THE EFFECT OF THE MANIPULATION OF BLOOD LACTATE ON THE INTEGRATED EMG OF THE VASTUS LATERALIS MUSCLE DURING INCREMENTAL EXERCISE

By

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ABSTRACT

This study was designed to test the hypothesis that the electromyographic signal recorded from a working muscle reflects changes in blood lactate concentrations.

A group of trained cyclists performed two incremental exercise tests on a cycle ergometer. The Control Trial was a incremental test with power increments of 23.5 watts per minute. Cadence was monitored and maintained at 90+/-1 revolutions per minute. The Experimental Trial consisted of a high intensity arm exercise protocol designed to elevate blood lactate above 8 mmol/l. The arm protocol was followed by five minutes of rest and the incremental exercise protocol used in the Control Trial.

Expired gases were sampled every fifteen seconds and calculated values for oxygen uptake, ventilation, excess CO_2, and R.Q. were averaged to give a mean value for each minute in both trials. Heart rate was monitored and recorded every minute for both trials.

Electromyographic data were sampled from the vastus lateralis of the right leg for the final eight seconds of each workload in both trials. The data were integrated for each pedal cycle and averaged to give a mean integrated value for each cycle (CIEMG) for each workload.

During both trials blood samples were drawn from the cephalic vein of the left arm during the last ten seconds of each workload. The anaerobic threshold (Tlac) was determined

using the log-log transformation as outlined by Beaver et al., (1985).

Control Trial lactate concentration showed a marked inflection point after an initial slow increase. The mean maximal lactate concentration was 18.21 +/- 5.54 in the Control Trials. This inflection point occurred at a mean lactate concentration of 5.58 +/- 1.05 mmol/l. The mean oxygen uptake at the inflection point was 2.28 +/- 0.37 l/min which represented a mean of 72.6 +/- 7.20 % of maximum. Experimental Trial mean plasma lactate at the beginning of incremental exercise was 26.61 +/- 8.86 mmol/l. The plasma lactate concentration decreased steadily for the initial loads to a mean low concentration of 10.78 +/- 5.78 mmol/l at Tlac and then increased to a mean of 19.08 +/- 6.66 mmol/l at test completion. Plasma lactate concentration was greater in the Experimental Trial at all workloads though the values tended to converge once Tlac was surpassed.

No visually identifiable inflection point in the plot of CIEMG vs Power could be determined in any of the plots. An analysis of the slope of the CIEMG vs. Power relationship was therefore performed. An analysis of variance demonstrated no significant difference in the slope of the relationship within or between trials in three different comparisons. The slope of the line was not statistically different when compared over: (a) the entire sample (b) pre Tlac and (c) post Tlac. Correlations performed between plasma lactate

concentrations and CIEMG were significant in five of six subjects during the Control Trial (r = 0.57 to 0.97). During the Experimental Trial only three of the six subjects showed significant correlations and they were in the opposite direction (r = -0.62 to -0.96). Correlations between power output and CIEMG were for all subjects in both trials (r = 0.92 to 0.99 Control, r = 0.91 to 0.99 Experimental).

The increase seen in CIEMG with increased power output reflects poorly the changes in blood lactate concentrations under the conditions of this investigation. Plasma lactate showed a dramatic increase in the Control Trial and a steady decrease from an initial high concentration followed by a marked increase in the final workloads of the Experimental Trial. In contrast the CIEMG increased in a near linear fashion for all subjects in both trials. The changes in CIEMG showed highly significant correlations with changes in $\dot{V}O_2$ or power output in both trials for all subjects. These results indicate that changes in the surface electromyogram are highly related to changes in power output. However the surface electromyogram changes are not driven by changes in lactate concentration under the conditions of this investigation and may not be a sensitive enough indicator of these changes to be employed in the determination of Tlac.

The Dynamics of Lactate and pH During Incremental Exercise..... 20

Mechanisms of Lactate Accumulation..... 21

Blood Lactate as an Estimator of Muscle Lactate..... 23

Dynamics of Lactate During Recovery from Exercise..... 24

pH Changes with Lactate Accumulation..... 26

Effects of Decreased pH on the Contractile Apparatus..... 27

Neuromuscular Response to Incremental Exercise..... 30

The Integrated EMG as an Indicator of Muscle Tension..... 30

Relationship Between Isometric Tension and EMG..... 31

Relationship Between Dynamic Contractions and EMG..... 34

The Integrated EMG as a Non-Invasive Indicator of the Lactate Threshold..... 36

Proposed Rationale for the use of IEMG as an Indicator of the Anaerobic Threshold..... 38

Possible Effects of Cadence on the Electromyographic Signal..... 41

Timing and Duration of Electrical Activity as a Possible Confounding Factor..... 43

3. PROCEDURES..... 44

Research design..... 44

Subjects..... 44

Control Condition Protocol..... 45

Experimental Condition Protocol..... 45

Pretrial Muscle Warming..... 46

Data Collection..... 46

Procedures for Lactate Sampling..... 46

Procedures for Lactate Determinations..... 47

Procedures for EMG Recording..... 47

Data Analysis..... 48

Integration of EMG Data..... 48

Timing and Duration of Electrical Activity..... 48

Determination of Lactate Threshold..... 48

Determination of Rate of Change (Slope) of CIEMG vs. Power Output..... 49

4. RESULTS..... 51

Description of Subjects..... 51

Analysis of Cardio-Respiratory Data..... 51

Control Trial Cardiorespiratory Data..... 51

Experimental Trial Cardiorespiratory Data... 53

Analysis of Lactate Data..... 53

Control Trial Lactate Data..... 54

Experimental Trial Lactate Data..... 56

Anaerobic Threshold Determination Using Plasma Lactate..... 57

Analysis of Integrated EMG Data..... 58

Control Trial Integrated EMG Data..... 58

Experimental Trial Integrated EMG Data..... 60

Examination of the Relationship Between Lactate and CIEMG..... 61

5. DISCUSSION..... 65

Cardiorespiratory and Metabolic Data..... 65

Response of EMG to Plasma Lactate..... 66

Plasma Lactate Changes in the Control Trial..... 66

The Effect of Lactate on the CIEMG in the Control Trial..... 67

Plasma Lactate Changes in the Experimental Trial..... 71

The Effect of Lactate on the CIEMG During the Experimental Trial..... 73

Explanation of Conflicting Results..... 75

6. Summary, Findings, Conclusions and Recommendations..... 84

Summary..... 84

Findings..... 85

Conclusions..... 86

Recommendations..... 87

APPENDIX A - COMPLETE CARDIORESPIRATORY DATA..... 89

APPENDIX B - PLASMA LACTATE DATA..... 94

APPENDIX C - ELECTROMYOGRAPHIC DATA..... 95

APPENDIX D - INDIVIDUAL SUBJECT PLOTS OF CIEMG AND PLASMA LACTATE FOR BOTH TRIALS..... 96

APPENDIX E - SELECTED DATA SUMMARY TABLES..... 102

REFERENCES..... 104

LIST OF TABLES

1. Analysis of Variance of Selected Cardiorespiratory Measures..... 54

2. Slope of IEMG vs $\dot{V}O2$ Plot for the Total Exercise Period and Before and After the Lactate Threshold.. 60

3. Analysis of Variance of Slopes of IEMG vs $\dot{V}O_2$ Plots..... 61

4. Correlations Coefficients for CIEMG and Power Output and CIEMG and Plasma Lactate..... 64

LIST OF FIGURES

1. Plots of Mean Cardiorespiratory Data - Control and Experimental Conditions..... 52

2. Mean Plasma Lactate vs Oxygen Uptake - Control and Experimental Condition..... 55

3. Plot of CIEMG vs. Power Output - Control and Experimental Trial..... 59

4. Group Data - Mean CIEMG and Plasma Lactate vs. Power Output - Control and Experimental Trials..... 63

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Chapter 1

INTRODUCTION

During incremental exercise on a cycle ergometer changes occur in the human metabolic environment which eventually contribute to a decreased ability to perform muscular work. At a power output representing approximately 60% of maximal oxygen uptake the human organism cannot supply energy through aerobic glycolysis at an adequate rate to sustain performance (Davis, 1985). At this point the body begins to rely more heavily on anaerobic glycolysis. This change in the predominant energy supply system is indicated by changes in the metabolic environment. The point at which the transition takes place is commonly termed the anaerobic threshold.

