

THE EFFECT OF HOCKEY EQUIPMENT ON THERMOREGULATORY  
RESPONSES AND WORK PERFORMANCE DURING INTERMITTENT  
WORK DESIGNED TO SIMULATE A HOCKEY GAME

By

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## **Abstract**

This study was designed to determine whether hockey equipment decreases the dissipation of metabolically generated heat to an extent that impairs the performance of an intermittent exercise protocol. Fifteen university hockey players completed the protocol dressed in hockey equipment (EQ) or shorts, socks, and shoes (NE). The protocol, designed to simulate a hockey game, involved performing six "shifts" of a 45 s workout that varied in intensity followed by 255 s of intervening recovery to simulate one "period". Three "periods" were performed at 10 °C separated by 15 min "intermissions" at 21 °C. Exercise (102 %), recovery (104 %), and "intermission" (104 %) heart rates and rectal (101 %) and mean skin (117 %) temperatures and were higher ( $p < .05$ ) during EQ. Weight loss (700 %) and ad libitum water consumption (148 %) were also greater in EQ ( $p < .05$ ). Work production during the "game" (95 %) and blood lactate at the end of each "period" (92 %) were lower ( $p < .05$ ) in EQ even though oxygen consumption during exercise and recovery was not different. Ratings of perceived exertion obtained after each "period" were not different between the two conditions.

Heat dissipation was decreased by wearing hockey equipment, resulting in increased body heat storage and stronger thermoregulatory responses. Thus, the thermoregulatory strain imposed by the equipment resulted in decreased work performance.

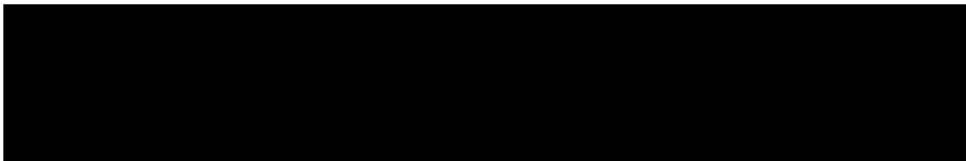
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## Table of Contents

Title Page	i
Abstract	ii
Table of Contents	iv
List of Tables	v
List of Figures	vi
Acknowledgements	x
Dedication	xi
Introduction	1
Limitations and Delimitations	4
Methods	6
Results	17
Discussion	39
Bibliography	51
Appendix: Review of Literature	57

## List of Tables

Table 1.	Mean (SD) anthropometric and fitness characteristics of the subjects.	13
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## List of Figures

Figure 1.	Time-course of the experimental protocol.	10
Figure 2.	Rectal temperature in NE and EQ and mean skin temperature in NE and EQ for one subject.	18
Figure 3.	Calf, thigh, chest, arm, back, and forehead temperatures in NE and EQ for one subject.	20
Figure 4.	Mean (SD) body weight in NE and EQ with and without equipment measured before and after the experimental condition and corrected for sweat loss.	22

Figure 5.	Mean body weight change and corrected weight change as a percentage of initial body weight in NE and EQ with and without equipment.	23
Figure 6.	Mean (SD) weight of the water consumed and sweat lost in NE and EQ and the increase in weight of the hockey equipment in EQ.	24
Figure 7.	Time-course of the heart rate response for one subject. Heart rate is illustrated every 20 s in NE and EQ.	26
Figure 8.	Mean (SD) peak exercise and lowest recovery and "intermission" heart rates for the simulated hockey game in NE and EQ.	27

Figure 9.	Mean (SD) peak exercise and lowest recovery heart rates for each "period" in NE and EQ.	28
Figure 10.	Time-course of oxygen consumption for one subject during alternate workouts in NE and EQ.	30
Figure 11.	Mean (SD) blood lactate concentration at rest, for all "periods" combined, and each "period" individually in NE and EQ.	32
Figure 12.	Time-course of mean work output for the first sprint, controlled cycling, and second sprint of each workout in NE and EQ.	34
Figure 13.	Mean (SD) work output during the first 15 s sprint of each workout for each "period" and the complete "game" in NE and EQ.	35

- Figure 14. Mean (SD) work output during each "period" and the complete "game" in NE and EQ. 36
- Figure 15. Mean (SD) rating of perceived exertion at the conclusion of each "period" in NE and EQ and after the physiological tests. 38

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## Dedication

This thesis is dedicated to the Watters who have been as a family to me and Dr. Art Quinney whom I can never adequately thank for the opportunities and encouragement he has given me.

## Introduction

Ice hockey is a high speed, contact game (Montgomery, 1988) played on a surface of ice in ambient temperatures between 4 and 12 °C (Green, 1978; Green et al., 1978). Players perform exercise of varying intensity during a "shift" of approximately 40 seconds. The "shift" is repeated after three to five minutes of inactivity and this exercise schedule may be repeated five to eight times during one 20 minute stop-time "period". A complete game consists of performing three 20 minute "periods" of play separated by 15 minutes of minimal or no activity (Green et al., 1976; Montgomery, 1979, 1988; Paterson, 1979). The predominant energy systems that meet this short-term, fluctuating work demand are the phosphagen and lactic acid systems, the latter of which produces lactic acid as a by-product, a compound which has been associated with a decrease in muscle force production (Sahlin, 1986). However, players attempt to delay the onset of fatigue by modifying the intensity and duration of the work interval during the time course of a complete game to maximize use of the phosphagen system and minimize lactate production (Green, 1979).

During muscular work, heat is released during metabolic energy expenditure (ATP hydrolysis) resulting in an increase in skeletal muscle temperature (Hill, 1949; Wilkie, 1968). This heat is conducted and convected

to the body surface where it is primarily dissipated through the evaporation of sweat. During prolonged exercise and/or exercise in hot, humid environments, blood flow to the working muscles is maintained at the expense of cutaneous blood flow, decreasing the convection of heat to the periphery. Higher core temperatures and an earlier cessation of exercise result (MacDougall et al., 1974; Rowell, Murray, et al., 1969). An increased tissue temperature and redistributed blood flow may also alter the relative contribution of energy from the different metabolic pathways, resulting in an increase in muscle and blood lactate concentrations (Fink et al., 1975; MacDougall et al., 1974; Rowell et al., 1968; Williams et al., 1962; Young et al., 1985). Therefore, the need to dissipate metabolic heat may decrease the intensity at which a hockey player can perform, resulting in less work accomplished before the onset of fatigue.

The ability of a hockey player to dissipate heat may be limited by the equipment worn (Mathews et al., 1969; MacDougall, 1979). Sporting equipment can: increase the thermal stress experienced, increase the severity of the thermoregulatory measures necessary for continued performance, and potentially decrease performance. However, the effect of wearing hockey equipment on the dissipation of metabolic heat produced during exercise has not been determined. Furthermore, a recent trend in new equipment to increased protection (Montgomery, 1988) has necessitated research to

investigate whether equipment has a deleterious effect on thermoregulation, metabolic energy contributions, and work performance in hockey players.

It is therefore the purposes of this investigation to determine whether wearing hockey equipment affects: 1) the dissipation of metabolically produced heat, 2) the metabolic energy contribution during intermittent anaerobic work, and 3) the performance of an intermittent anaerobic work protocol designed to simulate the work patterns in a hockey game.

## Limitations and Delimitation

1. The intermittent exercise protocol was limited in its capacity to simulate the fluctuations in intensity, play length, and rest periods experienced during a regular hockey game. The physiological responses to the work performed were therefore limited in the extent to which they reflected physiological responses to an actual hockey game.
2. Exercise was performed on a modified Monark 860 cycle ergometer (Somerville & Quinney, 1987) which is somewhat dissimilar to playing hockey. Cycling is not weight-bearing, uses different movement patterns, requires no upper body movement, and does not provide the convective air currents experienced during the motions of playing hockey. However, cycling is a reasonable simulation of skating and quantification of work performed can be obtained. Furthermore, a fan was directed on the subject while cycling to simulate the air flow a moving skater would experience and exercise was performed in an environmental chamber maintained at a temperature similar to an ice arena.

3. The physiological responses were also limited because testing was conducted without the presence of teammates, coaches, opponents, officials, and spectators, which may alter the emotional response mediated by the neural and endocrine system.
4. Percent change in blood volume was measured indirectly from changes in hematocrit and haemoglobin.

## Methods

### Subjects

Seventeen members of the University of Alberta Golden Bears Hockey team gave informed consent and volunteered to participate as subjects. All procedures received prior approval from the Committee on Research Involving Human Subjects at the University of Victoria. Results from two subjects were excluded as one subject withdrew for medical reasons and another declined further participation after completing one experimental session.

### Experimental Design

Data were collected while subjects performed an exercise protocol designed to simulate a hockey game under each of two conditions. One condition involved wearing a standardized set of hockey equipment (EQ): sports socks, full-length underwear, athletic support cup, shinguards, hockey socks, elbow pads, gloves, helmet with face visor, CBS Pro Upper Body Protector (Cooper Canada Limited), CG Pro Cooperall Girdle (Cooper

Canada Limited), breezer shell, practice sweater, and shoes. The second condition consisted of the same exercise protocol without equipment (NE). Subjects wore shorts, ankle socks, and shoes. Random assignment of conditions resulted in eight subjects completing NE first and seven subjects completing EQ first.

Testing began two months after the beginning of the 1989 hockey season. The two testing sessions were separated by less than seven days for thirteen subjects and five to eight weeks for three subjects due to involvement in a hockey tournament. Experimental sessions commenced at the same time of day to eliminate any differences due to circadian rhythms (Deryagina & Kraevskii, 1983) with the exception of two subjects who began NE six and nine hours later than the commencement of EQ. Subjects were asked to consume two litres of water and no alcohol on the preceding day and refrain from heavy food consumption, coffee, smoking, and formal exercise for four hours prior to data collection.

Subjects reported to the laboratory, inserted a rectal thermocouple to a depth of 12.0 cm, and were weighed ( $\pm$  0.1 kg) while wearing shorts and socks. Skin thermocouples were taped to the forehead, chest, back, upper arm, anterior thigh, and lateral calf. A tympanic thermocouple was inserted into the left ear (see temperature instrumentation). The subject was also fitted with a PE3000 Sport Tester (Polar Electro Ltd., Kempele, Finland) to

record heart rate every five seconds throughout the experimental session. The reliability and validity of these monitors has been previously established (Leger & Thivierge, 1988).

The subject rested in a chair in the laboratory at an ambient dry bulb temperature ( $T_{db}$ ) of 21.3 °C and wet bulb temperature ( $T_{wb}$ ) of 13.4 °C for five minutes while resting body temperatures and heart rates were recorded. Oxygen consumption was measured at rest and averaged every 30 s by a calibrated Beckman Metabolic Measurement Cart (Beckman Instruments, Inc., Illinois). A finger prick blood sample was obtained for the determination of resting blood lactate, hematocrit, and haemoglobin (see biochemical assays).

The subject was then dressed in hockey equipment, re-weighed (EQ only) and entered the environmental chamber ( $T_{db}=10.3$  °C,  $T_{wb}=6.8$  °C). After a five minute warmup at a resistance of 0.045 kg:kg body weight<sup>-1</sup>, an intermittent work protocol designed to simulate playing a hockey game was performed on a modified cycle ergometer (Somerville & Quinney, 1987) further modified for the quick application and removal of resistance and equipped to count pedal revolutions every five seconds to an accuracy of 0.2 revolutions. As illustrated in Figure 1, two 15 s maximal sprints (sprint one and sprint two) were performed at a resistance of 0.09 kg:kg body weight<sup>-1</sup> (Bar-Or, 1987) separated by 15 s of controlled cycling at 50 rpm and 0.045

kg·kg body weight<sup>-1</sup> to complete one 45 s workout or hockey "shift". Six workouts were performed, each followed by 255 s of rest, to complete one hockey "period". Three "periods" of six workouts were performed to complete a hockey "game" and each workout was designated by its "period" number and its location within the "period" (e.g. first workout of the three "periods" are 1:1, 2:1, and 3:1, in that order). The "periods" were separated by 15 min "intermissions" during which the subject rested in the laboratory ( $T_{db}=21.3$  °C,  $T_{wb}=13.4$  °C) outside the environmental chamber without helmet and gloves. The first workout of the second and third "periods" was preceded by a two minute controlled warmup at 50 rpm and a resistance of 0.045 kg·kg body weight<sup>-1</sup>.

A fan directed air onto the subject at 3.3 m·s<sup>-1</sup> during each workout to simulate the approximate air flow experienced by a moving skater. Water chilled to 10 °C was permitted ad libitum throughout the testing session. The subject was cautioned against heavy water consumption early in the first "period" as this seemed to enhance feelings of nausea. A second chilled water bottle was available for the subject to rinse his mouth. Only the volume of water ingested was measured. Wiping of sweat was not permitted except when the eyes were irritated. The subject was re-weighed while wearing equipment (EQ) and in shorts and socks before removal of the rectal probe (EQ and NE) at the conclusion of the third "period".

