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CHANGES IN BLOOD LACTATE CONCENTRATION DURING ACTIVE RECOVERY AT SUB-LACTATE THRESHOLD, LACTATE THRESHOLD, AND SUPRA-LACTATE THRESHOLD EXERCISE INTENSITIES

by

Sylvia J. Weihrer

A thesis presented
to the University of Ottawa
in fulfillment of the thesis
requirements for the degree of
Master of Science in Kinanthropology
in
Exercise Physiology

Sylvia J. Weihrer, Ottawa, Canada, 1991

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ABSTRACT

The purpose of this investigation was to identify the intensity of recovery exercise, relevant to endurance event performance, that would result in the most rapid times for half decrease in blood lactate concentration, following 2 minutes of cycling at maximal rates. Three recovery exercise intensities were investigated: (a) 7% of the maximal rate of oxygen consumption ($\dot{V}O_2max$) below lactate threshold oxygen consumption ($\dot{V}O_2$) (LT-7%), (b) lactate threshold $\dot{V}O_2$ (@LT), and (c) 7% of $\dot{V}O_2max$ above lactate threshold $\dot{V}O_2$ (LT+7%). Seven well-trained male cyclists (mean $\dot{V}O_2max$ 4.6 ± 0.49 $L \times min^{-1}$ participated as subjects in the investigation. Each subject completed seven separate cycling tests: (a) one combined lactate threshold-$\dot{V}O_2max$ test, (b) three constant work rate tests, and (c) three tests of recovery following supra-lactate threshold exercise (surge-recovery tests). The constant work rate tests served a dual purpose: to verify the individual lactate threshold, and to determine the baseline blood lactate concentration elicited by each recovery exercise intensity. The surge-recovery tests involved 2 minutes of cycling at $\dot{V}O_2max$ intensity immediately followed by 20 minutes of recovery at one of the three recovery intensities. The absolute values of blood lactate concentration were significantly different across the three recovery intensities ($p<.01$). At the end of the 20 minute recovery period the mean blood lactate concentrations were 1.57, 2.49, and 4.17 $mmol \times L^{-1}$ for the LT-7%, @LT%, and LT+7% recovery intensities, respectively. In order to examine the decrease in blood lactate concentration relative to the appropriate baseline of blood lactate concentration elicited by each recovery exercise intensity, times for half decrease in blood lactate concentration were calculated. There were no significant differences in these half decrease times between the three recovery intensities ($p<.01$). In other words, all three recovery intensities, even the one slightly above lactate threshold, were equally effective in decreasing blood lactate concentrations following the 2 minute surge.

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TABLE OF CONTENTS

ABSTRACT ii

ACKNOWLEDGEMENTS iii

INTRODUCTION AND RATIONALE 1
Statement of the Problem 6
Sub-Objectives 6
Experimental Hypothesis 7
Limitations 7
Abbreviations 7
Definitions 9

REVIEW OF LITERATURE 12
Lactate Threshold 12
Mode of Exercise 18
Exercise Protocols 18
Physiological Variables 20
Characterization of the Lactate Threshold Exercise Intensity 21
Detection Criteria 22
Lactate Metabolism 23
Lactate Production 23
Lactate 23
Pyruvate 25
Lactate Dehydrogenase 28
Factors Affecting the Rate of Lactate Production 30
Lactate Efflux from Muscle Cells 36
Lactate Entry into the Blood and Distribution Throughout the Body 37
Blood Lactate Elimination During Exercise 37
Explanations for the Blood Lactate Threshold Phenomenon 40
Muscular Fatigue 41
Definition of Muscular Fatigue 42
Review of Muscle Contraction 42
Possible Fatigue Sites and Mechanisms 45
Muscular Fatigue During Endurance Events 55
Blood Lactate Removal During Recovery Exercise 57

METHODOLOGY 67
Subjects 67
Subject Orientation 68
Procedure 68
Measurement of Oxygen Consumption 70
Measurement of Blood Lactate Concentration 71
Heart Rate Measurement 72
Bicycle and Cycle Ergometer 72

Combined LT-VO_2max Test 74
Criterion for VO_2max 76
Lactate Threshold Identification 76
Constant Work Rate Tests 77
Verification of the Lactate Threshold Exercise Intensity 79
Baseline Blood Lactate Concentrations 80
Surge-Recovery Tests 81
Analysis of Results and Statistical Design 83

RESULTS 86
Combined LT-VO_2max Test 86
Constant Work Rate Tests 87
Blood Lactate Concentration Analyses 93
Baseline Blood Lactate Concentrations 95
Oxygen Consumption Rate Analyses 96
Heart Rate Analyses 97
Surge-Recovery Tests 99
Pre-Recovery Periods 99
Blood Lactate Concentration Analyses 102
Oxygen Consumption Rate Analyses 103
Heart Rate Analyses 104
Recovery Periods 105
Blood Lactate Concentration Analyses 106
Oxygen Consumption Rate Analyses 109
Heart Rate Analyses 111
Times for Half Decrease of Blood Lactate 114

DISCUSSION AND CONCLUSIONS 118

BIBLIOGRAPHY 129

Appendix A: INFORMED CONSENT FORM

Appendix B: LT-VO_2max TEST RESULTS

Appendix C: POLYNOMIAL EQUATIONS FOR HALF-DECREASE TIMES

LIST OF FIGURES

1. Changes in blood lactate concentration with oxygen consumption 14

2. Blood lactate concentrations during 20 minutes of 15

3. Reaction sequence of the glycolytic pathway (Lehninger, 1982) 24

4. L-lactate (Lehninger, 1982) 25

5. Possible fates for pyruvate (Lehninger, 1982) 26

6. Reduction of pyruvate to lactate (Lehninger, 1982) 27

7. Hypothetical research design with recovery intensities defined 61

8. Blood lactate concentrations following maximal 63

9. Blood lactate concentration (mean ± standard error) 90

10. $\dot{V}O_2$ (mean ± standard error) versus time for the three 91

11. Heart rate (mean ± standard error) versus time for the three 92

12. $\dot{V}O_2$, blood lactate concentration, and heart rate 100

13. Blood lactate concentration (mean ± standard error) versus time 108

14. $\dot{V}O_2$ (mean ± standard error) 110

15. Heart rate (mean ± standard error) 113

16. Mean blood lactate concentration versus time during the recovery 116

LIST OF TABLES

1. General Subject Characteristics 67

2. Protocol for the LT-$\dot{V}O_2$max Test 74

3. Protocol for the Constant Work Rate Test 78

4. Protocol for the Surge-Recovery Test 82

5. Individual and Group Results from the LT-$\dot{V}O_2$max Test 87

6. Individual and Group Results from the Three Constant 89

7. Results from Three-Way ANOVA on Blood Lactate
Concentration 93

8. Results from Analysis of Simple Main Effects on Blood Lactate 94

9. Results from Three-Way ANOVA on VO_2 Measurements from 97

10. Results from Three-Way ANOVA on Heart Rate Measurements
from 98

11. Descriptive Statistics for Blood Lactate Concentration, ... 101

12. Results from Three-Way ANOVA on Blood Lactate
Concentration 102

13. Results from Three-Way ANOVA on VO_2 Measurements 104

14. Results from Three-Way ANOVA on Heart Rate Measurements 105

15. Mean Blood Lactate, VO_2, and Heart Rate Measurements 107

16. Results from Three-Way ANOVA on Blood Lactate
Concentration 109

17. Results from Three-Way ANOVA on VO_2 Measurements 111

18. Results from Three-Way ANOVA on Heart Rate Measurements
from 112

19. Mean Results from Analyses of Blood Lactate Concentration 115

20. Results from Two-Way ANOVA on Half-Times 117

INTRODUCTION AND RATIONALE

In endurance events, such as a running marathon or a cycling road race, submaximal exercise intensities must be maintained for prolonged durations. The length of time that an activity can be maintained is a function of the intensity at which the exercise is performed. That is, as exercise intensity increases, the length of time it can be maintained decreases (Astrand & Rodahl, 1986). Obviously, performance intensity is a critical issue for endurance athletes. These athletes must work at high intensities in order to be competitive, but they must also work for extended periods of time. Thus, during an endurance event, athletes must avoid intensities that are so high that they may result in premature fatigue.

The highest exercise intensity that can be maintained for prolonged durations is the one that produces the highest rate of oxygen consumption possible in conditions of steady-state blood lactate (Farrell, Wilmore, Coyle, Billing, & Costill, 1979; Tanaka & Matsuura, 1984). This intensity of activity is often referred to as the lactate threshold intensity. At this intensity the concentration of blood lactate is relatively stable and it represents a dynamic equilibrium between the rate of lactate entry into the blood and the rate of its elimination from the blood. Work at intensities even slightly above the lactate threshold intensity will create an imbalance between the rates of blood lactate appearance and of blood lactate elimination, such that there will be a progressive accumulation of lactate in the blood (Gladden, 1989). Endurance athletes working at their individual lactate threshold intensity would be able to

utilize the highest proportion of their maximal oxygen consumption (VO_2max) without accumulating lactate in the blood (Farrell et al., 1979; Stegmann, Kindermann, & Schnabel, 1981), and thus without suffering the consequences generally associated with such an accumulation.

The intensity of exercise intuitively chosen by endurance athletes during competition appears to be closely linked to the lactate threshold exercise intensity. The parameter that is most highly correlated (r=0.88-0.99) to long distance running performance (3.2 km - 42.2 km) is the running velocity at lactate threshold (Conconi, Ferrari, Ziglio, Droghetti, & Codeca, 1982; Farrell et al., 1979; Powers, Dodd & Garner, 1984; Sjodin & Schele, 1982; Williams & Nute, 1983). Very high correlations (r=0.94-0.99) have also been found between the running velocity at lactate threshold and the average marathon running velocity (Conconi et al., 1982; Farrell et al., 1979; Rhodes & McKenzie, 1984; Sjodin & Jacobs, 1981; Sjodin & Svedenhag, 1985). Other studies have investigated changes in blood lactate concentration in athletes before and after running a marathon. Costill (1970) reported low blood lactate concentrations following a marathon. Only athletes that sprinted to the finish demonstrated significant blood lactate accumulation (Costill, 1970; Maron, Horvath, & Wilkerson, 1975). Based on the results of these investigations, it appears that the intensity of activity chosen for at least a major portion of endurance event performance is either at or below the individual lactate threshold intensity.

While the majority of the work done in an endurance event will likely be at or below the lactate threshold intensity, there will, however, be circumstances that may force an athlete to work at suprathreshold intensities for varying durations. In their analysis of oxygen consumption through the course of a

competitive marathon, Maron, Horvath, Wilkerson, and Gilner (1976) found that oxygen consumption ranged between 68% and 100% of VO_2max depending on whether the athletes were running up or down a grade, or running on the level. Exercise intensities will rise above lactate threshold levels when athletes are involved in an uphill climb (Maron et al., 1976) or a sprint (Costill, 1970). Thus, factors such as terrain, competitive strategy and/or environmental conditions may push an endurance athlete into suprathreshold intensities of work several times within a competitive endurance event.

Work at suprathreshold intensities results in an imbalance between the rate of blood lactate appearance and the rate of blood lactate elimination. At suprathreshold exercise intensities, lactate production may be greatly increased, the efficiency of blood lactate removal may be reduced, or both may occur to some degree. In any case, the imbalance between lactate production and blood lactate removal will result in a progressive accumulation of lactate in the muscle and in the blood.

Elevated muscle and blood lactate concentrations are commonly associated with muscular fatigue. While the mechanisms of fatigue are still unclear, it is believed that the potential of a muscle to perform work is reduced by high muscle and blood lactate concentrations as well as high hydrogen ion concentrations (Karlsson, Bonde-Petersen, Henriksson, & Knuttgen, 1975; Schnabel, Kindermann, Schmitt, Biro, & Stegmann, 1982). Numerous studies have investigated the effect of various exercise intensities on the time to exhaustion. These studies have revealed that work at intensities even slightly above the lactate threshold intensity greatly reduces the time to exhaustion, compared to work at or below the lactate threshold intensity (Ribeiro, Hughes,

Fielding, Holden, Evans, & Knuttgen, 1986; Simon, Young, Gutin, Blood, & Case, 1983; Stegmann & Kindermann, 1982). Other studies have shown that the time to exhaustion decreases as a result of elevated pre-exercise lactate concentrations (Hogan & Welch, 1984; Karlsson et al., 1975). Thus, during prolonged exercise there appears to be a strong association between lactate accumulation and muscular fatigue.

Based on this fatigue association, athletes engaged in endurance competitions should avoid circumstances, such as work at suprathreshold intensities, that will result in an accumulation of muscle and blood lactate. Such circumstances, however, are often unavoidable. Therefore, if an endurance athlete does work at suprathreshold intensities, measures must be taken following the suprathreshold surge to ensure elimination of the accumulated blood lactate. The rapid elimination of blood lactate would reduce the risk of premature fatigue (Jacobs, 1986), and would enable the athlete to perform subsequent suprathreshold surges, should the demands of the event require them.

Blood lactate that has accumulated during a suprathreshold surge can be eliminated by decreasing the intensity of work. It has been well established that blood lactate elimination occurs during exercise (Belcastro & Bonen, 1975; Bonen & Belcastro, 1976; Davies, Knibbs, & Musgrove, 1970; Hermansen & Stensvold, 1972; McGrail, Bonen, & Belcastro, 1978; McLellan & Skinner, 1982; Stamford, Weltman, Moffatt, & Sady, 1981; Weltman, Stamford, & Fulco, 1979). In fact, there appears to be a critical intensity of exercise which results in an optimal rate of blood lactate removal. Studies have demonstrated that blood lactate removal rates increase as exercise intensities rise toward a critical level, and

then decrease as exercise intensities exceed this level (Belcastro & Bonen, 1975; Davies et al., 1970; McLellan & Skinner, 1982). However, the critical intensity of exercise that results in an optimal rate of decrease in blood lactate concentration has not yet been established.

Numerous investigators have searched for the exercise intensity that potentiates blood lactate removal. There have been fundamental differences in the methodological and conceptual approaches used in these studies. Discrepancies exist in the manner in which the recovery exercise intensities are defined. Exercise intensities have typically been defined relative to VO_2max. The exercise intensities should be defined relative to the lactate threshold, in order to achieve some degree of standardization of blood lactate kinetics between individuals. Fundamental problems also exist because of the different definitions used for lactate threshold. Discrepancies are also apparent in the methods employed to calculate blood lactate removal rates. As well, the question of exercise intensity for optimal blood lactate removal is usually asked within the context of an intermittent sport, where periods of maximal activity are usually followed by periods of inactivity. A contrasting context is an endurance event, where the suprathreshold surges occur intermittently throughout the course of the event. In the latter context, the proposed post-surge exercise intensity must be one that will elicit optimal blood lactate removal, and at the same time it must be one that is competitively feasible. As a result of the inconsistencies outlined, the exercise intensity that will potentiate the rate of decrease in blood lactate concentration, during an endurance event, is still unknown.

Statement of the Problem

The aim of this study was to identify the intensity of recovery exercise, relevant to endurance event performance, that would result in the most rapid times for half decrease in blood lactate concentration, following 2 minutes of cycling at $\dot{V}O_2max$ intensity. The recovery intensities that were investigated were defined relative to the individual lactate threshold exercise intensity. The blood lactate decrease half-times were calculated using individually determined baselines of blood lactate concentration. Since the context of the problem was a competitive endurance event, the recovery intensities investigated were those considered to be most applicable to endurance event performance; that is, intensities slightly below, at, and slightly above the individual lactate threshold exercise intensity.

Sub-Objectives

1. To determine the $\dot{V}O_2max$ and the individual lactate threshold exercise intensity of a group of endurance trained cyclists, using a continuous progressive incremental exercise test on a bicycle ergometer.
2. To verify the lactate threshold exercise intensities using continuous constant work rate tests at intensities of 7%$\dot{V}O_2max$ below, at, and 7%$\dot{V}O_2max$ above the individual lactate threshold $\dot{V}O_2$.
3. To determine individual baseline blood lactate concentrations for the 7%$\dot{V}O_2max$ below lactate threshold and the lactate threshold exercise intensities.
4. To assess the absolute and relative changes in blood lactate concentration during recovery exercise at intensities that are 7%$\dot{V}O_2max$ below, at, and 7%$\dot{V}O_2max$ above the individual lactate threshold $\dot{V}O_2$, following a 2 minute activity at $\dot{V}O_2max$ intensity.

Experimental Hypothesis

It was hypothesized that during recovery from 2 minutes of cycling at VO_2max intensity the times for half decrease in blood lactate concentration would be most rapid at the individual lactate threshold exercise intensity.

Limitations

The conclusions made from the results of this investigation can only be translated to highly trained endurance cyclists of ages similar to those of the cyclists that were actually tested. In addition, since the testing was conducted at the beginning of the competitive season, the conclusions can only be applied to cyclists who are in the same phase of their training.

Only one surge intensity and duration was used. It should be noted that many other types of surges may be encountered by a cyclist during an event. Only three intensities of recovery exercise were investigated. The duration of the recovery periods was constant. Each cyclist used an individually chosen cadence for the testing. This cadence was used in all of the tests performed by the cyclist. All of these experimental controls place limitations on the application of the results to competitive performance strategies.

Abbreviations

ADP - Adenosine diphosphate

AMP - Adenosine monophosphate

ANOVA - Analysis of variance

ATP - Adenosine triphosphate

BLC - Blood lactate concentration

bpm - Beats per minute

Ca^{2+} - Calcium Ion

CNS - Central nervous system

CO_2 - Carbon dioxide

df - Degrees of freedom

F - F-statistic

$\Delta G^{\circ\prime}$ - Standard free energy change

H_2O - Water

HR - Heart rate

IMP - Inosine monophosphate

K^+ - Potassium ion

K' - Dissociation constant

Km - Michaelis-Menten constant

kp - Kiloponds

LDH - Lactate dehydrogenase

LT - Lactate threshold

LT-7% - (($\dot{V}O_2$ at lactate threshold) - (7% of $\dot{V}O_2max$))

@LT - (($\dot{V}O_2$ at lactate threshold))

LT+7% - (($\dot{V}O_2$ at lactate threshold) + (7% of $\dot{V}O_2max$))

MS - Mean square

Na^+ - Sodium ion

NAD^+ - Nicatinamide adenine dineucleotide (oxidized form)

NH_3 - Ammonia

NH_4^+ - Ammonium ion

p - Probability

PFK - Phosphofructokinase

Pi - Inorganic phosphate

pK' - Negative logarithm of K'

rpm - Revolutions per minute

SS - Sum of squares

TCA - Tricarboxylic acid

Vmax - Turnover number

VO_2 - Rate of oxygen consumption

VO_2max - Maximal rate of oxygen consumption

Definitions

1. β-Oxidation: The sequential process of fatty acid catabolism to acetyl-CoA (Zubay, 1983).
2. Blood Lactate Decrease Half-Time: The time required to decrease blood lactate concentration (BLC) to a value equal to:
   (peak BLC - baseline BLC) / 2.
3. Coenzyme: A molecule possessing the physiochemical properties not found in the polypeptide chain of an enzyme that acts together with the enzyme to catalyze a biochemical reaction (Lehninger, 1982).
4. Gluconeogenesis: The formation of D-glucose from noncarbohydrate sources, such as lactate, pyruvate, and glycerol (Lehninger, 1982).
5. Glycogenolysis: The reaction pathway for glycogen degradation. This pathway involves the same reaction sequence as glycolysis once a glucose molecule has been removed from glycogen.
6. Glycolysis: The ten step sequence for glucose degradation. When glucose is degraded to acetyl-CoA, the process is called aerobic glycolysis; and it is called anaerobic glycolysis when glucose is degraded to lactate.

7. Dissociation Constant (K'): This constant describes the tendency of a molecule to lose a proton. A high K' indicates a strong acid (Lehninger, 1982).

8. Lactate Threshold: The lactate threshold is an intensity of exercise. It is best defined by explaining the manner in which it is determined. During activity of progressively increasing intensity, the lactate threshold exercise intensity is that intensity of exercise above which blood lactate concentrations abruptly increase and continue to increase. In continuous, constant rate work, the lactate threshold exercise intensity is the highest intensity that can be performed with steady-state blood lactate concentrations.

9. Michaelis-Menten Constant (Km): A parameter that is characteristic of the enzyme-substrate conditions (Zubay, 1983). It is the concentration of the specific substrate at which a given enzyme yields one-half maximal velocity (Lehninger, 1982).

10. Nicatinamide adenine dinucleotide (NAD^+): A biological carrier of reducing equivalents (that is, electrons) (Lehninger, 1982).

11. pH: A scale used to describe the concentration of free hydrogen ions in a solution, where pH is equal to the negative log of the hydrogen ion concentration (Sienko & Plane, 1979). The physiologic range of blood pH is between 6.8 and 7.8; normal blood pH is 7.4 (Jensen, 1980).

12. Respiratory Chain: A series of events in which coenzymes (NADH and $FADH_2$) are oxidized through the transfer of electrons, down a chain of electron carriers, to oxygen (Zubay, 1983).

13. Standard Free Energy Change ($\Delta G^{\circ\prime}$): The standard free energy change of a reaction (Lehninger, 1982).

14. Turnover Number (Vmax): The number of substrate molecules converted into product per second per mole of enzyme present when the enzyme is fully complexed with the substrate (Lehninger, 1982).

REVIEW OF LITERATURE

The objective of this research was to identify the intensity of recovery exercise, relevant to endurance event performance, that would result in the most rapid times for half decrease in blood lactate concentration, following a standard duration of activity at VO_2max intensity. In order to achieve this objective a number of particulars were investigated. First, because the intensity of recovery exercise was most appropriately defined relative to the lactate threshold, a suitable working definition of this threshold was identified. Second, because the intensity of recovery exercise was evaluated in terms of its influence on the rate of decrease in blood lactate concentration, the processes and mechanisms of lactate metabolism were investigated. Third, since the impetus for research into blood lactate removal was based on the association between lactate accumulation and fatigue, the factors responsible for muscular fatigue were investigated. Finally, the influence of active recovery on changes in blood lactate concentration, and the efforts of previous investigators to identify an optimal recovery exercise intensity were also investigated. Research into these four areas enabled the development of the most effective experimental design for examining the relationship between recovery exercise intensity and blood lactate decrease half-times.

Lactate Threshold

Lactate threshold is a term that is used to describe a particular intensity of exercise. It is a phenomenon that is based on the characteristic changes in

blood lactate concentration apparent with increasing intensities of exercise. This characteristic response is evident during exercise that involves progressively increasing intensities, as well as during exercise that involves continuous work at constant intensities. In exercise that is progressively incremental, blood lactate concentrations generally rise slightly with each increase in exercise intensity and VO_2 (Brooks, 1985). However, at a specific intensity of exercise, blood lactate concentrations abruptly increase and continue to increase at a relatively high rate until exhaustion (see Figure 1) (Brooks, 1985). In this type of exercise the lactate threshold exercise intensity is that intensity above which blood lactate concentrations abruptly increase.

A lactate threshold is also evident during continuous constant rate work. When increasing intensities of constant rate work are examined, blood lactate concentrations at low work rates will reach a steady-state level. This steady-state concentration will increase slightly with increasing constant work rates (Ribeiro et al., 1986; Scheen, Juchmes, & Cession-Fossion, 1981). However, an exercise intensity will eventually be reached that will result in a progressive accumulation of lactate in the blood (see Figure 2). In this type of exercise, the lactate threshold intensity is the highest intensity that can be performed with steady-state blood lactate concentrations.

The criteria, and the terms used to describe this specific intensity of exercise are not universal. Lactate threshold is only one of a plethora of terms used to describe an exercise intensity that corresponds to specific changes in blood lactate concentrations. Some of the other terms include: aerobic threshold, anaerobic threshold, aerobic-anaerobic threshold, onset of blood lactate accumulation, onset of plasma lactate accumulation, lactate turning

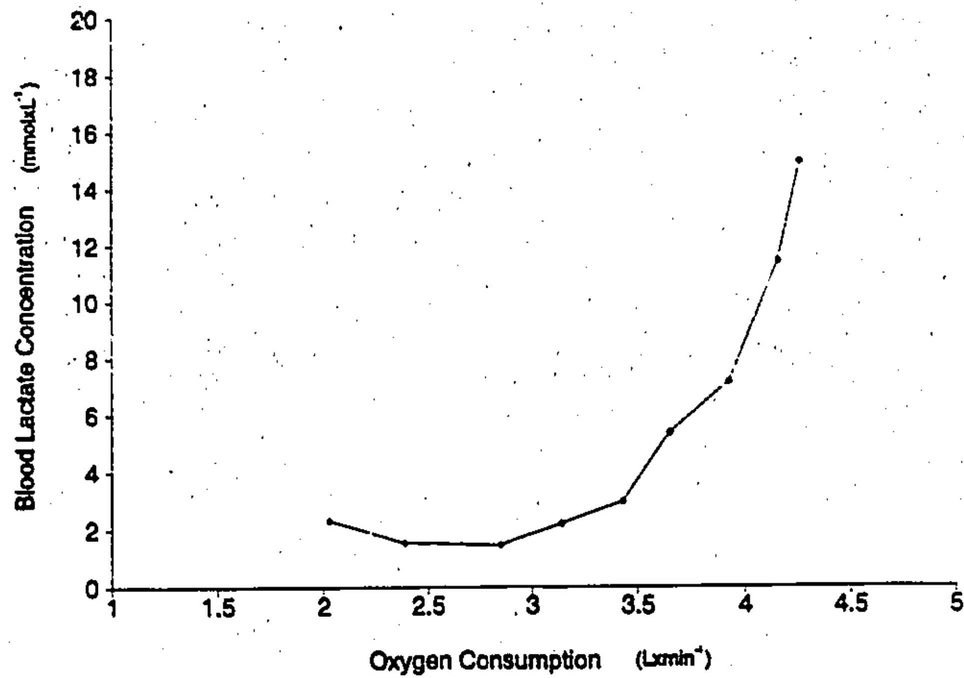


Figure 1. Changes in blood lactate concentration with oxygen consumption during a progressively incremental cycling test (unpublished data gathered in 1988 at the University of Ottawa (Kinanthropology Department): Ottawa, Canada.

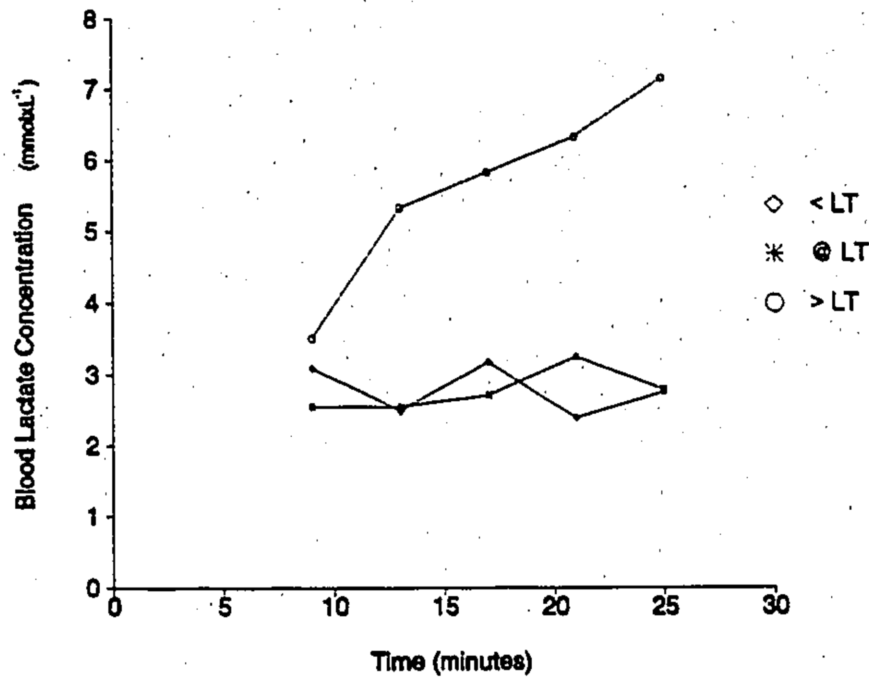


Figure 2. Blood lactate concentrations during 20 minutes of cycling at three different exercise intensities - below lactate threshold, at lactate threshold, and above lactate threshold (unpublished data gathered in 1988 at the University of Ottawa (Kinanthropology Department): Ottawa, Canada.

point, maximal steady-state, individual lactate threshold, excess lactate, and aerobic capacity (Jacobs, 1986). The criteria used to define this exercise intensity vary with the terms used to describe it. Some of these terms refer to the exercise intensity above which blood lactate accumulation occurs, others refer to the exercise intensity which causes blood lactate concentrations to rise above resting levels, and still others refer to the exercise intensity that elicits a specific concentration of blood lactate.

A great deal of the disparity surrounding this issue results from the lack of a concrete model to explain the physiological mechanisms underlying the blood lactate response. Until recently this point was most frequently referred to as the anaerobic threshold. The marked rise in blood lactate concentration, apparent when exercise intensities exceed lactate threshold, was once believed to indicate the onset of anaerobic metabolism (that is, the onset of lactate production) within the active muscle tissue (Wasserman & McIlroy, 1964). Local tissue hypoxia was the explanation provided as the cause of this anaerobic metabolism. More recent research suggests that hypoxic conditions do not exist in the muscle during submaximal exercise (Brooks, 1985). More importantly, it has become evident that some degree of anaerobic metabolism, or lactate production, occurs at all exercise intensities - even at rest. Therefore, the use of the term anaerobic threshold is misleading, since there is no exercise intensity which signifies the beginning of anaerobic metabolism and/or lactate production.

The term lactate threshold is not meant to imply a threshold for lactate production; rather, it refers to the threshold for the onset of lactate accumulation in the blood. The concentration of lactate in the blood at any

instant represents the net result of a number of processes: lactate production, lactate efflux from production sites, lactate distribution, and blood lactate elimination. At intensities of exercise equal to or below the lactate threshold, the rate of appearance of blood lactate and the rate of disappearance of blood lactate will be equal, and as a result, there will be no net accumulation of lactate in the blood. At intensities of exercise greater than the lactate threshold there will be an imbalance between the rates of lactate appearance and disappearance, and as a result, lactate will progressively accumulate in the blood. Therefore, the term lactate threshold refers to the threshold for the balance between the rate of appearance of lactate in the blood and the rate of disappearance of blood lactate. This term is applicable to research that investigates the response of the whole body to a particular intensity of exercise. Therefore, for the purposes of this paper, the term lactate threshold will be employed.

