0.232 (0.012)	0.228 (0.010)	0.239 (0.015)
CRT	0.450 (0.020)	0.447 (0.010)	0.408 (0.009)	0.437 (0.019)	0.429 (0.014)	0.452 (0.023)	0.428 (0.019)	0.425 (0.015)	0.431 (0.014)

Table 27: Reaction time results during simulated game.

No differences between any conditions in either SRT or CRT.
Values are means +/- SE

DISCUSSION

The primary impact of the hockey protective equipment was a 12% (+/-3.3%) reduction in peak power and a 14.5% (+/- 2.3%) reduction in mean power in the 5 x 6 test performed at the completion of the simulated game in the pad condition. This was most likely secondary to an increased reliance upon anaerobic glycolysis during the simulated game which may have been driven by several factors and resulted in elevated plasma lactate concentrations entering the final sprint series. Subjects experienced a combination of mild elevations in core temperature, moderate elevations in skin temperature, and a moderate level of dehydration. Any of these, alone or in concert, may lead to decreases in stroke volume which may have been due to reduced blood volume, reduced central venous pressure, or lower ventricular filling time secondary to elevated heart rate. Reductions in stroke volume can lead to decreases in cardiac output, mean arterial pressure, and ultimately reduced leg blood flow. This reduction in blood flow to active muscles in the legs would contribute to an increased reliance upon anaerobic metabolism, reduced aerobic contributions to power output, and a reduced rate of PCr resynthesis. All of these mechanisms could have contributed to the elevated lactate production during the simulated game, and may have played a role in the reduced power output seen during the final 5 x 6 test.

This study was also designed to replicate the energy demands of ice hockey which is challenging because of the highly complex and intermittent nature of the energy output

during a game. This discussion must therefore, begin with a description of the success or failure of the protocol to duplicate such an event.

Validation of Protocol

The time motion data of Green et al (145, 146) was used as a model for the simulated game. In these studies, total playing time averaged 1441 sec (1152-1723 s) and was broken down into an average of 19.1 (14.5-24.3) shifts. Each shift comprised of work and rest intervals with an average of 2.3 play stops per shift. Total work time per shift averaged 77.6 sec (57.9-88.1 s) and each of the intra-shift rest intervals averaged 28.0 sec (25.7-30.5 s). There was 236 sec of recovery time between shifts (159-291 s). Our simulated game total playing time of 1350 s distributed over 18 shifts with 2 play stops of 25 s duration per shift interspersed with three 25s work intervals, and a resting time of 250s between shifts matched the published data quite well.

Published data also exists for heart rate and blood lactate concentration during ice hockey competition. During ice hockey, heart rate ranges from 89 to 95% of maximal heart rate with peak on-ice heart rate of 97-100% of maximum and mean off-ice heart rate ranging from 59-71% of maximum (145, 148, 149). Our protocols peak working heart rate of 93-98% of maximum and mean working and resting heart rate of 84% and 63% of maximum are consistent with these data. Published blood lactate concentration display a large range of variability, due to the intermittent and variable nature of the game, with values ranging as high as 11 mmol/L to as low as 2.9 mmol/L (155). In our protocol, the plasma lactate concentration immediately following each period of work averaged 9.9 mmol/L

which when using a conversion factor of 0.6 to 0.7 to account for Donnan equilibrium forces and differences in erythrocyte and plasma lactates (163-165) yields blood lactate concentrations of 5.9-6.9 mmol/L.

Typical values of voluntary dehydration during ice hockey average 3% with maximum values approaching 10% of body mass (146, 155). Water intake for the simulated game was developed during pilot testing to match the voluntary dehydration experienced during ice hockey play. The resulting reduction in body mass of 2.57% in the pad condition was also consistent with these data.

Plasma lactate concentration was elevated by 42%-64% during the simulated game protocol during the pad condition when compared to the no pad condition. Of particular interest, the plasma lactate obtained just prior (blood sample 7) to the fatigued 5 x 6 test was 64% (+/- 13.1%) higher in the pad versus no pad condition. This elevation was most likely due to a combination of an increase in glycolytic flux (discussed below) and a reduction in lactate clearance (76) experienced during the simulated game. Increases in glycolytic flux have several implications in the development of fatigue as high levels of blood and intramuscular lactate have been associated with fatigue in high intensity intermittent exercises such as the 5 x 6 test utilized here (14). High levels of anaerobic metabolism lead to increased concentrations of intracellular $[H^+]$, which has been associated with reduced peak force, inhibition of glycolysis (166), and reductions in oxidative ATP production (167). However, questions have been raised regarding the role of pH and muscle fatigue because muscle pH has been shown to lag power recovery, thus

lower pH may be only one factor leading to fatigue (56, 168). Elevated reliance upon glycolysis would also lead to increased glycogen consumption and the possibility of exhausting glycogen stores. Increased glycogen consumption has been demonstrated in some (106, 123, 124) but not all studies (125-128) examining exercise in the heat, and reduced muscle glycogen has been associated with fatigue during intermittent sprinting protocols such as our 5 x 6 test (60). When examining the reduced power outputs (no change in the no pad condition and 12-15% reduction in the pad condition) and blunted lactate increases (68% in the no pad and 15% in the pad conditions respectively) experienced during the post game 5 x 6 test, it is hard to determine how much of the reduction in power and blunted increase in plasma lactate experienced in the pad condition was due to metabolic inhibition of glycolysis, reduced central drive to produce power, or limited metabolic substrate availability such as PCr and/or glycogen.

There were many factors evident in the pad condition of our protocol which would have contributed to the increased glycolytic flux during the simulated game. Subjects experienced an increased core temperature during the pad condition which has been shown to result in increases in blood lactate concentrations during exercise in the heat (75). Although no direct muscle temperatures were obtained, thigh and calf skin temperatures were elevated in the pad condition, indicating that active muscles experienced elevated temperatures as well. These elevations in muscle temperature have been show to increase glycolytic flux by a direct temperature effect which enhances enzyme kinetics (72, 81). Sympathetic nervous system activity was also elevated in the pad condition; represented by elevations in plasma norepinephrine concentrations,

although the large variability in epinephrine levels yielded non-significant findings

(107). It has previously been demonstrated that exercise in dehydrated conditions can

lead to elevated plasma epinephrine levels (106). Moreover, these elevations have been

shown to increase glycogenolytic rate by stimulating the conversion of phosphorylase to

its active form (107, 108).