# TIME-COURSE OF ONE EXPERIMENTAL SESSION

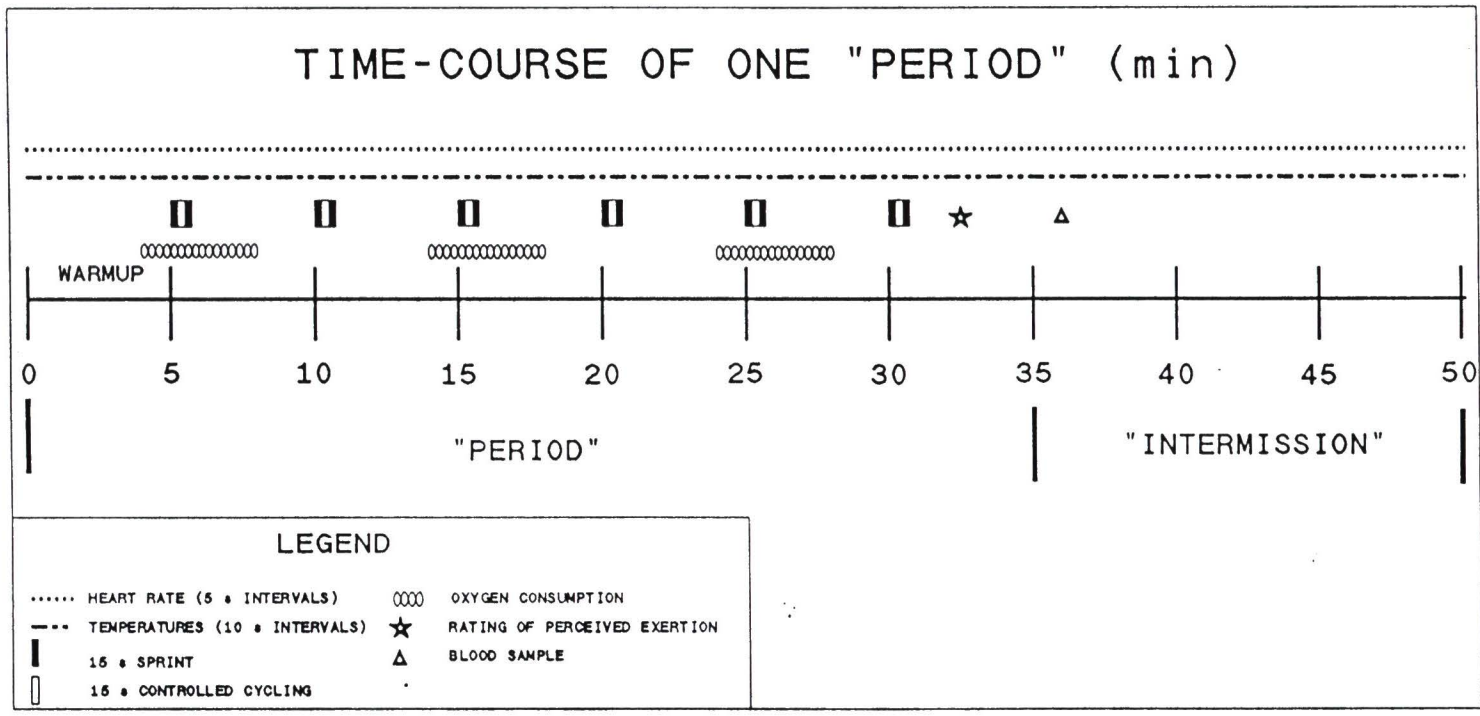
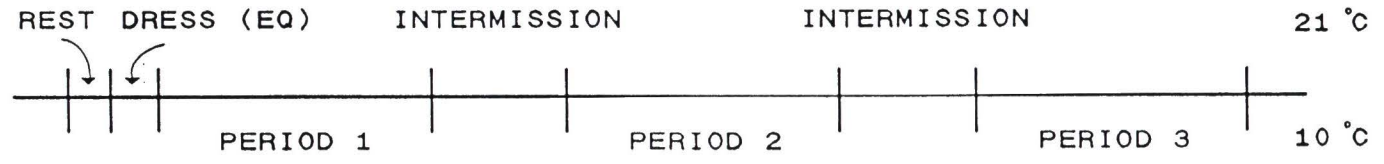


Figure 1. Time-course of the experimental protocol.

The dissipation of metabolically produced heat was evaluated by its effect on skin and core temperatures recorded every 10 s, heart rate recorded every 5 s, body weight, corrected weight (final weight - weight of water consumed), equipment weight (EQ only), and total water consumption. The metabolic energy contribution during intermittent anaerobic work was inferred from oxygen consumption averaged every 15 s for 60 s before, 45 s during, and 135 s after the first, third, and fifth workout of each "period" and blood lactate values obtained one minute after the conclusion of each "period". The variables calculated or measured to determine the performance of intermittent anaerobic work were: work performed during the two 15 s sprints and the intervening 15 s controlled cycling of each workout, the peak 5 s power output for each workout, and, at the conclusion of each "period", ratings of perceived exertion obtained from the Borg scale (Borg & Linderholm, 1967) with the descriptors used by Monahan (1988).

### Physiological Testing

Anthropometric and fitness characteristics of the subjects are presented in Table 1. Somatotype (Heath & Carter, 1967) was measured as this characteristic has been shown to influence heat dissipation (Docherty et al., 1986; Hayward et al., 1986). Maximum anaerobic power output was

determined during a 30 s Wingate test on a modified Monark cycle ergometer (Somerville & Quinney, 1987) at a resistance of 0.09 kg kg body weight<sup>-1</sup> (Bar-Or, 1987). Flywheel revolutions were determined for each 5 s interval and the 30 s mean and 5 s peak power outputs were calculated. Peak oxygen consumption (peak  $\dot{V}O_2$ ) was measured during a progressive cycle ergometer test to exhaustion. The following asymptotic protocol was used to determine the power output that would elicit peak  $\dot{V}O_2$ : subjects cycled at 120 W for two minutes, 180 W for the next two minutes, and at power outputs that increased by 30 W for each of the next four minutes and by 15 W for each additional minute until exhaustion. Metabolic measurements were taken and averaged every 30 s by a calibrated Beckman Metabolic Measurement Cart (Beckman Instruments, Inc., Illinois). Heart rate was measured during the last 15 s of each minute with a Sport Tester PE3000 (Polar Electro Ltd., Kempele, Finland). Ratings of perceived exertion (Borg & Linderholm, 1967) with the descriptors used by Monahan (1988) were obtained immediately after the anaerobic and aerobic power tests.

Table 1.

Mean (SD) anthropometric and fitness characteristics of the subjects.

Age (years)	21.4	(0.5)
Height (cm)	179.3	(5.3)
Weight (kg)	82.5	(7.8)
O scale rating	4.5	(1.2)
Somatotype		
Endomorphy	2.9	(0.6)
Mesomorphy	6.0	(1.2)
Ectomorphy	1.7	(0.8)
Anaerobic Power		
30 s (kJ)	23.7	(2.9)
30 s (kJ·kg <sup>-1</sup> )	0.29	(0.03)
Peak 5 s ( $\bar{W}$ )	993.9	(152.5)
Peak 5 s ( $\bar{W}$ ·kg <sup>-1</sup> )	12.1	(1.6)
Perceived exertion (Borg)	17	(1)
Aerobic Power		
Peak $\dot{V}O_2$ (L·min <sup>-1</sup> )	4.5	(0.5)
(mL·kg·min <sup>-1</sup> )	55.4	(6.6)
Perceived exertion (Borg)	18	(1)
Maximum Heart Rate (bpm)	188	(9)
Power Output at peak $\dot{V}O_2$ ( $\bar{W}$ )	353.5	(36.0)

## Temperature Instrumentation

Core (rectal and tympanic), skin (calf, thigh, chest, back, arm, and forehead), and laboratory and environmental chamber wet and dry bulb temperatures were recorded every ten seconds ( $\pm 0.1^{\circ}\text{C}$ ) using an Iso-thermex Electronic Thermometer (Columbus Instruments International Corporation, Ohio) and IBM-PC computer. These measurements were made during rest and throughout the experimental session except when subjects were dressing in the hockey equipment (EQ) and during movement into and out of the environmental chamber. The skin and ambient temperature probes were constructed by soldering copper-constantan wire to small copper disks and gluing with epoxy. Skin probes were taped to the skin with waterproof tape and the same probe was always used for each measurement site. The tympanic probe was made by twisting fine copper-constantan wire together and covering the end with cotton wool. After passing through a foam Ear Plug (Safety Supply Canada) which sealed the ear canal, the wire was taped to the side of the head to prevent movement. The accuracy of all probes was determined in a stirred water bath from 0.5 to 45.0  $^{\circ}\text{C}$  to be within 0.1  $^{\circ}\text{C}$  of a mercury thermometer.

## Biochemical Assays

Capillarized blood was obtained from a finger prick and analyzed for lactate concentration using a YSI Model 27 Industrial Analyzer (Yellow Springs Instrument Co., Inc., Yellow Springs, Ohio). The analyzer was calibrated prior to each analysis with 5 and 10 mmol·L<sup>-1</sup> lactate standards. The initial drop of blood was discarded and the subsequent blood drop was analyzed immediately. All values were corrected for changes in blood volume determined from hematocrit and haemoglobin measures (Dill & Costill, 1974) obtained on blood samples collected at the same time as the sample for blood lactate. Hematocrit was determined by centrifugation in a high speed microhematocrit centrifuge and haemoglobin by the cyanmethemoglobin method with Drabkins Reagent (Sigma Diagnostics, St. Louis, Missouri).

## Statistical Procedures

Repeated measures analyses of variance (ANOVA) by condition and by time with post-hoc Scheffé multiple comparisons were performed on the following dependent variables: unweighted mean skin and rectal temperatures at the beginning and end of each "intermission" and at the beginning of alternate workouts; lowest "intermission" and recovery and peak exercise

heart rates for each workout; body weight before, after, and corrected for sweat loss; water consumption; peak exercise and lowest recovery oxygen consumption for alternate workouts; blood lactate after each "period"; work output for each 15 s of the workout alone and in combination with each of the other 15 s segments; total work performed in each workout; peak 5 s power output for each workout; and rating of perceived exertion after each "period". Blood lactate and exercise and recovery heart rates and oxygen consumptions were also compared when expressed as a ratio of the work performed in the respective "period" or workout to eliminate the effect of any differences in work performed between the two conditions. A probability less than 0.05 was deemed statistically significant.

## Results

### Thermoregulation

#### Temperatures

Temperatures were measured every 10 s at all sites. Statistical comparisons were conducted between the values recorded at the beginning and conclusion of rest and each "intermission" and at the beginning of the first, third and fifth workout of each "period".

Core Temperatures. Tympanic temperatures were not analyzed as a hesitancy of the subjects to insert the probe deep enough into the auditory canal resulted in the probe frequently acting as an additional skin probe rather than a measure of core temperature. The time-course response of rectal temperature for one subject is illustrated in Figure 2. Rectal temperatures (n=10) were not different between NE and EQ during rest but showed a condition by "period" interaction ( $p < .05$ ) resulting in the first "period" rectal temperatures being lower for both conditions than those in EQ during "periods" two and three. Measures in "periods" two and three of NE were also lower than "period" three of EQ.

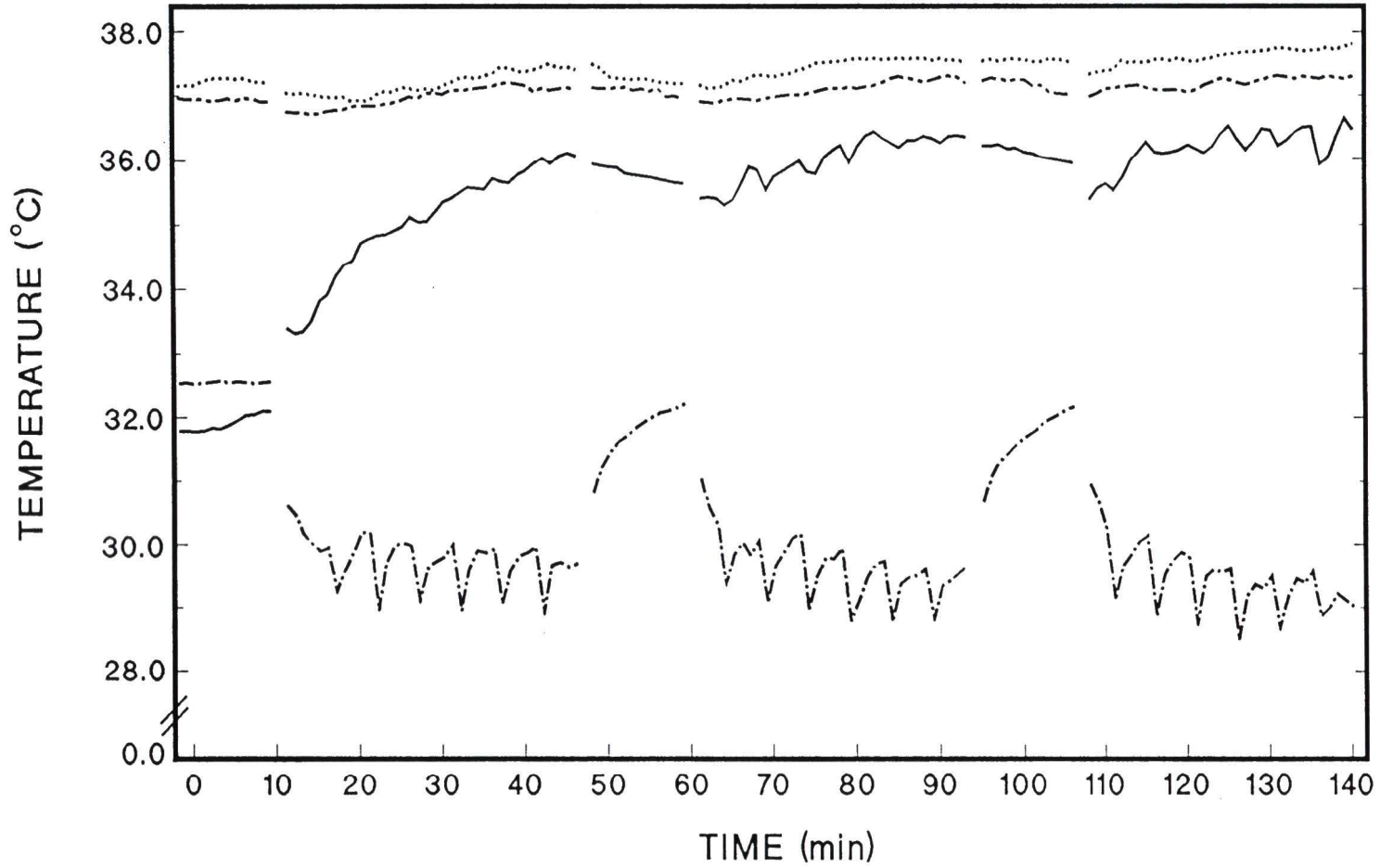


Figure 2. Rectal temperature in NE (-----) and EQ (.....) and mean skin temperature in NE (-.-.-.-) and EQ (——) for one subject.

