4-1-1999

A Comparison of Active and Passive Recovery on Lactate Concentration and Subsequent Performance of Repeated Work Bouts in University Ice Hockey Players

Michelle L. Lau
University of Nebraska at Omaha

Follow this and additional works at: https://digitalcommons.unomaha.edu/studentwork

A Comparison of Active and Passive Recovery on Lactate Concentration and

Subsequent Performance of Repeated Work Bouts in University Ice Hockey Players

A Thesis Presented to the School of Health, Physical Education and Recreation and

the Faculty of the Graduate College

University of Nebraska

In Partial Fulfillment of the Requirements for the Degree Master of Science

University of Nebraska at Omaha

by
Michelle L. Lau

April 1999

UMI Number: EP73291

UMI

Dissertation Publishing

UMI EP73291

ProQuest

TABLE OF CONTENTS

THESIS ACCEPTANCE

Acceptance for the faculty of the Graduate College,
University of Nebraska, in partial fulfillment of the
requirements for the degree Masters of Science,
University of Nebraska at Omaha.

Committee

_Richard W. _____ (signature)_

Name	Department/School

HPER

_____ (signature)

Name | Department/School

Psychology

_____ (signature)

Name | Department/School

NPER

Chairperson: _Kris Beng_

Date: _3-23-79_

Abstract

This study examined the effect of active and passive recovery on lactate concentration and subsequent performance of repeated work bouts in 18 male college ice hockey players. Using a repeated measure design, subjects performed a series of skating tests before and after a 15-minute recovery. The skating test consisted of skating a course for 7 shifts lasting 40 sec per shift with 90 sec rest between shifts. The recovery active (low intensity cycling) or passive (sitting) recovery lasted for 15 minutes followed by an identical seven shift skating test. Lactate was measured at rest, 3-5 min following the first skating test and 12-15 min into the recovery period. A 2 x 2 analysis of variance with repeated measures of distance and heart rate revealed significant differences between period 1 and 2 for both variables ($p=0.002$ and $p = 0.001$ respectively). There was no interaction between periods and recovery for either distance or heart rate. Passive versus active recovery also showed no statistically significant difference for distance skated or heart rate. No significant difference was found in lactate between active or passive recovery. However, lactate at 3-5 min was greater than at 12-15 min ($p<0.001$). Pearson correlation coefficient showed no relationship for lactate changes during active recovery to skating distance ($r = 0.11$, $p>0.05$) or HR ($r = 0.21$, $p>0.05$) in the second period. No relationship was found among heart rate during period 2 for either active or passive 12 min lactate values. There appeared to be a trend for greater skating distance in period 2 when active recovery was used, but the difference was not significant. It was concluded that active recovery did not enhance lactate removal or subsequent performance of repeated work bouts.

Acknowledgements

TABLE OF TABLES

TABLE OF FIGURES

Chapter I

Introduction

Various sports require a variety of skills and physical demands that an athlete must perform efficiently to excel. Ice hockey is a sport that requires athletes to utilize the speed of a sprinter, the power of a football player, and the finesse of a golfer, while performing on ice skates. The intermittent nature of this fast-pace sport requires the speed to chase a puck or for breaking away, power for checking and shooting, and precision to handle the puck all of which places great physiological and motor skill demands on the ice hockey player.

The ability of the exercise scientist or coach to properly prepare these athletes for competition is important. The metabolic areas important to performance are the anaerobic (ATP-PC or glycolysis) and aerobic (oxidative phosphorylation) energy systems. Seliger, Kostka, Grusova, Kovac, Machovcova, Pauer, Pribylova, and Urbankova (1972) found that glycolysis and ATP-PC provide 69% of the energy demand of ice hockey, while the role of oxidative phosphylation is 31%. Due to the intensity and the length of the shifts (30 to 80 seconds) the ice hockey player's main energy requirement is anaerobic metabolism, while the aerobic metabolism aids in the recovery process between shifts and periods.

Another important limiting factor in ice hockey performance is the accumulation of lactate, due to the highly intense efforts for 30 to 80 seconds. Lactate contributes to muscle fatigue and if high levels accumulate performance will be limited for 30 seconds to 15 minutes depending on the amount of lactate formed (Billat, 1996). Several studies

have observed that active recovery was the best method to facilitate lactate clearance following exercise other than hockey (Weltman & Regan, 1983; Bond, Adams, Tearney, Gresham, & Ruff, 1991; Thiriet et al. 1993). Weltman, Stamford, Moffatt, and Katch (1977) found that active recovery or 20 minute passive recovery resulted in a significantly ($p < 0.01$) greater subsequent exercise performance and enhanced the rate of lactate removal during recovery. Ahmaidi et al. (1996) found that higher outputs during a force-velocity test were better maintained with an active recovery protocol versus a passive recovery. These studies showed that active recovery could aid in subsequential exercise or work bouts, but other research contradicts these findings. Bond et al. (1991) demonstrated a reduction in lactate with active recovery, but subsequent isokinetic muscle function was unaffected by active or passive recovery modes. Watson and Hanley (1986) also found that no improvement occurred during maximal constant load cycling performance following active recovery.

Another study by Watson and Hanley (1986) is the only current active recovery study performed on ice hockey players. They found that active recovery enhanced lactate removal, but subsequent performance was unaffected by any treatment. Their findings leave some questions because the simulated ice hockey task was not designed to mimic an actual game situation and the duration of the shift may also have failed to replicate the game situation. To date research has not clearly determined whether active recovery is beneficial to subsequential exercise performance in hockey players. Therefore the purpose of this study was to determine the effect of active recovery on lactate concentration and subsequent performance of repeated work bouts in ice hockey players.

2

Chapter II

The Problem

Purpose

The purpose of this study was to compare the effect of active and passive recovery on lactate concentration and subsequent performance of repeated work bouts in ice hockey players.

Hypotheses

- Removal of lactate following repeated work bouts will be greater after active recovery than passive recovery.

- Hockey players will cover a greater distance in period two following active recovery versus passive recovery.

Delimitations

Eighteen male ice hockey players (M = age of 21.7) on the University of Nebraska at Omaha team were subjects in a study in which repeated work bouts of ice skating were performed followed by an active or passive recovery mode. At the completion of the recovery another set of repeated work bouts were performed.

Limitations

The limitations of this study would be the ice condition (surface), ability to simulate a game situation, motivation, and diet.

- Ice Conditions: as play continues the ice becomes choppy, and friction increases which slows skating speed.

- Game Situation: players experience numerous stops and starts during a game as well

as stop in play due to penalties or when the player leaves the playing surface for recovery between shifts. These characteristics are difficult to account for in a study.

- Motivation: motivating the athlete to perform at game level is difficult in a test atmosphere without spectators and competition.

- Diet: nutritional status was uncontrolled . Differences in diet such as carbohydrates, protein intake, and use of supplements (e.g., creatine) could affect the performance considering that ice hockey players rely heavily on the anaerobic system for performance.

Definition of Terms

- On-ice time: is the actual amount of playing time for one shift.

- Shifts: is one work bout lasting 40 seconds in this hockey study.

- Period: in college ice hockey is 20 minutes with three periods total that are interspersed with two 15 minute intermissions.

Significance of the Study

Active recovery appears to offer a physiological basis for enhancing work performance, but currently is not used in hockey or other team sports. If active recovery enhances lactate removal and subsequent performance in ice hockey players, then the use of active recovery in university players should be further examined to determine the optimal rate and duration of exercise during recovery.

Chapter III

Review of Literature

Ice hockey is a sport that requires various physical demands and skills for the athlete to excel. A review of these physical demands and active versus passive recovery methods will be examined with regards to ice hockey players.

Physical Demands of Ice Hockey

The three main energy systems that the human body calls upon to perform any type of movement are utilized to great extents in ice hockey. The ATP-PC system provides the quick burst of strength needed for sprinting down the ice, while anaerobic glycolysis allows the player to perform at high intensity for longer periods. Oxidative phosphylation is also important in the sport of ice hockey to help aid in recovery from high-intensity work. The main area of focus in this section will be the anaerobic energy system (power and capacity), aerobic energy system (VO_2max and heart rate), and lactate (accumulation and production) with regards to ice hockey.