The increase in glycolytic flux may have also been due to a reduction in leg blood flow

and ensuing aerobic energy provision. During exercise in the heat; dehydration, elevated

skin blood flow, and increased heart rate, each independently contribute to decreased

stroke volume, cardiac output, and mean arterial pressure (77, 79, 80, 99-101) all of

which are crucial in maintaining leg blood flow (117). The finding of reduced leg blood

flow during exercise and thermal stress has met with mixed results (117, 118) with some

authors postulating that the thermal stress must be significant enough to effect cardiac

output and mean arterial pressure before reductions in leg blood flow become evident

(117).

Subjects were dehydrated by over 100% more during the pad condition versus the no-pad

condition due to greater sweating rates. The elevated sweating rate appeared to overshoot

what was necessary to match cooling requirements as only 67% of secreted sweat

evaporated in the pad condition. Elevated skin temperature may have contributed to the

greater sweating rate because increases in skin temperature have been shown to

accentuate core temperature sweat response as well as independently drive sweat rate

(160). Moreover, dehydration is associated with lower blood volume, greater heart rate,

greater glycolytic flux, and premature fatigue compared to euhydrated conditions (95,

105). Although we did not measure skin blood flow, the greater sweating rates and skin temperature suggest that skin blood flow was increased during the pad versus the no-pad condition. This greater skin blood flow indicates a redistribution of blood to the periphery, and thus a lower central venous pressure (78, 79). Stroke volume may have been further compromised by a reduced filling time associated with the greater working and resting heart rates during the pad versus no-pad condition (80).

Lower leg blood flow in the pad condition, if present, may have also reduced the aerobic energy available during exercise and recovery. This reduction during exercise would limit aerobic ATP production, a crucial component in maintaining power output in the latter stages of repeat exercise (14, 53). This reduction in aerobic ATP production would have led to increased reliance upon glycolysis during the simulated game and could have independently contributed to the reduced power outputs seen during the fatigued 5 x 6 test in the pad condition. Reduced leg blood flow could also impair the ability to restore PCr as this restoration is dependent upon oxygen availability (52). If present, reduced levels of PCr would contribute to both an increased reliance upon glycolysis during the simulated game as well as to the fatigue experienced in the post game 5 x 6 test (53).

Distinct from the peripheral factors of fatigue is the possibility that the reduction in power output following the simulated game in the pad condition was due to a reduced central drive (83). Central fatigue is strongly related to elevations in core temperature in the range of 38.6-40.3 ºC and as such would not seem to have played a dominant role during our protocol because core temperatures averaged 37.5ºC with peak values of

37.9ºC. However, a "pacing" effect has been demonstrated by subjects during elevated

environmental temperatures in which subjects self-selected a reduced power output

during exercise in the heat even with normothermic core temperatures, which supports a

role for elevated body temperature in inducing central fatigue (87). However, Thomas et

al (88) demonstrated that elevations in core temperature as opposed to skin temperature

lead to a reduction in central drive. Elevated RPE, commonly seen during exercise and

dehydration (102-104), and present during our simulated game, provided evidence of an

altered perception of work intensity. The simulated game was performed at the same

work intensity in both conditions, but subjects rated it as 30-53% more intense in the pad

condition. This may have been partially due to the increased plasma lactate

concentrations which have been shown to correlate with RPE values (136), but RPE has

also been shown to increase in dehydrated conditions independent of blood lactate

concentrations (100).

We evaluated simple cognitive functions by the use of the SRT and CRT, but were not

able to demonstrate any reduction in performance during the protocol. Exercise has been

shown to have an inverted "U" effect on the performance of cognitive tasks (140) with

increased levels of arousal linked to increased in heart rate and/or perceived exertion.

Prior research has showed mixed results when looking at cognitive performance exercise

and thermal challenge with studies showing no effect (142), decreased performance,

(143), or improvements in performance (140). As in studies evaluating physical

performance, the interpretation of the data can be confounded by concomitant

dehydration. Dehydration, independent of hyperthermia has been shown to result in

depressed cognitive performances (140), impairing cognitive abilities such as perceptive discrimination, and short term memory, but did not reaction time (144).

Plasma volume readings were made more difficult by the high intensity exercise performed immediately prior to several measurements which has been shown to result in plasma volume shifts of 12% - 20% (13, 14). The plasma volume differences which were evident during blood samples 4 and 6 (taken at the end of 15 minute period breaks) had recovered by the end of the final 45 min rest interval while P_{osm} values remained elevated (289 +/- 1.7 versus 283 +/- 1.6 mOsmol/kg H2O, for pad versus no pad respectively). Even with these elevations in P_{osm} there were no detectable differences in AVP levels, which is strongly related to changes in P_{osm} (169). This lack of AVP response may have been due to the inhibition of AVP release during drinking (170) or due to the inhibitory effects of elevated blood pressure experienced during exercise (171).

The recovery of plasma volume at the end of the 45 min rest interval during which no water was ingested was most likely due to the increased P_{osm} values and a shift of albumin from the interstitial space to the vascular compartment. Gillen et al (19) demonstrated a recovery of PV following repeated bouts of cycle ergometry during which plasma volume fell by 15%. After one hour of seated recovery with no fluid replacement, PV had recovered to baseline, despite an overall body mass loss of 820g. Moreover, plasma albumin and total protein content increased enough to account for the entire PV restoration (19).

The increases in core and skin temperatures, and sweat loss, were due to the inhibition of heat transfer imparted by the protective equipment worn. While there are no prior studies of thermoregulation and hockey protective equipment, there have been studies published examining the effects of American football uniforms. These uniforms have been shown to significantly impair both convective and evaporative cooling during exercise (42, 44) and even more so during recovery (45). In our protocol, when examining the skin and core temperatures in relation to the work intervals, it was clear that both skin and core temperature continued to increase after exercise in the post exercise period. These continued temperature elevations were due to the elimination of the airflow from the fan which simulated the effects of skating velocity, and would mimic the loss of airflow experienced during the time spent sitting on the bench between shifts.