Skin Temperatures. The unweighted mean skin temperatures for one subject are illustrated in Figure 2. Mean skin temperature (n=16) demonstrated a condition by time interaction ( $p < .05$ ). During EQ, "periods" two and three had higher mean skin temperatures than "period" one. Without equipment, skin temperatures decreased with time as "period" one had higher temperatures than "periods" two and three. During EQ, mean skin temperatures were greater than the resting values and the NE mean skin temperatures ( $p < .05$ ). When equipment was not worn, mean skin temperatures during the workouts and at the beginning of the intermissions were lower than the resting values ( $p < .05$ ). A measurement site by condition interaction ( $p < .05$ ) showed NE arm and chest temperatures to be lower than all other sites (Figure 3). Forehead temperature was not different between the two conditions.

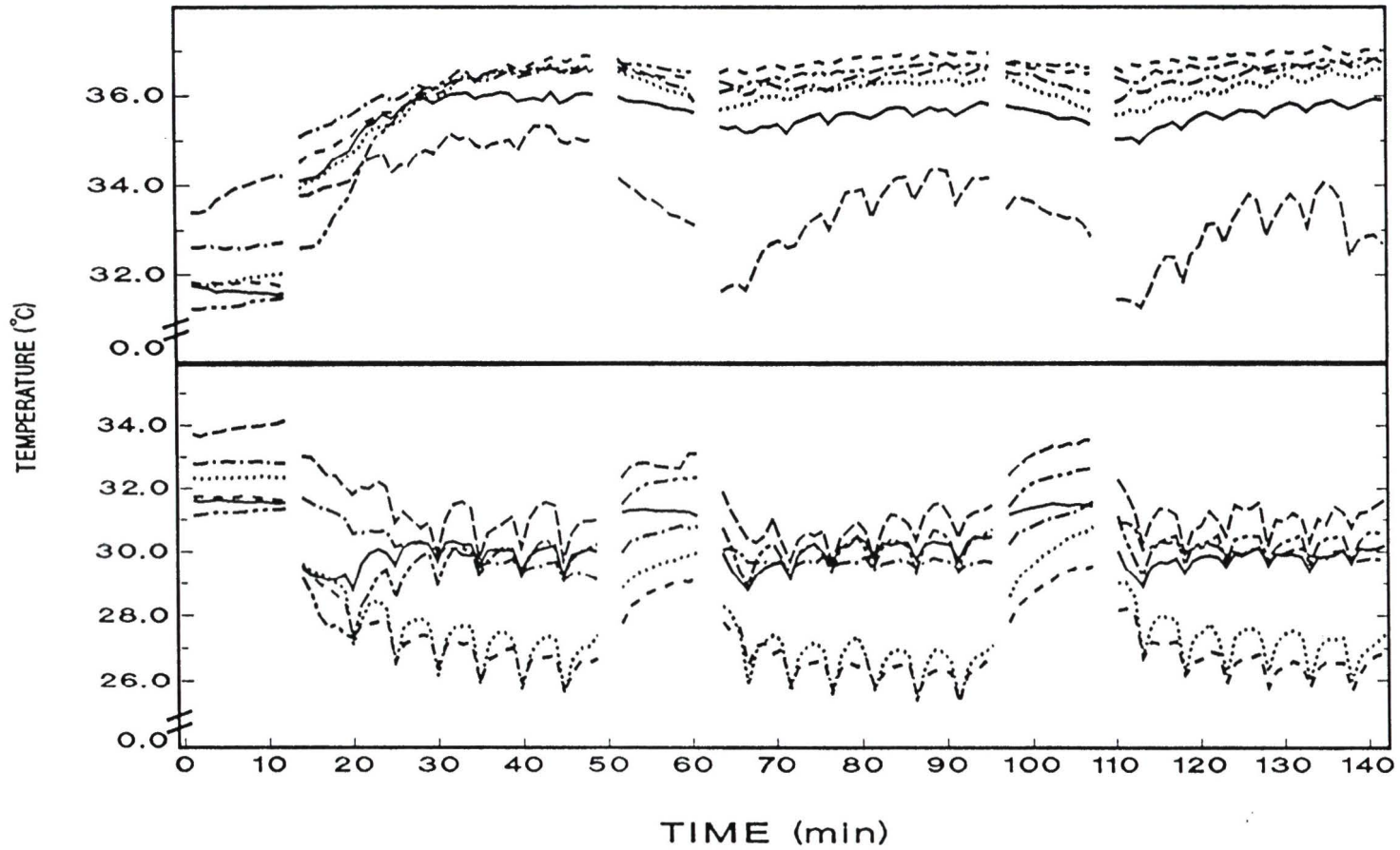


Figure 3. Calf (—), thigh (---), chest (.....), arm (-.-.-), back (-.-.-), and forehead (-.-.-) temperatures in NE (lower) and EQ (upper) for one subject.

### **Weight Changes**

Body weight and percent weight change ( $n = 15$ ) are shown in Figures 4 and 5. Initial, final, and corrected (final weight - water consumed) weights showed a condition by time interaction ( $p < .05$ ) as weight did not change during NE but decreased during EQ. Corrected weights were lower than initial and final weights for each condition. Final and corrected weights in EQ (undressed) were lower than in NE. Dressing in the 8.2 kg of equipment (EQ) increased the weight of the subjects 10% ( $p < .05$ ). More water was consumed during EQ than during NE ( $p < .05$ ) (Figure 6). The weight of sweat lost during EQ was greater than during NE and was also greater than the weight of the water consumed in either condition ( $p < .05$ ).

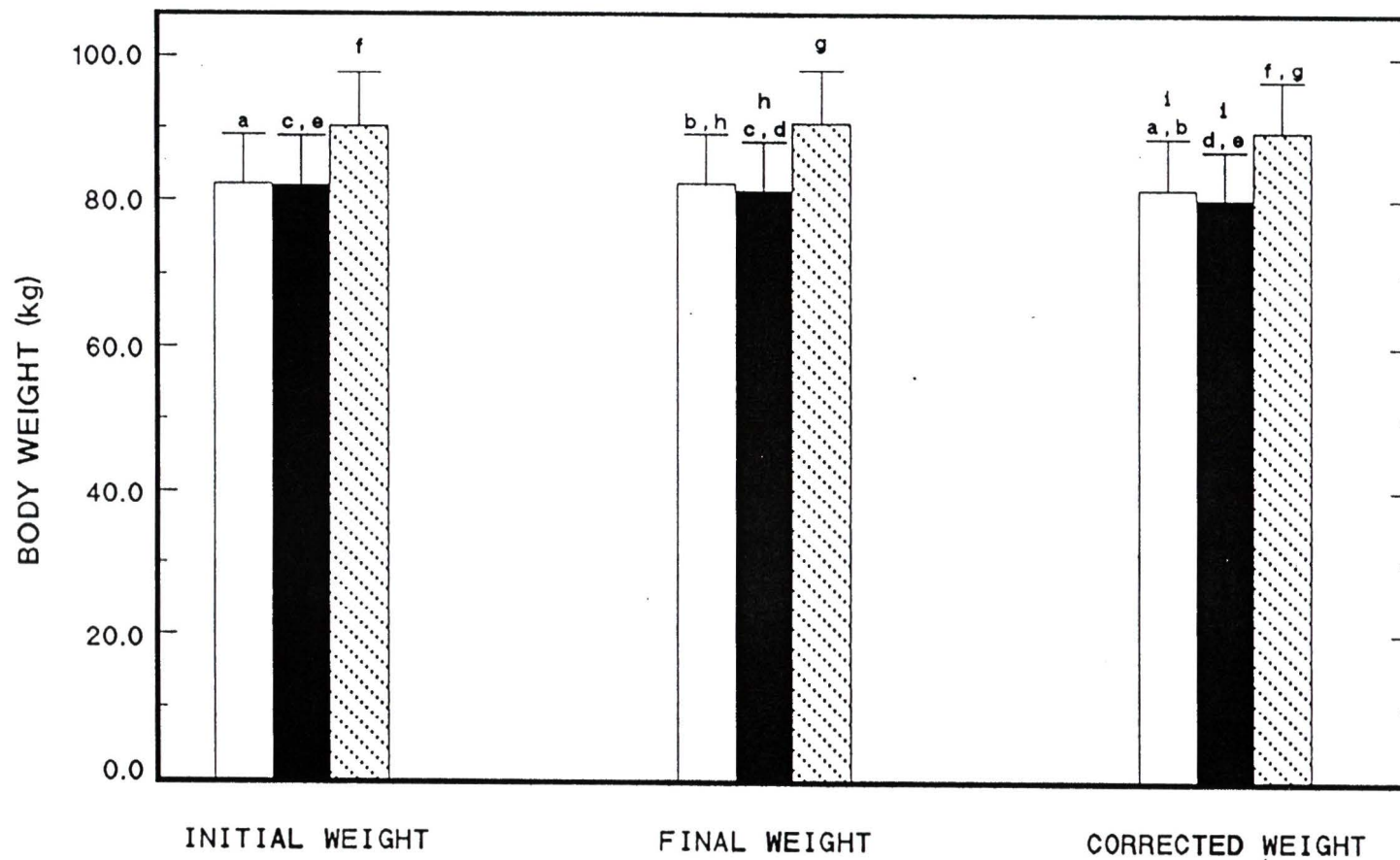


Figure 4. Mean (SD) body weight in NE (□) and EQ with (▨) and without equipment (■) measured before and after the experimental condition and corrected for sweat loss. Paired letters indicate significance ( $p < .05$ ).

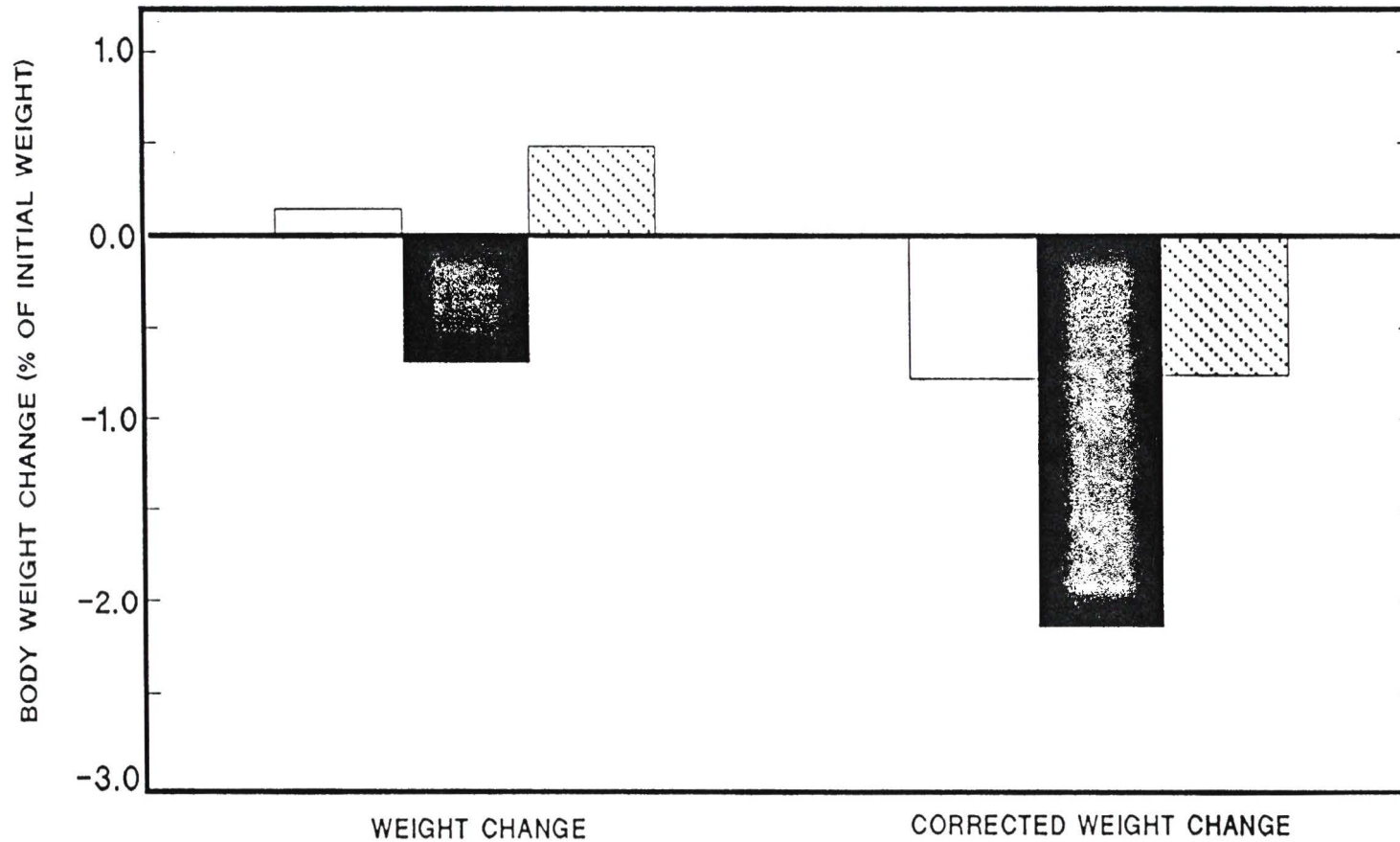


Figure 5. Mean body weight change and corrected weight change as a percentage of initial body weight in NE ( □ ) and EQ with ( ▨ ) and without equipment ( ■ ).