Anaerobic Energy System

A study using 13 players from a national representative team indicates that ice hockey is an activity that demonstrates a submaximal metabolic rate with 69% of the energy demand coming from anaerobic metabolism and 31% aerobic metabolism (Seliger et al., 1972). Watson and Sargeant (1986) compared anaerobic power and capacity in 24 university or junior A ice hockey players. This study tested the players for anaerobic power and capacity using three different methods. The methods were two on-ice tests versus the laboratory Wingate cycling test. The findings in this study showed anaerobic

power and capacity values for a 30 sec Wingate cycling test to be 10.1 ± 0.02 watt•kg^{-1}

for peak power and 7.7 ± 0.2 watt•kg^{-1} for capacity (Watson & Segreant, 1986). There

was no significant relationship ($r = 0.32$), $p > 0.05$ between the Wingate and on-ice testing

for anaerobic power, while the on-ice testing produced higher values for both anaerobic

power and capacity (peak power ranged 11.5 to 11.9 while the capacity range mean was

9.3 to 9.7 watt•kg^{-1}) (Watson & Sergeant, 1986). Smith, Quinney, Steadward, Wenger,

and Sexsmith (1982) tested the anaerobic power of the Canadian Olympic Team (1980)

using a 30 sec Wingate test on a modified Monark cycle and found overall team value of

11.7 watt•kg^{-1} for peak power. A study of the physiological profiles of National Hockey

League regulars during the 1985-86 season demonstrated peak anaerobic power values

for defensemen to be 12.04 watt•kg^{-1} and forwards was 12.0 watt•kg^{-1}, while anaerobic

capacity was 9.54 and 9.1 watt•kg^{-1} respectively (Rhodes, Cox, & Quinney, 1986).

Aerobic Energy System

The aerobic system aids in the recovery process after anaerobic exercise. The

level of aerobic fitness necessary for hockey players has been studied by several

researchers. Previous research that monitored game heart rates were used to predict

VO_{2max} during the game by comparing heart rate values obtained in a treadmill test.

These comparisons were used to predict on-ice aerobic energy requirement. The range

for heart rate for three periods of a game was 170 to 174 bpm using telemetered

recordings which was equivalent to 70 to 80% VO_{2max} in university players (Green et al.

1976). Paterson (1979) found a mean on-ice energy expenditure to be in excess of 80%

of the VO_{2max} which was 50ml/kg/min in elite boys and college players. They also found

peak heart rates were to be in excess of 95% of HR max during each shift on the ice in elite boys and intercollegiate players. Cunningham, Telford, and Swart (1976) found in their study of 15 boys from a competitive hockey team to have an average VO_{2max} of 56.6 ml/kg/min. The VO_{2max} in young boys and college players appears to be similar, even though they compete at different levels. A study by Houston and Green (1976) observed an average VO_{2max} of 55.4 ml/kg/min in major junior A and university players. The junior A players had a slightly greater VO_{2max} (56.3 ml/kg/min in forwards and 55.6 ml/kg/min in defensemen), while the university players VO_{2max} was 54.6 ml/kg/min forwards and 53.6 ml/kg/min defensemen. A longitudinal study by Cox, Miles, Verde, Levine, and Bartolozzi (1993) examined the physical and physiological characteristics of National Hockey League players between 1980 to 1991. Cox et al. (1993) most notable finding was that 58% of the players had a VO_{2max} less than 55 ml/kg/min in 1980, but by 1991 only 15% of the players VO_{2max} was less than 55 ml/kg/min. The mean VO_{2max} values were provided for 1991 only, which were 62.4 ml/kg/min for team Canada and 60.2 ml/kg/min for NHL regulars. Watson and Hanley (1986) found values for VO_{2max} on the treadmill to range between 54.3 to 63.8ml/kg/min (Mean of 58.4 $\pm$ 3.2 ml/kg/min) in university and junior players.

A study of two training methods (ice hockey or combination of ice hockey and low intensity cycling) in college hockey players both failed to elicit any alterations in VO_{2max}, HR_{max}, and VE_{max} (Daub, Green, Houston, Thomson, Fraser, and Ranney, 1983).

Lactate Levels in Hockey Players

The previously cited studies indicate that ice hockey players not only must possess high anaerobic capacity but also have a reasonable level of aerobic capacity to perform. The aerobic capacity plays a great role in the recovery process of the hockey player. Lactate removal occurs via the lactate shuttle which is facilitated by the players aerobic metabolism. The number of shifts and the length of the shift used by hockey players can affect their playing ability. The longer the shift the higher the lactate values reached. Green, Daub, Painter, and Thomson (1978) determined that the players' VO_{2max} is an important factor in the degree of glycogen depletion that will occur following a game. They reported a 60% reduction in glycogen following a game was reported in university players. The effects of a low VO_{2max} on the blood lactate and muscle glycogen was also seen in one Canadian and one Swedish hockey player. The results of the study showed the Canadian player to have a VO_{2max} of 55ml/kg/min, while the Swedish player VO_{2max} was 65ml/kg/min, which was associated with a higher blood lactate values (approximately 7mMol/L and 2.5mMol/L respectively for 90 sec of work) and more rapid depletion of glycogen in the Canadian player (Green, 1979). These studies both suggest the importance of a higher VO_{2max} even though hockey is a sport that requires great deal of anaerobic energy.

Several studies have demonstrated that highly intense exercise of a few seconds or minutes is impaired if blood lactate levels are elevated prior to exercise (Jacobs, 1986; Yates, Gladden, & Cresanta, 1983; Hogan & Welch, 1984; Karlsson, 1975.). Impairment in performance will also occur even if the exercise was performed by a muscle group

other than the original muscle group that created the initial lactate production and accumulation (Jacobs, 1986; Yates et al., 1983). The impairment can be insignificant if the recovery period is 20 minutes or the lactate levels do not exceed 4 to 6 mMol/L when exercise resumes (Weltman & Regan, 1983; Weltman et al., 1977).

In a study using endurance trained men, who performed a supermaximal cycling exercise that caused exhaustion within 3 to 6 minutes, existing high levels of lactate concentration did not affect the maximal aerobic power, but a reduction in total work output was observed (Klausen, Knuttgen, & Foster, 1972). Yates, Gladden, and Cresanta (1983) showed that elevated lactate levels due to maximal dynamic leg exercise caused a 25% reduction in a subsequential static arm endurance task. Jacobs (1986) showed the blood lactate levels of forwards and defensemen during a competitive ice hockey game were above 6 mMol/L seven minutes into the first period. As play continued into the second and third periods, blood lactate remained elevated above 6mMol/L for both the forwards and defensemen. Weltman and Regan (1983) and Weltman et al. (1977) noticed that if an athlete is allowed at least 20 minutes of recovery or lactate levels does not exceed 4 to 6mMol/L, when exercise resumes no impairment in performance occurs. From Jacobs' (1986) findings the ice hockey players remain at or above 6 mMol/L throughout the majority of the second and third periods. The highest levels seen were throughout the third period and occurred in the defensemen reaching a level of 11 to 11.5 mMol/L (Jacobs, 1986). After about seven minutes into the game the hockey players' blood lactate levels ranged between 6 to 11.5 mMol/L throughout the game even though they received two 20-minute recovery bouts between periods.

A unpublished observation cited in Houston and Green (1976) found blood lactate levels following each period of play in excess of 5 mMol/L. They also found lactate levels following a short duration supramaximal treadmill run to exhaustion in juniors and university ice hockey players to range between 7.2 to 21.7 mMol/L. The mean lactate level for the junior forwards was 12.2 mMol/L and for defensemen it was 13.0 mMol/L, while the university players' mean values were 12.3 and 13.3 mMol/L respectively (Houston & Green, 1976). Watson and Sargeant's (1986) study of university and junior A players, while performing a 30 sec Wingate test, repeated sprint skate, and Sargeant Anaerobic skate test found lactate values of 10.8 ± 1.5 mMol/L, 10.7 ± 1.9 mMol/L, and 11.5 ± 1.6 mMol/L, respectively. Another study of university and junior A hockey players found a mean lactate value of 12.1 ± 1.1 mMol/L following six 45 seconds work bouts (skating on an oval course) with 90 seconds rest between work bouts (Watson & Hanley, 1986). These studies show high levels of lactate being produced in ice hockey players.

