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The effect of static stretching on lactate removal during recovery from high intensity exercise

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The Effect of Static Stretching on Lactate Removal During Recovery From High Intensity Exercise

by

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in

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ABSTRACT

The purpose of this study was to compare the effectiveness of static stretching on lactate removal following high intensity exercise to a passive and a low intensity cycling recovery. Eight male subjects, ages 19-50, with an average $VO2_{max}$ of 46.1 $\pm$ 8.2 ml/kg.min., performed a high intensity cycling protocol followed by one of the three possible recovery periods: sitting, stretching, or cycling. To determine blood lactate concentration, a fingertip blood sample was taken at minute 0, 3, 8, 16, 24, and 32 of the recovery periods. Blood lactate was significantly lower in the cycling and stretching recoveries versus the sitting recovery. Compared to the sitting recovery, lactate half-time was 44% faster in the cycling recovery and 24% faster in the stretching recovery. These results indicate that stretching provides moderate benefits in the reduction of lactate following exercise.

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CHAPTER 1
INTRODUCTION

Lactate concentration in muscle and blood increases during exercise. At high intensities of exercise, the amount of lactate produced cannot be balanced by utilization; lactate accumulates and fatigue results. The type and length of recovery period following exercise affect the rate of lactate clearance. Unless lactate levels are lowered to reasonable levels before an additional bout of exercise, an individual's subsequent performance will be compromised.

The ability to lower lactate levels effectively is important in many athletic events. For example, track competitions often require athletes to perform several heats before a final and in ice hockey, players are sent out in shifts. Lactate that is not utilized from initial exercise bouts will become a negative factor in later exercise.

An active recovery lowers blood lactate levels more effectively than a passive recovery. Lactate can be converted to pyruvate directly in the active muscles and used as an energy source. Moderate exercise during recovery also increases blood flow which enhances the transport of lactate to sites where it can be utilized: the liver and other skeletal muscles. Lactate can be oxidized or serve as a gluconeogenic precursor at these distal locations.

Stretching is a useful activity for increasing flexibility and is often recommended as a warm-up or cool down activity. Since holding stretched positions statically requires muscular effort, it may be considered intermediate between an active and non-active recovery. The purpose of this study was to determine the effects of a static stretching routine on lactate removal following exercise.

Statement of the Problem

Lactate removal from blood during recovery from high intensity exercise is faster when low-intensity exercise is used than when recovery is passive or non-active. Static stretching is an activity requiring muscular effort but which is passive in nature.

Purpose of the Study

The purpose of this study was to evaluate the effectiveness of a static stretching program on lactate removal during recovery from high intensity exercise relative to a passive and a low intensity exercise recovery program.

Need for the Study

Stretching is a popular activity following exercise. If another bout of exercise is to be performed, though, the time spent stretching

needs to be justifiable. If lactate levels are not lowered sufficiently before subsequent exercise, performance will suffer. Consequently the time the individual spent stretching could have been better spent by lowering lactate levels through proven low intensity aerobic activities.

It is currently not known how effective stretching exercises are in utilizing lactate. This study was designed to see if stretching produces effects more similar to a passive or a proven active recovery. This information would provide guidance in the selection of stretching as a recovery exercise.

Limitations

1. Since the sample size was relatively small and homogeneous, the generalization of the results should be done with caution.

2. Twenty static stretching exercises were used. The results may not be the same for other types of stretches or for stretches performed in a different manner.

Assumptions

1. Subjects gave a maximum effort on the $VO2_{max}$ test.

2. Subjects performed the stretching exercises as instructed.

3. 35% of $VO2_{max}$ is an optimal intensity of exercise for lactate removal on a cycle ergometer (Belcastro & Bonen, 1975).

4. Peak blood lactate levels occur 3 minutes after cessation of high intensity, interval cycling (Koziris & Montgomery, 1991).

5. Lactate levels peaked at similar values in each subject following the high intensity exercise protocol.

Statement of Hypotheses

Null Hypothesis:

H_0: Blood lactate concentration is the same after static stretching as after a passive recovery.

Research Hypotheses:

H_1: Blood lactate concentration will be lower after stretching exercises than after passive recovery.

H_2: Blood lactate concentration will be lower after low-intensity aerobic exercise than after stretching exercise.

Definition of Terms

Static Stretching is the holding of stretches for approximately 15 seconds at the point where muscle tension prevents further movement.

Low Intensity Aerobic Exercise is exercise at 35% of $VO2_{max}$.

High Intensity Exercise is exercise at 120% of $VO2_{max}$.

CHAPTER 2
REVIEW OF THE RELATED LITERATURE

Introduction

Lactate is a commonly used measure in exercise studies. Stretching, as an accepted component of physical fitness, has also been studied extensively. Related literature is presented under the following topics: (1) Role of Lactate: Historical Perspective, (2) Lactate and Exercise Performance, (3) Fate of Lactate During Recovery, (4) Rate of Lactate Removal, (5) Static Stretching, (6) Stretching During Recovery, (7) Summary.

Role of Lactate: Historical Perspective

In a series of experiments, Fletcher & Hopkins (1907) demonstrated that the amount of lactic acid in excised frog muscle would increase in an anaerobic environment, but would not develop when oxygen was present. A rise in lactic acid levels was also noted when frog muscles were contracted to fatigue. Early studies came to the conclusion that lactic acid resulted from the breakdown of muscle glycogen in the absence of oxygen (Hill & Lupton, 1923). It was

determined that the amount of glycogen that disappeared during muscular activity equaled the amount of lactic acid that was produced. During recovery from activity, the majority of the lactic acid in a muscle would be converted back to glycogen while a small fraction would be oxidized. The term oxygen debt was used to describe the amount of excess oxygen consumed following an activity. The oxidation of the lactic acid remaining in muscle following activity was equilibrated with the oxygen debt (Hill & Lupton, 1923).

Margaria, Edwards, and Dill (1933) divided oxygen debt into an alactacid and lactacid component. They asserted that excess lactic acid was not produced at workloads less than two thirds of the maximum metabolic rate and consequently could not be responsible for oxygen debt at these low workloads. An alactacid debt was thought to be repaid rapidly following exercise and was hypothesized to be due to the oxidation of fuels other than lactic acid to resynthesize endogenous phosphagens. The lactacid debt was correlated with the slow decline in oxygen consumption and was believed to be due to the energy needed for the resynthesis of glycogen in muscle.

A different fate of lactate in muscle was proposed by Cori and Cori (1929). In the "Cori cycle" lactate leaves the muscle, circulates through the bloodstream to the liver where it is resynthesized to glucose for future use in muscles. Krebs (1964) provided evidence supporting the Cori cycle in a study involving the gluconeogenic

capacity of rats. After exercise which increased lactate levels, the rate of gluconeogenesis in rat kidney cortex was found to increase significantly. Krebs and Woodford (1964) also argued that lactate must be converted to glycogen in the liver and kidney because several key enzymes necessary to the process were not present in muscle.