The metabolic characteristics of the blood and muscle during incremental exercise reflect increasing muscular power output. As the power output of a muscle increases the rate at which energy must be supplied increases. The rate of energy production determines the predominant energy system and therefore the end product of glycolysis. At submaximal power outputs the aerobic energy system favors the production of pyruvate which is completely metabolized to carbon dioxide and water via the TCA cycle and the electron transport system. However as power output increases toward maximal levels the production of lactate is favored over pyruvate oxidation (Wasserman, 1986). The amount of lactate produced in the muscle is related to the intensity, mode and duration of work,

the training status of the individual, as well as the glycogen content and type of muscle fibre involved (Farrel et al., 1979, Graham, 1978). The unbalancing of muscular lactate production and utilization results in a inflection point in a plot of blood lactate vs. oxygen uptake. This inflection point in blood lactate is used as an invasive estimation of the anaerobic threshold or lactate threshold (Tlac).

During the performance of muscular work the human body adjusts the functional coupling of cardiovascular and respiratory activity to supply the cells with an adequate supply of oxygen. The changes in the demand for oxygen as the body adjusts to the increased metabolic need reflect accurately the intensity of the work being performed. This is evidenced by the linear relationship between oxygen uptake and power output. As the power output increases the ability of the circulation to supply all cells with oxygen may be inadequate. This brings about a change in the redox state of the working muscle such that the production of lactate is favored over pyruvate entry into the mitochondrion (Wasserman, 1986, and Wasserman et al., 1986) increasing the concentration of muscle lactate. Prior to the production of lactate due to an altered redox state the increased energy need is met by a rapid acceleration of glycolysis whose end product, pyruvate, is produced at a rate greater than the rate at which it can be oxidized completely (Wasserman et al., 1985). This results in an increased production of lactate. These changes indicate the

body's increased reliance on muscle glycogen and anaerobic glycolysis. This increased reliance results in an unbalancing of muscular lactate utilization and production.

At physiological pH lactate is 99% dissociated and must be buffered. Some portion of this buffering is performed by the bicarbonate system (Wasserman et al. 1967). This results in increased hydrogen ion concentration in the muscle which decreases muscle pH. The increase in intracellular lactate concentrations is linearly related to the decrease in intracellular muscle pH (Sahlin, 1978) and has been highly correlated with changes in ventilatory parameters due to the increased CO_2 production. The following set of equations summarizes the process.

$$HLA \xleftarrow{\text{Lactate Dehydrogenase}} H^+ + LA^- \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad 1$$

$$H^+ + HCO_3^- \xleftarrow{\text{Carbonic Anhydrase}} H_2CO_3 \quad \ldots\ldots\ldots\ldots\ldots \quad 2$$

$$H_2CO_3 \longleftrightarrow H_2O + CO_2 \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad 3$$

Because of the relationship between ventilation, changes in lactate concentration, and the subsequent change in pH numerous studies have demonstrated the usefulness of these ventilatory changes in estimating the anaerobic threshold. The anaerobic threshold (Tvent) is estimated to occur at the percentage of oxygen uptake where a non-linear increase in $\dot{V}_E$ and excess CO_2 occurs accompanied by an increased $\dot{V}_E/\dot{V}O_2$ ratio without a concomitant increase in $\dot{V}_E/\dot{V}CO_2$ ratio (Davis, 1985). While some studies have have demonstrated a high correlation between Tlac and Tvent during incremental exercise tests

others have introduced conditions where a dissociation of the two inflection points has been evident (Farrel and Ivy, 1987). Despite the uncertainty of this relationship these two methods, one invasive and one non-invasive, have proven useful as estimators of the transition point in energy production from predominantly oxidative phosphorylation to anaerobic glycolysis resulting in lactate production.

Several studies (Moritani, 1980, Nagata et al., 1981, Miyashita et al., 1981, Moritani et al., 1984, Viitasalo et al., 1985) have demonstrated a relationship between muscle activation as determined by surface electromyogram and the anaerobic threshold. These studies have shown that with incremental increases in power output there is a point where a sudden increase in Integrated EMG (IEMG) occurs. The sudden increase seems to exceed that expected due to increased power output. This inflection point occurs at a percentage of oxygen uptake which correlates highly with the percentage of maximal oxygen uptake at which the inflection point in ventilatory parameters or blood lactate concentration occurs (Moritani, 1980, Nagata, 1981). These studies have concluded that since IEMG seems to respond to changes in lactate the Integrated EMG could serve as a non-invasive indicator of Tlac.

The electromyogram reflects the firing rate, amplitude, recruitment pattern and fibre type of motor units of the muscle being recorded (Basmajian, J.V., 1978, Komi et al.,

1970, Moritani, 1986). The theoretical basis of increased electrical activity with increased force development stems from studies by Milner-Brown et al. (1972) and Milner-Brown et al., (1976). These studies demonstrated that increased force was achieved by initially increasing the number of fibres recruited and later increasing the firing rate of the fibres involved. The orderly recruitment of fibres from small to large and the greater amplitude and maximum firing frequency of the larger fibres produce increasing recorded electrical activity. Any changes in the metabolic environment which affect the recruitment pattern or firing frequency within a certain muscle or alter the activation pattern of a muscle group will be reflected in changes in the recorded surface electromyogram and the integrated EMG.

There have been numerous studies which characterized the relationship between the electromyogram and muscle tension or force in both dynamic and isometric contractions of unfatigued muscle. Some studies have indicated a linear relationship between isometric contraction force and IEMG (Lippold, 1952, Inman et al., 1952, Close et al., 1960, Dejong et al., 1967, Moritani et al., 1986) while others have demonstrated non-linear relationships (Zuniga et al., 1969, Komi et al., 1970, Kuroda et al., 1970, Heckathorne et al., 1981).

During dynamic cycling exercise a linear relationship was demonstrated between EMG activity (Root Mean Square) of the lateral quadriceps and load (Bigland-Ritchie and Woods, 1974).

This relationship was later extended by Petrofsky et al., (1979) who "showed that in the absence of fatigue" the linear relationship existed for loads up to an independently determined maximum.

The exact nature of the relationship is difficult to characterize. Measures of force or muscle tension represent summations of activity within a muscle or group of muscles that may not be reflected by the surface electromyogram which provides a statistical sampling of the electrical activity. The majority of evidence suggests that a linear relationship exists between the recorded electromyogram signal and measured force. However evidence indicating some other type of relationship suggests that caution must be used in attributing changes in the electromyographic signal to variables other than increased force or muscle tension.

The presence of fatigue in a muscle alters the relationship between the IEMG and force. The relationship has been shown to change with sustained submaximal isometric contractions, in a muscle that was previously fatigued and during sustained maximal contractions (Komi, 1984). These changes have been related in some instances to changes in the metabolic environment.

The accumulation of lactate in the muscle decreases the pH in the muscle and subsequently in the blood (Hermansen et al., 1977, Sahlin, 1978). The decrease in muscular pH has been suggested to be the cause of fatigue for high intensity

exercise. Evidence has shown that the decrease in muscle pH affects the excitation contraction coupling of muscle contraction. Several steps in the contractile process are affected by a decrement in muscular pH. They include: (a) increased Ca^{++} requirement (Donaldson, 1978); (b) decrease in myosin ATPase activity (Schadler cited in Sahlin 1978); (c) an increase in the binding constant of the sarcoplasmic reticulum for Ca^{++} (Nakamura and Schwartz, 1972). The decreased ability of the muscle to produce force correlates highly with increasing levels of lactate and has been hypothesized to be the result of a combination of these mechanisms.