Active vs. Passive Recovery

The previous research on lactate demonstrates how high lactate levels can affect athletic performance. The levels of lactate seen in ice hockey players during competition and in the laboratory environment also indicate a potential impairment to performance during repeated work bouts. The purpose of this section is to analyze the role of active (AR) and passive (PR) recovery on lactate levels and subsequent performance.

Bond et al. (1991) studied active and passive recovery using five healthy males while performing a cycle ergometer test for 60 seconds at 150% of the subjects' VO_{2max}.

10

The test was followed by a 20-minute recovery period, which consisted of either sitting on the ergometer (passive) or pedaling at 30% of the subject's VO_{2max} (active). At completion of the recovery period the subjects performed an isokinetic muscle test evaluating peak torque output and fatigue of the quadriceps muscle group. They found that the rate of lactate removal during active recovery was significantly greater ($p < 0.05$) when compared to passive recovery (3.5mM and 7.1mM, respectively). Bond et al., (1991) also determined that active recovery enhanced lactate removal, but subsequent isokinetic muscle function was unaffected by either recovery modes. A study of active recovery in 10 healthy males performing an incremental exercise test and two force-velocity tests on a cycle ergometer found a significantly ($p < 0.01$) lower lactate level with active recovery than with passive recovery when using higher braking forces (Ahmaidi et al., 1996). This study also observed higher power outputs with the active recovery mode, but the findings were significant only at higher braking forces ($p < 0.01$).

Sixteen healthy male gymnasts performed a series of four exhaustive cycling ergometer tests at 130% of maximal aerobic power (MAP) for two minutes (Thiriet, et al. 1993). Each series of exercise was followed by a 20 minute recovery, which consisted of either passive sitting on a chair at cycle level, active leg recovery (pedaling at 30% MAP), or active arm recovery (pedaling at 30% MAP). They found that passive recovery resulted in significantly higher lactate concentration following each subsequent recovery than active leg or arm, with active leg having the lowest lactate concentration. Another finding of this study was that duration of subsequent exercise was unaffected in active leg and arm, but was significantly shorter in passive recovery ($p < 0.03$). Thiriet et al. (1993)

11

also found a significant decrease in work during the second exercise repetition only when passive recovery was employed ($p<0.01$). Siebers and McMurray (1981) used six female swimmers to evaluate the recovery procedure after exercise. The swimmers performed a front crawl for 2 minutes at 90% VO_{2max} using a swimming ergometer, which was followed by 15 minute recovery period. The recovery consisted of either walking on land for 10 minutes and then sitting for 5 minutes or swimming continuous lengths of front crawl at a moderate pace for 10 minutes and then sitting 5 minutes. Subjects then performed a 200 yard front crawl swam as fast as possible from a diving start followed the recovery period. A significant ($p<0.05$) reduction in lactate concentration was observed with swimming recovery as compared to walking as a method of recovery (Siebers & McMurray, 1981). Swimming reduced the lactate by $53.3\pm3\%$ compared to $38.5\pm3\%$ for walking recovery. They found no significant differences in 200 yard swim times between swimming or walking recovery ($p<0.05$). Fifteen minutes was determined to be an adequate recovery period to remove lactate following high intense work bouts lasting three minutes or less, but the results indicated that neither recovery significantly influenced subsequent performance or the recovery from subsequent performance. Weltman et al. (1977) evaluated active and passive recovery in 11 male university students after pedalling on a cycle ergometer for one minute all-out at 5.5 kg. The recovery modes used included sitting at cycle level for either 10 or 20 minutes (passive) and pedaling at 60 rpm at 1.0 kg breathing room air or 100% oxygen for either 10 or 20 minutes in duration (active). The subsequent performance was performing the same cycle ergometer task. They found a significant effect for active versus passive recovery

and for 10 versus 20 minute recovery, with active and 20 minute recovery resulting in significantly enhanced rate of lactate removal during recovery ($p < 0.001$) and a higher subsequent power production ($p < 0.001$). The use of 100% oxygen during recovery had no significant effect on subsequent performance or lactate removal ($p > 0.05$).

Weltman and Regan (1983) performed another study of active and passive recovery using nine healthy male university students while performing a maximal constant load test on a cycle ergometer at 60 rpm. The subjects in this study were required to perform the exercise at 110% of VO_{2max}, which was followed by a 20 minute recovery. The recovery consisted of sitting at cycle level (PR) or pedaling at 40% VO_{2max} (AR) which was then followed by another maximal performance test (110% VO_{2max}). They found a significantly ($p < 0.05$) lower blood lactate concentration with active recovery then passive recovery (4.0±1.3mM and 5.6±1.2mM, respectively). Another major finding of this study was the poor relationship ($r = 0.08$) between elevated blood lactate concentration and subsequent exercise performance. No significant difference was found in performance time when active or passive recovery were employed. Gupta, Goswami, Sadhukhan, and Mathur (1996) studied 10 male athletes performing a cycle ergometer test at 150% VO_{2max} for one minute interspersed with 15 seconds of rest. Significant differences were demonstrated in half-life of lactate with different recovery modes. Active recovery (pedaling at 30% VO_{2max}) demonstrated the fastest half-life of lactate when compared to massage or passive (sitting) recovery (15.7±2.5 min, 21.8±3.5 min, and 21.5±2.8 min respectively).

Currently only one active and passive recovery study exists using ice hockey

players as the subjects. Watson and Hanley (1986) used eight ice hockey players to determine the effect of two different active recovery modes on the lactate concentration and subsequent performance. The exercise stimulus consisted of a maximal 45 second skate around a 133.3 m oval course six times with 90 seconds rest between each 45 second trial. A recovery period of 15 minutes followed the exercise bout. The recovery period was two different modes of active recovery (skating or bench-stepping at a heart rate of 120 b•min^{-1}) and passive recovery (sitting on players bench). The duration of the recovery modes was 10 minutes while blood samples were collected three minutes prior to the beginning of the recovery period. A significantly (p<0.05) lower lactate level was observed during bench-stepping (6.1±2.2 mmol•l^{-1}) than for passive recovery (8.1±1.6 mmol•l^{-1}), while skating performance was not significantly different from either bench stepping or passive. Watson and Hanley (1986) discovered that bench stepping enhanced lactate removal, but subsequent performance was unaffected by any of the recovery modes. They actually found a significantly lower performance in the second work bout than the initial work bout for all recovery modes.

Several studies have been conducted using ice hockey players in regards to physiological profile, but only one study has evaluated active and passive recovery in ice hockey players. Few studies have examined active and passive recovery and the role that recovery plays on subsequential performance. After evaluating these studies further examination is need to determine the effect that active and passive recovery plays on subseqeunt performance. The studies on active and passive recovery are summarized in Table 1.

Table 1. Summary of Studies Comparing Active and Passive Recovery and Subsequent Performance.

Author	N	Subjects	Exercise Intensity	Active Recovery	LA Level after exercise	Enhance Performance (Yes/No)
Ahmaidi et al. (1996)	10	male healthy	incremental aerobic test & FV test	32% VO_{2max}	cycling ergometer	Yes
Bond et al. (1991)	5	male active	150% VO_{2max}	30% VO_{2max}	9.1 to 9.2 mM cycling ergometer	No
Gupta et al. (1996)	10	male walker & runners	150% VO_{2max}	30% VO_{2max}	3-10 mM cycling ergometer	N/A
Siebers et al. (1981)	6	female swimmers	90% VO_{2max}	moderate	96.7±1.8 mg/100ml swimming or walking	No
Thireit et al. (1993)	16	male gymnastic students	130% VO_{2max}	30% VO_{2max}	cycling ergometer	Yes
Watson et al. (1986)	8	male ice hockey	91.7% HR_{max}	127±8 b·min⁻¹ (S) and 136±8 b·min⁻¹ (B)	12.1 mmol·l⁻¹ bench-stepping or skating	No
Weltman et al. (1977)	11	male non-athletes	all-out	60rpm	115 - 132 mg% cycling ergometer	Yes
Weltman et al. (1983)	9	male swimmer 6, diver, runner, & biker	92-94% VO_{2max}	40% VO_{2max}	9.1-10.2 mM cycling ergometer	No

Chapter IV

Methods

Subjects

Nineteen male college ice hockey players attending the University of Nebraska at Omaha were used as subjects and provided informed consent approved by the IRB.