Lactate kinetics and the role of lactic acid in oxygen debt have evolved from earlier beliefs. The production of lactate is no longer thought to be dependent on a lack of oxygen, but can be produced when more than adequate oxygen is available (Brooks, 1986). Lactate is present during rest and exercise as a result of carbohydrate breakdown in the glycolytic pathway. The end result of the breakdown of glycogen is pyruvate. The fate of pyruvate is determined by two competing enzymes: lactate dehydrogenase (LDH) and pyruvate dehydrogenase (PDH). The majority of pyruvate is converted to acetyl CoA by PDH for oxidation in the Krebs cycle, while only a small percentage forms lactic acid via LDH. However, the rate of lactic acid production does increase when excess pyruvate is present such as during high intensity exercise or when oxygen supply is limited. Under these conditions there is an increased dependence on anaerobic glycolysis which creates more pyruvate than can be converted by PDH for oxidation in the Krebs cycle. The result is increased action of LDH on the pyruvate present and an increased formation of lactic acid.

The role of lactic acid in oxygen debt is no longer considered exclusive but one of a number of factors. Excess post-exercise oxygen consumption may be affected by creatine phosphate resynthesis, levels of catecholamines, and elevated tissue temperature, as well as lactate metabolism. Brooks (1991) has put forth a "lactate shuttle hypothesis" which describes lactate as a beneficial metabolic intermediate during sustained exercise. Lactate can be oxidized locally in muscle fibers or be quickly transported to other distal oxidative muscle fibers to serve as an energy source. This acts to both clear lactate from the system and spare the utilization of other stored fuels. Lactate also serves an important role in gluconeogenesis which can occur in the liver as well as skeletal muscle. Lactate is shuttled to the sites where it can be put to use immediately as metabolic substrate or be stored for later use.

Lactate and Exercise Performance

At rest, lactate concentration is usually 1 mmol per liter of blood, while blood lactate levels as high as 30 mmol have been measured after multiple bouts of intense exercise (Gollnick, Bayly, & Hodgson, 1986). During sustained light exercise, lactate concentration will only increase slightly as there is a balance between production and utilization. With increasing intensity of exercise, steady state blood lactate concentration can no longer be maintained. The point at which the rate of lactate production

exceeds the rate of clearance has been termed the anaerobic or lactate threshold. This has been defined as an absolute blood lactate concentration of 2 mmol to 4 mmol, or the intensity at which lactate concentration begins to increase. The exact percentage of $VO2_{max}$ at which this occurs is variable and depends on an individual's state of training (Hurley et al., 1984). Lactate concentration will continue to increase until the individual cannot continue to exercise.

Exercise fatigue is a complex topic as there is the possibility of both central and peripheral origins. The excess production of lactic acid during strenuous exercise plays a role in peripheral muscular fatigue. A significant correlation has been demonstrated between increasing lactate levels and decreasing muscle contractile force (Fitts & Holloszy, 1976). Lactic acid, at physiological pH, dissociates to lactate and hydrogen ions. It is the hydrogen ions that have two main negative effects on muscle contractility: decreasing the pH level of muscle and interfering with the binding of calcium to troponin. A decreased pH level impairs the activity of enzymes in the glycolytic pathway and in the contraction process. The affinity of phosphofructokinase for fructose-6-phosphate has been shown to be reduced when pH levels are lowered in vitro (Trivedi & Danforth, 1966). Hydrogen ions also interfere with muscle contraction by binding to the calcium binding site on troponin which in turn prevents actin-myosin cross bridge formation (Fuchs, Reddy, & Briggs, 1970).

Many studies have examined the effect of pre-existing high blood lactate levels on exercise performance. The specific methods and results of these studies have varied. In 1977, Weltman, Stamford, Moffat, and Katch measured the power output of untrained subjects in a 1 minute all out cycling test. After an initial bout and varied recovery periods, the power test was performed again. No significant correlation ($r = -.19$) was found between lactate level and power output on the second test. Ainsworth, Serfass, and Leon (1993) conducted a similar experiment using trained cyclists and 45 second cycling bouts. In this case there was a significant correlation between net power output and net blood lactate for a nine ($r = .6$) and twelve ($r = .63$) minute recovery period but not for a six ($r = .35$) minute break between bouts.

Klausen, Knuttgen, and Forster (1972) exercised subjects at a supra maximal level until exhaustion (approximately 5 minutes) on a cycle ergometer. Following a recovery period, blood lactate levels averaged 10 mmol and the subjects performed the same supra maximal exercise until exhaustion. No significant differences were found in maximum oxygen uptake. There was a tendency towards a reduced time to exhaustion, but it was not significant.

On the other hand, Karlsson, Flemming, Henriksson, and Knuttgen (1975) and Pendergast, Leibowitz, Wilson, and Cerretelli (1983) did find a significant reduction in performance time after lactate levels were elevated by an initial bout of exercise. Karlsson et al. (1975) employed maximal leg exercise after maximal arm

exercise and vice versa, while Pendergast et al. (1983) exercised subjects on a treadmill. However, in both studies there was no significant reduction in maximum oxygen uptake.

Overall, in regards to exercise performance, high pre-existing lactate levels do seem to reduce time to exhaustion but not maximal oxygen uptake. A relationship with power output needs further examination.

Fate of Lactate During Recovery: Oxidation vs. Glyconeogenesis

Lactate appears in muscle as an end product of glycolysis during exercise and at rest. At low intensities of exercise there is a balance between production and utilization as lactate can be oxidized by the producing muscles or distant muscles including the heart (Brooks, 1991). During intense exercise, lactate will continue to accumulate until the individual becomes fatigued. After exercise ceases, the excess lactate in the muscle and blood is removed in order to return to normal, resting levels. The specific fate of lactate after exercise has been studied by several researchers with different conclusions. There is a debate as to whether the majority of lactate is oxidized after exercise or is used to form glycogen. The specific location at which the majority of lactate is oxidized or processed to form glycogen is also under question.

The belief that lactate is primarily converted to glycogen directly in muscle was first put forth by Meyerhof in the 1920s.

Bendall and Taylor (1970) reconfirmed Meyerhof's conclusions in a study utilizing isolated frog sartorious muscles. Lactate was added to prepared bathing solutions containing the frog muscles and large quantities of glycogen were noted to form.

Similar results were documented by Hermansen and Vaage (1977) following exhaustive exercise in humans. They found that only 10% of the lactate produced in the muscle left to the bloodstream after exercise. Of the remaining lactate in the muscle, they concluded it would require an unreasonable large A-V oxygen difference to oxidize the majority of this lactate in the muscle. Since no significant muscle uptake of glucose was measured, the large increase in muscle glycogen synthesis was attributed to lactate conversion directly in the muscle.

On the other hand, research by Brooks, Brauner, and Cassens (1973) indicates that the majority of lactate is oxidized after exercise. In this study, rats were injected with labeled lactate after exhaustive exercise. During a post-exercise period of 2 hours, 75% of the labeled lactate was traced to expired CO2. Glycogen synthesis was also measured in fasted, exercise exhausted rats. No significant synthesis of glycogen in the liver or muscles was found. Based on the lack of evidence of glycogen synthesis and the labeled lactate results, it was concluded that the ultimate fate of lactate after exercise was mainly oxidative.

Brooks and Gaesser (1980) conducted a similar experiment to better quantify the end points of lactate utilization following

exhaustive exercise in rats. Results indicated that the labeled lactate was mainly oxidized (45.18%) with other significant destinations including glycogen (18.3%), $HCO3^-$ (17.72%), and protein (8.57%).