In dynamic incremental cycling exercise to determine maximal oxygen uptake, an inflection point in IEMG, apparently unrelated to changes in workload, has been shown to occur. (Moritani, 1980, Nagata et al., 1981, Miyashita et al., 1981, Moritani et al., 1984, Viitasalo et al., 1985). This inflection point, caused by changes in the electrical characteristics of the muscle, is hypothesized to be the result of the decreased pH caused by increased lactate concentration (Tesch et al. (1983) in Komi 1984).

This explanation is logical because both increased lactate concentrations and increased electrical output can be shown to be related to increased percentages and recruitment of fast glycolytic fibres (Tesch et al., 1978, Tesch et al., 1983). The increase in IEMG with fatigue results from the failure of some working fibres resulting in increased

recruitment of fast twitch glycolytic fibres as well as increased firing rate of some already recruited fibres (Häkkinen et al. 1986). This change in muscle fibre recruitment and firing frequency would result in altered electrical characteristics in the muscle as a result of the greater amplitude and firing frequency of the fast twitch fibres. This, in combination with the increasing work load could result in an accelerated, non-linear increase, or inflection point in the IEMG of the working muscle.

However several studies (Edwards et al., 1972, Karlsson et al., 1975, Broman, 1977, Petrofsky, 1980, Komi 1984, Boubrit, 1983) have pointed out that the time course of recovery from exercise for lactate and pH requires one to several hours depending upon conditions of recovery while the recovery of maximum twitch tension and amplitude as well as mean power frequency of the EMG signal have shown an almost immediate recovery to pre-exercise or resting levels. Mills and Edwards (1984) examined changes in the electromyogram of myophosphorylase deficient patients who are incapable of producing lactate. They demonstrated changes similar in direction to normal controls but greater in magnitude. This indicated that under the conditions cited above the electrical characteristics of a muscle are independent of the metabolic environment and using EMG as an indicator of underlying physiological changes may not be valid.

The initial study using incremental cycle ergometer

exercise utilized constant power output ergometers and allowed cadence to range from 50 to 80 revolutions per minute (Moritani, 1984). Because power output is the product of the applied force and cadence, a constant power output is possible with any combination of the two variables. It is quite likely that these different combinations cause the muscle to respond differently depending on whether the increase in power output is achieved through increasing force or by increasing cadence. There is evidence to suggest that changes in cadence at constant power output alters metabolic response (Coast and Welch 1985). A change in the metabolic response of the working muscle could indicate an altered activation pattern. Such alterations could have an effect on the electromyographic signal recorded from the working muscle. Further it is possible that some predictable changes in cadence occur during incremental work which could result in the observed changes in the IEMG. It is important that cadence be monitored and addressed as a possible contributor to observed changes in the IEMG with incremental exercise.

Statement of the Problem

Previous studies have demonstrated a relationship between both Tlac and an Integrated EMG inflection point. The conclusion that IEMG could be used as an indicator of the observed dramatic increase in blood lactate concentrations during incremental exercise suggests a high degree of sensitivity. If this method of lactate threshold

determination is to be of practical use the implied sensitivity is a necessity. Such a high degree of sensitivity has not been demonstrated. If the proposed relationship does exist between blood lactate and IEMG it should respond predictably to changes in blood lactate accumulation under varied conditions.

Hypotheses

1. It was hypothesized that a strong positive correlation would be seen between the percentage of maximal oxygen uptake at which an inflection point in blood lactate occurred and the percentage of maximal oxygen uptake at which an inflection point in CIEMG occured in the Control Condition.

2. In the Experimental Condition it was hypothesized that the inflection point would be shifted to the left in the lactate vs. power output plot. Further the CIEMG vs. power output plot would show a similar shift in the inflection point.

Significance of the Study

The significance of this study is that it will provide data which clarify the relationship between changing amounts of lactate and the electrical characteristics of muscle. The integration of data concerning the metabolic state of the muscle and its effect on the electrical functioning or vice versa is important because of the multifactorial nature of

muscle fatigue.

It should provide a starting point for further studies which could focus on monitoring, manipulation and description of some of the other factors which are known to effect both muscle function and IEMG.

In addition, if a valid relationship is supported between lactate and IEMG during incremental exercise some initial practical applications may be made. These applications would require further study into the application of IEMG in the monitoring of training intensity and its effectiveness as compared to other more conventional methods.

Definition of Terms

Incremental Exercise Test. A test performed on a cycle ergometer with four minutes of unloaded cycling and increasing in steps of 23.5 watts per minute until the subject could no longer maintain the prescribed cadence of 90 revolutions per minute.

Anaerobic Threshold. The point in incremental exercise to maximum where energy is increasingly supplied by anaerobic glycolysis. This term will be used interchangeably with Tlac.

Tlac. The intersection point in a log-log plot of Lactate concentration vs. Oxygen uptake of two regression lines. One from test commencement to a visually determined dividing point and one from the dividing point to maximum (Beaver et al., 1985). Tlac will be determined for each subject from their Control Trial data.

Tvent. The percentage of oxygen uptake where a non-linear increase in $\dot{V}_E$ and excess CO_2 occurs accompanied by an increased $\dot{V}_E/\dot{V}O_2$ ratio without a concomitant increase in $\dot{V}_E/CO_2$ ratio (Davis, 1985).

Integrated Electromyogram (IEMG). The integral of the full wave rectified EMG signal.

Cycle Integrated Electromyogram (CIEMG). The integrated value for each burst of activity (from TDC to TDC) at each work load is calculated. Each cycle is averaged to give a mean integrated per cycle value (CIEMG) for each workload.

Top Dead Centre (TDC). The position of the pedal at the top of the cycle, when the crank is perpendicular to the floor. A TTL pulse of +/- 5 volts indicates the position of the crank.

Delimitations

Measurements of pCO_2, pO_2, pH, and HCO^-_3 will not be performed on arterial blood samples which then could be combined with arterial lactates for a more sensitive and accurate determination of lactate threshold (Nagata et al. 1981). The determination of the lactate threshold from venous lactate measurements in the arm is a valid reflection of the changes in overall blood lactate (Simon et al., 1986). Venous blood lactate concentrations will be taken to reflect the direction of change in muscle lactate concentrations only.

Chapter 2

REVIEW OF THE LITERATURE

This review is divided into four major subsections: (a) physiological responses to incremental exercise; (b) the existence and significance of the anaerobic threshold; (c) the dynamics and interactions of lactate and pH during incremental exercise; (d) the neuromuscular response to exercise.

Physiological Responses to Incremental Exercise

At the onset of exercise the human body begins a series of adaptations which are aimed at maintaining cellular homeostasis. During incremental exercise from low intensity to maximal effort a typical pattern of responses is evident in a number of different measures. Skinner and McClellan (1979) divided these responses into three distinct phases moving from almost purely aerobic metabolism through a transition phase to almost purely anaerobic metabolism.

During the first phase the demand for oxygen by the working tissues increases resulting in greater extraction of oxygen from the blood causing a slight decrease in the fraction of expired oxygen (F_EO_2%). Concomitantly the amount of carbon dioxide produced increases causing a slight increase in the fraction of expired CO_2 (F_ECO_2%). Linear increases occur in Ventilation ($\dot{V}_E$), Heart Rate, and Oxygen uptake ($\dot{V}O_2$). That this stage is aerobic is indicated by only small

increases in the respiratory quotient (R or $\dot{V}CO_2/\dot{V}O_2$) which remains between 0.7 and 0.8. As well blood lactate [La] which is an indicator of the degree of anaerobiosis, rises only slightly above rest values.