Experimental Design

Using a repeated measures design subjects performed a series of skating tests before and after 15 minutes recovery. The skating test consisted of seven work bouts lasting 40 seconds with 90 seconds of rest between each work bout. The active recovery (low intensity cycling) or passive (sitting) lasted for approximently 12 to 15 minutes followed by a second identical skating test. This study was designed to achieve a work-rest ratio (40: 90 sec) commonly used in college hockey. In order for this to occur the subjects skated the time equivalent of one period of play, then recovered during a 15 minute intermission and followed by another skating period. Subjects performed the skate tests in pairs. The second skater was started 20 sec after the completion of the first skater's 40 sec work bout. In each pair one subject was randomly assigned to active recovery, while the other was assigned to passive recovery for the first test day. On the second day of testing the opposite type of recovery was performed from the first test. Tests were conducted with at least two days separating test days with the exception of three atheletes who skated with only one day of rest due to ice time restraints. Also due to avaliable ice time two different locations were used in this study.

Procedures

Skating Test. The subjects were dressed in hockey gear and carried their hockey stick while performing each shift. All subjects performed a series of seven shifts each consisting of 40 sec of skating alternated with 90 sec of rest. This work-rest ratio was based on game data (video) from one UNO game. The findings showed a mean of 6.6 shifts played per period with each shift lasting 30 to 50 seconds. The 90 seconds was based on the statistics collected on 100 NHL time on ice leaders. (www.NHL.com) The mean rest between shifts was 91 seconds.

The work portion of the test measured the distance skated in an obstacle course for 40 seconds. The course consisted of forward skating (41.1 meters at Benson and 42 meters at Aksarben ice rinks) followed by a stop and change of direction. The subject then skated backward across the ice (18.2 meters at Benson and 23.0 meters Aksarben ice rinks) and then stopped to change direction again. At this time the subject skated forward to the next face off dot ice (18.2 meters at Benson and 23.0 meters Aksarben ice rinks). The subject then stopped again and skated backward (41.1 meters at Benson and 42 meters at Aksarben ice rinks) followed by another stop. At this point the subject then skated forward (18.2 meters at Benson and 23.0 meters Aksarben ice rinks) and then stopped. The subject then skated backwards to the start (18.2 meters at Benson and 23.0 meters Aksarben ice rinks). The distance (m) covered in each 40 sec work bout was marked and measured. The sum of the distances covered in each work bout was summed to represent the total skating distance in each period. The course consisted of a hexagonal pattern that required forward, backward and lateral skating to set points on the ice. The course was designed to mimic actual playing of the game, with inherent

multiple changes of direction. Figure 1 displays the course.
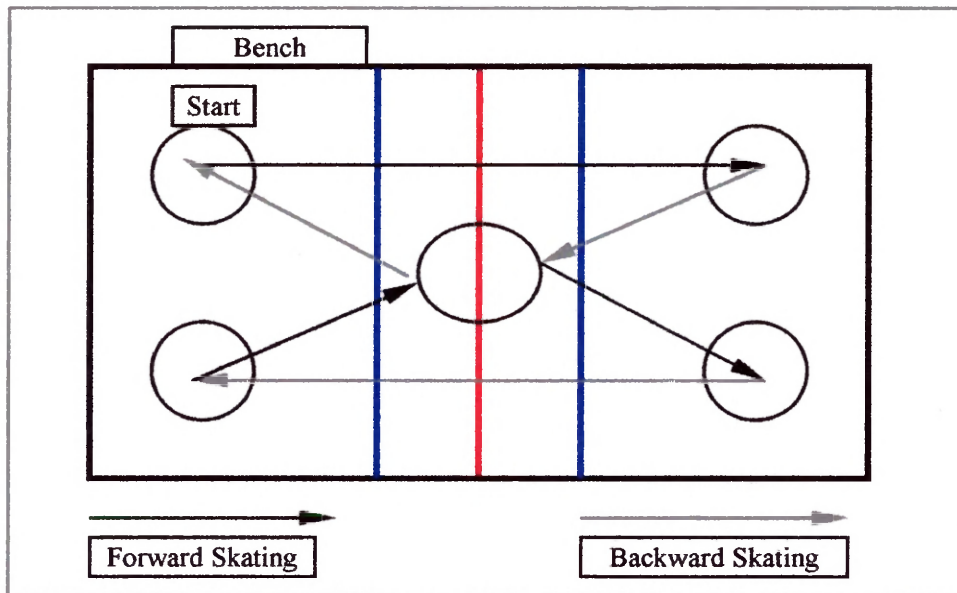
Figure 1. Diagram of Skating Test

A whistle was used to signal the completion of the shift, while a verbal ("ready, set, go"!) comand was used to start. Subjects rested for 90 sec while standing off to the side of the starting spot before performing the next shift. After the seven shifts were completed the subjects then performed either an active or passive recovery. Active recovery consisted of pedaling a cycle egrometer at a self-selected work rate between 50 and 70 rpm for about 12 minutes. The subject sat on the cycle immediately following the completion of the seventh shift and rested without pedaling for three minutes. At three minutes a blood sample was collected and the subject started pedaling. The 3 minutes on the cycle without pedaling allowed blood lactate to reach a peak level which occurs about 3 to 5 min following exertion. The passive recovery consisted of sitting on the players bench for 15 minutes which is the normal length of intermission in a college hockey

game. At the completion of the 15 minute recovery period subjects then returned to the

ice following two minutes of light skating before the next skating test began. The

subjects then performed the identical skating test performed in period one.

Heart Rate. Heart rate (HR) was monitored with a Polar watch and recorded

immediately following the completion of a shift. Heart rate was also collected for

descriptive purposes during recovery every two minutes beginning with the 2nd minute.

The heart rate after each shift was used to determine the relative work effort for each

shift.

Blood Lactate. One drop of blood was collected from a finger puncture to determine

lactate concentration in each subject at rest (before warm-up), 3 to 5 minutes following

period 1 and at 12 to 15 of the recovery period. An Accusport™ lactate analyzer (Yellow

Springs, Model 121) was used to determine lactate concentration and calibrated before

each test per manufacturer's instructions. Code strips to calibrate the analyzer were

enclosed in each new box of lactate strips. Code stripes were used each time a blood

sample was collected and analyzed.

Statistical Analysis

Descriptive statistics including mean and standard deviation were calculated for

each variable. Three two-way analyses of variance with repeated measures were used to

compare the effect of recovery mode on skating distance, lactate level, and heart rate.

Pearson correlation coefficient was used to determine relationships among lactate, HR

and skating distance in period 2. The 0.05 level of significance was used in all tests. The

software program used for two way analysis of variance was SPSS for MS Windows

Release 6.0, while Graph Pad Instat version 3.0 for Win 3.1 was used for the Pearson r.