A compromise between these varied results would state that the actual fate of lactate is balanced with approximately even amounts being oxidized and used for gluconeogenesis in muscle. A study by Astrand, Hultman, Juhlin-Dannfelt, and Reynolds (1986) supports this conclusion. In this case, of a mean production of 830 mmol of lactate, 330 mmol was the maximum amount oxidized and 360 mmol was calculated to be used for muscle glycogen synthesis.

Rate of Lactate Removal

The rate of lactate removal depends on the type and intensity of recovery activity. An active recovery is superior to a passive recovery due to increased blood circulation and energy requirements. Since lactate is both oxidized and converted to glycogen at various sites, the maintenance of high blood circulation aids rapid removal; lactate is transported quickly to sites were it can be temporarily stored or utilized. High blood circulation also maintains a concentration gradient between the muscle and blood which promotes a faster removal of lactate from fatigued muscles. An active recovery period is also beneficial as the lactate can be reconverted to pyruvate and used as an energy source (Gladden, 1989).

Different exercise intensities during recovery have been studied in order to determine the optimum percentage of $VO2_{max}$ for lactate decline. An exercise recovery above the anaerobic threshold has been shown to be too high (McLellan & Skinner, 1982; Stamford, Weltman, Moffatt, & Sady, 1981). Any benefit of increased blood circulation and energy requirements will be counteracted by excess lactate production.

Bonen and Belcastro (1975) compared lactate removal at rest and at exercise intensities of 29.7%, 45.3%, 61.8%, and 80.8% of $VO2_{max}$ after 6 minutes of work on a cycle ergometer at 89% of $VO2_{max}$ They found that the 29.7% and 45.3% levels were significantly better than the others. Based on a prediction equation, they calculated that 32% of $VO2_{max}$ would be the most effective recovery intensity.

Dodd, Powers, Callender, and Brooks (1984) tested the effectiveness of a combined high and low intensity recovery: exercise at 65% of $VO2_{max}$ for 7 minutes followed by 35% for 33 minutes. While the combined recovery reduced lactate levels better than rest and a 65% $VO2_{max}$ exercise recovery, it was not significantly different that a 35% $VO2_{max}$ exercise recovery which proved to be optimal.

Hermansen & Stensvold (1972) found that a higher intensity of exercise recovery resulted in maximal lactate removal for well-trained subjects performing treadmill running. An average of 63% of

$VO2_{max}$ proved to be the most effective versus recovery exercise intensities ranging from 30% to 80% $VO2_{max}$.

Self-selected recovery exercises can take on varying intensities and also different forms such as intermittent jogging. Belcastro and Bonen (1975) found that self-selected intensities did not significantly differ in the effectiveness of lactate removal from optimal intensities. In 1976, Bonen and Belcastro compared two self-selected types of recovery: free jogging and free intermittent jogging, with a passive recovery. They found that both self-selected activities were superior to the passive recovery and further, that continuous free jogging was significantly better than intermittent activity.

In summary, the optimum intensity of recovery exercise to utilize lactate should be below an individual's anaerobic threshold and approximately 35% of $VO2_{max}$ Self-selected rates often prove just as effective. Continuous activities provide more benefit than intermittent ones. The high intensity found to be optimal by Hermansen and Stensvold may be explained by the fact that the subjects were well-trained and were still exercising well below their anaerobic threshold at 60% - 70% $VO2_{max}$ (Stamford et al., 1981).

Static Stretching

Stretching is a form of exercise. There are three general types of stretching: static, ballistic, and proprioceptive neuromuscular facilitation (PNF). Static stretching involves positioning and holding

joints at the point where muscle tension prevents further movement. Ballistic stretching uses repetitive bouncing movements to move joints from an unrestricted state to a point of muscular tension and back again. Proprioceptive neuromuscular facilitation can take on many different forms but generally consists of the alternating contraction and relaxation of muscle groups with the aid of another person. Since PNF requires assistance and ballistic stretching has been associated with muscle soreness, static stretching can be considered a more convenient, safer method (de Vries, 1986).

Static stretching has been shown to increase the range of motion around a joint (de Vries, 1962). Adequate flexibility is necessary for athletic performance; if body segments cannot be moved through requisite ranges, performance will be impaired. In sports such as gymnastics, the requirements for joint flexibility are extreme which necessitates superior ranges of motion (Shellock & Prentice, 1985). Increased flexibility not only allows movements to be correctly performed but can benefit athletic performance from a biomechanical perspective: a larger range of motion allows increased velocity and momentum to be generated over the movement. Chances of injury during exercise are also reduced by increased flexibility as an adequate range of motion provides a measure of safety against over extension and joint damage (Alter, 1988).

Stretching During Recovery

Stretching is logically performed during a recovery period from exercise as connective tissues are more elastic after a rise in tissue temperature. Stretches can be performed more effectively and safely after exercise has raised body temperature (Shellock & Prentice, 1985). Stretching following exercise has also been linked to the relief of muscle soreness. There is a debate about the actual causes of muscle soreness as several theories have been presented. De Vries (1961b) conducted a study which provides evidence for a role of static stretching in the prevention of soreness. Subjects performed unaccustomed exercise in both arms after which one arm was stretched while the other arm served as a control. Muscular distress was reported to be significantly less in the stretched arm. In another related study, de Vries (1961a) used electromyographic analysis to study muscle activity before and after static stretching. Muscle tension was found to be significantly reduced in six out of seven subjects after stretching was employed.

Since static stretching is a form of light exercise, it should have an effect on lactate kinetics. In static stretching, agonist muscles are contracted in order to stretch the antagonist muscles. Once tension is developed in the antagonist muscles, the agonist muscles must perform isometric contractions to hold the stretch. The muscles actively involved in stretching could use lactate as an energy source. The muscular requirements of stretching would also elevate heart

rate more than a passive recovery. An increased blood circulation would increase transport of lactate to other sites and also help maintain a concentration gradient between fatigued muscles and the bloodstream.

Summary

Excessive build up of lactate in muscle during exercise contributes to fatigue. Unless lactate levels are lowered before a subsequent bout of exercise, performance will be impaired. Specifically, several studies have showed a decreased time to exhaustion when exercise is performed with high pre-existing lactate levels. The rate of lactate removal is affected by the type and intensity of recovery activity. An active recovery is superior to a passive one.

Static stretching can be used as a recovery activity following exercise. The general benefits of stretching include an increased range of motion around joints and the possibility of reduced muscle tension and soreness. Stretching should also have some effect on lactate clearance since is a light form of exercise, but the presence and magnitude of any effect is currently unknown.

CHAPTER 3
METHODOLOGY

Subjects

Eight males volunteered to participate in the study. Approval to use human subjects was granted in November 1994 by the UNLV Institutional Review Board. Informed consent was obtained in accordance with the guidelines set forth by the Institutional Review Board. Subject characteristics are presented in Table 1.

Table 1 Subject Characteristics

Subject	Age	Height(in)	Weight(lbs)	$VO2_{max}$(ml/kg.min)
1	28	68.5	148	54
2	24	75	176	42.5
3	31	72	166	42.6
4	26	68.5	157	43.7
5	19	68	140	64.3
6	24	72	169	42.9
7	50	66	165	37.8
8	32	71	182.5	40.7
Mean	29	70.4	162.9	46.1
Std.Dev.	9	2.7	13.2	8.2

Design

A repeated measures design was used. Each of the subjects first performed a maximum oxygen uptake test on a cycle ergometer. The following week each participant performed a high intensity exercise protocol followed by one of three possible recovery regimens: sitting, stretching, or cycling. The subjects performed each of the three recovery regimens on separate occasions in a counterbalanced order. To determine blood lactate concentration, fingertip blood samples were taken during the recovery periods.

