THE RELATIONSHIP BETWEEN AN INCREASED AEROBIC POWER AND THE EXCESS POST EXERCISE OXYGEN CONSUMPTION

by

EDWARD W. CANNON

BPE, University of British Columbia, 1991

A THESIS SUBMITTED IN PARTIAL FULFILLMENT OF

THE REQUIREMENTS FOR THE DEGREE

MASTER OF HUMAN KINETICS

in

THE FACULTY OF GRADUATE STUDIES

School of Human Kinetics

We accept this thesis as conforming to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

September, 1997

In presenting this thesis in partial fulfilment of the requirements for an advanced degree at the University of British Columbia, I agree that the Library shall make it freely available for reference and study. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by the head of my department or by his or her representatives. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Department of HUMAN KINETICS

The University of British Columbia
Vancouver, Canada

Date OCT. 15/97

DE-6 (2/88)

ABSTRACT

As a result of aerobic training, the rate and magnitude of the recovery VO_2 following submaximal exercise at the same absolute workloads is decreased (Hagberg et al., 1980). To date there has been little research associated with the effects of an increased aerobic power on the recovery VO_2 following supramaximal exercise. The purpose of this study was to determine the effects of an increased aerobic power on the excess post exercise oxygen consumption (EPOC) after a supramaximal exercise test. A secondary purpose was to determine the relationships between the increased aerobic power and the recovery VO_2 rate and magnitude.

Ten untrained males participated in a six week training study. The subjects performed pre and post training VO_{2max} tests and Anaerobic Speed Tests (ASTs). EPOC volume and EPOC rate components (τ_1 and τ_2) as well as post exercise blood lactate response were measured following a 2 minAST. Significant differences were evident between pre and post training relative and absolute VO_{2max} scores (46.38 ± 3.74 $ml \cdot kg^{-1} \cdot min^{-1}$ vs. 51.82 ± 5.21 $ml \cdot kg^{-1} \cdot min^{-1}$ and 3.61 ± 0.42 $L \cdot min^{-1}$ vs. 4.00 ± 0.44 $L \cdot min^{-1}$; $p<0.05$). EPOC volume was significantly decreased following the endurance training program (9.13 ± 1.68 L vs. 7.49 ± 1.73 L; $p<0.05$). Significant differences were found between the pre and post training fast VO_2 recovery rate (τ_1) (2.69 ± 0.19 min. vs. 2.29 ± 0.33 min. ; $p<0.05$) and the pre and post training slow VO_2 recovery rate (τ_2) (43.74 ± 5.12 min. vs. 39.63 ± 4.24 min.; $p<0.05$). Post exercise blood lactate response was significantly decreased following the training program (15.28 ± 1.80 $mmol \cdot L^{-1}$ vs. 13.36 ± 1.55 $mmol \cdot L^{-1}$; $p<0.05$). A significant relationship was found between the change in VO_{2max} and the change in blood lactate concentration ($r=0.73$; $p<0.05$). No significant relationships were evident between

VO_{2max}, EPOC volume, or EPOC recovery rates ($p>0.05$). The results of this study indicate that aerobic training can decrease the VO_2 recovery volume and rate, as well as decrease the blood lactate response associated with anaerobic exercise. However, the rate and magnitude of the recovery VO_2 from supramaximal work appear to be independent of VO_{2max}.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ACKNOWLEDGMENTS

I would like to thank my thesis committee members for their guidance and input during the preparation and completion of this project. Special thanks go out to my graduate advisor Dr. Ted Rhodes for sharing his ideas and enthusiasm in the area of exercise physiology and for his support throughout my entire degree.

I would like to thank the subjects for their time and commitment in participating in this training study. As well, I would like to thank those students who volunteered hours of their time in administering and monitoring some of the training and testing.

One last thanks to the former Buchanan Lab technician, Mr. Dusan Benickey. Without his help, this project would not have been possible.

CHAPTER 1

1.0 Introduction

The elevated post exercise oxygen consumption observed in recovery from exercise has been investigated since the early 1900's. Much of the early work focused on lactic acid metabolism and its relationship to the post exercise VO_2. In response to this early research Hill and Lupton (1923) developed the classic "O_2 debt" hypothesis, based on the assumption that the primary fate of lactate in recovery was gluconeogenesis. Eighty percent of the lactate accumulated during exercise was thought to be reconverted to glycogen and 20% oxidized to CO_2 and H_2O.

The "O_2 debt" hypothesis was later modified and more fully developed in 1933 by Margaria et al. The rapid decline in observed post exercise VO_2 was termed the "fast component" or "alactacid" O_2 debt and was thought to represent a replacement of phosphagens in skeletal muscle. The extended VO_2 uptake was termed the "slow component" or "lactacid" O_2 debt and was thought to represent gluconeogenesis of the accumulated lactate.

In 1984 Gaesser and Brooks published a review paper on the O_2 debt. In order that an implied causality relationship did not exist in the terminology of the elevated VO_2 seen in recovery from exercise, they proposed the phenomenon be termed Excess Post Exercise Oxygen Consumption (EPOC). Gaesser and Brooks (1984) determined that post exercise 55-70% of the accumulated lactate was oxidized to CO_2 and H_2O, while less than 20% of the lactate was converted to muscle or liver glycogen. Their research implied that lactate was not so predominantly responsible for EPOC and that the factors affecting EPOC reflect a general metabolic disturbance after exercise.

EPOC and the factors which may influence the recovery oxygen consumption continue to be an important subject of research. Although debate continues regarding the biochemical, physiological, and physical reasons for the elevated post-exercise VO_2, EPOC remains a useful tool in comparing recovery from exercise.

Numerous publications exist that focus on aerobic power and its relationship to energy transfer in the body during exercise. Those individuals with higher aerobic capacities generally perform better on sustained muscular work activities. Research has shown that both central and peripheral aerobic power adaptations occur in response to endurance training activities (Sutton, 1992). As the aerobic energy system represents a supply and recovery system, it is generally accepted that an increased aerobic power allows an individual a faster time course to the adjustment to exercise as well as a faster time course to recovery. Hagberg et al. (1980) reported that a nine week aerobic training program increased the rate of the recovery oxygen consumption during submaximal exercise conditions at both the same absolute and relative work loads after training.

These aerobic training responses are magnified under supramaximal exercise conditions. Theoretically, the adaptive response of the aerobic energy system to supramaximal exercise allows the anaerobic system to function at a given intensity for a longer period of time as well as the ability to recover more quickly from the preceding exercise condition. Inconclusive evidence exists to support this recovery hypothesis.

Shortcomings exist in the literature concerning the relationship of aerobic power to recovery. Brehm and Gutin (1986), Freedman-Akabas et al. (1985), and Chad and Quigley (1991) provide evidence to suggest that there is no relationship between the recovery VO_2 and

maximal aerobic power. However, research by Hagberg et al. (1980) and Elliot et al. (1988), indicates a relationship between the recovery VO_2 and an individual's level of training.

The discrepancies in the literature on the relationship of aerobic power to EPOC may be attributed to different methodologies employed in the research. Those studies that have examined the relationship of aerobic power to recovery have looked at the speed of recovery in relation to the magnitude and duration of EPOC while few studies have analyzed the rate of the recovery VO_2. As well, these studies have analyzed the relationship of aerobic power to recovery under submaximal exercise conditions. A research design using an analysis of the rate of the recovery VO_2 from supramaximal exercise conditions in relation to an increased aerobic power may give greater insight into the effects of endurance training on the excess post exercise oxygen consumption.

1.1 Statement of the Problem

The purpose of this study was to determine if an increased aerobic power is related to the rate and magnitude of recovery of the excess post exercise oxygen consumption (EPOC) under supramaximal exercise conditions.

1.2 Primary Hypotheses

1. A six week aerobic training program will increase both absolute and relative aerobic power (VO_{2max}).

a.) VO_{2max} ($L \cdot min^{-1}$) (pre-training) < VO_{2max} ($L \cdot min^{-1}$) (post-training) $(p < 0.05)$

b.) VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) (pre-training) < VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) (post-training) $(p < 0.05)$

Rationale:

It is well documented that endurance training will increase aerobic power. The amount of training improvements depends on one's initial fitness level and the intensity of the training program (McArdle et al., 1991). Individuals with low initial fitness levels have a greater capacity for improvement than individuals with high initial fitness levels. Generally, an increase in aerobic power of 5-25% is expected from systematic endurance training with significant training improvements occurring in the first three weeks (Hickson et al., 1981). Therefore, it is hypothesized that a six week aerobic training program will bring about an increased aerobic power in the relatively untrained subjects recruited for this study.

2. The six week aerobic training program will affect recovery from supramaximal exercise in the following ways:

a.) increase the rates of recovery (τ_1 and τ_2) for the same absolute supramaximal workload administered from pre to post training.

i.) τ_1 (pre-training) > τ_1 (post training) ($p < 0.05$)

ii.) τ_2 (pre-training) > τ_2 (post training) ($p < 0.05$)

Rationale:

Early work by Margaria et al. (1933) showed the rate constant for the initial component of recovery oxygen consumption to be greater than the rate constant for the second component of the recovery VO_2. These observations are mathematically correct when VO_2 recovery curves are expressed in the exponential form:

$$VO_2 = A_1 e^{-k_1 t} + A_2 e^{-k_2 t}$$

The constants (k_1 and k_2) then satisfy the two component recovery VO_2 model with an associated initial fast phase of recovery VO_2 and a second slow phase of recovery VO_2. In the present study the VO_2 recovery curves will mathematically be expressed as:

$$VO_2 = A_1 e^{-t/\tau_1} + A_2 e^{-t/\tau_2}$$

The rate constants (τ_1 and τ_2) are reciprocals of the rate constants (k_1 and k_2) initially used by Margaria et al. (1933). It is therefore hypothesized that the rate constant for the initial component of the recovery VO_2 (τ_1) will be less than the rate constant for the second component of the recovery VO_2 (τ_2).

Berg (1947) recognized that physically trained individuals had a faster time course of recovery oxygen consumption from submaximal exercise tests. Associated with these faster recovery times, larger post training recovery rate constants (expressed as k_1 and k_2) were observed in trained than in untrained individuals. Hagberg et al. (1980) showed a decreased time to recovery at both the same absolute and relative workloads after training. These results were later supported in work by Frey et al. (1993) and Elliot et al. (1988). As the aerobic energy system represents both a supply and recovery system, it is hypothesized that the rate of recovery oxygen consumption from a supramaximal exercise test will decrease after the training program. In the present study, the exponential equations and rate constants will be expressed in the form:

$$y = Ae^{-t/\tau}$$

A decrease in the recovery rate constants should be observed from pre to post test measures.

b.) decrease the volume of post exercise oxygen consumption (EPOC) for the same absolute supramaximal workload administered from pre to post training.

EPOC (pre-training) > EPOC (post-training) ($p < 0.05$)

Rationale:

Research has shown total EPOC volume at a set absolute work rate to decrease following training (Hagberg et al., 1980; Girondola and Katch, 1973). In fact, Hagberg et al. (1980) recognized EPOC to be smaller at the same submaximal relative work rates following a nine week aerobic training program. In the present study, it is expected that a set absolute supramaximal workload administered after an aerobic training program will cause less of a homeostatic disturbance to the body resulting in a smaller EPOC. Therefore, it is hypothesized that the post-training EPOC will be significantly smaller in magnitude than the pre-training EPOC.

c.) decrease the blood lactate concentration ([BLa]) for the same absolute supramaximal workload administered from pre to post training.

[BLa] (pre-training) > [BLa] (post-training) ($p < 0.05$)

Rationale:

Specific adaptations take place during training which favor the production of less lactic acid (Holloszy and Coyle, 1984) or a more rapid rate of its removal at a given exercise intesity (MacRae et al., 1992). In the present study, it is therefore hypothesized that the post training blood lactate concentration will be significantly lower than the pre-training blood lactate concentration.

1.2.1 Secondary Hypotheses

1. There will be significant negative relationships between the relative VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) and the VO_2 rate constants (τ_1 and τ_2).

a.) Pre-Training:

i.) VO_{2max} and τ_1

ii.) VO_{2max} and τ_2

b.) Post-Training:

i.) VO_{2max} and τ_1

ii.) VO_{2max} and τ_2

Rationale:

Berg (1947) indicated that the time course of recovery VO_2 was shorter in the trained than in the untrained state. As well, the shorter time course of recovery was associated with an increased recovery rate in the trained state. Hagberg et al. (1980) showed an aerobic training program increased the rate of the recovery oxygen consumption at both the same absolute and relative work loads after training. Based on our double exponential O_2 recovery curve equation:

$$VO_2 = A_1 e^{-t/\tau_1} + A_2 e^{-t/\tau_2}$$

A faster time to recovery would be associated with smaller recovery rate constants (τ_1 and τ_2) and a slower time to recovery associated with larger recovery rate constants. In the present study, it is therefore hypothesized that significant negative relationships will exist between the pre-training VO_{2max} and the pre-training rate constants τ_1 and τ_2. As well, significant negative relationships will exist between the post-training VO_{2max} and the post-training rate constants τ_1 and τ_2.

2. There will be significant positive relationships between the change in VO_{2max} and the changes in recovery variables from pre to post trainining.

a.) Δ VO_{2max} and Δ EPOC volume.

b.) Δ VO_{2max} and Δ τ_1

c.) Δ VO_{2max} and Δ τ_2

d.) Δ VO_{2max} and Δ [BLa]

Rationale:

Early research suggests that the time course of recovery VO_2 from submaximal exercise is shorter in the trained than in the untrained state (Berg, 1947). These observations have been supported in more recent research on recovery curve analysis (Frey et al., 1993; Elliot et al., 1988; Hagberg et al., 1980). Research has also shown training to decrease EPOC volume (Hagberg et al., 1980; Girondola and Katch, 1973) and blood lactate concentration (MacRae, et al., 1992). In the present study, it is expected that the subjects with the greatest change in aerobic power between pre and post training will show the greatest change in EPOC volume, change in recovery rates (τ_1 and τ_2) and change in blood lactate concentration ([BLa]).Therefore, it is hypothesized that there will be positive correlations between the change in VO_{2max} and the changes in the measured recovery variables.

1.3 Significance of the Study

Common to all intermittent sports are the physical requirements demanded of players to perform various intensities of exercise over a prolonged period of time. Players may be required to perform a number of short, fast bursts of running interspersed with periods of jogging and walking. These variations in the intensity of activities require the athletes to integrate and utilize

both aerobic and anaerobic energy supply systems. A faster VO_2 rate of recovery under these varying exercise conditions would be beneficial to subsequent physical performance.

1.4 Delimitations

This study is delimited to:

1. the endurance training program applied to elicit a change in aerobic power.
2. male HKIN undergraduate students between the ages of 19-25 years of age.
3. the methodology and instruments utilized in determining and producing VO_{2max}, EPOC, τ_1, τ_2, and blood lactate concentrations.

1.5 Limitations

This study is limited by:

1. the subjects adherence to the training program.
2. subjects completing the six week training program.
3. the present understanding of EPOC.
4. the interpretation of recovery variables.
5. the perception of fatigue experienced by each subject.

1.6 Definitions

AST: a graded treadmill run to exhaustion.

Supramaximal Exercise: exercise intensity which requires a rate of energy release exceeding the maximal O_2 uptake (Medbo et al., 1988).

Excess Post Exercise Oxygen Consumption (EPOC): the summed volume of all O_2 derived processes, in excess of resting VO_2 values, that in response to exercise serves to restore metabolic homeostasis in the working muscles and the organs and tissues of the body (Roth et al., 1988).

O_2 Deficit: the difference between the total oxygen actually consumed during exercise and the total that would have been consumed had a steady rate of metabolism been reached immediately at the start of the exercise period (McArdle et al., 1991).

Steady State: a balance between the energy required by the working muscles and the rate of ATP production via aerobic metabolism (McArdle et al., 1991).

Ventilatory Threshold (VT): point (critical intensity) above which the aerobic energy response is of insufficient magnitude to supply the energy demanded, and there is an increased reliance on anaerobic processes with an accompanying accumulation of metabolic byproducts.

Excess CO_2 ($ExCO_2$): non metabolic CO_2 formed as a result of the hydrogen ions of lactic acid being buffered by bicarbonate. Used as a marker of anaerobic metabolism. The calculation of excess CO_2 will be based on the formula of Volkov et al. (1975) where:

$$ExCO_2 = VCO_2 - (RQ_{rest} - VO_2)$$

CHAPTER 2

2.0 Review of Literature

2.1 Historical Review - Recovery Oxygen Consumption/Developing a Mathematical Model

The analysis of recovery oxygen curves after physical activity dates back to the early 1920's. Hill and Lupton (1923) were the first to recognize that post exercise recovery curves for oxygen consumption were exponential in nature. Analysis by Margaria et al. (1933) and Margaria and Edwards (1934 a, b) led to a modification and explanation of the VO_2 recovery curve as a two component model. These researchers recognized that post exercise oxygen recovery was composed of two exponential curves, an initial rapid component lasting a few minutes, and a longer component of several hours duration depending upon the preceding exercise intensity. Margaria et al. (1933) termed the fast phase the "alactacid" component and the slow phase the "lactacid" component. They hypothesized that the fast phase represented a replacement of phosphagens in skeletal muscle and the slow phase represented gluconeogenesis of accumulated lactate.

Berg (1947), following the model put forth by Hill and Lupton (1923), applied a single exponential equation to post exercise recovery oxygen consumption and CO_2 production after moderate exercise in humans. Berg's model, however, was unable to fully explain his results.

Henry and DeMoor (1950) continued research into the application of a mathematical model to explain the post exercise recovery VO_2. These researchers applied double exponential equations to the oxygen recovery curves, allowing the curves to be separated into their fast (alactacid) and slow (lactacid) components (Margaria et al. 1933). Henry and DeMoor's

mathematical model fit the recovery curves extremely well and explained the two component recovery oxygen curves that were recognized by Margaria et al. (1933).

Henry and DeMoor (1956) later investigated the application of their mathematical model to explain recovery oxygen curves under varying exercise conditions. They found that increased work intensity on a bicycle ergometer produced a larger O_2 debt and recovery lag. The double exponential model accurately described the recovery curves and indicated an increasing lactacid component with increasing exercise intensity.

The two component oxygen recovery model (fast and slow) (Margaria et al., 1933) mathematically expressed as a double exponential equation (Henry and DeMoor, 1950) is the base behind current recovery curve analysis.