The lack of consistency in the terminology and criteria used for the lactate threshold exercise intensity also extends to the methodologies used in the identification of this intensity. In deciding on a methodology for the identification of the lactate threshold a number of factors must be considered. The key considerations include: (a) the mode of exercise that will be used, (b) the type of exercise protocol that will be used, (c) the physiological variable(s) that will be monitored, (d) the method that will be used to characterize the threshold, and (e) the criteria that will be used to identify the threshold. Each of these considerations will be addressed in the following sections.

Mode of Exercise

The first consideration is of the mode of exercise that will be employed. This is a critical consideration because the lactate threshold exercise intensity is specific to each mode of exercise. For example, the lactate threshold will occur at a different VO_2 for different activities performed by the same individual (Davis, Vodak, Wilmore, Vodak, & Kurtz, 1976; Withers, Sherman, Miller, & Costill, 1981). Therefore, if the lactate threshold of an athlete is evaluated, and the resulting information is to be applied to the athlete's training regime, the exercise performed during the evaluation should be as sport specific as possible. In addition, if the lactate threshold exercise intensity is determined for a group of individuals, and it is being compared between the individuals, then the mode of exercise during evaluation must be consistent for all individuals.

Exercise Protocols

There are a number of different exercise protocols that have been used to identify the lactate threshold exercise intensity. In many studies the lactate threshold is determined using the same protocol that is used to measure VO_2max. This is a popular practice because it enables the measurement of both parameters with a single test. The most common protocols involve either progressive incremental exercise (Aunola & Rusko, 1984; Neary, MacDougall, Bachus, & Wenger, 1985; Powers et al., 1984; Stegmann et al., 1981; Steinrauf, Schulz, Ryder, Brunn, & Cook, 1987; Yoshida, 1984), or continuously increasing exercise intensities (ramp tests) (Davis & Gass, 1981; Hughson & Green, 1982; Yeh, Gardner, Adams, Yantowitz, & Crapo, 1983). Stegmann et al. (1981) have suggested that ramp tests may be preferable in studies that closely assess the

relationship between workload and physiologic response. However, Yeh et al. (1983) were unable to identify the lactate threshold exercise intensity using a ramp test. In addition, Hughson & Green (1982) revealed that fast ramp tests (49.0 to 65.4 watts$\times$min^{-1}) resulted in overestimated lactate threshold exercise intensities. The problem with the ramp tests may be that they do not allow enough time for the mechanisms of blood lactate disappearance to adjust to the changes in lactate production and blood lactate appearance that result with each change in work rate. The adjustment of blood lactate disappearance at each work rate is essential to the identification of the lactate threshold exercise intensity. Without this adjustment the exercise intensity that results in an imbalance between the appearance and disappearance of blood lactate will be impossible to determine. Therefore, the progressive incremental protocol may be optimal for lactate threshold determination because it effectively displays the lactate threshold response.

Some progressive incremental protocols are more effective in identifying the lactate threshold exercise intensity than others. These protocols can vary in stage duration and in the size of the work increment imposed at each stage. Stage durations of 1 to 4 minutes have been used, but the most common duration is 3 minutes (Neary et al., 1985; Powers et al., 1984; Stegmann et al., 1981; Steinrauf et al., 1987). The 3 minute stage duration appears optimal for lactate threshold determination because it allows enough time for oxygen consumption to attain a steady-state (Yoshida, 1984). It also allows enough time for the lactate that is produced in the working muscles to diffuse to the extracellular space and to be subsequently dealt with by the available elimination mechanisms (Stamford, Weltman, & Fulco, 1978).

Progressive incremental protocols have varied more in the size of the work increment used between stages, than in the stage duration used. The work increment will obviously vary with the specific ergometer that is employed. In cycling tests, work increments have varied between 10 watts and 65 watts. In treadmill tests, work increments have usually involved augmenting the treadmill speed by 1 or 2 km per hour at each stage. Smaller work increments are generally utilized when a very sensitive determination of the lactate threshold exercise intensity is desired. Very small increments, however, will substantially increase the length of the test and this may create other problems, such as premature fatigue.

Physiological Variables

The lactate threshold exercise intensity has been estimated from direct and indirect measures. Ventilatory and/or gas exchange values are the indirect measures that have been used. Lactate threshold detection from these indirect measures is based on the assumption that blood lactate accumulation results in a breakaway ventilation response. This assumption has been greatly disputed (Brooks, 1985; Hughson & Green, 1982; Neary et al., 1985; Powers et al., 1984). Therefore, while the indirect detection method is appealing because it is non-invasive, it may not be valid.

The direct method of lactate threshold detection involves the use of the actual blood lactate concentration determined from blood samples taken at each exercise intensity. Obviously this method is the most valid since it actually measures blood lactate concentration. However, the direct method of lactate threshold determination is not free from controversy. A number of different

methods have been used for the actual sampling of blood lactate during the test. Blood samples have been obtained from arterial, mixed venous, venous, and capillary blood. The first three of these methods involve the use of an indwelling catheter. Yoshida (1984) has demonstrated that similar lactate threshold exercise intensity identification is possible using arterial or venous blood, provided the exercise duration is long enough. Capillary blood sampling is by far the simplest of these methods. As well as being less invasive than an indwelling catheter, the cost, equipment complexity, and professional involvement required for implementation of this method is considerably less. In addition, capillary blood sampling allows for reliable lactate threshold identification provided the stage durations are sufficiently long (Jacobs, 1986).

Characterization of the Lactate Threshold Exercise Intensity

Another factor that must be considered when determining the lactate threshold exercise intensity is the manner in which this exercise intensity is expressed. At times it is expressed as a rate of work; for example, a power output on a cycle ergometer or a treadmill running velocity. This method of expression may be misleading since the specific workload at lactate threshold has been shown to vary with the test protocol used (Yoshida, 1984). It has also been shown to vary with such factors as the level of glycogen depletion. For these reasons, lactate threshold is most commonly expressed as an oxygen consumption in $L \times min^{-1}$, or as a percentage of $\dot{V}O_2max$. The $\dot{V}O_2$ at lactate threshold has been shown to be reliable between test sessions (Aunola & Rusko, 1986), and between different types of progressive incremental test protocols (Yoshida, 1984).

Detection Criteria

The criteria used to identify the lactate threshold exercise intensity from direct measures, are quite similar among researchers. Some researchers identify the lactate threshold as the $\dot{V}O_2$ at the intensity of exercise just below the intensity that causes blood lactate concentrations to increase distinctly above resting levels (Aunola & Rusko, 1984; Aunola & Rusko, 1986; Yoshida, 1984). Others identify the lactate threshold as the $\dot{V}O_2$ at the intensity of exercise just before the non-linear increase in blood lactate concentration (Neary et al., 1985; Steinrauf et al., 1987). While the wording of these two criteria is different, in practice they usually result in the detection of a very similar lactate threshold exercise intensity.

In summary, for the purposes of this investigation, the lactate threshold will be defined as: (a) that intensity of exercise, during progressive incremental exercise, above which blood lactate concentrations abruptly increase and continue to increase; and (b) the highest intensity of exercise, during constant rate work, that can be performed with steady-state blood lactate concentrations. A number of test methods have been used to determine lactate threshold. Of these methods, accurate and reliable detection appears to be possible with a progressive incremental protocol. Stage durations of 3 minutes with moderate work increments should be incorporated into this type of protocol. Detection of the lactate threshold from capillary blood sampling appears to be a simple and reliable technique.

Lactate Metabolism

The lactate threshold exercise intensity is determined from the characteristic changes in blood lactate concentration that occur in response to varying intensities of exercise. The blood lactate concentration at any instant is the net result of a number of different processes: lactate production, lactate efflux from production sites, lactate entry into the blood, blood lactate distribution throughout the body via the circulatory system, and blood lactate uptake by various tissues. Each of these processess will be investigated in order to gain insight into the mechanisms underlying the lactate threshold phenomenon.

Lactate Production

This section will deal with the different aspects of lactate production; namely, lactate, pyruvate, lactate dehydrogenase, and the factors which affect the rate of lactate production.

Lactate.

Lactate is a product of carbohydrate catabolism. The specific metabolic pathways that produce lactate are glycogenolysis and glycolysis. Glycogenolysis is the breakdown of glycogen, and glycolysis is the breakdown of glucose. These two metabolic pathways differ only in their starting materials (see Figure 3); therefore, they will collectively be referred to as the glycolytic pathway, from this point on.

Lactate is a three carbon biomolecule with a carboxyl group and a hydroxyl group (see Figure 4). Lactate is a fairly strong acid - with a pK' of 3.86 at 25 °C

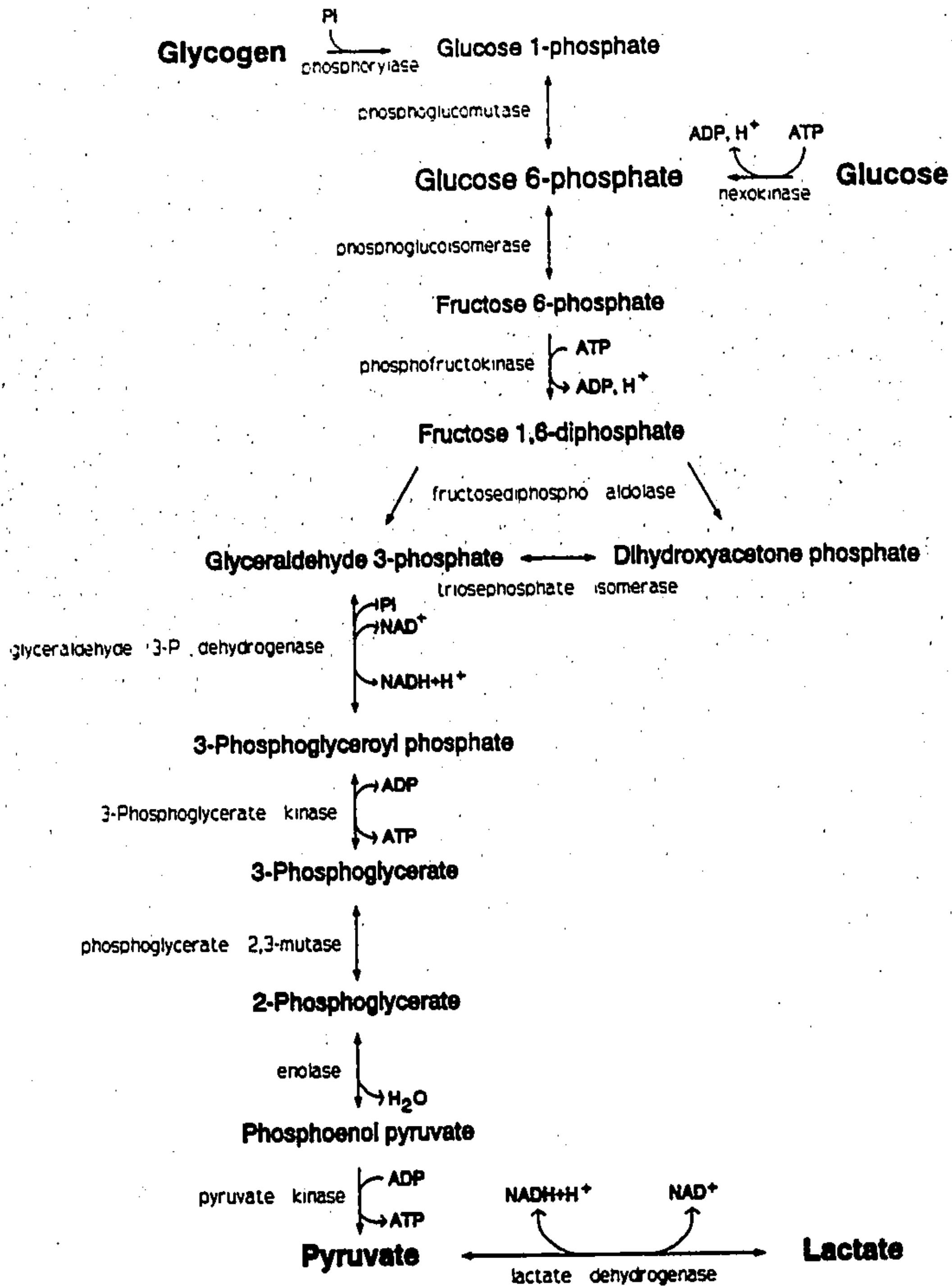


Figure 3. Reaction sequence of the glycolytic pathway (Lehninger, 1982).

(Lehninger, 1982). Thus, at physiological pH the molecule will be almost completely dissociated into hydrogen and lactate ions: H^+ and $CH_3CHOHCOO^-$. Therefore, the term lactate is used synonymously with lactic acid in physiological discussions.

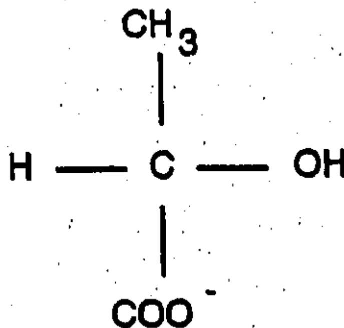


Figure 4. L-lactate (Lehninger, 1982).

Pyruvate.

The immediate precursor of lactate is pyruvate (see Figure 3 on page 24). Pyruvate is a key molecule because it is common to several metabolic pathways. Lactate is only one of a number of possible fates for pyruvate (see Figure 5); these fates are outlined below.

AMINO ACID FORMATION. The carbon skeleton of pyruvate can be used in the formation of certain amino acids. Pyruvate gives rise to L-alanine and L-valine; in part it can also be used to produce isoleucine and lysine (Zubay, 1983).

ACETYL-COA FORMATION. Pyruvate formed from the glycolytic catabolism of carbohydrates provides a large amount of acetyl-CoA for the tricarboxylic acid (TCA) cycle. The sequence of events which leads to the oxidative decarboxylation of pyruvate to form acetyl-CoA is catalyzed by a

structured cluster of enzymes and coenzymes known as the pyruvate dehydrogenase complex. This reaction takes place within the mitochondria of eukaryotic cells and is highly exergonic ($\Delta G^{\circ\prime}$=-8.0 kcal×mol^{-1}) (Zubay, 1983). The activity of this complex is inhibited by high concentrations of ATP, NADH (the reduced form of NAD^+), and citrate (Lehninger, 1982).

OXALOACETATE FORMATION. Pyruvate is also involved in a reaction that has an important anapleurotic purpose for the TCA cycle. In this reaction, pyruvate is used to produce oxaloacetate; the enzyme involved is pyruvate carboxylase, and the reaction requires ATP, CO_2, and H_2O. The formation of oxaloacetate from pyruvate is also an important step in one of the central pathways of gluconeogenesis, which is the formation of glucose from non-carbohydrate precursors (Lehninger, 1982).

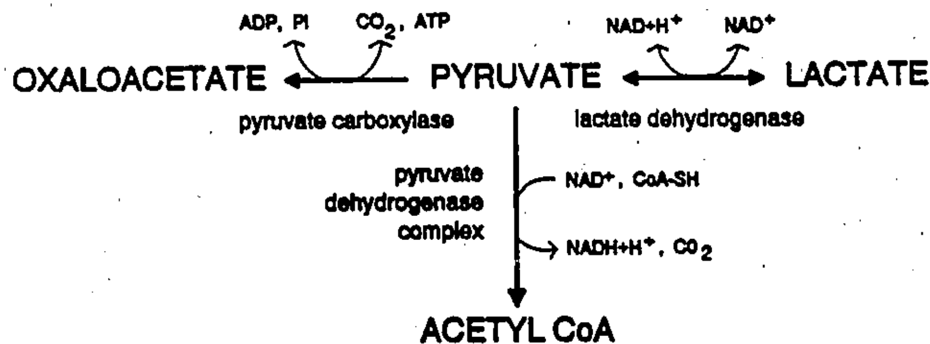


Figure 5. Possible fates for pyruvate (Lehninger, 1982).

LACTATE FORMATION. The pyruvate formed from carbohydrate catabolism can also be reduced to lactate. This reaction takes place in the cytoplasm and is catalyzed by the enzyme lactate dehydrogenase (LDH) (see Figure 6).

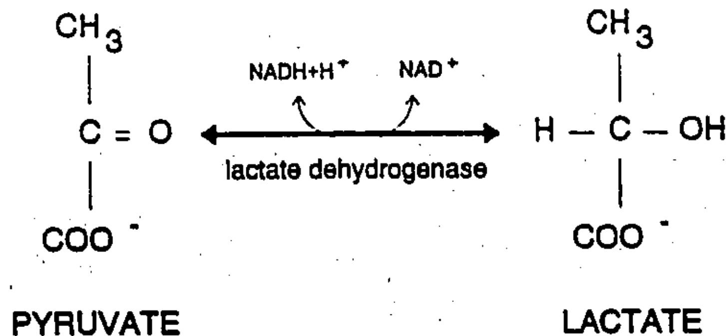


Figure 6. Reduction of pyruvate to lactate (Lehninger, 1982)

Ultimately the fate of pyruvate will depend on the type of cell that it is produced in, and on the metabolic state of the cell (Zubay, 1983). At times when the energy requirement of a cell is at normal resting levels the pyruvate that is produced can follow anabolic fates such as, amino acid formation for eventual protein synthesis, oxaloacetate formation for anapleurotic purposes or for glycogen synthesis, or acetyl-CoA formation for eventual fatty acid synthesis. At times when the activity of the cell is elevated (for example, in an active muscle cell) the pyruvate that is produced can be converted to acetyl-CoA which is further catabolized via the TCA cycle and the respiratory chain to CO_2 and H_2O; it can also be reduced to lactate.

The reduction of pyruvate to lactate is the dead end of glycolysis. Lactate cannot be catabolized further. Once it has been produced it can either leave the cell, accumulate within the cell, or be oxidized back to pyruvate - since the LDH step is reversible. While the reduction of pyruvate to lactate is a dead end, it serves an important function. This function has to do with the coenzyme NAD^+

which is required at the glyceraldehyde 3-phosphate dehydrogenase step of glycolysis (see Figure 3 on page 24). This coenzyme is supplied in aerobic conditions through the oxidation of NADH with molecular oxygen in the mitochondria (Lehninger, 1982). However, at times when the glycolytic activity is high, or when a cell must function anaerobically, these reducing equivalents are also obtained from the reduction of pyruvate to lactate. Pyruvate is used as an electron acceptor molecule for the hydride ion that must be removed from NADH in order to generate the necessary NAD^+ to maintain glycolytic activity (Zubay, 1983). Therefore, lactate production is a critical step in glycolysis, as it maintains the activity of the pathway in certain metabolic conditions.

The reduction of pyruvate to lactate results in the regeneration of NAD^+. Because the supply of NAD^+ may be limiting when mitochondrial function cannot keep pace with energy demands, this reaction has been especially implicated in anaerobic tissues or in anaerobic states. However, the formation of lactate from pyruvate is highly exergonic ($\Delta G°'$=-6.0 kcal$\times$mol^{-1}) (Zubay, 1983). In addition, the catalytic activity of LDH is greater than that of any other glycolytic enzyme and greater than that of enzymes providing alternate routes for pyruvate metabolism (Brooks, 1985). Therefore, when pyruvate is produced within a muscle cell lactate will inevitably be formed, regardless of the level of oxygenation.

<u>Lactate Dehydrogenase</u>.

Lactate dehydrogenase is the enzyme that catalyzes the reversible reduction of pyruvate to lactate. This enzyme occurs in five different isozyme forms (Lehninger, 1982). LDH is a tetrameric combination of two polypeptides termed M and H, which differ in composition and in sequence (Lehninger, 1982).

The five different LDH isozymes arise from the five possible tetrameric combinations of the M and H subunits: M_4, M_3H_1, M_2H_2, H_3M_1, and H_4. These forms have different Km values for pyruvate, they have different turnover numbers, and they are allosterically inhibited by pyruvate to different degrees. The M_4 and M_3H_1 LDH isozymes are collectively termed M-LDH. The M-LDH isozymes have low Km values for pyruvate; therefore, they have high affinities for pyruvate (Zubay, 1983). The M-LDH isozymes favor the rapid reduction of pyruvate to lactate even in very low concentrations of pyruvate (Lehninger, 1982). In addition, these isozymes have a high catalytic activity, relative to the other LDH isozymes, and they are not allosterically inhibited by pyruvate (Lehninger, 1982). The H_4 and H_3M_1 LDH isozymes are collectively termed H-LDH. The H-LDH isozymes have high Km values for pyruvate; therefore, they have higher affinities for lactate than for pyruvate (Zubay, 1983). The H-LDH isozymes favor the rapid oxidation of lactate to pyruvate (Lehninger, 1982). These isozymes are less active catalytically than the M-LDH isozymes. In addition, the H-LDH isozymes are strongly inhibited in high concentrations of pyruvate (Lehninger, 1982).

The functional significance of the different LDH isozyme forms is based on the relationship between muscle fiber composition, total LDH activity, and the distribution of LDH isozymes within the fiber (Skinner & McLellan, 1980). The Type II muscle fibers have a high M-LDH activity. These fibers have higher contractile rates, due to higher myosin ATPase activity (Gollnick & Hermansen, 1973), and they have fewer oxidative enzymes than the Type I fibers. Therefore, they are largely dependent on anaerobic glycolysis for ATP production (Mahler & Loke, 1985). Based on these characteristics and the high M-LDH activity in these fibers, it is clear that the Type II fibers are largely geared towards the production of lactate (Gollnick & Hermansen, 1973).

In contrast, the Type I skeletal muscle fibers and the cardiac muscle fibers are not geared towards lactate production, but rather to lactate utilization. These fibers demonstrate a high H-LDH activity. They have low contractile rates and a great oxidative capacity; therefore, they are largely dependent on aerobic energy production pathways to generate ATP. In addition, the Type I and cardiac muscle fibers have a greater resistance to fatigue than the Type II fibers. Based on these characteristics and on the high H-LDH activity in these fibers, it is clear that they are better suited to the utilization of lactate as a fuel than to the production of lactate. That is, the Type I and the cardiac muscle fibers can uptake exogenous lactate and utilize it as a fuel by oxidizing it to pyruvate and completely degrading it to CO_2 and H_2O in the mitochondria, through the TCA cycle.

Factors Affecting the Rate of Lactate Production.

The characteristic changes in blood lactate concentration, apparent in exercise of progressively increasing intensity, are related to the level of balance or imbalance between the rate of lactate production and the rate of blood lactate removal. This section will focus on the factors which affect the rate of lactate production. The following section will address blood lactate removal.

The rate of lactate production within each active muscle fiber is influenced by a number of different factors. Some of these factors are intrinsically mediated, while others are extrinsically mediated. These factors include: (a) the level of activation of the muscle fiber, (b) the composition of the muscle fiber, (c) the fuel supply of the muscle fiber, (d) the level of oxygenation of the muscle fiber, (e) the influence of catecholamines on the muscle fiber, and (f) the influence of certain metabolites within the cell on its metabolic activity. A discussion of each of these factors follows.

ACTIVATION OF THE MUSCLE FIBER. Fundamentally, the amount of lactate that is produced within a muscle fiber is dependent upon the level of activation of the fiber (that is, the rate at which the fiber is being recruited). The relationship between the activation of a fiber and the production of lactate within the fiber stems from the link that both of these factors have with the glycolytic rate. It has been demonstrated, in the previous section, that lactate production is largely a consequence of pyruvate production. As such, lactate production will be directly related to the rate of glycolysis. The rate of glycolysis is largely determined by the cell's requirement for ATP resynthesis, and this requirement is defined by the level of activation of the fiber.

The glycolytic rate is adjusted to meet the muscle fiber's requirement for ATP by certain enzymes along the glycolytic pathway whose activities are affected by the concentrations of key metabolites. There are two regulatory enzymes that control the entry of glucose residues into the glycolytic pathway. The first is hexokinase - which phosphorylates free glucose as it enters the cell. In skeletal muscle hexokinase is an allosteric enzyme that is inhibited by the product of its reaction, glucose 6-phosphate (Lehninger, 1982). The second regulatory enzyme is glycogen phosphorylase, which provides glucose residues to glycolysis from glycogen. In skeletal muscle, glycogen phosphorylase exists in an active and an inactive form, the relative amounts of each form determining the rate of glycogen breakdown (Lehninger, 1982). Besides controlling the rate of entry of glucose residues into glycolysis, there are two other regulation points along the glycolytic pathway. The phosphofructokinase (PFK) step is the first one that is regulated. PFK is a complex allosteric enzyme with many stimulatory and inhibitory modulators. In skeletal muscle the rate of PFK activity is regulated by the concentration of its substrates - ATP and

fructose 6-phosphate, and by its products - ADP and fructose 1,6-diphosphate. Other important modulators of PFK are AMP, citrate, Mg^{2+}, and phosphate (Lehninger, 1982; Zubay, 1983). The most important inhibitory modulators are ATP and citrate; the most important stimulatory modulators are AMP and fructose 1,6-diphosphate. The second site of regulation along the glycolytic pathway is the pyruvate kinase step. The activity of pyruvate kinase is lowered by high ATP concentrations, high acetyl-CoA concentrations, and by an abundance of long-chain fatty acids (Lehninger, 1982).

The concentration of ATP undoubtedly has a strong modulating effect on the rate of glycolysis in skeletal muscle. It is apparent that the glycolytic rate will be low when ATP concentrations are high, and when acetyl-CoA is being supplied to the mitochondria in adequate amounts by the β-oxidation of fatty acids. In these conditions PFK activity will be inhibited as will pyruvate kinase activity. Hexokinase will subsequently be inhibited when glucose-6-phosphate begins to accumulate because of the reduced activities of PFK and pyruvate kinase. Conversely, the glycolytic rate will be high when the concentration of ATP in the muscle fiber has fallen, and when the concentration of ADP has risen. These two changes will stimulate glycolysis by activation PFK and pyruvate kinase.

Increased activation or recruitment of a muscle fiber will increase the utilization of ATP for contractile work, and therefore, will increase the requirement of the fiber for ATP resynthesis. The resulting changes in ATP concentration will activate glycolysis. As a result, pyruvate production will increase. The enhanced production of pyruvate will result in an enhanced production of lactate. Therefore, the rate of lactate production within a muscle fiber will be directly related to the level of activation of the fiber.

Decreased recruitment of a muscle fiber will decrease the demand for ATP resynthesis in the fiber. The resulting increase in ATP concentration within the cell will have an inhibitory effect on glycolysis. As a result, pyruvate production, and consequently lactate production, will decrease. However, even when a muscle fiber is inactive (in the contractile sense) glycolysis will be active to some degree. Glycolysis will always be active because the cell requires a certain amount of carbohydrate (pyruvate) to facilitate the activity of the TCA cycle (Walsh & Banister, 1988). Therefore, even at rest glycolysis will be active, pyruvate will be produced, and subsequently lactate will be produced.

MUSCLE FIBER COMPOSITION. The rate of lactate production within a muscle fiber is directly related to the level of activation of the fiber; however, the absolute amount of lactate that is produced will greatly depend on the physiological and biochemical characteristics of the muscle fiber that is recruited. The two skeletal muscle fiber types that will be addressed are the Type I and the Type II fibers. The characteristics of these fibers differ greatly. The Type I skeletal muscle fibers have a high oxidative capacity, a high fatigue resistance, a low ability to generate power, and a high H-LDH isozyme distribution. Relative to the Type I fibers, the Type II skeletal muscle fibers have a lower oxidative capacity, a lower mitochondrial density, a smaller capillary to fiber ratio, a low resistance to fatigue, a greater glycolytic capacity, a much greater power generating ability, and a high M-LDH isozyme distribution. It is obvious from these characteristics that the Type I fibers are geared towards aerobic metabolism and the Type II fibers are geared towards anaerobic metabolism. As such, recruitment of Type II fibers will result in a much greater production of lactate than the recruitment of Type I fibers (Bauer, Reichman, & Hofer, 1986).

MUSCLE FIBER FUEL SUPPLY. The rate of lactate production within a muscle fiber is dependent upon the level of activation of the fiber, as well as the characteristics of the fiber. In addition, the amount of lactate that is produced will depend on the availability of substrates to the fiber. For example, increasing the free fatty acid concentration in the blood has been shown to decrease the concentration of blood lactate during exercise (Costill et al., 1977). Decreasing muscle glycogen stores has a similar effect on the blood lactate concentrations during exercise (Hughes, Turner, & Brooks, 1982). In contrast, when muscle glycogen stores are elevated above normal resting levels, lactate production during exercise will be above that seen with normal glycogen levels (Richter & Galbo, 1986). Therefore, the rate of lactate production within a muscle cell will be affected by the level of substrate availability, as glycolytic activity appears to be higher when muscle glycogen stores are high as opposed to when fatty acid availability is high.

MUSCLE FIBER OXYGENATION. The rate of lactate production within a muscle fiber may also vary with the amount of oxygen that is supplied to the fiber. Performing exercise while breathing hypoxic gas has been shown to increase blood lactate concentration, while breathing hyperoxic gas generally decreases the blood lactate concentration, at the same relative exercise intensity (Hogan & Welch, 1984, 1986). From these results it may be interpolated that lactate production within a muscle fiber will be reduced if the fiber is well oxygenated, thus allowing maximization of the oxidative capacity of the fiber. These results, however, may be misleading since the decrease in lactate production apparent while breathing hyperoxic gas may be due to the toxicity of the gas on certain glycolytic enzymes (Walsh & Banister, 1988).

INFLUENCE OF CATECHOLAMINES. The concentration of catecholamines in the blood can also influence the rate of lactate production within a muscle fiber. Characteristically, blood catecholamine concentration increases curvilinearly with increasing exercise intensities. Lactate production in muscle tissue that is not actively contracting is affected by the blood catecholamine concentration (Richter, Ruderman, & Galbo, 1983; Sahlin, Henriksson, & Juhlin-Dannfelt, 1984). The effect of catecholamines on contracting skeletal muscle tissue is more difficult to verify; however, it is expected to be similar to the effect produced in inactive tissues (Walsh & Banister, 1988).