Procedures

The maximum oxygen uptake test was performed on a Schwinn BioDyne cycle ergometer. The subject was fitted with a Vantage heart rate monitor (Polar Inc., Stanford CT), nose clip, and head gear with a non-rebreathing valve and mouthpiece. Oxygen consumption was measured with a Vista metabolic measurement system (Vaccumed Inc., Ventura, CA). At zero resistance, the subject was allowed to practice maintaining a cadence of 75 rpm. The subject was informed that the test would be stopped when a cadence of 75 could no longer be maintained. The $VO2_{max}$ test was initiated at a resistance of 1 kp. Resistance was increased every 2 minutes based on the subject's fitness level and degree of exhaustion. Expired air was analyzed every 30 seconds. Heart rate and rating of perceived

exertion were recorded every two minutes. At the conclusion of the test, a final heart rate was recorded and the resistance was lowered to zero to allow the subject to cool down.

The following week each subject returned to the laboratory to perform the high intensity exercise protocol followed by one of the three recovery regimens. A fingertip blood sample was taken to determine resting blood lactate concentration for use as a baseline. The high intensity exercise was performed on a Schwinn BioDyne cycle ergometer. Heart rate was monitored with a Vantage heart rate monitor. The subject was told to maintain a cadence of 75 rpm during the test. After a three minute warm-up at .5 kp, the resistance was set to a workload equivalent to 120% of the subject's $VO2_{max}$. The subject was instructed to maintain the 75 rpm cadence for 45 seconds. After 45 seconds, the subject was told to stop pedaling and sit motionlessly on the cycle ergometer for 30 seconds. After the rest period, the subject exercised again at 120% $VO2_{max}$ followed by another rest period, and then a final identical exercise bout.

Immediately following the high intensity exercise the subject began one of the three possible recovery regimens. The passive recovery consisted of sitting motionlessly in a chair adjacent to the cycle ergometer just used for the high intensity exercise. In the low intensity cycling recovery, the resistance on the cycle ergometer was set to a level equivalent to 35% of the subject's $VO2_{max}$. The subject was instructed to continue pedaling at the cadence of 75 bpm. In the

static stretching recovery, the experimenter led the subject through a series of lower extremity static stretches. The routine lasted eight minutes and was repeated four times. See Figure 1 for the specific stretches and the time spent holding each stretch. Each recovery period lasted a total of 32 minutes.

Heart rate was recorded and a fingertip blood sample was taken at minute 0, 3, 8, 16, 24, and 32 into each recovery period. Just prior to collection time an alcohol wipe was used to clean one of the subject's fingertips. Wearing latex gloves, the experimenter pricked the subject's fingertip with a semi-automatic sterile lancet. Approximately 5 drops of blood were drawn into a Microtainer Brand tube with EDTA (Becton Dickinson & Co., Rutherford, NJ) within 30 seconds. After each sample of blood was taken the container was sealed, vortexed, and refrigerated for later analysis. Blood samples were diluted 1:1 with water and lactate measured on a YSI Model 23L Lactate Analyzer (Yellow Springs Instrument Co., Yellow Springs, OH).

Statistical Analysis

A 3 x 7 factorial repeated measures analysis of variance (ANOVA) was used to analyze both blood lactate and heart rate during the recovery periods. Factor A consisted of the different recovery types: sitting, stretching, and cycling. Factor B was time including a baseline, 0, 3, 8, 16, 24, and 32 minute level. Blood

lactate half-times and the percentage of peak lactate at each measurement time were calculated in each group. Lactate half-times were analyzed with a one way repeated measures ANOVA, while a factorial ANOVA (3 x 5) was used to analyze the percentage of peak lactate data. The significance level in all ANOVAs was set at $p < 0.05$. Post hoc comparisons were made using the Tukey (HSD) method. Effect sizes were calculated for the average blood lactate values, percentages of peak lactate, and lactate half-times.

Figure 1 Static Stretching Routine

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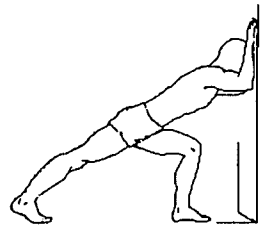

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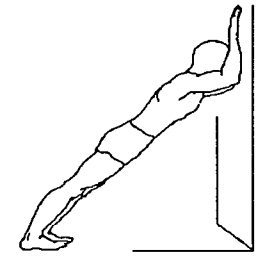

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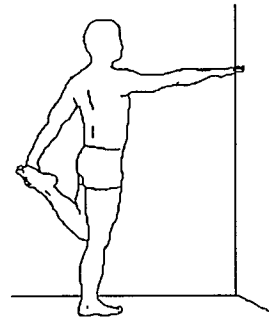

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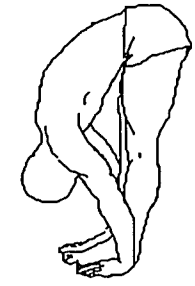

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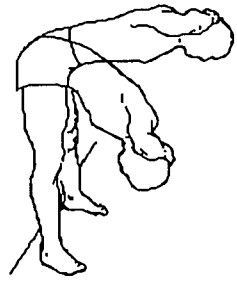

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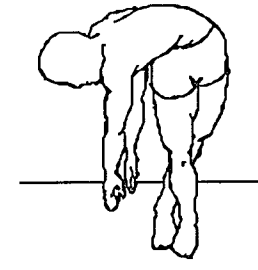

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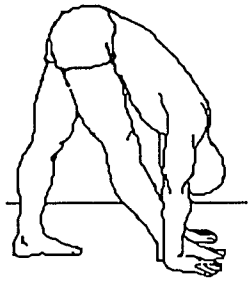

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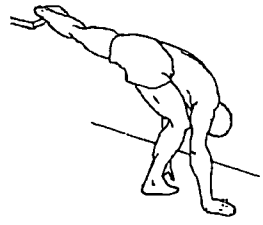

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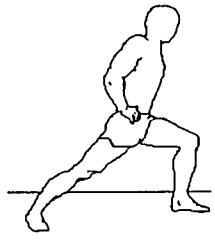

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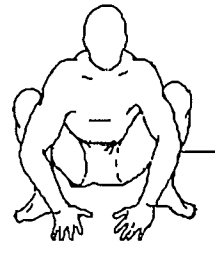

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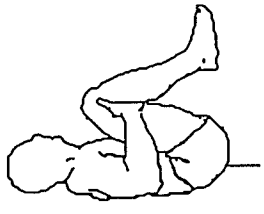

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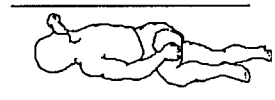

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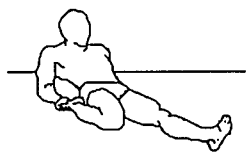

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14.