2.2 Mechanisms Governing Oxygen Consumption Curve Components

Following the development of a mathematical model to explain recovery oxygen curves, research into the mechanisms underlying the curve components was undertaken. Early work on the elevated recovery oxygen consumption focused on lactic acid metabolism and its relationship to the post exercise VO_2. From this work, Hill and Lupton (1923) formulated the classical "O_2 debt" hypothesis explaining the excess post exercise oxygen consumption. This O_2 debt hypothesis served as an accepted term to describe the post exercise metabolism phenomenon until many years later when Gaesser and Brooks (1984) introduced the term "excess post exercise oxygen consumption" (EPOC). In this literature review the O_2 debt will be used to describe the post exercise recovery VO_2 in studies that took place prior to 1984. In studies after 1984, the elevated post exercise metabolism will be referred to as EPOC.

In three separate articles, Huckabee (1958 a,b,c) concluded that "Excess Lactate" produced during exercise showed the same time patterns as the oxygen recovery curve and thus could be used as a predictor for total O_2 debt. Huckabee proposed that O_2 debt is directly related to excess lactate production. This theory challenged previous research at the time which indicated that an alactic phase of recovery oxygen consumption existed, to which other mechanisms were accountable besides a lactate $\longrightarrow$ pyruvate mechanism.

Further research served to contradict Huckabee's findings. Knuttgen (1962), and Margaria et al. (1963) observed that venous lactate increase was not evident until a "critical work intensity" was attained. As work intensity surpassed this "threshold intensity", a rapid increase in the O_2 debt was evident.

Knuttgen (1962) made use of four different work rates on a bicycle ergometer ranging from 300-1600 kg-m/min. At the lower work rates, the resultant lactate or excess lactate was unable to predict the O_2 debt. As work intensity reached a "critical level" a rapid increase in O_2 debt as well as lactate and excess lactate was observed. Although a temporal relationship was observed between recovery curves for O_2 debt, excess lactate, and lactate, the findings failed to provide evidence of a causal relationship. The accumulated O_2 debt was greater than the theoretical O_2 equivalents of the maximum increase in not only excess lactate but also the change in lactate. The findings supported the two component model of recovery oxygen consumption.

In 1963, Margaria et al. provided further evidence to support a two component recovery oxygen curve model. In support of Knuttgen's research, Margaria and associates also observed that an increase in venous lactate did not occur until a "critical work intensity" had been attained.

At lower work levels an O_2 debt was still evident, though venous lactate concentration remained close to resting values.

In direct support of the findings of Knuttgen (1962) and Margaria et al. (1963), Wasserman and McIllroy (1964) and Wasserman et al. (1964) introduced the concept of the "Anaerobic Threshold". The anaerobic threshold was thought to represent a transition point in energy metabolism above which endurance performance capacity became severely limited. The anaerobic threshold was explained as a maximal balance between lactate production and elimination during continuous exercise. They suggested that an increase in arterial blood lactate does not occur until exercise reaches a specific work load intensity. Wasserman et al. (1965) recognized that at low work intensities lactate levels remained at or near rest values and therefore could not explain the elevated VO_2 associated with recovery from sub-threshold exercise.

In response to the discrepancies in the literature on excess lactate and O_2 debt relationships, Thomas et al. (1965) attempted to reproduce Huckabee's original investigation. Thomas was unsuccessful in finding a correlation between excess lactate and O_2 debt thus providing yet more evidence to discredit the excess lactate and O_2 debt relationship.

As the simplistic excess lactate/O_2 debt relationship proved to be misleading, researchers began to investigate the mechanisms governing each component of the oxygen recovery curves.

In the first of two papers, Piiper et al. (1968) investigated the relationship of the O_2 debt to high energy phosphate depletion in the gastrocnemius muscle of the dog. During in vitro muscle stimulation a decreased concentration of CP and oxygenated myoglobin was observed. The researchers proposed that the O_2 deficit incurred at the beginning of exercise was associated with a decrease in concentration of high energy phosphates (ATP and CP), particularly CP. They

suggested that the fast component of the recovery VO_2 after exercise was responsible for the homeostatic return of these high energy phosphates to baseline.

DiPrampero and Margaria (1968) analyzed ATP, ADP and CP concentrations following contractions of in vivo dog gastrocnemius muscle. These researchers observed a decrease in CP concentration with increasing exercise intensity while ATP and ADP concentrations remained constant at steady state. They proposed that the steady state ratio of oxygen consumption to the alactic O_2 debt is identified with the speed constant of the resynthesis of phosphagens in skeletal muscle.

Cerretelli et al. (1969) performed further work in the analysis of the mechanisms governing the components of the O_2 debt. Isotonic contractions of dog gastrocnemius muscle preparations were used to determine the energy equivalents of the alactic and lactic components of the O_2 debt in anaerobic work conditions. The results of the experiment confirmed the validity of partitioning the O_2 debt into a fast and a slow component.

Piiper and Spiller (1970) provided further evidence to support the CP resynthesis/alactic repayment hypothesis. The researchers investigated the repayment of O_2 debt and the resynthesis of high energy phosphates in the gastrocnemius muscle of the dog. They recognized resynthesis of CP in recovery from exercise occurred with a time course similar to that of the fast component of O_2 debt repayment, being close to complete in two minutes. The researchers hypothesized that the high-energy phosphates resynthesized in the recovery from exercise could energetically be explained by the fast component of the O_2 debt repaid.

An extensive study by Knuttgen (1970), analyzed the O_2 debt after steady state sub-maximal bicycle ergometer exercise of varying intensities and durations. An in depth analysis into

each recovery curve was undertaken. Factors contributing to the fast component appeared to be relatively independent of metabolic level at the lower work intensities (45%-65% VO_{2max}). After an initial basal rate, an increase in work intensity was accompanied by a linear increase in the fast component, and an exponential increase in the slow component. Interestingly, at a constant workload the duration of work showed a tendency toward lower values for the fast component, and an increase in the slow component. Knuttgen postulated that a partial repayment of the fast component contributing factors may occur during the extended work conditions.

Knuttgen (1970) recognized the lack of a strong relationship between the slow component and blood lactate, concentrations, particularly when the time of exercise was extended. He suggested that the elevated VO_2 in recovery from exercise was attributable to a general disturbance of the body's resting conditions during exercise. Again, these results only added more questions about the mechanisms governing the post exercise VO_2.

Whipp et al. (1970) investigated the efficiency of non-steady state exercise under a constant workload and various exercise durations to the relationship of O_2 debt. The protocol consisted of a constant workload on a cycle ergometer at exercise durations from 1-10 minutes. Whipp and associates suggested that the difference in efficiency between short and long term exercise could be accounted for by a delay in repayment of alactic debt prior to steady state exercise conditions, and elevation in blood lactate concentrations reaching peak values during the second and third minutes of exercise.

Further evidence in support of the CP resynthesis/alactic repayment hypothesis was provided by Harris et al. (1976). The researchers investigated the time course of CP resynthesis in the quadriceps muscle of man during recovery from exhausting dynamic exercise and isometric

contractions sustained to fatigue. The time course of CP resynthesis in recovery was found to be biphasic exhibiting a fast and slow component. These observations supported those made by Piiper and Spiller (1970) in that the kinetics of CP resynthesis during recovery were similar to the O_2 debt repayment.

2.3 Supramaximal Exercise and the Recovery VO_2:

Historically, research associated with recovery oxygen consumption from supramaximal work began to be undertaken (DiPrampero et al., 1973; Freund and Gendry, 1978; Roberts and Morton, 1978). DiPrampero et al. (1973) investigated the total accumulated O_2 debt and the kinetics of its repayment after supra-maximal exercise. The results indicated that the recovery oxygen consumption decreased with a similar trend as that observed after aerobic exercise. The recovery curve could be mathematically described by a double exponential equation. Kinetic analysis indicated a $t_{1/2}$ of 25-30 s for the fast component and a $t_{1/2}$ of between 15-20 min for the slow component of recovery VO_2.

Roberts and Morton (1976) examined both alactic and total O_2 debts following supramaximal treadmill tests to exhaustion. As well, a procedure for the measurement of total alactic debt was evaluated. The researchers recognized the substantial contribution the anaerobic energy system makes to short term, supramaximal activity encountered in many team games and thus the practicality of the measurement of anaerobic capacity. They compared their results with maximal alactic O_2 debt values from the literature and concluded the method developed for the measurement of the alactic portion of the oxygen debt to be both reliable and valid.

Katch (1973) performed one of the more extensive studies on supramaximal work and the recovery VO_2. The researcher investigated the kinetics of oxygen uptake and recovery for supramaximal cycle ergometer work of short duration. Thirty five subjects pedaled a loaded cycle ergometer (5.5 kg resistance) at maximal speed for one minute. Recovery VO_2 was monitored for 15 minutes after the exercise. The net VO_2 uptake was reported as 4.89 L while the calculated half times were 28.8 s for the fast component of recovery and 11.76 min. for the slow component. Comparison of the curve parameters obtained in this study with published data at that time showed large differences for the post exercise oxygen recovery and the slow component of the recovery curve. The kinetics and magnitude of the fast component of recovery, however, were similar to other data available in the literature. The proportion of slow component recovery to total VO_2 recovery magnitude was approximately 69%, thus indicating that the slow component is more responsible for the EPOC magnitude after supramaximal work of short duration.

Recent work associated with high intensity exercise and VO_2 kinetics has shifted to the VO_2 response at the start of exercise (Barstow et al., 1996; Gaesser and Poole, 1996). A systems analysis approach has been undertaken to determine the underlying mechanisms responsible for the rate of VO_2 uptake. Attempts have been made to determine steady state and dynamic linearity in predictive equations for the VO_2 response at the start of exercise. Two schools of thought on the mechanisms responsible for the VO_2 response have emerged from this analysis. Some authors point to O_2 transport as the rate limiting step in VO_2 kinetics (Hughson, 1990), while others suggest that the rate of increase in O_2 utilization is controlled by the biochemical processes of O_2 utilization (Whipp and Mahler, 1980).

More recently, Bell et al. (1997) looked at the relationship between aerobic fitness and the recovery VO_2 from intermittent exercise in endurance trained cyclists. Subjects in this study performed three, one minute cycle tests at 125% VO_{2max} interspersed with five minutes of stationary recovery. Ten minute EPOCs of 4.12 L were reported. The half-time for the fast component of recovery VO_2 was 55.6 s and the half-time for the slow component was 53.8 s. These correspond to τ values of 80.23 sec. for τ_1 and 77.63 sec. for τ_2 (computed from the relationship - $t_{1/2} = \ln 2 * \tau$) . The fact that a faster "slow component" than "fast component" was found in this study is questionable. There is no research to support such a finding. In fact, numerous investigators have demonstrated a significant slow phase of recovery VO_2 kinetics that becomes more prominent the higher the preceding exercise condition (Margaria et al., 1933; Knuttgen, 1962; Davies et al., 1972). The length of time for which EPOC was recorded may provide some answers for the relatively fast slow component. For the purpose of the study, the researchers only monitored recovery VO_2 for ten minutes. The effect of a short recovery time is to decrease the τ_2 constant resulting in an apparent slow component that is smaller (faster) than the true value (Katch, 1973). This still does not account for a faster "slow component" than "fast component" of recovery VO_2.

Bell et al. (1997) do not provide evidence to support a relationship between VO_{2max} and the recovery VO_2. They documented no significant correlations between VO_{2max}, net EPOC, EPOC recovery rates, or blood lactate removal after intermittent high intensity exercise. These authors indicate that the lack of relationships between the measurements of aerobic fitness and metabolic recovery after the high-intensity, intermittent exercise bouts suggests that the

physiological factors underlying the different assessments of aerobic fitness are probably too diverse to be used as indicators for the ability to recover from high-intensity, intermittent exercise.

2.4 Effect of Exercise Intensity and Duration on EPOC

Recent analysis of recovery oxygen consumption has focussed on the relative effects of exercise intensity and duration on total EPOC magnitude. Researchers attempting to control body mass have tried to manipulate and maximize the energy expenditure in recovery from exercise. Many studies have found exercise intensity to be the prime determinant of EPOC magnitude (Hagberg et al., 1980; Bahr et al., 1987; Gore and Withers, 1990; Bahr and Sejersted, 1991a; Bahr et al., 1992; Smith and McNaugton, 1993; Frey et al., 1993). Recently, Sedlock (1994) indicated that the changes in magnitude of EPOC are generally a function of the preceding relative exercise intensity (the percentage of VO_{2max} of the preceding exercise). Other researchers have determined duration of exercise to be primarily responsible for the total magnitude of EPOC (Knuttgen, 1970; Chad and Wenger, 1985; 1988; Sedlock, 1991). With some finding no effect of either duration or intensity of exercise on EPOC (Freedman-Akabas et al., 1985; Kaminsky et al., 1987; Maresh et al., 1992).

A few researchers have found a threshold intensity which must be reached in order to produce significant elevations in EPOC. Gore and Withers (1990a and b) and Bahr and Sejersted (1991a and b) estimated the effect of exercise intensity on EPOC to be associated with a >50% VO_{2max} threshold value while Hagberg et al. (1980) estimated the threshold slightly higher at >65% VO_{2max}. At these intensities a linear relationship is observed between duration of exercise and EPOC (Hagberg et al., 1980; Bahr et al., 1987; Gore and Withers, 1990a and b; Frey et al.,

1993). Interestingly, Bahr and Sejersted (1991a and b) suggest the exercise intensity threshold associated with a prolonged EPOC may possibly equal the lactate threshold. They indicate that the exercise intensity threshold responsible for the prolonged EPOC seems to occur near the percentage of VO_{2max} that is commonly associated with an untrained individual's anaerobic threshold.

2.5 Anaerobic Muscular Fatigue

Muscular fatigue may be defined as the inability of a muscle to contract at the required intensity. Factors responsible for limiting a muscles contractile capability will limit an individual's ability to perform. There are many integrated factors which dictate the performance of an exercise task and it is probably a combination of these factors which alter the metabolic efficiency and impair one's ability to perform (Wenger and Reed, 1976).

Metabolic inefficiency is the result of a decrease in available energy stores. Direct intramuscular sources of energy available to muscle cells are in the form of the high energy phosphates adenosine triphosphate (ATP) and creatine phosphate (CP). In fact, the contractile machinery of muscle cells can only directly use ATP as a fuel source (Wenger and Reed, 1976). Research has shown that physical exercise results in a marked decrease in the muscle content of CP and only a slight change in ATP concentration (Harris et al., 1976). These observations theoretically justify CP's primary role in phosphorylating ADP. Under any circumstances, a decrease in ATP or the associated high energy phosphate CP will limit an individual's ability to perform work.

The inability to continue high intensity work of 1-2 minutes is related to an accumulation of metabolic by-products. The buildup of excess metabolites prevents the subsequent regeneration of ATP and CP. As well, accumulated metabolic by-products directly effect the contractile machinery of the actin-myosin complex of muscle cells (Wenger and Reed, 1976).

The primary determinant of anaerobic muscular fatigue in work of 1-2 minutes duration is an increased lactate production by anaerobic glycolysis. Directly associated with the increase in lactate production is a decrease in the glycolytic rate and a subsequent decrease in the regeneration of intramuscular stores of the ATP and CP. The increased lactate concentration results in a lower muscle and blood pH which in turn reduces the reaction velocity of phosphofructokinase (PFK). As well, an associated decreased muscle pH and increased cellular acidity affects the permeability of cell membranes to Na^+ and K^+. Thus, the affected cells exist in a hyperpolarized state. As the permeability of the cell membrane to Na^+ and K^+ is important for eliciting action potentials, a decreased pH and associated hyperpolarized state of the cell could function to impair the ability of a muscle to contract. This effect is more pronounced when recruiting fast glycolytic (FG) muscle fibers as their resting membrane potential (-85mv) is lower than slow oxidative (SO) muscle fibers (-70mv). This becomes particularly important in anaerobic fatiguing exercise as the predominantly recruited muscle fibers are fast glycolytic type II a and b fibers. (Wenger and Reed, 1976).

Furthermore, increased lactate production and decreased pH are associated with an accumulation of H^+ in the cell cytosol. An increased H^+ concentration may compete with Ca^+ for the binding sites on the troponin complex of the actin filament. Thus, a resultant decrease in

functional actin-myosin complexes would be evident and therefore the contraction magnitude required in high intensity work would not be possible (Wenger and Reed, 1976).

Muscular fatigue can be limited or offset by adaptive training responses. Theoretically, training results in less of a decline in ATP and CP concentrations in the same exercising individual for a given workload. Factors governing the lowered decrease in high energy phosphate concentrations have been suggested by a number of researchers (Holloszy et al., 1984). One suggestion is that aerobic training results in a more rapid rise in O_2 delivery at the onset of exercise. As well, an improvement in O_2 availability to the contractile fibers occurs post training, resulting in an increased extraction of O_2 from the blood (Hagberg et al., 1980).

2.6 Mechanisms and Metabolic Components of EPOC:

The excess post exercise oxygen consumption (EPOC) appears to be dependent upon a number of interrelated factors. Postulated mechanisms behind the homeostatic disturbance and subsequent recovery from exercise appear to be: replenishment of the high energy phosphates ATP and CP, reloading the body's oxygen stores in the blood and muscle, increased energy cost of ventilation and circulation, metabolism of lactate, repletion of glycogen, response to exercise induced catecholamine release, oxidation of fat, increased substrate cycling rate, elevation of body temperature (Q_{10} effect), compensatory increased protein synthesis, ionic redistribution, thermic effect of food, and an elevated physiological functioning (Gaesser and Brooks, 1984; Bahr et al., 1992).

Traditionally, the elevated VO_2 seen in recovery from exercise has been broken up into two components; a rapid component and a slow component.

The rapid phase of recovery VO_2 has been attributed to the restoration of the high energy phosphates ATP and CP depleted during exercise, as well as the reloading of hemoglobin and muscle myoglobin stores (Bahr and Sejersted, 1991a and b). The kinetics and relative time course of the rapid phase of recovery VO_2 and CP resynthesis have been shown to be very similar (Harris et al., 1976; Knuttgen and Saltin, 1972; Mahler and Homsher, 1982). As much as 10% of the recovery VO_2 has been found to reload blood hemoglobin stores. As well, 2-5% goes to restoring the O_2 dissolved in body tissues and that which is bound to muscle myoglobin (McArdle et al., 1991).

The increased ventilation associated with the recovery from exercise requires that the respiratory muscles increase their O_2 supply. Also, in order to match the increased ventilation, the heart rate increases to allow for efficient gas exchange and for the delivery of O_2 to the tissues, thus the post exercise tachycardia contributes to the recovery O_2 (McArdle et al., 1991). Although both these factors affect the recovery VO_2 their contribution is relatively small compared with total EPOC magnitude.

The processes associated with the fast phase of recovery VO_2 are known to be limited to within the first few minutes and < 1 hour after exercise (Bahr and Sejersted, 1991a and b).