Glycogenolysis is activated by catecholamines through the cascade effect initiated by activating adenyl cyclase. Activation of adenyl cyclase results in the eventual conversion of glycogen phosphorylase b to the more active form - glycogen phosphorylase a (Lehninger, 1982; Zubay, 1983). Therefore, glycogen utilization can be activated or enhanced by an increase in catecholamine concentration, the result of which will be an increase in the production of lactate. Since the Type II skeletal muscle fibers have a higher glycolytic capacity than the Type I fibers, the production of lactate due to catecholamine influence will be greater in the Type II fibers (Walsh & Banister, 1988). In conclusion, it appears that catecholamines may have some influence on lactate production during exercise; however, their affect does not account for the total increase in lactate production resulting from acute exhaustive exercise (Cartier & Gollnick, 1985).

INFLUENCE OF METABOLITES. Muscle fibers that are contracting at fairly high rates will derive some energy from the adenylate kinase reaction which converts two moles of ADP to ATP and AMP. A subsequent reaction

converts AMP to IMP and NH_3. This reaction is catalysed by the enzyme adenylate deaminase. NH_4^+, AMP and IMP may influence the production of lactate by activating certain enzymes along the glycolytic pathway (such as PFK) (Griffiths & Rahin, 1978). This finding is supported by the high correlation that has been demonstrated between PFK activity and AMP deaminase activity (r=0.97) (Winder, Terjung, Baldwin, & Holloszy, 1974). Therefore, the production of lactate may be enhanced by elevated levels of NH_4^+, AMP and IMP. This enhancement will likely have a greater effect on the Type II skeletal muscle fibers because of the increased glycolytic capacity of these fibers.

In summary, it is apparent that a wide range of factors influence the rate at which lactate is produced within skeletal muscle. Of these factors, the two which appear to have the most significant influence are the level of activation of the individual muscle fibers and the type of fibers that are being recruited. The level of lactate production can also be modulated by fuel supply, oxygen supply, catecholamines, and metabolites.

Lactate Efflux from Muscle Cells

The lactate and hydrogen ions that are produced in muscle cells can remain there or be released to the extracellular space and the blood (Hultman & Sahlin, 1980). Simple diffusion is commonly presented as the transport mechanism for lactate/lactic acid across the sarcolemmal membrane (Jensen, 1980). However, some research demonstrates that the lactate transport mechanism can be saturated; therefore, it may involve more than just simple diffusion (Donovan & Brooks, 1983; Eldridge, T'so, & Chang, 1974). Whether lactate leaves the cell in a dissociated or an undissociated state is also controversial. There is some

support, however, for the view that lactate passes through the sarcolemmal membrane in an undissociated form (Mainwood & Renaud, 1985; Walsh & Banister, 1988)

The rate at which lactate leaves the muscle cell is affected by both the extracellular pH and the extracellular concentration of lactate. Extracellular acidosis and/or high extracellular lactate concentrations will reduce lactate efflux, while extracellular alkalosis and/or low extracellular lactate concentrations will enhance lactate efflux (Hultman & Sahlin, 1980).

Lactate Entry into the Blood and Distribution Throughout the Body

The lactate that diffuses from muscle cells into the extracellular space can diffuse into the blood capillaries that are near the muscle cell and in this manner travel to various tissues throughout the body.

Blood Lactate Elimination During Exercise

Lactate production increases with increased exercise intensity, yet below the lactate threshold intensity the blood lactate concentration remains fairy low and does not progressively accumulate. It is obvious then that there are mechanisms which remove lactate from the blood, and that these mechanisms increase in parallel with increases in lactate production, at least until the lactate threshold exercise intensity is reached. The possible routes for lactate elimination during exercise will be presented, along with the factors which may affect the rate of lactate elimination.

The lactate/lactic acid that is produced within a muscle cell can travel from the cell into the interstitia and then into the blood. It is then distributed throughout the body by means of the circulatory system. Since lactate is a partially catabolysed glucose unit, it can still serve as an energy source for tissues that have the metabolic machinery needed to further degrade the 3-carbon molecule.

Lactate can be reconverted to pyruvate via the lactate dehydrogenase step in glycolysis (refer to page 28). As previously discussed, tissues with the greatest potential for the conversion of lactate to pyruvate are those that contain a relatively higher proportion of the H-LDH isozyme than the M-LDH isozyme. The H-LDH isozyme is found in high distributions in tissues that function, to a great extent, aerobically, such as the cardiac muscle, the Type I skeletal muscle, the liver, and the kidneys. The cardiac muscle tissue and the Type I skeletal muscle fibers can use blood lactate as a fuel; once inside the muscle cell lactate can be converted to pyruvate, the pyruvate can enter the mitochondria where it can be completely oxidized to CO_2 and H_2O by means of the TCA cycle. The lactate taken up by the liver is predominantly used in the synthesis of glucose and glycogen. In the liver, lactate is converted to pyruvate, then to oxaloacetate. Oxaloacetate is a TCA cycle intermediate but it is also a precursor to phosphoenol pyruvate which is a molecule in the gluconeogenesis pathway. In this manner blood lactate is used by the liver to form glucose and glycogen. However, relatively small amounts of lactate are taken up by the liver during exercise (Hultman & Sahlin, 1980).

The main route of blood lactate elimination during exercise is through oxidation (Brooks, 1985). Since the skeletal muscle represents the major site of

oxidative metabolism, it has been proposed that the most significant site of lactate removal is the skeletal muscle (Brooks, 1985; McGrail et al., 1978). While initially it appears paradoxical that lactate is both produced and eliminated by the skeletal muscle, this is actually possible because skeletal muscle is composed of both Type I and Type II fibers (Gollnick & Hermansen, 1973; Issekutz, Shaw, & Issekutz, 1976). While the Type II fibers are the main producers of lactate during exercise, the Type I fibers, with their high level of capillarization, have a great potential for lactate elimination from the blood (Tesch, Daniels, & Sharp, 1982). The Type I fibers can similarly remove lactate from the interstitia, before it ever reaches the blood.

The rate at which lactate is eliminated from the blood during exercise is a function of several factors. These factors include: (a) exercise intensity, (b) blood flow rates, (c) level of intramuscular glycogen stores, and (d) blood lactate concentrations. Increasing metabolic activity increases the blood lactate removal rate (Eldridge, 1975; McGrail et al., 1978). The increased metabolism in the active skeletal muscle tissues increases their requirement for fuel. It is hypothesized that in Type I skeletal muscle and in the cardiac muscle the increased demand for fuel increases the use of available blood lactate and lactate in the interstitia. The use of such exogenous lactate may actually have a carbohydrate sparing effect on these tissues, because the lactate would provide a large portion of the carbohydrate supply required to maintain TCA cycle activity.

There are three other factors which influence the rate of blood lactate elimination. Increasing blood flow rates to tissues which remove lactate from the blood will increase the potential for lactate elimination by these tissues, and

obviously the reverse is also true. Increasing the concentration of intramuscular glycogen will increase the use of endogenous carbohydrate as an energy supply, and therefore, will increase lactate production and reduce blood lactate elimination. And finally, the concentration of lactate in the blood appears to have an effect on the rate at which it is eliminated. At very high blood lactate concentrations the removal of lactate from the blood is enhanced (Depocas, Minaire, & Chatonnet, 1969). It is possible that there is a mass action effect caused by the high blood lactate concentration which enhances its removal by facilitating diffusion into removal tissues (Depocas et al., 1969; Issekutz et al., 1976).

Explanations for the Blood Lactate Threshold Phenomenon

The characteristic changes in blood lactate concentration apparent in exercise of progressively increasing intensity may be the result of a number of different events that occur within and without each active muscle fiber. One explanation for this phenomenon is that at a certain exercise intensity the supply of oxygen to the muscle fiber becomes inadequate, and anaerobic production of ATP is initiated to maintain the required supply of ATP in the contracting fiber. Another explanation is that at certain exercise intensities the predominant use of free fatty acids as a fuel no longer provides ATP at an adequate rate. Consequently, the glycolytic rate is enhanced to satisfy the ATP requirements, and lactate is produced because of the augmented glycolytic activity. Yet another explanation identifies the recruitment of progressively larger motor units as the cause of the increased blood lactate concentration during progressive exercise. As the recruitment of the large, Type II fibers increases both spatially and temporally, lactate production increases and blood

lactate elimination cannot meet the increased rate of entry of lactate into the blood. The increase in blood lactate concentration may also be the result of a reduced capacity to remove lactate from the blood. It has also been suggested that the influence of catecholamines on the metabolism of muscle fibers is responsible for the blood lactate threshold. Other factors that have been implicated causally in the blood lactate threshold are temperature, lactate efflux from production sites, and endogenous metabolite activation of glycolysis.

Each of the explanations has supportive evidence; however, for none is the evidence conclusive. It may well be that blood lactate accumulation begins at a specific exercise intensity because of the combined effect of some or all of these factors. Nevertheless, it is apparent that at a specific exercise intensity the concentration of blood lactate begins to progressively increase and this increase is due to a disequilibrium between the rate of lactate entry into the blood and the rate of blood lactate removal.

Muscular Fatigue

The mechanisms responsible for muscular fatigue have been an elusive aspect of exercise physiology. The reason being that this area of exercise physiology is highly complex. The following discussion of muscular fatigue is divided into four sections. In the first section muscular fatigue will be defined. The second section consists of a brief review of the major processes involved in muscle contraction. This review provides the background necessary for an understanding of the third section, which deals with a number of the different fatigue sites and fatigue mechanisms that have been identified by various researchers. The final section consists of a discussion of the possible mechanisms of muscular fatigue during endurance events.

Definition of Muscular Fatigue

Muscular fatigue is a very common aspect of physical activity. It occurs when a muscle, or a group of muscles, is used repeatedly. This repeated use eventually results in a decrease in the tension producing ability of the muscle. In general, muscular fatigue is defined as the inability to maintain a required or expected level of muscular performance. The degradation in muscular performance is usually described in terms of either a decrease in force production, movement velocity, or work output. For the purposes of this paper, muscular fatigue is defined as the inability to maintain an expected level of work output.

Review of Muscle Contraction

Even though muscular fatigue is a common phenomenon, it is very complex and its underlying causes are not well understood. A great deal of the complexity surrounding muscular fatigue arises from the number of steps involved in the excitation and contraction of a muscle fiber. A brief review of these steps follows.

Muscular contraction often begins with the generation of a signal in the brain. This signal is translated to a muscle cell by a specialized nerve cell called a motoneuron. The cell body of a motoneuron is located in the spinal cord. It can receive many excitatory and inhibitory inputs from a number of different sources. When the excitatory inputs to the cell body are enough to override the inhibitory inputs, an action potential will be generated at the cell body that will travel down the axon of the motoneuron. The axon extends to the muscle it is associated with. It branches at various points to produce a number of corollary axons that lead to different muscle fibers within the same muscle.

The next stage of signal transmission involves the transfer of the signal from the motoneuron to the muscle fiber. The point where the terminal end of the axon (axon terminal) meets with the muscle cell is called the neuromuscular junction. Here the axon terminal and the muscle cell membrane are separated by a small gap. Transmission of a nerve impulse across this gap is achieved by use of a chemical transmitter, acetylcholine, which is stored in membrane bound vesicles on the terminal.

When an action potential reaches the axon terminal, it depolarizes the membrane at the terminal. This depolarization causes the membrane permeability to Ca^{2+} to change. As a result, Ca^{2+} diffuses into the terminal and triggers the release of acetylcholine into the neuromuscular junction. The acetlycholine rapidly diffuses across the gap to the motor end plate membrane of the muscle cell, where there are receptors for it. The permeability of the motor end plate membrane to Na^{+} and K^{+} is greatly increased by the binding of acetylcholine to the receptor sites. This permeability change results in a depolarization of the motor end plate membrane, and thus, the signal is translated from the motoneuron to the muscle cell.

The depolarization of the motor end plate membrane causes an action potential to be propagated over the muscle cell membrane (sarcolemma). Once an action potential is initiated at a point on the sarcolemma, it travels laterally over the length of the muscle fiber. To produce contraction the action potential must be translated to the contractile proteins at the interior of the muscle cell. This is achieved by T-tubules which run perpendicular to the muscle fibers. The T-tubules are believed to be responsible for translation of excitation from the sarcolemma to the sarcoplasmic reticulum. The excitation coupling between

the T-tubules and the sarcoplasmic reticulum results in the release of Ca^{2+} from the terminal cisternae of the sarcoplasmic reticulum into the environ of the contractile proteins - actin and myosin.

The Ca^{2+} influx allows the actin and myosin filaments to interact. This interaction is the basis of muscle fiber contraction and it is termed the cross bridge cycle. The cross bridge cycle is inactive in resting muscle cells because the interaction between the actin and myosin filaments is inhibited; that is, the active site for myosin binding on the actin filament is not exposed. With the large influx of Ca^{2+} from the terminal cisternae of the sarcoplasmic reticulum the actin active site is exposed and the cross bridge cycle is disinhibited.

The cross bridge cycle is made up of a series of steps which ultimately results in muscle fiber contraction. ATP plays a key role at two points in the cycle. The cycle involves the enzyme actomyosin ATPase. The myosin head has a binding site for actin and an actomyosin ATPase site. Only the high energy form of the myosin head can bind to the actin filament. The high energy myosin head is formed when a molecule of ATP is hydrolyzed by actomyosin ATPase. When the high energy myosin head binds to the actin filament, the stored energy from ATP hydrolysis is released, and this release results in the movement of the cross bridge. In order for the myosin head to release the actin, ATP must bind again to the myosin. When this binding occurs the myosin head is released from the actin and the bound ATP can once again be hydrolyzed to produce a high energy myosin head. In such a manner the cross bridge cycle can be continuously repeated. The cycle will cease, and the muscle will relax, when Ca^{2+} is removed from the contractile protein environ, causing the actin active site to no longer be exposed to the high energy myosin head.

Following muscle contraction, a number of different processes must be activated to return the muscle to its resting state. To return the motor end plate membrane to its resting state the receptor bound acetylcholine must be broken down. This is accomplished by an enzyme called acetylcholinesterase which is located at the motor end plate membrane. In order to reestablish the resting membrane potential of the sarcolemma, an ATP requiring process must be active. This process involves the enzyme Na^+/K^+ ATPase, and is referred to as the Na^+/K^+ pump. Through the Na^+/K^+ pump the resting intracellular and extracellular concentrations of Na^+ and K^+ are actively restored. In order to stop the cross bridge cycle Ca^{2+} must be resequestered into the sarcoplasmic reticulum. This is achieved through a process which requires ATP, involves the enzyme Ca^{2+} transport ATPase, and is referred to as the Ca^{2+} pump. The activity of these three processes returns the muscle cell to its resting state, and thus enables it to rapidly respond to subsequent motoneural activation.

Possible Fatigue Sites and Mechanisms

When a muscle exhibits fatigue it may be the result of a decreased or deteriorated function at any of the steps along the series of events leading to muscle contraction. Different researchers have attributed fatigue to different steps or to different mechanisms at a step. A number of the proposed fatigue sites and mechanisms will be presented here in succession from those in the brain to those in the contractile elements of the muscle fiber.

The first potential site of fatigue is the brain. Central fatigue is the term that is used to describe a decrease in muscle force due to a decreased motor drive from the central nervous system (CNS). With a lower CNS drive, muscle

activation cannot be maintained at the required level. Central fatigue has often been attributed to psychological factors such as inadequate motivation or a decreased ability to tolerate pain. Some research, however, has suggested that central fatigue may be physiological - the decreased motor drive from the CNS may be a response to afferent feedback from the muscle. This feedback may reach the brain in two ways: (a) via afferent feedback from the sensory apparati of the muscle, or (b) through the passage of substances within the blood, such as NH_3, across the blood brain barrier (Green, 1987).

Another mechanism of central fatigue focuses on the motoneuron. It has been suggested that since the cell body of a motoneuron is a focal point for a great deal of excitatory and inhibitory input, it is possible that the excitability of a motoneuron or a motoneural pool could be decreased by an increase in local inhibitory input (Green, 1987). The effect of decreasing the excitability of a motoneuron would be a decrease in muscle force production, providing CNS activation remains constant. Thus, there are three proposed mechanisms for central fatigue: (1) modification of the CNS motor drive by afferent feedback from the muscle, (2) modification of the CNS motor drive by substances carried in the blood, and (3) modification of the excitability of a motoneuron or motoneural pool. However, whether or not these mechanisms are actually active, and whether or not they are responsible for muscular fatigue has not yet been determined.

Muscular fatigue due to central mechanisms may be more applicable to certain types of activities than others. Some researchers have found evidence which suggests that the fatigue resulting from high intensity activity may be due to central factors. For instance, high intensity exercise involving Type II

muscle fibers results in elevated levels of blood NH_3 (Meyer, Dudley, & Terjung, 1980). The passage of NH_3 in the blood across the blood brain barrier may be a metabolic feedback pathway which results in a decreased motor drive. It has also been suggested that the Type II motor units, being more fatigable than the Type I motor units, would be better candidates for the selective motoneural pool inhibition, which was previously outlined as a possible mechanism of fatigue (Green, 1987). Contrary to these views, other researchers have found little evidence of central fatigue during high intensity exercise if subjects are well motivated. In studies which compared maximal voluntary contractions to electrically stimulated contractions researchers found little difference in the force that was generated with each, if the subjects were well motivated (Gibson & Edwards, 1985). From their research, Bigland-Ritchie & Woods (1984) have concluded that central fatigue can be overcome in maximal contractions of 45 to 60 seconds in duration. It is generally accepted that in short duration high intensity exercise, central fatigue is not the critical fatiguing mechanism at work. In prolonged activity, however, the role of central fatigue has not been extensively evaluated (Bigland-Ritchie & Woods, 1984).

In many cases, muscular fatigue is apparent even though the motor drive from the CNS is adequate. In such situations fatigue is not due to central factors, but must be attributed to peripheral factors. In fact, muscular fatigue is most frequently associated with some aspect of the peripheral contractile events - either there is failure of the motoneural action potential transmission, or of one or more of the events leading to contraction within the muscle fiber.

The first potential peripheral fatigue site is the motoneuron. A neuromuscular block will occur if a motoneuron is unable to translate an

excitatory motor drive into a regenerative action potential at the muscle cell membrane. A number of possible mechanisms have been identified for neuromuscular block. One possible mechanism is that excitation is lost at the axonal branch points of a motoneuron (Green, 1987). Another possible mechanism for neuromuscular block is that the integrity of the motoneural membrane is disrupted. A loss of integrity of the membrane would change its electrical characteristics, and these characteristics are critical for signal transmission (Green, 1987). Yet another mechanism may be that the amount of acetylcholine being released from the axon terminal is not enough to depolarize the motor end plate membrane (Green, 1987). These three possible mechanisms for neuromuscular block must be investigated further before any conclusions can be made concerning their role in muscular fatigue and their specificity to certain types of activities. Presently, there seems to be agreement that failure of the motoneuron is a rare occurrence (Bigland-Ritchie & Woods, 1984; Clamann, 1987).

Muscular fatigue is more commonly associated with failure of some aspect of contraction within the muscle cell. The first site of concern is the motor end plate membrane. If there is failure at this site no action potential will develop (Green, 1987). It has been speculated that a cause of failure may be that for some reason the membrane becomes incapable of responding to acetylcholine (Clamann, 1987). If this occurs the motor end plate membrane will not depolarize and the action potential will not be generated. This mechanism, however, is only speculation and as such muscular fatigue is rarely attributed to failure at the motor end plate membrane.

The sarcolemma is the next potential site of fatigue within the muscle cell. Muscular fatigue will result if the sarcolemma fails to regenerate an action potential (Green, 1987). Normal sarcolemma function is dependent on an intact membrane. It has been suggested that in prolonged activity there may be reversible membrane damage (Green, 1987; Tibbits, 1987). A loss of membrane integrity will change the permeability of the membrane to ions and to molecules, and thus will affect the ability of the membrane to depolarize (Green, 1987). A loss of membrane integrity will also affect other membrane processes that are critical to normal cell function, such as pH regulation (Tibbits, 1987). Sarcolemmal failure will also occur if the resting membrane potential has not been reestablished before the next action potential arrives. The Na^+/K^+ ATPase enzyme is responsible for reestablishing the resting membrane potential. This enzyme requires ATP to function and it is regulated by the concentration of Na^+ and K^+, and by free fatty acids, insulin, and catecholamines (Green, 1987). Thus, the activity of the enzyme can be decreased through local substrate depletion or through enzyme impairment (Tibbits, 1987). If the activity of the Na^+/K^+ ATPase is decreased, repolarization of the sarcolemmal membrane will require more time. With an increased repolarization time, the rate at which the muscle cell can be effectively stimulated will be reduced, and subsequently the force producing capacity of the muscle will decrease.

Sarcolemmal failure has been evidenced in human muscle fibers in vitro and in vivo. In vitro it usually occurs in response to high frequency stimulation, and it results in a rapid loss of force (Gibson & Edwards, 1985). In vivo, during a sustained voluntary contraction, there is a natural fall in firing frequency. It has been suggested that this natural decrease may be a protective mechanism built in to the system to avoid incurring sarcolemmal failure (Gibson & Edwards,

1985). Other researchers have suggested that the decrease in firing frequency may simply be an attempt at signal conservation; where only enough signal is relayed as can be utilized at any point in time (Bigland-Ritchie & Woods, 1984). The latter suggestion implies a great deal of afferent feedback, since the motor drive would have to be continuously modified to suit the active state of the muscle cell. The case for sarcolemmal failure during high intensity activity is poor, since there are built-in protective mechanisms to guard against it. However, sarcolemmal failure during prolonged activity may result from a loss of integrity of the sarcolemma, and/or a decreased activity of Na^+/K^+ ATPase.

The next potential fatigue site within the muscle cell is the T-tubule. The T-tubule may fail to couple excitation from the sarcolemma to the sarcoplasmic reticulum. One speculated mechanism is that at some point the T-tubules may begin to retain Ca^{2+} (Green, 1987).

The sarcoplasmic reticulum is also considered a potential fatigue site. In prolonged activity both reduced Ca^{2+} release from the sarcoplasmic reticulum and reduced Ca^{2+} uptake by the sarcoplasmic reticulum have been reported (Green, 1987). A reduction in the amount of Ca^{2+} that is released would decrease the number of cross bridges possible, thus reducing the force of contraction. A reduction in the uptake of Ca^{2+} from the contractile protein environ would increase the relaxation time. The mechanism responsible for Ca^{2+} retention is unknown; however, there is a possible explanation for reduced Ca^{2+} uptake.

A reduction in Ca^{2+} uptake into the sarcoplasmic reticulum may be caused by a modification of the activity of the enzyme responsible for Ca^{2+} uptake - Ca^{2+} ATPase. This enzyme has a number of effector controls (Green, 1987). In

vitro, reversible alterations in sarcoplasmic reticulum function, attributable to changes in Ca^{2+} ATPase activity, have resulted from prolonged exercise (Tibbits, 1987). It has been suggested that an increased concentration of ADP decreases the free energy of hydrolysis in Ca^{2+} ATPase (Tibbits, 1987). As well, the enzyme may be inhibited by a decreased concentration of ATP or by a decreased pH (Tibbits, 1987). If Ca^{2+} ATPase activity is decreased the result will be an increase in relaxation time, because the actin-myosin dissociation will be delayed. Gibson and Edwards (1985) proposed that the decreased Ca^{2+} ATPase activity was also a protective mechanism built-in to counteract fatigue, since it results in a prolonged force maintenance for each action potential. It appears that the sarcoplasmic reticulum may be a site for muscular fatigue, especially in prolonged activities.

Certain aspects of the cross bridge cycle may also be vulnerable to fatigue. Actomyosin ATPase is the enzyme that hydrolyzes ATP to provide the energy for cross bridge movement. This enzyme has a number of regulators. For example, high concentrations of the products of ATP hydrolysis: ADP, P_i, and Mg^+ may alter the activity of actomyosin ATPase and may reduce the free energy of ATP hydrolysis (Green, 1987). In addition, low ATP concentrations, or high hydrogen ion concentrations will reduce the activity of actomyosin ATPase (Gibson & Edwards, 1985). Thus, even when the release of Ca^{2+} from the sarcoplasmic reticulum is adequate to promote optimal actin-myosin association, contraction may still fail because of a depletion of ATP or an accumulation of metabolites (Clamann, 1987). Impairment of the cross bridge cycle appears to be associated more with activities of high intensity than with prolonged activities of lower intensity (Green, 1987).

Certain metabolic factors, such as the rate of ATP resynthesis, may also play a significant role in muscular fatigue. An adequate supply of ATP is needed: (a) by actomyosin ATPase for the cross bridge cycle, (b) by Ca^{2+} ATPase for resequestering Ca^{2+} into the sarcoplasmic reticulum, and (c) by Na^+/K^+ ATPase for restoring the resting membrane potential of the sarcolemma. Obviously, the supply of ATP is critical to the contractile process within the muscle cell. It is well known that the concentration of ATP within a muscle cell is fairly stable and cannot be driven below approximately 60 to 70% of the resting level, even after exhaustive exercise (Noble, 1986). If ATP hydrolysis could continue while ATP resynthesis was inactive, the intracellular stores of ATP would be rapidly depleted and the cell would die. It follows then, that there are mechanisms within the muscle cell which carefully match the rates of ATP hydrolysis and resynthesis. Thus, ATP will not be continuously hydrolyzed and used by actomyosin ATPase, Ca^{2+} ATPase, or Na^+/K^+ ATPase unless it is being resynthesized at an adequate rate. As such, the fatiguing mechanism may be related to the factors which influence the rates of ATP resynthesis and hydrolysis.

The rate at which ATP can be resynthesized in the muscle cell is dependent upon the energy production pathway that is being predominantly used at any point in time. The major pathway used for ATP resynthesis in the muscle cell will vary with the intensity of activity that is being performed, and with the type of muscle cell that is being recruited. During low to moderate intensity activity ATP resynthesis occurs primarily through oxidative pathways. Such intensities of activity can be continued for prolonged durations before fatigue is apparent. Obviously, oxidative ATP production is capable of maintaining a balance between ATP hydrolysis and ATP resynthesis for these activity

intensities. The muscular fatigue induced by prolonged activity is commonly attributed to glycogen depletion in the active muscle fibers. This reasoning is based on the fact that at the point of fatigue there are usually ample reserves of fat substrate remaining; however, the body's carbohydrate supply is depleted (Bergstrom, Hermansen, Hultman, & Saltin, 1967; Bergstrom & Hultman, 1967). Fatigue may occur when the body's carbohydrate reserves are depleted because it appears that an obligatory amount of carbohydrate is required to facilitate the activity of the TCA cycle. Therefore, when the carbohydrate reserves become depleted, aerobic glycolysis is not active at a rate that can maintain the required level of TCA cycle activity. Consequently, the rate at which ATP can be resynthesized will be reduced, and the work output will drop.

During high intensity activities ATP resynthesis occurs predominantly through anaerobic glycolysis. If ATP is being resynthesized through anaerobic glycolysis, the lactate and hydrogen ion concentrations in the muscle cell will rise. In the past, lactate accumulation within a muscle cell was implicated as the primary cause of muscular fatigue during high intensity exercise. This reasoning was based on the observation that the intracellular concentrations of lactate at the point of exhaustion were extremely high - as much as 20 to 30 times as high as those seen at rest. While lactate accumulation itself has not been proven as a cause of muscular fatigue (Tesch, 1980), it has definitely demonstrated a very strong association with it (Hermansen, 1981). This association may be a reflection of the close relationship between lactate and hydrogen ion concentration. When the anaerobic glycolytic metabolic pathway is active there is a rapid production of both lactate and hydrogen ions. The hydrogen ions that are produced can efflux from the muscle, or be buffered by one of the buffering systems within the cell, such as the bicarbonate or the

protein buffering systems; however, a great deal of the hydrogen ions that are produced will accumulate in the muscle cell during maximal intensity activity. The result of this accumulation will be a drop in intracellular pH. Intracellular pH levels of 6.4 to 6.5 have been reported at the point of exhaustion from maximal repeated exercise (Hermansen, 1981).

High intracellular concentrations of hydrogen ion and the resulting drop in pH have been implicated in two possible fatigue mechanisms. The activities of two key enzymes in the glycolytic sequence - phosphorylase and phosphofructokinase (PFK), are modified by changes in intracellular pH. These enzymes have a decreased activity at reduced pH. The enzyme inhibition appears to be a protective mechanism which prevents continued hydrogen ion production, thereby reducing the risk of protein denaturation and the destruction of other acid labile cell components due to low pH. Therefore, a feedback mechanism exists where the activity of the anaerobic glycolytic pathway is severely retarded when the end products of the pathway accumulate in the cell. Fatigue will result when the activity of the anaerobic glycolytic pathway is reduced because of the resulting decrease in the rate of ATP resynthesis.

If the contractile proteins were allowed to hydrolyze ATP while the main resynthesis pathway was inactive the intracellular stores of ATP would quickly be depleted and the cell would die. Therefore, decreased intracellular pH also triggers mechanisms which control the activity of the contractile proteins so that ATP is never completely depleted. The Ca^{2+} sensitivity of the contractile proteins decreases with decreasing intracellular pH. It appears that the accessibility of the binding sites is altered by the myofibrillar charge which is

pH dependent (Mainwood & Renaud, 1985). Thus, the decreased pH causes a drop in the number of Ca^{2+} that can bind to troponin, and this results in fewer actin-myosin interactions and a decreased contractile ability (Tesch, 1980). The accumulation of hydrogen ions and low intracellular pH also decreases the activity of actomyosin ATPase and Ca^{2+} ATPase. Thus, decreased intracellular pH affects the contractile activity of a muscle fiber by reducing the rate at which ATP can be hydrolyzed and by reducing the rate of ATP resynthesis.

It is apparent that changes in intracellular pH may affect force generation through two mechanisms. The first of these involves the reduced activity of the regulatory enzymes of glycolysis caused by the decreased pH. The second involves the impairment of the contractile process caused by the increased hydrogen ion concentration (Hermansen, 1981). There is a close relationship between intracellular pH and the intracellular lactate concentration immediately following exercise (Tesch, 1980). Therefore, while lactate accumulation cannot be proven as the main causation of fatigue, it is certainly a fatigue indicator because of its close relationship to intracellular pH (Tesch, 1980).