The present results are consistent with published papers examining the combined effects of hyperthermia and dehydration during high intensity intermittent exercise in the heat. Elevated muscle and core temperatures and mild dehydration impaired repeated sprint performance with reductions in mean power of 10% when exercising in hot (40°C) versus neutral (20°C) conditions (121). Moreover, subjects in hot conditions consumed less oxygen during repeated sprints and displayed a trend towards increased glycogen consumption (P = 0.06) during a fatiguing protocol. Maximal hand grip strength was reduced by 8% following exercise in the heat as opposed to neutral conditions demonstrating some degree of central fatigue (121). Morris et al (122) found that total intermittent running distance to fatigue was decreased in hot (33°C) versus moderate (17°C) conditions. Moreover, within the intermittent running protocol sprint times were

reduced more profoundly in the heat compared to moderate conditions. The comprised performances were associated with greater core and muscle temperatures, RPE, blood and muscle lactate concentrations as well as plasma NE concentration in the hot versus moderate conditions. Accompanying the increases in blood and muscle lactate was a trend ($P=0.055$) towards increased glycogen use (34%) in the heat.

Limitations of the Study

This experiment was limited by the need for a laboratory environment to allow for collection of an extensive amount of data and for strict control of work intensity. As such, it is limited by the validity of the protocol used to simulate the sport of ice hockey. The simulation relied entirely on power output from the legs to generate fatigue, which is not typical for the sport of ice hockey. While dominated by leg power, the sport also involves battles for position and puck control, shooting, passing, and checking, none of which were simulated in our protocol. One aspect of the hockey pads which may have contributed to premature fatigue may be that they provided resistance to movement at the knee and/or hip. Subjects denied feeling any resistance to pedaling when wearing the pads, a fact supported by the similar plasma lactate concentrations during the initial 5 x 6 test (7.72 +/- 0.72 versus 7.30 +/- 0.73 mmol/L pad versus no pad respectively). In an attempt to further evaluate the resistance of the pads; four subjects performed a continuous exercise protocol where they cycled at 100 W for 10 minutes both with and without the lower extremity pads on (shin pads and hockey pants). We recorded continuous oxygen consumption and heart rate during both intervals. Both oxygen consumption and heart rate were elevated by 2-3% when wearing the lower pads

indicating that any extra work output used to overcome the mechanical resistance of the pads was minimal.

Controlling the pedaling speed may have accentuated the lactate response to the simulated game. It has been shown that as muscle temperature increases, efficiency increases at higher pedaling or contraction velocities (73, 74, 82). Perhaps if given the option, subjects may have self selected a higher pedaling speed which would have allowed for reduced resistances at the same power outputs. This increased efficiency may have reduced some of the reliance on glycolysis and ensuing elevations in plasma lactate concentration which was at least partially responsible for the increased fatigue experienced while wearing the pads.

Overall Summary

The main impact of the wearing of hockey protective equipment was the inhibition it provided to both convective and evaporative cooling. This resulted in elevated skin and core temperatures as well as a doubling of water loss. These results may have contributed to decrements in cardiac output and mean arterial pressure, and subsequently leg blood flow, which when coupled with direct temperature effects as well as catecholamine stimuli, led to an increased reliance upon glycolytic pathways of ATP regeneration during the simulated game. This increased reliance upon glycolysis led to elevated plasma lactate concentrations and possibly reduced intramuscular pH. The lower intramuscular pH, when combined with a reduction in the ability to provide aerobic resynthesis of ATP and PCr, may have reduced power output in the 5 x 6 test performed

following the simulated game in the pad condition. Also possible was a reduced CNS

drive to exercise which may have been reflected by elevated RPE experienced during the

simulated game.

Future studies should examine matching water losses, or possible modifications to the

protective equipment to allow for greater air flow next to the skin which would enhance

both convective and evaporative cooling, allowing for a reduced sweat rate which would

lead to a reduction in dehydration.