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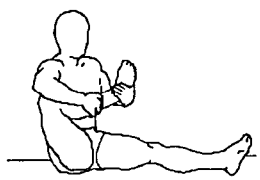

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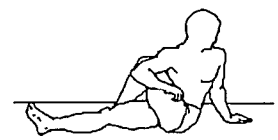

15 seconds each leg

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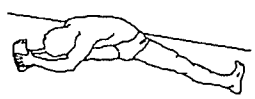

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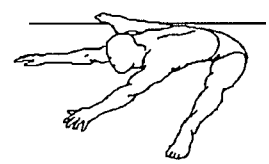

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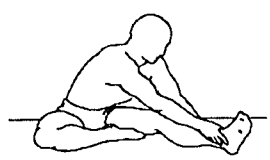

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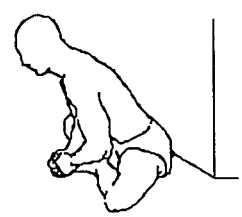
20.



15 seconds (Alter, 1988)

CHAPTER 4
RESULTS

Lactate

Average blood lactate in each recovery period is shown in Table 2 and Figure 2. The factorial ANOVA for blood lactate resulted in a significant main effect for factor A, $F_{(2,14)} = 6.24$, $p < .0115$. Blood lactate was significantly lower in the stretching and cycling recoveries compared to the sitting recovery. The main effect for factor B (time) was also significant, $F_{(6,42)} = 147.68$, $p < .0001$. Lactate levels increased significantly from baseline to peak and then declined significantly over time during the recovery periods. No significant interaction was found between recovery type and time. Effect sizes between the mean lactate values in each recovery condition from minute 8 to 32 are shown in Table 3.

Lactate half-time was calculated for each subject as the time for peak lactate to decrease by 50%. The results are shown in Table 4. There was no significant difference in blood lactate half-times between the three recovery conditions. However, compared to the sitting recovery, cycling half-time was 44% faster and stretching

half-time was 24% faster. Effect sizes for lactate half-times are shown in Table 5.

The percentage of peak lactate at minute 8, 16, 24, and 32 was calculated for each subject using minute 3 as the peak lactate value. Average percentages in each recovery period are shown in Table 6 and Figure 3. Factorial ANOVA analysis revealed a significant main effect for factor B, $F_{(3,21)} = 225.2$, $p < .0001$. The percentage of peak lactate declined significantly between each measurement time from minute 8 to 32. No significant interaction or main effect for factor A (recovery type) was found. Effect sizes between recovery conditions are shown in Table 7.

Heart Rate

Average heart rates during each of the recovery periods are shown in Figure 4. A significant interaction was found between recovery type and time, $F_{(12,84)} = 15.65$, $p < .0001$. There was no difference in baseline heart rates (75, 74, 75) or peak heart rates at time 0 (167, 167, 167) between the sitting, stretching, and cycling recoveries, respectively. At minute 8, 16, 24, and 32, the average cycling heart rate was significantly higher than the average heart rates in the stretching and sitting recovery periods.

Table 2 Average Blood Lactate In Each Recovery Condition

Average Lactate In Each Recovery Condition

	TIME (Min.)						
	Baseline	0	3	8	16	24	32
Sitting	2.3 + .3	10.6 + .4	11.3 + .4	9.9 + .3	8.1 + .5	6.4 + .4	5.1 + .5
Stretching	1.9 + .2	9.6 + .4	10.4 + .4	9.4 + .4	6.8 + .5	5.0 + .4	3.9 + .5
Cycling	2.5 + .3	9.7 + .5	10.2 + .6	8.8 + .6	6.5 + .8	4.7 + .4	4.0 + .4

Lactate values are mmol/L, mean ± SE.

Figure 2 Average Blood Lacate In Each Recovery Condition

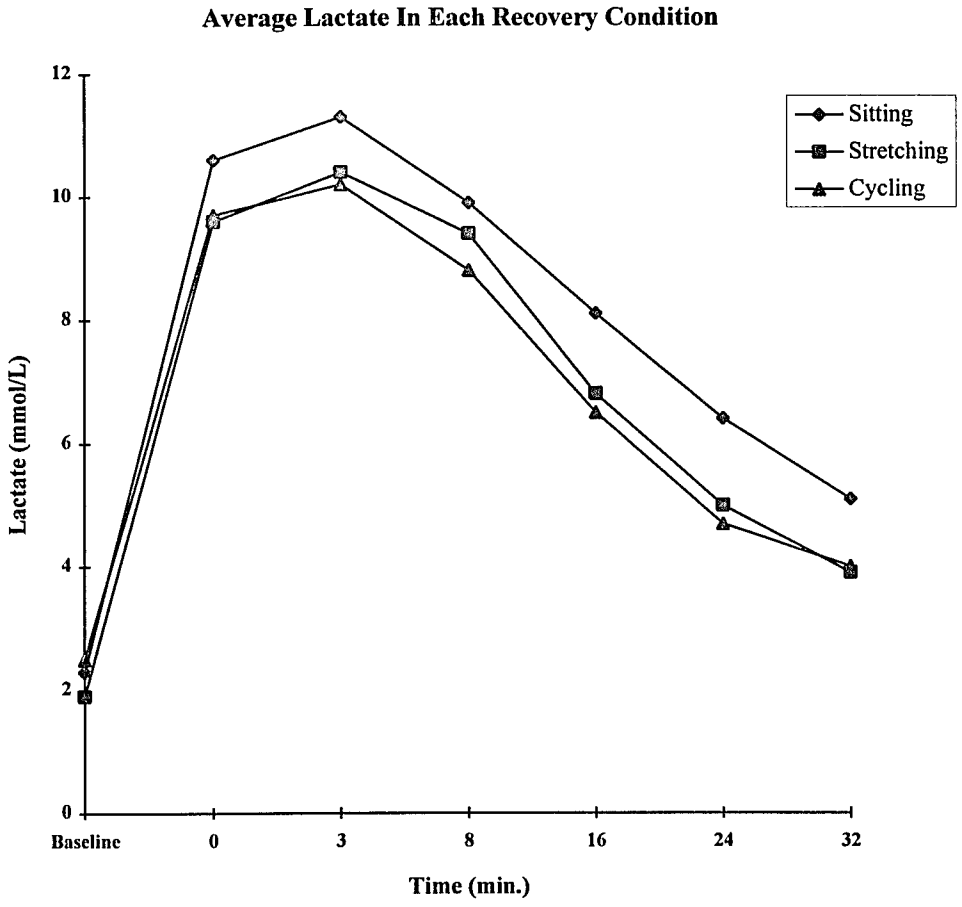


Table 3 Average Blood Lactate Effect Sizes

Comparison of Lactate Effect Sizes Between Recovery Conditions

	Sitting versus Stretching	Sitting versus Cycling	Stretching versus Cycling
8 minutes	0.49	0.79	0.39
16 minutes	0.96	0.89	0.16
24 minutes	1.27	1.7	0.27
32 minutes	0.89	0.91	0.08

Table 4 Lactate Half-times In Each Recovery Condition

Comparison of Lactate Half-times

	Recoveries		
	Sitting	Stretching	Cycling
1/2 time (min.)	26 ± 4	21 ± 3	18 ± 2

Mean ± SE.