Muscular Fatigue During Endurance Events

Based on the previous discussion, it is apparent that during prolonged, moderate intensity activity fatigue may be caused by a number of possible mechanisms. The continuous, repetitive nature of this type of activity supports the view that reversible damage to either the motoneural membrane and/or the sarcolemmal membrane may be responsible for fatigue. Another possible mechanism for fatigue in prolonged activities is substrate depletion, namely, carbohydrate depletion. In addition, the effects of central fatigue during

prolonged activities remains to be investigated. At this point, it appears that the most probable causes of fatigue during prolonged, moderate intensity activity are sarcolemmal membrane damage, motoneural membrane damage, and/or carbohydrate depletion.

While the fatigue resulting from prolonged, moderate intensity work appears to be the most relevant for application to endurance events; the fatigue resulting from high intensity activity cannot be overlooked within this context. Most endurance events are predominantly made up of activity at moderate intensities - very close to the individual lactate threshold. However, activity intensities can climb to suprathreshold levels within the event if certain circumstances such as terrain, weather, or competitive strategy dictate that they must. The possible mechanisms of fatigue resulting from high intensity activity have been previously outlined. This type of fatigue appears to be largely due to the affect of various metabolites on the rate of ATP resynthesis and on the rate of ATP hydrolysis. The two metabolites of key concern are lactate and hydrogen ions. These are produced rapidly in Type II muscle fibers with the increased glycolytic activity resulting from high rates of activity. It is obvious that following suprathreshold intensity surges of activity within an endurance event, strategies should be employed to ensure adequate lactate and hydrogen ion removal so that the fatigue induced by these metabolites can be avoided.

The section which follows discusses studies that have investigated different strategies for recovering from high intensity activities. From the previous discussion on fatigue it was revealed that the high proton load experienced by the Type II muscle fibers due to high intensity activity was a primary fatiguing

influence. In order to recover from such high intensity activity it follows that the proton load must be reduced. Lowering extracellular lactate and raising extracellular pH enhances lactate efflux and probably hydrogen ion efflux as well (Hultman & Sahlin, 1980). The effect of various recovery strategies following high intensity activity is usually assessed by monitoring changes in blood lactate concentration. This practice may have initially been employed because lactate was thought of as a primary fatiguing metabolite; however, it is still valid since "the uptake and disposal of lactate in the blood will most probably correspond to similar changes in hydrogen ion" (Hultman & Sahlin, 1980).

Blood Lactate Removal During Recovery Exercise

It is a well-established fact that the rate of decrease in blood lactate concentration, following a period of high intensity exercise, is faster if the recovery period is active as opposed to passive (Belcastro & Bonen, 1975; Bonen & Belcastro, 1976; Davies et al., 1970; Depocas et al., 1969; Hermansen & Stensvold, 1972; McGrail et al., 1978; McLellan & Skinner, 1982; Stamford et al., 1981; Weltman et al., 1979). The increased blood lactate removal, apparent during active recovery, is explained by the fact that blood lactate can be used as a substrate by the heart and the Type I skeletal muscle tissue (McGrail et al., 1978). If the activity of these tissues is augmented, as it is during active recovery, the requirement for fuel will be increased, and subsequently the uptake of lactate by these tissues will be enhanced (Depocas et al., 1969; Eldridge, 1975; Issekutz et al., 1976). In addition, active recovery will cause an increase in the blood flow to these tissues, thereby further increasing the potential for blood lactate uptake at these sites (Astrand & Rodahl, 1986).

Blood lactate removal is enhanced by an active recovery period; however, elimination will be sub-optimal if the recovery intensity is too high. Belcastro and Bonen (1975) found that recovery intensities of up to 45% $\dot{V}O_2$max were more effective in removing lactate from the blood than intensities of 62 and 81% $\dot{V}O_2$max. Davies et al. (1970) found that at exercise intensities beyond 40% $\dot{V}O_2$max blood lactate removal was diminished. Hermansen and Stensvold (1972) found that recovery intensities of up to 60 to 70% $\dot{V}O_2$max enhanced blood lactate removal, but higher intensities decreased it.

There are two common explanations for the reduced blood lactate removal apparent at high recovery exercise intensities. One explanation is that as exercise intensities increase, blood flow is redirected away from tissues that remove lactate (Walsh & Banister, 1988). Musch, Haidet, Freidman, Pitetti, & Stray-Gundersen (1984) found that blood flow to the liver and inactive skeletal muscle was reduced during exercise. A second explanation is that as recovery exercise intensities increase, the recruitment of Type II skeletal muscle and the firing rate of these fibers will also increase; as a result, lactate production will increase (Nagata, Muro, Moritani, & Yoshida, 1981). Since the objective of recovery is to reduce the concentration of blood lactate, any increase in the rate of lactate production is counterproductive. Therefore, an exercise intensity that results in a decreased blood flow to tissues which uptake lactate, and/or an increased lactate production, will cause the rate of blood lactate removal to decrease.

Active recovery will enhance blood lactate removal. However, if the intensity of the recovery exercise is too high blood lactate removal will be diminished. Thus, there appears to be an optimal intensity of recovery exercise

which potentiates blood lactate removal. It is hypothesized that the optimal recovery exercise intensity for blood lactate removal is the lactate threshold intensity. This intensity appears to represent the point at which blood lactate elimination mechanisms have been potentiated and blood lactate production has not yet exceeded this elimination potential. A number of studies have attempted to identify the optimal recovery exercise intensity for blood lactate removal. However, because the results and the methodologies of these studies have been inconsistent, the optimal intensity of recovery exercise is still unknown.

Most of the studies that have attempted to identify the optimal exercise intensity for blood lactate removal have examined recovery exercise intensities relative to $\dot{V}O_2max$. Optimal recovery intensities of 40, 45, and 60 to 70% $\dot{V}O_2max$ were identified by Davies et al. (1970), Belcastro and Bonen (1975), and Hermansen and Stensvold (1972), respectively. Weltman et al. (1979) compared two exercise intensities: 40 and 65% $\dot{V}O_2max$, which they identified as below and above the lactate threshold, respectively. They found that recovery at 40% $\dot{V}O_2max$ resulted in a more effective blood lactate removal than recovery at 65% $\dot{V}O_2max$. In a similar approach, Stamford et al. (1981) compared recovery intensities of 40 and 70% $\dot{V}O_2max$, which they identified as below and above the lactate threshold, respectively. They found that blood lactate removal was not significantly different between these two recovery intensities, if experimentally determined baseline blood lactate concentrations were used. While the results from these studies are not entirely conflicting, they definitely lack consistency. These results must be interpreted with care, since significant conceptual and methodological inadequacies exist in most of the studies that produced them.

The most obvious inadequacy is that researchers have examined blood lactate removal at recovery exercise intensities defined as a percentage of VO_2max. Blood lactate removal is not a function of a percentage of VO_2max, it is primarily a function of the degree of imbalance between the rates of lactate production and of blood lactate elimination. The point of balance between these two rates is the lactate threshold exercise intensity. It is well known that this intensity occurs at different percentages of VO_2max for different individuals and even for the same individual at different trained states (Coyle, Coggan, Hopper, & Walters, 1988). An intensity of exercise defined as a percentage of VO_2max could represent an intensity that is below lactate threshold for some individuals and above threshold for others (see Figure 7) (Brooks, 1985). By defining recovery exercise intensities relative to VO_2max, researchers have been comparing very different rates of lactate production and elimination for each of their subjects, at each recovery intensity. As a result, high interindividual variations in blood lactate removal have been noted (Belcastro & Bonen, 1975). This variation is reduced when recovery intensities are expressed relative to the lactate threshold (McLellan and Skinner, 1982). Clearly, the use of a percentage of VO_2max to standardize recovery exercise intensity is inappropriate when the variable under investigation is blood lactate. In such situations, recovery exercise intensities should be standardized by defining them relative to the lactate threshold exercise intensity.

There has also been a lack of consistency in the method used to determine the rate of blood lactate removal. This rate of removal is usually the criterion used to evaluate the effectiveness of the different recovery exercise intensities; therefore, the method used to determine this rate is an important consideration. Belcastro and Bonen (1975) described the rate of blood lactate removal as a

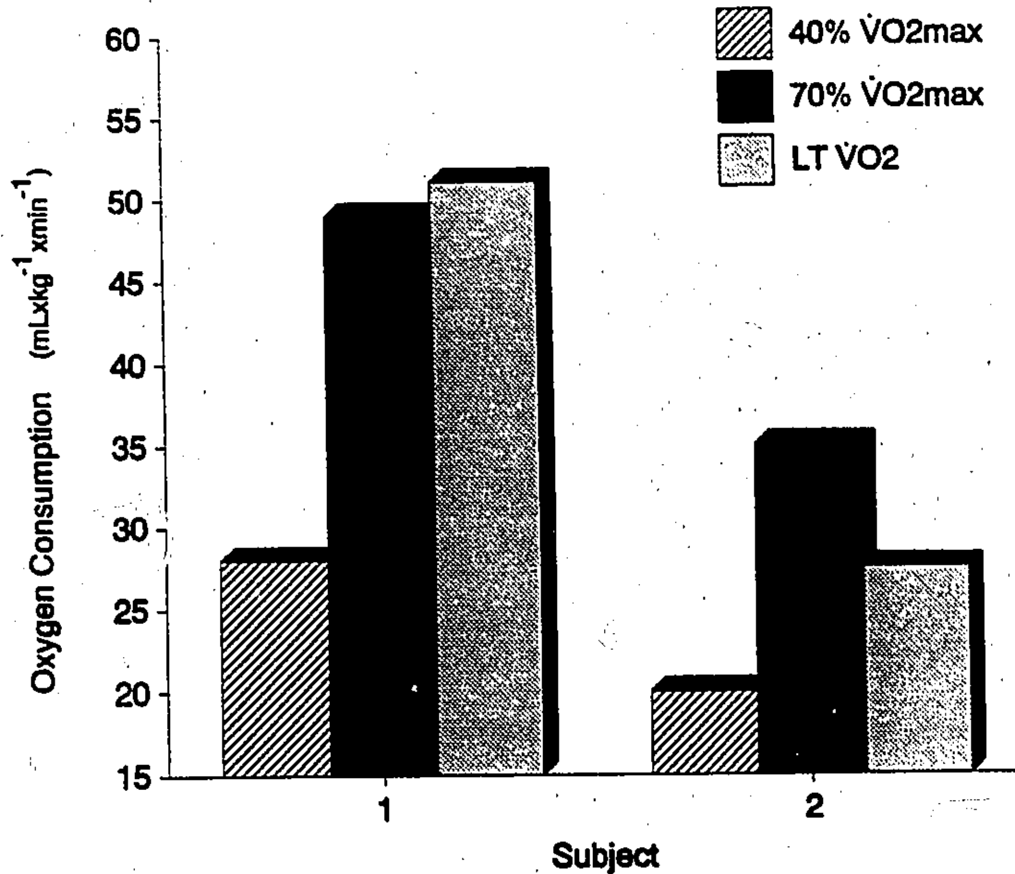
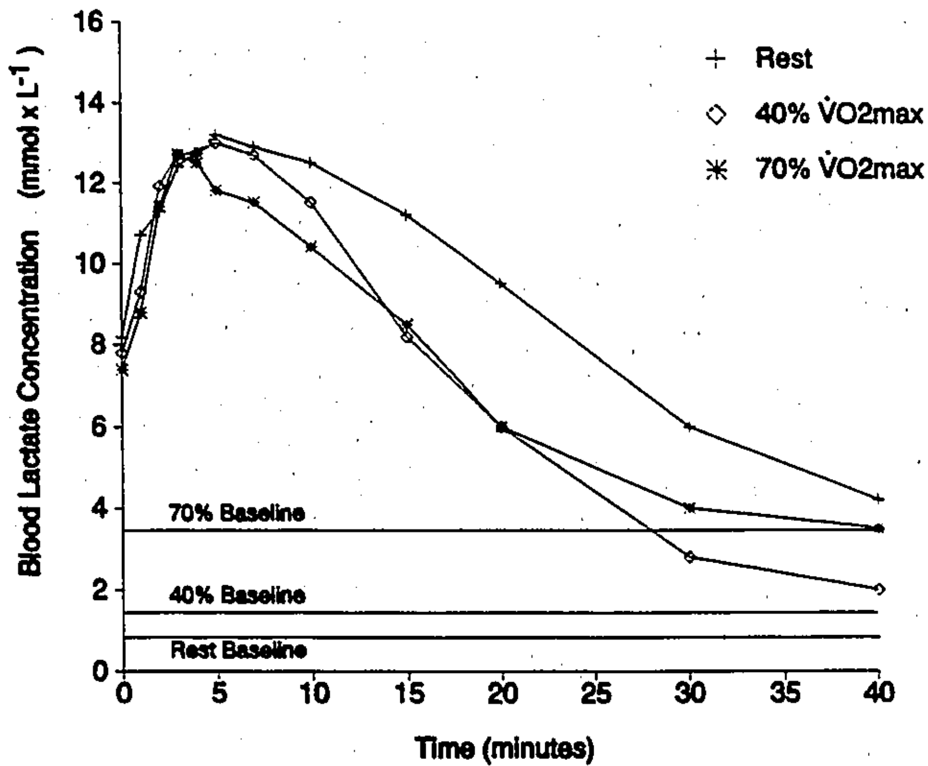


Figure 7. Hypothetical research design with recovery intensities defined as 40 and 70% $\dot{V}O_2max$ for 2 subjects. Subject 1 demonstrates lactate threshold at 73% $\dot{V}O_2max$, which is above the highest recovery exercise intensity. Subject 2 demonstrates lactate threshold at 55% $\dot{V}O_2max$, which is between the two recovery exercise intensities. The recovery exercise intensities expressed as a percentage $\dot{V}O_2max$ clearly represent very different relative rates of lactate production and elimination for each subject.

linear decay, and expressed the decay as a percent change in lactate concentration per minute from the lactate concentration at time zero of the recovery period. However, the recovery blood lactate response is not always linear (McLellan & Skinner, 1982; Stamford et al., 1981). Blood lactate concentrations characteristically increase during the first few minutes of recovery, after which they decrease, and continue to decrease throughout the recovery period towards a particular asymptote of blood lactate concentration (see Figure 8) (Hermansen & Stensvold, 1972; Stamford et al., 1981; Weltman et al., 1979). Therefore, the linear decay method of describing blood lactate removal rates is often inappropriate.

Davies et al. (1970) recognized the non-linear response of blood lactate disappearance with time. These researchers plotted the log of blood lactate concentration against time, for the first ten minutes of recovery, and used the slope of the resulting line as the blood lactate removal rate. The calculation of times for half removal of blood lactate was the next stage of refinement made to blood lactate removal calculations. In the study by Stamford et al. (1981), individual single-component exponential curves of blood lactate disappearance, which started from peak blood lactate concentrations, were used to describe blood lactate removal during recovery. From the parameters of these curves, blood lactate removal half-times were determined for each exercise intensity. McLellan and Skinner (1982) similarly calculated half-times from individual blood lactate removal curves to compare removal rates between exercise intensities.

The calculation of blood lactate removal half-times from single component exponential curves is a method which accounts for the non-linearity of blood lactate removal. The half-times for blood lactate removal, however, will vary



<u>Figure 8.</u> Blood lactate concentrations following maximal work for three recovery intensities: rest, 40% V̇O_2max, and 70% V̇O_2max. Experimentally determined baseline blood lactate concentrations for each recovery intensity are included.

NOTE: From "Exercise recovery above and below anaerobic threshold following maximal work" by B. A. Stamford, A. Weltman, R. Moffat, and S. Sady, 1981, <u>Journal of Applied Physiology: Respiratory Environmental Exercise Physiology</u>, p. 840-844.

considerably depending on the concentration of blood lactate used as the baseline value in the calculation (Stamford et al., 1981). In the study by McLellan and Skinner (1982) half-time values were calculated using a standard baseline blood lactate concentration of 1.0 $mmol \times L^{-1}$. The use of a standard baseline blood lactate concentration for different recovery exercise intensities is invalid. During recovery exercise, blood lactate concentrations will decrease towards an asymptote that is characteristic to the individual and to the exercise intensity used. This asymptote appears to be equivalent to the steady-state blood lactate concentration observed during constant work at each recovery exercise intensity (Stamford et al., 1981). As exercise intensities approach lactate threshold the concentration of blood lactate observed during steady-state work will progressively increase (Hermansen & Stensvold, 1972). Therefore, blood lactate removal for a recovery exercise performed at an intensity near the lactate threshold intensity cannot be evaluated using a standard 1.0 $mmol \times L^{-1}$ baseline because the blood lactate concentration asymptote for work at such an intensity is generally much higher than 1.0 $mmol \times L^{-1}$ (Stamford et al., 1981). If a standard baseline is used, the blood lactate decrease half-time will be overestimated for any recovery exercise intensity that generates a blood lactate concentration asymptote greater than the standard.

A study which used experimentally determined blood lactate baselines to evaluate blood lactate removal at different recovery exercise intensities was done by Stamford et al. (1981). These researchers measured blood lactate removal at two active recovery intensities - 40 and 70% VO_2max. Before the recovery exercises were investigated, baseline blood lactate concentrations were determined for each recovery exercise intensity. Steady-state work at 40%

$\dot{V}O_2max$ resulted in blood lactate concentrations that averaged 1.4 mmol$\times L^{-1}$. Steady-state work at 70% $\dot{V}O_2max$ resulted in blood lactate concentrations that averaged 3.5 mmol$\times L^{-1}$. When blood lactate removal rates were evaluated for these two recovery exercise intensities, recovery at 40% of $\dot{V}O_2max$ produced a significantly faster half-time if a resting blood lactate baseline (0.9 mmol$\times L^{-1}$) was used. However, if the experimentally determined baselines were used (1.4 mmol$\times L^{-1}$ and 3.5 mmol$\times L^{-1}$) there were no significant differences in half-time between the two recovery exercise intensities. That is, recovery at 70% $\dot{V}O_2max$ was just as effective in reducing blood lactate as recovery at 40% $\dot{V}O_2max$.

The results from the study by Stamford et al. (1981) are significant even though there were some methodological problems in the study. Stamford et al. (1981) used exercise intensities relative to $\dot{V}O_2max$ and not relative to the lactate threshold. The problems associated with the use of recovery intensities expressed as a percentage of $\dot{V}O_2max$ have been discussed earlier. These researchers were also able to measure baseline blood lactate concentrations at an exercise intensity which they claimed to be above lactate threshold (70% $\dot{V}O_2max$). If their subjects were truly above lactate threshold at the 70% $\dot{V}O_2max$ intensity, then they should not have been able to measure baseline blood lactate concentrations, since exercise intensities above lactate threshold cause blood lactate concentrations to progressively increase. Despite these two failings, Stamford et al. (1981) were successful in demonstrating a very important aspect of blood lactate decrease during active recovery. They demonstrated that the rate of decrease is dependent upon the baseline blood lactate concentration observed at a particular intensity of recovery exercise. They also demonstrated that results are misinterpreted if the correct baseline concentration is not used when rates of blood lactate decrease are calculated.

That is, the results from Stamford et al. (1981) would have been similar to the results of Davies et al. (1970) and McLellan and Skinner (1982) if experimentally determined baseline blood lactate concentrations had not been used to calculate the rate of blood lactate decrease.

In conclusion, many studies have investigated the response of blood lactate concentration to various intensities of recovery exercise. In applying the results of these studies, a number of factors must be considered. First, most of these studies used recovery exercise intensities that were defined relative to $\dot{V}O_2max$, and not relative to the lactate threshold. As a result, these studies have compared the responses of their subjects to very different exercise intensities, in terms of the rates of lactate production and of blood lactate elimination. A second critical factor is the method used to calculate the rate of blood lactate removal. Since individual blood lactate removal rates are often non-linear, they must be calculated accordingly. A key consideration in the calculation of times for half decrease in blood lactate is the blood lactate concentration used as a baseline. Removal half-times should be calculated using experimentally determined baselines for each recovery exercise intensity. The last consideration is the scenario to which the experimental results are being applied. The effectiveness of a particular recovery exercise intensity will depend on whether it occurs within an athletic event or between events. None of the previous studies on blood lactate removal during recovery exercise have adequately accounted for all of these factors. Therefore, an experimental design was needed where individual lactate threshold exercise intensities would be accurately determined; where recovery exercise intensities would be defined relative to the lactate threshold; and where decreases in blood lactate concentration would be assessed relative to pre-determined baselines of blood lactate concentration for each recovery exercise intensity.

METHODOLOGY

This chapter describes the experimental design that was used to investigate the effect of three different recovery exercise intensities upon the rate of decrease in blood lactate concentration following a standard activity of suprathreshold intensity. A detailed description of all pertinent aspects of the experimental design follows.

Subjects

Seven male endurance trained cyclists were used as subjects in this experimentation. The general subject characteristics are presented in Table 1.

Table 1

General Subject Characteristics

Subject	Age (years)	Height (cm)	Weight (kg)	$\dot{V}O_2max$ ($L \times min^{-1}$)	Cadence (rpm)
1	32.0	183.4	68.6	5.00	114
2	34.0	183.7	72.7	4.14	90
3	41.2	176.5	75.7	4.35	100
4	30.6	184.1	75.6	5.03	105
5	31.9	169.2	66.4	4.37	104
6	33.6	171.8	67.9	4.02	100
7	27.1	183.7	80.0	5.27	114
$\bar{X}$	32.9	178.9	72.4	4.60	104
σ	4.3	6.4	5.0	0.49	8

Subject Orientation

Prior to the actual testing, the group of potential subjects met with the researcher. The exercise protocols were outlined to the subjects at this time. The subjects were also introduced to the equipment that they would be interfacing with during the experimentation.

The subjects were shown the bicycle that they would be riding during the testing. Measurements, such as seat height and handle bar displacement, were made to ensure that the cyclists were compatible with the bicycle. At the end of the orientation session, when the potential subjects had a full understanding of the demands and requirements of the experimentation, they were asked to sign informed consent forms. The consent forms that were used have been included in Appendix A of this document.

Procedure

The research design for the experimentation required that each subject perform seven cycling tests. The first of the tests was a combined lactate threshold $\dot{V}O_2max$ test (LT-$\dot{V}O_2max$ test). The LT-$\dot{V}O_2max$ test served three important functions: (a) it established the $\dot{V}O_2max$ of the subject, (b) it identified the approximate $\dot{V}O_2$ at the lactate threshold exercise intensity, and (c) it established the relationship between $\dot{V}O_2$ and cycling resistance, in kiloponds (kp), for each subject.

Following the combined LT-$\dot{V}O_2max$ test, three cycling tests which involved constant rate work; that is, cycling at a constant resistance and a constant cadence, were performed by each subject. The time lapse between the LT-

$\dot{V}O_2max$ test and the first constant work rate test was no less than 24 hours and no greater than 72 hours. The three intensities of constant rate work were: (a) subthreshold (lactate threshold $\dot{V}O_2$ minus 7% $\dot{V}O_2max$ (LT-7%)), (b) threshold (lactate threshold $\dot{V}O_2$ (@LT)), and suprathreshold (lactate threshold $\dot{V}O_2$ plus 7% $\dot{V}O_2max$ (LT+7%)). These constant work rate tests served two important functions; namely, they finely tuned the lactate threshold $\dot{V}O_2$ prediction, and they established individual baselines of blood lactate concentration for the lactate threshold and subthreshold work rates. The time span between each constant work rate test was no less than 24 hours and no greater than 72 hours.

The combined LT-$\dot{V}O_2max$ test and the constant work rate tests provided the information necessary to proceed to the three final tests - the surge-recovery tests. These surge-recovery tests involved a standardized suprathreshold intensity ($\dot{V}O_2max$) period of work, immediately followed by a recovery period at a constant rate of work. Three different intensities of recovery activity were used, thus, the three tests. The intensities of recovery that were used were the LT-7%, @LT, and LT+7% intensities that were established in the preceding constant work rate tests. The time lapse between the last constant work rate test and the first surge-recovery test was no less than 24 hours and no greater than 72 hours. The time span between each surge-recovery test was no less than 24 hours and no greater than 72 hours.

The dietary and training habits of the athletes may have had an affect on lactate production and/or utilization during the cycling tests (Ivy, Costill, Van Handel, Essig, & Lower, 1981; Jacobs, 1986). Even so, strict diet and training guidelines were not imposed on the subjects. However, in order to minimize

within subject differences between test sessions, the subjects were instructed to eat and train consistently throughout the test period.

The physiological parameters which were measured throughout the testing included $\dot{V}O_2$, blood lactate concentration, and heart rate. A description of the methods that were used to make these measurements is provided, as are detailed descriptions of the protocols used in the LT-$\dot{V}O_2max$, constant work rate, and surge-recovery tests.

Measurement of Oxygen Consumption

The open circuit method was used to determine $\dot{V}O_2$. Throughout each exercise test the subject breathed through a two-way, non-rebreathing, T-shaped valve. Inspiratory volumes were measured using a Morgan Ventilometer Mark 2. Expiratory gases travelled through a length of hose into a mixing box. From the mixing box a small sample of the gas (0.6 L) was passed through a tube containing calcium sulfate. This sample was then analyzed for oxygen and carbon dioxide content with the Amtek S-3A Oxygen Analyzer and the Amtek CD-3A Carbon Dioxide Analyzer, respectively. The output from these three devices was integrated with the Pulmonary Exercise System Software produced by the S & M Instrument and Physiodyne Instrument Companies. In this manner, $\dot{V}O_2$ measurements were made every 30 seconds of each cycling test.

The ventilometer, oxygen analyzer, and carbon dioxide analyzer were calibrated prior to and every 2 hours within each period of testing. The ventilometer was calibrated using a 4L syringe. The oxygen and carbon dioxide analyzers were calibrated using a sample of gas with known concentrations of O_2 and CO_2.

Measurement of Blood Lactate Concentration

At specific times during each cycling test approximately 30 μL of blood were drawn from a fingertip into a heparinized capillary tube. Prior to puncturing the fingertip with a lancet, the skin surface was cleansed with an alcohol swab. Once the blood had been collected, the skin perforation was again cleansed with an alcohol swab, and then covered with a tissue. From the blood sample collected in the capillary tube, 20 μL was immediately transferred, using a pipet, into a vial containing 380 μL of diluting solution. Within 6 hours of taking the blood samples, they were analyzed for blood lactate concentration. The blood lactate concentration analyses were performed by pipeting 100 μL of this mixture into the Kontron Medical Lactate Analyzer 640.

The Kontron Medical Lactate Analyzer determines the concentration of lactate in a sample by introducing an enzyme, cytochrome b_2, to it. This enzyme catalyses the oxidation of lactate to pyruvate. During the oxidation an electrochemically active substance is produced in proportion to the pyruvate. With the production of this substance, a measurable current is developed. The increase in current is linearly related to the concentration of lactate in the sample; and thus, the concentration of lactate can be quantified.

The lactate analyzer was calibrated using standard lactate samples of 5 mmol$\times$L^{-1} and 10 mmol$\times$L^{-1}. The calibration was conducted prior to performing any lactate analyses, on any test day. The calibration of the analyzer was monitored by measuring the lactate concentration of the 5 mmol$\times$L^{-1} standard every 5 to 10 samples - it was recalibrated whenever necessary.

Heart Rate Measurement

Heart rates were monitored throughout each cycling test using the Sport Tester PE3000 Heart Rate Monitor. This heart rate monitor enabled heart rate sampling at 5 second intervals. The heart rates were available for immediate observation. In addition, they were stored in the memory of the heart rate monitor. Following the cycling test, the heart rate data that had been stored in the memory of the monitor was downloaded into an IBM personal computer, using the PE3000 software, in order to obtain a hardcopy of the data. It should be noted that while the heart rate data was collected at 5 second intervals, only the heart rate at each minute of each test was actually used in the result analyses.

Bicycle and Cycle Ergometer

All of the subjects performed each cycling test on the same bicycle. This bicycle was of the dropped handlebar variety, with a 53.3 cm frame. Adjustments could be made to the seat height and to the handle bar position. In this way, the size of the bicycle was modified to accommodate each cyclist. In addition, two crank/pedal systems were available to accommodate cyclists that used either the Look system or the standard system.

The bicycle was mounted on a trainer, which supported the frame under the crank arms. The trainer was bolted to a larger frame to which a standard Monarch cycle ergometer was also mounted. An elongated chain had been constructed which travelled around the front and back sprockets of the bicycle as well as the sprocket of the ergometer. The connection of the two cycles in this manner allowed the resistance of the bicycle to be controlled through the

resistance control of the ergometer. This set-up enabled the cycling resistance to be standardized. It also ensured that the resistance could be repeated between test sessions. This apparatus was constructed so that the cyclist could perform the tests in the standard cycling position.

The bicycle had a Cateye Solar Cyclocomputer (Model CC-2000) mounted on it. The Cateye provided immediate cadence information to the cyclists. This feature enabled the cyclist to accurately maintain a prescribed cadence. The same cadence was used in all of the cycling tests performed by a particular subject. This cadence was one that was comfortable for the cyclist over the range of resistances that were used, and it was within the range of 90 to 115 revolutions per minute (rpm) (refer to Table 1 on page 67). The prescribed cadence was determined by the cyclist and researcher during the first test session. Once this individual cadence had been chosen it was used consistently by the subject in all cycling tests.

Prior to each cycling test the bicycle was adjusted to the subject. The same adjustments were made for each cycling test. The Monarch cycle ergometer was calibrated at three different times during the experimentation. Adjustments to the cycling resistances, based on the results of the calibration, were never required. During each cycling test the resistance on the cycle ergometer was monitored every 15 seconds, and adjusted as necessary. This procedure was required because the tension on the flywheel had a tendency to decrease during the course of a test.

Combined LT-$\dot{V}O_2$max Test

The protocol used for the combined LT-VO_2max test is outlined in Table 2. Prior to each test, height, weight, resting blood lactate, and resting heart rate measurements were made. After the bicycle had been adjusted to the subject, the subject began cycling. At this time the subject and researcher decided upon the cadence that was to be used by this subject throughout all of the cycling tests. The subject was instructed to monitor his own cycling cadence throughout the test; however, intermittent cadence checks were frequently made by the researcher to ensure that the prescribed cadenced was being maintained.