A prolonged EP0C component has been observed for as much as 8-48 hours into recovery (Bahr et al., 1987; Maehlum et al., 1986; Gore and Withers, 1990a). Much of the debate over the length of EPOC may be attributed to different methodologies used in the experimental procedures. The prolonged EPOC component has been suggested to be a result of several factors, including elevated concentrations of catecholamines, increased substrate cycling rate (substrate synthesis), increased body temperature, tissue repair and associated increased protein synthesis,

ionic redistribution, and the thermic effect of food (Gaesser and Brooks, 1984; and Gore and Withers, 1990a).

Elevated concentrations of catecholamines have been proposed as a mechanism behind the prolonged EPOC component (Gaesser and Brooks, 1984). Exercise at higher intensities activates the sympathetic nervous system and elevates the concentration of plasma catecholamines. Gaesser and Brooks (1984) speculate that catecholamines elevate mitochondrial respiration by stimulating energy requiring processes in the cell. Norepinephrine has been shown to increase the permeability of cell membranes to Na^+ and K^+ ions thus contributing to a disruption in ionic homeostasis. Bahr et al. (1992) show a correlation between mean increase in plasma norepinephrine over the first hour of recovery and EPOC ($r=0.70$, $p<0.005$), while Frey et al. (1993) show a slightly higher correlation ($r=0.78$, $p<0.05$).

Catecholamines have also been found to increase the rate of TG-FA cycling-which is an energy requiring process (Bahr and Sejersted, 1991a and b). Bahr et al. (1990) have demonstrated the rate of TG-FA cycling to be elevated for at least 3 hours after exercise.

Shephard (1984) has provided evidence showing the increased concentration of plasma catecholamines associated with exercise is not evident below exercise intensities of 70% VO_{2max}. Interestingly, Bahr and Sejersted (1991a) speculate there appears to exist a threshold exercise intensity (possibly equal to the lactate threshold) to exceed in order to trigger the metabolic processes responsible for the prolonged EPOC component.

Bahr et al. (1990a and b) provide evidence to suggest a shift in substrate utilization from CHO to fat accompanies an increased cycling rate. These researchers recognized post exercise

RER values to be reduced, indicating an increased reliance on FA oxidation and possible increased rate of TG-FA cycling.

The return to homeostasis of body temperature and post exercise VO_2 have been shown to be closely associated (Gaesser and Brooks, 1984). The increased body temperature probably effects EPOC by altering mitochondrial energetics; increasing respiration and decreasing phosphorylation-coupling efficiency (Gaesser and Brooks, 1984). Hagberg et al. (1980) determined that 60-70% of the slow component of recovery VO_2 could be accounted for by the effect of temperature on metabolism. In contrast, Maresh et al. (1992) found no significant correlation between rectal temperature and the post exercise VO_2 under exercise conditions of 60-70% VO_{2max} duration for 20-40 minutes. Differences in results may be due to different modes of exercise or muscle mass involved in the tests.

An increased rate of substrate (futile) cycles has been partly attributed to the slow component of recovery VO_2 (Bahr et al., 1987). Bahr et al. (1987) suggests that EPOC may be partly attributed to the mobilization of fat from adipose tissue and an increased rate of cycling between triacylglycerol and fatty acids. There is evidence to suggest that an O_2 uptake of 1ml $O_2 \cdot min^{-1} \cdot kg^{-1}$ could be accounted for by substrate cycles operating at only 3-17% of maximal capacity (Gaesser and Brooks, 1984).

Research by Viru (1987) indicates that protein metabolism is decreased during exercise. In the post exercise period, a compensatory increased rate of protein metabolism is evident. This increased rate of protein metabolism is partly accountable for by post exercise tissue repair.

Exercise causes a disruption in ionic homeostasis which must be corrected for in recovery. The catecholamine norepinephrine has been shown to increase the disruption of Na^+ and K^+

homeostasis, thus resulting in an increased permeability of the cell membrane to Na^+ and K^+ (Gaesser and Brooks, 1984). As well, the hormones thyroxine and glucocorticoids have been shown to increase Na^+/K^+ pump activity (Gaesser and Brooks, 1984).

In the post-exercise period Ca^{++} redistribution must take place within the muscle and across the cell membrane. Oxidative phosphorylation may also be affected by increased amounts of Ca^{++} within the mitochondria (Gaesser and Brooks, 1984).

The thermic effect of food (TEF) is known to contribute to VO_2 uptake. The digestion, absorption, transport, and storage of food places additional energy requirements on the body which must be met by an increase in O_2 uptake. It has been shown that 1 hour after ingestion of a 4900 KJ meal, VO_2 increased to a maximum of approximately 23% above resting levels (Gore and Withers, 1990).

To date, many attempts have been made to determine the specific mechanisms responsible for the elevated post exercise oxygen consumption. As yet, an all encompassing theory is far from being found. Gaesser and Brooks (1984) have offered an interesting approach to analyzing the recovery VO_2. They contend that because the site of oxygen consumption in the cell is the mitochondrion, the explanation of the elevated post exercise VO_2 may be found at the level of this cellular organelle. The metabolic rate will return to baseline when all the factors influencing mitochondrial respiration have returned to control levels (Gaesser and Brooks, 1984).

Debate continues regarding the biochemical, physiological and physical reasons for the elevated post exercise VO_2. Consensus is that the elevated recovery oxygen consumption is necessary to restore the body to its pre-exercise condition and is largely the result of the preceding metabolic and physiologic events during exercise (Stainsby and Barclay, 1970).

2.7 Lactate:

The "O_2 debt" hypothesis put forth by Hill and Lupton (1923) was an attempt to link the post exercise recovery VO_2 with the metabolism of lactate. This hypothesis was formulated based on the assumption that the primary fate of lactate in recovery was gluconeogenesis. Eighty percent of the lactate accumulated during exercise was thought to be reconverted to glycogen and twenty percent oxidized to CO_2 and H_2O. Since this time numerous studies have provided substantial evidence of a dissociation between the kinetics of the post exercise VO_2 and changes in blood and muscle lactate concentrations (Gaesser and Brooks, 1984). Although a great deal of research has been directed towards the investigation of lactate production and removal associated with exercise, the issue remains unclear due to the complexity of the factors controlling lactate dynamics.

Lactic acid serves as the by-product of anaerobic glycolysis. Lactate production occurs when lactate dehydrogenase (LDH) catalysis the conversion of pyruvate to lactate:

$$\text{pyruvate} + \text{NADH} + \text{H}^+ \longrightarrow \text{lactate} + \text{NAD}$$

The relative kinetics of glycolysis, LDH, and mitochondrial respiration determine the rate of lactate production.

The pK of lactic acid is 3.8, therefore, at physiological pH values, lactic acid dissociates to a proton (H^+) and a lactate anion ($C_3H_5O_3^-$). Thus the metabolism of lactate ($C_3H_6O_3$) must be considered when lactate is formed or removed via oxidation or gluconeogenesis or other pathways (Brooks, 1986).

In skeletal muscle, lactate production occurs when the energy demands cannot be supplied totally by oxidative processes (Cortes et al., 1988). As exercise intensity increases, the metabolic demands of muscle exceed the capacity of aerobic metabolism. Thus, a supplementation of energy is supplied by the anaerobic process of glycogen degradation to lactate (Stainsby, 1986). Plasma lactate is known to increase in concentration exponentially with increasing exercise intensity (Gollnick et al., 1986).

Traditionally, lactate has been considered a metabolic end product that accumulated during exercise giving rise to fatigue (Brooks, 1986). In recent years however, the major metabolic fate of lactate after exercise has been shown to be oxidation (Gaesser and Brooks, 1984). Gaesser and Brooks (1984) determined that 55-70% of the accumulated lactate was oxidized, post exercise, to CO_2 and H_2O, while less than 20% of the lactate was converted to muscle or liver glycogen. Brooks (1986) contends that lactate serves as an advantageous metabolic intermediate between carbohydrate storage forms and the metabolic end products CO_2 and H_2O.

Studies using isotopically labeled lactate have shown that the removal of lactate occurs in the liver, heart, skeletal muscle, brain, and kidney. Of particular interest is the proposed "Lactate Shuttle Hypothesis" by Brooks (1986). He outlines the role skeletal muscle and the heart play in the removal of lactate. He states that most of the lactate which appears in the blood will be removed and combusted by oxidative muscle fibers in both the same active muscle bed and in the heart.

The predominant metabolic fate of lactate appears to be directed towards energy production in the mitochondria. Gaesser and Brooks (1984) state that a post exercise elevated

concentration of lactate may be viewed as a reservoir of carbon. The accumulated lactate may serve as a source of ATP production or as a source of carbon skeletons for the synthesis of glucose, glycogen, amino acids, and TCA cycle intermediates (Gaesser and Brooks, 1984).

2.8 Oxygen Uptake During Exercise and Recovery as a Result of Training:

Berg (1947) appears to be the first researcher to address the relationship between training status and post exercise oxygen consumption. He recognized that trained individuals exhibited increased VO_2 recovery rates from moderate exercise.

Early work in the analysis and explanation of the VO_2 recovery curve led to the application of double exponential equations of the form:

$$VO_2 = A_1 e^{-k_1 t} + A_2 e^{-k_2 t}$$

As recovery rates are represented by half-time constant functions (half-time ($t_{1/2}$) = ln 2/k), a slow or fast recovery from exercise was found to be represented by a large or small half time constant respectively. Thus, Berg (1947) generalized to say that physically fit individuals tend to have smaller recovery half time constants.

Henry and Demoor (1950 and 1956) provided further insight into the rate of the recovery VO_2 and its relationship to training status. They stated that the recovery rate constants exhibit reliable individual differences and are altered by factors such as athletic training. Although a foundation for research regarding training and its relationship to recovery rates had been set,

much of the work with post exercise oxygen consumption began to be undertaken in the mechanisms governing the recovery VO_2.

Typically, research indicates that post training oxygen uptake during exercise and recovery is decreased for the same absolute workload (Robinson and Harman, 1941; Knehr et al., 1942; Crescitelli and Taylor, 1944; Henry and Berg 1950; Cotes and Meade, 1959; Douglas and Becklake, 1968; Cunningham and Faulkner, 1969; Girandola and Katch, 1973; Hagberg et al., 1980). Authors cite an increase in mechanical efficiency as the cause of this decreased O_2 uptake during exercise.

Renewed interest in the relationship of training status to the rate of the post exercise recovery oxygen consumption was provided by Hagberg et al. (1980). The researchers evaluated the effects of endurance exercise training on the VO_2 adjustment to and recovery from submaximal exercise. It was found that post training an increase in the recovery rate of oxygen consumption during submaximal exercise was evident at the same absolute as well as relative work loads. As well, they found the O_2 debt to be smaller at the same absolute work rate and not significantly different at the same relative work rate post training. Hagberg et al. (1980) provided further information in support of the early research associated with training status and VO_2 recovery rates suggesting that an individual's training status may be an important determinant in the time course of the recovery VO_2.

Further research into the response of an individual's post exercise recovery oxygen consumption to training has found varying results. Brehm and Gutin (1986) and Freedman-Akabas et al. (1985) found that recovery VO_2 was not significantly different between fit and unfit subjects. Further evidence discrediting Hagberg's results were provided by Chad and Quigley

(1991) and Sedlock (1994). Chad and Quigley (1991) used trained and untrained women on a 30 minute cycle ergometer test, and failed to find any significant difference in the rate of the recovery VO_2 uptake between the trained and untrained subjects. Recently, Sedlock (1994) found no significant differences for either magnitude or duration of EPOC between fit and unfit subjects who exercised to 300 kilocalories on a cycle ergometer. In this study all subjects exercised at the same relative intensity, therefore, it could be assumed that the relative disruption to homeostasis was similar for both groups.

Elliot et al. (1988) provided evidence in support of Hagberg's results. A cycle ergometer test at 80% max. HR for 10 and 30 minutes indicated a direct correlation ($r=-0.70$, $p<0.01$) between aerobic endurance capacity and the recovery energy expenditure.

Frey et al. (1993) compared recovery VO_2 between trained and untrained women after a cycle ergometer test at 80% VO_{2max} and 65% VO_{2max} to a set energy expenditure (300 kcal). They showed that total EPOC was larger in the trained subjects yet, the time to recovery was shorter. The authors indicated, as others had suggested previously, that training status affects primarily the fast component and the initial phase of the slow component of EPOC.

Recent work on the effects of training on gas exchange kinetics has focused on the VO_2 uptake at the onset of exercise in specific populations (Babcock et al., 1994; Barstow et al., 1996). Babcock et al. (1994) examined the kinetics of gas exchange at the on-transient of exercise in older men (72 yr.) after 6 months of aerobic cycle training. The authors were able to show that with a vigorous training program the kinetics of gas exchange were made faster and approached values reported in young subjects (33-36 sec). They found a 20% increase in VO_{2max} and showed

a significant decrease (48.7%) in τ for O_2 uptake kinetics (62.2s to 31.9s). Interestingly, the correlation of the decrease in τVO_2 with the increase of VO_{2max} was not significant.

Recently, Barstow et al. (1996) examined the effects of training on gas exchange kinetics in patients with spinal cord injury. Subjects in this study performed two, 30 minute training sessions per week of cycle ergometer exercise invoked by functional electrical stimulation (FES) of the legs. Peak VO_2 was significantly greater and the Mean Response Time On (MRT_{on}) and Mean Response Time Off (MRT_{off}) for τVO_2 became significantly faster following training. There was, however, great individual variability in the extent of the improvements. No correlation's were found between the percentage improvement in either MRT_{on} or MRT_{off} for VO_2 and the percentage increase in peak VO_2. Interestingly, the authors found that the training decreased the intersubject variability of both MRT_{on} and MRT_{off}.

2.9 Supramaximal Exercise:

Supramaximal exercise requires a rate of energy release exceeding the maximal oxygen uptake (Medbo et al., 1988). Energy used for supramaximal exercise is supplied by intramuscular stores of high energy phosphates, glycolysis, and aerobic metabolism.

The energy requirements for exhausting exercise up to two minutes in duration are mainly met by the high energy phosphates (ATP and CP) and anaerobic glycolysis. Typically, the maximal amount of ATP formed by aerobic and anaerobic processes in fatiguing exercise of approximately two minutes in duration has been defined as the anaerobic capacity (Medbo et al., 1988). The stores of ATP and CP and the extent to which lactate can accumulate are limited thus,

under appropriate exercise conditions the maximal accumulated O_2 deficit seems to provide an accurate estimate of the anaerobic capacity (Medbo et al., 1988).

Medbo et al. (1988) presented a method for quantifying the anaerobic capacity based on a determination of the maximal accumulated O_2 deficit. The method involved an estimation of the O_2 demand made by extrapolating the linear relationships between treadmill speed and the O_2 uptake at submaximal intensities. The maximal accumulated O_2 deficit, and thus an individual's anaerobic capacity, could then be quantified after measuring the O_2 uptake during one exhausting bout of exercise lasting 2-3 minutes.

CHAPTER 3

3.0 Methodology

3.1 Research Design:

This study employed a single group RM design. Subjects were measured pre and post training on the dependent variables of VO_{2max}, EPOC, blood lactate concentration, and the rate constants τ_1, and τ_2.

Dependent variables are:

1. VO_{2max}.
2. EPOC.
3. blood lactate concentration
4. τ_1 and τ_2.

Independent variable:

1. endurance training program.

3.2 Subjects

Ten male HKIN undergraduate students between the ages of 19-25 years were recruited for this study. The subjects were relatively untrained, that is they were not on an endurance training program and they exercised for less than four hours a week. All subjects had a pre-training VO_{2max} of less than 50 $ml \cdot kg^{-1} \cdot min^{-1}$. The study was carried out according to the ethical guidelines laid down by the University of British Columbia Clinical Screening Committee for research and other studies involving human subjects.

3.3 Pre-Experimental Protocol / Testing Preparation

Prior to giving consent, subjects visited the lab in order to become familiar with the equipment used as well as the tests to be performed. Practice trial tests were then performed to familiarize the subjects with treadmill running and the degree of fatigue they could expect in the actual test sessions. At this time, all subjects completed a written informed consent form and a physical activity readiness questionnaire (PAR-Q).

3.4 Experimental Protocol and Procedures:

All testing was performed at the University of British Columbia in the John M. Buchanan Exercise Science Laboratory. Subjects were required to perform six treadmill tests (four pre-intervention tests and two post-intervention tests). These tests were performed on separate days with a minimum of two days rest in between.

Subjects were instructed to refrain from eating or drinking, with the exception of water, for two hours prior to any of the tests. As well, subjects were asked to not consume caffeine or alcohol or engage in physical exercise 24 hours prior to testing.

Subject's anthropometric data (height and weight) was collected on the first testing day, prior to the VO_{2max} test, in the pre and post-intervention phases of the study. All metabolic data acquisition was made using a Beckman Metabolic Measuring Cart interfaced with a Hewlett Packard 3052A Data Acquisition System. This system recorded respiratory gas exchange variables every 15 seconds.

Subjects underwent three phases of the study:

1. Pre-intervention phase

2. Intervention phase

3. Post-intervention phase

Pre-Intervention Phase

All subjects were tested on one VO_{2max} test and three AST's over a period of two weeks. The performance of each test was separated by at least two days rest. An RMR measurement was also made during the pre-intervention phase of the study.

Data recorded during the AST's were fifteen second heart rates and time to fatigue. The first AST was run at 8 mph and 20% grade and the second at 8 mph and 12% grade.

The third AST was run with a treadmill speed of 8 mph and an individually determined percent grade was used in order to produce a test of approximately two minutes. This percent grade was determined from each subject's time to fatigue recorded in the first two AST's.

Intervention Phase:

Endurance Training Program

The endurance training program consisted of a six week training period. Subjects were required to exercise three times a week for 30 minutes each session. All training was progressive with respect to the calculated pre-training ventilatory threshold (VT). A progression in exercise intensity over the six weeks took place as follows:

week 1 - treadmill speed at 16% below VT:

week 2 - treadmill speed at 12% below VT.

week 3 - treadmill speed at 8% below VT.

week 4 - treadmill speed at 5% below VT.

week 5 - treadmill speed at 3% below VT.

week 6 - treadmill speed at pre-training VT.

note - A five minute warm-up and cool-down period at a self selected exercise intensity were required with each exercise session.

The training program took place and was monitored on treadmills in the J.M. Buchanan Exercise Science Laboratory. All subjects completed the six week training program.

Post Intervention Phase:

The subjects were tested on one VO_{2max} test as well as one two minute AST (2min AST). The 2min AST was performed at the same workload and time duration as in the pre-intervention phase.

3.5 Measurements

3.5.1 Heart Rate (HR)

Heart rate was monitored with a Sport Tester PE 3000 heart rate monitor. Heart rate was measured every minute during the VO_{2max} tests. Heart rate was also recorded each minute during the RMR calculation and each minute of recovery from the AST's. Fifteen second heart rate recordings were made during the performance of the AST's.