Table 5 Lactate Half-time Effect Sizes

Comparison of Lactate Half-time Effect Sizes

	Recovery Comparisons		
	Sitting versus Stretching	Sitting versus Cycling	Stretching versus Cycling
Effect Size	0.55	0.95	0.41

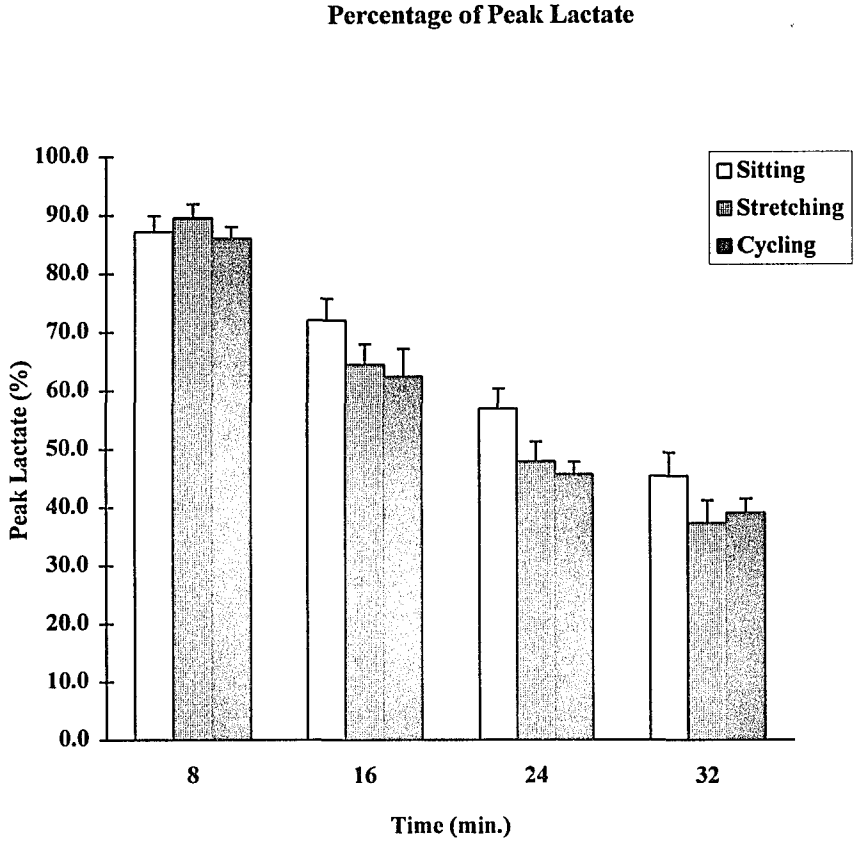
Table 6 Percentage Of Peak Lactate During Each Recovery Condition

Percentage of Peak Lactate

	Time (min.)			
	8	16	24	32
Sitting	87.2%	72.0%	56.9%	45.3%
Stretching	89.5%	64.4%	47.8%	37.2%
Cycling	86.0%	62.4%	45.6%	39.0%

Based on 100% at 3 minute measurement time.

Figure 3 Percentage of Peak Lactate During Each Recovery Condition



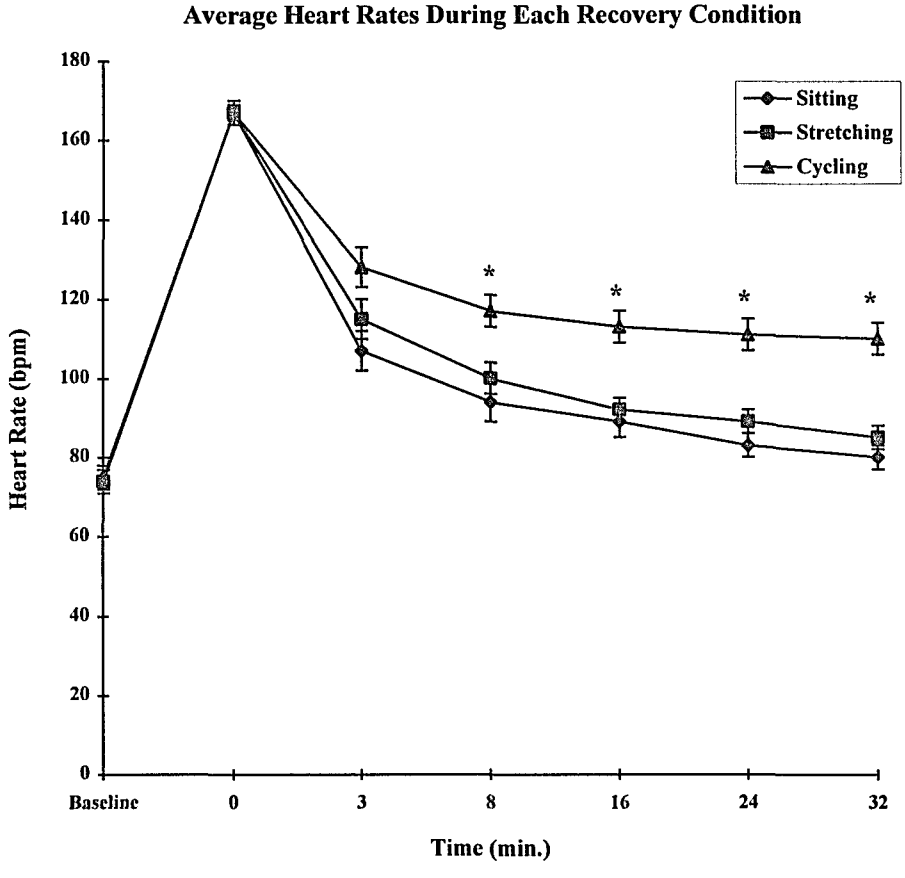
Based on 100% at 3 minute measurement time, mean + SE.

Table 7 Percentage Of Peak Lactate Effect Sizes

Comparison of Percentage of Peak Lactate Effect Sizes

	Sitting versus Stretching	Siting versus Cycling	Stretching versus Cycling
8 minutes	0.31	0.19	0.52
16 minutes	0.65	0.76	0
24 minutes	0.85	1.32	0.26
32 minutes	0.65	0.61	0.18

Figure 4 Average Heart Rates During Each Recovery Condition



* significantly different from sitting and stretching recoveries, $p<0.05$

CHAPTER 5

DISCUSSION

An active recovery results in a faster decline in blood lactate levels than a passive recovery. The basis of the faster reduction in the active recovery is an increased blood circulation and energy requirement. After exercise, excess lactate can be oxidized or converted to glycogen by skeletal muscle or the liver (Brooks & Gaesser, 1980). An elevated metabolic rate allows more lactate to be oxidized while the increased blood circulation results in a faster clearance and transport of lactate from the muscle to other sites where it can be immediately used as an energy source or stored for later use (Gladden, 1989).

In general, the results of this study are in agreement with previous research on the rate of lactate decline. Blood lactate levels in the cycling recovery were significantly lower than the levels in the sitting recovery. Recovery cycling intensity was set at 35% of $VO2_{max}$ which has previously proven more effective than a passive recovery (Belcastro & Bonen, 1975). Other studies have found significant differences in lactate between active and passive recoveries at 10 - 15 minutes into the recovery periods (Bonen

& Belcastro, 1976; Stamford et al., 1981). In this study, the effect sizes between the sitting and cycling recoveries at minute 8, 16, 24, and 32 were .79, .89, 1.7, and .91, respectively. An effect size of .2 indicates a small difference between means, .5 is a moderate difference, and .8 a large difference (Thomas, Salazar, & Landers, 1991). The large effect sizes from minute 8 to 32 indicate meaningful differences between the sitting and cycling recoveries. Heart rate in the cycling recovery was significantly higher than heart rate in the sitting recovery from minute 8 to 32. The higher heart rate in the cycling recovery is indicative of the higher energy demand and suggests increased clearance and transport of the muscle lactate to other sites.