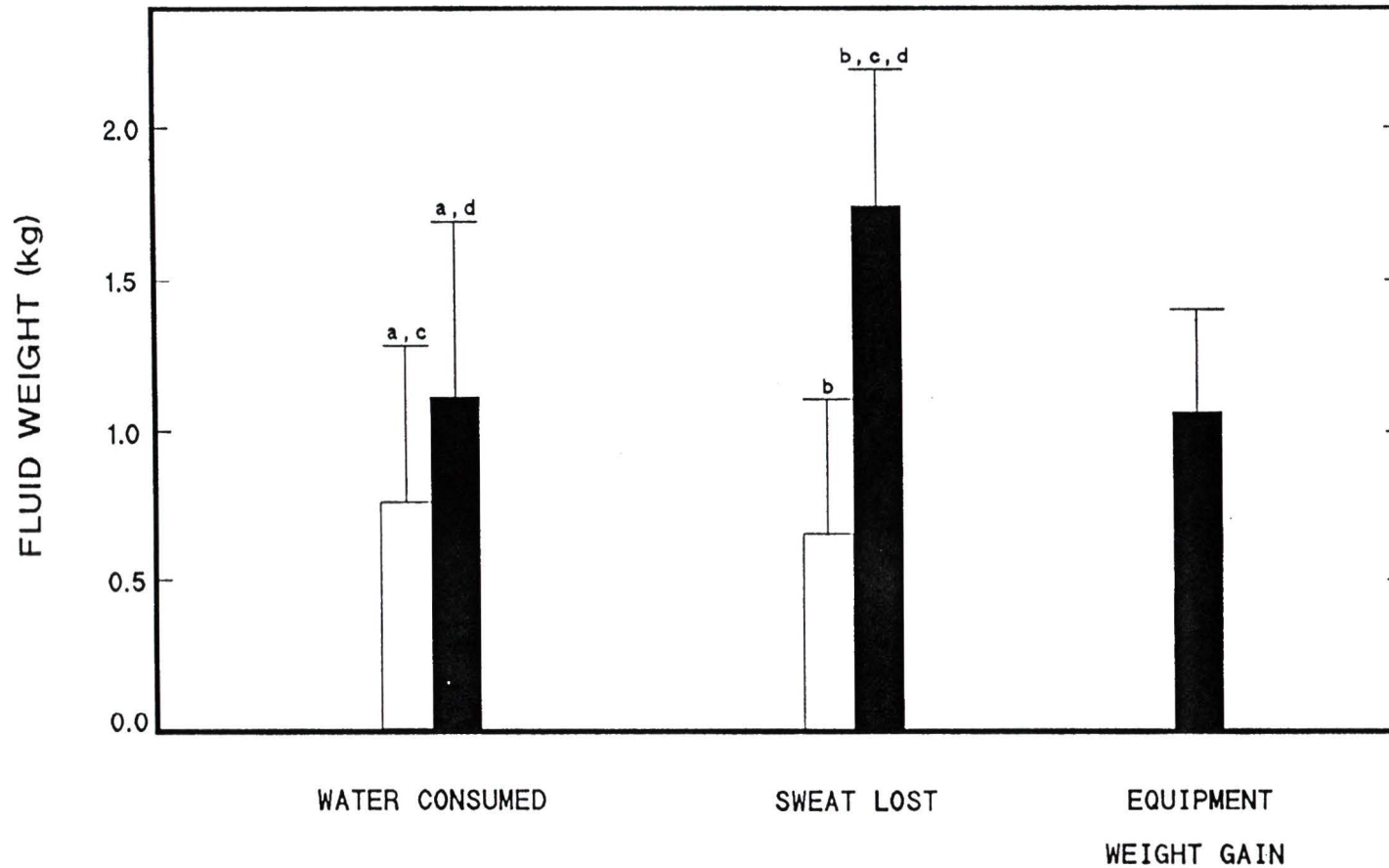


Figure 6. Mean (SD) weight of the water consumed and sweat lost in NE (□) and EQ (■) and the increase in weight of the hockey equipment in EQ. Paired letters indicate significance ( $p < .05$ ).

## Heart Rate

Although heart rate was recorded every 5 s, statistical comparisons were made only on the lowest value recorded at rest, during recovery from each workout, and during the "intermissions"; and on the highest heart rate recorded in response to each workout (n=13). The time-course of the heart rate response for one subject is illustrated in Figure 7. The "intermission", recovery, and exercise heart rates each showed an overall condition effect of being lower during NE than EQ ( $p < .05$ ) (Figure 8). When each "period" was analyzed separately, recovery and exercise heart rates were only lower during NE compared to EQ during the third "period" ( $p < .05$ ) (Figure 9). Exercise and recovery heart rates were also analyzed after being expressed as a ratio of the work performed in the respective workout to decrease the effect of any differences in work performed between the two conditions. Heart rates were again lower during NE than during EQ for both exercise and recovery ratios ( $p < .05$ ). Peak and low heart rates during exercise and recovery in NE were 89 to 91 and 50 to 61%, respectively, of maximum heart rates obtained during the peak  $\dot{V}O_2$  test. During EQ, exercise and recovery heart rates were 90 to 93 and 52 to 64% of maximum heart rates, respectively.

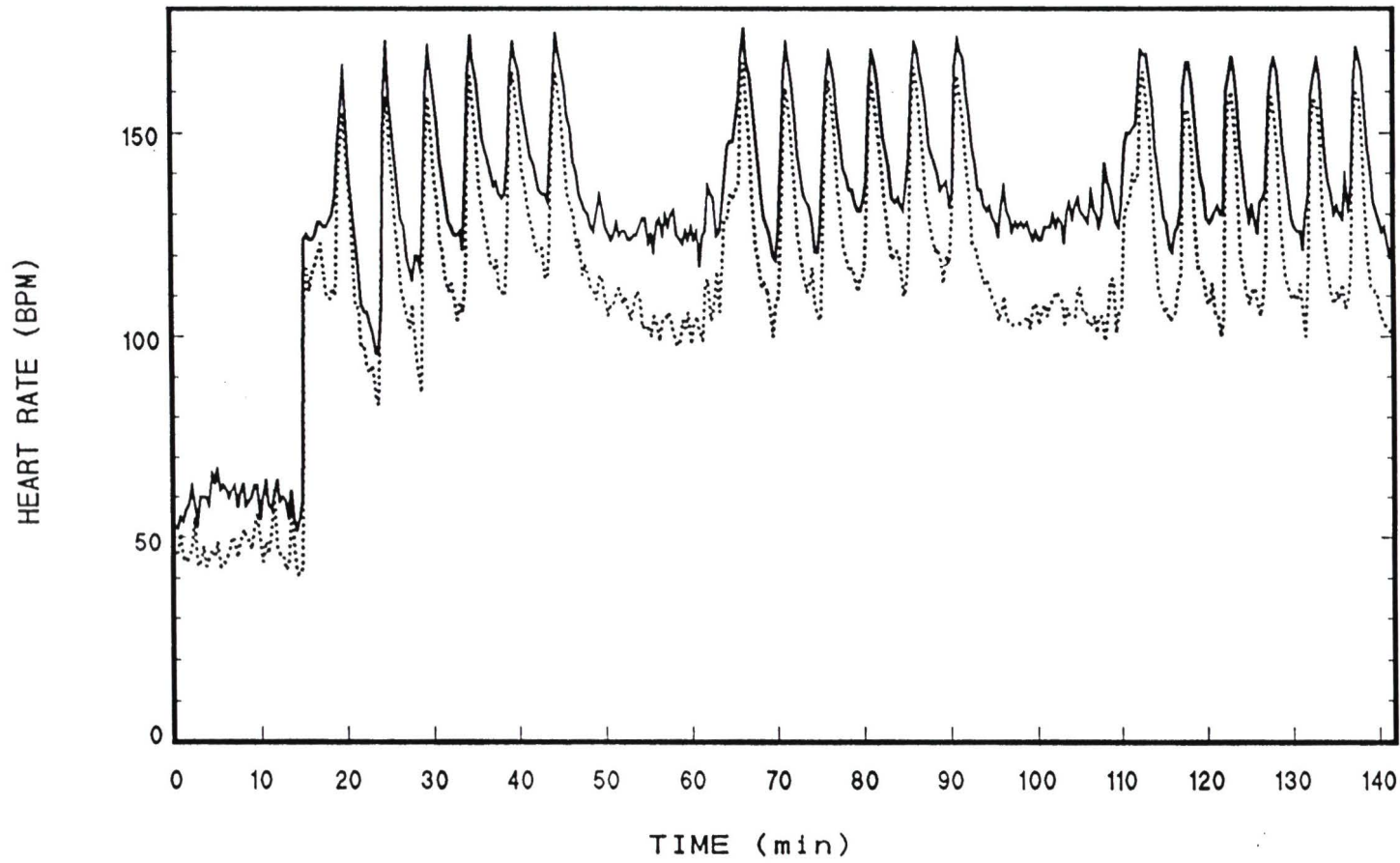


Figure 7. Time-course of the heart rate response for one subject. Heart rate is illustrated every 20 s in NE (.....) and EQ (—).

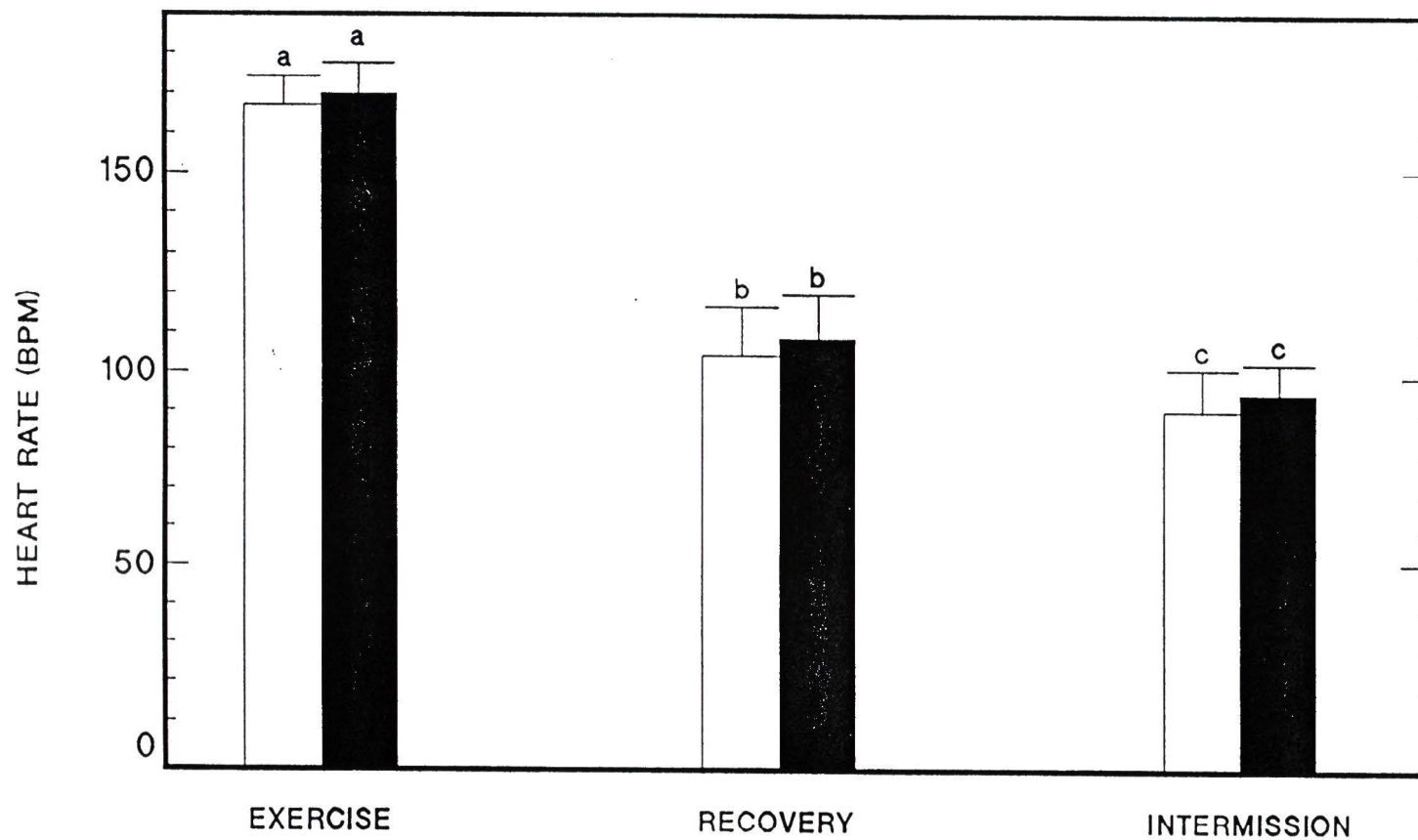


Figure 8. Mean (SD) peak exercise and lowest recovery and "intermission" heart rates for the simulated hockey game in NE (□) and EQ (■). Paired letters indicate significance ( $p < .05$ ).