Chapter V

Results

The removal of lactate following repeated work bouts with either active and passive recovery was not significantly different. The interaction between recovery and lactate values accross time were also not significantly different, while the main effect time was significantly different ($F=63.51$, $df=1,11$, $p<0.001$). A significantly higher lactate was seen at 3-5 min following exercise than for 12-15 min into recovery ($F=22.04$, $df=2,22$, $p<0.001$). The relative lactate values (multiples of resting lactate) also displayed no significant difference between active and passive recovery. These results do not support the hypothesis that the removal of lactate would be greater following active recovery. Figure 2 represents the mean lactate values at rest, 3-5 min following exercise, and 12-15 min into recovery.
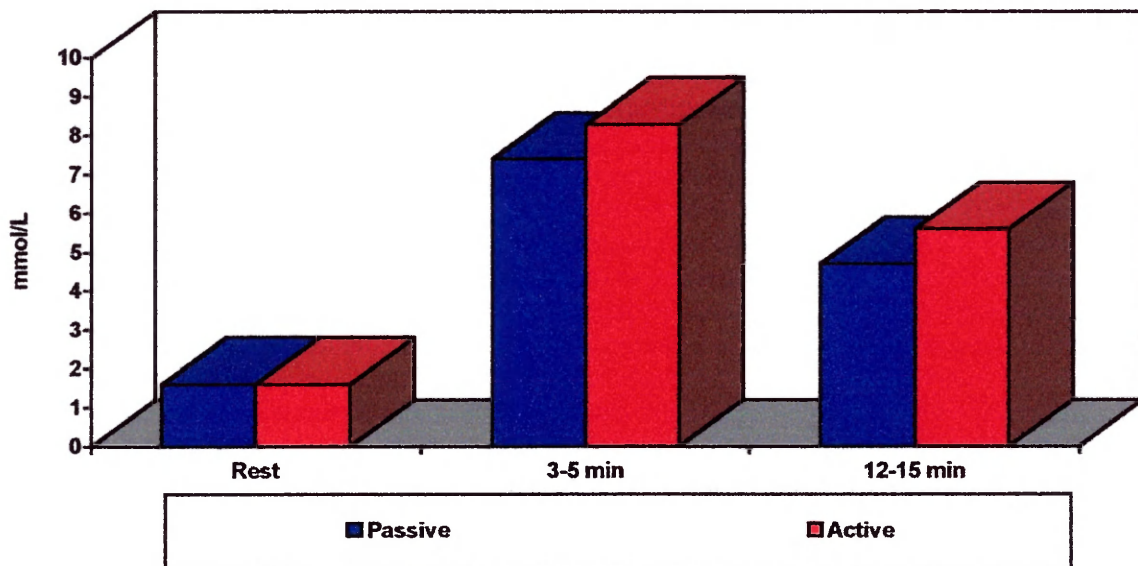
Figure 2. Mean Lactate Value at Rest, 3-5 Minutes Following Skating Test, and 12-15 Minutes into Recovery Period.

The second hypothesis stated that a greater skating distance will be covered in period two following active recovery versus passive recovery. Statistically significance difference occured between periods (F=12.68, df = 1, 17, p = 0.002). The distance skated in period 2 across both passive and active recovery was greater in period 2, with a mean of 1594 versus 1616 m, respectively. Figure 3 displays the total distance covered each period. No significant differences were found for the main effect recovery mode (F = 0.33, df = 1,17, p = 0.572) or for interaction between period and recovery (F = 1.76, df = 1,17, p = 0.202).

Figure 3. Total Distance Skated for Periods 1 and 2 While Performing Either a Passive or Active Recovery Between Periods.

No significant differences were found in skating distance for each shift when active or passive recovery was performed between periods. However, a clear trend favoring active recovery occurred. Figure 4 shows the distance skated each shift for period 1 and 2.

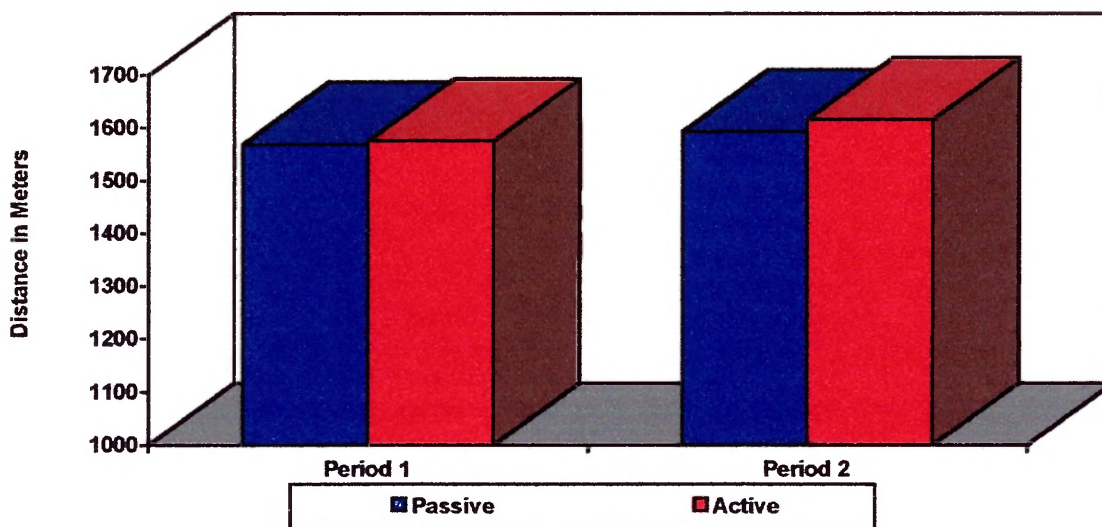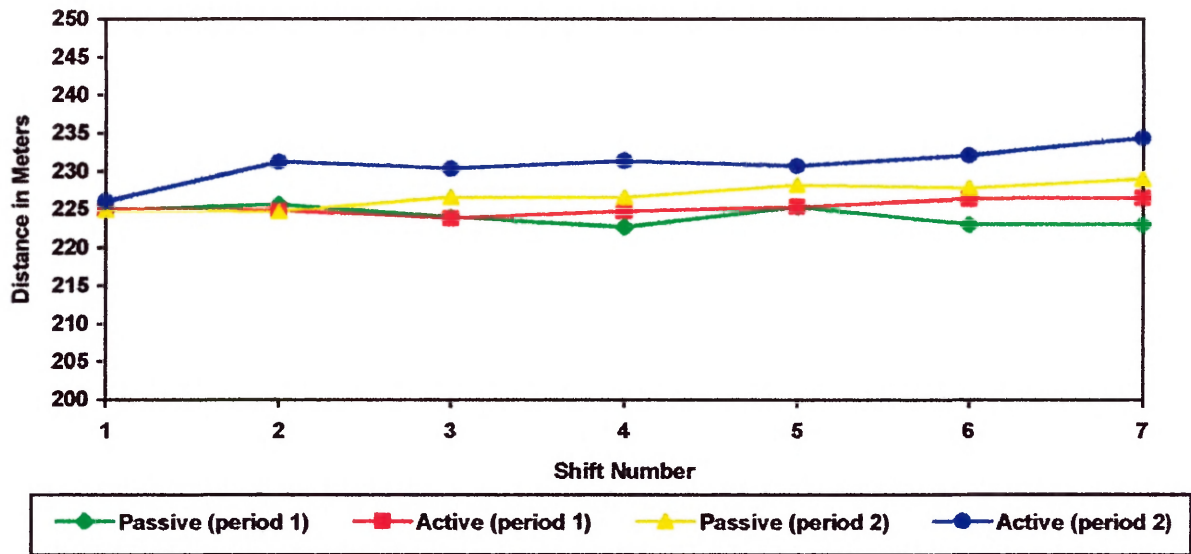**Figure 4. Skating Distance for Individual Shifts During Period 1 and 2 with Either Active or Passive Recovery.**

Heart rate was also measured to determine if heart rate varied due to the recovery used. A significantly higher HR in period 2 than period 1 ($F = 12.54$, $df = 1,17$, $p = 0.001$), while no significant difference was found between recovery modes or interaction between period and recovery. Table 2 represents the mean data for lactate, distance skated and heart rate.

Table 2. The mean values for lactate, distance, and heart rate during period 1and 2 following either passive or active recovery (M ± SD).