3.5.2 Oxygen Consumption (VO_2)

Oxygen consumption was measured by the Beckman Metabolic Cart every 15 seconds during the pre and post intervention VO_{2max} tests and the pre and post intervention 2min AST's. Oxygen consumption was also measured every 15 seconds during the RMR calculations.

3.5.3 Resting Metabolic Rate (RMR)

RMR was measured pre and post training (both measurements were made at the same time of day). After an overnight fast (minimum of 8 hours), subjects reported to the lab. Subjects were required to be dropped off at the lab the morning of the test. RMR was then measured during a thirty minute metabolic gas acquisition period. Subjects lay supine on a standard trainers bench while metabolic gasses were collected (using the Beckman Metabolic Cart). RMR was calculated as the mean rate of oxygen consumption (relative and absolute) for the last five minutes of the thirty minute session.

3.5.4 Resting HR

Resting HR was calculated during the 30 minute metabolic gas acquisition period used in determining RMR. Resting HR was calculated as the mean HR ($bts \cdot min^{-1}$) for the last five minutes of the 30 minute session.

3.5.5 Maximal Oxygen Uptake (VO_{2max})

VO_{2max} was determined using a continuous treadmill protocol. A five minute warm-up at an individually set treadmill speed preceded the test. The VO_{2max} involved running on a level

treadmill starting at 5 mph and this speed was increased by .5 mph every minute until volitional fatigue (Taylor et al., 1953). A nose clip and mouthpiece were worn by the subjects during the performance of the VO_{2max} test. A left and right tube were attached at either side of the mouthpiece. The left tube was open at the distal end to allow room air into the mouthpiece. The right tube transferred expired air to the Beckman Metabolic Measuring Cart. Two of three criteria were met in order to ensure attainment of VO_{2max}:

1. Heart rate within 10% of age predicted maximum.
2. Leveling off of VO_2 with an increase in workload.
3. RER greater than 1.1.

3.5.6 AST (Anaerobic Speed Test)

The Anaerobic Speed Tests were run at 8 mph on a 20% grade and at 8 mph on a 12% grade. The AST's began with the treadmill set at the respective percent grade and the velocity of the treadmill at eight mph. Subjects began by straddling the moving treadmill belt and the researcher gave a five second countdown to the start of the test. A timer was started when the researcher had reached time zero and at this point the subject stepped onto the treadmill belt and proceeded to run to fatigue. Fatigue was measured as the time from the start of the test to the time at which the subject was no longer able to maintain treadmill speed. At this time the subject stepped off and straddled the treadmill belt. The clock was then stopped and the time to fatigue was recorded.

3.5.7 Two Minute AST (2min AST) and Measurement of Maximum EPOC (maxEPOC)

Maximum EPOC was measured for 30 minutes following each subject's 2min AST. The 2min AST test was individually determined by estimating from the two previous AST's (8mph on 20% and 12% grade) a percent grade that provided a test of approximately 2 minutes in duration. Subjects were fitted with the same noseclip and mouthpiece used in the VO_{2max} test. The treadmill was set at the pre-determined percent grade and the treadmill belt was running at a velocity of eight mph. The 2min AST started with the subjects straddling the moving treadmill belt and breathing through the mouthpiece. The same protocol used to initiate the 20% and 12% grade AST's was used to initiate the start of the 2min AST. Time to fatigue was again recorded in the same manner as in the previous AST's. When the subject reached fatigue and stepped off and straddled the treadmill, the metabolic data acquisition system was turned on. At this time the treadmill was stopped and the elevation was taken to 0% grade. A standard trainers bench was then lifted onto the treadmill and placed in position for the subject to lie down on. The time between the completion of the 2min AST and the time the subject was lying in a supine recovery position was about 15 seconds. Metabolic measurements were made over the next 30 minutes.

3.5.8 Ventilatory Threshold (VT)

VT was determined through metabolic data acquisition made during the VO_{2max} test. VT was represented by a disproportionate (nonlinear) increase in the excess CO_2 ($ExCO_2$) elimination curve over time. This determination was made by visual inspection of two observers not directly involved in the study, who had experience using excess CO_2 to measure VT. Observations were made separately and an agreement was reached on the location of the threshold. VT was then

equated to a treadmill speed by measuring the velocity of the treadmill at the determined threshold point.

3.5.9 Blood Lactate

Blood lactate measurements were made at 3 minutes after the 2min AST. Blood lactate samples were taken by finger prick samples and analyzed with an Accusport portable blood lactate analyzer.

3.6 Data Analysis:

Analysis of the data was as follows:

1. analysis of the recovery curves to determine the rate constants τ_1 and τ_2.

2. EPOC magnitude was determined by analyzing the area under each VO_2 recovery curve.

3. Hotelling's T^2 test was used to determine mean differences between:

a) pre-intervention VO_{2max} and post-intervention VO_{2max}

b) pre-intervention EPOC and post-intervention EPOC.

c) pre-intervention τ_1 and post-intervention τ_1.

d) pre-intervention τ_2 and post-intervention τ_2.

e) pre-intervention blood lactate concentration and post-intervention blood lactate concentration.

5. Pearson Product Moment Correlation Coefficient (r) was used to determine relationships between:

a) pre-intervention VO_{2max} and the pre-intervention variables τ_1 and τ_2.

b) post-intervention VO_{2max} and the post-intervention variables τ_1 and τ_2.

c) the change in VO_{2max} and the change in EPOC magnitude.

d) the change in VO_{2max} and the changes in τ_1 and τ_2.

e) the change in VO_{2max} and the change in blood lactate concentration.

3.6.1 VO_2 Recovery Rate

The VO_2 recovery curve was fit with a double exponential equation of the form:

$$VO_2 = A_1 e^{-t/\tau_1} + A_2 e^{-t/\tau_2}$$

Equation variables:

VO_2 = oxygen consumption at time "t"

A_1 = fast component linear parameter

τ_1 = fast component non-linear parameter

A_2 = slow component linear parameter

τ_2 = slow component non-linear parameter

A scatter plot formation of minute EPOC values in log form was plotted against time. Individual recovery data was split into two components (fast and slow) and fit to separate exponentials.

$$\text{fast: } y_1 = A_1 e^{-t/\tau_1} \qquad \text{slow: } y_2 = A_2 e^{-t/\tau_2}$$

Constants were determined by carrying out linear regressions:

If $y = A e^{-t/\tau}$

Then $\log y = \log A - t/\tau (\log e)$

Therefore $\log y = \log A - t(.434)/\tau$ (1)

If log y is plotted against t, the result is a straight line slope with the intercept log A. Values were obtained by regression analysis of semi-log data. If m and b are the linear regression constants i.e.

$$y = mx + b \quad (2)$$

then, comparing equation (1) and (2) gives:

$$m = -(.434)/\tau \quad \text{and} \quad b = \log A$$

giving $\tau = -(.434)/m$ and $A = 10^b$ (3) and (4)

Sample VO_2 Recovery Rate Calculation:

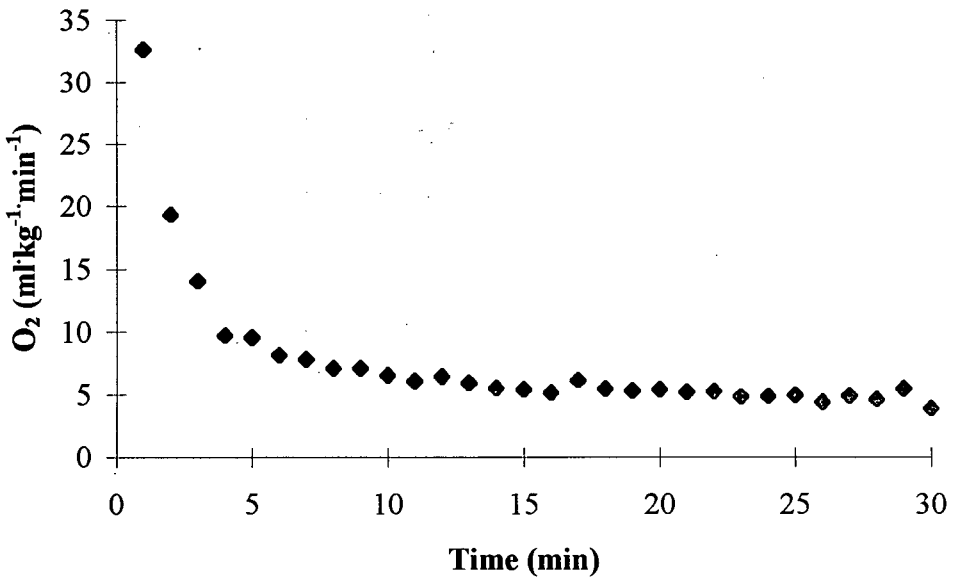

Figure 1. EPOC for subject 2.

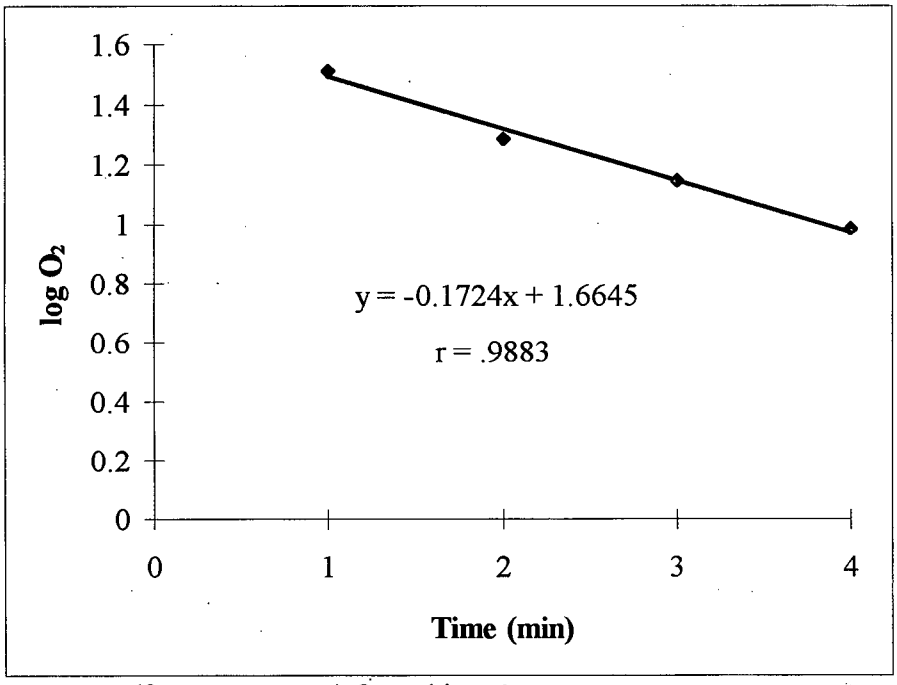

Figure 2. Recovery rate (fast component) for subject 2.

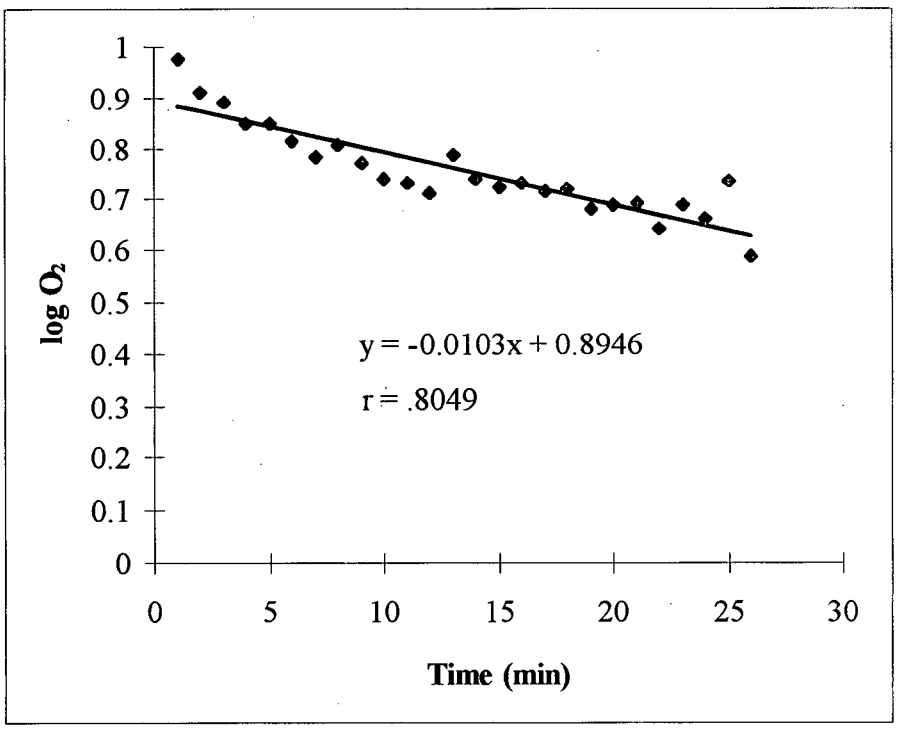

Figure 3. Recovery rate (slow component) for subject 2.

3.6.2 EPOC Magnitude

EPOC magnitude was determined by analyzing the area under each individual's recovery curve. The net VO_2, above resting values, for each minute value of the recovery period was summed in order to give total EPOC magnitude (Sedlock, 1991).

$\sum_{j=1}^{30} x_j$ -where x represents the net EPOC value for the minute j.

CHAPTER 4

4.0 Results

4.1 Descriptive Data

Ten male subjects completed the six week aerobic training program. All subjects were in good health and injury free throughout the duration of the study. Individual physical data for the ten subjects is presented in Table 1. Mean physiological data for the ten subjects and the results of the Hotelling's T^2 test performed on pre and post training data are presented in Table 2.

Table 1: Physical Data

Subject	Age	Height (cm)	Weight (kg)	
			pre-training	post-training
1	24	184.3	88.6	83.5
2	21	162.9	63.2	62.8
3	20	183.3	86.3	86.3
4	20	176.3	73.7	72.5
5	21	174.0	64.0	63.6
6	24	178.2	100.8	99.6
7	24	165.9	67.6	68.9
8	21	184.0	77.8	79.8
9	20	185.9	88.2	86.7
10	25	174.7	76.2	75.5
$\overline{X}$	22.38	176.53	79.51	78.66
SD	2.07	8.53	12.34	11.48

Table 2. Significant differences between pre and post training values (N=10) for VO_{2max}, τ_1, τ_2, EPOC, EPOCfast, EPOCslow, and blood lactate.

	$\overline{X} \pm$ (SD)		Significant
	pre training	**post training**	
VO_{2max} ($L \cdot min^{-1}$)	3.61 (0.42)	4.00 (0.44)	$p = 0.001$ *
VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)	46.38 (3.74)	51.82 (5.21)	$p < 0.001$ *
τ_1 (min^{-1})	2.69 (0.19)	2.29 (0.33)	$p = 0.005$ *
τ_2 (min^{-1})	43.74 (5.12)	39.63 (4.24)	$p = 0.033$ *
EPOC (Litres)	9.13 (1.68)	7.49 (1.73)	$p < 0.001$ *
EPOC ($ml \cdot kg^{-1}$)	116.89 (18.21)	95.38 (16.55)	$p = 0.001$ *
Lactate ($mmol \cdot L^{-1}$)	15.28 (1.80)	13.36 (1.55)	$p < 0.001$ *
EPOCfast (Litres)	4.46 (0.95)	3.81 (0.80)	$p = 0.003$ *
EPOCfast ($ml \cdot kg^{-1}$)	57.84 (4.63)	49.11 (5.25)	$p = 0.001$ *
EPOCslow (Litres)	4.67 (1.18)	3.71 (1.30)	$p < 0.001$ *
EPOCslow ($ml \cdot kg^{-1}$)	59.05 (17.31)	46.57 (16.04)	$p = 0.003$ *

* indicates that the values are significantly different at $p < 0.05$.
Values are means with standard deviations in parenthesis ($\overline{X} \pm$ SD).

4.2 Changes in Dependent Variables

Dependent variables are presented in Table 2. Hotelling's T^2 test indicated significant differences between pre and post training absolute and relative VO_{2max} (Figures 4 and 5) and EPOC values (Figures 8 and 9) ($p < 0.05$). Hotelling's T^2 test also indicated significant differences between pre and post training recovery rates (τ_1 (Figure 6) and τ_2 (Figure 7)) and blood lactate values (Figure 10) ($p < 0.05$). EPOC magnitude was broken down into its two components, EPOCfast and EPOCslow. The Hotelling's T^2 test results indicated significant differences between absolute and relative pre and post training EPOCfast (Figures 11 and 12) and EPOCslow components (Figures 13 and 14) ($p < 0.05$).

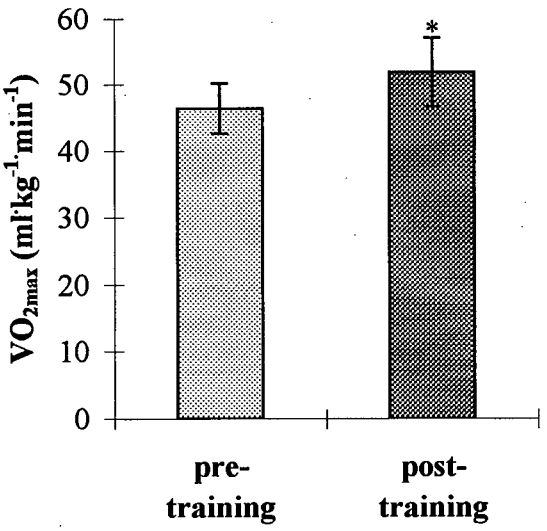

Figure 4. The relative change in VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

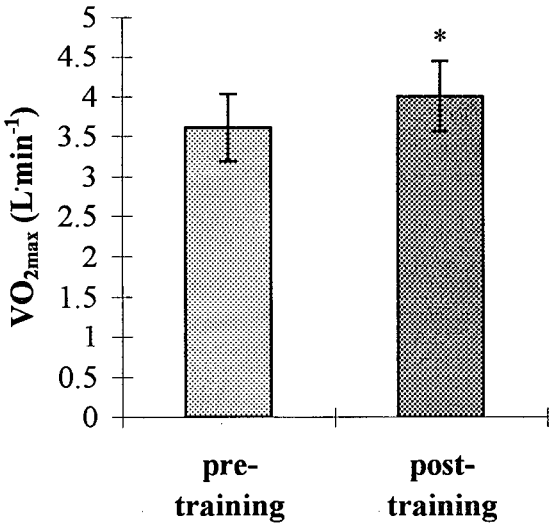

Figure 5. The absolute change in VO_{2max} ($L \cdot min^{-1}$) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

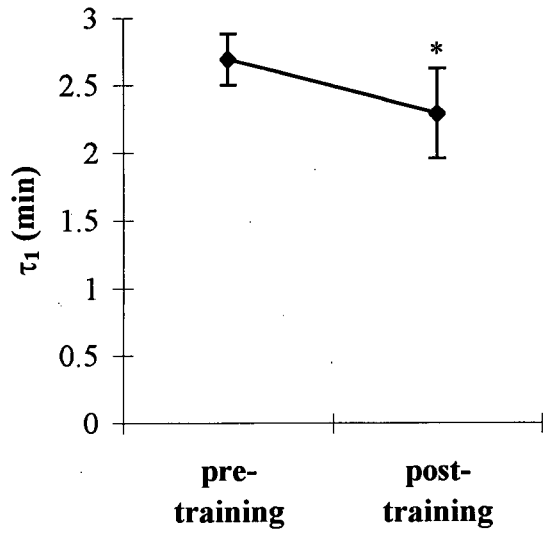

Figure 6. The change in τ_1 from pre to post training. The value of τ_1 is given as a rate in minutes.
* indicates that the values are significantly different at $p < 0.05$.