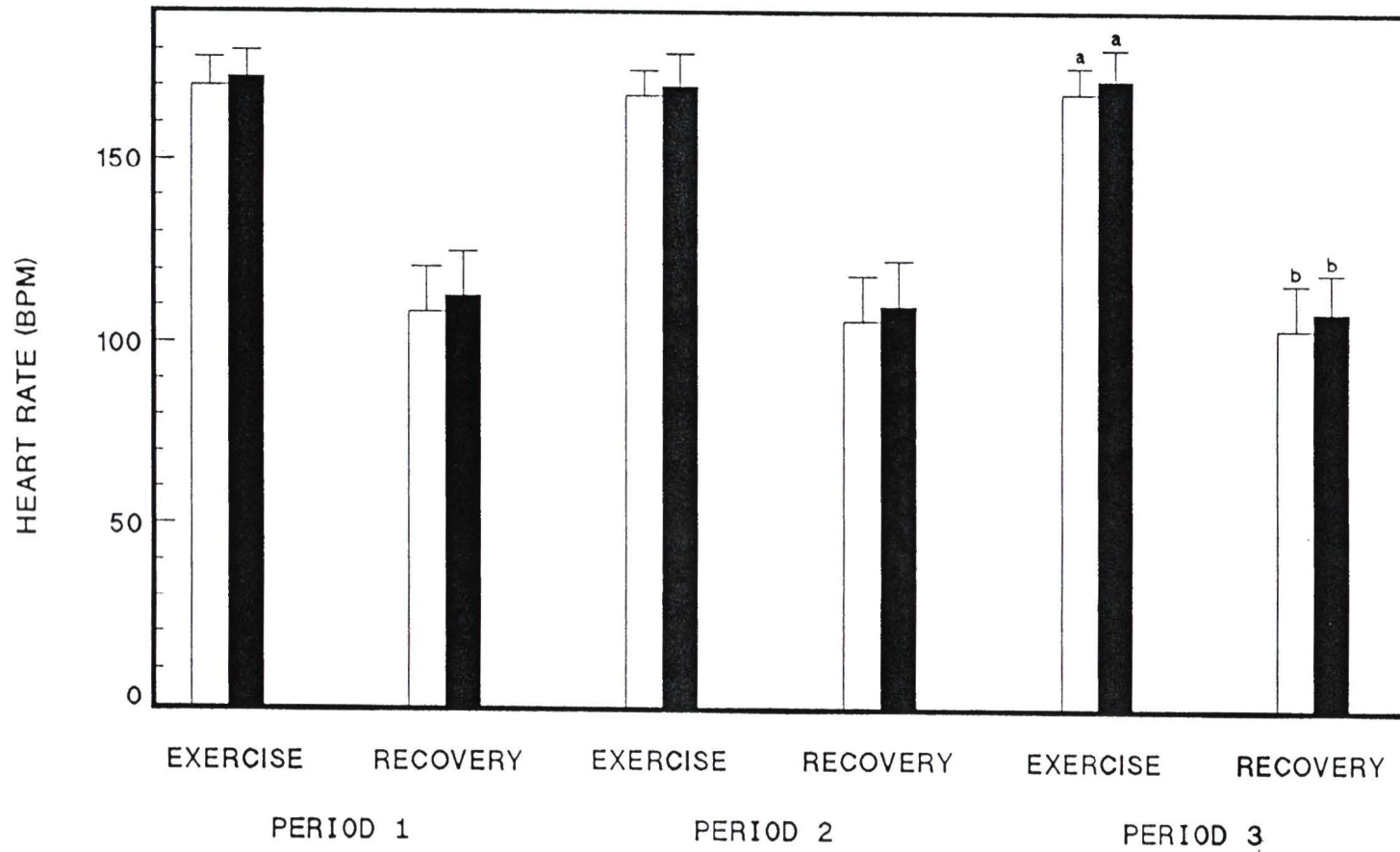


Figure 9. Mean (SD) peak exercise and lowest recovery heart rates for each "period" in NE ( □ ) and EQ ( ■ ). Paired letters indicate significance (p<.05).

## Energy Contribution

### **Oxygen Consumption**

Oxygen consumption was measured each 15 s for 60 s before, 45 s during, and 135 s after alternate workbouts within each "period" (Figure 10). Statistical comparison of the highest 15 s oxygen consumption, obtained during the second sprint, and on the recovery value recorded 135 s after the workout (n=13) showed no difference between NE and EQ in oxygen consumed at rest, during exercise, or during recovery. The exercise and recovery oxygen consumption values were also analyzed after being expressed as a ratio of the work performed in the respective workout to determine whether differences in work performance confounded the results. There remained no condition effect during exercise or recovery. Oxygen consumption was 89 to 102 and 18 to 23% of peak  $\dot{V}O_2$  during exercise and recovery, respectively.

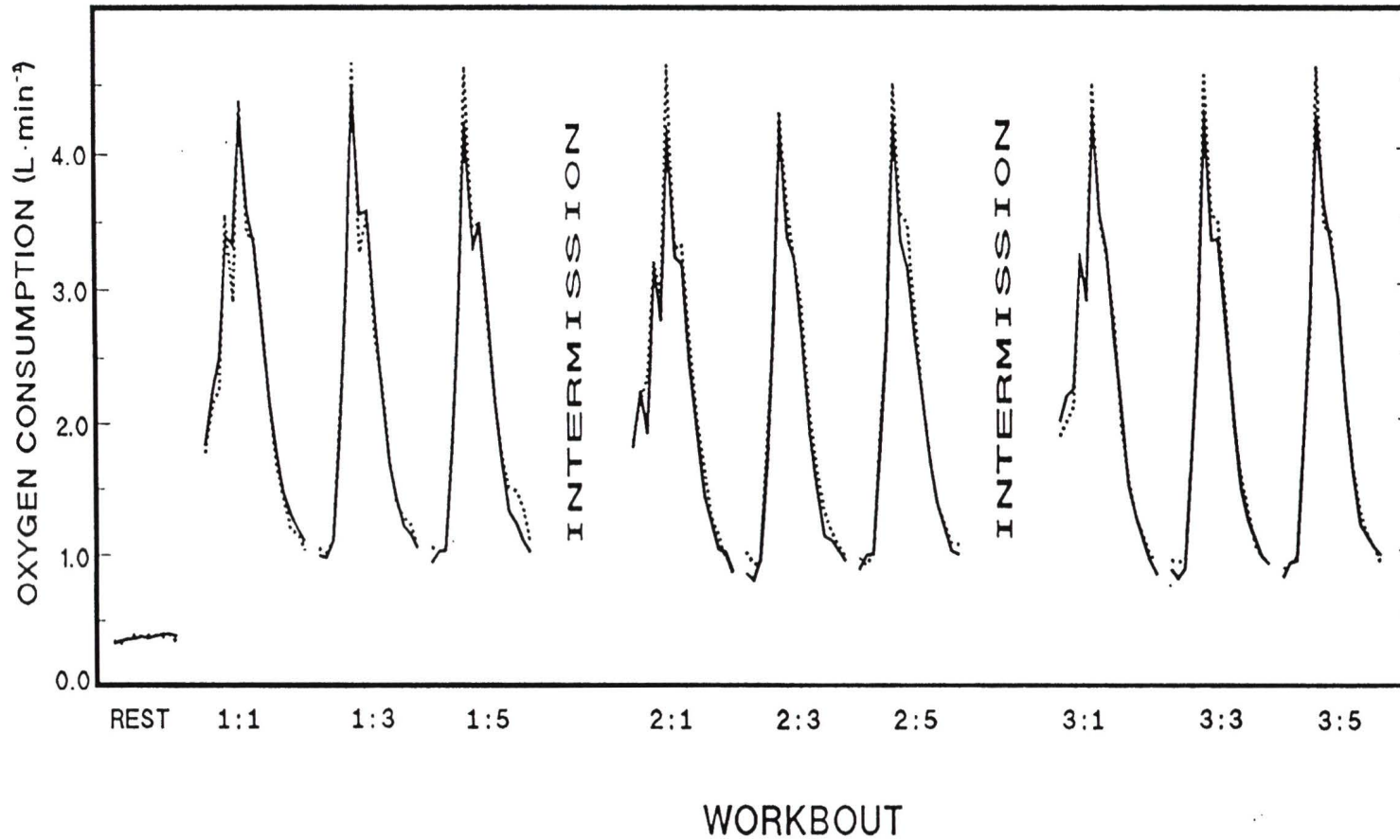


Figure 10. Time-course of oxygen consumption for one subject during alternate workbouts in NE (.....) and EQ (—).

**Blood Lactate**

Blood lactate concentration (n=15), corrected for changes in blood volume, showed an overall effect of being greater ( $p < .05$ ) during NE than during EQ (Figure 11). There was no difference between the two conditions when expressed as a ratio of the work performed during the preceding "period". Lactate concentrations after each period were higher than at rest ( $p < .05$ ) but there was no difference between the three "periods".

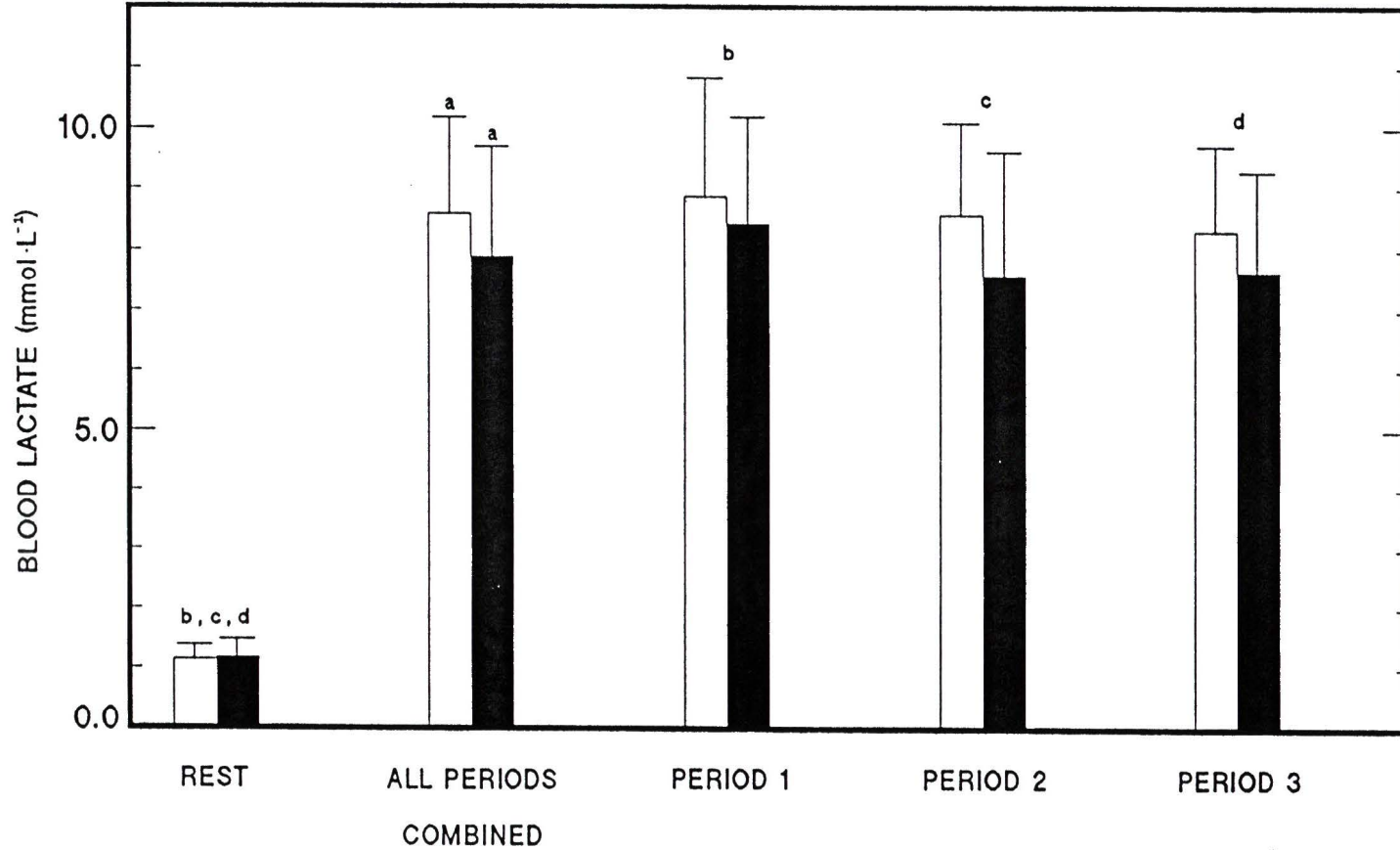


Figure 11. Mean (SD) blood lactate concentration at rest, for all "periods" combined and each "period" individually in NE (□) and EQ (■). Paired letters indicate significance ( $p < .05$ ).

## Work Performance

### Work

There were six 45 s workbouts in each of the three "periods" and each workout was comprised of an initial 15 s sprint followed by 15 s controlled cycling and a final 15 s second sprint (Figure 12). There was an overall condition effect ( $p < .05$ ) with more work ( $n = 14$ ) performed in NE than EQ during the first sprint of each workout (Figure 13). A similar effect was found for the first sprint in combination with either of the other two 15 s segments and the entire 45 s workout (Figure 14). When each "period" was analyzed separately, the first sprint showed no difference in "periods" one or three but more work occurred in NE than EQ ( $p < .05$ ) during "period" two. The complete 45 s workout was not different between the two conditions in "period" one but work performed in NE was greater than in EQ for both "periods" two and three ( $p < .05$ ). There was no difference between NE and EQ during the controlled cycling, second sprint, these two combined, or during the peak 5 s power output for each workout. Average power outputs during the workbouts were 136 to 163 and 128 to 161% for NE and EQ, respectively, of the average power output that elicited peak  $\dot{V}O_2$ .