Table 2

Protocol for the LT-$\dot{V}O_2$max Test

Time (min)	Stage	Resistance (kp)	Timing of Blood Lactate Measurements (min)
0 to 5	warm-up	1.00	4:45 to 5:00
5 to 8	1	1.50	7:45 to 8:00
8 to 11	2	2.00	10:45 to 11:00
11 to 14	3	2.25	13:45 to 14:00
14 to 17	4	2.50	16:45 to 17:00
17 to 20	5	2.75	19:45 to 20:00
20 to 23	6	3.00	22:45 to 23:00
[a]			

[a] Continue with these increments and measurements until test termination.

During the test VO_2 was determined every 30 seconds. Fingertip blood samples were taken during the last 15 seconds of each stage. Heart rate measurements were sampled at 5 second intervals throughout the test. The resistance of the cycle ergometer was closely monitored to ensure that it remained at the appropriate level throughout each stage. If the subject reached the point of exhaustion prior to the prescribed blood sampling time, a sample was collected immediately after test termination.

Subjects were instructed to remain as relaxed as possible during the first few stages of the test so that blood lactate concentrations would not be exaggerated during this phase due to the stress of the impending maximal activity. However, once the subject appeared to have surpassed lactate threshold (apparent through changes in breathing rate, heart rate, and level of fatigue), verbal encouragement was provided to heighten the motivational level of the subject, so that a maximal VO_2max would be attained.

The test was terminated when any of the following events occurred:

1. The subject was unable to continue activity.
2. The subject was unable to maintain the required cadence after repeated efforts to do so.
3. The subject showed visible signs of extreme fatigue, such as a loss of co-ordination, facial pallor, etc..

Following completion of the LT-VO_2max test, the resistance on the cycle ergometer was decreased to 1 kp, and the subject was encouraged to continue cycling for 5 minutes. At 5 minutes the final blood sample was taken. VO_2 and heart rate measurements were not made during this recovery period.

Criterion for $\dot{V}O_2max$.

The $\dot{V}O_2$ data from the test were immediately analyzed to determine whether or not a true $\dot{V}O_2max$ had been attained. $\dot{V}O_2max$ was attained if any of the following criteria were demonstrated.

1. A levelling or decrease in $\dot{V}O_2$ with increasing workload (change in $\dot{V}O_2$ of less than or equal to 2 $mL \times kg^{-1} \times min^{-1}$).
2. A respiratory quotient of greater than or equal to 1.0.
3. A heart rate in excess of 200 beats per minute.

If none of the $\dot{V}O_2max$ criteria had been exhibited, a $\dot{V}O_2max$ verification test would have been conducted. Each subject demonstrated a clear $\dot{V}O_2max$ during the initial test (according to the first criterion); therefore, no verification tests were performed.

Lactate Threshold Identification.

The lactate threshold was identified subjectively through a visual detection method. A graph of blood lactate concentration ($mmol \times L^{-1}$) versus $\dot{V}O_2$ ($L \times min^{-1}$) was used to detect the lactate threshold exercise intensity for each subject. The lactate threshold exercise intensity was identified as that intensity ($\dot{V}O_2$ ($L \times min^{-1}$)) just before blood lactate concentrations exhibited a marked rise (greater than or equal to 1 $mmol \times L^{-1}$), and exhibited a continued increase in blood lactate concentration with subsequent increases in workload. In this manner, the lactate threshold was predicted from the combined LT-$\dot{V}O_2max$ test. This intensity was verified with the three separate constant work rate tests.

Constant Work Rate Tests

The three constant work rate tests involved cycling at three intensities: subthreshold (LT-7%), lactate threshold (@LT), and suprathreshold (LT+7%). These intensities were calculated from the results of the combined test; therefore, they were specific to each individual. Only one constant work rate test was performed on any day by a subject.

In order to determine the work rates that were to be used by the subjects in the constant work rate tests, a linear regression analysis was performed on the individual $\dot{V}O_2$ (L×min^{-1}) and cycling resistance data from the combined LT-$\dot{V}O_2max$ test. Utilizing the data from all stages of the combined test, except the warm-up stage, the final complete stage, and the final incomplete stage (if there was one), an equation was determined which described the relationship between $\dot{V}O_2$ and cycling resistance. With this equation it was possible to predict the cycling resistance required to elicit a specific $\dot{V}O_2$. For instance, if a subject obtained a $\dot{V}O_2max$ of 4.26 L×min^{-1}, and his lactate threshold exercise intensity was predicted to be at 3.43 L×min^{-1}, then the subthreshold exercise intensity was equal to (3.43-(7%(4.26))), or 3.13 L×min^{-1}; the lactate threshold exercise intensity was equal to 3.43 L×min^{-1}; and the suprathreshold exercise intensity was equal to (3.43+(7%(4.26))), or 3.73 L×min^{-1}. The following equation was derived from the linear regression of the $\dot{V}O_2$ and cycling resistance (kp) data for this subject: $\dot{V}O_2 = 0.44 + (1.06 \times kp)$. Therefore, by inserting the desired $\dot{V}O_2$ values for each of the work rates, the cycling resistance required to obtain these $\dot{V}O_2$ were obtained. In this example, the following cycling resistances were obtained: 2.5 kp, 2.8 kp, and 3.1 kp, for the LT-7%, @LT, and LT+7% intensities, respectively. Since the cycling cadence was a

constant for each cyclist, only the cycling resistance had to be modified to change the work rate.

The protocol for the constant work rate tests is outlined in Table 3. Prior to each test, weight, resting blood lactate, and resting heart rate measurements were made. After the bicycle had been adjusted to the subject, he began warming up for the test. As in the combined test, the subject was instructed to monitor his own cycling cadence throughout the test; however, intermittent cadence checks were frequently made by the researcher to ensure that the prescribed cadenced was being maintained.

Table 3

Protocol for the Constant Work Rate Test

Time (min)	Stage	Resistance (kp)	Timing of Blood Lactate Measurements (min)
0 to 5	warm-up	1.00	----
5 to 8	warm-up	1.50	----
8 to 28	constant work	[a]	11:45 to 12:00 15:45 to 16:00 19:45 to 20:00 23:45 to 24:00 27:45 to 28:00
28 to 33	recovery	1.00	----

[a] The actual resistance used was specific to each cyclist.

During the test VO_2 was measured every 30 seconds. Heart rates were measured every 5 seconds. Blood lactate samples were taken in the last 15 seconds of the warm-up, and thereafter, every 4 minutes of the constant work rate portion of the test. The resistance of the cycle ergometer was closely monitored to ensure that it remained at the appropriate level throughout each test. Following the constant work rate period, a recovery period of 5 minutes, at approximately 1.0 kp, was completed by each subject. No physiological measurements were made either during or after the recovery period.

Verification of the Lactate Threshold Exercise Intensity.

The first constant work rate test involved cycling at the intensity corresponding to the predicted individual lactate threshold intensity (@LT). If constant work at the @LT intensity resulted in stable blood lactate concentrations (standard deviation (σ) of less than 0.6 mmol$\times$L^{-1}), then it was assumed that the lactate threshold estimation was either correct or it was underestimated; the next constant work rate test was performed at the LT+7% intensity. If the blood lactate concentrations during the LT+7% constant work rate test were unstable (σ greater than or equal to 0.6 mmol$\times$L^{-1}), then it was assumed that the lactate threshold intensity had been estimated accurately; the final test was then performed at the LT-7% intensity. If, however, the blood lactate concentrations were still stable during the second constant work rate test (LT+7%), then it was assumed that the lactate threshold had been underestimated; a further constant work rate test at LT+14%VO_2max was then conducted (the LT+7% intensity became the new @LT intensity). The new intensities for sub-threshold, at threshold, and suprathreshold were therefore verified. On the other hand, if the blood lactate concentrations during the first constant work rate test (@LT) were unstable, then it was assumed that the

lactate threshold prediction had been overestimated. The @LT intensity became the LT+7% intensity, and the two constant work rate tests performed subsequently were at the LT-7% and the LT-14% intensities (the LT-7% intensity became the new @LT intensity).

In summary, in order to determine the actual lactate threshold intensity, constant work rate tests were performed until the following conditions were satisfied:

1. A subthreshold intensity was identified which elicited stable blood lactate concentrations.
2. A lactate threshold intensity was identified, at a workload 7% $\dot{V}O_2max$ higher than the subthreshold intensity, which also elicited stable blood lactate concentrations.
3. A suprathreshold intensity was identified, at a workload that was 7% $\dot{V}O_2max$ higher than the lactate threshold intensity, which elicited a progressive increase in blood lactate concentrations.

For all subjects, these intensities were identified during the first three constant work rate tests performed.

<u>**Baseline Blood Lactate Concentrations.**</u>

The baseline blood lactate concentrations were calculated from the lactate samples taken during the 20 minute period of constant work. The baseline concentration was equal to the mean of the five blood lactate concentration measures; that is, those sampled from minutes 4 to 20 of the 20 minute constant work period. These baseline blood lactate concentrations were specific to the intensity of the constant work and to each subject. The required blood lactate response to the suprathreshold work rate was a progressively increasing

blood lactate concentration. For this reason, a baseline blood lactate calculation could not be validly made for the suprathreshold intensity. The baseline blood lactate concentration determined for the lactate threshold intensity was used as the baseline for the suprathreshold intensity. This was the most appropriate adjustment, since the aim of the recovery period was to decrease the blood lactate concentration to at least that exhibited at the lactate threshold intensity.

Surge-Recovery Tests

The three surge-recovery tests involved cycling for 2 minutes at the final intensity achieved during the LT-$\dot{V}O_2max$ test. This suprathreshold work was followed by recovery at the three different intensities identified in the constant work rate tests as: LT-7%, @LT, and LT+7%. Only one surge-recovery test was performed on any day by a subject. The order in which a subject was exposed to the three recovery intensities was randomized.

The protocol for the surge-recovery tests is outlined in Table 4. Prior to each test, weight, resting blood lactate, and resting heart rate measurements were made. After the bicycle had been adjusted to the subject, he began warming up for the test. As in the other tests the subject was instructed to monitor his own cycling cadence; however, intermittent cadence checks were frequently made by the researcher to ensure that the prescribed cadence was being maintained.

During the test $\dot{V}O_2$ was measured every 30 seconds. Heart rates were measured every 5 seconds. Blood lactate samples were taken following the warm-up, the pre-surge period, the surge, every minute of the first 5 minutes of the recovery, and every 3 minutes thereafter, until the end of the recovery

Table 4

Protocol for the Surge-Recovery Test

Time (min)	Stage	Resistance (kp)	Timing of Blood Lactate Measurements (min)
0 to 5	warm-up	1.00	4:45 to 5:00
5 to 10	warm-up	@LT[a]	9:45 to 10:00
10 to 12	surge	MAX[a]	----
12 to 32	recovery	[a]	12:00 to 12:15 13:00 to 13:15 14:00 to 14:15 15:00 to 15:15 16:00 to 16:15 17:00 to 17:15 20:00 to 20:15 23:00 to 23:15 26:00 to 26:15 29:00 to 29:15 32:00 to 32:15
32 to 37	final recovery	1.00	----

[a] The actual resistance used was specific to each cyclist.

period (see Table 4). Several samples were taken during the first 5 minutes of recovery to ensure that the peak blood lactate concentration was accurately determined. The resistance on the cycle ergometer was closely monitored to ensure that it remained at the appropriate level throughout each test. No physiological measurements were made either during or after the final recovery period.

Analysis of Results and Statistical Design

Data analyses from the constant work rate test results were conducted to determine whether or not the three intensities of constant work demonstrated significantly different physiological responses. This was established through separate comparisons of the blood lactate concentrations, the $\dot{V}O_2$, and the heart rates for each of the constant work rate intensities. Therefore, three separate three-way analyses of variance (ANOVA) with repeated measures on two factors were performed. The three independent variables were: (a) the three constant work rate intensities, (b) the 7 subjects, and (c) the measurement sampling times within each constant work rate period. The repeated measures were the recovery intensities, and the measurement sampling times.

The first stage in the analysis of the data from the surge-recovery tests was to determine whether or not the standardized pre-recovery period of each surge-recovery test imposed similar levels of physical stress for each subject. This was established through a comparison of the pre-recovery blood lactate concentrations, $\dot{V}O_2$, and heart rates. Therefore, three separate three-way ANOVA with repeated measures on two factors were performed. The three independent variables were: (a) the three recovery intensities, (b) the 7 subjects, and (c) the measurement sampling times. The repeated measures were the recovery intensities and the sampling times.

A three-way factorial ANOVA with repeated measures was also used to determine whether or not there were significant differences in blood lactate concentrations between the three recovery intensities. The independent variables were: (a) the eleven times of blood lactate sampling during the

recovery period, (b) the three recovery intensities, and (c) the subjects. The repeated measures were the recovery intensities and the sampling times. Similar analyses were also performed for the VO_2 data and the heart rate data, in order to determine whether or not there were any significant differences in these physiological parameters due to the different recovery intensities.

The final analysis involved calculating the time for half decrease of blood lactate for each of the three recovery conditions. In order to determine the half-times, an equation was derived for the blood lactate concentration versus time curve developed for each subject and each recovery intensity. This equation was derived using the Slidewrite Plus (1987) Graphics Package. This software derives approximating formulas for either linear, exponential, logarithmic, power, or polynomial functions. In all cases, the polynomial function, to the fourth or fifth order resulted in the best approximation of the blood lactate recovery curve (refer to Appendix C). The polynomial equations were approximated using a least squares polynomial regression method ($y=a_0+a_1x+a_2x^2+...+a_nx^n$). In order to calculate the half-times for a subject at a specific recovery intensity, the half blood lactate concentration had to be calculated. The half blood lactate concentration was equal to the average of the peak blood lactate concentration demonstrated in the recovery period and the baseline blood lactate concentration for that recovery intensity. This half blood lactate concentration was entered into the specific polynomial function, and this function was solved for time, using Newton's method (Gerald & Wheatley, 1985). The individually determined half-time for each recovery intensity was analyzed for significant differences using a 3X7 ANOVA with repeated measures. The two independent variables were: (a) the three recovery intensities, and (b) the 7 subjects. The repeated measure was the recovery intensity. The dependent variable was the half-time.

Statistical analyses were performed using the SAS Statistical Software Package (release 5.18) on the mainframe computer system at the University of Ottawa. All post hoc multiple comparison analyses were performed using Tukey's method. The level of significance for all analyses was .01.

RESULTS

In general, the experimentation provided very consistent results across all of the cycling tests. That is, the results of the analyses performed for the constant work rate tests clearly established that each of the three intensities represented significantly different rates of work. Similarly, the analyses performed for the surge-recovery tests revealed that each recovery intensity resulted in a physiological response that was significantly different from that of the other two recovery intensities. However, when the blood lactate recovery responses were assessed with consideration of the appropriate blood lactate concentration baseline, it was evident that none of the intensities resulted in significantly different rates of decrease in blood lactate. A detailed presentation of the analyses that were performed follows.

Combined LT-$\dot{V}O_2max$ Test

The first cycling test that each subject performed was the combined LT-$\dot{V}O_2max$ test. The results from the combined LT-$\dot{V}O_2max$ test are presented in Table 5. The mean absolute $\dot{V}O_2max$ was 4.60 $L \times min^{-1}$. The individual lactate threshold intensity was predicted from the blood lactate concentration measurements made during the test (refer to Appendix B). This prediction was verified with the constant work rate tests, and adjusted when necessary. The most accurate individual lactate threshold intensity appears in Table 5. The mean absolute $\dot{V}O_2$ at lactate threshold was 3.62 $L \times min^{-1}$; this value corresponded to 79% of the mean $\dot{V}O_2max$.

Table 5

Individual and Group Results from the LT-$\dot{V}O_2max$ Test

Subject	$\dot{V}O_2max$ ($L \times min^{-1}$)	$\dot{V}O_2max$ ($mL \times kg^{-1} \times min^{-1}$)	$\dot{V}O_2$ @ LT ($L \times min^{-1}$)	LT % of $\dot{V}O_2max$
1	5.00	72.9	4.01	79.7
2	4.14	56.9	3.29	79.3
3	4.35	57.5	3.39	76.5
4	5.03	66.5	3.74	74.0
5	4.37	65.8	3.71	84.2
6	4.02	59.2	3.32	81.9
7	5.27	65.9	4.07	77.1
$\overline{X}$	4.60	63.5	3.65	79.0
σ	0.49	5.9	0.33	3.4

Constant Work Rate Tests

An equation for the linear relationship between VO_2 and workload was determined for each subject from the results of the LT-VO_2max test. This relationship was used, in conjunction with the VO_2 at the predicted lactate threshold intensity and the VO_2max, to estimate the workloads corresponding to the LT-7%, @LT, and LT+7% intensities for each subject. Using this method, the individual lactate threshold intensity was predicted accurately for 5 subjects. It was overestimated for 2 subjects. Nevertheless, only three constant work rate tests had to be performed by each subject in order to accurately establish the three work intensities: LT-7%, @LT, and LT+7%.

Constant work at the LT-7% intensity resulted in a mean VO_2 that was 7.3% of the mean VO_2max below the mean lactate threshold VO_2. Constant work at

the LT+7% intensity resulted in a mean $\dot{V}O_2$ that was 7.2% of the mean $\dot{V}O_2max$ above the mean lactate threshold $\dot{V}O_2$. When the work rates were assessed individually, the LT-7% intensity resulted in $\dot{V}O_2$ values that ranged between 3% and 13% of $\dot{V}O_2max$ below the individual lactate threshold $\dot{V}O_2$. The LT+7% intensity resulted in $\dot{V}O_2$ that ranged between 3% and 10% of the $\dot{V}O_2max$ above the individual lactate threshold $\dot{V}O_2$.

The mean $\dot{V}O_2$, blood lactate concentration, and heart rate measurements demonstrated by each subject during each constant work rate test are presented in Table 6. The standard deviations for the blood lactate concentration measurements are also presented to demonstrate that the criterion deviation of greater than or equal to 0.6 $mmol \times L^{-1}$ was met by each subject in the suprathreshold (LT+7%) constant work rate test. Graphical representations of the group results for blood lactate concentration, $\dot{V}O_2$, and heart rate, in the constant work rate tests, are presented in Figure 9, Figure 10, and Figure 11, respectively.

In order to determine whether or not the three constant rides represented significantly different intensities of exercise, a separate three-way factorial ANOVA with repeated measures on two factors was performed for each dependent variable: blood lactate concentration, $\dot{V}O_2$, and heart rate. The three independent variables in the analyses were: (a) the intensity of constant work, either LT-7%, @LT, or LT+7%; (b) the measurement sampling time within each segment of constant work; and (c) the 7 subjects. The two repeated measures were the intensity and the measurement sampling time.

Table 6

Individual and Group Results from the Three Constant Work Rate Tests

	Workload (kp)	Mean $\dot{V}O_2$ (L×min^{-1})	Mean HR (bpm)	Mean BLC (mmol×L^{-1})	σ BLC (mmol×L^{-1})
SUBJECT 1					
LT-7%	2.0	3.33	154	2.24	0.26
@LT	2.3	4.01	161	3.42	0.33
LT+7%	2.6	4.39	174	8.23	1.42
SUBJECT 2					
LT-7%	2.4	3.12	150	1.39	0.16
@LT	2.6	3.29	163	3.82	0.39
LT+7%	2.8	3.72	167	5.82	0.70
SUBJECT 3					
LT-7%	1.9	2.93	132	1.25	0.09
@LT	2.2	3.39	149	2.85	0.26
LT+7%	2.5	3.60	160	4.74	0.64
SUBJECT 4					
LT-7%	2.3	3.61	160	1.44	0.11
@LT	2.5	3.74	169	2.82	0.16
LT+7%	2.7	4.25	180	5.30	1.04
SUBJECT 5					
LT-7%	2.3	3.40	177	1.50	0.16
@LT	2.5	3.71	184	2.80	0.17
LT+7%	2.7	3.91	186	4.45	0.80
SUBJECT 6					
LT-7%	2.2	3.01	150	1.48	0.03
@LT	2.5	3.32	175	4.14	0.39
LT+7%	2.8	3.70	179	4.78	0.69
SUBJECT 7					
LT-7%	2.1	3.77	148	1.27	0.20
@LT	2.3	4.07	157	2.26	0.21
LT+7%	2.5	4.25	168	4.51	0.71
GROUP					
LT-7%	2.2	3.31	153	1.51	
@LT	2.4	3.65	166	3.16	
LT+7%	2.7	3.98	173	5.41	

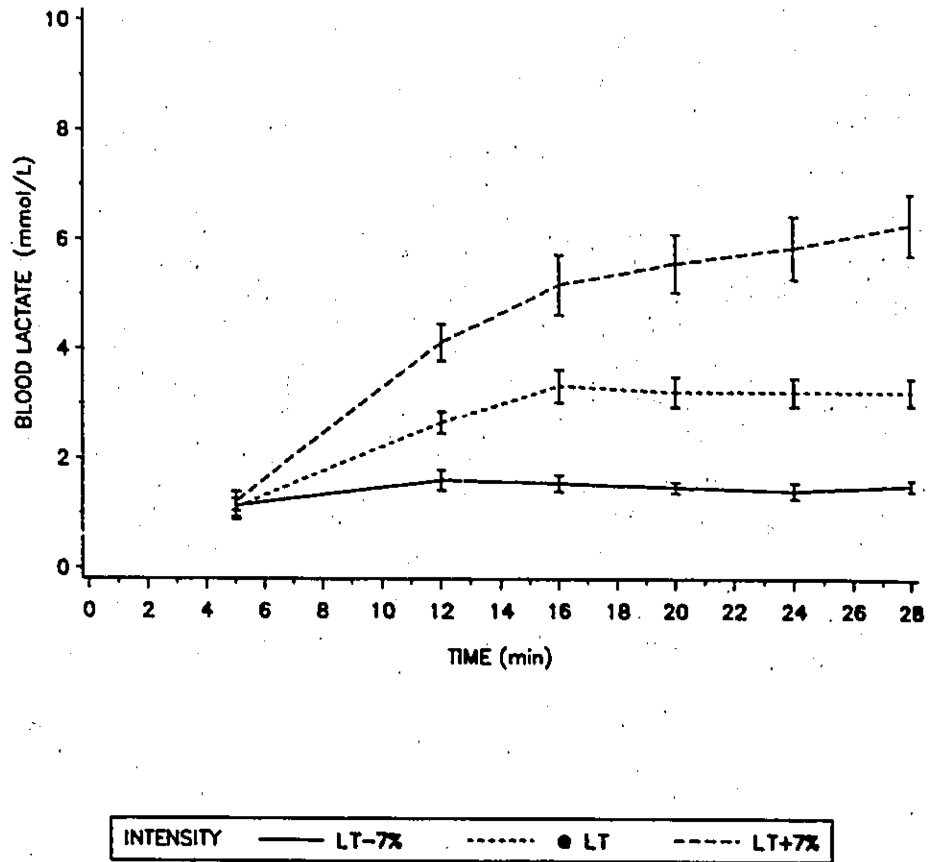


Figure 9. Blood lactate concentration (mean ± standard error) versus time for the three constant work rate tests. The constant work rate portion of the test occurred during minutes 8 through 28.

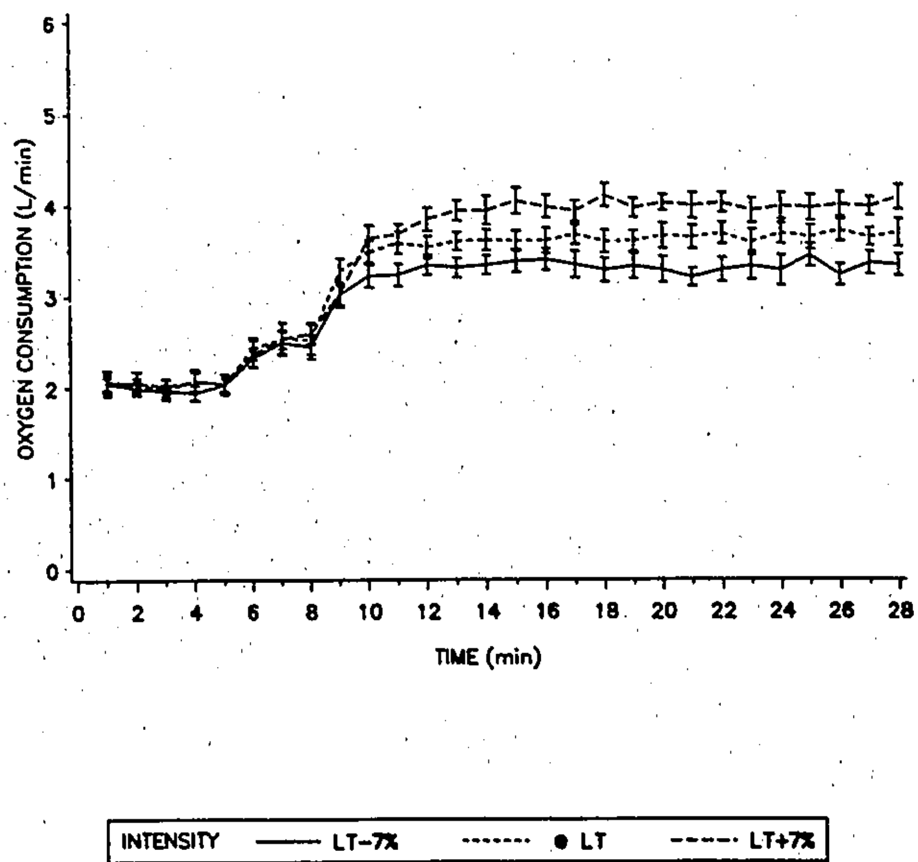


Figure 10. VO_2 (mean ± standard error) versus time for the three constant work rate tests.

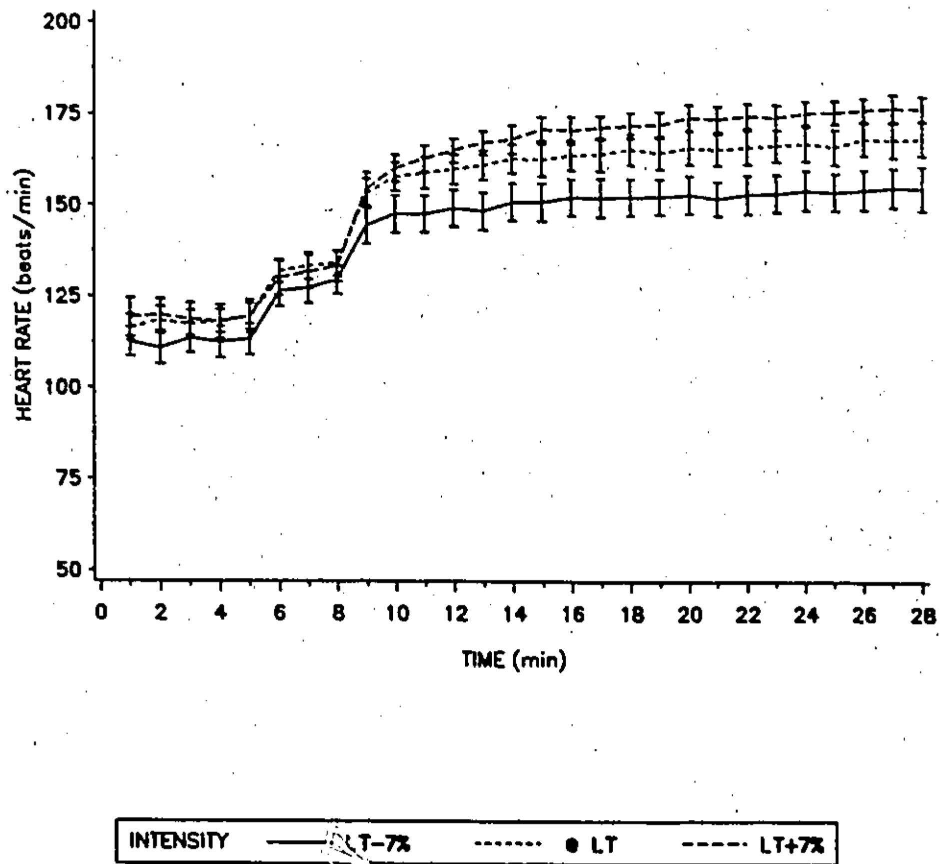


Figure 11. Heart rate (mean ± standard error) versus time for the three constant work rate tests.

Blood Lactate Concentration Analyses

The results from the ANOVA performed on the five blood lactate concentration measures taken from minutes 4 to 20 of the constant work segments are presented in Table 7. The ANOVA demonstrated that there were significant main effects due to the subjects, the work intensity, and the blood lactate sampling time. In addition, there was a significant interaction between the work intensity and the sampling time. Because of the significant interaction, two analyses of simple main effects were performed: one for work intensity at each level of sampling time, and the other for sampling time at each level of work intensity. The results of these analyses are presented in Table 8.

Table 7

Results from Three-Way ANOVA on Blood Lactate Concentration Measurements from Constant Work Rate Tests

Source of Variation	SS	df	MS	F
Subjects (S)	38.65	6	6.44	14.98[a]
Treatments	288.19	14	20.59	
Intensity (I)	267.67	2	133.84	311.26[a]
BLC Sampling Time (T)	8.34	4	2.09	4.86[a]
Interaction (IxT)	12.18	8	1.52	3.53[a]
Residual	36.24	84	0.43	
Total	363.08	104		

[a] $p < .01$

The analysis of simple main effects revealed that there was a significant intensity effect at each level of blood lactate sampling time. By applying post

Table 8

Results from Analysis of Simple Main Effects on Blood Lactate Concentration Measurements from Constant Work Rate Tests

Source of Variation	SS	df	MS	F
Intensity (I)	267.67	2	133.84	311.26[a]
I at T_1	22.19	2	11.10	25.81[a]
I at T_2	46.16	2	23.08	53.67[a]
I at T_3	59.28	2	29.64	68.93[a]
I at T_4	70.09	2	35.05	81.51[a]
I at T_5	82.12	2	41.06	95.49[a]
BLC Sampling Time (T)	8.34	4	2.09	4.86[a]
T at I_1	0.15	4	0.04	0.09
T at I_2	1.19	4	0.30	0.70
T at I_3	19.18	4	4.80	11.16[a]
Interaction (I×T)	12.18	8	1.52	3.53[a]
Residual	36.24	84	0.43	

[a] $p < .01$

hoc procedures, it was evident that for each sampling time the blood lactate concentration for the LT-7% intensity was significantly lower than that of the other two intensities. It was also evident that at each sampling time the blood lactate concentration for the @LT intensity was significantly lower than that of the LT+7% intensity. These results established that the three constant work rate intensities represented significantly different cycling intensities in terms of blood lactate concentration.