The second stage or the transition phase is characterized by continuing linear increases in Heart Rate and $\dot{V}O_2$ and an initial rise in blood lactate, usually to levels approximately twice that of resting values. This increase in lactate causes an increased production of H^+ ions thereby decreasing pH (Hermansen and Osnes, 1972, Sahlin, 1978, Metzger and Fitts, 1987). These ions are buffered via several mechanisms. These mechanisms include metabolic buffering processes such as the utilization of creatine phosphate, the production of ionosine monophosphate resulting from the combination of hydrogen ions with ATP and the oxidation of amino acids. These metabolic processes account for approximately 40% of the available buffering capacity. The remaining buffering capacity is via physico-chemical processes which account for approximately 60% of the total buffering capacity (Parkhouse and McKenzie, 1984). These include the absorption of hydrogen ions by plasma proteins and inorganic phosphate. Sahlin (1978) reported that approximately 15 - 18 % of the hydrogen ions produced are buffered via the bicarbonate system according to the following simplified equations.

$$HLa + NaHCO_3 \longleftrightarrow NaLa + H_2CO_3 \quad \ldots\ldots\ldots\ldots\ldots\ldots \quad 4$$

$$H_2CO_3 \longleftrightarrow H_2O + CO_2 \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad 5$$

The increased production of CO_2 as a result of the dissociation of the carbonic acid in equation five results in a steady increase in the F_ECO_2. The respiratory centers respond to the decreased pH and the increased production of CO_2 by increasing $\dot{V}_E$ resulting in a marked increase in $\dot{V}CO_2$. A distinct increase in the $\dot{V}_E/\dot{V}O_2$ ratio is seen due to an increase in $\dot{V}_E$ and $\dot{V}CO_2$ greater than the increase in $\dot{V}O_2$. Because of the disproportionate increase in $\dot{V}_E$ in comparison with $\dot{V}O_2$ there is a decreased extraction of oxygen from the inspired air resulting in an increased F_EO_2%. This combination of responses, non-linear increases $\dot{V}_E$ and $\dot{V}CO_2$ with a rise in F_EO_2 without concomitant increases in $\dot{V}CO_2$, along with increased lactate concentration was proposed to represent the anaerobic threshold (Wasserman et al., 1973). These changes occur at power outputs which correspond to oxygen uptakes of approximately 40-60% of maximum.

The final phase outlined by Skinner and McClellan (1978) occurs as the power output approaches approximately 65-90%. The linear increases in Heart Rate and $\dot{V}O_2$ continue until near maximal levels where a leveling off is typically seen. The increase in $\dot{V}_E$ continues but is not adequate to compensate for the increased levels of CO_2 thus a decrease in $\dot{V}CO_2$ occurs and the F_EO_2 continues to rise. A rapid increase in La is evident and continues until maximal power output is reached. These changes, decreased F_ECO_2, a sharp increase in La and hyperventilation have been identified by MacDougall, (1978)

and Green et al. (1979) as representing the anaerobic threshold. These adaptations to incremental exercise result from the increased power output and reflect changes in the relative contribution of aerobic and anaerobic energy systems to the energy supply of the working muscle.

The Existence and Significance of the Anaerobic Threshold

Precise physiological mechanisms responsible for a threshold have not yet been determined. Further while the anaerobic threshold concept is of practical importance using it to interpret underlying physiological interactions is difficult. Despite controversy concerning the interpretation of the relevant data and the subsequent terminology associated with this evidence (Davis, 1985, Brooks, 1985), there is a general agreement on a functional definition. The anaerobic threshold can be defined as the point during incremental exercise above which the energy utilized is increasingly derived from anaerobic glycolysis (Jones and Ehrsam, 1982, Helal et al., 1987). Anaerobic glycolysis depends primarily on glycogen reserves as fuel and generates metabolites which appear to indirectly contribute to the inhibition of energy production and thus is self-limited in its ability to supply energy over long periods of time (Coyle and Coggan, 1984). The existence of the anaerobic threshold is empirically supported by research which correlates anaerobic threshold determinations with predictions of a maximal maintainable rate of work.

Prediction of Performance Using Anaerobic Threshold Determinations. Rhodes and Mckenzie, (1984) determined the anaerobic threshold using ventilatory excess CO_2. Performance times in a marathon were then predicted using the running velocity at the determined threshold. They demonstrated a high correlation (r = 0.94) between predicted and actual performance times suggesting that running velocity at the anaerobic threshold represents optimal running pace or a pace which theoretically would be difficult to exceed and maintain because doing so would cause greater reliance on anaerobic glycolysis. Tanaka and Matsura (1984) performed a similar study to examine the differences in marathon performance predictive ability between the quantitatively defined (ie 4mmol) onset of blood lactate accumulation (OBLA) and the anaerobic threshold defined as the point of systematic increase of lactate above a resting base-line value. The running velocity at the anaerobic threshold as determined by the latter method, showed a higher correlation with the predicted marathon race velocity than did OBLA. Farrel et al. (1979) investigated the relationship between the accumulation of plasma lactate and distance running performance over various distances. They demonstrated that regardless of competitive levels the treadmill velocity and $\dot{V}O_2$ which corresponded with the systematic increase of plasma lactate above a base-line correlated very highly (r = 0.91) with distance running performance. These investigations support

the existence of the anaerobic threshold as defined by Jones and Ehrsam (1982) and Helal et al. (1987). They establish the practical significance of the threshold or a transition phase but do not provide evidence which clearly demonstrates the responsible mechanisms.

Fibre Type and Substrate Depletion During Incremental Exercise. The existence of the anaerobic threshold is also empirically supported by research examining fibre type distribution and substrate depletion.

There are two primary types of muscle fibres which comprise human skeletal muscle. Gollnick et al. (1972) distinguished the fibres on the basis of their myosin ATPase activity while others have identified the different types on the basis of oxidative enzyme concentrations or glycolytic capacity (Essen et al., 1975). The types have been variously named but for the purposes of this investigation the fibres will be referred to using the nomenclature Slow Oxidative (SO) and Fast Glycolytic (FG). This nomenclature is useful in that it denotes both the contractile characteristics (Fast, Slow) and the predominant type of energy supply system (Glycolytic, Oxidative). An intermediate fibre, Fast Oxidative Glycolytic (FOG) has also been identified. The SO fibres are enzymatically and structurally designed to function primarily during low intensity aerobic work. These fibres are rich in mitochondrion and the enzymes necessary for oxidative metabolism. Muscle glycogen levels which are the primary

energy source for anaerobic glycolysis are relatively small in these fibres. FG fibres function optimally at high levels of muscular work. They have fewer mitochondrion and enzyme profiles which are best suited for energy production via anaerobic glycolysis (Gollnick and Hermansen, 1973). The FOG fibres have characteristics of both the SO and FG fibres and may alter characteristics somewhat depending upon the type of training they have undergone. During incremental exercise recruitment of the different fibres depends on the intensity of the work being performed with the SO fibres being recruited first at low intensities and FG fibres being recruited later (Milner-Brown et al., 1973). Essen, (1978) demonstrated that during submaximal exercise the SO fibres had a more pronounced depletion of glycogen than the FG fibres while during sustained high intensity exercise glycogen depletion was more marked in FG fibres supporting the increasing role of glycolytic fibres as power output increases. Tesch et al. (1978) found a positive correlation between the percentage of FG fibres and muscle lactate concentration. Ivy et al. (1980) demonstrated high correlations between the percentage of SO fibres and both maximal oxygen uptake and muscle respiratory capacity. Consequently Ivy et al. (1980) noted a high correlation between both the relative and absolute lactate thresholds and the percentage of SO fibres in the vastus lateralis muscle of humans. The higher the percentage of SO fibres the higher the percentage of $\dot{V}O_2$ at which the lactate

threshold occurred. It is suggested that this is due to the high glycolytic capacity and greater lactate production of FG fibres which are recruited in greater numbers as power output increases.