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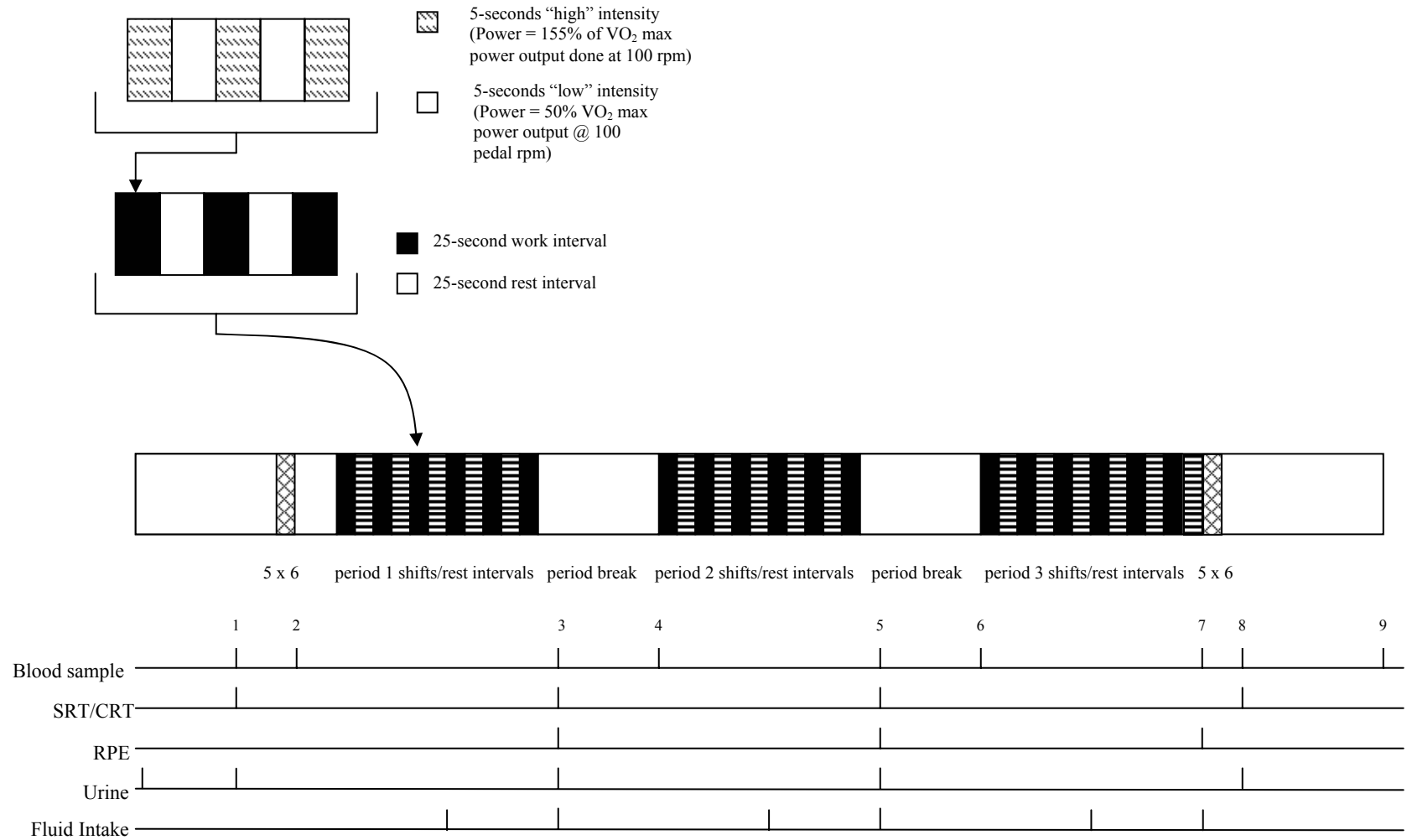
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Citation	Exercise Protocol	Environmental Conditions	Power Results	Other Significant Differences	Subject Condition
Falk et al	Repeated 15 sec Wingate Tests (15s WAnT:30s active rest) x 5 60 min passive recovery (15s WAnT:30s active rest) x 5 *WAnT: maximal cycle ergometer test using 7.5% of body mass as resistance.*	Thermo-neutral : (22 C / 40% Rh) Hot: (35 C / 30% Rh)	Subjects in the hot condition made greater peak (719.6 W) and mean (636.2 W) power in the initial series of sprints versus the thermo-neutral (664.4, and 584.0 W respectively) condition, but could not maintain this in the second series (hot peak/mean = 688.4 / 631.8 neutral peak/mean = 704.8 / 607.4). As shown, the neutral group actually improved performance in the second vs first series. This resulted in a greater calculated drop-off in peak and mean power in the hot vs. thermo-neutral condition. Fatigue within the series of sprints was the same in both conditions. *Peak Power = avg of 1st 5 seconds of power for all five sprints in a series of sprints.* *Mean Power = avg of entire 15 seconds of power output for each series of sprints.*	PV: B Core: A Skin: A	Subjects were hyperthermic, but not dehydrated (allowed to rehydrate to mach body mass losses in the rest interval)
Backx et al	Repeated 30 sec Wingate Tests (30s WAnT:30s active rest) x 3 60 min passive recovery (30s WAnT:30s active rest) x 3	Normal: (22 C / 30% Rh) Wet: (30 C / 85% Rh) Hot: (40 C / 40% Rh)	No difference in Peak or Mean Power between conditions. In all conditions, subjects made greater peak power in first vs third sprint of each series.	HR: B BLa: B Uosm: B	Subjects were hyperthermic, but not dehydrated (allowed to rehydrate to mach body mass losses in the rest interval
Ball et al	Two 30 sec Wingate Tests 30 s WAnT:4 min passive rest:30s WAnT	Normal: (18.7 C / 40% Rh) Hot: (30.1 C / 55% Rh)	PPO, MPO were not different in sprint one versus two in either conditions PPO was significantly higher in heat vs normal when averaged over both sprints (909 vs 650 W) MPO was significantly higher in heat vs normal when averaged over both sprints (612 vs 496 W) % decline from PPO to min power output was significantly greater in the heat vs normal in sprint #1 (48% decline vs 34% decline)	BLa: B	Subjects were hyperthermic, but not enough time elapsed for significant dehydration to occur.
Morris et al	Modified LIST protocol: **Part A: [**Walk (3x20m) : Sprint (1x15m) : walk (1x3m) : Cruise (3x20m) : Jog (3x20m):] x11 = 1 set. Three minutes rest after each set, total of 5 sets. (total exercise time of 90 minutes) These sets are followed by **Part B:** which consisted of repeated 60s run : 60s rest at 100% VO$_2$ max until fatigue. Walk = 1.54 m/s Sprint = maximal Walk = 4s Cruise = 90% VO$_{2max}$ Jog = 45% VO$_{2max}$	Hot: (HT) (30 C / 24% Rh) Moderate: (MT) (16 C / 50% Rh)	Exercise time was 78 min in the HT and 105 min in MT (didn't finish LIST in heat) 15 m sprints took longer to complete in the HT and had a greater rate of decline in performance over the repeated protocol as opposed to MT Total distance completed in Part A and B combined was 25% less when performed in the heat. **Not exactly same amount of work so they compared equivalent times during LIST (not endpoints for MT).**	RPE: A (during A) HR: A BLa: B PV: B Core: A	Subjects entered both tests hydrated and were allowed to drink ad libitum, this seemed to prevent any dehydration as plasma volumes and body mass were maintained, making it far less likely that any fatigue experienced was due to dehydration.
Morris et al	Modified LIST protocol: **Part A: [**Walk (3x20m) : Sprint (1x15m) : walk (1x3m) : Cruise (3x20m) : Jog (3x20m):] x11 = 1 set. Three minutes rest after each set, total of 5 sets. (total exercise time of 90 minutes) These sets are followed by **Part B:** which consisted of repeated 60s run : 60s rest at 100% VO$_2$ max until fatigue. Jog = 49% VO$_{2max}$	Hot: (HT) (30 C / 66 % Rh) Moderate: (MT) (20 C / 71% Rh)	Not everyone could finish HT trial LIST Distance covered in Part B was reduced in Hot condition **Not exactly same amount of work so they compared equivalent times during LIST (not endpoints for MT).**	RPE: A (during A) HR: A BLa: B PV: B Core: A	Subjects were allowed to rehydrate ad libitum in both tests (twice as much intake in Hot). This prevented any loss of body mass.