The analysis of simple main effects also revealed that, at the LT+7% intensity, sampling time had a significant effect on blood lactate concentration. Post hoc analyses demonstrated that the blood lactate concentration measured

at minute 4 of the 20 minute constant work period at LT+7% was significantly lower than that measured at any of the four subsequent times. In addition, the blood lactate concentration measured at minute 8 was significantly lower than that measured at minute 20 of the constant work at LT+7%. These results are noteworthy. They indicate that the blood lactate concentration measurements taken during the 20 minute constant work segments: (a) were not significantly different for the LT-7% and the @LT intensities, and (b) increased over the 20 minute work period for the LT+7% intensity.

A significant main effect was also demonstrated for the subjects. It was expected that there would be differences between the subjects in the physiological parameters measured. These differences are not relevant to the current investigation. Therefore, post hoc procedures will not be applied to determine where the inter-subject differences lie, either for this analysis, or for any of the subsequent analyses.

Baseline Blood Lactate Concentrations.

The previous analyses have demonstrated a significantly different blood lactate concentration response to each constant work intensity. The mean blood lactate concentration at the LT-7% intensity was 1.51 $mmol \times L^{-1}$, and the mean blood lactate concentration at the @LT intensity was 3.16 $mmol \times L^{-1}$. For both of these work intensities the blood lactate concentration measures did not demonstrate any significant change from the beginning to the end of the 20 minute constant work segment. In addition, the individual standard deviation in blood lactate concentration was less than 0.6 $mmol \times L^{-1}$. Therefore, these mean values can validly be used as blood lactate concentration baselines for their respective exercise intensities. The mean blood lactate concentration at the LT+7% intensity was 5.41 $mmol \times L^{-1}$. Blood lactate concentrations were

increasing across the 20 minute constant work segment at this intensity. Therefore, no baseline of blood lactate concentration could validly be determined for the LT+7% work intensity. This is in keeping with the lactate threshold definition which specifies that work above the lactate threshold intensity will cause a progressive rise in blood lactate concentration (refer to pages 12 and 13).

Oxygen Consumption Rate Analyses

The results from the ANOVA performed on the $\dot{V}O_2$ measures taken from minutes 4 to 20 of the constant work segments of the constant work rate tests are presented in Table 9. These results demonstrated that there were significant main effects due to the work intensity. Post hoc procedures were applied to the $\dot{V}O_2$ data to determine between which work intensities there were significant differences. The post hoc analyses clearly revealed that the mean $\dot{V}O_2$ for the LT-7% intensity (3.31 $L \times min^{-1}$) was significantly lower than that of both the @LT intensity (3.65 $L \times min^{-1}$) and the LT+7% intensity (3.98 $L \times min^{-1}$). In addition, the mean $\dot{V}O_2$ for the @LT intensity was significantly lower than that of the LT+7% intensity. These results correspond well with the blood lactate concentration results, since they demonstrate that each of the three constant work rate intensities represented significantly different exercise intensities in terms of $\dot{V}O_2$.

The fact that there was not a significant main effect of sampling time on $\dot{V}O_2$ indicates that the $\dot{V}O_2$ measurements were stable over the constant work period for each of the three work intensities.

Table 9

Results from Three-Way ANOVA on $\dot{V}O_2$ Measurements from Constant Work Rate Tests

Source of Variation	SS	df	MS	F
Subjects (S)	52.66	6	8.78	323.99[a]
Treatments	51.51	95		
Intensity (I)	49.45	2	24.73	912.55[a]
$\dot{V}O_2$ Sampling Time (T)	0.70	31	0.02	0.85
Interaction (I×T)	1.36	62	0.02	0.81
Residual	15.42	570	0.03	
Total	119.59	671		

[a] $p < .01$

Heart Rate Analyses

The results from the ANOVA performed on the heart rate measures taken from minutes 4 to 20 of the constant work segments of the constant work rate tests are presented in Table 10. Significant main effects were apparent for the work intensity and the time of heart rate sampling. Post hoc procedures revealed that all three work intensities produced significantly different heart rate responses. The mean heart rate at the LT-7% intensity (153 bpm) was significantly lower than those of both other work intensities. The mean heart rate at the @LT intensity (166 bpm) was significantly lower than that of the LT+7% intensity (173 bpm). These results correspond well with the blood lactate concentration and VO_2 results.

Table 10

Results from Three-Way ANOVA on Heart Rate Measurements from Constant Work Rate Tests

Source of Variation	SS	df	MS	F
Subjects (S)	34822.23	6	5803.70	352.63[a]
Treatments	25753.40	47		
Intensity (I)	24012.88	2	12006.44	729.51[a]
HR Sampling Time (T)	1600.35	15	106.69	6.48[a]
Interaction (IxT)	140.17	30	4.67	0.28
Residual	4641.20	282	16.46	
Total	65216.83	353		

[a] $p < .01$

The post hoc analyses also revealed that there was a slight increase in heart rate across the 20 minute constant work segment. The heart rates at minutes 19 and 20 were significantly greater than those measured from minutes 4 to 12 of the constant work segment.

Based on the results of the analyses performed on the blood lactate concentration, $\dot{V}O_2$, and heart rate measurements taken during the constant work segments of the constant work rate tests, it was clearly evident that each work intensity was significantly different from the others.

Surge-Recovery Tests

A number of analyses were required for this phase of the test results. The presentation of the results for the surge-recovery tests will be divided into two sections. In the first section, the results from the pre-recovery periods of the tests will be presented. In the second section, the results from the recovery periods of the tests will be presented.

Pre-Recovery Periods

The individual cycling intensities applied during the three pre-recovery phases (warm-up, @LT, and surge) were identical for each of the three surge-recovery tests performed by a subject. Therefore, in order to validly compare the results from the three separate surge-recovery tests performed by each subject, it was first necessary to establish that these standard pre-recovery periods of the tests were not significantly different from each other. During each 12 minute pre-recovery period, measurements of blood lactate concentration, VO_2, and heart rate were made. These measurements were utilized to determine whether or not the responses to the three pre-recovery periods were similar. The mean blood lactate concentration, VO_2, and heart rate measurements for the three pre-recovery periods are presented in Table 11. Diagrammatic representations of the physiological responses to the pre-recovery periods are presented in Figure 12.

A separate three-way ANOVA with repeated measures on two factors was performed for each of the three dependent variables: blood lactate concentration, VO_2, and heart rate. The independent variables were: (a) the recovery intensity, either LT-7%, @LT, or LT+7%, (b) the measurement sampling

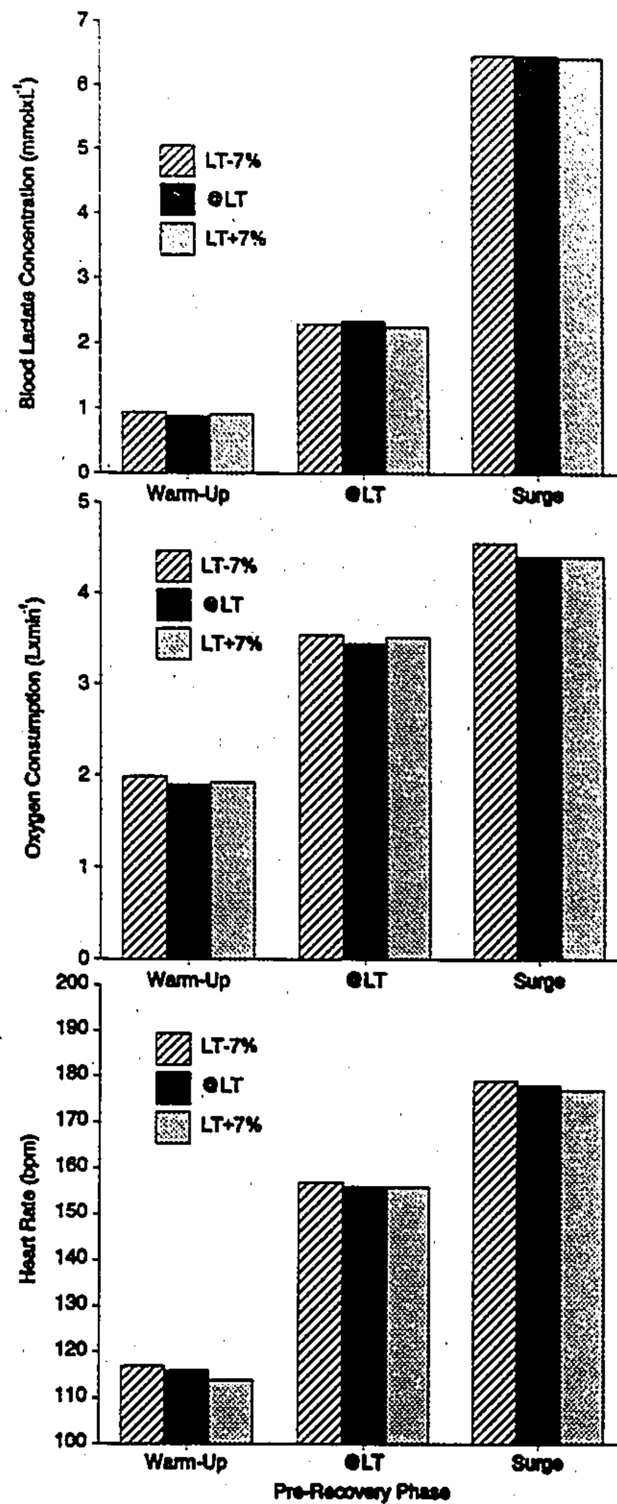


Figure 12. $\dot{V}O_2$, blood lactate concentration, and heart rate responses to pre-recovery periods of surge-recovery tests.

Table 11

Descriptive Statistics for Blood Lactate Concentration, $\dot{V}O_2$, and Heart Rate Measurements During the Three Phases of the Pre-Recovery Periods

PHASES		LT-7%	@LT	LT+7%
		Blood Lactate Concentration (mmol×L^{-1})		
Warm-Up	$\bar{X}$	0.93	0.88	0.91
	σ	0.30	0.32	0.31
@LT	$\bar{X}$	2.30	2.35	2.26
	σ	0.79	0.74	0.92
Surge	$\bar{X}$	6.48	6.47	6.44
	σ	1.47	1.23	1.31
		$\dot{V}O_2$ (L×min^{-1})		
Warm-Up	$\bar{X}$	1.99	1.90	1.93
	σ	0.24	0.23	0.28
@LT	$\bar{X}$	3.54	3.44	3.52
	σ	0.36	0.27	0.33
Surge	$\bar{X}$	4.56	4.41	4.48
	σ	0.43	0.38	0.47
		Heart Rate (bpm)		
Warm-Up	$\bar{X}$	117.1	116.6	114.1
	σ	9.1	8.9	10.4
@LT	$\bar{X}$	157.8	156.5	156.6
	σ	12.2	12.4	11.4
Surge	$\bar{X}$	179.1	178.6	177.0
	σ	10.6	9.7	9.3

time, and (c) the 7 subjects. The two repeated measures were the recovery intensity and the sampling time.

Blood Lactate Concentration Analyses.

The results from the ANOVA performed on the three blood lactate concentration measurements taken during each pre-recovery period are presented in Table 12. The blood lactate concentration measurements were taken at 5, 10, and 12 minutes of each pre-recovery period. The results of the ANOVA demonstrated that the blood lactate concentrations measured during the pre-recovery periods were not significantly different across the three tests. Therefore, in terms of blood lactate concentration, the three pre-recovery periods were similar.

Table 12

Results from Three-Way ANOVA on Blood Lactate Concentration Measurements from Pre-Recovery Periods

Source of Variation	SS	df	MS	F
Subjects (S)	24.94	6	4.16	9.39[a]
Treatments	350.78	8		
Intensity (I)	0.01	2	0.01	0.02
BLC Sampling Time (T)	350.74	2	175.37	396.13[a]
Interaction (IxT)	0.03	4	0.01	0.02
Residual	21.25	48	0.44	
Total	396.97	62		

[a] $p < .01$

A significant main effect due to blood lactate sampling time was apparent. This result was expected since the sampling occurred at the completion of periods of different intensities of work (refer to surge-recovery test protocol in Table 4 on page 82). The post hoc analysis confirmed that the blood lactate concentration measured immediately following the 5 minute warm-up period was significantly lower than that of the subsequent two measures. In addition, the analysis confirmed that the blood lactate concentration measured immediately following the 5 minute period at the lactate threshold intensity of exercise was significantly lower than that measured following the 2 minute period at the surge intensity.

Oxygen Consumption Rate Analyses.

The mean of the last four $\dot{V}O_2$ measurements from the warm-up phase was compared to the mean of the last four measurements from the lactate threshold phase, and the last measurement from the surge phase of the pre-recovery period. The results from the ANOVA performed on the $\dot{V}O_2$ measurements taken during the pre-recovery periods are presented in Table 13. These results demonstrated that the $\dot{V}O_2$ measurements made during the pre-recovery periods were not significantly different across the three tests. Therefore, in terms of $\dot{V}O_2$, the three pre-recovery periods were not significantly different. This result is consistent with the results from the blood lactate concentration measurement analyses.

The ANOVA demonstrated a significant main effect due to $\dot{V}O_2$ sampling time. This result was again expected, since the sampling times represented different work intensities. Post hoc analyses confirmed that the measures of $\dot{V}O_2$ collected during the warm-up phase were significantly lower than those

Table 13

Results from Three-Way ANOVA on $\dot{V}O_2$ Measurements from Pre-Recovery Periods

Source of Variation	SS	df	MS	F
Subjects (S)	5.40	6	0.90	45.18[a]
Treatments	69.49	8		
Intensity (I)	0.14	2	0.07	3.50
$\dot{V}O_2$ Sampling Time (T)	69.34	2	34.65	1738.15[a]
Interaction (IxT)	0.01	4	0.00	0.18
Residual	0.96	48	0.02	
Total	75.85	62		

[a] $p < .01$

collected in the subsequent two phases. The analyses also confirmed that the measures of VO_2 collected during the lactate threshold phase were significantly lower than the VO_2 measured during the surge phase.

Heart Rate Analyses.

The final analysis for the pre-recovery periods was performed on the heart rate data. The mean of the last two heart rates from the warm-up phase was compared to the mean of the last two heart rates from the lactate threshold phase, and the maximal heart rate measured during the surge phase. The results from the ANOVA are presented in Table 14. These results directly paralleled those of the blood lactate concentration and VO_2 measurement analyses. That is, there were no significant differences in heart rate across the three pre-recovery periods, and there was a significant main effect due to heart rate sampling time.

Table 14

Results from Three-Way ANOVA on Heart Rate Measurements from Pre-Recovery Periods

Source of Variation	SS	df	MS	F
Subjects (S)	4915.36	6	819.23	9.88[a]
Treatments	42127.66	8		
Intensity (I)	47.06	2	23.53	0.28
HR Sampling Time (T)	42067.30	2	21033.65	253.07[a]
Interaction (IxT)	13.30	4	3.33	0.04
Residual	3979.57	48	82.91	
Total	51022.59	62		

[a] $p < .01$

In summary, these results clearly demonstrated that the three separate pre-recovery periods were not significantly different from each other in terms of the blood lactate concentrations, the VO_2 values, and the heart rates they elicited. Therefore, it is possible to make valid comparisons of the three recovery periods.

Recovery Periods

Several analyses were performed to determine whether or not the three recovery intensities produced significantly different recovery responses. Once again, the blood lactate concentration, VO_2, and heart rate measurements taken during the recovery periods were the factors of comparison. The sampling times for these parameters during each 20 minute recovery period were: 1, 2, 3, 4, 5, 8, 11, 14, 17, and 20 minutes. The mean value for each parameter at each

sampling time in each of the three recovery intensities is presented in Table 15. A separate three-way ANOVA with repeated measures on two factors, was performed for each of the three dependent variables: blood lactate concentration, $\dot{V}O_2$, and heart rate. The three independent variables were: (a) the intensity of recovery, either LT-7%, @LT, or LT+7%, (b) the 10 measurement sampling times, and (c) the 7 subjects. The two repeated measures were the recovery intensity and the sampling time.

Blood Lactate Concentration Analyses.

The mean blood lactate concentration measurements for each recovery intensity are graphically presented in Figure 13. The results from the ANOVA performed on the recovery blood lactate concentration measurements are presented in Table 16. These results demonstrated significant main effects due to both the recovery intensity and the sampling time. Post hoc analyses revealed that the blood lactate measurements were significantly different across the three recovery intensities, with the measurements during recovery at LT-7% being the lowest, and those during recovery at LT+7% being the highest. The post hoc analyses on the blood sampling times revealed that blood lactate concentrations were fairly stable over the first 3 minutes of the recovery periods. However, from 3 to 11 minutes the blood lactate concentrations demonstrated a significant decrease between each sampling time. After the 11 minute sampling time, significant differences in blood lactate concentration between the remaining sampling times were not apparent.

Table 15

Mean Blood Lactate, $\dot{V}O_2$, and Heart Rate Measurements from Recovery Periods

Recovery Sampling Time (minutes)	Recovery Intensity		
	LT-7%	@LT	LT+7%
	Blood Lactate Concentration (mmol×L^{-1})		
1	7.49	7.61	7.70
2	7.06	7.35	7.52
3	6.33	6.61	6.97
4	5.49	5.78	6.45
5	4.73	5.35	6.02
8	3.12	3.96	5.11
11	2.29	3.17	4.61
14	1.84	2.83	4.41
17	1.68	2.63	4.34
20	1.57	2.49	4.17
	$\dot{V}O_2$ (L×min^{-1})		
1	4.03	4.13	4.25
2	3.59	3.82	3.96
3	3.44	3.63	3.89
4	3.41	3.56	3.93
5	3.36	3.56	3.86
8	3.28	3.56	3.82
11	3.32	3.56	3.90
14	3.28	3.50	3.86
17	3.27	3.57	3.86
20	3.24	3.51	3.87
	Heart Rate (bpm)		
1	169.1	170.6	172.7
2	158.9	162.7	168.3
3	153.4	160.3	166.3
4	152.7	159.3	167.0
5	153.6	160.1	168.6
8	156.4	163.0	170.1
11	157.0	163.6	171.7
14	156.0	164.6	173.2
17	156.4	164.1	173.3
20	155.1	164.3	173.1

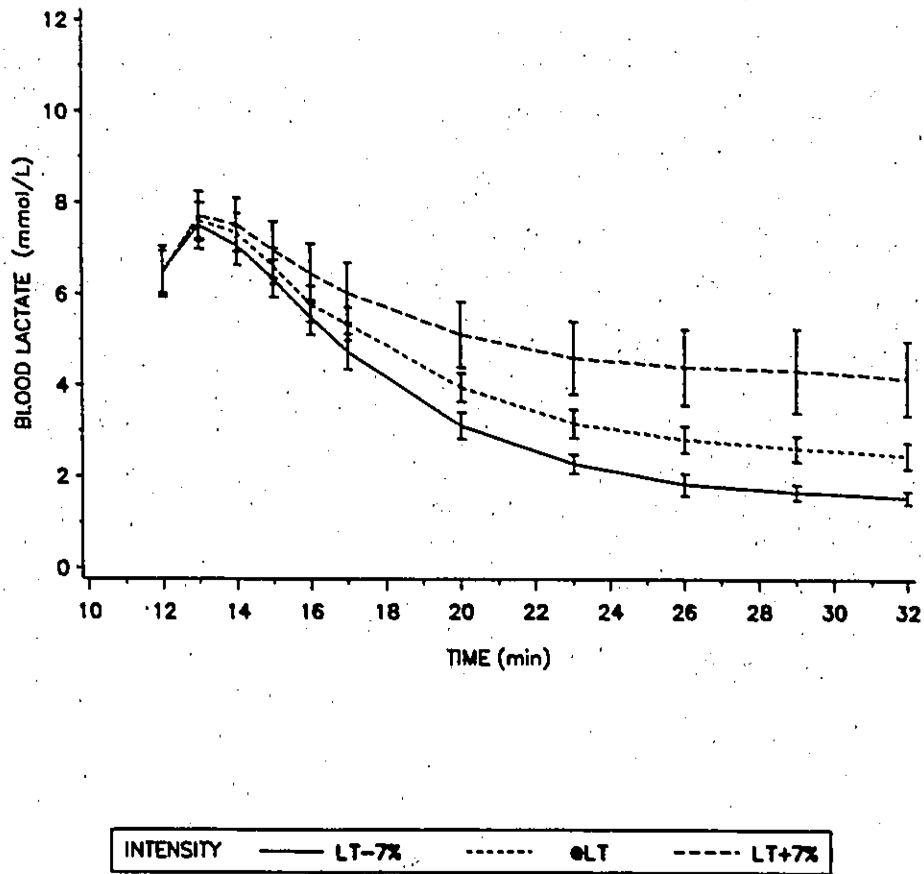


Figure 13. Blood lactate concentration (mean ± standard error) versus time during the recovery period of the surge-recovery tests.

Table 16

Results from Three-Way ANOVA on Blood Lactate Concentration Measurements from Recovery Periods

Source of Variation	SS	df	MS	F
Subjects (S)	163.18	6	27.20	30.64[a]
Treatments	804.18	29		
Intensity (I)	87.70	2	43.85	49.40[a]
BLC Sampling Time (T)	685.94	9	76.22	85.86[a]
Interaction (IxT)	30.54	18	1.70	1.92
Residual	154.46	174	0.89	
Total	1121.82	209		

[a] $p < .01$

Oxygen Consumption Rate Analyses.

The mean VO_2 measurements for each recovery intensity are graphically presented in Figure 14. The results from the ANOVA performed on the recovery VO_2 measurements are presented in Table 17. These results demonstrated significant main effects due to both the recovery intensity and the sampling time. Post hoc analyses revealed that the VO_2 for all three recovery intensities were significantly different from each other. The LT-7% intensity demonstrated the lowest rates, and the LT+7% intensity demonstrated the highest rates. Post hoc analyses performed for the VO_2 sampling times revealed that the VO_2 during the first minute of the recovery period was significantly higher than that measured for all other sampling times. Additionally, none of the VO_2 values, from minutes 2 to 20 of the recovery period, were significantly different from each other.

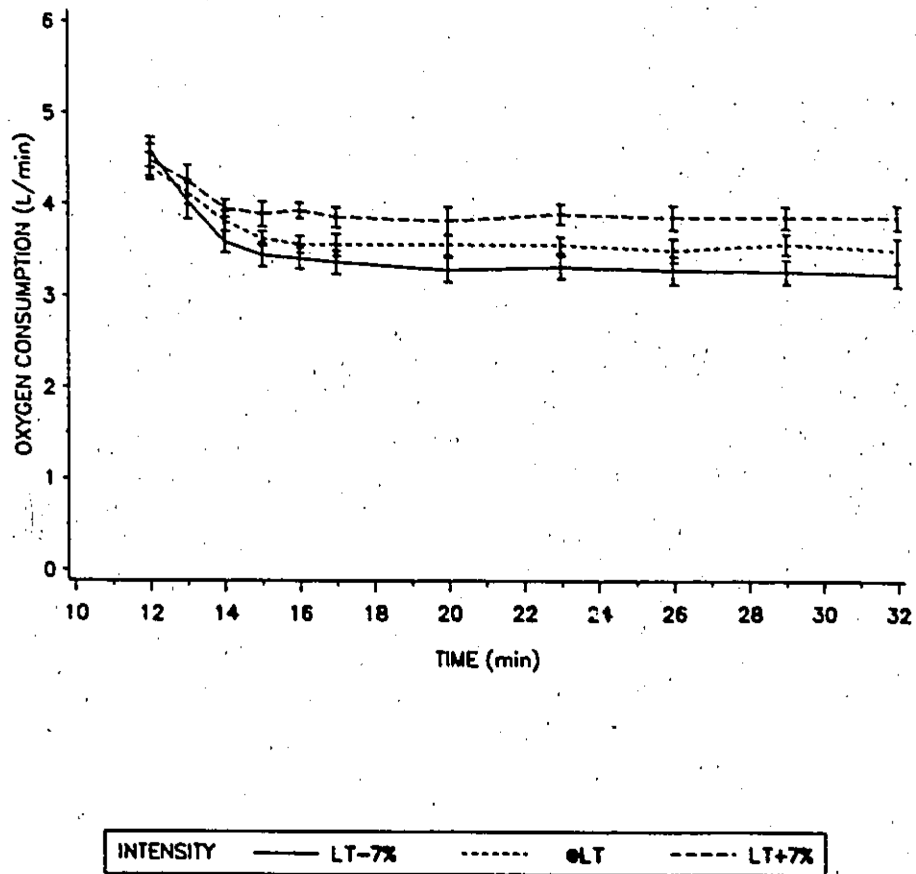


Figure 14. $\dot{V}O_2$ (mean ± standard error) versus time during the recovery period of the surge-recovery tests.

Table 17

Results from Three-Way ANOVA on $\dot{V}O_2$ Measurements from Recovery Periods

Source of Variation	SS	df	MS	F
Subjects (S)	16.00	6	2.66	96.63[a]
Treatments	12.94	29		
Intensity (I)	7.25	2	3.63	131.86[a]
$\dot{V}O_2$ Sampling Time (T)	4.90	9	0.54	19.62[a]
Interaction (IxT)	0.79	18	0.04	1.45
Residual	4.79	174	0.28	
Total	33.73	209		

[a] $p < .01$

Heart Rate Analyses.

The mean heart rate measurements for each recovery intensity are graphically presented in Figure 15. The results from the ANOVA performed on the recovery heart rate measurements are presented in Table 18. These results were similar to those for blood lactate concentration and $\dot{V}O_2$. That is, there was a significant main effect on heart rate due to both the intensity of the recovery period and the measurement sampling time. The post hoc analyses performed for the intensity effect revealed that the heart rates measured during recovery at each of the three different intensities were significantly different from each other. The heart rates during recovery at LT-7% were the lowest, and those during recovery at LT+7% were the highest. The post hoc analyses performed for the heart rate sampling times demonstrated results that were identical to those of the oxygen consumption sampling times. That is, the heart rate during the first minute of recovery was significantly higher than all

subsequent heart rates, and there were no significant differences in heart rate from minutes 2 to 20 of the recovery periods.

The results of the analyses performed on the blood lactate concentration, VO_2, and heart rate data gathered during the recovery periods of the surge-recovery tests clearly demonstrated that the recovery response was significantly different for each of the recovery intensities, in terms of the aforementioned physiological parameters. The LT-7% recovery intensity consistently demonstrated significantly lower blood lactate concentration, VO_2, and heart rate measurements than both the @LT and the LT+7% recovery intensities. Additionally, the @LT recovery intensity consistently demonstrated significantly lower blood lactate concentration, VO_2, and heart rate measurements than the LT+7% recovery intensity.

Table 18

Results from Three-Way ANOVA on Heart Rate Measurements from Recovery Periods

Source of Variation	SS	df	MS	F
Subjects (S)	21763.39	6	3627.23	295.99[a]
Treatments	8521.41	29		
Intensity (I)	6065.55	2	3032.78	247.48[a]
HR Sampling Time (T)	1955.22	9	217.25	17.73[a]
Interaction (IxT)	500.64	18	27.81	2.27
Residual	2132.32	174	12.26	
Total	32417.12	209		

[a] $p < .01$

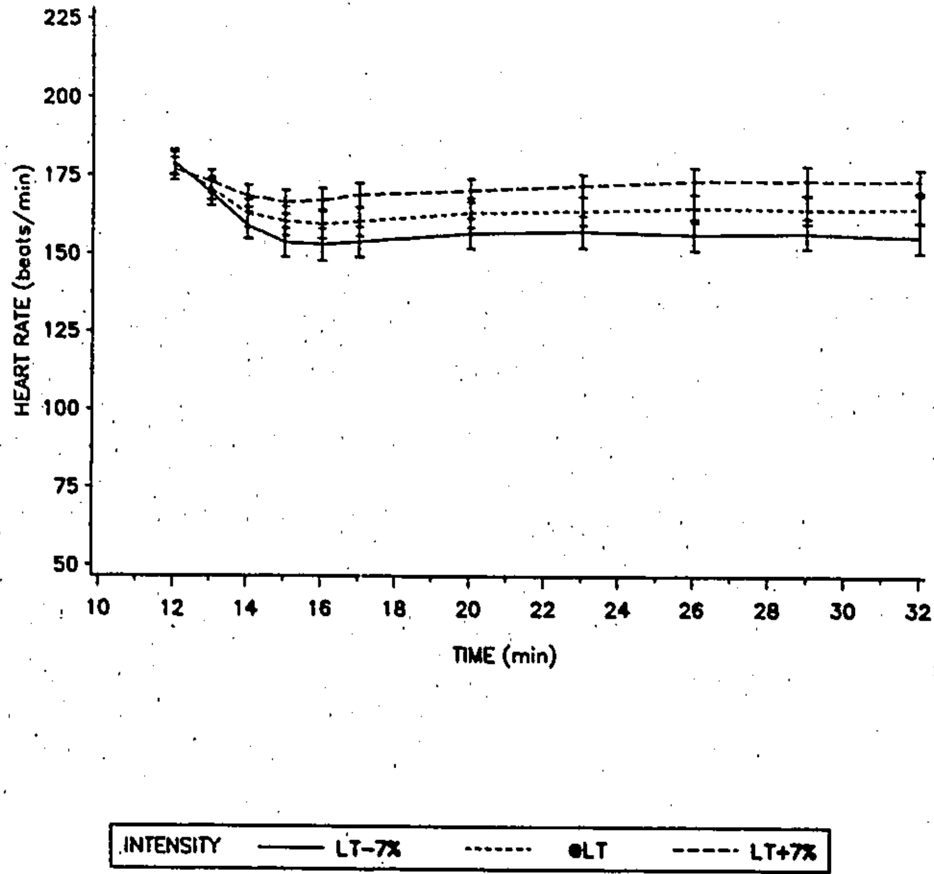


Figure 15. Heart rate (mean ± standard error) versus time during the recovery period of the surge-recovery tests.