The empirical evidence for the existence of a transition phase or anaerobic threshold is substantial. These investigations also demonstrate a link between the changing profile of muscle fibre types being recruited for force production and the changing metabolic profile during incremental exercise.

The Dynamics and Interactions of Lactate and pH During Incremental Exercise

The production of lactate in the muscle has been shown to be highly related to metabolic rate (Donovan and Brooks, 1983, Brooks et al., 1984). As power output increases the recruitment of the FG fibres increases (Milner-Brown et al., 1973). These fibres have a high glycolytic capacity and their recruitment results in the production of pyruvate at a rate which exceeds the ability of the pyruvate dehydrogenase complex to convert pyruvate to acetyl CoA and enter the mitochondrion. The excess pyruvate produced in the muscle is then converted into Lactate [La] resulting in increased La concentration. It has been suggested that the increased glycolytic metabolism was due to a deficit in oxygen supply which inhibited energy production via oxidative metabolism (Hill et al., 1924). It has become clear through tracer

kinetic experiments that the production and utilization of lactate is a constant ongoing process which occurs both at rest and during exercise (Eldridge et al., 1974, Eldridge, 1975, Issekutz B. Jr. et al., 1976, Issekutz B. Jr., 1984). The results of these investigations invalidate the theory that accelerated anaerobic metabolism and the subsequent increase in muscle lactate concentration is due solely to an inadequate oxygen supply.

Mechanisms for Lactate Accumulation. Wasserman (1986) and Wasserman et al. (1986), proposed that there are two mechanisms responsible for the accumulation of lactate in working muscle. (a) Glycolysis increases so rapidly that pyruvate is produced at a rate which is too fast for it to be moved into the mitochondrion for complete oxidation. This results in the accumulation of pyruvate in the cytosol which causes the accelerated formation of lactate. The formation of lactate allows maintenance of glycolytic metabolism. (b) The mitochondrial shuttle which carries the reduced NAD into the TCA cycle to transfer electrons and protons to coenzymes for eventual combination with oxygen is no longer capable of maintaining the redox state. Wasserman et al. (1986) point out that this might occur if the oxygen available to the cytochrome oxidase reached a value low enough to change the cell redox state to favor the conversion of pyruvate to lactate and accelerated glycolysis. This model of mechanisms for lactate accumulation is supported by research (Wasserman

et al., 1985) showing that a transition in the mechanism for the increase in lactate concentration seems to occur during incremental exercise. These authors examined the lactate/pyruvate ratios and showed that at work intensities up to the Tlac the increase in lactate was paralleled by increases in its precursor pyruvate. This is a necessity for the mass action conversion of pyruvate to lactate, the basis of the mechanism proposed in one above. However beyond some critical oxygen uptake represented by Tlac the increase in lactate far exceeded the increase in pyruvate concentration. This supports the second mechanism described above since the increase in lactate would have to come from a change in redox state where the production of lactate from pyruvate would be favored. The accumulation of lactate is due to a greater increase in production of lactate via the mechanisms described above, than the increase in the ability to remove lactate. The removal of lactate during exercise is however considerable.

The results of tracer studies by Issekutz (1984) and Eldridge et al. (1974) indicate that lactate is an important substrate during exercise and that the major avenue for lactate removal is oxidation. Brooks (1986) demonstrated that approximately 50% of the lactate produced at rest was oxidized and that during exercise at 50% of maximum, oxidation accounted for nearly 90% of the lactate removal. The same investigation indicated that both the relative and absolute

oxidation rates of lactate increased with increasing power output. Despite the use of lactate as a substrate during exercise the production of lactate during exercise eventually exceeds the ability of the various metabolic pathways to remove it. This results in the accumulation of lactate both in the muscle and the blood that is seen during incremental exercise.

Blood Lactate as an Estimator of Muscle Lactate. The appearance of lactate in the blood is subsequent to its production in the muscle. The concentration of lactate in the blood is dependent upon the balance of production, utilization and removal in muscle.

Diamant et al. (1968) provided evidence which suggested a concentration gradient for lactate from muscle to blood. Karlsson (1971) and Jorfeldt et al. (1978) found significantly higher lactate concentrations in human muscle than in venous blood during work and reported that several minutes were required for blood lactate to equilibrate with muscle lactate. Similar results were reported by Hirche et al. (1971) for isolated dog gastrocnemius with a concentration gradient existing at all times between muscle lactate and blood lactate during exercise. The gradient favored movement of lactate into venous blood. These authors also pointed out that the gradient was increased markedly at power outputs above 70% of maximum. They concluded that the production of lactate in working muscle was greater than its release into the blood.

Graham (1978) reported that blood lactate concentrations depend upon blood flow, sampling time and muscle fibre type and did not always represent accurately absolute muscle lactate values.

The evidence above suggests that estimation of absolute lactate concentrations of muscle from blood lactate concentrations is not valid. Karlsson (1971) measured blood lactate values immediately following maximal exercise which were approximately half the simultaneously measured muscle lactate values. However the evidence also indicates that during incremental exercise beginning from rest the direction of change is similar. That is, during incremental exercise increases in blood lactate reflect increases in muscle lactate of the working muscle.

Dynamics of Lactate During Recovery from Exercise. The increased lactate concentration which accompanies incremental exercise disturbs the homeostasis of the muscle cell eventually reaching a point where it can no longer function. The return of lactate concentrations to pre-exercise levels has been shown to depend on both the duration, intensity and mode of exercise used to produce the increased concentration, and on the intensity of activity following the exercise (Brooks and Fahey, 1984). Brooks (1986) estimated that up to 70% of the lactate produced in exhaustive exercise in rats was removed via oxidation during recovery. Approximately 20% of the lactate was converted into muscle and liver glycogen,

5-10% were converted into protein constituents, less than two percent was traced to glucose and lactate and the remaining ten percent was not specifically located. These estimates are supported by tracer studies (Issekutz, 1984, and Eldridge et al., 1974) which also indicate that the major avenue for lactate removal was oxidation. The results from these studies indicate the importance of lactate as a substrate during exercise. The linear relationship demonstrated between lactate extraction and arterial lactate concentration in muscle during both rest and exercise (Brooks, 1986) clearly indicates that an elevated lactate concentration results in increased uptake by working muscle. Belcastro and Bonen (1975) demonstrated that elevated blood lactate levels produced by exercise above Tlac can be reduced by exercise at an intensity below Tlac. Such evidence highlights the balance between lactate production, efflux, and utilization which ultimately determines measured concentrations in the blood or muscle. Submaximal levels of exercise will lower lactate to pre-exercise concentrations more rapidly than passive recovery (Jorfeldt, 1970, Essen et al., 1975, Hermansen et al., 1973, and Issekutz et al., 1976). Lactate appears to be used preferentially as a substrate particularly in the case of prior elevated lactate concentrations.