Appendix A: table of studies comparing exercise in the heat to exercise in thermoneutral conditions

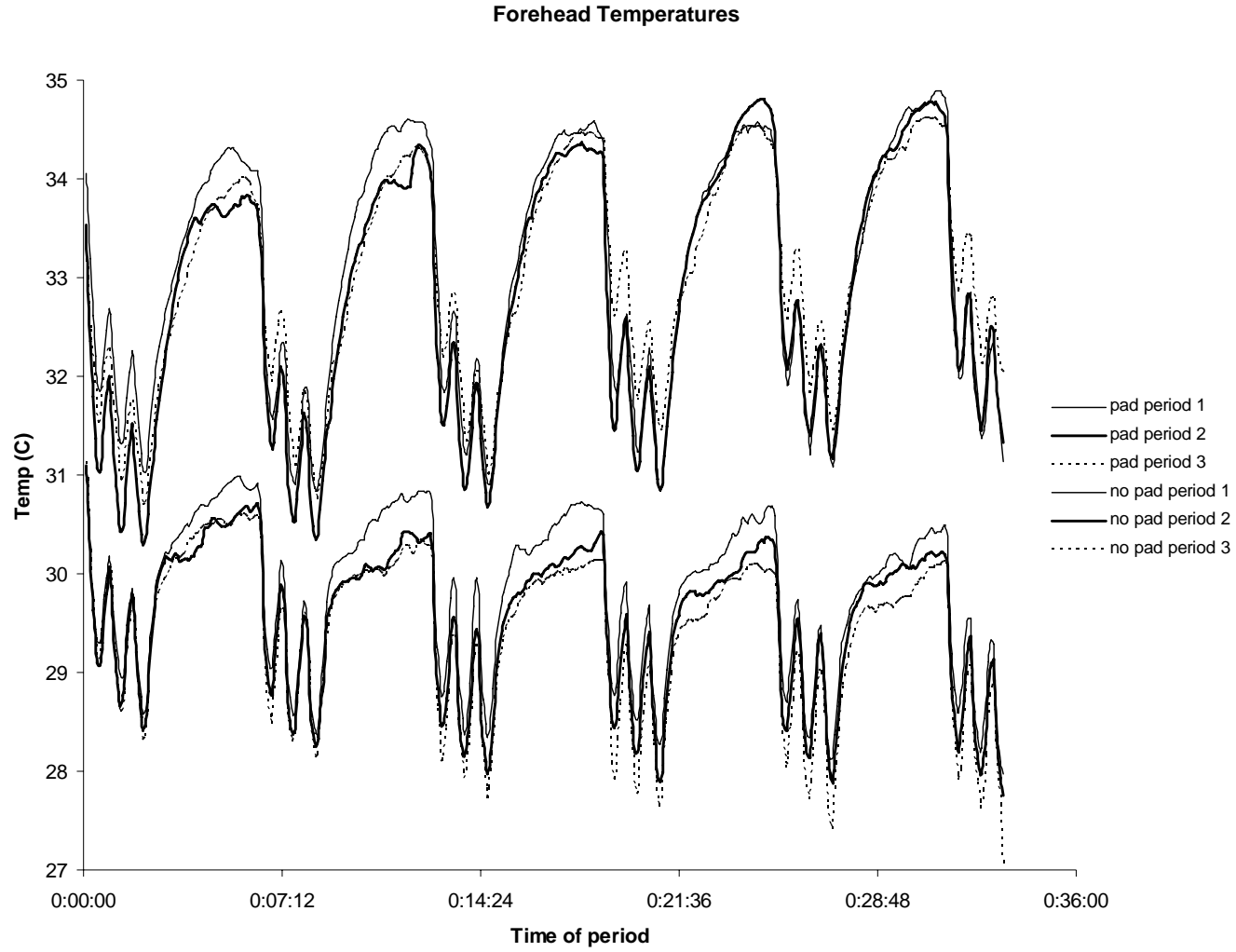| Citation	Exercise Protocol	Environmental Conditions	Power Results	Other Significant Differences	Subject Condition
Maxwell, Neil, Gardner, Nimmo (1999)	MART performance (alternating 20s sprint with 100s passive rest at increasing velocities until failure, appx 12 min) Test #1 done either Cool or Hot conditions Test #2 done either dehydrated or euhydrated at start.	Test #1: Cool: (21.3 C / 49% Rh) Hot: (32.8 C / 81% Rh) Test #2 Hypohydrated by 2% of body mass or Euhydrated	Test #1: Time to failure on MART was significantly less (138 vs 150 seconds) in hot vs cool conditions Test #2: Time to failure on MART was significantly less (148 vs 154 seconds) in hypohydrated vs euhydrated conditions.	Test #1: muscle biopsies pre and post Endurance: less Glycogen: NSD BLa: NSD Test #2 Endurance: less Core: 0.8C increase HR: higher	In Test #1, subjects were hyperthermic, but not dehydrated at start of exercise. They did not rehydrate during exercise which lasted appx 12 minutes. In Test #2, subjects were not at elevated environmental temps, but started out either euhydrated or hypohydrated by 2%.
Ftaiti, Grelot, Coudreuse, Nicol (2001)	Run for 40 min at 65% of maximal aerobic velocity while wearing an impermeable tracksuit.	Moderate: (22.5 C / 55% Rh)	When compared from rest to post exercise, maximal knee extensor torque was decreased when measured isometrically (12%), and at 60 degrees per second (17%). Values were not affected at 240 degrees per second. These reductions were accompanied by even greater reductions in EMG activity (39% and 25% respectively).	Only one group	Subjects were both hyperthermic (tympanic temps appx 40 C) and dehydrated (appx 2%) during experiment.
Maxwell, Aitchison, Nimmo (1996)	20 minute warmup then MART in either hot or cool conditions. Test with warmup lasted appx 34 minutes.	Hot: (33 C / 80% Rh) Cool: (21 C / 38% Rh)	Earlier fatigue in MART (151 s vs 140 s) cool vs hot conditions. Note: all samples taken after completion of MART which was different endpoint between conditions.	Power: fatigue HR: SD BLa: NSD Core: SD Skin: SD BM: SD	Subjects drank predetermined amount of saline solution before MART.