	Lactate (mmol/L) N=12			Distance (meters) N=18		Heart Rate (bpm) N=18	
Recovery	Rest	3-5 min	12-15 min	Period 1	Period 2	Period 1	Period 2
Passive	1.6 ± 0.83	7.4 ± 2.74	4.7 ± 2.75	1570 ± 16.91	1594 ± 15.51	172 ± 9.16	175 ± 9.64
Active	1.6 ± 0.52	8.3 ± 3.07	5.6 ± 2.49	1577 ± 15.57	1616 ± 16.76	173 ± 8.71	177 ± 8.88

Pearson correlation coefficients were calculated to determine relationships among delta lactate, HR and skating distance in period 2. No significant relationships were

23

found between the delta lactate (i.e, peak LA - LA at 12-15 min) during active recovery and skating distance in period 2 ($r = 0.11$, $p>0.05$). This indicates a weak trend for greater skating distance to be associated with a greater delta lactate. The correlation of delta lactate during active recovery to HR in the second period also displayed no significant relationship ($r = 0.21$, $p>0.05$), but also indicated that a greater delta lactate was weakly associated with a greater HR.

Heart rate during the second period showed no significant relationship to 12 min lactate for either active or passive recovery ($r = -0.09$ and $r = 0.15$, $p>0.05$, respectively). Distance skated in period 2 for active recovery had no relationship to 12 min lactate values ($r = -0.32$, $p = 0.19$). However, a weak trend exists for greater skating distance to be related with lower lactate level at 12-15 min. Skating distance following passive recovery also had no relationship to 12 min lactate values ($r = -0.31$, $p = 0.22$). Both skating distance and HR were negative correlations that weakly support a trend for greater skating distance to be associated with lower 12-15 min lactate.

Chapter VI

Discussion

The main findings of this study were that active recovery did not produce a lower lactate level at 12 min of recovery and that skating distance was not enhanced. Although no significant differences were noted in these variables, there appears to be a trend for active recovery to demonstrate a greater skating distance in the second period. The difference in distance skated in the second period for active and passive recovery appears to be small, (1616 m - 1594 m = 22 m respectively), but this difference could be meaningful in a game. A small advantage in skating distance could be a matter of the player reaching the puck before his opponent scores or to score a goal. Differences in hockey teams might well be reflected in such small variations in skating distance.

The inability of this investigation to show enhancement in performance following active recovery is in agreement with several other studies (Bond et al., 1991; Siebers et al., 1981; Watson et al., 1986; and Weltman et al., 1983). Watson and Hanley (1986) found no benefit in skating performance in ice hockey players when active recovery (bench-stepping or skating) or passive recovery (sitting) was used. The exercise intensity elicited during the skating test for the present study was 87.9% HR_{max} (mean HR for all conditions) versus 91.7% HR_{max} reported by Watson and Hanley (1986), while peak lactate levels were 7.9 mmol/L and 12.1 mmol/L, respectively. Bond et al. (1991) study of active males pedaling on a cycle ergometer for 60 sec at 150% VO2max followed by a 20 min active recovery of pedaling at 30% VO2max displayed no enhancement in subsequental isokinetic muscle performance for either active or passive recovery. A

25

swim study using six female swimmers also showed no advantage in active recovery. Subjects swam the front crawl for 2 min at 90% Vo2max followed by a 10 min recovery of moderate swimming or walking. There was no enhancement in a subsequent 200 yard front crawl for time (Siebers & McMurray, 1981). Another study using nine healthy males performing maximal constant load exercise on a cycle ergometer at 110% VO2max followed by 20 min recovery of sitting or pedaling at 40% Vo2max found no enahncement in subsequent performance (Weltman & Regan, 1983). This study also found a poor reationship ($r = 0.08$) among elevated blood lactate concentration and subsequent exercise peformance.

Other researchers have displayed enhancement in performance following active recovery methods (Ahmaidi et al., 1996; Thireit et al., 1993; Weltman et al., 1977). All three studies used a cycle ergometer at various exercise intensity during the work phase with a moderate intensity during the recovery. Ahmaidi et al. (1996) subjects performed incremental aerobic test and force velocity test followed by an active recovery of pedaling on cycle ergometer at 32% VO_{2max}. Thireit et al. (1993) male gymnast performed both arm or leg pedaling at 130% VO_{2max} with active recovery consisting of arm or leg pedaling at 30% VO_{2max}. The third study by Weltman et al. (1977) had the subjects perform an all-out pedaling task at 5.5 kg resistance for 1 min which was followed by active recovery of pedaling at 1.0 kg at 60 rpm.

The HR_{max} found in the present study had a mean value of 87.9% HR_{max}, while Ahmaidi et al. (1996) reached 98% HR_{max}. The mean HR_{max} of 87.1% for period 1 was well below HR_{max} values found during game situation for elite boys and intercollegiate

26

players. These players reached on ice values of 95% HR_{max} and in most cases the HR_{max} was reached during each shift (Paterson, 1979; Green et al., 1976).

The following studies reported no enhancement in performance despite a significant drop in lactate following active recovery. These studies all reached high lactate values following exercise that ranged between 9.1 – 12.1 mmol/L (Bond et al., 1991; Siebers et al., 1981; Watson et al., 1986; and Weltman et al., 1983). Weltman and Regan's (1983) study produced lactate levels of 9.1 to 10.2 mM following a cycling ergometer test performed at 92-94% VO_{2max}. Another study of female swimmers exercising (swimming) at 90% VO_{2max} produced lactate values of 9.9 to 11.3 mmol/L (Siebers et al., 1981). Bond et al. (1991) male subjects pedalled on a cycle ergometer at 150% VO_{2max} obtained lactate values of 9.1 to 9.2 mM. Weltman et al. (1977) demonstrated lactate levels of 115-132 mg% in non-athletic males.

The incapacity to enhance lactate removal with active recovery may be due to the self-selection exercise intensity used during the active recovery. Possibly the intensity used was to light to maintain blood flow through the active muscles. Belcastro and Bonen (1975) and Bonen and Belcastro (1976) found that effective removal of lactate during recovery can be accomplished by using a self-selected exercise intensity. Watson and Hanley (1986) demonstrated a reduction in lactate when bench stepping was used during recovery, but the controlled skating recovery did not show a reduction in lactate versus resting values. They believed that the high degree of gliding during the controlled skating during recovery might not have produced the same degree of oxidative fiber involvement which may have limited lactate removal.

The appropriate intensity for active recovery to provide mainly aerobic metabolism and avoid build up of lactate was suggested by Skinner and McLellan (1980) to be at a heart rate of 130-150 bpm which is equivalent to the aerobic threshold in younger athletes. Heart rate in the present study during active recovery only reached 117 bpm, which may explain the lack of enhanced lactate removal. The HR of 117 bpm was 59% of the HR_{max} which according to Paterson (1979) is just below the recovery heart rates (60 to 75% HR_{max}) found between shifts of a game situation. Consequently the exercise intensity used by the present subjects in recovery may have been too low to affect the lactate level.

Several limitations occurred in this study. The relatively low lactate level achieved following the first skating test (7.9 mmol/L) might be due to the player remaining on the ice between shifts which simulated an active recovery between shifts. Lactate levels may have dropped between shifts thereby minimizing peak lactate. Consequently this study did not fully simulate the true game situation of sitting on the player's bench between shifts. The design of the study included sitting on the bench between shifts but due to an unexpected failure in the heating system at the ice rink cooled the rink temperature to 0° C, which made it very difficult for players to remain warm between shifts. Consequently, they requested staying on the ice and moving between shifts. The easy gliding on the skates between shifts likely provided a higher degree of lactate removal than anticipated. This effect was seen in the Watson and Hanley, (1986) study. They found that controlled skating during recovery provided a drop in lactate following recovery, but it was not significantly different the passive

recovery.

Another problem with the cold temperature is that it caused malfunctions with the lactate analyzer. The battery-operated equipment malfunctioned at times causing missing data for several subjects. This explains the sample size of 12 for lactate analysis.