The accumulation of lactate is in itself not detrimental to the muscle function (Sahlin, 1986) and in fact forms a useful metabolic substrate of considerable importance

necessary in the maintenance of glycolytic energy production. The fate of the lactate accumulated above that which can be metabolized by active muscle, inactive muscle, the liver, and the heart does however have important implications for the understanding of the effects that increased anaerobic metabolism may have on the contractile mechanism.

pH Changes with Lactate Accumulation. The low pK of lactic acid (3.9) results in it being almost entirely dissociated at cellular pH and necessitates that the resulting protons be buffered immediately. Sahlin (1978) demonstrated that the production of lactate results in an equivalent release of hydrogen ions causing a decrease in intramuscular pH. This investigation demonstrated a linear relationship between Total Muscle pH and lactate + pyruvate concentration after both isometric and dynamic exercise. The accumulation of protons and their measured effect on pH is a result of interactions between proton production and proton removal. Protons are produced due to the dissociation of the acids which are the end products of two pathways for energy production. Oxidation produces the weak acid CO_2 while glycolysis produces the stronger lactic acid. Due to the low pK of lactic acid it produces greater amounts of protons explaining the greater drop in pH during work which requires use of the glycolytic pathways. The removal of protons which are produced in the muscle cell depend essentially on two processes (a) movement of acid or bases across the cell

membrane, (b) the removal of the acid or supply of the base from the extracellular space via circulating blood. At some point the appearance of protons is exceeded by their removal resulting in a decrease in muscle pH. Hermansen and Osnes (1972) demonstrated a decrease in muscle pH from a resting value of 6.93 to a low of 6.40 after both intermittent and continuous exercise. Sahlin et al. (1972), and Sahlin et al. (1976) found similar values with a decrease from 7.08 at rest to 6.60 and 6.56 following dynamic and isometric exercise respectively. Metzger and Fitts (1987) investigated the changes in muscle pH with both high and low frequency stimulation to fatigue of in vitro preparations of rat diaphragm muscle. Their results showed comparable decrements in pH from resting values of 7.06 to a low value of 6.33 following fatiguing contractions.

All of the above studies measured decrements in the pH of working muscle. A relationship has been shown to exist between this decrease in the pH of the muscle and its ability to perform.

Effects of Decreased pH on the Contractile Mechanism. The decrease in muscular pH or increase in H^+ ions has been suggested to be the cause of fatigue for high intensity exercise.

Sahlin (1986) identified four steps in the contractile process which previous investigators have shown to be affected by the decrement in muscular pH. (a) An increased Ca^{++}

requirement, (b) decreased maximal tension with decreased muscle pH (Donaldson, 1978), (c) a decrease in myosin ATPase activity as the pH decreased from 6.5 to 7.5 (Schadler, 1967), and (4) an increase in the binding constant of the sarcoplasmic reticulum for Ca^{++} with decreased pH (Nakamura and Schwartz, 1972). This increased affinity of the sarcoplasmic reticulum for Ca^{++} could interfere in the excitation contraction coupling. Any of these changes could effect the ability of the affected muscle cells to contract.

The detrimental effect of decreased pH is supported by investigations which have demonstrated increased ability of muscles to perform work by increasing the buffering ability of both the muscle and the extracellular fluid (Costill et al., 1984). These authors also demonstrated a decrease in pH to be accompanied by a corresponding decrease in HCO^{-}_{3}. This would be predicted by equation 7 which in the presence of hydrogen ions and HCO^{-}_{3} would cause a shift to the right.

$$HLA \longleftrightarrow H^{+} + LA^{-} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad 6$$

$$H^{+} + HCO_{3}^{-} \longleftrightarrow H_{2}CO_{3} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad 7$$

$$H_{2}CO_{3} \longleftrightarrow H_{2}O + CO_{2} \quad \ldots\ldots\ldots\ldots\ldots\ldots\ldots \quad 8$$

Mainwood and Renaud (1985) reported that while changes in pH are clearly related to the ability of the muscle to develop tension they cannot be considered to be causal. The authors reported that the recovery of force development could be delayed over an hour by maintaining an extracellular pH of 6.4. In the same experiment an increase in the extracellular

pH from 6.4 to 8.0 during recovery resulted in a rapid increase in force development. This increase was more rapid than would be predicted due to the increased proton efflux. The authors postulated that there may be other more direct mechanisms at work in the recovery process. They supported this idea with results from further experiments employing proton efflux inhibitors. These inhibitors had no effect on the recovery of twitch tension at a pH of 7.4. Of particular relevance to the present investigation it was reported that a similar decrease in developed tension was seen during stimulation of frog sartorius muscle with external pH's of 6.4 and 8.0. This result is difficult to explain because proton load increases with intensity and decreased external pH has been shown to decrease the efflux of protons. Once again it would appear that some other factor may participate in the inbility to decrease tension although is likely that the result may be due to methodology. In a more recent study Mainwood et al., (1987) decreased lactate efflux from isolated skeletal muscle by increasing extracellular pH. Under these conditions they showed that muscle tension did not return to normal during recovery but that there was a further decrease in tension. These authors also showed that muscles loaded with a lactate load similar to that seen in fatigue reversed lactate flux and produced a reduction in intracellular pH. The artificial lactate load increased twitch duration and time to peak tension. The alterations were similar to those seen

in normally fatigued muscle but were smaller in magnitude.

It is clear that there is a relationship between muscle function, lactate and pH changes. The relationship is complex and it seems likely that other factors are involved. The model presented by Mainwood must be applied cautiously since the lactate loads utilized (20 - 30 umol) are less than the lactate loads observed in humans during exercise.

The cascade of events resulting in an increased lactate concentration and subsequent decreased blood and muscle pH have been related to observed changes in the electrical profile of muscle. These changes could result in previously recruited fibres becoming ineffective in force production resulting in changes in the electrical characteristics of the entire muscle as it adapted in order to continue to produce the required level of tension.

Neuromuscular Response to Exercise

The Integrated EMG as an Indicator of Muscle Tension.

The electrical characteristics of the two primary fibre types are also different and are related to the metabolic capabilities of the fibre. The FG fibres have a higher recruitment threshold and are therefore recruited primarily at higher levels of force output than the SO fibres (Tanji and Kato, 1972). The SO fibres are enervated by alpha motorneurons which are smaller in diameter than those innervating the FG fibres resulting in lower nerve conduction velocities and longer time to peak force development for the

SO fibres. The FG fibres have a greater peak amplitude and firing frequency and produce greater peak forces than the SO fibres (Milner-Brown et al., 1973). Thus, due to the recruitment pattern and the greater amplitude and firing frequency of fibres recruited at higher tensions it is clear that with increasing load their should be an increase in the electrical output of the muscle.

The surface electromyogram reflects the firing rate, amplitude, recruitment pattern, and fibre type of motor units of the muscle being recorded (Basmajian, J.V., 1978, Komi et al., 1970, Moritani 1986). There have been numerous studies which characterized the relationship between the electromyogram and muscle tension or force in both dynamic and isometric contractions.

Relationship Between Isometric Tension and EMG. Zuniga et al. (1969), examined the relationship between tension and continuously averaged EMG for tensions covering the full range of voluntary isometric contractions of the biceps brachii muscle and reported that a parabolic curve provided a much better fit than a linear one. The authors contend that the difference in the relationship shown by their data and some previous data resulted from the use of a full range of tensions (up to maximum). The linearity of their data occurred at loads that had not been previously used in investigating the relationship. However the coefficient of variation for their average EMG data was 53% and ranged from 45 to 68% in

individual subjects. Further observation of their data shows that the line calculated for the linear regression falls well within the error bars on the plots of EMG vs. tension. Komi and Buskirk (1970) supported the existence of a non-linear relationship while researching the reproducibility of different electrode types in electromyographic recording. A quadratic relationship was exhibited between mean IEMG and and isometric tension of the biceps brachii muscle. Kuroda et al. (1970) reported a linear relationship between average EMG and force while force was maintained at submaximal levels. Once the level of force exceeded submaximal levels the linearity of the relationship decayed. These authors used a linear-plus exponent function to describe the EMG-force relationship over the entire range of forces. Heckathorne and Childress (1981) also used a cineplastic preparation of the human biceps brachii muscle of an amputee in which tension was recorded directly from the distal biceps tendon via a surgically produced tunnel. These authors demonstrated a non-linear relationship between IEMG and tension at several muscle lengths for both isometric and isotonic contractions. Heckathorne and Childress suggest that the linearity demonstrated by other researchers may be due to the synergistic action of other muscles in a normal intact limb.