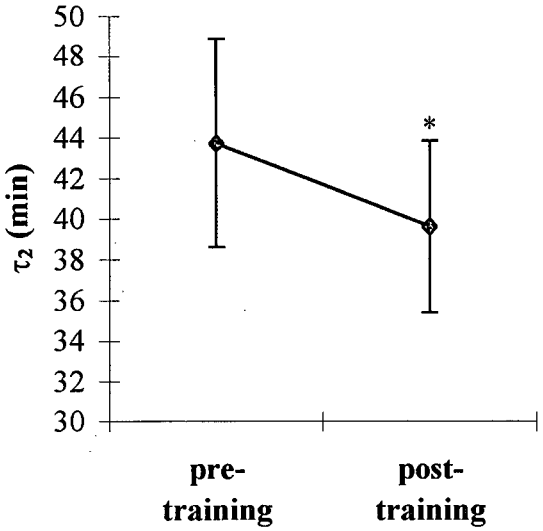

Figure 7. The change in τ_2 from pre to post training. The value of τ_2 is given as a rate in minutes.
* indicates that the values are significantly different at $p < 0.05$.

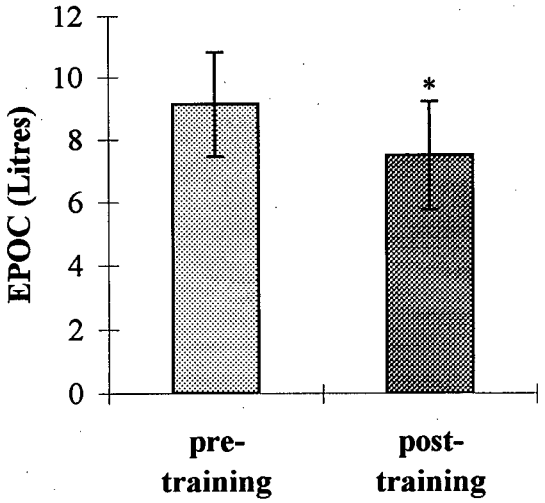

Figure 8. The absolute change in EPOC volume (Litres) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

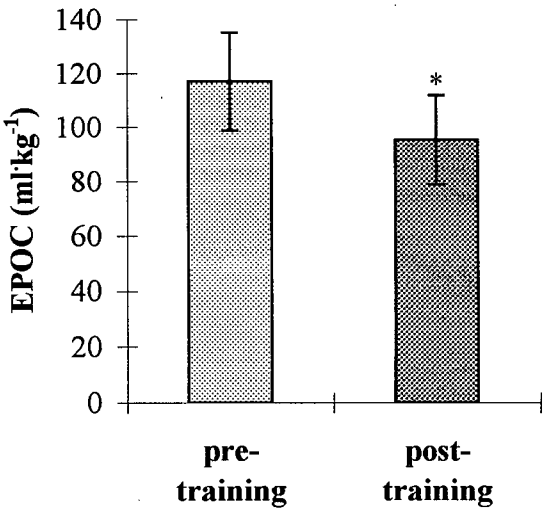

Figure 9. The relative change in EPOC volume ($ml \cdot kg^{-1}$) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

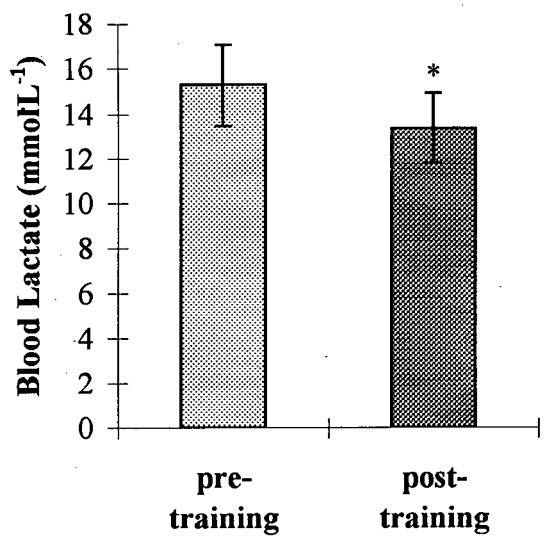

Figure 10. The change in blood lactate concentration ($mmol \cdot L^{-1}$) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

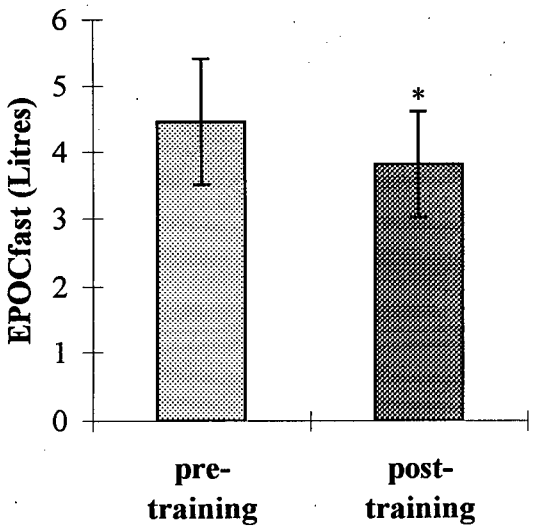

Figure 11. The absolute change (Litres) in the fast component EPOC volume (EPOCfast) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

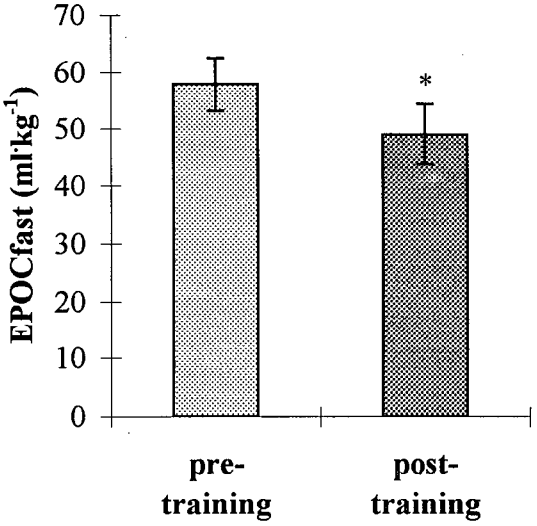

Figure 12. The relative change ($ml \cdot kg^{-1}$) in the fast component EPOC volume (EPOCfast) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

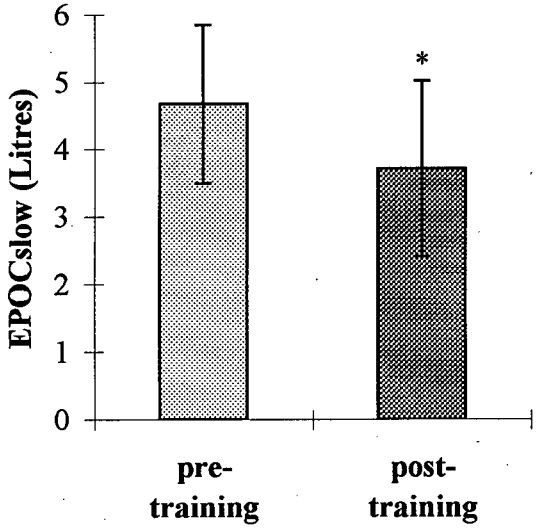

Figure 13. The absolute change (Litres) in the slow component EPOC volume (EPOCslow) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

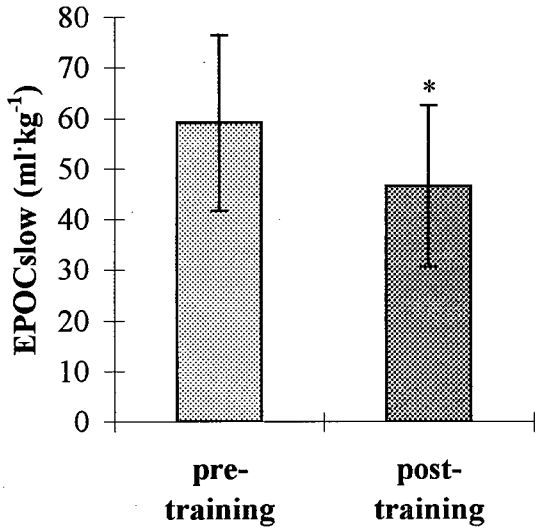

Figure 14. The relative change ($ml \cdot kg^{-1}$) in the slow component EPOC volume (EPOCslow) from pre to post training.
* indicates that the values are significantly different at $p < 0.05$.

4.3 Relationships

Relative and absolute comparisons were made among scores. For relative comparisons, VO_{2max} was expressed in $ml \cdot kg^{-1} \cdot min^{-1}$ and EPOC, $EPOC_{fast}$, and $EPOC_{slow}$ were expressed in $ml \cdot kg^{-1}$. For absolute comparisons, VO_{2max} was expressed in $L \cdot min^{-1}$ and EPOC, $EPOC_{fast}$, and $EPOC_{slow}$ were expressed in Litres.

Table 3. Correlation coefficients for VO_{2max} and τ_1 and τ_2.

	VO_{2max} pre-training (relative)	**VO_{2max} post-training (relative)**	**VO_{2max} pre-training (absolute)**	**VO_{2max} post-training (absolute)**
τ_1 pre-training	-0.0273 (p = 0.470)	-	-0.0337 (p = 0.463)	-
τ_1 post-training	-	-0.4797 (p = 0.080)	-	0.2826 (p = 0.214)
τ_2 pre-training	-0.1287 (p = 0.362)	-	-0.0628 (p = 0.432)	-
τ_2 post-training	-	-0.4956 (p = 0.073)	-	0.3720 (p = 0.145)

A correlation matrix outlining the relationships between VO_{2max} and the rate constant recovery variables is presented in Table 3. Non significant negative correlations were found between both pre and post training relative VO_{2max} and pre and post training rate constants. Although these correlations were not significant, these trends in the data would indicate that the higher the relative VO_{2max}, the faster the fast and slow component rates of recovery from the supramaximal exercise condition.

Table 4. Correlation coefficients for the change in VO_{2max} and the changes in the measured recovery variables.

-	**ΔVO_{2max} (relative)**	**ΔVO_{2max} (absolute)**
Δ EPOC (relative)	0.5133 (p = 0.065)	-
Δ EPOC (absolute)	-	0.3349 (p = 0.172)
$\Delta \tau_1$	0.2405 (p = 0.252)	0.0579 (p = 0.437)
$\Delta \tau_2$	0.3011 (p = 0.199)	0.0844 (p = 0.408)
Δ [BLa]	0.7272 (p = 0.009) *	0.4864 (p = 0.077)

* indicates that the values are statistically significant at $p < 0.05$.

Non significant positive correlations were found between the change in VO_{2max} and the changes in EPOC and changes in recovery rate constants between pre and post training. Although these correlations were not significant, the positive trends in the data would indicate that a greater change in VO_{2max} is associated with a greater change in EPOC and change in recovery rate constants. There was a significant correlation found between the relative change in VO_{2max} and the change in blood lactate concentration ($r = 0.7272$; $p < 0.009$) (Fig. 15).

$EPOC_{slow}$ Volumes

Table 5. Correlation coefficients for EPOC volume and the EPOCslow volumes.

	EPOC pre-training (relative)	**EPOC post-training (relative)**	**EPOC pre-training (absolute)**	**EPOC post-training (absolute)**
EPOCslow pre-training (relative)	0.8354 ($p < 0.001$) *	-	-	-
EPOCslow pre-training (absolute)	-	-	0.9673 ($p < 0.001$) *	-
EPOCslow post-training (relative)	-	0.9493 ($p < 0.001$) *	-	-
EPOCslow post-training (absolute)	-	-	-	0.9037 ($p < 0.001$) *

* indicates that the values are statistically significant at $p < 0.05$.

Table 5 identifies the relationships between the relative and absolute EPOC volumes and the relative and absolute EPOCslow volumes. Significant positive correlation's were found between the pre training EPOC volumes and the pre training EPOCslow volumes (relative, $r = 0.8354$; absolute, $r = 0.9673$) ($p < 0.05$) (Figures 16 and 17). Significant positive correlation's were also found between post training EPOC volumes and post training EPOCslow volumes (relative, $r = 0.9493$; absolute, $r = 0.9037$) ($p < 0.05$) (Figures 18 and 19).

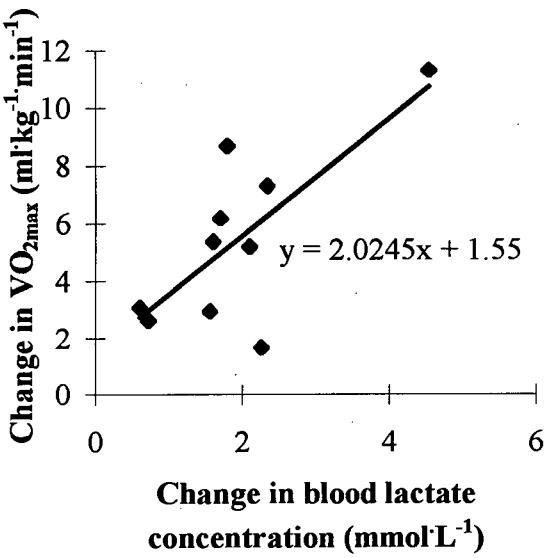

Figure 15. The correlation between the change in VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) and the change in blood lactate concentration ($mmol \cdot L^{-1}$) from pre to post training.

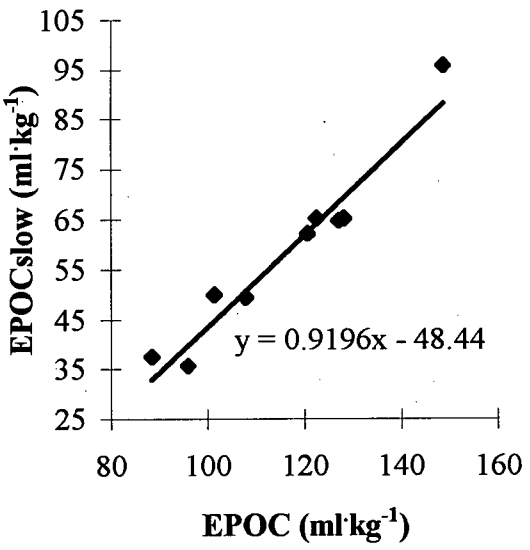

Figure 16. The correlation between the relative pre-training EPOCslow volume ($ml \cdot kg^{-1}$) and the relative pre-training EPOC volume ($ml \cdot kg^{-1}$).

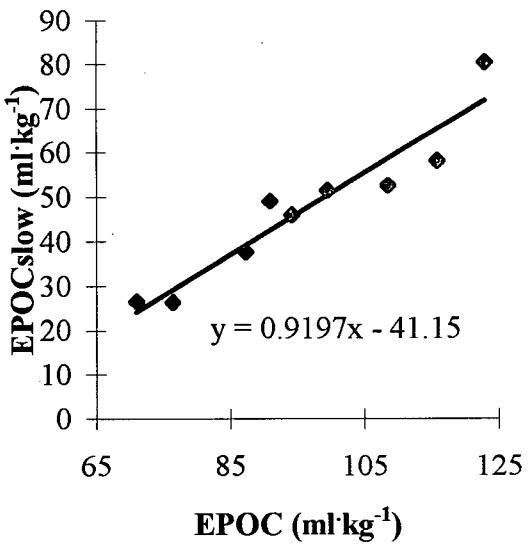

Figure 17. The correlation between the relative post-training EPOCslow volume ($ml \cdot kg^{-1}$) and the relative post-training EPOC volume ($ml \cdot kg^{-1}$).

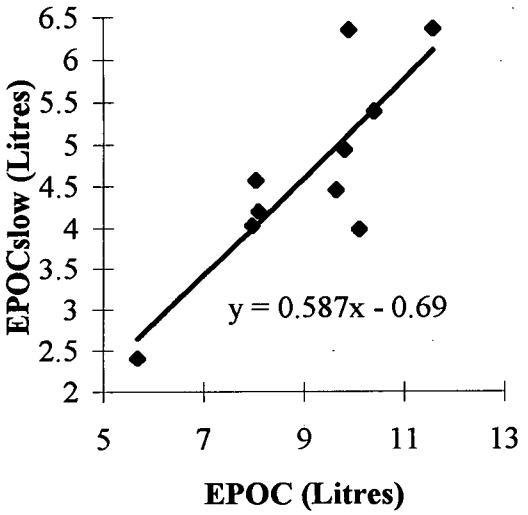

Figure 18. The correlation between the absolute pre-training EPOCslow volume (Litres) and the absolute pre-training EPOC volume (Litres).

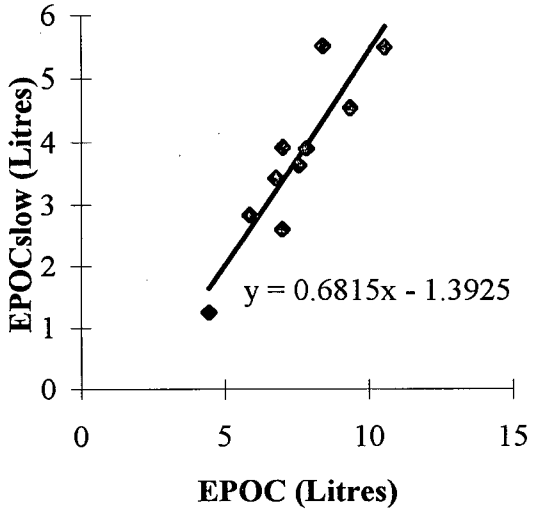

Figure 19. The correlation between the absolute post-training EPOCslow volume (Litres) and the absolute post-training EPOC volume (Litres).