Watson and Hanley (1986) showed a greater skating distance in the first work bout (period 1) for all three recovery mode (M = 334 ± 10 m bench-stepping, 334 ± 8 m skating, and 337 ± 12 m rest), while the second work bout (period 2) distance skated was 330 ± 9 m, 330 ± 9 m, and 330 ± 102 m, respectively. The present study found a greater skating distance in the second period following both active and passive recovery (231 ± 17 m and 227 ± 16 m, respectively) than the distance skated in period 1 (225 ± 16 m and 224 ± 17 m, respectively). The present study and Watson and Hanley (1986) both found no significant difference in distance following active or passive recovery. The present study did find a significantly ($p=0.002$) greater skating distance in period 2 for active and passive recovery may be due to a self pacing mechanism by the players. Most players consistently skated nearly the same distance each shift until the last shift here they pushed themselves to achieve a greater distance. This pacing mechanism was more noticable when the players skated on the second test day. A greater distance skated in the second period probably explains the significantly higher HR and lactate in period 2 than in period 1 ($p=0.001$). A trend for greater distance skated following active recovery (see figure 4.) occured. A greater distance was skated in shifts 2 through 7 for active versus passive recovery.

Although no significant relationships were found between the delta lactate (i.e,

peak LA - LA at 12-15 min) during active recovery and skating distance in period 2 ($r = 0.11$, $p>0.05$). These results indicate a weak trend for greater skating distance to be associated with a greater delta lactate. The correlation of delta lactate during active recovery to HR in the second period also displayed no significant relationship ($r = 0.21$, $p>0.05$), but also indicated that a greater delta lactate was associated with a greater HR.

Heart rate during the second period showed no significant relationship to 12-15 min lactate for either active or passive recovery ($r = -0.09$ and $r = 0.15$, $p>0.05$, respectively). Distance skated in period 2 for active recovery had no relationship to 12 min lactate values ($r = -0.32$, $p = 0.19$). However, a weak trend exists for greater skating distance to be related with lower lactate level at 12-15 min. Skating distance following passive recovery also had no relationship to 12 min lactate values ($r = -0.31$, $p = 0.22$). Both skating distance and HR were negative correlations that weakly support a trend for greater skating distance to be associated with lower 12-15 min lactate.

Chapter VII

Summary, Recommendations, and Conclusions

The purpose was to determine the affect of active and passive recovery on lactate concentration and subsequent skating performance of repeated work bouts. It was hypothesized that lactate removal would be enhanced by active recovery and a greater distance would be skated in period 2 following active recovery. Active recovery provides an increase blood flow which allows more lactate to be carried away by the lactate shuttle, so it was hypothesized that this greater removal would allow the players to skate further. The subjects were college players from the University of Nebraska at Omaha NCAA Division I team. Using a repeated measures design the subjects performed a series of skating tests before and after 15 minutes recovery. The skating test consisted of seven shifts lasting 40 seconds with 90 seconds of rest between each shift. The active recovery (low intensity cycling) or passive (sitting) lasted for approximently 12 to 15 minutes followed by a second identical skating test. Results indicated that active recovery had no effect on lactate removal or skating distance.

Further evaluation is needed to determine whether active recovery can truly play a role on enhanced performance following repeated work bouts. To evaluate this the intensity of the exercise, intensity of the active recovery, and the recovery period should be examined extensively. Another recommendation when conducting a study on athletes is request the presence of one of the coaches to aid in motivation.

A trend appeared for greater skating distance in period 2 when active recovery was used, but the effect was not significant. Consequently, the following conclusions are

warranted:

1. Active recovery did not enhance subsequent performance of repeated work bouts.

2. Lactate removal was not enhanced by active recovery in comparison with passive recovery.

References

Ahmaidi, S., Granier, P., Taoutaou, Z., Mercier, J., Dubouchaud, H., & Prefaut, C. (1996) Effects of active recovery on plasma lactate and anaerobic power following repeated intensive exercise. **Medicine and Science in Sports and Exercise.**, 28; 450-456.

Belcastro, A.N., & Bonen, A. (1975) Lactic acid removal rates during controlled and uncontrolled recovery exercise. **Journal of Applied Physiology**, 39; 932-936.

Billat L.V. (1996) Use of blood lactate measurements for prediction of exercise performance and for control of training: Recommendations for long-distance running. **Sports Medicine**, 22; 157-175.

Bond, V., Adams, R.G., Tearney, R.J., Gresham, K., & Ruff, W. (1991) Effects of active and passive recovery on lactate removal and subsequent isokinetic muscle function. **Journal of Sports Medicine and Physical Fitness**, 31; 357-361.

Bonen, A., & Belcastro, A.N. (1976) Comparison of self-selected recovery methods on lactic acid removal rates. **Medicine and Science in Sports**, 8; 176-178.

Cox, M.H., Miles, D.S., Verde, T.J., Levine, M.J., & Bartolozzi, A.R. (1993) Physical and physiological characteristics of NHL players over the last decade. **Medicine and Scence in Sports and Exercise**, 25; Suppl, 951.

Cunningham, D.A., Telford, P., & Swart, G.T. (1976) The cardioplumonary capacities of young hockey players: age 10. **Medicine and Science in Sports**, 8; 23-25.

Daub, W.B., Green, H.J., Houston, M.E., Thomson, J.A., Fraser, I.G., & Ranney, D.A. (1983) Specificity of physiologic adaptations resulting from ice-hockey training. *Medicine and Scence in Sports and Exercise*, 15; 290-294.

Gupta, S., Goswami, A., Sadhukhan, K., & Mathur, D.N. (1996) Comparative study of lactate removal in short term massage of extremities, active recovery and a passive recovery period after supramaximal exercise sessions. **International Journal of Sports Medicine**, 17; 106-110.

Green, H.J. (1979) Metabolic aspects of intermittent work specific regard to ice hockey. **Candian Journal of Applied Sports Science**, 4; 29-34.

Green, H.J., Daub, B.D., Painter, D.C., & Thomson, J.A. (1978) Glycogen depletion patterns during ice hockey performance. **Medicine and Scence in Sports and Exercise**, 10; 289-293.

Green, H., Bishop, P., Houston, M., McKillop, R., Norman, R., & Stothart, P. (1976) Time-motion and physiological assessments of ice hockey performance. **Journal of Applied Physiology.** 40; 159-163.

Hogan, M.C. & Welch, H.C. (1984) Effect of varied lactate levels on bicycle ergometer performance. **Journal of Applied Physiology: Respiratory Environmental Exercise Physiology,** 57; 507-513.

Houston, M.E., & Green, H.J. (1976) Physiological and anthropometric characteristics of elite canadian ice hockey players. **Journal of Sports Medicine,** 16; 123-128.

Jacobs, I. (1986) Blood lactate. Implications for training and sports performance. **Sports Medicine,** 3; 10-25.

Klausen, K., Knuttgen, H.G., & Forster, H.V. (1972) Effect of pre-existing high blood lactate concentration on maximal exercise performance. **Scandavian Journal of Clinical Laboratory Investation,** 30; 415-419.

Karlsson, J., Bonde-Petersen, F., Henriksson, J., & Knuttgen, H.G. (1975) Effects of previous exercise with arms or legs on metabolism and performance in exhaustive exercise. **Journal of Applied Physiology,** 38; 763-767.

Paterson, D.H. (1979) Respiratory and cardiovascular aspects of intermittent exercise with regard to ice hockey. **Candian Journal of Applied Sports Science,** 4; 22-28.

Rhodes, E.C., Cox, M.H., & Quinney, H.A. (1986) Comparison of national hockey league regular players on selected physiological parameters. **Medicine and Scence in Sports and Exercise.,** Suppl 46; 274.

Rhodes, E.C., Cox, M.H., & Quinney, H.A. (1988) Physiological monitoring of national hockey league regulars during the 1985-86 season. **Candian Journal of Applied Sports Science,** Suppl, 36P.

Seliger, V., Kostka, V., Grusova, D., Kovac, J., Machovcova, J., Pauer, M., Pribylova, A., & Urbankova, R. (1972) Energy expenditure and physical fitness of ice-hockey players. **Internationale Zeitschrift Fuer Angewandte Physiologie Einschliesslich Arbeitsphyiologie,** 30: 283-291.