Times for Half Decrease of Blood Lactate

In order to establish which recovery intensity resulted in the highest rate of decrease in blood lactate, an analysis of the change in blood lactate concentration with respect to the appropriate blood lactate baseline for the specific recovery intensity was performed. This analysis involved determining and comparing the times for half decrease of blood lactate for each subject at each recovery intensity. The individual and group results for the times for half decrease in blood lactate concentration are presented in Table 19.

It should be noted that the blood lactate concentration baseline used for the LT+7% intensity was that of the @LT intensity. This adjustment was necessary because a blood lactate concentration baseline could not validly be determined for the LT+7% intensity. This issue has been addressed previously on pages 80 and 81.

With the exception of subject 1 at the LT+7% recovery intensity, all of the subjects recovered to at least the half blood lactate concentration at all recovery intensities. The mean recovery responses, for all three recovery intensities, are presented graphically in Figure 16. The baseline blood lactate concentrations for the recovery intensities have also been presented in Figure 16.

The mean time for half decrease in blood lactate concentration was 5.46 minutes for the LT-7% intensity, 4.81 minutes for the @LT intensity, and 5.82 minutes for the LT+7% intensity. In order to determine whether or not the times were significantly different from each other, a two-way (3X7) ANOVA with repeated measures on one factor was performed. The two independent variables in the analysis were (a) the recovery intensity, either LT-7%, @LT, or

Table 19

Mean Results from Analyses of Blood Lactate Concentration Decreases During Recovery Periods

Subject	Intensity	Peak BLC (mmol×L^{-1})	Baseline BLC (mmol×L^{-1})	Half BLC (mmol×L^{-1})	Half-Time (minutes)
1	LT-7%	7.18	2.24	4.71	5.20
	@LT	8.16	3.42	5.79	5.25
	LT+7%	9.92	3.42	6.67	∞
2	LT-7%	9.14	1.39	5.27	6.13
	@LT	7.26	3.82	5.54	2.95
	LT+7%	6.14	3.82	4.98	4.21
3	LT-7%	7.70	1.25	4.48	5.59
	@LT	8.64	2.85	5.75	5.97
	LT+7%	8.34	2.85	5.60	6.29
4	LT-7%	5.22	1.44	3.33	4.58
	@LT	5.80	2.82	4.31	4.81
	LT+7%	6.42	2.82	4.62	4.31
5	LT-7%	8.24	1.50	4.87	4.46
	@LT	8.80	2.80	5.80	5.28
	LT+7%	9.00	2.80	5.90	8.04
6	LT-7%	8.52	1.48	5.00	5.52
	@LT	8.34	4.14	6.24	4.53
	LT+7%	8.20	4.14	6.17	6.34
7	LT-7%	6.96	1.27	4.12	6.46
	@LT	6.70	2.26	4.48	5.32
	LT+7%	6.18	2.26	4.22	5.75
Group (η=6)[a]					
	LT-7%	7.63	1.39	4.51	5.46
	@LT	7.59	3.12	5.35	4.81
	LT+7%	7.38	3.12	5.25	5.82

[a] Not including Subject 1.

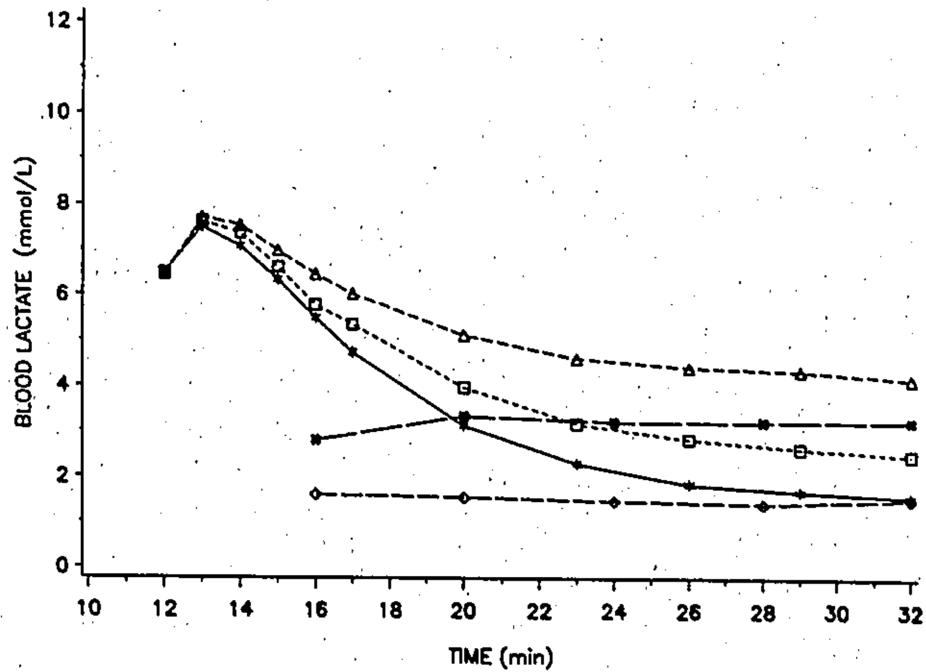


Figure 16. Mean blood lactate concentration versus time during the recovery period of the surge-recovery tests. The baseline blood lactate concentrations for the LT-7% and the @LT intensities are also presented.

LT+7%; and (b) the 7 subjects. The repeated measure was the recovery intensity. The dependent variable was the time for half decrease. The results from the ANOVA are presented in Table 20. These results demonstrated that there were no significant differences in the times for half decrease in blood lactate concentration between the three recovery intensities. Therefore, half blood lactate decrease occurred just as rapidly at the LT+7% recovery intensity as it did at either of the two lower recovery intensities.

Table 20

Results from Two-Way ANOVA on Half-Times for Blood Lactate Decrease

Source of Variation	SS	df	MS	F
Subjects (S)	7.22	5	1.44	1.23
Intensity (I)	3.16	2	1.58	1.35
Residual	11.68	10		
Total	22.06	17		

DISCUSSION AND CONCLUSIONS

The aim of this investigation was to identify the intensity of recovery exercise that would result in the most rapid time for half decrease in blood lactate, following a standard duration of activity at $\dot{V}O_2max$ intensity. The unique features of this investigation were: (a) that the intensities of recovery exercise were defined relative to the individual lactate threshold intensity; (b) that the recovery intensities chosen for investigation were those that would likely be used within a competitive endurance event, that is, slightly below, at, and slightly above the individual lactate threshold intensity; and (c) that the changes in blood lactate concentration were evaluated with respect to baseline blood lactate concentrations which were determined individually for each recovery exercise intensity. It was hypothesized that recovery at the individual lactate threshold intensity would result in the most rapid times for half decrease in blood lactate.

The results of this investigation clearly demonstrated that the absolute reduction in blood lactate concentration during recovery was significantly different across the three recovery exercise intensities. When recovery exercise was performed at the subthreshold intensity, blood lactate was reduced to the lowest concentrations during the 20 minute recovery period. Conversely, when recovery exercise was performed at the suprathreshold intensity (LT+7%) the highest recovery blood lactate concentrations were observed. These results had been anticipated, as it is well known that during moderate to heavy exercise the concentration of blood lactate is directly related to exercise intensity (Ribeiro et

al, 1986, Scheen et al., 1981). Thus, it was not surprising that significantly different blood lactate concentrations were apparent across the three recovery exercise intensities. However, a comparison of blood lactate concentrations on such absolute terms gives little insight into the recovery ability of the system relative to the exercise intensity that is being performed. An unbiased comparison of changes in blood lactate concentration must include consideration of the baseline blood lactate concentration that will be induced by each intensity of recovery exercise.

In order to conduct such a comparison, times for half decrease in blood lactate concentration were calculated using individually determined baselines of blood lactate concentration. In terms of these times for half decrease there were no significant differences between the three recovery exercise intensities. In other words, the decrease in blood lactate concentration, following the 2 minute surge at $\dot{V}O_2max$ intensity, was equally rapid for all three of the recovery intensities investigated when the appropriate baselines of blood lactate concentration were considered. Therefore, the original hypothesis must be rejected, since recovery exercise performed at the lactate threshold intensity did not result in statistically superior times for half decrease in blood lactate.

In terms of the times for half decrease in blood lactate, the results of this investigation parallel those of Stamford et al. (1981). These researchers investigated two active recovery intensities: 40% $\dot{V}O_2max$ and 70% $\dot{V}O_2max$. They found that the lowest active recovery intensity resulted in the greatest absolute decrease in blood lactate over a 40 minute recovery period. However, when the active recovery intensities were assessed with respect to their appropriate baselines of blood lactate concentration, neither were significantly

different in terms of their resulting blood lactate recovery kinetics. Davies et al. (1970) and Belcastro and Bonen (1975) also demonstrated significantly faster absolute decreases in blood lactate at relatively low exercise intensities (40% $\dot{V}O_2max$). However, because neither of these latter studies considered blood lactate concentration baselines in their analyses of blood lactate decrease kinetics, they concluded that the low exercise intensity was the most effective for blood lactate decrease. To this end, the results of the present investigation confirm the conclusions made by Stamford et al. (1981): "that interpretation of the effectiveness of exercise recovery above and below (lactate threshold) with respect to blood lactate disappearance is influenced by the baseline blood lactate concentration".

There are two aspects of the results from this investigation that warrant further discussion. First, 6 of the 7 subjects demonstrated an ability to reduce blood lactate concentrations, when the intensity of the recovery exercise was above lactate threshold (LT+7%). This result was unexpected, since during constant rate work at the same intensity all of the cyclists demonstrated significant increases in blood lactate concentration. A further observation was that during recovery at the lactate threshold intensity the blood lactate concentrations decreased to baseline levels after the first 11 minutes and fell to levels well below the baseline during the remaining 9 minutes. In contrast, during recovery at the subthreshold intensity blood lactate concentrations only dropped to baseline levels after 20 minutes of recovery (refer to Figure 16 on page 116). Both of these observations lead to the possible conclusion that following the 2 minute surge at $\dot{V}O_2max$ intensity, lactate disappearance from the blood was enhanced and/or lactate entry into the blood was reduced at the @LT and the LT+7% recovery intensities, relative to the rates of these processes during the constant work rate tests.

The common supposition is that when exercise intensities exceed the lactate threshold intensity, the equilibrium between lactate appearance and blood lactate disappearance will be destroyed and blood lactate accumulation will be inevitable (Gladden, 1989). Yet, in the current investigation, when the cyclists recovered at the LT+7% intensity, the decrease in blood lactate concentration was significant and progressive over the entire 20 minute recovery period. That this observation is the result of a training effect during the experimentation is unlikely. The subjects were exposed to the three recovery intensities randomly, and they were highly trained (endurance) competitive cyclists. The trained state of the subjects is reflected in the mean VO_2max of 4.6 $Lxmin^{-1}$ and in the lactate threshold which occurred at a mean of 79% of $\dot{V}O_2max$. It is doubtful that the $\dot{V}O_2max$ test and the three constant work rate tests, all of which preceded the surge-recovery tests, would have elicited a significant change in the lactate threshold exercise intensity of these well trained cyclists.

An explanation for these observations may lie within the mechanisms of blood lactate disappearance and those of blood lactate appearance. Some aspect(s) of either or both of these processes may have been modified as a result of the 2 minute surge at $\dot{V}O_2max$ intensity, since the surge was essentially the only protocol feature that was different between the constant work rate tests and the surge-recovery tests.

The large lactate load that was induced by the surge may have facilitated blood lactate disappearance in a number of ways. Two of the factors which are known to improve lactate uptake are high extracellular lactate concentrations and low extracellular pH. Assuming that the high post-surge blood lactate

concentrations reflected high lactate concentrations both within the working muscle cells and within the extracellular space, the uptake of lactate by tissues capable of oxidizing the substrate may have been facilitated. In addition, elevated blood lactate concentrations are known to inhibit free fatty acid mobilization (Astrand, 1986). As such, conditions which would promote the use of lactate as a fuel source by the active tissues were very likely created during recovery from the surge. Lactate and hydrogen ion concentrations are known to be positively correlated. This fact is significant in that the transport of pyruvate across the inner mitochondrial membrane depends on both the existence and the size of the pH gradient across the membrane (Zubay, 1983). Based on the blood lactate concentrations measured, it is a reasonable assumption that the surge created such a pH gradient. Thus, the inward transport of pyruvate across the inner mitochondrial membrane may have been augmented during the recovery phase of the ride. By improving the transport of pyruvate into the mitochondria, the availability of this substrate for oxidation via the TCA cycle would also have been improved.

In summary, three mechanisms have been proposed which may have, either singularly or in combination, resulted in an augmented blood lactate removal capacity following the 2 minute surge at $\dot{V}O_2max$: (a) improvements in the ability of tissues to take up lactate because of elevated extracellular lactate concentrations and reduced extracellular pH, (b) augmented use of lactate as a fuel because of its accessibility and because of inhibited free fatty acid mobilization, and (c) enhanced ability for pyruvate transport across the inner mitochondrial membrane due to the enhanced pH gradient across the membrane.

In addition to the possible effects of the lactate load on blood lactate kinetics during active recovery, the maximal intensity activity of the surge itself may have provided a stimulus for enhanced lactate utilization and reduced blood lactate appearance. The initial drop in energy charge within the active muscle cells resulting at the onset of surge activity would have provided a stimulus for enhanced glycolytic activity and enhanced TCA cycle activity. It can be speculated that if TCA cycle activity were to remain at augmented levels and the rate of glycolysis were to be reduced, following the 2 minute surge, the ability of the active muscle cells to oxidize exogenous lactate would be enhanced. In order to support this hypothesis, it must be established that: (a) TCA cycle activity would be enhanced by the surge, and (b) TCA cycle activity would remain enhanced after the surge during active recovery.

A primary regulator of TCA cycle activity is the pyruvate dehydrogenase complex (Lehninger, 1982). This enzyme system catalyzes the series of reactions responsible for the oxidative decarboxylation of pyruvate to acetyl-CoA. The pyruvate dehydrogenase complex is present in the mitochondria in both active and inactive forms. The inactive form is converted to the active form when there is a decrease in the mitochondrial ATP to ADP ratio and when there is a rise in the intracellular Ca^{2+} concentration (Lehninger, 1982). The 2 minute surge at maximal intensity could have provided both of these stimuli. These same stimuli would have resulted in an increased glycolytic rate, thereby providing pyruvate dehydrogenase with ample amounts of the substrate it requires for acetyl-CoA formation. Therefore, the surge may provide a stimulus for maximizing the concentration of active pyruvate dehydrogenase in the mitochondria, allowing for potentiation of the cells capacity to oxidize pyruvate and subsequently lactate.

If two activities are performed at the same intensity, one with a preceding surge of short duration and one without, it is conceivable that for the activity preceded by the surge the rate of the TCA cycle would be relatively higher. The difference in TCA cycle rate between the two situations would be the result of a number of interrelated factors. First, the surge provides maximal stimulation for the TCA cycle; that is, the surge provides the necessary stimuli for maximizing the intramitochondrial concentrations of active pyruvate dehydrogenase. Second, the build-up of metabolites during the surge reduces the glycolytic capacity within the active muscle tissue (refer to page 54), and thus creates a dependence on mitochondrial processes for ATP during active recovery. Third, the surge results in an accumulation of endogenous and exogenous pyruvate and lactate, thereby providing an ample supply of the necessary substrate for the TCA cycle. Finally, the build-up of metabolites during the surge enhances pyruvate uptake by the mitochondria because of the existence of a pH gradient across the inner mitochondrial membrane. Therefore, it is conceivable that the TCA cycle activity remains at a higher rate during activity preceded by a surge of short duration.

The elevated TCA cycle activity during recovery would enhance the utilization of endogenous and exogenous lactate, since lactate is a precursor of pyruvate. Lactate uptake in the Type I and cardiac muscle tissue may be improved as an indirect result of this enhanced capacity for pyruvate oxidation following the surge. This enhanced capacity would reduce pyruvate accumulation in the cytosol and thus the inhibitory effect of high pyruvate concentrations on the H isozyme of LDH would be reduced. This would allow continued conversion of lactate to pyruvate, and therefore, continued uptake of lactate from the interstitia and blood.

Several possible effects of the surge and of the resulting lactate load on blood lactate uptake mechanisms have been presented. However, there are a number of questions that remain unanswered. For example, during constant work above lactate threshold, a progressive increase in blood lactate concentration was apparent. Why did the lactate load generated during the constant work rate test not instigate the response apparent during the surge-recovery ride at the same intensity? The difference in the lactate load between that generated by the surge and that generated during the 20 minute ride at LT+7% was in the rate of accumulation. Perhaps the proposed mechanism is rate specific. Another difference between the two lactate loads is that prior to the recovery ride the processes of oxidative metabolism had likely been maximally stimulated, thereby possibly creating an enhanced capacity to oxidize lactate, while there was no such maximal stimulation during the constant ride.

Further questions are suggested from the fact that enhanced blood lactate removal capacities were apparent for a minimum of 20 minutes of active recovery, during which time blood lactate concentrations dropped to baseline levels, if not lower. In a similar investigation conducted by Stamford et al. (1981), the enhanced removal capacity was apparent for at least 40 minutes of active recovery. These results raise the following questions:

1. How long would the enhanced capacity for blood lactate removal continue?
2. How would the enhanced capacity for blood lactate removal be influenced by another surge?
3. At what percentage of VO_2max above lactate threshold would recovery be ineffective in terms of decreases in blood lactate concentration? That is, is there another lactate threshold that is

specific to exercise performed at constant intensities that has been preceded by a surge of short duration? And if so, at what percentage of VO_2max does this threshold occur, and how is it influenced by the length and intensity of the surge?

The results of this investigation demonstrated that there were no significant differences in the times for half decrease in blood lactate concentration between the three recovery exercise intensities investigated. This would imply that the endurance athlete can effectively recover (in terms of blood lactate concentration), from a surge of short duration within a competitive event, at an intensity that is slightly above lactate threshold. In addition, since the surge activity seems to initiate a response which enhances the oxidative capacity of the system, endurance athletes may improve their performances by incorporating a short surge of activity within their pre-event warm-up routine. Both of these recommendations must be considered carefully, since blood lactate concentration has only been associated with muscular fatigue. That is, there may be other fatiguing influences that must be considered in addition to the blood lactate concentration. These influences include: glycogen depletion, blood NH_3 concentration, excitability of motoneurons, contractility of muscle cells, etc. (refer to pages 55 and 56).

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

1. The absolute decrease in blood lactate concentration was significantly greater during recovery exercise performed at an intensity slightly below the lactate threshold, in comparison to recovery performed at and slightly above the lactate threshold exercise intensity.

2. The absolute decrease in blood lactate concentration was significantly lower during recovery exercise performed at an intensity slightly above the lactate threshold, in comparison to recovery performed at and slightly below the lactate threshold exercise intensity.
3. The times for half decrease in blood lactate concentration during active recovery, calculated relative to individually determined baseline blood lactate concentrations, were not significantly different for any of the recovery exercise intensities investigated. It therefore seems reasonable to conclude that analyses of the response of blood lactate concentration to various recovery exercise intensities should incorporate consideration of the baseline blood lactate concentration induced by the specific recovery exercise intensity.
4. The processes which govern blood lactate appearance and removal may have been influenced by the short duration surge and/or by the lactate load resulting from the surge.

In conclusion, these results have provided greater insight into the phenomenon termed the lactate threshold. It is quite possible that this threshold is not only specific to the individual and to the mode of exercise employed, but also to the specific protocol of exercise used in its determination. That is, the data suggest that the lactate threshold evident during constant work preceded by a 2 minute surge at maximal intensities was different than the one evident during constant work without the preceding surge. Further research should be conducted using similar intensity gradients but a larger range of intensities to investigate this postulation. In addition, further research

should investigate how the intensity and the duration of the surge itself affects blood lactate decrease during recovery. The answers to such questions would be useful in providing the endurance athlete with performance guidelines and racing strategies.

BIBLIOGRAPHY

Astrand, P., & Rodahl, K. (1986). Textbook of work physiology: Physiological bases of exercise. New York: McGraw-Hill.

Aunola, S., & Rusko, H. (1984). Reproducibility of aerobic and anaerobic thresholds in 20-50 year old men. European Journal of Applied Physiology, 53, 260-266.

Aunola, S., & Rusko, H. (1986). Aerobic and anaerobic thresholds determined from venous lactate or from ventilation and gas exchange in relation to muscle fiber composition. International Journal of Sports Medicine, 7, 161-166.

Bauer, H. P., Reichman, H., & Hofer, W. (1986). Perfusion of psoas muscle of the rabbit: metabolism of a homogeneous muscle composed of 'fast glycolytic' fibres. International Journal of Biochemistry, 18, 67-72.

Belcastro, A. N., & Bonen, A. (1975). Lactic acid removal rates during controlled and uncontrolled recovery exercise. Journal of Applied Physiology, 39(6), 932-936.

Bergstrom, J., & Hultman, E. (1967). A study of the glycogen metabolism during exercise in man. Scandinavian Journal of Clinical and Laboratory Investigations, 19, 218-228.

Bergstrom, J., Hermansen, L., Hultman, E., & Sahlin, B. (1967). Diet, muscle glycogen and physical performance. Acta Physiol Scand, 71, 140-150.

Bigland-Ritchie, B., & Woods, J. J. (1984). Changes in muscle contractile properties and neural control during human muscular fatigue. Muscle and Nerve, 7, 691-699.

Bonen, A., & Belcastro, A. N. (1976). Comparison of self-selected recovery methods on lactic acid removal rates. Medicine and Science in Sports, 8(3), 176-178.

Brooks, G. A. (1985). Anaerobic threshold: review of the concept and directions for future research. Medicine and Science in Sports and Exercise, 17(1), 22-31.

Cartier, L.-J., & Gollnick, P. D. (1985). Sympathoadrenal system and activation of glycogenolysis during muscular activity. Journal of Applied Physiology, 58, 1122-1127.

Clamann, H. P. (1987). Fatigue mechanisms and contractile changes in motor units of the cat hindlimb. Canadian Journal of Sports Sciences, 12(Suppl.1), 20S-25S.

Conconi, R., Ferrari, M., Ziglio, P. G., Droghetti, P., & Codeca, L. (1982). Determination of the anaerobic threshold by a noninvasive field test in runners. Journal of Applied Physiology, 52, 869-873.

Costill, D. L. (1970). Metabolic responses during distance running. Journal of Applied Physiology, 28(3), 251-255.

Costill, D. L., Coyle, E., Dalsky, G., Evans, W., Fink, W., & Hoopes, D. (1977). Effects of elevated plasma FFA and insulin on muscle glycogen usage during exercise. Journal of Applied Physiology, 43(4), 695-699.

Coyle, E. F., Coggan, A. R., Hopper, M. K., & Walters, T. J. (1988). Determinants of endurance in well-trained cyclists. Journal of Applied Physiology, 64(6), 2622-2630.

Davies, C. T. M., Knibbs, A. V., & Musgrove, J. (1970). The rate of lactic acid removal in relation to different baselines of recovery exercise. Int. Z. angew. Physiol., 28, 155-161.

Davis, H. A. & Gass, G. C. (1981). The anaerobic threshold - as determined before and during lactic acidosis. European Journal of Physiology, 47, 141-149.

Davis, J. A., Vodak, P., Wilmore, J. H., Vodak, J., & Kurtz, P. (1976). Anaerobic threshold and maximal aerobic power for three modes of exercise. Journal of Applied Physiology, 41(4), 544-550.

Depocas, F., Minaire, Y., & Chatonnet, J. (1969). Rates of formation and oxidation of lactic acid in dogs at rest and during moderate exercise. Canadian Journal of Physiology and Pharmacology, 47, 603-610.

Donovan, C. M., & Brooks, G. A. (1983). Endurance training affects lactate clearance, not lactate production. American Journal of Physiology, 244 (Endocrinol. Metab. 7), E83-E92.

Eldridge, F. L. (1975). Relationship between turnover rate and blood concentration of lactate in exercising dogs. Journal of Applied Physiology, 39(2), 231-234.

Eldridge, F. L., T'so, L., & Chang, H. (1974). Relationship between turnover rate and blood concentration of lactate in normal dogs. Journal of Applied Physiology, 37(3), 316-320.

Farrell, P. A., Wilmore, J. H., Coyle, E. F., Billing, J. E., & Costill, D. (1979). Plasma lactate accumulation and distance running performance. Medicine and Science in Sports, 11(4), 338-344.

Gerald, C. F. & Wheatley, P. O. (1985). Applied Numerical Analysis. Reading, Massachusetts: Addison - Wesley Publishing Company.

Gibson, H., & Edwards, R. H. T. (1985). Muscular exercise and fatigue. Sports Medicine, 2, 120-132.

Gladden, L. B. (1989). Lactate uptake by skeletal muscle. Exercise and Sport Sciences Reviews, 17, 115-155.

Gollnick, P. O., & Hermansen, L. (1973). Biochemical adaptations to exercise: Anaerobic metabolism. Exercise and Sport Sciences Reviews, 1, 1-43.

Green, H. J. (1987). Neuromuscular aspects of fatigue. Canadian Journal of Sports Sciences, 12(Suppl.1), 7S-19S.

Griffiths, J. R., & Rahin, Z. H. A. (1978). Glycogen as a fuel for skeletal muscle. Biochemistry Society Transactions, 6, 530-534.

Hermansen, L. (1981). Effect of metabolic changes on force generation in skeletal muscle during maximal exercise. In: R. Porter, & J. Whelan (Eds.), Human muscle fatigue: Physiological mechanisms (pp. 75-88). London: Pitman Medical.

Hermansen, L., & Stensvold, I. (1972). Production and removal of lactate during exercise in man. Acta Physiol Scand, 86, 191-201.

Hogan, M. C., & Welch, H. G. (1984). Effect of varied lactate levels on bicycle ergometer performance. Journal of Applied Physiology, 57(2), 507-513.

Hogan, M. C., & Welch, H. G. (1986). Effect of altered arterial O_2 tensions on muscle metabolism in dog skeletal muscle during fatiguing work. American Journal of Physiology, 251, C216-C222.

Hughes, E. F., Turner, S. C., & Brooks, G. A. (1982). Effects of glycogen depletion and pedaling speed on "anaerobic threshold". Journal of Applied Physiology, 52(6), 1598-1607.

Hughson, R. L., & Green, H. J. (1982). Blood acid-base and lactate relationships studied by ramp work tests. Medicine and Science in Sports and Exercise, 14 (4), 297-302.

Hultman, E., & Sahlin, K. (1980). Acid-base balance during exercise. Exercise and Sport Sciences Reviews, 8, 41-128.

Issekutz, B., Shaw, W. A. S., & Issekutz, A. C. (1976). Lactate metabolism in resting and exercising dogs. Journal of Applied Physiology, 40(3), 312-319.

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

Jensen, D. (1980). The principles of physiology. New York: Appleton-Century-Crofts.

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(5), 763-767.

Lehninger, A. L. (1982). Principles of biochemistry. New York: Worth Publishers.

Mahler, D. A., & Loke, J. (1985). The physiology of marathon running. The Physician and Sportsmedicine, 13(1), 85-97.

Mainwood, G. W., & Renaud, J. M. (1985). The effect of acid-base balance on fatigue of skeletal muscle. Canadian Journal of Physiology and Pharmacology, 63, 403-416.

Maron, M. B., Horvath, S. M., & Wilkerson, J. E. (1975). Acute blood biochemical alterations in response to marathon running. European Journal of Applied Physiology, 34, 173-181.

Maron, M. B., Horvath, S. M., Wilkerson, J. E., & Gilner, J. A. (1976). Oxygen uptake measurements during competitive marathon running. Journal of Applied Physiology, 40(5), 836-838.

McGrail, J. C., Bonen, A., & Belcastro, A. N. (1978). Dependence of lactate removal on muscle metabolism in man. European Journal of Applied Physiology, 39, 89-97.

McLellan, T. M., & Skinner, J. S. (1982). Blood lactate removal during active recovery related to the aerobic threshold. International Journal of Sports Medicine, 3, 224-229.

Meyer, R. A., Dudley, G. A., & Terjung, R. L. (1980). Ammonia and IMP in different skeletal muscle fibers after exercise in rats. Journal of Applied Physiology, 49, 1033-1041.

Musch, T. A., Haidet, G. C., Freidman, D. B., Pitetti, K. H., Stray-Gundersen et al. (1984). Distribution of regional blood flow at different percentages of maximal oxygen consumption in the untrained dog. (Abstract). Medicine and Science in Sports and Exercise, 16, 178.

Nagata, A., Muro, M., Moritani, T., & Yoshida, T. (1981). Anaerobic threshold determination by blood lactate and myoelectric signals. Japanese Journal of Physiology, 31, 585-597.

Neary, P. J., MacDougall, J. D., Bachus, R., & Wenger, H. A. (1985). The relationship between lactate and ventilatory thresholds: Coincidental or cause and effect? European Journal of Applied Physiology, 54, 104-108.

Noble, B. J. (1986). Physiology of exercise and sport. St. Louis: Times Mirror/Mosby College Publishing.

Powers, S. K., Dodd, S., & Garner, R. (1984). Precision of ventilatory and gas exchange alterations as a predictor of the anaerobic threshold. European Journal of Applied Physiology, 52, 173-177.

Rhodes, E. C., & McKenzie, D. C. (1984). Predicting marathon time from anaerobic threshold measurements. The Physician and Sportsmedicine, 12 (1), 95-98.

Ribeiro, J. P., Hughes, V., Fielding, R. A., Holden, W., Evans, W., & Knuttgen, H. G. (1986). Metabolic and ventilatory responses to steady state exercise relative to lactate thresholds. European Journal of Applied Physiology, 55, 215-221.

Richter, E. A., & Galbo, H. (1986). High glycogen levels enhance glycogen breakdown in isolated contracting skeletal muscle. Journal of Applied Physiology, 61, 827-831.

Richter, E. A., Ruderman, N. B., & Galbo, H. (1983). Alpha and beta adrenergic effects on muscle metabolism in contracting, perfused muscle. In: Knuttgen et al. (Eds.), Biochemistry of exercise Vol 13, (pp. 766-772). Champaign, Illinois: Human Kinetics Publishers Inc..

Sahlin, K., Henriksson, J., & Juhlin-Dannfelt, A. (1984). Intracellular pH and electrolytes in human skeletal muscle during adrenaline and insulin infusions. Clinical Science, 67, 461-464.