The main purpose of the present study was to determine if stretching is effective in lowering lactate levels following high intensity exercise. Stretching, while essentially a passive activity, requires muscular effort which should elevate metabolic activity and consequently accelerate lactate decline. In fact, the results did show that stretching provided benefits compared to the sitting recovery. Blood lactate levels were significantly lower in the stretching recovery compared to the sitting recovery. Certain effect sizes also indicate that the stretching recovery was more closely aligned with the cycling recovery than the sitting recovery. At minute 16, 24, and 32 the effect sizes between the sitting and stretching recoveries were .96, 1.27, and .89 which provides evidence of a meaningful difference between means while the effect sizes between the stretching and

cycling recoveries at these same times were only .16, .27, and .08, respectively.

Heart rates during the stretching recovery were only slightly higher than the values in the sitting recovery. The small elevation in stretching heart rates provides some basis for better lactate clearance and utilization than the sitting recovery. The stretching heart rates may even be deceptively low due to the timing of the measurement. The stretching heart rate will have varied during the different standing and sitting stretches due to the positioning of the body. The actual measure was taken at the end of group of sitting stretches which may have resulted in a lower heart rate than if the measure was taken during the standing stretches.

Lactate half-times provide information on the rate of lactate decline. No significant differences were found in half-times between the three recovery conditions. However, effect size analysis indicates that there are meaningful differences between the half-times. The effect size between the sitting half-time (26 minutes) and the cycling half-time (18 minutes) was .95 which indicates a large difference. Effect sizes between the sitting versus stretching recovery and cycling versus stretching recovery were .55 and .41, respectively. This suggests that the stretching half-time is intermediate between the other two recoveries. In relation to the sitting recovery, the cycling recovery results in a superior rate of decline while the stretching recovery provides moderate benefits.

The percentages of peak lactate at minute 8, 16, 24, and 32 were also not significantly different between the three recovery conditions. The stretching and cycling percentages were consistently lower than the sitting values from minute 16 through 32, though. Effect sizes during these times show moderate to large differences between the stretching and cycling recoveries versus the sitting recovery while only small differences are present between the stretching and cycling recoveries.

The relative work intensity attained during the stretching recovery was not determined. Previous studies have disagreed on the optimal intensity at which to work in order to lower lactate levels during recovery; effective intensities have ranged from 30% - 60% of $VO2_{max}$ (Bonen & Belcastro, 1975; Hermansen & Stensvold, 1972). At very high intensities, there is the possibility that the anaerobic threshold will be crossed and lactate production will exceed utilization. Heavy workloads may increase blood flow to the heart and skeletal muscle which would hasten lactate decline, but blood flow to the liver may also be significantly reduced which would impair lactate clearance. At the other extreme, very low workloads have a minimal effect on energy demands and blood flow which will have little impact on lactate removal. The stretching recovery was most likely performed at a very low percentage of $VO2_{max}$ since the cycling heart rates were significantly higher than those in both the stretching and sitting recovery. The lower workload during the stretching recovery compared to cycling can

explain the relatively slower lactate decline as not as much lactate would be needed as an immediate energy source and the corresponding lower heart rate would have a reduced effect on lactate clearance and transport rates.

The moderate benefits of stretching may also be due to the activity being intermittent rather than continuous. Each stretch was held for approximately fifteen seconds before switching to a new position. While the transition between different stretches and between sitting and standing stretches was short there was still a small rest period which would add up over the course of the recovery. A previous study has shown that lactate recovery time is significantly faster when continuous rather than intermittent activity is performed (Bonen & Belcastro, 1976).

Rapid blood lactate removal following exercise is important if another bout of exercise is to be performed. Blood lactate in muscle can impair muscle contractions by impairing glycolytic enzymes and interfering in actin-myosin cross bridge formation. Previous studies have found a reduced time to exhaustion when exercise is performed with pre-existing high lactate levels (Karlsson, Flemming, Henriksson, and Knuttgen, 1975; Pendergast, Leibowitz, Wilson, and Cerretelli, 1983). The amount of time between exercise bouts is an important factor to consider when selecting a recovery activity. In this study, from minute 8 to 32, effect size analysis indicated there was a meaningful difference between the sitting and cycling recoveries. Comparison of the sitting and stretching recovery periods showed

there was a meaningful difference starting at minute 16 and continuing to minute 32. The practical significance of this information is that given a short time (5 minutes) between intense exercise bouts it does not make a significant difference in terms of lactate reduction what type of recovery activity an individual performs. After approximately 10 minutes a low intensity aerobic recovery will effectively lower lactate while a stretching recovery will not show meaningful benefits until after approximately 15 minutes. After approximately 30 minutes there was still a difference between the sitting recovery and the stretching and cycling recoveries. However, the lactate levels are approaching resting levels by this time regardless of the type of activity performed after exercise.

While low intensity aerobic exercise has proven a superior method of lowering lactate levels, stretching is also beneficial in terms of lactate reduction. Stretching can provide other benefits following exercise as well: an increased range of motion which can enhance performance and decrease the chances of injury, and provide the possibility of reduced muscle tension and soreness. Low intensity aerobic exercise such as jogging may also be impractical in some situations where stretching can easily be performed. Overall, the selection of a recovery exercise should be based on the time between exercise bouts, any additional benefits, and ease of performance.

CHAPTER 6

CONCLUSION

The ability to lower lactate levels after an exercise bout is important to subsequent exercise performance. Excess lactate that is not utilized before the start of another bout of exercise impairs muscular contraction and causes a reduced time to exhaustion. Previous research has shown that an active recovery results in a faster decline in blood lactate levels than a passive recovery due to increased energy requirements and heart rate. Static stretching is a relatively passive activity, but does require isometric muscle contractions to hold stretches. The purpose of this study was to determine the effectiveness of static stretching in lowering lactate levels following high intensity exercise relative to a passive and low intensity cycling recovery. The low intensity cycling recovery and static stretching recovery resulted in a significantly lower lactate levels than the sitting recovery. Lactate half-time in the stretching recovery was intermediate between the half-times in the sitting and cycling recoveries. These results indicate that stretching provides moderate benefits in the reduction of lactate following exercise.

APPENDIX I

INFORMED CONSENT FORMS

INFORMED CONSENT

General Form

Title: Effect of Static Stretching on Lactate Removal During Recovery

Purpose:

You are being asked to participate in a research study designed to learn what effect stretching has on blood lactate levels following high intensity exercise. The effects of stretching will be compared to two other types of recoveries: sitting passively, and cycling at a low intensity.

Subjects:

You have been asked to participate in this study because you are in good health.