4.4 Results of Hypotheses:

4.4.1 Primary Hypotheses:

1. a.) VO_{2max} ($L \cdot min^{-1}$) < VO_{2max} ($L \cdot min^{-1}$) accept
(pre-training) (post-training)

b.) VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) < VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) accept
(pre-training) (post-training)

2. a.) i.) τ_1 (pre-training) > τ_1 (post training) accept

ii.) τ_2 (pre-training) > τ_2 (post training) accept

b.) EPOC (pre-training) > EPOC (post-training) accept

c.) [BLa] (pre-training) > [BLa] (post-training) accept

4.4.2 Secondary Hypotheses

1.) Significant negative relationships between the relative VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$) and the VO_2 rate constants (τ_1 and τ_2).

a.) Pre-Training:

i.) VO_{2max} and τ_1 reject

ii.) VO_{2max} and τ_2 reject

b.) Post-Training:

i.) VO_{2max} and τ_1 reject

ii.) VO_{2max} and τ_2 reject

2.) Significant positive relationships between the change in VO_{2max} and the changes in recovery variables from pre to post training.

a.) Δ VO_{2max} and Δ EPOC volume. reject

b.) Δ VO_{2max} and Δ τ_1 reject

c.) Δ VO_{2max} and Δ τ_2 reject

d.) Δ VO_{2max} and Δ [BLa] accept

CHAPTER 5

5.0 Discussion

5.1 Introduction

Since the mid 1920's, the majority of the research investigating training and the recovery VO_2 after exercise has focused on recovery from submaximal exercise conditions. It is well documented that significant physiological changes take place with training. These changes include an increased aerobic power (VO_{2max}) as well as a smaller and more rapid rate of recovery VO_2 for the same absolute work load administered post training. However, there is a paucity of information on the effects of an endurance training program to the recovery VO_2 associated with supramaximal exercise. In fact, the majority of research in this area has focused on metabolic recovery from exercise intensities at or below 75% of subjects VO_{2max}. There are also discrepancies in the literature pertaining to the relationship that exists between aerobic power (VO_{2max}) and the recovery VO_2. Bell et al. (1997), Brehm and Gutin (1986), Freedman-Akabas et al. (1985), and Chad and Quigley (1991) provide evidence to suggest that there is no relationship between the recovery VO_2 and maximal aerobic power. However, research by Hagberg et al. (1980) and Elliot et al. (1988) indicates a relationship between recovery VO_2 and an individual's level of training.

The major findings from this study suggest that endurance training improves the rate and magnitude of the recovery VO_2 associated with supramaximal exercise. However, no relationships were found between aerobic power and the recovery VO_2, thus indicating that the rate and magnitude of the recovery VO_2 after supramaximal exercise are independent of VO_{2max}.

5.2 VO_{2max}

It is well documented that endurance training can increase aerobic power. The amount of training improvements depend to a large extent on one's initial fitness level. Individuals with low initial fitness levels have a greater capacity for improvement than individuals with high initial fitness levels. Generally, an increase in aerobic power of 5-25% is expected from systematic endurance training with significant training improvements occurring in the first three weeks (Hickson et al., 1981). In the present study, the sample of relatively untrained subjects showed a significant improvement in relative aerobic power (10.50%) as well as in absolute aerobic power (9.75%). The six week aerobic training program produced an increase in relative aerobic power from 46.38 $ml \cdot kg \cdot min^{-1}$ to 51.82 $ml \cdot kg \cdot min^{-1}$ and an increase in absolute aerobic power from 3.61 $L \cdot min^{-1}$ to 4.00 $L \cdot min^{-1}$. More frequent training appears to lead to greater improvements in peak VO_2. Hagberg et al. (1980) reported a 24% increase in VO_{2max} after a nine week exercise program in which the subjects trained at a high intensity, six days per week, for 30 to 40 minutes a session.

A training induced increase in aerobic power, as seen in the present study, may be attributed to a combination of a number of physiological changes that take place with training. These may include specific aerobic system changes such as an increased capacity of mitochondria to generate ATP aerobically by oxidative phosphorylation; an increase in both the size and number of mitochondria and an increase in the level of aerobic system enzymes; an increase in skeletal muscle myoglobin content; an increase in the trained muscles' capacity to mobilize and oxidize fat; an increase in the trained muscles capability to oxidize CHO; and a selective hypertrophy of different muscle fibers to specific overload training. Related cardiovascular and respiratory changes, which could function to increase aerobic power, may also take place in response to

training. Some related functional and dimensional changes that may take place with training are a cardiac hypertrophy, an increase in plasma volume and total hemoglobin concentration of the blood; a decrease in resting and submaximal exercise heart rate; an increase in the heart's stroke volume, an increase in maximal cardiac output; and an increased arterial-venous O_2 difference (McArdle et al., 1991).

5.3 Recovery Rate and Training

Researchers have demonstrated that aerobic endurance training can improve the rate of the recovery VO_2 (Hagberg et al., 1980) . Hagberg et al. (1980) showed a decreased time to recovery at both the same absolute and relative workloads after training. These researchers analyzed the recovery curves as a single exponential and reported half-times of 23 to 35 seconds for work rates of 50% and 70% VO_{2max} before training and 58% (previously 70%) and 70% after training. Hagberg et al. (1980) reported a 21.03% reduction in recovery rate half-times, from 31.0 s to 23.3 s, for the same absolute work performed pre and post training (50% of VO_{2max}). As well, a 24.79% reduction in recovery rate half times, from 35.5 s to 26.7 s, was found for the same absolute 70% VO_{2max} workload administered pre and post training. In the present study, the recovery curves were fit to double exponential equations. A decrease in the rates of both the fast component rate constant (τ_1) and the slow component rate constant (τ_2) were observed post-training in this study. The pre-training fast component rate constant (τ_1) was computed as 2.69 $\pm$ .19 min ($t_{1/2}$ = 1.86 $\pm$ .13 min) and the post-training fast component rate constant was found to be 2.29 $\pm$ .33 min ($t_{1/2}$ = 1.59 $\pm$ .23 min). These values are slightly higher than others reported in the literature; though, the majority of research on recovery VO_2 kinetics has focused on recovery

from submaximal exercise conditions. In fact a trend can be found for longer (slower) fast component recovery rates for workloads which require an energy release higher than maximal VO_2 uptake and for those which also involve a sustained lactic acidosis. In the present study the subjects were working at supramaximal exercise intensities and induced a significant lactic acidosis. This may explain the longer recovery rates reported in the present study as compared to other values reported in the literature.

Through metabolic modeling the fast component VO_2 recovery rate has been linked to PCr resynthesis. If a link exists between O_2 consumption and [PCr] then recent research on PCr kinetics indicates that the VO_2 recovery rate is slower the more intense the exercise. McCann et al. (1995) reported that the rate of recovery of [PCr] is slower for intense than light or moderate exercise.

The pre-training slow component rate constant (τ_2) was computed as 43.74 $\pm$ 5.12 min ($t_{1/2}$ = 30.32 $\pm$ 3.55) and the post-training slow component rate constant was found to be 39.63 $\pm$ 4.24 min ($t_{1/2}$ = 27.47 $\pm$ 2.86 min). Comparison of slow component recovery rates in the literature yields a wide range of values. The discrepancies in the literature may be linked to the varying intensities and durations utilized in the preceding exercise conditions. Numerous investigators have demonstrated a significant slow component of the recovery VO_2 kinetics that becomes more prominent the higher the preceding work rate (Margaria et al., 1933; Knuttgen, 1962; Davies et al., 1972). This supports the findings in the present study. Subjects in this study ran on a treadmill at an individually determined intensity to elicit fatigue at two minutes. Exercise of this intensity requires an energy release greater than maximal VO_2 uptake and is known to produce high levels of lactic acid. Though debate continues regarding the effect of lactate on the recovery VO_2,

research has indicated that although it is not solely responsible for the VO_2 uptake in recovery from exercise, it does have an influence. Researchers have recently reported significant relationships between peak blood lactate at the end of exercise and the resultant EPOC (Bahr et al., 1992; Frey et al., 1993; Rhodes and Roberts, 1994). The slow component of the recovery VO_2 has been suggested to be a result of several factors, including elevated concentrations of catecholamines, increased substrate cycling rate (substrate synthesis), increased body temperature, tissue repair and associated increased protein synthesis, ionic redistribution, and the thermic effect of food (Gaesser and Brooks, 1984; Gore and Withers, 1990a).

In the present study the changes in recovery rate after training correspond to a 14.86% decrease in τ_1 and a 9.40% decrease in τ_2. In general, VO_2 kinetics follow changes in chronic usage of the muscles, speeding with training (Casaburi et al., 1992; Hagberg et al., 1980) and slowing with bed rest (Convertino et al., 1984). A greater training stimulus would, theoretically, result in greater changes in VO_2 kinetics. This would explain the greater change in VO_2 kinetics in the study by Hagberg et al. (1980), corresponding with a greater training stimulus. In the Hagberg study, subjects exercised at a high intensity for 30-40 minutes per session, 6 days per week, for 9 weeks. In the present study, subjects exercised at an intensity near threshold for 30 minutes per session, 3 days a week, for 6 weeks. The faster VO_2 kinetics observed after training are consistent with improved O_2 delivery to, and/or enhanced aerobic capacity of the contracting muscles during and after exercise. Endurance exercise training typically results in improvements in exercise tolerance and these results are thought to be due to a combination of central (cardiac) and peripheral (skeletal muscle) adaptations, which result in a greater capacity to deliver O_2 from the lungs to the mitochondria of the contracting muscles (Barstow et al., 1996).

The mechanisms governing the changes in VO_2 kinetics following training can be linked to training induced changes in the biochemical utilization of O_2 as well as changes in O_2 transport. From a cellular O_2 utilization standpoint, specific mitochondria changes take place with training. Holloszy and Coyle (1984) have indicated that mitochondria from trained skeletal muscle show a greater capacity to generate ATP aerobically by oxidative phosphorylation. Other studies have documented an increase in both the size and number of mitochondria and a potential twofold increase in the level of aerobic system enzymes after training (Barnard et al., 1970).

The importance of the mitochondria changes to the faster VO_2 kinetics following training have been indicated by research on VO_2 kinetics in different muscle fiber types. The underlying fiber type composition of the contracting muscles has in fact been shown to have implications to determining the time constant for the rate of muscle O_2 consumption (QO_2). Type I fibers exhibit characteristics (such as more mitochondria and capillary density), consistent with a faster time constant than Type II (especially IIb) fibers (Vrbova, 1979). The specificity of the VO_2 time constant to fiber type has also been indicated by research on PCr dynamics. Kushmerick et al. (1992) have shown that the change in PCr for a given increase in QO_2 is also greater in Type IIb than Type I fibers.

In terms of O_2 transport, central (cardiac output) as well as peripheral (skeletal muscle) blood flow changes take place with training. It is well documented that significant increases in cardiac output take place with training. The heart's increased outflow capacity after training results directly from an improved stroke volume. Some peripheral blood flow adaptations, more closely associated with skeletal muscle, also take place with training. Skeletal muscle capillarity, the capillary to fiber ratio and the capillary density (approx. 20%) have been shown to be

increased with endurance training (Ingjer, 1979). As well, changes in muscle blood flow occur with training. These blood flow changes have been indicated to result from altered control of resistance arterioles, which modulate redistribution of blood flow to active skeletal muscle during exercise (Martin et al., 1989).

A number of studies have documented the importance of blood flow changes to VO_2 kinetics. It has been shown that slowing of the kinetics of circulatory adjustments with β-blockade (Petersen et al., 1983) or reducing arterial O_2 content with hypoxia (Springer et al., 1991) or carbon monoxide breathing (Koike et al., 1990) results in slower kinetics for VO_2. Longer time constants for VO_2 are also found in patients with cardiovascular (Zhang et al., 1993) and pulmonary disease (Nery et al., 1982). These conditions may represent variable mixtures of reduced muscle oxidative capacity or reduced O_2 delivery.

5.4 EPOC Magnitude

The majority of research on EPOC magnitude has focused on measuring VO_2 uptake after prolonged submaximal exercise. Few studies have measured the recovery VO_2 following short bouts of supramaximal exercise involving a significant anaerobic contribution and high levels of blood lactate (Hermansen, 1969; Bahr et al., 1992; Rhodes and Roberts, 1994).

Bahr et al. (1992) compared EPOC values from three separate intermittent two minute exercise bouts on a cycle ergometer at 108% VO_{2max}. They measured EPOC one hour into recovery and reported values of 7.8 ± 0.7 L (3 * 2 min.), 6.7 ± 0.4 L (2 * 2 min.), and 5.6 ± 0.4 L (1 * 2 min.). These values are much lower than the EPOCs recorded in this study. In this study, pre-training EPOCs of 9.13 ± 1.68 L and post training EPOCs of 7.49 ± 1.73 L were recorded

after 30 minutes of recovery from a supramaximal 2min AST. The higher EPOCs in this study are most likely attributed to the larger muscle mass engaged in the AST running test as opposed to the smaller muscle mass involved in the cycling protocol used by Bahr et al. (1992).

In comparison, Rhodes and Roberts (1994) found larger EPOCs from four separate sprint conditions involving either an isokinetic device (APM) or free sprinting. They reported 30 minute EPOCs of 15.16 ± 2.59 L, 11.38 ± 2.72 L, 9.88 ± 2.80 L, and 9.09 ± 2.51 L for a 2min AST, 5 APM, 10 free, and 5 free, respectively. Gitto et al. (1996) recorded 30 minute EPOCs of 9.29 ± 2.22 L, 9.04 ± 2.03 L, and 7.56 ± 1.13 L for 2 min, 20%, 15% - 1 minute ASTs, respectively. The 2 min ASTs used in these studies as well as in the present study were administered identically. The larger 2 min EPOCs found by Rhodes and Roberts (1994) and by Gitto et al. (1996) most likely are attributed to the subject pools; eight highly trained sprinters and a group of elite soccer players, respectively. Both sets of subjects would most likely have a larger muscle mass and a greater lactate tolerance in comparison to the subjects used in this study.

Endurance training has been shown to have a significant effect on the volume of O_2 consumed in recovery from exercise (Hagberg et al., 1980). Hagberg et al. (1980) compared pre and post training EPOC values after nine weeks of aerobic endurance training and found that total EPOC magnitude was reduced at the same absolute workload after training. They recorded ten minute pre and post training EPOC values at 50% and 70% of VO_{2max} and found values of 0.70 ± 0.07 L and 1.41 ± 0.15 L, respectively. Ten minute EPOC values were then recorded at the same absolute work rates post training and yielded values of 0.49 ± 0.04 L and 1.04 ± 0.07 L. These post-training values correspond to a reduction in EPOC magnitude of 30% and 26.21%, respectively. The larger EPOC values recorded in the present study are probably linked to the

longer time for which EPOC was recorded, the higher intensity of the preceding exercise condition and the running as opposed to a cycling protocol. In the present study, a reduction in EPOC magnitude from pre to post training of 17.96% was found. The larger percentage difference in EPOC values between pre and post training in the Hagberg study as compared to the present study may be attributed to a greater training stimulus. In the study by Hagberg et al. (1980) subjects trained at a high intensity, six days a week for 30-40 minutes a session. The training program in the present study was not as frequent or intense as the one in the study by Hagberg et al. (1980). Subjects in the present study exercised at an intensity near threshold for three days a week at 30 minutes per session.

The discrepancies in the EPOC values reported in previous literature and those recorded in this study may be attributed to the preceding exercise conditions for which EPOC was measured as well as the length of time that EPOC was recorded. The intensity and duration of the preceding exercise condition have been shown to have a significant affect on the resultant recovery VO_2. Recently, Sedlock (1994) indicated that the changes in magnitude to EPOC are generally a function of the preceding relative exercise intensity (the percentage of VO_{2max} of the preceding exercise). A few researchers have found a threshold intensity which must be reached in order to produce significant elevations in EPOC. Gore and Withers (1990a and b) and Bahr and Sejersted (1991a and b), estimated the effect of exercise intensity on EPOC to be associated with a >50% VO_{2max} threshold value while Hagberg et al. (1980) estimated the threshold slightly higher at >65% VO_{2max}. At these intensities a linear relationship is observed between duration of exercise and EPOC (Hagberg et al., 1980; Bahr et al., 1987; Gore and Withers, 1990a and b; Frey et al., 1993). Interestingly, Bahr and Sejersted (1991a and b) suggest the exercise intensity threshold

associated with a prolonged EPOC may possibly equal the lactate threshold. They indicate that the exercise intensity threshold responsible for the prolonged EPOC seems to occur near the percentage of VO_{2max} that is commonly associated with an untrained individual's anaerobic threshold. In the present study, subjects were working upwards of 100% of their VO_{2max}; much higher than threshold. This may explain the much larger EPOC values recorded in this study compared to a number of earlier studies which focused on recovery from submaximal exercise conditions. The EPOC magnitude values reported in this study are within a comparable range to those found in some more recent work utilizing supramaximal exercise conditions (Bahr et al., 1992; Rhodes and Roberts, 1994; Gitto and Rhodes, 1996).

A prolonged EPOC component has been observed for as much as 8-48 hours into recovery (Bahr et al., 1987; Maehlum et al., 1986; Gore and Withers, 1990a), although, much of the debate over the length of EPOC may be attributed to different methodologies used in the experimental procedures. Unfortunately, evidence in support of a prolonged EPOC component cannot be taken from this study. For the purpose of this study, EPOC was only monitored for 30 minutes into recovery. At this time though, no subject had reached their asymptotic recovery VO_2 baseline. The prolonged EPOC component has been suggested to be a result of several factors, including elevated concentrations of catecholamines, increased substrate cycling rate (substrate synthesis), increased body temperature, tissue repair and associated increased protein synthesis, ionic redistribution, and the thermic effect of food (Gaesser and Brooks, 1984; and Gore and Withers, 1990a).

5.5 Lactate

Early research indicated that blood lactate was almost exclusively responsible for the elevated VO_2 seen in recovery from exercise (Hill and Lupton, 1923; Margaria et al., 1933). The "O_2 debt" hypothesis explaining the recovery VO_2 was based on the assumption that the primary fate of lactate in recovery from exercise was gluconeogenesis. Eighty percent of the lactate accumulated during exercise was thought to be reconverted to glycogen and 20% oxidized to CO_2 and H_2O. More recently, Gaesser and Brooks (1984) determined that post exercise 55-70% of the accumulated lactate was oxidized to CO_2 and H_2O, while less than 20% of the lactate was converted to muscle and liver glycogen. Their research implied that lactate was not so predominantly responsible for the recovery VO_2 and that the factors affecting VO_2 in recovery from exercise reflect a general metabolic disturbance to the body.