Contrary to these results some investigators have indicated linear relationships. In an early study, Lippold (1952), demonstrated a linear relationship between the

amplitude of the integrated EMG and the tension produced in a voluntary isometric contraction of the gastrocnemius muscle. Inman et al. (1952) using both inserted wire electrodes and surface electrodes, reported a similar relationship in cineplastic muscle preparations of amputees and concluded that the IEMG could be used as an index of muscle tension during isometric contractions of the biceps brachii muscle. Edwards and Lippold (1956), showed that the previously demonstrated linear relationship between IEMG and tension of an unfatigued muscle was maintained with a previously fatigued muscle with only variations in the slope of the relationship being evident. Close et al. (1960), used indwelling wire electrodes placed in the soleus muscle to determine the relationship between action potential counts and both isometric and isotonic contractions. They demonstrated a linear relationship between isometric tension and action potential counts for ten second isometric contractions at six different muscle lengths. These authors results also suggested that a linear relationship existed between tension developed in isotonic contractions and IEMG. Dejong and Freund (1967), studied the relationship between the twitch tension and the amplitude of the action potential in the adductor pollicis brevis muscle. They concluded that a strong linear association existed between the action potential amplitude and the twitch tension with electrically evoked contractions via the motor nerve. Devries (1968), reported a

linear relationship between maximal isometric tension of the elbow flexor group and the root mean square of the EMG. More recently Moritani and Devries (1978) demonstrated a similar linear relationship between the integrated EMG and force of voluntary isometric contractions of the elbow flexor muscle group. They demonstrated that a linear function fit the data better than either exponential, quadratic, or power functions for both standardized group data and individual data.

Relationship Between Dynamic Contractions and EMG. There has been less research done on the relationship between dynamic repetitive contractions and EMG. Bigland-Ritchie & Joseph (1974), used dynamic cycling exercise to demonstrate a linear relationship between EMG activity (Root Mean Square) of the lateral quadriceps and load for unfatigued muscle at submaximal loads. This relationship was later extended by Petrofsky (1980) who showed that in the absence of fatigue the linear relationship existed for loads up to an independently determined maximum. In contrast during dynamic exercise at power outputs equal to or greater than 60% of maximum their was a steady increase in the amplitude of the RMS EMG that was unrelated to changes in workload. It was concluded that fatigue complicated the relationship between electrical output and tension and made EMG invalid as an indicator of muscle tension.

The variety of experimental protocols, EMG recording and processing techniques make it difficult to compare the results

from the preceding investigations. The preponderance of evidence suggests however that the relationship between the IEMG and tension or force as recorded by surface electrodes is in fact linear for an unfatigued muscle performing both isometric and isotonic contractions. As well IEMG recorded during dynamic repetitive exercise such as cycling has been demonstrated to have a linear relationship in the absence of fatigue (Petrofsky et al., 1980). This research was confirmed by pilot data collected in our laboratory which demonstrated a clearly linear relationship between power output and IEMG during brief bouts of dynamic cycling exercise for increasing power outputs, decreasing power outputs and with randomly selected power outputs. It is also clear from the reviewed research that fatigued muscle responds differently to increased loads with electrical output showing increases that appear unrelated to changes in power output. During incremental exercise to maximum a number of changes occur in the metabolic profile of the working muscle which do not occur in the muscle during brief periods of work. The electromyogram is sensitive to changes in fibre type, recruitment, firing rate, and amplitude of muscle. Therefore any changes in the metabolic environment which affect any of these parameters should be reflected in changes in the surface electromyogram and the Integrated EMG. In particular the accumulation of lactate and the resulting decrease in pH have been hypothesized to be responsible for some of the changes

seen in the electrical profile of the muscle during incremental exercise.

The Integrated EMG as a Non-Invasive Indicator of the Lactate Threshold

There have been several studies which demonstrated a relationship between the anaerobic threshold and muscle activation as determined by electromyography (Moritani 1980, Nagata et al., 1981, Miyashita et al., 1981, Moritani et al., 1984, Viitasalo et al., 1985). An initial investigation by Moritani (1980) provided data which was used to validate an IEMG-Power Method for the non-invasive determination of the anaerobic threshold.

The results indicated that an inflection point in the IEMG of the vastus lateralis appeared during incremental exercise at a oxygen uptake value which correlated very highly ($r = 0.99$ to 0.863, $p<0.0001$) with the oxygen uptake value at the anaerobic threshold. The anaerobic threshold was determined using cardiorespiratory measures or Tvent as earlier defined. Nagata et al. (1981) validated further this method of non-invasive determination of the anaerobic threshold by showing high correlation ($r = 0.921$ $p<0.001$) between the anaerobic threshold determined using arterial blood lactate, PO_2, PCO_2, HCO^-_3 and pH with the anaerobic threshold determined using the IEMG-Power method. In addition Nagata et al. (1981) demonstrated an abrupt increase in the frequency band width at 70% of peak frequency which occurred immediately after the

anaerobic threshold. The increase in the width of this frequency band, which was postulated to be the most active was taken as an indication of previously reported decreases in mean power frequency noted in muscular fatigue. Moritani (1984) supported his own hypothesis by demonstrating high correlations between blood lactate and both IEMG and Mean Power Frequency (MPF) (0.977 to 0.857, $p<0.001$ and -0.962 to -0.862, $p<0.001$ respectively) during incremental forearm exercise to exhaustion. Viitasalo et al. (1985) monitored five different muscles separately during both the ascending and descending phases of the pedal cycle. The results of tests at five different power outputs indicated an apparent non-linearity in the IEMG of all muscles at a power output identified by the authors as the aerobic threshold (Skinner and McClellan, 1979) but no further change in the non-linearity at the anaerobic threshold. The MPF was also monitored but no difference was noted above and below the anaerobic threshold. These studies contend that with increases in power output there is a point where a sudden increase in Integrated EMG (IEMG) activity occurs which is apparently unrelated to changes in workload. This inflection point occurs at a percentage of oxygen uptake which correlates well with the percentage of maximal oxygen uptake at which Tlac occurs. All of these studies have concluded that this relationship would allow for the use of Integrated EMG as a non-invasive indicator of Tlac or the anaerobic threshold.

Proposed Rationale for the Use of the IEMG as an Indicator of the Anaerobic Threshold. The inflection point in the IEMG vs. Power plot is caused by changes in the firing frequency and/or the amplitude of the muscle above that resulting from increased load. As previously discussed, accumulation of lactate in the muscle decreases the muscle pH (Hermansen et al., 1977, Sahlin 1978). It is hypothesized that these changes are the result of the decreased muscle pH. Research reviewed earlier indicated that changes in pH interfere with the function of the contractile apparatus. It is proposed that this interference debilitates certain fibres and necessitates changes to maintain tension or power output. Hakkinen et al. (1986) showed decreased pH to be related to increased recruitment of fast twitch glycolytic fibres as well as increased firing rate of already recruited fibres. This change in muscle fibre recruitment and firing frequency would result in altered electrical characteristics in the total muscle as a result of the greater amplitude and firing frequency of the fast twitch fibres. These altered characteristics in combination with the increasing work load could result in an accelerated, non-linear increase, or inflection point in the Integrated EMG of the working muscle.

The presence of fatigue in a muscle, defined as the inability to maintain or produce a given force, alters the relationship between the IEMG and force. Komi (1984) points to three well documented effects of fatigue on the IEMG.

1) An increase in IEMG activity is observed during a sustained submaximal isometric contraction. The slope of the relationship is dependent on the level of tension with the increase being greater at higher tensions. The time to exhaustion is also shortened with increasing tension.

2) The relationship between IEMG and force is shifted to the left across the entire range of forces in fatigued muscle.

3) During maximal contractions maximum force decreases and the maximum IEMG also declines.

It is clear that alterations in the electrical characteristics of muscle occur as the muscle reaches maximal levels and fatigues. These changes have been related in some instances to changes in the metabolic environment in particular increases in lactate concentration and decreases in pH.