Procedures:

If you decide to volunteer, you will first perform a maximal oxygen uptake test on a stationary bicycle. This is a physically demanding test lasting between 8-12 minutes which requires one to start exercising at an easy workload and gradually progress to a very strenuous level. The following week you will perform a high intensity exercise protocol followed by one of three possible recovery regimens. The high intensity protocol is 3 cycling bouts of 45 seconds at 120% of VO2 max. with a 30 second rest break between each effort. The three possible recovery periods each last 32 minutes and consist of sitting passively in a chair, performing a standardized routine of static stretches, or cycling at 35% VO2 max. You will perform all three recovery regimens, each on a separate day. Each recovery regimen will be preceded by the high intensity exercise protocol. During the recovery period at minute 0, 3, 8, 16, 24, and 32, one of your fingers will be pricked with a small sterile lancet to obtain a blood sample (approximately 5 drops).

Risks:

There are risks involved in participating in this study. The maximal oxygen uptake test and high intensity exercise protocol are very physically demanding. You may experience dizziness, nausea, and muscle soreness. Arrhythmias and heart attacks while possible,

are extremely unlikely. You will be monitored closely and the tests will be terminated on your request or if the examiner notes significant signs of intolerance.

The taking of fingertip blood samples poses an infectious risk; there is the possibility of transmitting diseases. Sterile lancets, alcohol wipes, and the use of latex gloves will effectively eliminate this risk. You may experience some soreness in your fingers from being pricked.

During the stretching recovery there is a possibility of stretching too far and pulling a muscle. Since no bouncing stretches are being used, the chances of a pulled muscle are very low.

Benefits:

The risks from participating in this study are minimal. By participating you will learn your maximum oxygen uptake which is a common measurement of physical fitness.

Confidentiality:

All data will be kept on file at the UNLV exercise physiology laboratory and only research personnel will have access to these files. Your identity will not be revealed in any presentation of the results of this study.

Right To Refuse Or Withdraw:

You may refuse to participate in any part of this study and you may withdraw at any time without jeopardy to your standing in the Department of Kinesiology or UNLV.

Questions:

If you have any questions, please ask us. If you have additional questions later, Kevin Pitt will answer them at 732-3234. You will be given a copy of this form to keep.

I have read the description of this study and agree to participate. I understand that any questions I may have regarding my participation in this study will be answered and that I am free to withdraw from the study at any time without penalty.

_____	_____	_____
Subject's Signature	Subject's Name (Print)	Date

INFORMED CONSENT

Maximal Oxygen Uptake

Test: The test you are about to take is to determine maximal oxygen uptake. This test involves riding a cycle ergometer while heart rate is taken and expired breaths are analyzed. Each few minutes the workload is increased until maximum heart rate is attained. (This means that although the workload is increased, the heart rate does not increase.) This is about the point of maximum oxygen uptake.

This test requires you to work at your maximum ability and therefore is a demanding, vigorous, and a stressful test. Depending on your physical fitness, the test will last between 8-12 minutes. This test should only be taken by those who have been cleared by a physician who has indicated that there are no contraindications to the required stress. There are discomforts and possible dangers to the test. Muscle soreness, nausea, breathlessness, dizziness, and lightheadedness may occur. Maximum care, supervision, and preparation will be taken to minimize any hazard or danger. The test will be stopped any time the subject is not adapting well to the activity or when any major discomfort arises. The subject may stop the testing or withdraw from the test at any time. The test may be fatal for an individual with any history or symptoms of coronary artery disease.

In signing the consent form, I acknowledge that I understand the test procedure, the possible dangers, and certify that there is no medical reason why I should not participate in the test.

_____	_____	_____
Subject's Signature	Subject's Name (Print)	Date
_____	_____	_____
Witness' Signature	Witness' Name (Print)	Date

APPENDIX II
INDIVIDUAL DATA

Table 8 Individual Blood Lactate Values In Each Recovery Condition

SITTING

	TIME (Min.)						
Subject	Baseline	0	3	8	16	24	32
1	1.8	11.4	13	10.6	9	7	5.8
2	2	12.2	11.8	9.4	6	5.4	4.8
3	4	11	10	10.2	9.4	8	7.6
4	2	8.4	10.8	10	7	5	3.4
5	2	10.8	12.2	10.2	9.8	7.2	5.6
6	1.6	10.2	10.8	9.4	8.2	6.8	5
7	2.4	9.6	10.2	8.2	7.2	5.8	4
8	2.6	11.2	11.6	10.8	8.4	6	4.6
Mean	2.3	10.6	11.3	9.9	8.1	6.4	5.1
Std. Dev.	0.8	1.2	1.0	0.8	1.3	1.0	1.3

STRETCHING

Subject	Baseline	0	3	8	16	24	32
1	1.2	10.6	12.8	10.8	8	5.6	4.3
2	1.6	10.2	9.8	8.8	6	4.6	3.2
3	3	10	9.2	10	7.4	6	4.6
4	1.6	7.8	11	9.4	5.8	4.2	3.6
5	1.2	9.6	9.4	8	5.6	3.8	2.6
6	2.2	11	10.4	10.2	8	4.4	4
7	2.2	8.8	9.4	7.4	4.6	4	2.2
8	1.8	8.8	10.8	10.2	8.6	7.4	6.8
Mean	1.9	9.6	10.4	9.4	6.8	5.0	3.9
Std. Dev.	0.6	1.1	1.2	1.2	1.4	1.2	1.4

CYCLING

Subject	Baseline	0	3	8	16	24	32
1	1.8	9.6	12.2	11	7.4	4.6	3.4
2	3.6	10.6	10.6	8.6	5.4	5.6	4
3	2.8	8	9	7.2	5	4.6	3.6
4	1.8	9.6	9.8	9.4	6	3.8	3.4
5	1.6	9.6	11.6	9.7	7.4	5.8	5.4
6	3	12.4	7.8	6.2	3.8	3.2	3.4
7	2	8.2	8.4	7.2	5.6	3.8	2.8
8	3.2	9.8	12	11	11	5.8	5.8
Mean	2.5	9.7	10.2	8.8	6.5	4.7	4.0
Std. Dev.	0.8	1.4	1.7	1.8	2.2	1.0	1.1

Table 9 Individual Heart Rate Measures In Each Recovery Condition

SITTING

	TIME (Min.)						
Subject	Baseline	0	3	8	16	24	32
1	65	172	111	92	87	85	83
2	70	170	97	95	83	75	74
3	75	165	115	97	92	84	75
4	86	169	113	94	91	81	78
5	78	176	130	115	105	95	93
6	79	172	109	103	100	94	85
7	66	154	78	69	73	75	68
8	81	155	106	88	78	77	83
Mean	75	167	107	94	89	83	80
Std. Dev.	7	8	15	13	11	8	8

STRETCHING

Subject	Baseline	0	3	8	16	24	32
1	68	167	116	91	86	85	84
2	65	174	109	96	84	81	82
3	80	164	122	106	100	88	85
4	86	169	125	100	87	84	82
5	83	173	126	110	92	97	96
6	72	175	120	110	102	102	98
7	65	154	85	82	82	75	70
8	76	162	119	108	105	100	85
Mean	74	167	115	100	92	89	85
Std. Dev.	8	7	13	10	9	10	9

CYCLING

Subject	Baseline	0	3	8	16	24	32
1	65	168	132	126	122	120	120
2	70	178	128	115	106	106	110
3	78	165	117	111	101	101	100
4	85	170	143	132	129	127	125
5	81	172	152	123	125	120	118
6	83	165	128	121	116	118	116
7	62	156	103	97	94	90	87
8	75	160	120	108	108	104	104
Mean	75	167	128	117	113	111	110
Std. Dev.	8	7	15	11	12	12	12

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