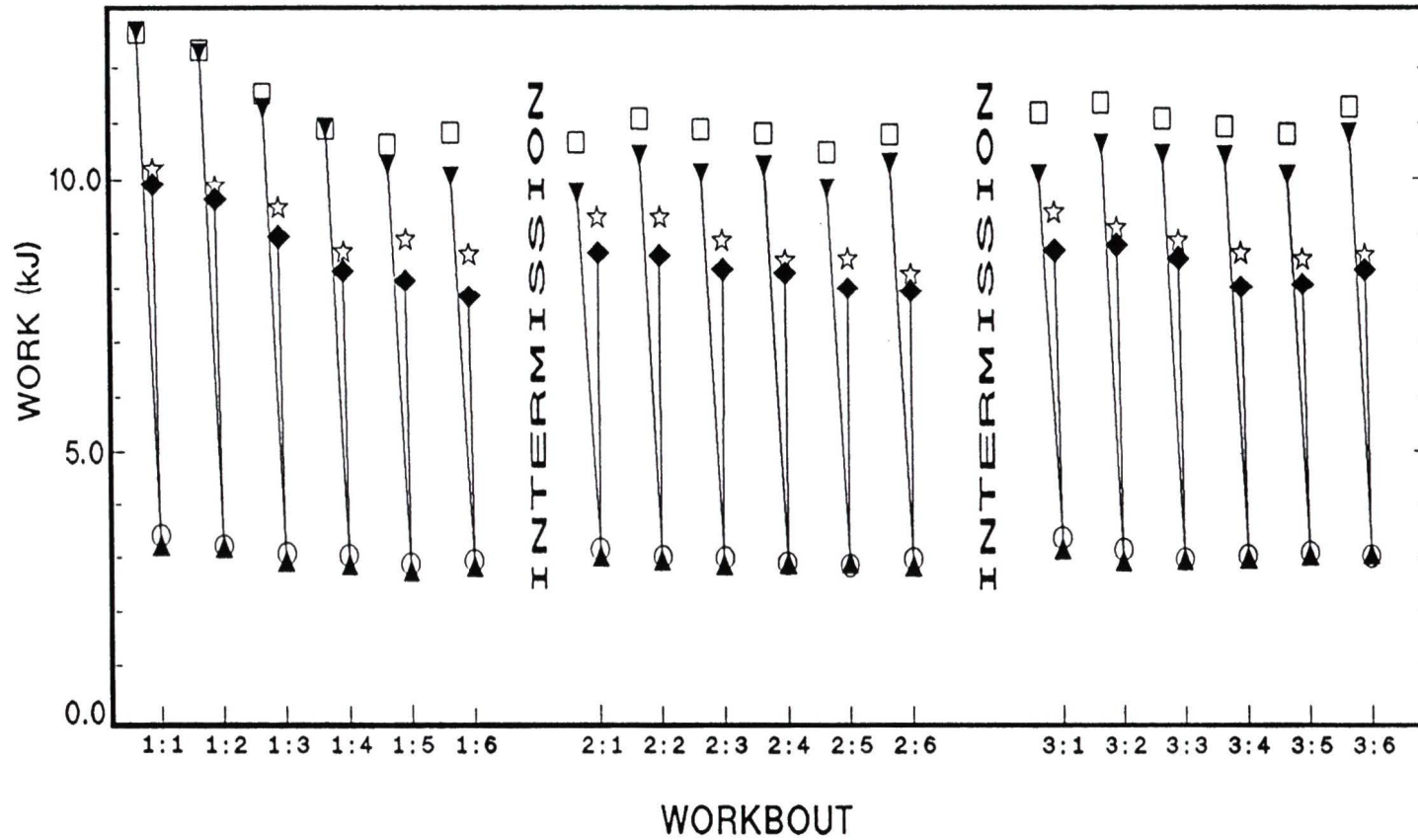


Figure 12. Time-course of mean work output for the first sprint (□, ▼), controlled cycling (○, ▲), and second sprint (☆, ◆) of each workbout in NE and EQ, respectively.

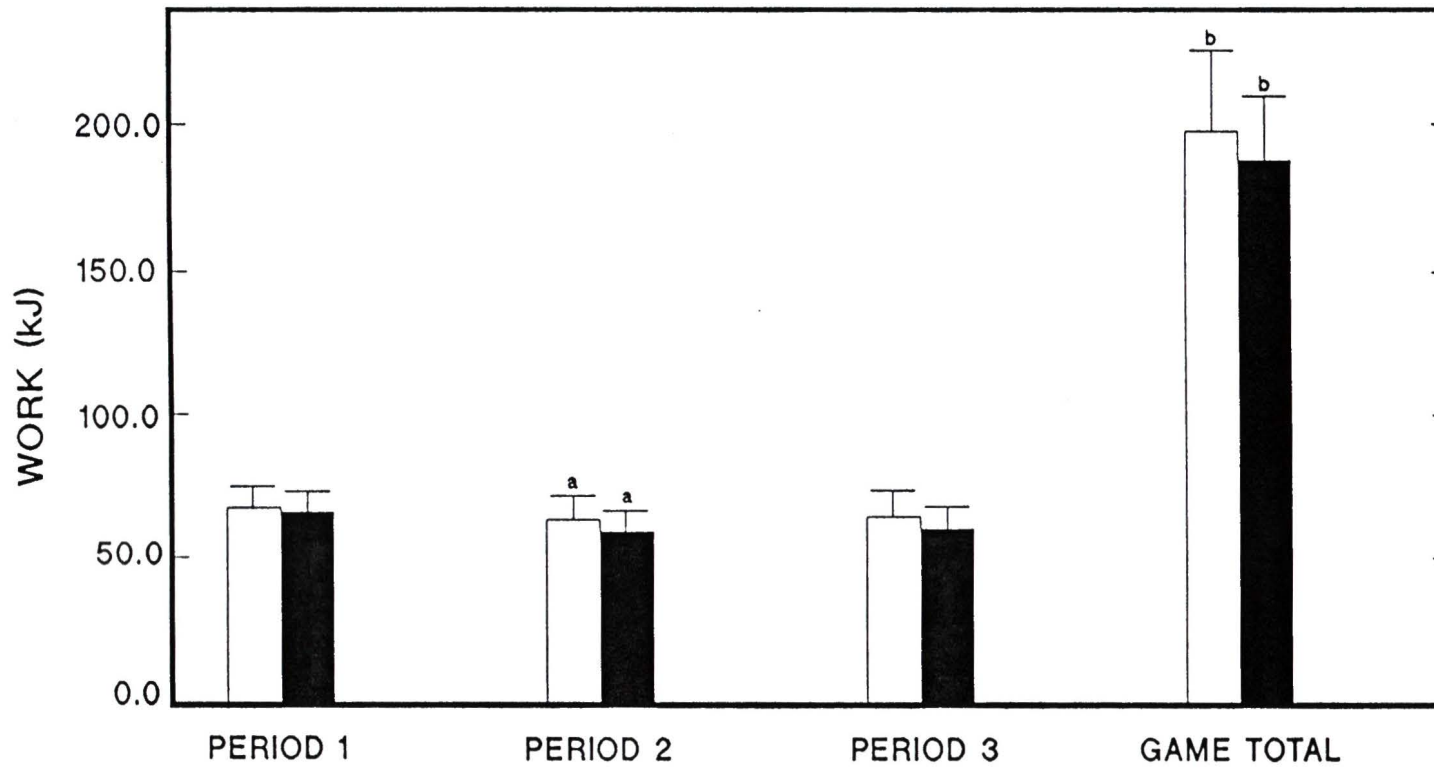


Figure 13. Mean (SD) work output during the first 15 s sprint of each workout for each "period" and the complete "game" in NE ( □ ) and EQ ( ■ ). Paired letters indicate significance ( $p < .05$ ).

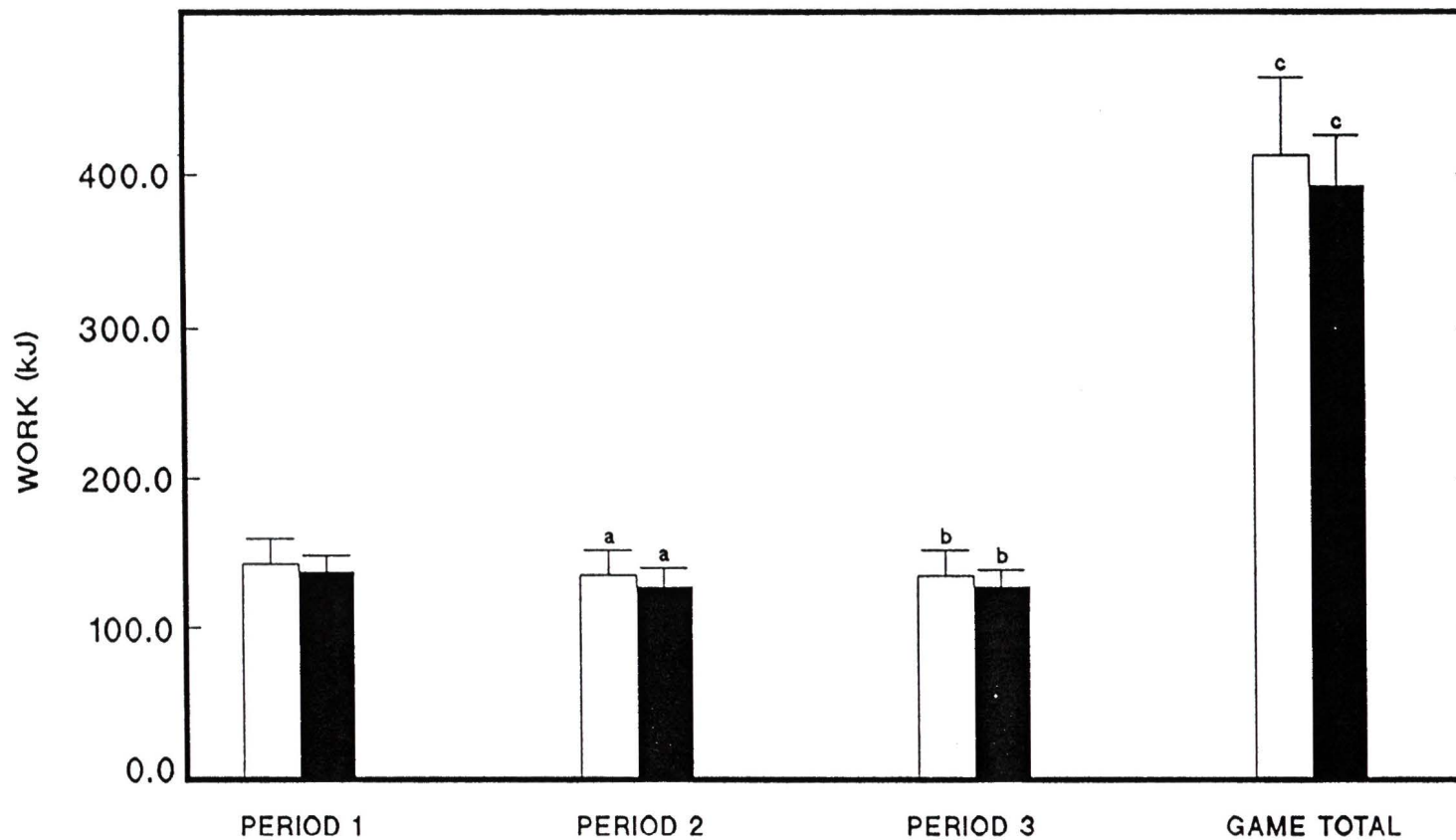


Figure 14. Mean (SD) work output during each "period" and the complete "game" in NE ( □ ) and EQ ( ■ ). Paired letters indicate significance ( $p < .05$ ).

**Perceived Exertion**

Ratings of perceived exertion ( $n=15$ ) were not different between the two conditions (Figure 15). When both conditions were analyzed together, "period" one received a lower rating than "period" three ( $p<.05$ ).