Inconsistencies in the literature still remain regarding the effects of lactate on the recovery VO_2. Some recent research has indicated significant relationships between peak blood lactate at the end of exercise and the resultant EPOC; Bahr et al. (1992), $r = 0.86$, $p < .0005$, Frey et al. (1993), $r = 0.90$, $p < 0.05$, and Rhodes and Roberts (1993), $r = 0.87$, $p < 0.05$. Although high correlations were found in these studies between peak blood lactate and EPOC, these researchers contend that a large portion of EPOC cannot be accounted for by lactate removal (Bahr et al., 1992: Frey et al., 1993). Rhodes and Roberts (1994), in fact state that peak blood lactate at the end of exercise and the resultant EPOC may be spuriously related by the effect of the intensity of the preceding exercise condition.

In the present study, no significant relationships were found between lactate and EPOC. Several other investigators have also found no significant relationships between blood lactate and

EPOC (Knuttgen, 1970; Bahr and Sejersted, 1991a and b; Roth et al., 1988). In fact, Roth et al. (1988), found no significant effect on EPOC through manipulating end exercise blood lactate levels. Interestingly, Knuttgen (1970), was able to show substantial slow component EPOC magnitudes in recovery from exercise eliciting small blood lactate changes. These results contradict early work which indicated that blood lactate concentration was predominantly responsible for the recovery VO_2 after exercise. In the present study the mean peak blood lactates derived from the 2min ASTs were 15.28 $mmol \cdot L^{-1}$ (pre training) and 13.36 $mmol \cdot L^{-1}$ (post training). These values are similar to those reported in other high intensity/short duration tests from other studies; Medbo et al., (1988), 13.4 $mmol \cdot L^{-1}$; Medbo and Burgers (1990), 14.9 $mmol \cdot L^{-1}$; Rhodes and Roberts (1994), 14.83 $mmol \cdot L^{-1}$; and Gitto and Rhodes (1996), 15.74. In comparing data from this study and that of Rhodes and Roberts (1994) and Gitto and Rhodes (1996), lactate values were within 2.38 $mmol \cdot L^{-1}$ while there was a 7.67 Litre difference in EPOC values.

More recent research on the recovery VO_2 has shown that EPOC reflects a general metabolic disturbance to the body and that a number of physiological mechanisms increase their energy demands during exercise and into recovery and these systems contribute to the recovery VO_2. Bangsbo et al. (1990) has indicated that other tissues as well as muscle must be responsible for the recovery VO_2 because whole body EPOC is much greater than can be accounted for by local muscle events.

Physiological adaptations take place during training which would correspond with lower blood lactate levels for a set absolute workload administered post training (McArdle et al., 1991). The decreased blood lactate concentration post training may reflect lower lactate production

and/or a greater lactate removal rate. In fact, Karlsson et al. (1972) have shown muscle lactate concentration to be decreased at the same absolute and relative submaximal work rates following training. These authors indicate that at the subcellular level one of the biochemical properties responsible for the recovery VO_2 is decreased in the trained state. In the present study a decrease in blood lactate concentration was observed post training. A pre-training blood lactate concentration of 15.28 ± 1.80 mmol·L^{-1} and a post training blood lactate concentration of 13.36 ± 1.55 mmol·L^{-1} were recorded after a 2 min supramaximal exercise test. This corresponds to a 12.57% change in blood lactate concentration from pre to post training.

It is interesting to note that some of the physiological training adaptations associated with blood lactate and EPOC changes would correspond with Brook's "Lactate Shuttle" hypothesis. Brooks (1986) contends that a major fraction of the lactate produced during exercise may be oxidized within the same active muscle bed by Type I fibers. Type I fibers have in fact been shown to possess the respiratory capacity sufficient to oxidize large quantities of lactate (Bonen and Belcastro, 1976; Brooks, 1986; Mazzeo et al., 1986; Stanley, 1991). Lactate within these fibers is metabolized in the Krebs Cycle via reconversion to pyruvate. Specific endurance training is known to increase both the size and number of mitochondria as well as increase the level of aerobic system enzymes. (Barnard et al., 1970). Theoretically, a training induced increase in the size and number of mitochondria as well as an increase in aerobic system enzymes would allow for the capacity to oxidize more lactate post training. Brooks (1986) has also shown that inactive skeletal muscle takes up circulating lactate during exercise of other muscle groups.

5.6 Relationships

Early research indicated that there were individual differences in the ability to recover from exercise. Berg (1947) recognized that trained individuals exhibited faster VO_2 recovery rates from moderate exercise. Henry and Demoor (1950 and 1956) stated that the recovery rate constants exhibit reliable individual differences and are altered by factors such as athletic training. Since this time, cross sectional studies have provided inconsistent results on the training-recovery relationship. Some cross sectional studies have shown faster kinetics in trained vs. sedentary individuals (Cerretelli et al., 1979), higher VO_{2max} when examining individuals differing in VO_{2max} (Ebfeld et al., 1987; Zhang et al., 1991), and athletes differing in VO_{2max} (Powers et al., 1985). Other cross sectional studies provide inconsistent results to the literature, suggesting there is no relationship between VO_{2max} and recovery VO_2 kinetics. Chad and Quigley (1991) failed to find a significant difference in the rate of the recovery VO_2 uptake between trained and untrained females after a 30 minute cycle ergometer test. Bell et al. (1997) examined the relationship between aerobic fitness and the recovery VO_2 kinetics from high intensity intermittent exercise in a cross sectional sample of endurance trained cyclists. They reported no significant correlations between aerobic power (VO_{2max}) and between the fast (τ_1) and slow (τ_2) rates of recovery VO_2. These authors indicate that the lack of relationships between the measurements of aerobic fitness and metabolic recovery after the high-intensity, intermittent exercise bouts suggests that the physiological factors underlying the different assessments of aerobic fitness are probably too diverse to be used as indicators for the ability to recover from high-intensity, intermittent exercise. In the present study there were no correlations found between VO_{2max} and τ_1 and τ_2 when

examining the data as a cross sectional sample. There was, however, a trend for higher aerobic powers being associated with faster recovery rates. It is possible that the relatively small sample size and the homogeneity of the group did not allow for this relationship to be found. The results from this study support some of the recent work on the recovery rate-training status relationship in failing to indicate a relationship between an individuals rate of recovery and their level of aerobic fitness (VO_{2max}).

In the present study, correlations made between the change in VO_{2max} and the change in the recovery rate constants also failed to provide any significant relationships. Although significant differences were found between pre and post training VO_{2max} and pre and post training τ_1 and τ_2, those subjects who made the greatest change in VO_{2max} did not necessarily have the greatest change in recovery rate. There was, however, a trend in the data for those individuals who made a larger change in VO_{2max} from pre to post training to make a larger change in the recovery rates from pre to post training. Again, it is possible that the relatively small sample size and the homogeneity of the group did not allow for this relationship to be found. In the present study, there was a 10.22% increase in VO_{2max}, a 14.87% decrease in τ_1VO_2, and a 9.40% decrease in τ_2VO_2 for recovery oxygen consumption. The relatively small differences and small changes in VO_{2max} and τVO_2's in the present study may also have been another reason for finding no significant correlation's between VO_{2max} and τVO_2. The results from this study are consistent with some recent work on VO_2 kinetics and training. Recent work has focused on the VO_2 uptake at the start of exercise in specific populations. Babcock et al. (1994) and Barstow et al. (1996) documented large differences in both pre and post training VO_{2max} and pre and post training τVO_2 at the onset of exercise. Babcock et al. (1994) reported a 20% increase in VO_{2max} and showed a

significant decrease (48.7%) in τ for O_2 uptake kinetics at the start of exercise. Barstow et al. (1996) showed an 11% increase in VO_{2max} and a 25.94% decrease in τVO_2 at the start of exercise. Although these differences were found in the data, no correlation's were found between the decrease in τVO_2 and the increase in VO_{2max}. Barstow et al. (1996) also examined the change in the recovery rate of VO_2 uptake in relation to the increase of VO_{2max} after the training program. No correlation's were found between the decrease in the recovery τVO_2 (19.60%) and the increase in VO_{2max} (11%).

5.7 Summary

The present study demonstrated that aerobic training can decrease the rate and magnitude of the recovery VO_2 as well as decrease the blood lactate response associated with an absolute supramaximal work bout. These results suggest that the adaptive response(s) of the aerobic energy system with training allows for more efficient recovery from the preceding exercise condition. The results also demonstrate that the recovery VO_2 from supramaximal work appears to be independent of VO_{2max}. The findings from this study as well as those from other recent research suggests that the numerous physiological factors underlying the assessment of VO_{2max} are probably too diverse to allow for this single measure of aerobic power to be used as an indicator of recovery VO_2 ability (Barstow et al., 1996; Bell et al., 1997). Changes in the rates and volume of the recovery VO_2 may be more closely related to changes in the oxidative capacity of mitochondria within specific muscles that to whole body oxygen consumption as reflected by VO_{2max}.

BIBLIOGRAPHY

Anderson, P., Henrickson, J. (1977) Capillary supply of the quadriceps femoris muscle of man: adaptive response to exercise. Journal of Physiology, 270, 677-690.

Anderson, P., Saltin, B. (1985). Maximal perfusion of skeletal muscle in human. Journal of Physiology (London), 366, 233-249.

Babcock, M.A., Paterson, D.H., Cunningham, D.A. (1994). Effects of aerobic endurance training on gas exchange kinetics of older men. Medicine and Science in Sports and Exercise, 26 (4), 447-452.

Bangsbo, J., Gollnick, P.D., Graham, T.E., Juel, C., Kiens B., Mizuno, M., Saltin, B. (1990). Anaerobic energy production during exhaustive exercise in humans. Journal of Physiology, 422, 539-559.

Bahr, R., Ingnes, I., Vaage, O., Sejersted, O.M., Newsholme, E.A. (1987). Effect of duration of exercise on excess postexercise O_2 consumption. Journal of Applied Physiology, 62 (2), 485-490.

Bahr, R., Hansson P., Sejersted, O.M. (1990a). Triglyceride/fatty acid cycling is increased after exercise. Metabolism, 39 (9), 993-999.

Bahr, R., Opstad, P.K., Medbo, J.I., Sejersted, O.M. (1990b). Strenuous prolonged exercise elevates resting metabolic rate and causes reduced mechanical efficiency. Acta Physiologica Scandinavica, 114, 555-563.

Bahr, R., Sejersted, O.M. (1991a). Effect of intensity of exercise on excess postexercise O_2 consumption. Metabolism, 40 (8), 836-841.

Bahr, R., Sejersted, O.M. (1991b). Effect of feeding and fasting on excess postexercise oxygen consumption. Journal of Applied Physiology, 71 (6), 2088-2093.

Bahr, R., Gronnerod, O., Sejersted, O.M. (1992). Effect of supramaximal exercise on excess postexercise O_2 consumption. Medicine and Science in Sports and Exercise, 16(1), 29-43.

Barnard, R.J., Foss, M.L., Tipton, C.M. (1970). Oxygen debt: involvement of the Cori cycle. Int. Z. angew. Physiol. 28:105-119.

Barstow, T.J., Scremin, A.M.E., Mutton, D.L., Kunkel, C.F., Cagle, T.G., Whipp, B.J. (1996). Changes in gas exchange kinetics with training in patients with spinal cord injury. Medicine and Science in Sports and Exercise, 28 (10), 1221-1228.

Barstow T.J. (1994). Characterization of VO_2 kinetics during heavy exercise. Medicine and Science in Sports and Exercise, 26 (11), 1327-1334.

Bell, G.J., Snydmiller, G.D., Davies, D.S., Quinney, H.A. (1997). Relationship between aerobic fitness and metabolic recovery from intermittent exercise in endurance athletes. Canadian Journal of Applied Physiology, 22 (1), 78-85.

Berg, K.E. (1991). Comparison of energy expenditure in men and women at rest and during exercise recovery. The Journal of Sports Medicine and Physical Fitness, 31 (3), 351-356.

Berg, W.E. (1947). Individual differences in respiratory gas exchange during recovery from moderate exercise. American Journal of Physiology, 149, 597-610.

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

Brehm, B.A., Gutin, B. (1986). Recovery energy expenditure for steady state exercise in runners and nonexercisers. Medicine and Science in Sports and Exercise, 18 (2), 205-210.

Brooks, G.A. (1991). Current concepts in lactate exchange. Medicine and Science in Sports and Exercise, 23, (8), 895-906.

Brooks, G.A. (1986). The lactate shuttle during exercise and recovery. Medicine and Science in Sports and Exercise, 18, (3), 360-368.

Casaburi, R., Barstow, T.J., Robinson, T., Wasserman, K. (1992). Dynamic and steady-state ventilatory and gas exchange responses to arm exercise. Medicine and Science in Sports and Exercise, 24, 1365-1374.

Cerretelli, P., Pendergast, D., Paganelli, W.G., Rennie, D.W. (1979). Effects of specific muscle training on VO_2 on-response and early blood lactate. Journal of Applied Physiology, 47, 761-769.

Cerretelli, P., diPrampero, P.E., Piiper, J. (1969). Energy balance of anaerobic work in the dog gastrocnemius muscle. American Journal of Physiology, 217 (2), 581-585.

Chad, K.E., Quigley, B.M. (1991). Exercise intensity: effect on post exercise oxygen uptake in trained and untrained women. Journal of Applied Physiology, 70 (4), 1713-1719.

Chad, K.E., Wenger, H.A. (1985). The effects of duration and intensity on the exercise and post exercise metabolic rate. The Australian Journal of Science and Medicine in Sport, 17 (4), 14-18.

Chad, K.E., Wenger, H.A. (1988). The effect of exercise duration on the exercise and post-exercise oxygen consumption. Canadian Journal of Sport Sciences, 13 (4), 204-207.

Convertino, V.A., Goldwater, D.J., Sandler, H. (1984). VO_2 kinetics of constant-load exercise following bed-rest-induced deconditioning. Journal of Applied Physiology, 57, 1545-1550.

Cortes, C.W., Kreider, R.B., Al-Mandalawi, S., Lamberth, J., Johnson, K.D., Thompson, W.R., Anderson, H.N. (1988). Lactate clearance during active and passive recovery. Annals of Sports Medicine, 4, (1), 26-28.

Cotes, J.E., Meade, F. (1959). Physical training in relation to the energy expenditure of walking and factors controlling respiration during exercise. Ergonomics, 2, 195-205.

Crescitelli, F., Taylor, C. (1944). The lactate response to exercise and its relationship to physical fitness. American Journal of Physiology, 141, 630-639.

Cunningham, D.A., Faulkner, J.A. (1969). The effect of training on aerobic and anaerobic metabolism during a short exhaustive run. Medicine and Science in Sports and Exercise, 1, 65-69.

Davies, C.T.M., diPrampero, P.E., Cerretelli, P. (1972). Kinetics of cardiac output and respiratory gas exchange during exercise and recovery. Journal of Applied Physiology, 32 (5), 618-625.

Diprampero, P.E., Margaria, R. (1968). Relationship between O_2 consumption, high energy phosphates and the kinetics of the O_2 debt in exercise. Pflugers Archiv European Journal of Physiology, 304, 11-19.

Diprampero, P.E., Peeters, L., Margaria, R. (1973). Alactic O_2 debt and lactic acid production after exhausting exercise in man. Journal of Applied Physiology, 34 (5), 628-632.

Douglas, F.G., Becklake, M.R. (1968). Effect of seasonal training on maximal cardiac output. Journal of Applied Physiology, 25, 600-605.

Ebfold, C., Hoffman, U., Stegemann, J. (1987). VO_2 kinetics in subjects differing in aerobic capacity: investigation by spectral analysis. European Journal of Applied Physiology. 56, 508-515.

Elliot, D.L., Goldberg, L., Kuehl, K.S. (1988). Does aerobic conditioning cause a sustained increase in the metabolic rate? American Journal of Medicine and Science, 296 (4), 249-251.

Freedman-Akabas, S., Colt, E., Kissileff, H.R., Pi-Sunyer, X.F. (1985). Lack of sustained increase in VO_2 following exercise in fit and unfit subjects. American Journal of Clinical Nutrition, 41, 545-549.

Freund, H., Gendry, P. (1978). Lactate kinetics after short strenuous exercise in man. European Journal of Applied Physiology, 39, 123-135.

Frey, G.C., Byrnes, W.C., Mazzeo, R.S. (1993). Factors influencing excess post-exercise oxygen consumption in trained and untrained women. Metabolism, 42 (7), 822-828.

Gaesser, G.A., Brooks, G.A. (1984). Metabolic bases of excess post-exercise oxygen consumption: a review. Medicine and Science in Sports and Exercise, 16, (1), 29-43.

Gaesser, G.A., Poole,

Girandola, R.N., Katch, F.I. (1973). Effects of physical conditioning on changes in exercise recovery O_2 uptake and efficiency during constant-load ergometer exercise. Medicine and Science in Sports and Exercise, 5 (4), 242-247.

Gollnick, P.D., Bayly, W.M., Hodgson, D.R. (1986). Exercise intensity, training, diet and lactate concentration in muscle and blood. Medicine and Science in Sports and Exercise, 18, 334-340.

Gore, C.J., Withers, R.T. (1990a). Effect of exercise intensity and duration on postexercise metabolism. Journal of Applied Physiology, 68 (6), 2362-2368.

Gore, C.J., Withers, R.T. (1990b). The effect of exercise intensity and duration on the oxygen deficit and excess post-exercise oxygen consumption. European Journal of Applied Physiology, 60, 169-174.

Hagberg, J.M., Hickson, R.C., Ehsani, A.A., Holloszy, J.O. (1980). Faster adjustment to and recovery from submaximal exercise in the trained state. Journal of Applied Physiology, 48 (2), 218-224.

Hagberg, J.M., Mullin, J.P., Nagle, F.J. (1980). Effect of work intensity and duration on recovery O_2. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology, 48 (3), 540-544.

Harris R.C., Edwards, R.H.T., Hultman, E., Nordesjo, L.-O., Nylind, B., Sahlin, K. (1976). The time course of phosphorylcreatine resynthesis during recovery of the quadriceps muscle in man. Pflugers Archiv European Journal of Physiology, 367, 137-142.

Henry, F.M., Berg, W.E. (1950). Physiological and performance changes in athletic conditioning. Journal of Applied Physiology, 3, 103-111.

Henry, F.M., DeMoor, J.C. (1950). Metabolic efficiency of exercise in relation to work load at constant speed. Journal of Applied Physiology, 2 (9), 481-487.

Henry, F.M., DeMoor, J.C. (1956). Lactic and alactic oxygen consumption in moderate exercise of graded intensity. Journal of Applied Physiology 8 (6), 608-614.

Hermansen L. (1969). Anaerobic energy release. Medicine and Science in Sports, 1 (1), 32-38.

Hill, A.V., Lupton, H. (1923). Muscular exercise, lactic acid and the supply and utilization of oxygen. Q.J. Med., 16, 135-171.

Hickson, R.C., Bomze, H.A., Holloszy, J.O. (1981) Time Course of the adaptive responses of aerobic power and heart rate to training. Medicine and Science in Sports and Exercise, 13

Holloszy, J.O., Coyle, E.F. (1984). Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. Journal of Applied Physiology, 56 (4), 831-838.