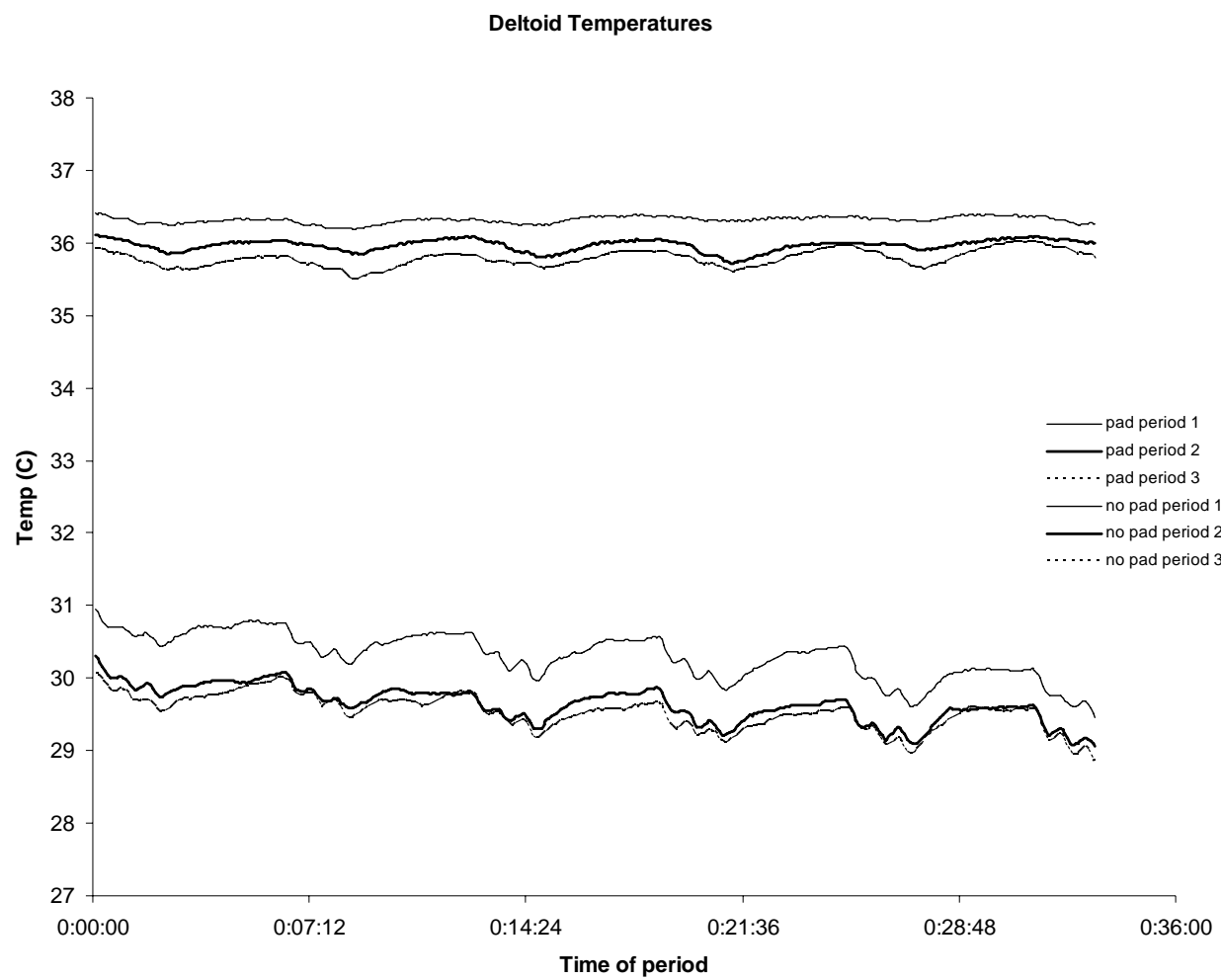Appendix B: Table of exercise under hyperthermic and dehydrated conditions.

5-seconds "high" intensity
(Power = 155% of VO_2 max
power output done at 100 rpm)

5-seconds "low" intensity
(Power = 50% VO_2 max
power output @ 100
pedal rpm)

25-second work interval

25-second rest interval

5 x 6   period 1 shifts/rest intervals   period break   period 2 shifts/rest intervals   period break   period 3 shifts/rest intervals   5 x 6