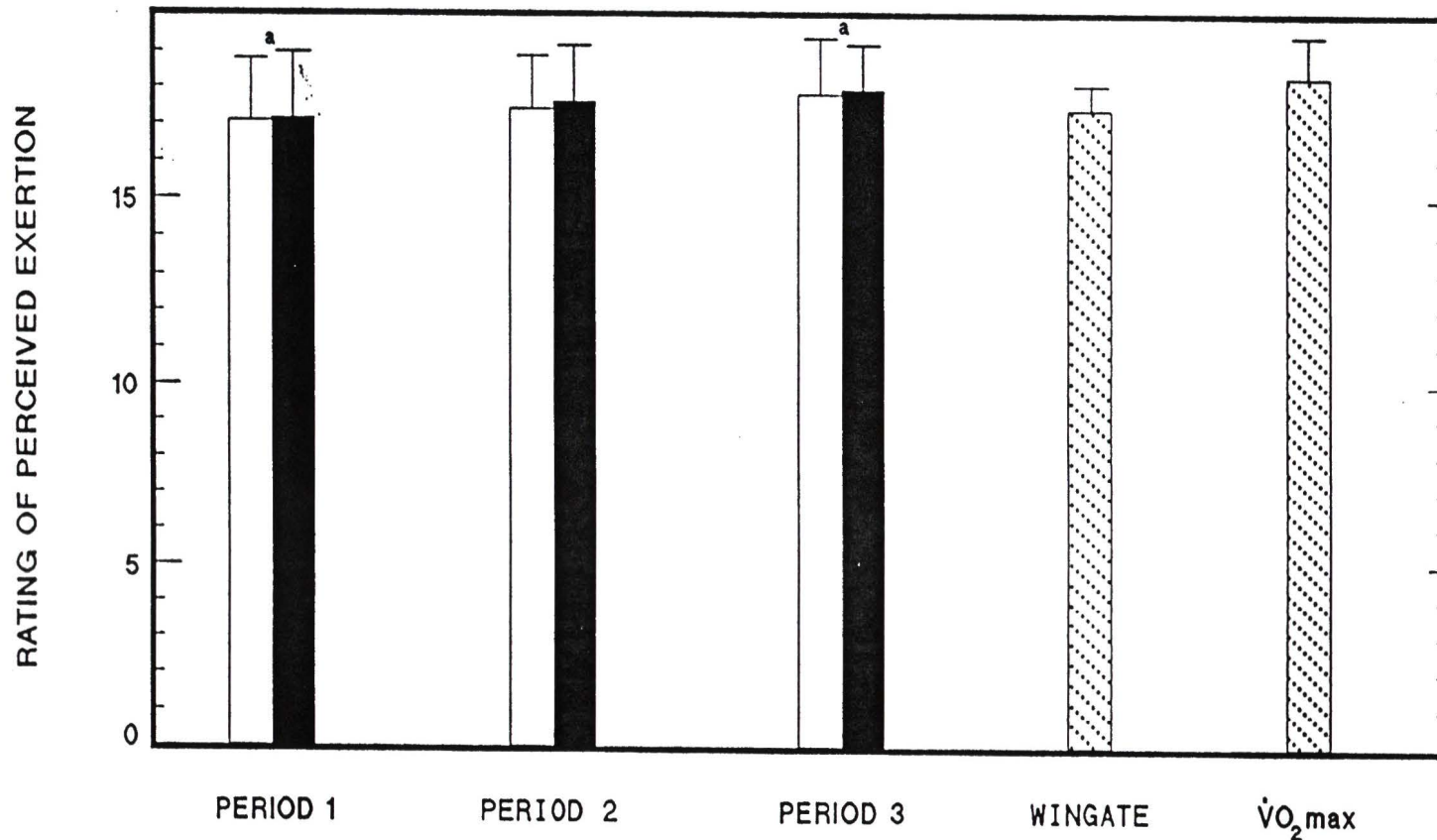


Figure 15. Mean (SD) rating of perceived exertion at the conclusion of each "period" in NE (□) and EQ (■) and after the physiological tests (▨). Paired letters indicate significance ( $p < .05$ ).

## Discussion

An intermittent exercise protocol designed to simulate a hockey game was performed with and without recently developed hockey equipment to evaluate thermoregulatory, metabolic, and work output responses to protective equipment. Core and skin temperatures, exercise and recovery heart rates, and weight loss were all greater and work output and blood lactate were lower when equipment was worn. Ratings of perceived exertion at the end of each "period" and oxygen consumption during exercise and recovery were not different between the two conditions.

Although ice hockey is played in cool (4 to 12 °C) ambient conditions, the present study would indicate that because of the equipment worn, players must dissipate, not conserve heat. Work output and, concomitantly, metabolically produced heat were lower in equipment. Therefore, the increased mean skin and core temperatures in equipment were likely due to the hockey equipment acting as a heat loss barrier, increasing body heat storage and evoking heat dissipation mechanisms while decreasing their efficiency (Goldman, 1968, 1969; Mathews et al., 1969).

Skin blood flow has been observed to increase in an attempt to dissipate heat (Wenger et al., 1975; Wyss et al., 1974). This may also have

contributed to the higher skin temperatures in the present study. Roberts and Wenger (1979) suggested that an increased skin temperature may result from increased capacitance in cutaneous veins to enhance heat transfer from the body core via the blood to the skin where it is transferred to the air by the evaporation of sweat. This peripheral vasodilation may partly account for the interaction between the skin temperature measurement sites and experimental condition. Aulick et al. (1981) have shown that the arms were used to dissipate vascular heat from the central circulation during leg exercise. Furthermore, the temperature of arterial blood in the arms was about 3 °C higher than vascular blood. In the present study, the temperature of the arm was the highest skin temperature when equipment was worn, indicating the use of the arms as a viable avenue for heat dissipation. Without equipment, arm and chest temperatures were lowest, likely due to either peripheral vasoconstriction in the nonworking limbs upon exposure to the cool ambient temperatures (Senay, 1972) or an increase in the core-to-skin thermal gradient which could decrease the peripheral blood flow necessary for heat dissipation (Strydom & Holdsworth, 1968). Forehead temperature was the lowest and highest of all skin temperatures with and without equipment respectively. There was no significant difference between the two conditions. The lower temperature of the forehead when wearing equipment could have been due to evaporation of sweat in this area as the forehead probe was closest to the

only exposed skin of the players. The maintenance of forehead temperature with different experimental conditions may also be important for the maintenance of brain temperature. Forehead temperature and sweat rate are maintained when sweat rate is decreased elsewhere in dehydrated subjects (Caputa & Cabanac, 1988) and performance is improved when the forehead is cooled by fanning (Hirata et al., 1987).

Subjects exhibited higher exercise and recovery heart rates when equipment was worn. This finding is similar to the higher heart rates observed in response to the performance of continuous work at constant or progressive workloads in high ambient temperatures (Claremont et al., 1975; Smolander et al., 1986; Williams et al., 1962) or while wearing a nylon shell (Powers et al., 1982), football equipment (Cooter et al., 1975; Mathews et al., 1969), or a suit perfused with warm water (MacDougall et al., 1974; Rowell, Murray, et al., 1969) compared to normal or cool conditions. Higher heart rates during thermal stress and work of progressive or low intensity (26 to 39% of  $\dot{V}O_2\text{max}$ ) were accompanied by a decreased stroke volume, increased or maintained cardiac output, decreased central blood volume, and decreased total peripheral resistance as blood was distributed to the periphery to dissipate heat (Rowell, Murray, et al., 1969; Williams et al., 1962). During more intense work (45 to 70% of  $\dot{V}O_2\text{max}$ ), higher heart rates and lower stroke volumes were also observed but the thermoregulatory vasodilation in

the periphery was decreased by an increased vasoconstrictor tone that was necessary to maintain a higher metabolic demand for oxygen transport (Hirata et al., 1983; Nadel et al., 1979; Rowell, Murray, et al., 1969). Heart rate and cardiac output were decreased while stroke volume and central blood volume increased when skin temperature was decreased to 26.9 °C by perfusing a suit with water at 10 °C (Rowell, Murray, et al., 1969). Thus, the higher heart rates observed in the present study during exercise and recovery in equipment were likely accompanied by decreased stroke volumes and a decreased peripheral resistance with increased skin blood flow. A decreased muscle blood perfusion may have also accompanied these circulatory changes, decreasing substrate supply, waste removal, and work output. Peripheral blood flow was likely lower during the condition without equipment, allowing an increased central blood volume, stroke volume, and muscle blood flow and lower heart rate.

Although work intensities in the present study were higher (128 to 163% of peak  $\dot{V}O_2$  workload) than those in the aforementioned studies, the short duration of the work interval may have decreased the duration of the vasoconstrictor response, allowing an increased peripheral blood flow to quickly resume after the workout in equipment, decreasing central blood volume and stroke volume, and resulting in the increased heart rate during recovery. As hyperemic blood flow in muscles after exercise has been shown

to be increased by the frequency or force of muscle contraction to a plateau at 40% of maximal voluntary contraction (Richardson & Shewchuk, 1980), this response should not have been different between the two conditions. However, this competition between muscle and periphery for blood flow when equipment was worn may have decreased muscle waste removal and substrate supply, decreasing work performance.

Heart rate may also have been increased by dehydration as more weight was lost when equipment was worn. Dehydration has been shown to decrease stroke volume and increase heart rate and body temperatures (Nielsen, 1986; Strydom & Holdsworth, 1968). Sweat was produced to evaporate and decrease the skin temperature (Goldman, 1968), increasing the core-to-skin temperature gradient (Sen Gupta et al., 1984), and decreasing the volume of blood in the peripheral circulation needed to dissipate the same amount of heat (Roberts & Wenger, 1979; Strydom & Holdsworth, 1968). Wearing football equipment has been shown to decrease the surface area from which sweat can evaporate directly from the skin and decrease the heat lost by evaporation as the point of evaporation was translocated from the skin to the equipment (Mathews et al., 1969). Therefore, the evaporation of sweat when equipment was worn was not as effective as exposed skin in 10 °C air in cooling the skin, decreasing the transfer of heat from the core. As a result, a stronger central drive to dissipate heat may have been evoked in equipment.

Weight lost due to sweating when equipment was not worn was 0.7 kg compared to 1.8 kg lost when equipment was worn. However, the equipment worn gained 1.1 kg. This resulted in less than 0.7 kg of evaporated sweat, as some dripping of sweat was observed, and decreased cooling due to the decreased effectiveness of the sweat that did evaporate. As a result, skin temperature was not decreased and skin blood flow was likely maintained at a higher level, increasing the stress on the cardiovascular system when equipment was worn (Roberts & Wenger, 1979).

The lower work output while wearing equipment during the "game" in the present study can be primarily attributed to less work being performed in the second and third "periods" of this condition. Evaluations of the effect of protective equipment or high ambient temperatures on work output have not been previously conducted using short, intermittent work at high intensity as in the present study. Studies are usually conducted with continuous work at fixed or increasing submaximal intensities and equated between the conditions under investigation to more clearly determine the physiological responses. However, when work output is allowed to vary, it has been shown to be lower during continuous exercise at 70% of  $\dot{V}O_2\text{max}$  in a suit perfused with water at rectal temperature compared to normal conditions and increased with the perfusion of water at 18 °C (MacDougall et al., 1974). Work duration was shorter in hot dry and hot humid conditions compared to comfortable

conditions at 65, 82, and 98 Watts (Sen Gupta et al., 1984). The number of hand-grip contractions at 20% of maximum that subjects could perform was decreased when esophageal temperature was increased 0.5 °C by leg immersion (Hirata et al., 1987). Conversely, cooling with trunk ice packs has been shown to increase work duration in running dogs (Kozlowski et al., 1985). Although the type of work was different, the decreased work output observed in the present study when wearing equipment is similar to the effect of increased body temperature on performance.

Sargeant (1987) found increasing muscle temperature resulted in an increase in both power output and rate of fatigue during a 20 s sprint. Muscle temperatures, although not measured in the present study, may have been higher in equipment since skin and core temperatures were higher than when equipment was not worn. This does not explain the lower work in the first sprint when equipment was worn. Also, the peak 5 s power output which occurred during the first sprint was not different between the two conditions. It is possible that differences in metabolic energy contribution, and not differences in muscle temperature may explain these results. Neither oxygen consumption nor blood lactate was different between the two conditions when analyzed in proportion to the work output for each condition.

The oxygen consumption during exercise was 89 to 102% of peak  $\dot{V}O_2$  in the present study. Sakate (1978) has described maximal oxygen

consumption as a quadratic function of ambient temperature. Other investigators have not addressed this equation (Sen Gupta et al., 1977, 1984; Smolander et al., 1986; Strange Petersen & Vejby-Christensen, 1973; Williams et al., 1962). However, the described relationships between oxygen consumption and ambient temperature are not in contradiction with this equation. Rowell, Brengelmann et al. (1969) discussed four factors that may explain this parabolic relationship: 1) an increased metabolic cost during work in the heat, 2) inadequate circulation to redistribute blood to meet the metabolic demand, 3) the increased mechanical efficiency of muscle at increased muscle temperatures decreasing oxygen consumption, 4) the  $Q_{10}$  effect. These factors were not all evaluated during the present study. The lack of change in oxygen consumption between the two conditions may have been the result of a complex interaction between all the factors associated with both an increase or decrease in oxygen consumption. According to the equation of Sakate (1978), wearing equipment would have to be equivalent to exercising at 38.9 °C for oxygen consumption to equal that consumed at 10.3 °C without equipment. As mean skin temperature was above 35 °C after the middle of the first "period" and above 36 °C after the middle of the second "period", the effective ambient temperature of wearing hockey equipment may not have been different from 38.9 °C. Roberts and Wenger (1979) and Nielsen et al. (1990) found exercising subjects at 40 °C to result

in mean skin temperatures of 36 and 35.2 °C, respectively. Therefore the difference in work performance between the two conditions cannot be attributed to differences in aerobic energy production.

During submaximal or progressive work in the heat or warm water perfused suits, blood lactate has been observed to be higher than during normal conditions (Claremont et al., 1975; Fink et al., 1975; MacDougall et al., 1974; Strange Petersen & Vejby-Christensen, 1973; Williams et al., 1962; Young et al., 1985). Also, no difference during incremental work at 25 or 40 °C has been observed (Smolander et al., 1986). The observation of higher than expected blood lactate has been attributed to decreased removal due to a decreased splanchnic blood flow, the  $Q_{10}$  effect on glycolysis, a decreased rate of lactate efflux (Young et al., 1985), and a decreased blood flow to the muscles increasing glycogen use and lactate production (Fink et al., 1975). In the present study, blood lactate was proportional to work output and was higher when equipment was not worn. It was not increased by working at increased body temperatures induced by wearing hockey equipment as would be expected from the other studies cited. Rather, if blood lactate can be taken to reflect the activity of the lactic energy system, this system would seem to account for the difference in work output. The disagreement with other studies may be due to the differences in work protocol. The short exercise duration and recovery time between workouts may have allowed adequate

blood flow for lactate removal and substrate supply when equipment was not worn. When equipment was worn, a greater proportion of the blood supply may have been redistributed to the periphery, possibly decreasing the removal of waste and supply of substrates. Skin temperature was higher in "periods" two and three than "period" one when equipment was worn, which may indicate greater peripheral blood flow during these "periods" when work output was decreased.