Huckabee, W.E. (1958a). Relationships of pyruvate and lactate during anaerobic metabolism. I. Effects of infusion of pyruvate or glucose and of hyperventilation. Journal of Clinical Investigations. 37, 244-254.

Huckabee, W.E. (1958b). Relationships of pyruvate and lactate during anaerobic metabolism. II. Exercise and formation of O_2 debt. Journal of Clinical Investigations. 37, 255-263.

Huckabee, W.E. (1958c). Relationships of pyruvate and lactate during anaerobic metabolism. III. Effect of breathing low-oxygen gases. Journal of Clinical Investigations. 37, 264-271.

Hughson, R.L. (1990). Exploring cardiorespiratory control mechanisms through gas exchange dynamics. Medicine and Science in Sports and Exercise, 22 (1), 72-79.

Ingjer, F. (1979). Effects of endurance training on muscle fibre ATP-ase activity, capillary supply and mitochondrial content in man. Journal of Physiology, 294, 419-432.

Kaminsky, L.A., Kanter, M.M., Lesmes, G.R., Laham-Saeger, J. (1987). Excess oxygen consumption following exercise of different intensity and duration. Canadian Journal of Sport Sciences, 12 (4), 237-239.

Kaminsky, L.A., Padjen, S., Lattam-Saeger, J. (1990). Effect of split exercise sessions on excess post-exercise oxygen consumption. British Journal of Sports Medicine, 24 (2), 95-98.

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

Karlsson, J., Nordesjo, L.O., Jorfeldt, L., Saltin, B. (1972). Muscle lactate, ATP and CP in working muscles during exhaustive exercise in man. Journal of Applied Physiology, 19, 598-602.

Katch, V.L. (1973). Kinetics of oxygen uptake and recovery for supramaximal work of short duration. International Z. angew, Physiology, 31, 197-207.

Katch, V.L., Gilliam,T., Weltman, A. (1978). Active versus passive recovery from short-term supramaximal exercise. The Research Quarterly, 49 (2), 153-161.

Knehr, C.A., Dill, B., Neufeld, W. (1942). Training and its effects on man at rest and at work. American Journal of Physiology, 136, 148-156.

Knuttgen, H.G. (1962). Oxygen debt, lactate, pyruvate and excess lactate after muscular work. Journal of Applied Physiology, 17 (4), 639-644.

Knuttgen, H.G. (1970). Oxygen debt after submaximal physical exercise. Journal of Applied Physiology, 29(5), 651-657.

Knuttgen, H.G., Saltin, B. (1972). Muscle metabolites and oxygen uptake in short-term submaximal exercise in man. Journal of Applied Physiology, 32, 690-694.

Koike, A., Wasserman, K., McKenzie, D., Zanconato, S., Weiler-Ravell, D. (1990). Evidence that diffusion limitation determines O_2 uptake kinetics during exercise in man. Journal of Clinical Investigations, 86, 1698-1706.

Kushmerick, M.J., Meyer, R.A., Brown, T.R. (1992). Regulation of oxygen consumption in fast- and slow- twitch muscle. American Journal of Physiology, 263: C598-C606.

MacRae, H.S-H., Dennis, S.C., Bosch, A.N., Noakes, T.D. (1992). Effect of training on lactate production and removal during progressive exercise. Journal of Applied Physiology, 72, 1649-1656.

Martin, W.H., Coggan, A.R., Spina, R.J., Saffitz, J.E. (1989). Effects of fiber type and training on β-adrenoceptor density in human skeletal muscle. American Journal of Physiology (Endocrinol. Metab. 20) E736-E742.

McArdle, W.D., Katch, F.I., Katch, V.L. (1991). Exercise Physiology - Energy, Nutrition, and Human Performance. Lea and Febiger (3rd edition).121-137.

Medbo, J.I., Burgers, S. (1990). Effect of training on the anaerobic capacity. Medicine and Science in Sports and Exercise, 22, 501-507.

Medbo, J.I., Mohn, A.C., Tabata, I., Bahr, R., Vaage, O., Sejersted, O.M. (1988). Anaerobic capacity determined by maximal accumulated O_2 deficit. Journal of Applied Physiology, 64 (1), 50-60.

Maehlum, S., Grandmontagne, M., Newsholme, E.A., Sejersted, O.M. (1986). Magnitude and duration of exercise postexercise oxygen consumption in healthy young subjects. Metabolism, 35 (5), 425-429.

Mahler, M., Homsher, E. (1982). Metabolic rate changes in parallel with creatine level during non-steady states in frog skeletal muscle. Fed. Proc. 41, 979.

Maresh, C.M., Abraham, A., DeSouza, M.J. Deschenes, M.R., Kraemer, W.J., Armstrong, L.E., Maguire, M.S., Gabaree, C.L., Hoffman, J.R. (1992). Oxygen consumption following exercise of moderate intensity and duration. European Journal of Applied Physiology, 65, 421-426.

Margaria, R., Cerretelli, P., diPrampero, P.E. Massari, C., Torelli, G. (1963). Kinetics and mechanisms of oxygen debt contraction in man. Journal of Applied Physiology, 18 (2), 371-377.

Margaria, R., Edwards, H.T. (1934a). The sources of energy in muscular work performed in anaerobic conditions. American Journal of Physiology, 108, 341-348.

Margaria, R., Edwards, H.T. (1934b). The removal of lactic acid from the body during recovery from muscular exercise. American Journal of Physiology, 107, 681-686.

Margaria, R., Edwards, H.T., Dill, D.B. (1933). The possible mechanisms of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction. American Journal of Physiology, 106, 689-715.

Mazzeo, R.S., Brooks, G.A., Schoeller, D.A., Budinger, T.F. (1986). Disposal of blood lactate in humans during rest and exercise. Journal of Applied Physiology, 1, 232-241.

McCann, D.J., Mole, P.A., Caton, J.R. (1995). Phoshphocreatine kinetics in humans during exercise and recovery. Medicine and Science in Sports and Exercise, 27 (3), 378-387.

McLellan, T.M., Skinner, J.S. (1982). Blood lactate removal during active recovery related to the anaerobic threshold. International Journal of Sports Medicine, 3, (4), 224-229.

McLoughlin,P., McCaffery, N., Moynihan, J.B. (1991). Gentle exercise with a previously inactive muscle group hastens the decline of blood lactate concentration after strenuous exercise. European Journal of Applied Physiology, 62, 274-278.

Nery, L.E., Wasserman, K., Andrews, D.J., Huntsman, J., Hansen, E., Whipp, B.J. (1982). Ventilatory and gas exchange kinetics during exercise in chronic airways obstruction. Journal of Applied Physiology, 56, 1594-1602.

Paterson, D.H., Whipp, B.J. (1991). Assymetries of oxygen uptake transients at the on and offset of heavy exercise in humans. Journal of Applies of Physiology, 443, 575-586.

Peterson, E.S., Whipp, B.J., Davis, J.A., Huntsman, D.J., Brown, H.V., Wasserman, K. (1983). Effects of β-adrenergic blockade on ventilation and gas exchange during exercise in humans. Journal of Applied Physiology. 54, 1306-1313.

Piiper, J., diPrampero, P.E., Cerretelli, P. (1968). Oxygen debt and high energy phosphates in gastrocnemius muscle of the dog. American Journal of Physiology. 215 (3), 523-531.

Piiper, J., Spiller, P. (1970) Repayment of O_2 debt and resynthesis of high energy phosphates in gastrocnemius muscle of the dog. Journal of Applied Physiology. 28 (5), 657-662.

Powers, S.K., Dodd, S., Beadle, R.E. (1985). Oxygen uptake kinetics in trained athletes differing in VO_{2max}. European Journal of Applied Physiology, 54, 306-308.

Rhodes, E.C., Roberts, G.A. (1994). A comparison of the metabolic function between isokinetic and free sprint performance. Medicine and Science in Sports and Exercise, 26.

Roberts, A.D., Morton, A.R. (1978). Total and alactic oxygen debts after supramaximal work. European Journal of Applied Physiology, 38, 281-289.

Robinson, S., Harmon, P.M. (1941). The effects of training and of geletin upon certain factors which limit muscular work. American Journal of Physiology, 133, 161-169.

Roth, D.A., Stanley, W.C., Brooks, G.A. (1988). Induced lactacidemia does not affect postexercise O_2 consumption. Journal of Applied Physiology, 65, 1045-1049.

Sahlin, K., Alvestrand, A., Brandt, R., Hultman, E, (1978). Intracellular pH and bicarbonate concentration in human muscle during recovery from exercise. Journal of Applied Physiology, 45, 474-480.

Sedlock, D.A. (1991). Effect of exercise intensity on postexercise energy expenditure in women. British Journal of Sports Medicine, 25 (1), 38-40.

Sedlock, D.A. (1992). Post-exercise energy expenditure after cycle ergometer and treadmill e exercise. Journal of Applied Sport Science Research, 6, 19-23.

Sedlock, D.A. (1994). Fitness level and postexercise energy expenditure. Journal of Sports Medicine and Physical Fitness, 34, 336-342.

Sedlock, D.A., Fissinger, J.A., Melby, C.L. (1989). Effect of exercise intensity and duration of post-exercise energy expenditure. Medicine and Science in Sports and Exercise, 21, 662-666.

Shephard, R.J. Biochemistry of Physical Activity. Charles C. Thomas Publisher, Illinois, 1984.

Smith, J., McNaughton, L. (1993). The effects of intensity of exercise on excess post-exercise oxygen consumption and energy expenditure in moderately trained men and women. European Journal of Applied Physiology, 67, 420-425.

Springer, C.S., Barstow, T.J., Wasserman, K., Cooper, D.M. (1991). Oxygen uptake and heart rate responses during hypoxic exercise in children and adults. Medicine and Science in Sports and Exercise, 23, 71-79.

Stainsby, W.M. (1986). Biochemical and physiological bases for lactate production. Medicine and Science in Sports and Exercise, 18 (3), 341-343.

Stainsby, W.M., Barclay, J.K. (1970). Exercise metabolism: O_2 deficit, steady level O_2 uptake and O_2 uptake for recovery. Medicine and Science in Sports, 2, 177-186.

Stainsby, W.N., Brooks, G.A. (1990). Control of lactic acid metabolism in contracting muscles during exercise. Exercise and Sport Sciences Reviews, 18, 29-63.

Stanley, W.C. (1991). Myocardial lactate metabolism during exercise. Medicine and Science in Sports and Exercise, 23, (8), 920-924.

Stanley, W.C., Gertz, E.W., Wisneski, J.A., Neese, R.A., Brooks, G.A. (1984). Glucose and lactate turnover in man during rest and exercise studied with simultaneous infusion of ^{14}C-glucose and ^{13}C-lactate. Medicine and Science in Sports and Exercise, 16, 136.

Sutton, J.R. (1992). Limitations to Maximal Oxygen Uptake. Sports Medicine, 13 (2), 127-133.

Taylor, H.L., Buskirk, E., Henschel, A. (1953). Maximal oxygen intake as an objective measure of cardiorespiratory performance. Journal of Applied Physiology, 8, 73-80.

Tesch, P.A., Wright, J.E. (1983). Recovery from short-term intense exercise its relation to capillary supply and blood lactate concentration. European Journal of Applied Physiology, 52, 98-103.

Thomas, H.D. Gaos, C., Vaughn, C.W. (1965). Respiratory oxygen debt and excess lactate in man. Journal of Applied Physiology. 20 (5), 898-904.

Viru, A. (1987). Mobilisation of structural proteinns during exercise. Sports Medicine, 4, 95-128.

Volkov, N.I., Shirdovets, E.A., Borilkevich, V.E. (1975) Assessment of aerobic and anaerobic capacity of athletes in treadmill running. European Journal of Applied Physiology, 34 (2), 121-130.

Vrbova, G. (1979). Influence of activity on some characteristic properties of slow and fast mamalian muscles. In: Exercise and Sports Sciences Reviews, Volume 7, edited by R.S. Hutton and D.I. Miller. The Franklin Institute Press, 181-213.

Wasserman, K., Burton, G.G., van Kessel, A.L. (1964). The physiological significance of the "Anaerobic Threshold." The Physiologist, 7 (3), 279.

Wasserman, K., Burton, G.G., van Kessel, A.L. (1965). Excess lactate concept and oxygen debt of exercise. Journal of Applied Physiology, 20 (6), 1299-1306.

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

Wenger, H.A., Reed, A.T. (1976). Metabolic factors associated with muscular fatigue during aerobic and anaerobic work. Canadian Journal of Applied Sport Sciences, 1, 43-47.

Weltman, A., Stamford, B.A., Moffat, R.J., Katch, V.L. (1982). Exercise recovery, lactate removal, and subsequent high intensity exercise performance. Research Quarterly, 48, 786-796.

Whipp, B.J., Mahler, M. (1980). Dynamics of gas exchange during exercise. In: Pulmonary Gas Exchange, Volume II, J.B. West. (Ed.). New York: Academic Press, 33-96.

Whipp, B.J., Seard, C., Wasserman, K. (1970). Oxygen deficit-oxygen debt relationships and efficiency of anaerobic work. Journal of Applied Physiology, 28 (4), 452-456.

Whipp, B.J., Wasserman, K. (1972). Oxygen uptake kinetics for various intensities of constant-load work. Journal of Applied Physiology, (3), 351-356.

Wolfe, R.R., Klein, S., Carraro, F., Weber, J.M. (1990). Role of triglyceride-fatty acid cycle in controlling fat metabolism in humans during and after exercise. American Journal of Applied Physiology, 258, E382-E389.

Zhang, Y.Y., Johnson, M.C., Chow, N., Wasserman, K. (1991). The role of fitness on VO_2 and VCO_2 kinetics in response to proportional step increases in work rate. European Journal of Applied Physiology, 63, 94-100.

APPENDIX

Table 6. Individual and Mean VO_{2max} Scores:

Subject	VO_{2max} ($ml \cdot kg^{-1} \cdot min^{-1}$)		VO_{2max} ($L \cdot min^{-1}$)	
	pre-training	post-training	pre-training	post-training
1	45.32	52.63	4.02	4.39
2	47.67	58.96	3.01	3.70
3	46.41	48.07	4.03	4.15
4	48.08	51.04	3.54	3.68
5	49.06	51.70	3.15	3.29
6	36.49	39.59	3.68	3.94
7	45.53	51.72	3.08	3.62
8	47.11	55.80	3.67	4.45
9	49.03	54.40	4.20	4.72
10	49.06	54.26	3.74	4.10
means	46.38	51.82	3.61	4.00
SD	3.74	5.21	.42	.44

Table 7. Individual and Mean Rate Constants (τ) Values (minutes):

Subject	τ_1 - fast component (minutes)		τ_2 - slow component (minutes)	
	pre-training	post-training	pre-training	post-training
1	3.02	2.64	43.27	40.75
2	2.52	1.92	42.22	28.95
3	2.52	2.50	43.84	42.42
4	2.98	2.33	55.43	42.26
5	2.67	2.34	38.51	38.17
6	2.61	2.59	43.88	41.37
7	2.79	2.17	47.85	41.22
8	2.63	2.46	42.76	42.72
9	2.51	2.44	43.06	41.89
10	2.61	1.56	36.59	36.56
means	2.69	2.29	43.74	39.63
SD	.19	.33	5.12	4.24

Table 8. Individual and Mean Rate Half-Times ($t_{1/2}$)(minutes):

Subject	$t_{1/2}$ - fast component (minutes)		$t_{1/2}$ - slow component (minutes)	
	pre-training	post-training	pre-training	post-training
1	2.10	1.83	29.99	28.25
2	1.75	1.33	29.26	20.07
3	1.75	1.73	30.39	29.41
4	2.07	1.61	38.42	29.29
5	1.85	1.62	26.69	26.46
6	1.81	1.79	30.42	28.68
7	1.94	1.50	33.17	28.57
8	1.82	1.71	29.64	29.61
9	1.74	1.69	29.84	29.04
10	1.81	1.08	25.36	25.34
means	1.86	1.59	30.32	27.47
SD	.13	.23	3.55	2.86

Table 9. Individual and Mean EPOC Values:

Subject	EPOC (Litres)		EPOC (ml.kg.min-1)	
	pre-training	post-training	pre-training	post-training
1	9.65	7.85	107.93	99.5
2	7.98	5.87	127.12	94.18
3	10.41	9.37	120.61	108.51
4	8.10	7.01	101.47	90.88
5	5.69	5.47	88.42	76.44
6	10.11	7.00	95.88	71.00
7	9.91	8.43	148.69	122.84
8	9.81	7.59	128.09	87.30
9	11.59	10.57	122.57	115.81
10	8.05	6.77	128.09	87.30
means	9.13	7.49	116.89	95.38
SD	1.66	1.73	18.21	16.55

Table 10. Individual and Mean Fast Component EPOC Volumes (EPOCfast):

Subject	Fast Component (Litres)		Fast Component (ml.kg-1.min-1)	
	pre-training	post-training	pre-training	post-training
1	5.19	4.21	58.50	51.13
2	3.95	3.03	62.43	48.25
3	5.02	4.83	58.50	56.00
4	3.90	3.09	51.46	41.79
5	3.28	3.20	50.98	50.15
6	6.12	4.14	60.29	44.45
7	3.56	2.92	52.87	42.40
8	4.87	3.96	62.94	49.60
9	5.23	5.08	57.45	57.73
10	3.48	3.34	62.94	49.60
means	4.46	3.81	57.84	49.11
SD	0.95	0.79	4.63	5.25

Table 11. Individual and Mean Slow Component EPOC Volumes (EPOCslow):

Subject	Slow Component (Litres)		Slow Component (ml.kg-1.min-1)	
	pre-training	post-training	pre-training	post-training
1	4.46	3.91	49.43	51.41
2	4.03	2.84	64.69	45.93
3	5.39	4.54	62.11	52.51
4	4.20	3.92	50.01	49.09
5	2.41	1.25	37.44	26.29
6	3.99	2.61	35.59	26.55
7	6.35	5.51	95.82	80.44
8	4.94	3.63	65.15	37.70
9	6.36	5.49	65.12	58.08
10	4.57	3.43	65.15	37.70
means	4.67	3.71	59.05	46.57
SD	1.18	1.30	17.31	16.04

Table 12. Individual and Mean Blood Lactate Values ($mmol \cdot L^{-1}$):

Subject	Blood Lactate ($mmol \cdot L^{-1}$)	
	pre-training	**post-training**
1	14.96	12.62
2	18.16	13.62
3	14.62	12.36
4	15.12	13.56
5	17.22	16.50
6	11.70	11.10
7	15.20	13.50
8	16.30	14.50
9	15.80	14.20
10	13.70	11.60
means	15.28	13.36
SD	1.80	1.55