Scheen, A., Juchmes, J., & Cession-Fossion, A. (1981). Critical analysis of the "anaerobic threshold" during exercise at constant work loads. European Journal of Applied Physiology, 46, 367-377.

Schnabel, A., Kindermann, W., Schmitt, W. M., Biro, G., & Stegmann, H. (1982). Hormonal and metabolic consequences of prolonged running at the individual anaerobic threshold. International Journal of Sports Medicine, 3, 163-168.

Sienko, M. J., & Plane, R. A. (1979). Chemistry principles and applications. New York: McGraw-Hill.

Simon, J., Young, J. L., Gutin, B., Blood, D. K., & Case, R. B. (1983). Lactate accumulation relative to the anaerobic and respiratory compensation thresholds. Journal of Applied Physiology, 54(1), 13-17.

Sjodin, B., & Jacobs, I. (1981). Onset of blood lactate accumulation and marathon performance. International Journal of Sports Medicine, 2(1), 23-26.

Sjodin, B., & Schele, R. (1982). Oxygen cost of treadmill running in long distance runners. In: Komi (Ed.), Exercise and sport biology (pp. 61-67). Champaign, Illinois: Human Kinetics Publishers.

Sjodin, B., & Svedenhag, J. (1985). Applied physiology of marathon running. Sports Medicine, 2, 83-99.

Skinner, J. S., & McLellan, T. H. (1980). The transition from aerobic to anaerobic metabolism. Research Quarterly for Exercise and Sport, 51(1), 234-248.

Stamford, B. A., Weltman, A., & Fulco, C. (1978). Anaerobic threshold and cardiovascular responses during one- versus two-legged cycling. Research Quarterly, 49, 351-362.

Stamford, B. A., Weltman, A., Moffatt, R., & Sady, S. (1981). Exercise recovery above and below anaerobic threshold following maximal work. Journal of Applied Physiology, 51(4), 840-844.

Stegmann, H., & Kindermann, W. (1982). Comparison of prolonged exercise tests at the individual anaerobic threshold and the fixed anaerobic threshold of 4 mmol$\times$L^{-1} lactate. International Journal of Sports Medicine, 3, 105-110.

Stegmann, H., Kindermann, W., & Schnabel, A. (1981). Lactate kinetics and individual anaerobic threshold. International Journal of Sports Medicine, 2 (3), 160-165.

Streinrauf, L. K., Schulz, A., Ryder, K., Brunn, P., & Cook, D. (1987). Detection of the lactic acid threshold. Indiana Medicine, 80(6), 545-548.

Tanaka, K., & Matsuura, Y. (1984). Marathon performance, anaerobic threshold, and onset of blood lactate accumulation. Journal of Applied Physiology, 57 (3), 640-643.

Tesch, P. (1980). Muscle fatigue in man. Acta Physiol Scand, S 48, 403-416.

Tesch, P. A., Daniels, W. L., & Sharp, D. S. (1982). Lactate accumulation in muscle and blood during submaximal exercise. Acta Physiol Scand, 114, 441-446.

Tibbits, G. F. (1987). Cellular adaptation of skeletal muscle to prolonged work. Canadian Journal of Sports Sciences, 12(Suppl.1), 26S-32S.

Walsh, M. L., & Banister, E. W. (1988). Possible mechanisms of the anaerobic threshold. Sports Medicine, 5, 269-302.

Wasserman, K., & McIlroy, M. B. (1964). Detecting the threshold of anaerobic metabolism in cardiac patients during exercise. The American Journal of Cardiology, 14, 844-852.

Weltman, A., Stamford, B. A., & Fulco, C. (1979). Recovery from maximal effort exercise: Lactate disappearance and subsequent performance. Journal of Applied Physiology, 47(4), 677-682.

Williams, C., & Nute, M. L. G. (1983). Some physiological demands of a half-marathon race on recreational runners. British Journal of Sports Medicine, 17, 152-161.

Winder, W. W., Terjung, R. L., Baldwin, K. M., & Holloszy, J. O. (1974). Effect of exercise on AMP deaminase and adenylosuccinase in rat skeletal muscle. American Journal of Physiology, 22, 11411-1414.

Yeh, M. P., Gardner, R. M., Adams, T. D., Yantowitz, F. G., & Crapo, R. O. (1983). "Anaerobic threshold": Problems of determination and validation. Journal of Applied Physiology, 55(4), 1178-1186.

Yoshida, T. (1984). Effect of exercise duration during incremental exercise on the determination of anaerobic threshold and the onset of blood lactate accumulation. European Journal of Applied Physiology, 53, 196-199.

Zubay, G. (1983). Biochemistry. Don Mills, Ontario: Addison-Wesley.

Appendix A

INFORMED CONSENT FORM

INFORMED CONSENT FORM

For Research on Recovery Exercise Intensities in Cycling

When a research project that studies individuals is undertaken by a member of the University of Ottawa, the Ethics Committee of the University requires the written consent of the participants. The intention is to ensure that the project is adequately explained and that all potential risks and/or benefits are mentioned before the subject agrees to participate.

This research project is being conducted by Sylvia Weihrer, a graduate student in the Department of Kinanthropology at the University of Ottawa. The project is being supervised by Dr. Jim Thoden who is a faculty member in the same department. The project investigates the effect of different intensities of cycling on the change in blood lactate concentration following two minutes of cycling at a maximal intensity in male endurance cyclists between the ages of 20 and 30. Seven cycling tests will be performed by each cyclist over a 10 to 20 day period. No more than one cycling test will be performed on any day.

The first cycling test will be conducted to determine the maximal aerobic power (1) and to estimate the lactate threshold (2) exercise intensity of each cyclist. A minimum of three cycling tests, involving 25 minutes of submaximal intensity activity, will subsequently be performed to verify the lactate threshold exercise intensity. The final three cycling tests will involve two minutes of cycling at maximal aerobic power, which will immediately be followed by 20 minutes of

cycling at a submaximal intensity.

During the cycling tests, expired gas will be collected continuously from a mouthpiece through which the cyclist breathes; therefore, the cyclist will be breathing into a two-way, non rebreathing valve which simply separates inspired from expired air without interfering with respiration. In addition, approximately 30 microlitres of blood will be sampled at two minute intervals from a fingertip throughout each cycling test (total = 10 to 12 samples), and heart rates will be monitored continuously using a Sport Tester PE3000 heart rate monitor.

Following a cycling test, the fingertip(s) which were punctured for blood sampling may be tender. The tenderness may persist for one or two days. Any other physical discomfort that may be experienced either during or after a cycling test will be that usually experienced during cycling at maximal and submaximal intensities. The subjects are requested to seek advice from their physician (or Campus Medical Services) on their intention to enter into maximal aerobic power testing (also referred to as maximal oxygen consumption testing). Campus Medical Services are aware of the conduct and nature of such testing in our laboratories.

Following completion of all cycling tests, subjects will receive a copy of their test results, and counselling, with respect to the results, will be provided upon request.

Further information concerning this research project can be acquired by contacting Sylvia Weihrer at 998-2227 during the day.

I, ______________________________, willingly agree to participate in this study. I understand that it is possible at any time to end my involvement. I understand that my results will remain strictly confidential and that I will not be identifiable in the presentation of the results of the study.

______________________	______________________	____________
NAME OF PARTICIPANT	SIGNATURE OF PARTICIPANT	DATE
______________________	______________________	____________
NAME OF PARTICIPANT	SIGNATURE OF PARTICIPANT	DATE

1 Maximal aerobic power is defined as the maximal amount of oxygen that can be taken up by the body, from room air, while performing progressive exercise that continues until the subject can no longer maintain the work rate.

2 The lactate threshold exercise intensity is defined as the highest intensity of exercise that can be continued for periods longer than 8 to 10 minutes without generating a progressive increase in blood lactate (the dissociated form of lactic acid). The lactate threshold exercise intensity is the intensity of exercise normally chosen for performance of endurance events.

Appendix B

LT-VO_2MAX TEST RESULTS

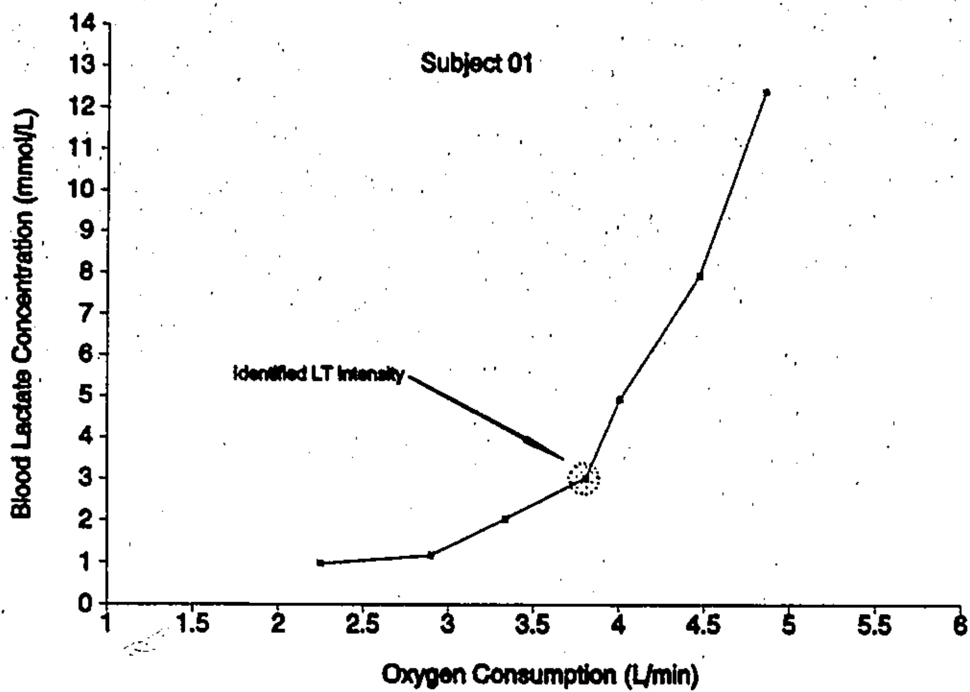


Figure B-1. Relationship between oxygen consumption and blood lactate concentration during each stage of the LT-VO_2max test for subject 01.

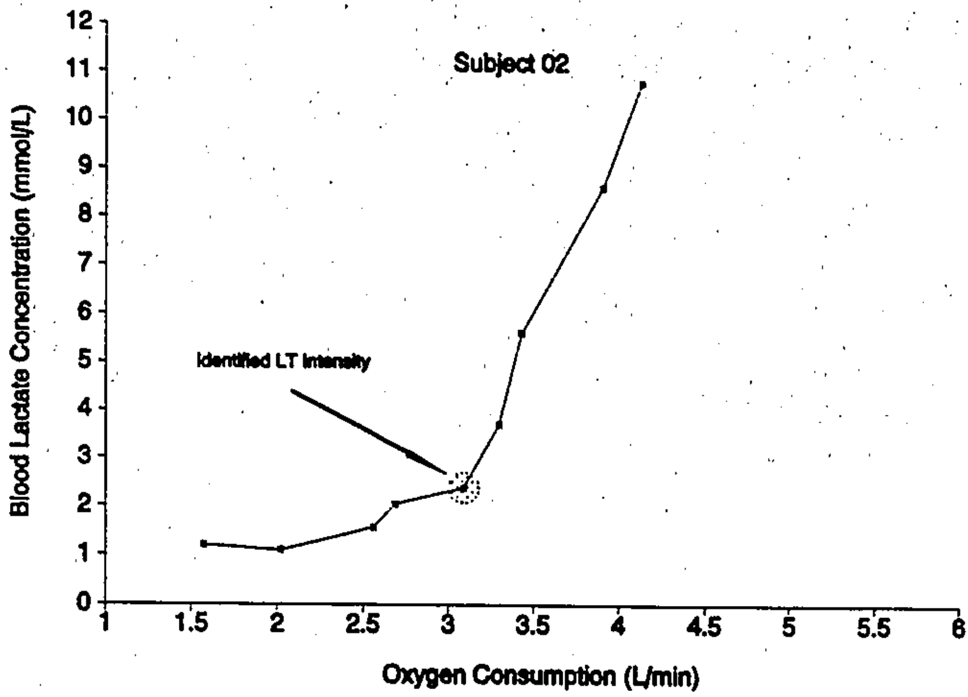


Figure B-2. Relationship between oxygen consumption and blood lactate concentration during each stage of the LT-VO_2max test for subject 02.

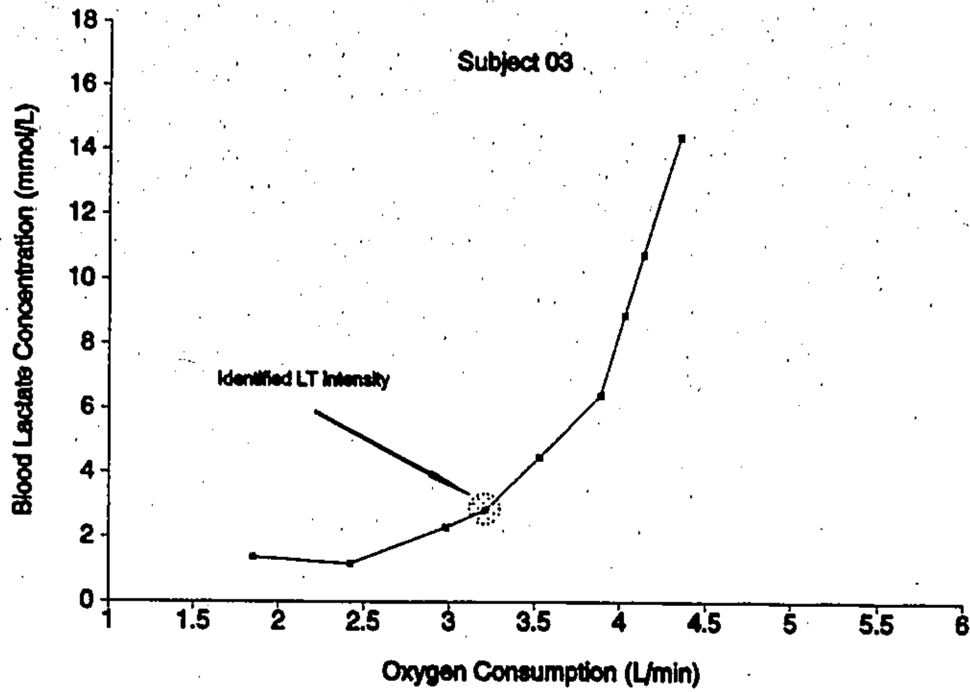


Figure B-3. Relationship between oxygen consumption and blood lactate concentration during each stage of the LT-VO_2max test for subject 03.

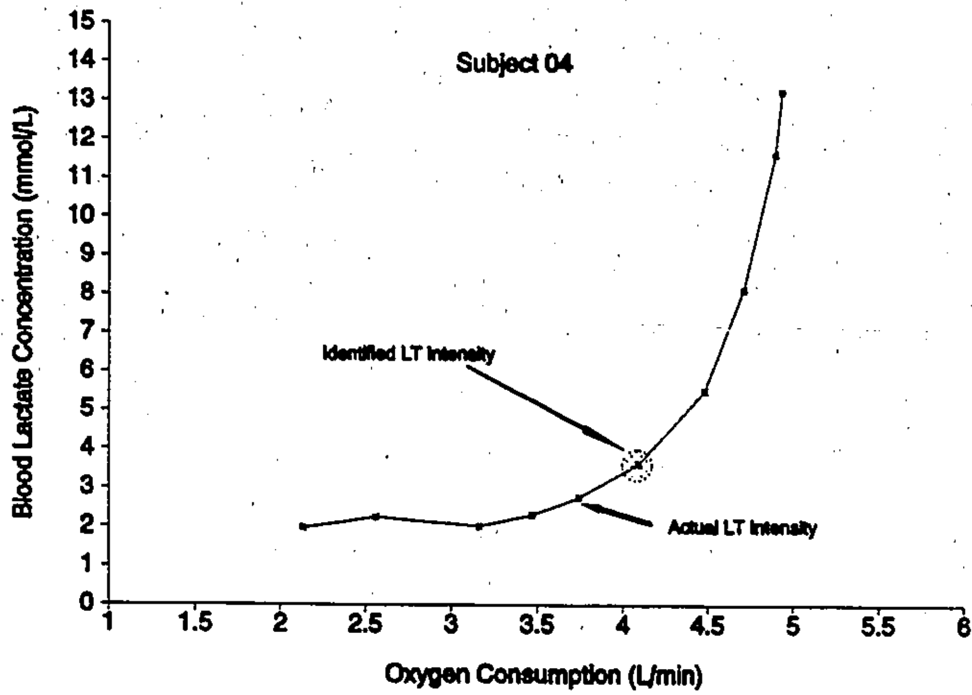


Figure B-4. Relationship between oxygen consumption and blood lactate concentration during each stage of the LT-$\dot{V}O_2$max test for subject 04.

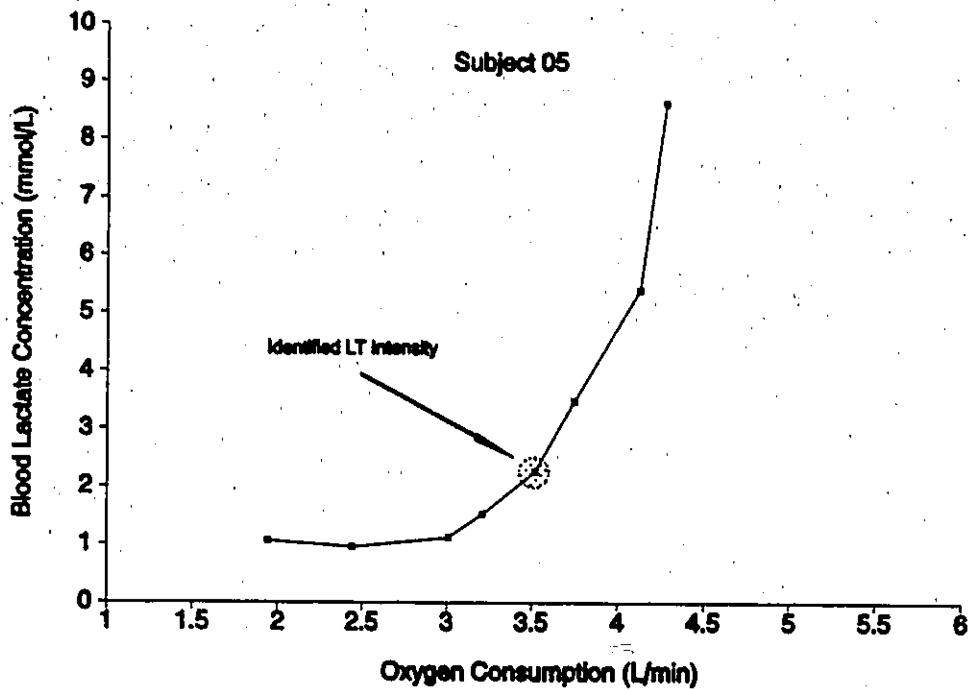


Figure B-5. Relationship between oxygen consumption and blood lactate concentration during each stage of the LT-VO_2max test for subject 05.

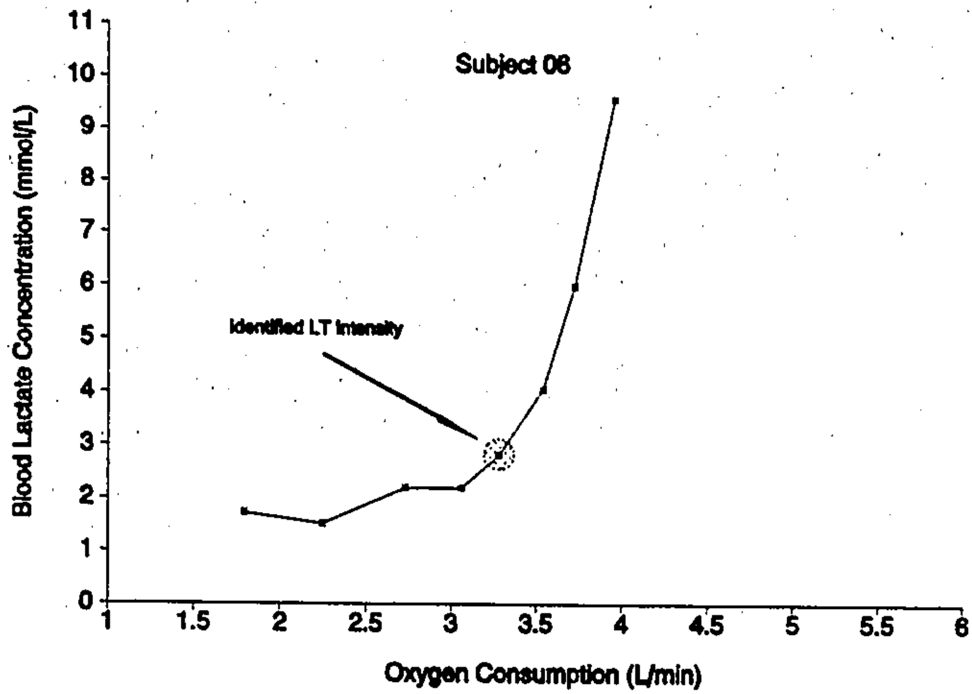


Figure B-6. Relationship between oxygen consumption and blood lactate concentration during each stage of the LT-VO_2max test for subject 06.

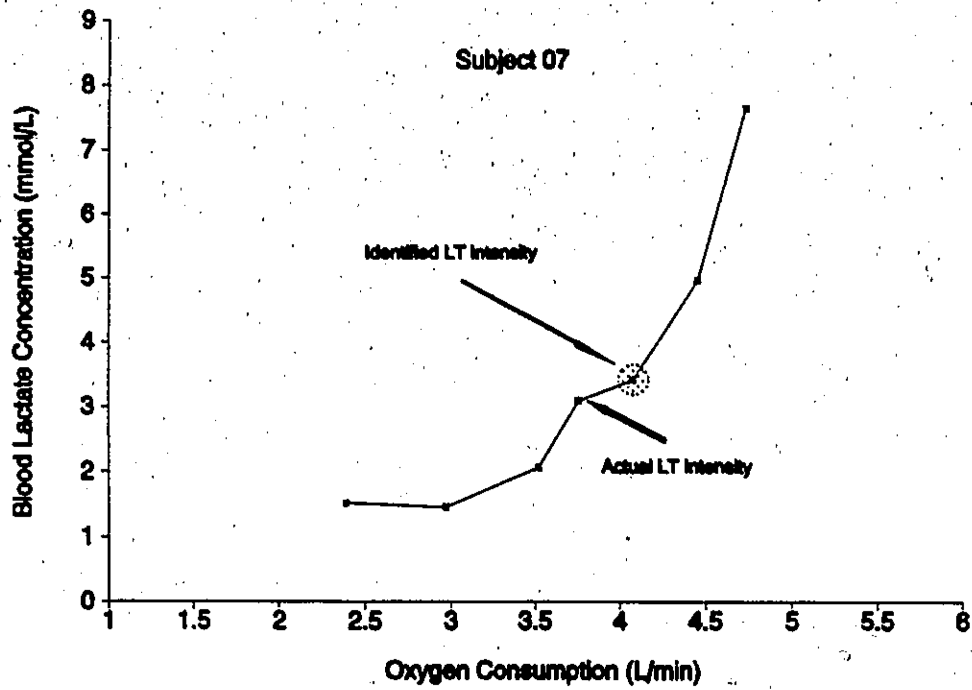


Figure B-7. Relationship between oxygen consumption and blood lactate concentration during each stage of the LT-VO_2max test for subject 07.

Appendix C

POLYNOMIAL EQUATIONS FOR HALF-DECREASE TIMES

POLYNOMIAL EQUATIONS FOR HALF-DECREASE TIMES

Standard Polynomial Format

The following polynomial was the standard format of those derived from the blood lactate concentration versus time recovery curves to estimate the half-times for blood lactate concentration decrease (refer to page 83 in Chapter III):

$$y = a_0 + a_1x + a_2x^2 + a_3x^3 + a_4x^4$$

where:

y = half blood lactate concentration (BLC),

i.e. y = peak BLC - ((peak BLC - baseline BLC) ÷ 2)), and

x = time for half decrease in BLC (half-time).

The polynomial equations for each subject and each recovery intensity will be presented in this Appendix. The correlation coefficient for each polynomial (r) will also be presented.

Subject 01

LT-7%.

Polynomial: $y = 9.675 - 1.381x + 0.101x^2 - 0.004x^3$; $r = 0.999$

$y = 7.18 - ((7.18 - 2.24) \div 2)$

$y = 4.71$

Solving for x:

$x = 5.20$

@LT.

Polynomial: $y = 10.527 - 1.483x + 0.160x^2 - 0.012x^3 + 0.001x^4$;

$r = 0.999$

$y = 8.16 - ((8.16 - 3.42) \div 2)$

$y = 5.79$

Solving for x:

$x = 5.25$

LT+7%.

Polynomial: $y = 11.084 - 0.813x + 0.150x^2 - 0.018x^3 + 0.001x^4$;

$r = 0.998$

$y = 9.92 - ((9.92 - 3.42) \div 2)$

$y = 6.67$

Solving for x:

$x = \infty$

Subject 02

LT-7%.

Polynomial: $y = 9.428 - 0.092x - 0.228x^2 + 0.029x^3 - 0.001x^4$;

$r = 1.0$

$y = 9.14 - ((9.14 - 1.39) + 2)$

$y = 5.27$

Solving for x:

$x = 6.13$

@LT.

Polynomial: $y = 8.508 - 1.201x + 0.063x^2 + 0.001x^3$; $r = 0.995$

$y = 7.26 - ((7.26 - 3.82) + 2)$

$y = 5.54$

Solving for x:

$x = 2.95$

LT+7%.

Polynomial: $y = 6.154 + 0.256x - 0.228x^2 + 0.030x^3 - 0.002x^4$;

$r = 0.999$

$y = 6.14 - ((6.14 - 3.82) + 2)$

$y = 4.98$

Solving for x:

$x = 4.21$

Subject 03

LT-7%.

Polynomial: $y = 8.475 - 0.614x - 0.052x^2 + 0.008x^3$; $r = 0.999$

$y = 7.70 - ((7.70 - 1.25) \div 2)$

$y = 4.48$

Solving for x:

$x = 5.59$

@LT.

Polynomial: $y = 11.134 - 1.473x + 0.141x^2 - 0.010x^3$; $r = 1.0$

$y = 8.64 - ((8.64 - 2.85) \div 2)$

$y = 5.75$

Solving for x:

$x = 5.97$

LT+7%.

Polynomial: $y = 8.630 - 0.181x + 0.026x^2 - 0.001x^3$; $r = 0.998$

$y = 8.34 - ((8.34 - 2.85) \div 2)$

$y = 5.60$

Solving for x:

$x = 6.29$

Subject 04

LT-7%.

Polynomial: $y = 5.331 + 0.232x - 0.276x^2 + 0.036x^3 - 0.002x^4;$

$r = 0.998$

$y = 5.22 - ((5.22 - 1.44) \div 2)$

$y = 3.33$

Solving for x:

$x = 4.58$

@LT.

Polynomial: $y = 5.734 + 0.343x - 0.241x^2 + 0.028x^3 - 0.001x^4;$

$r = 0.999$

$y = 5.80 - ((5.80 - 2.82) \div 2)$

$y = 4.31$

Solving for x:

$x = 4.81$

LT+7%.

Polynomial: $y = 8.470 - 1.451x + 0.189x^2 - 0.017x^3 + 0.001x^4;$

$r = 1.0$

$y = 6.42 - ((6.42 - 2.82) \div 2)$

$y = 4.62$

Solving for x:

$x = 4.31$

Subject 05

LT-7%.

Polynomial: $y = 9.720 - 1.448x + 0.091x^2 - 0.002x^3$; $r = 0.998$

$y = 8.24 - ((8.24 - 1.50) \div 2)$

$y = 4.87$

Solving for x:

$x = 4.46$

@LT.

Polynomial: $y = 9.622 - 0.749x - 0.025x^2 + 0.008x^3$; $r = 0.999$

$y = 8.80 - ((8.80 - 2.80) \div 2)$

$y = 5.80$

Solving for x:

$x = 5.28$

LT+7%.

Polynomial: $y = 9.484 - 0.360x - 0.069x^2 + 0.012x^3 - 0.001x^4$;

$r = 0.999$

$y = 9.00 - ((9.00 - 2.80) \div 2)$

$y = 5.90$

Solving for x:

$x = 8.04$

Subject 06

LT-7%.

Polynomial: $y = 8.786 - 0.041x - 0.263x^2 + 0.035x^3 - 0.002x^4$;

$r = 0.999$

$y = 8.52 - ((8.52 - 1.48) \div 2)$

$y = 5.00$

Solving for x:

$x = 5.52$

@LT.

Polynomial: $y = 8.543 - 0.059x - 0.197x^2 + 0.027x^3 - 0.001x^4$;

$r = 1.0$

$y = 8.34 - ((8.34 - 4.14) \div 2)$

$y = 6.24$

Solving for x:

$x = 4.53$

LT+7%.

Polynomial: $y = 8.317 + 0.020x - 0.155x^2 + 0.022x^3 - 0.001x^4$;

$r = 0.999$

$y = 8.20 - ((8.20 - 4.14) \div 2)$

$y = 6.17$

Solving for x:

$x = 6.34$

Subject 07

LT-7%.

Polynomial: $y = 7.576 - 0.741x + 0.059x^2 - 0.007x^3$; $r = 0.999$

$y = 6.96 - ((6.96 - 1.27) + 2)$

$y = 4.12$

Solving for x:

$x = 6.46$

@LT.

Polynomial: $y = 7.139 - 0.361x - 0.059x^2 + 0.008x^3$; $r = 1.0$

$y = 6.70 - ((6.70 - 2.26) + 2)$

$y = 4.48$

Solving for x:

$x = 5.32$

LT+7%.

Polynomial: $y = 6.792 - 0.633x + 0.045x^2 - 0.003x^3$; $r = 1.0$

$y = 6.18 - ((6.18 - 2.26) + 2)$

$y = 4.22$

Solving for x:

$x = 5.75$