However, muscle blood flow was not elevated during submaximal (20 to 60%  $\dot{V}O_2$ max) work in the heat (40 °C) or a suit perfused with 45 °C water (Nielsen et al., 1990; Savard et al., 1988). It therefore seems unlikely that muscle blood flow would be compromised during recovery when equipment was worn. Savard et al. (1988) postulated that exercise that elicited maximal heart rate may compromise muscle blood flow; however, near maximal heart rates were only achieved for a few seconds, if at all, in the present study. Nielsen et al. (1990) felt a lack of sensitivity in the muscle blood flow measurement system may have masked any differences that did exist. However, they attributed a decrease in performance in the heat to a decrease in function in the motor centers or decreased motivation due to decreased central nervous system function at higher temperatures. Bruck and Olschewski (1987) found precooling to increase endurance time and concluded that increased body temperatures would increase negative feedback

in circulatory, thermal, and muscular discomfort, decreasing motivation and the drive to exercise. Similarly, a decreased interval time in the heat during the performance of spontaneous work and rest intervals was found to be associated with the quicker attainment of a critical heart rate (Vogt et al., 1983). As heart rates and body temperatures were higher when equipment was worn, circulatory and thermal discomfort may have decreased the drive to exercise in this condition.

Although work performance was different between the two conditions, ratings of perceived exertion were not. These ratings, obtained from the Borg scale, are somewhat related to heart rate when exercise is of medium intensity (Williams & Eston, 1989). Therefore, the high intensity of the work performed may have affected the accuracy of this measure. The values obtained may also have been affected by the increased heart rate due to thermoregulatory stress. This result underlines the need to assess protective equipment with physiological measures obtained in an environment that simulates sport competition.

In summary, wearing hockey equipment could elicit the following responses: Core and skin temperatures may be increased, possibly due to decreased heat dissipation while wearing equipment. Heart rates may be higher, possibly due to dehydration and the peripheral displacement of blood to conduct heat to the skin where it can be dissipated by evaporation. Weight

loss may be greater in equipment, but heat loss through sweat evaporation could possibly be less as most of the sweat produced may be absorbed by the equipment. Oxygen consumption may not be different between the two conditions as any possible decrease that may accompany a possible decreased work performance may be offset by the  $Q_{10}$  response. Although blood lactate concentration may be lower in equipment, as work output may also be lower, these values could be equal when normalized for work output. The peripheral displacement of blood possibly decreasing muscle blood flow, waste removal, and substrate supply may have attributed to any differences that may have occurred in work performance. The effect increased body temperatures could have on the central nervous system may also have been a factor. These responses could possibly have combined to decrease work output when equipment was worn. Therefore, it may be of an advantage for hockey players to obtain adequate protection from minimal equipment, remove equipment whenever possible, and ensure adequate hydration to prevent dehydration possibly hindering performance.

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## Literature Review

Whenever muscular work is performed, the hydrolysis of ATP that occurs to provide the necessary energy also releases heat (Hill, 1949; Wilkie, 1968). If work is continued for a period of time, the temperature of the exercising muscle increases in proportion to the intensity of the work performed (Aikas et al., 1962). The increase in muscle blood flow that occurs at the onset of exercise serves to remove much of the heat produced from the muscle to the body core. This prevents muscle temperature from reaching damaging temperatures and activates hypothalamic sensors which integrate the dissipation of heat from the body (Nadel, 1987).

The increased core temperature that accompanies exercise (Aikas, et al., 1962; Claremont et al., 1975; Goodman, et al., 1985) serves to initiate sweating which cools the skin through evaporation (Roberts & Wenger, 1979). This results in an increase in the temperature gradient from the core to the periphery, allowing heat to be conducted by the blood down the gradient to the skin (Aulick et al., 1981; Davies, 1979, Nadel, 1987). An increase in the capacitance of cutaneous veins occurs as body temperature increases, decreasing flow velocity in these vessels, and allowing an increased heat transfer to the skin for dissipation (Roberts & Wenger, 1979). This causes the

displacement of blood to the periphery, decreasing central blood volume and stroke volume and increasing heart rate (Rowell, Murray, et al., 1969). The volume of displaced blood in the periphery and its effect on the cardiovascular system is a function of the work intensity, ambient temperature, relative humidity, and hydration state of the subject.

At high work intensities, there is no major redistribution of blood to the skin as the high demand for oxygen transport is maintained at the expense of thermoregulation (Hirata et al., 1983; Nadel et al., 1979; Rowell, Murray, et al., 1969). When ambient conditions are cool and dry, a greater core-to-skin temperature gradient can be maintained and less peripheral blood flow is necessary to conduct heat to the skin. If combined with high ambient temperatures, the duration of intense work is reduced as body temperature rapidly obtains a critical level due to the decreased heat dissipation (MacDougall et al., 1974; Sen Gupta et al., 1984). Moderate work intensities in the heat are associated with an increased heart rate, increased cardiac output, decreased stroke volume, decreased central blood volume, and a decrease in total peripheral resistance compared to work in cooler ambient conditions (Rowell, Murray et al., 1969; Williams et al., 1962).

The volume of blood displaced is partially dependent on the relative humidity. In low humidity, sweat evaporates freely, cooling the skin, and decreasing the skin blood flow necessary to transfer heat from the body core.

In high humidity, sweat evaporation is hampered and the skin is not effectively cooled. A higher skin blood flow must therefore be maintained to conduct heat from the body core (Roberts & Wenger, 1979). Similarly, when dehydrated, the body conserves fluid, decreasing the rate of sweat production and, therefore, the cooling that can be obtained. Conversely, a well hydrated subject can sweat sufficiently to decrease skin temperature, decreasing the peripheral blood flow required (Strydom & Holdsworth, 1968). Therefore, increased work intensity, ambient temperature, relative humidity, and dehydration will each increase the thermal stress experience and, in combination, will have a multiplying effect.

The wearing of clothing or sporting equipment decreases the body surface area from which evaporation can occur directly from the skin (Goldman, 1968, 1969) and translocates the point of evaporation away from the skin, resulting in less heat being removed by any sweat that evaporates from the clothing (Mathews et al., 1969). Thus, heat dissipation is decreased and physiological responses similar to exercising in warm ambient temperatures with high humidity are observed. Wearing a nylon shell resulted in higher heart rates and rectal temperatures during exercise at 60%  $\dot{V}O_2$ max compared to when only shorts were worn (Powers et al., 1982). Wearing football equipment resulted in higher heart rates, rectal and skin temperatures, and weight lost due to sweating during 30 min of walking on a

treadmill compared to wearing shorts and a backpack the weight of the equipment or only shorts. However, the calculated heat lost due to sweating was less in the equipment due to decreased sweat evaporation and the translocation of the point of evaporation from the skin to the surface of the equipment (Mathews et al., 1969).

The increased sweat rate while wearing football equipment was an attempt to increase heat loss by evaporation. This would decrease skin temperature and increase heat dissipation so that work output could be maintained (Sen Gupta et al., 1984). However, the net result would have been a decrease in work output for two reasons: a portion of the excess sweat produced would have been absorbed by the equipment worn, increasing its weight which has been shown to increase sprint and anaerobic endurance times in hockey players (Montgomery, 1982) and, an increased fluid loss would increase dehydration, decreasing blood volume and increasing the osmolality of body fluids, resulting in increased peripheral vasoconstriction and body temperatures, and the inhibition of sweating, respectively. Therefore, work output would be decreased or terminated prematurely as body temperature increased (Nielsen, 1986).

Metabolic changes, which may or may not be associated with cardiovascular strain, are also observed during work in the heat. Maximal oxygen consumption was observed by Sakate (1978) to be a quadratic function

of ambient temperature at 50% relative humidity with the maximal value observed at 25.4 °C. Other investigators have not addressed this equation. However, the described relationships between oxygen consumption and ambient temperature can be explained by this equation (Sen Gupta et al., 1977, 1984; Smolander et al., 1986; Strange Petersen & Vejby-Christensen, 1973; Williams et al., 1962). The factors that account for this relationship may also contribute to the observation of increased (Consolazio et al., 1963; MacDougall et al., 1974; Nielsen et al., 1990), similar (Powers et al., 1982; Rowell, Brengelmann, et al., 1969), or decreased (Smolander et al., 1986; Strange Petersen & Vejby-Christensen, 1973; Williams et al., 1962) oxygen consumption during submaximal exercise in the heat or wearing a suit perfused with water at warm temperatures. Rowell, Brengelmann et al. (1969) assert that the differences observed may be due to differences in subject acclimatization, conditioning, and skill level. This latter study found the oxygen cost of sweating and increased cardiac frequency too small to measure and concluded that an increased mechanical efficiency in skeletal muscle, offset by the  $Q_{10}$  effect, resulted in the lack of difference they observed.

Improved performance with increased muscle temperature during 20 s work has been observed by Sargeant and Jones (1978) and Sargeant (1987). These authors found that by varying muscle temperature by immersion in

water baths maximal peak force and power were increased 2-10% per °C, depending on the pedal speed. However, as the rate of fatigue was also increased at the higher muscle temperatures, the extrapolation of these results to long term work is questionable.

The redistribution of blood to the periphery for heat dissipation has been cited as a possible explanation for decreased oxygen consumption during submaximal work in the heat due to a decreased perfusion of the working muscles (Sen Gupta et al., 1984; Williams et al., 1962). Work output was maintained through anaerobic metabolism and an increase in blood lactate was observed (Fink et al., 1975; Williams et al., 1962). However, leg muscle blood flow, lactate production, and oxygen extraction were not different during walking or cycling in the heat (Nielsen et al., 1990; Savard et al., 1988). Both increased (Fink et al., 1975) and no change (Young et al., 1985) in muscle glycogen utilization have been observed. The lack of sensitivity of the muscle blood flow measures (Nielsen et al., 1990) and differences in work intensity have been cited as possible explanations for the discrepancies. Savard et al. (1988) felt that muscle blood flow might be reduced to maintain blood pressure if maximal heart rate was reached. A decreased removal of blood lactate from decreased splanchnic blood flow (Rowell et al., 1968), alterations in neuromuscular recruitment in the heat, the  $Q_{10}$  effect on glycolysis, and differences in the availability and utilization of blood glucose

(Young et al., 1985) may have contributed to the differences observed.

The study of thermoregulatory responses to heat stress is usually conducted with continuous work at fixed or increasing submaximal intensities and equated between the conditions under investigation to more clearly determine the physiological responses. When work output is allowed to vary, it has been shown to be lower when body temperature is higher (Hirata et al., 1987; MacDougall et al., 1974; Sen Gupta et al., 1984). If the peripheral displacement of blood decreases blood flow in working muscles during work in the heat and an increase in anaerobic metabolism compensates to maintain work output, decreased work duration times in the heat could be explained by an increase in muscle lactate levels, as these have been associated with fatigue (Sahlin, 1986). The effect of heat on muscle metabolism may also limit endurance as higher body temperatures have been shown to shift the equilibrium between high-energy phosphate breakdown and resynthesis to lower values of ATP and CP (Kozlowski et al, 1985). The effect of an increase in body temperature on the central nervous system may also play a role. Nielsen et al. (1990) concluded that the termination of work in the heat may be associated with a reduction in the function of motor centers and the inability to recruit the motor units required for activity. Bruck and Olschewski (1987) found precooling to improve performance. They traced the temperature dependency of performance to circulatory, thermal and muscular

discomfort. Increasing body temperature was seen to increase the negative feedback from these factors, counteracting motivation, and reducing the drive to exercise. During the performance of spontaneous work and rest intervals at three different ambient temperatures, Vogt et al. (1983) observed decreased interval times in the highest ambient condition as the time to reach a critical heart rate was decreased due to the increased body heat load.

The effect of thermoregulation on sports performance has been primarily evaluated in endurance events, with little attention given to sports where activity is intermittent or variable in intensity. Cohen et al. (1981) found rectal temperatures to be elevated after rugby matches, attributable, in part, to the sweaters and socks worn. Fox et al. (1966) and Mathews et al. (1969) evaluated thermoregulatory responses to football equipment during continuous walking exercise on a treadmill while work output was controlled. The decreased evaporative surface increased body temperatures, heart rate, and weight loss due to sweating. Therefore, both intermittent activity and the protective equipment worn may influence the thermoregulatory responses of sports participants.

MacDougall (1979) has discussed thermoregulation in ice hockey and concluded that performance could be affected. Ice hockey is played in cool (4 to 12 °C) ambient temperatures (Green, 1978; Green et al., 1978) with an intermittent production of metabolic heat during 40 to 90 s "shifts"

interspersed with 3 to 5 min of inactivity (Green et al., 1976, 1978; Montgomery, 1988; Paterson, 1979). Body weight loss during a hockey game has been reported as 2 to 3 kg indicating an increased sweat rate in an attempt to dissipate metabolically produced heat (Green et al., 1978; MacDougall et al., 1979). The extensive protective equipment worn would decrease heat dissipation (Montgomery, 1979, 1988) and result in the player exercising in a warm microclimate with a high relative humidity. As already discussed, this would possibly result in: an increased body temperature affecting motivation and motor unit recruitment; an increased peripheral displacement of blood to compensate for the decreased core-to-skin thermal gradient due to the inhibition of sweat evaporation; dehydration from increased sweating in an attempt to increase the thermal gradient; a possible decrease in muscle blood flow and oxygen consumption from competition for peripheral blood flow; and, the  $Q_{10}$  effect increasing glycolysis and lactate production. In discussing energy metabolism in ice hockey, Green (1979) concluded that "optimal performance in ice hockey depends on a maximal involvement of the aerobic system for ATP resynthesis while minimizing the glycolytic involvement" (p. 33). Thus, the dissipation of metabolically produced heat is of importance to the hockey player if optimal performance is to be obtained. With the recent trend to increased protection (Montgomery, 1988), the effect of hockey equipment on heat dissipation needs

to be objectively evaluated, allowing players to obtain adequate injury and contact protection without inhibiting work output by the thermoregulatory measure evoked.

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