1   2   3   4   5   6   7   8   9

Blood sample

SRT/CRT

RPE

Urine

Fluid Intake

Appendix C: Graphical Representation of Protocol

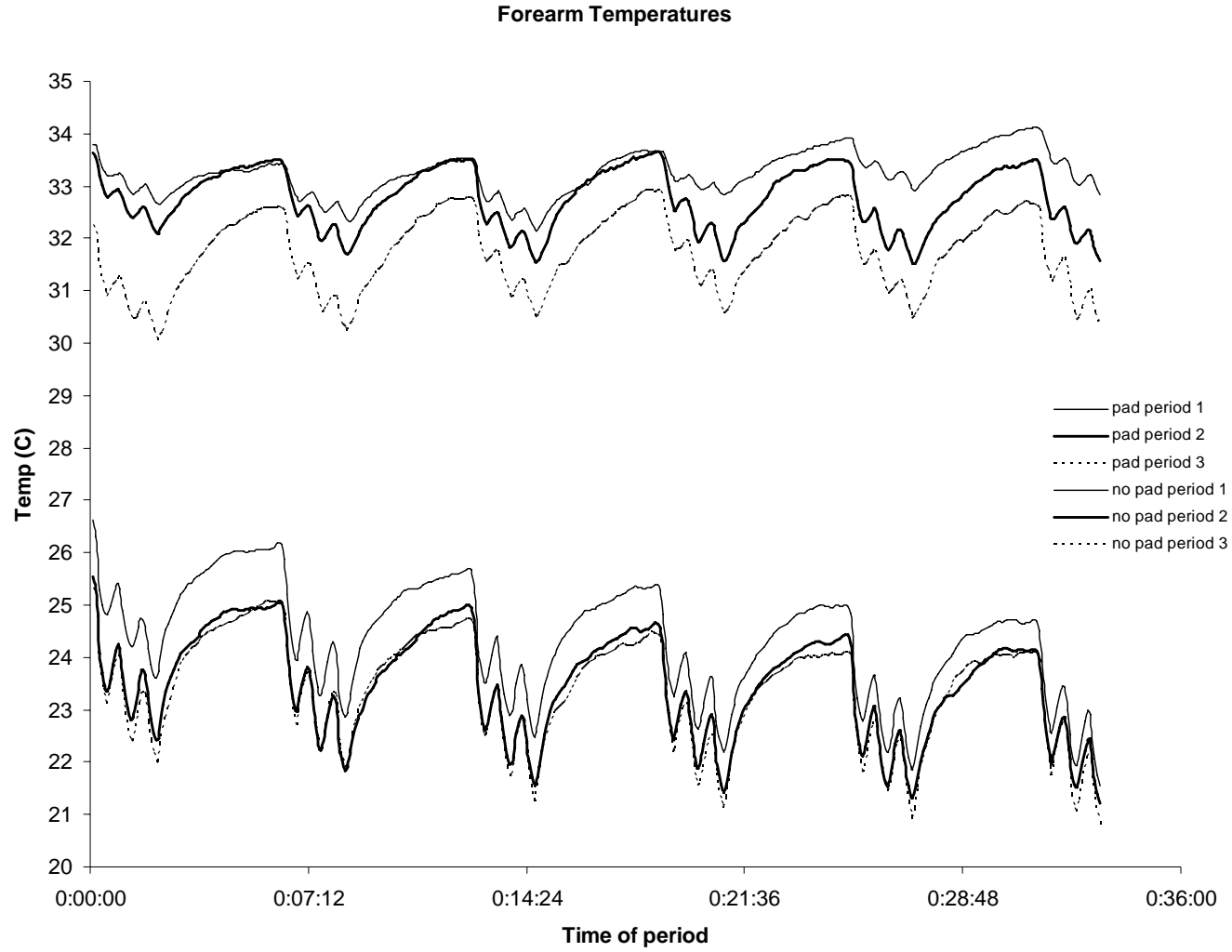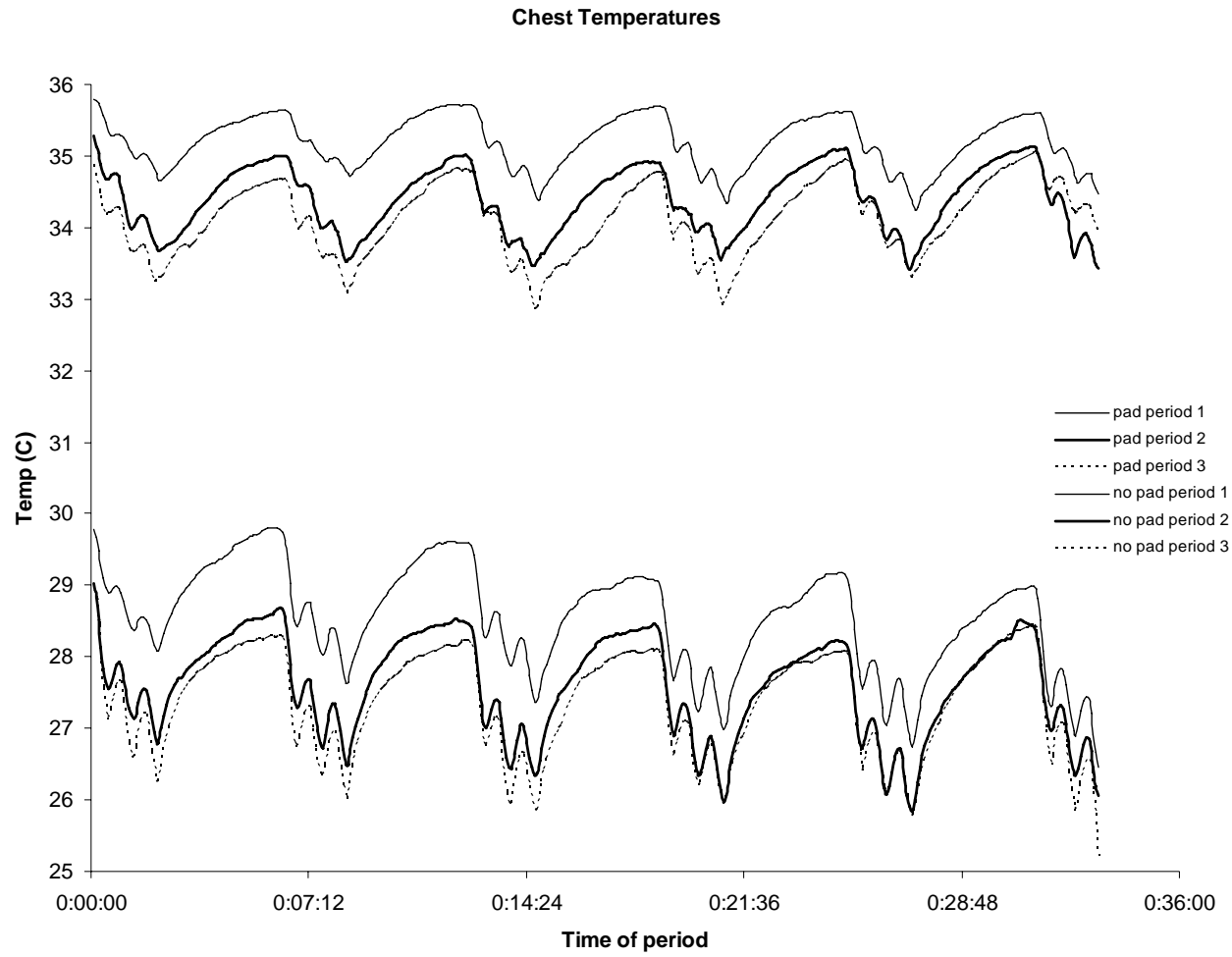| Start and Finish Times for Task		Test Items
Start	Finish	
0	42	Arrive, catheter in, void (urine specimen #1, check urine SG), bodyweight both with and without pads, temp sensors on, core temp sensor in, put gear on, baseline
42	45	Blood sample #1 *base line values* Urine specimen #2. Get body weight with pads on
45	54	Move into chamber SRT#1, CRT#1 then set up on bike
54	62	Warm up - 5 min at 80 rpm and 1.5 kg - 5 second sprints at minute 3 and 4. - 3 min at 80 rpm and 1 kg
62	65	Cool down / stretch
65	67:30	Rested 5 x 6 s sprints
67:30	69	Blood sample a. Immediately after last recovery interval take blood sample # 2
67:30	72:30	Rest for 5 min (timer starts right at end of last sprint)
72:30	105:50	Simulate period 1
105:50	120:50	Period break #1 a. Immediately after last shift take Blood sample # 3 and drink. b. RPE #1 c. SRT #2 d. CRT #2 e. Urine specimen #3. f. At the 13:00 mark get Blood sample # 4
120:50	154:10	Simulate period 2
154:10	169:10	Period break #2 g. Immediately after last shift take Blood sample # 5 and drink. h. RPE #2 i. SRT #3 j. CRT #3 k. Urine specimen #4 l. Get bodyweight with all gear on, then sit in ambient conditions until 117:00 mark, get back on bike for next period. m. At the 11:00 mark get Blood sample # 6
170	203:20	Simulate Period 3
203:20	207:30	Rest interval before fatigued 5x6 a. Immediately after last shift take Blood sample #7 and drink. b. RPE #3
207:30	210	Fatigued 5 x 6 sprints (simulating shift #6) a. Blood sample #8 (at end of last rest interval)
210	Appx 270	End of game interval a. Do last SRT /CRT #4 b. Get out and take off gear c. Collect Urine specimen #5 d. Dry bodyweight e. Be seated for 45 minutes for last blood sample Blood sample #9