Siebers, L.S. & McMurray, R.G. (1981) Effects of swimming and walking on exercise recovery and subsequent swim performance. **Research Quarterly Exercise and Sports,** 52; 68-75.

Skinner, J.S. & McLellan, T.M. (1980) The transition from aerobic to anaerobic

metabolism. **Research Quarterly,** 51; 234-248.

Smith, D.J., Quinney, H.A., Steadward, R.D., Wenger, H.A., & Sexsmith, J.R. (1982)
Physiological Profiles of the Canadian Olypic hockey team (1980) **Candian Journal of
Applied Sports Science,** 7; 142-146.

Thiriet, P., Gozal, D., Wouassi, D., Oumarou, T., Gelas, H., & Lacour, J.R. (1993) The
effect of various recovery modalities on subsequent performance, in consecutive
supramaximal exercise. **Journal of Sports Medicine and Physical Fitness,** 33; 118-129.

Watson, R.C., & Sargeant, T.L.C. (1986) Laboratory and on-ice test comparisons of
anaerobic power of ice hockey players. **Candian Journal of Applied Sports Science,**
11; 218-224.

Watson, R.C., & Hanley, R.D. (1986) Application of active recovery techniques for a
simulated ice hockey task. **Candian Journal of Applied Sports Science,** 11; 82-87.

Weltman, A. & Regan, J.D. (1983) Prior exhaustive exercise and subsequent, maximal
constant load exercise performance. **International Journal of Sports Medicine,** 4; 184-
189.

Weltman, A., Stamford, B.A., Moffatt, R.J., & Katch, V.L. (1977) Exercise recovery,
lactate removal, and subsequent high intensity exercise performance. **Research
Quarterly Exercise and Sports,** 48; 786-796.

Yates, J.W., Gladden, L.B., & Cresanta, M.K. (1983) Effects of prior dynamic leg
exercise on static effort of the elbow flexors. **Journal of Applied Physiology:
Respiratory Environmental Exercise Physiology,** 55; 891-896.

<center>**Appendix A**</center>

IRB #: <u>339-98</u>
ADULT INFORMED CONSENT FORM

THE EFFECT OF ACTIVE RECOVERY ON LACTATE CONCENTRATIONS AND SUBSEQUENT PERFORMANCE OF REPEATED WORK BOUTS IN UNIVERSITY ICE HOCKEY PLAYERS

INVITATION TO PARTICIPATE

You are invited to participate in this research study. The following information is provided in order to help you make an informed decision whether or not to participate. If you have any questions please do not hesitate to ask.

BASIS FOR SUBJECT SELECTION

You are eligible to participate in this study because you are a healthy male ice hockey players between the ages of 19 and 29 years. You may participate only if you are free from any cardiovascular, metabolic, and/or muscle or joint risk factors.

PURPOSE OF THE STUDY

The purpose of this study is to determine the effect of active recovery on lactate concentrations and subsequent performance of repeated work bouts in university ice hockey players.

EXPLANATION OF PROCEDURES

You will be asked to avoid eating food for four hours prior to testing and to avoid heavy exertion or exercise 12 hours prior to participation. After obtaining an informed consent, you will be asked to complete a concise medical and exercise history. You will be required to wear full hockey gear and carry your stick during the test. During this time you will be asked to provide blood for lactate analysis by finger puncture. A heart rate monitor will then be attached to the chest. You will then be allowed to warm up and then skate the test course.

You will be asked to perform a series of skating tests before and after 15 minutes of recovery. The skating test will consist of eight shifts lasting 50 seconds with 90 seconds of rest between each shift. A recovery period of active or passive lasting for approximately 15 minutes will follow. The recovery performed will be randomly assigned prior to starting the test. Active recovery will consist of pedaling on a bicycle at

a self selected pace between 50 and 75 rpm for about 10 minutes, while passive recovery is sitting on the players bench for 15 minutes. After completion of the recovery period the second period will begin and you will be asked to perform the same skating task performed in the first period.

Blood samples will be collected via a finger puncture before exercise, about 3 minutes following the first period and upon completion of the recovery period.

You will be asked to perform both types of recovery (active or passive), which means that you will perform the test twice with at least two days rest separating tests.

POTENTIAL RISKS AND DISCOMFORTS

The following are the risks and discomforts you could potentially experience during this study:

Maximal skating intervals: Possible risks associated with maximal skating intervals include heart attack, abnormal heart rhythms, abnormal blood pressure, stroke, shortness of breath, dizziness, reduced coordination, muscle soreness and musculoskeletal injuries from a fall.

Finger stick blood sampling: There may be minor pain associated with the finger stick. There is also a chance of bruising and a slight possibility of infection.

POTENTIAL BENEFITS TO SUBJECTS

You may benefit by obtaining information on which type of recovery might aid your performance while playing ice hockey.

POTENTIAL BENEFITS TO SOCIETY

Others team sports like basketball, football, and soccer may also benefit from this study. Any sport that requires an athlete to perform at a high intensity and then stop before performing another bout of highly intense exercise may benefit from this study.

IN CASE OF EMERGENCY CONTACT PROCEDURE

In the event of a research related injury or if you experience an adverse reaction please immediately contact one of the investigators listed at the end of the consent form.

EMERGENCY CARE AND COMPENSATION IN CASE OF INJURY

If you are injured or have an adverse reaction because of this research emergency medical treatment will be available at the Nebraska Health System (NHS) hospitals at no cost to

you. No additional compensation will be provided. Agreeing to this does not mean you are giving up any legal rights you may have.

FINANCIAL OBLIGATIONS

The tests will be provided to you free of charge.

ASSURANCE OF CONFIDENTIALITY

Any information obtain during this study which could identify you will be kept strictly confidential. The information obtained during the study may be published in a scientific journal or presented at meetings but your identity will be kept strictly confidential. The Institutional Review Board (IRB) has access to the research records of the subject.

RIGHTS OF RESEARCH SUBJECTS

Your rights as a research subject have been explained to you. If you have any questions concerning the rights of research subjects you may contact the University of Nebraska Institutional Review Board (IRB), (402) 559-6463.

VOLUNTARY PARTICIPATION AND WITHDRAWAL

You are free to decide not to participate in this study or to withdraw at any time without adversely affecting relationship with the investigators or the University of Nebraska. Your decision will not result in loss of benefits to which you are otherwise entitled.

DOCUMENTATION OF INFORMED CONSENT

YOU ARE VOLUNTARILY MAKING A DECISION WHETHER OR NOT TO PARTICIPATE IN THIS RESEARCH STUDY. YOUR SIGNATURE CERTIFIES THAT THE CONTENT AND MEANING OF THE INFORMATION ON THIS CONSENT FORM HAVE BEEN FULLY EXPLAINED TO YOU AND THAT YOU HAVE DECIDED TO PARTICIPATE HAVING READ AND UNDERSTOOD THE INFORMATION PRESENTED. YOUR SIGNATURE ALSO CERTIFIES THAT YOU HAVE HAD ALL YOUR QUESTIONS ANSWERED TO YOUR SATISFACTION. IF YOU THINK OF ANY ADDITIONAL QUESTIONS DURING THIS STUDY PLEASE CONTACT THE INVESTIGATORS. YOU WILL BE GIVEN A COPY OF THIS CONSENT FORM TO KEEP.

_____   _____
Signature of Subject   Date

IN MY JUDGMENT THE SUBJECT IS VOLUNTARILY AND KNOWINGLY GIVING INFORMED CONSENT AND POSSESSES THE LEGAL CAPACITY TO

GIVE INFORMED CONSENT TO PARTICIPATE IN THIS RESEARCH STUDY.

_____   _____
Signature of Witness   Date

IN MY JUDGMENT THE SUBJECT IS VOLUNTARILY AND KNOWINGLY
GIVING INFORMED CONSENT AND POSSESSES THE LEGAL CAPACITY TO
GIVE INFORMED CONSENT TO PARTICIPATE IN THIS RESEARCH STUDY.

_____   _____
Signature of Investigator   Date

Primary Investigators
Shelle Lau, B.S. Graduate Student

Secondary Investigator
Kris Berg, Ed. D. Professor