Appendix D: Timeline of Simulated Game

Forehead Temperatures
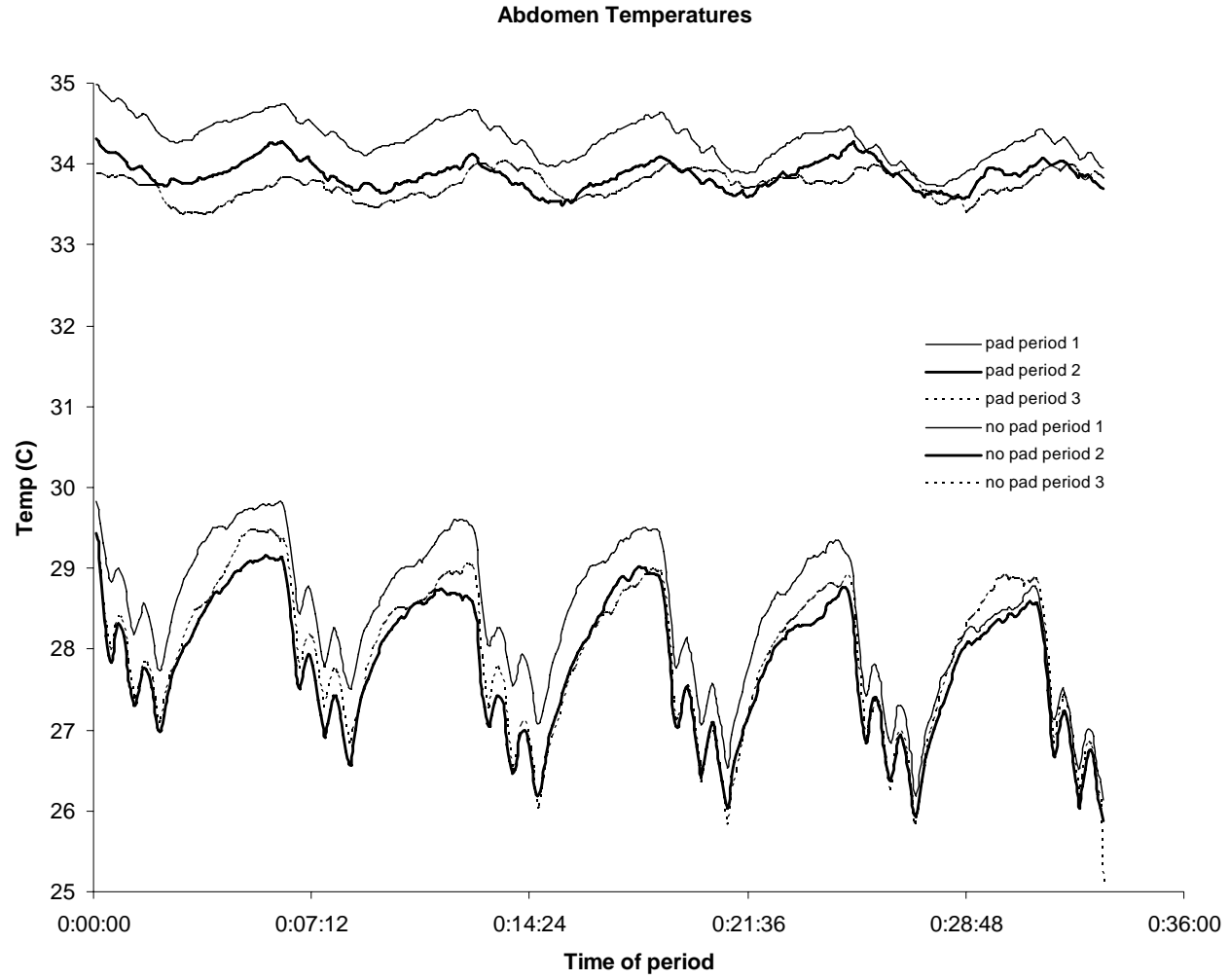

Appendix E: Skin Temps

Deltoid Temperatures

Appendix E: Skin Temps

Forearm Temperatures

Appendix E: Skin Temps

Chest Temperatures

Appendix E: Skin Temps

Abdomen Temperatures

Appendix E: Skin Temps

Back Temperatures

Appendix E: Skin Temps

Thigh Temperatures

Appendix E: Skin Temps

Calf Temperatures

Appendix E: Skin Temps

core temps period 1
pad vs no pad

Appendix F: Core Temperatures

core temps period 2
pad vs no pad

Appendix F: Core Temperatures

core temps period 3
pad vs no pad

Appendix F: Core Temperatures