One measure that has been used in the analysis of changes in muscle function is the mean power frequency or power spectrum. During fatiguing contractions the power spectrum has been shown to shift to lower frequencies (Moritani et al., 1986). Komi (1984) cites studies (Komi and Tesch, 1979, Viitasalo and Komi, 1978 and Tesch et al., 1983) which showed a negative relationship between percentage of fast twitch fibres which produce high levels of lactate and the decrease in mean power frequency of the EMG signal. Tesch et al. (1983) indicated a negative relationship between mean power frequency and both increasing levels of lactate and percentage of fast

twitch fibres. Moritani et al. (1984) showed the marked increase in venous blood lactate was accompanied by a statistically significant increase in RMS amplitude and decrease in mean power frequency during incremental forearm exercise. However several studies (Edwards et al., 1972, Karlsson et al., 1975, Broman, 1977, Petrofsky, 1980, Komi 1984, Boubrit, 1983) have pointed out that the time course of recovery from exercise for lactate and pH requires one to several hours depending upon conditions of recovery while the recovery of maximum twitch tension and amplitude as well as mean power frequency have shown an almost immediate recovery to pre-exercise or resting levels. Mills and Edwards (1984) studied subjects with myophosphorylase deficiency who are incapable of producing lactate and demonstrated a frequency shift in their EMG signal greater than that observed in normal controls. These studies in combination with the conclusion by Mainwood and Renaud (1986) that in certain types of fatigue "...intracellular lactacidosis is not the main cause of the suppressed tension..." (p. 648) point to the necessity for further investigation into the proposed relationship between IEMG and lactate. The relationship has come under recent scrutiny. Boubrit (1983) showed a dissociation of the previously demonstrated relationship between decreased MPF and lactate in recovery re-examined the relationship. Helal et al. (1987) examined the response of the power and frequency of the EMG signal during incremental exercise in nine subjects.

A breakpoint was found in the PEMG which occurred at a power output of 275 watts similar to the breakpoint in lactate which occurred at 250 watts. The mean EMG data was found not to represent correctly represent the individual data to the characteristic high inter-subject variability. Therefore the individual subject data was examined. They observed the breakpoint in the PEMG of the vastus lateralis in only five of the nine subjects. Similarly only six of the nine show a decrease in the MPF and the decrease occurred at the final workload.

Possible effects of cadence on the electromyographic signal. The initial study which examined the relationship between IEMG and lactate during incremental cycling exercise utilized a constant power output ergometer. Cadence was allowed to vary between 50 and 80 revolutions per minute. (Moritani, 1980). Coast and Welch (1985) and Boning et al. (1984) have both demonstrated a parabolic relationship between oxygen uptake and cadence at a constant power output.

Force and cadence may be combined in many combinations to provide the same power output. It is likely that different combinations of cadence and force, producing equivalent power outputs, would result in an entirely different recruitment strategy by the muscle. Such recruitment strategies would be reflected in the recorded electromyographic signal. The work by Coast and Welch (1985) suggests the existence of an optimal cadence which would support the premise of altered recruitment

strategy for different cadences. Also, changes in cadence could affect the IEMG simply because with a constant sampling period increases or decreases in cadence would result in similar increases or decreases in the total amount of activity per sampling period such variation should be considered in analysis of electromyographic data.

In subsequent studies however (Nagata et al., 1981, Miyashita et al., 1981, Viitasaalo et al., 1985), similar results were obtained and cadence was "...maintained constant". These studies did not report monitoring of cadence over the course of the test nor was cadence used as the criterion for determining the end of the test. The use of IEMG makes a constant cadence important since variations will alter the amount of activity which is recorded in a given period of time. In a recent study Helal et al. (1987) maintained cadence "rigourously" at 80 rpms. They also reported inflection points in recorded EMG but in only five of nine subjects.

It is important that cadence be monitored and controlled when examining changes in IEMG of a muscle. The results from the investigations above suggest cadence is unrelated to the observed inflection points. However the area needs further investigation to ascertain if the observed relationship between lactate and IEMG is not simply an artifact of variations in cadence which alter the recruitment pattern of the muscle.

Timing and duration of electrical activity as a possible confounding factor. A further possible confounding factor that has not been examined is possible changes in the timing and duration of the electrical activity in relation to the sampling interval. If such changes occurred in a systematic manner with incremental exercise they could result in systematic changes in the IEMG of the working muscle. Timing and duration should be monitored because of the possible contribution of these factors to changes in the recorded electromyographic signal. Elimination of these factors would allow stronger conclusions regarding the effects of the metabolic state of the muscle on the electromyographic signal. The surface electromyogram represents a statistical summation of the electrical activity in a muscle and may not be sensitive enough to detect metabolic changes at the level of the Tlac. If IEMG is to be used in the non-invasive determination of the anaerobic threshold or Tlac this sensititvity is required. Evidence demonstrating a breakdown in the proposed relationship between lactate and the IEMG suggest that the relationship may be coincidental.

Chapter 3

PROCEDURES

The purpose of this study was to examine the effect of changes in blood lactate concentration during an incremental exercise test on the Integrated EMG of the vastus lateralis. These changes were observed in order to test the validity of the IEMG-Power Method for the determination of the lactate threshold. This chapter contains a description of the methodology and instrumentation used to obtain and analyze the experimental data as follows: (a) Research Design; (b) Subjects; (c) Control Condition Protocol; (d) Experimental Condition Protocol; (e) Data Collection; (f) Data Analysis.

Research design

An attempt was made to test the validity of the IEMG - Power Method for determining the lactate threshold by correlation of the percent oxygen uptake at the point of inflection in a Mean Plasma Lactate vs Power plot with the percent of oxygen uptake at the point of inflection in the IEMG vs Power plot. Because no inflection point could be determined in either condition from the IEMG vs Power plots an analysis of the rate of change of the IEMG vs Power plot (Slope) was performed to determine if any difference could be detected between the Control and Experimental Conditions.

Subjects

The subjects were six trained male cyclists accustomed to

maximal efforts. All subjects had at least two years of racing experience.

Control Condition Protocol

In the Control Condition an incremental cycle ergometer test was used to determine maximal oxygen uptake. The protocol consisted of four minutes of unloaded cycling at 90 revolutions per minute (rpm) with subjects receiving visual feedback to assist in maintenance of cadence within +/-1. Increases in power output of 23.5 watts/minute continued until the subject reached a work load which caused the cadence to drop below 90 rpms for more than five seconds. Expired gases were monitored and the following ventilatory parameters V_E, VO_2, Excess CO_2, F_EO_2, F_ECO_2, Respiratory Quotient (R.Q.) were calculated and recorded. Heart Rate (M.H.R.) was monitored and recorded for each workload. Lactate measures were taken at rest and then once during the final eight seconds of each workload. Raw EMG was also amplified, filtered and sampled at 500 hz for the final eight seconds of each workload and stored on floppy disk for later analysis.

Experimental Condition Protocol

The Experimental Condition utilized a high intensity arm exercise protocol on a Monark Arm Ergometer to produce elevated blood lactates. Each subject performed two three minute bouts of arm work at a power output equal to between 0.90 and 1.00 watt per kilogram of body weight. The two

exercise bouts were separated by one minute of rest. A rest period of five minutes followed the completion of the second bout of arm exercise. At the end of five minutes a blood sample was drawn and used to determine the Post Arm Exercise lactate concentration. The Control Condition protocol was repeated immediately following the sampling of the blood.

Pre-trial Muscle Warming. All trials were preceded by the warming of the vastus lateralis muscle by application of an external hot pack to achieve a surface temperature of approximately 40 degrees centigrade to minimize the effects of muscle temperature on the amplitude and conduction velocity of the action potential.

Data Collection

Procedures for Lactate Sampling. In the Control Condition samples of approximately 2 ml were taken from the cephalic vein of the arm at rest and then once every minute after the onset of exercise until test completion. During the Experimental Condition a sample was taken five minutes after the arm exercise protocol and then once every minute as in the Control Condition. Sampling occurred during the final eight seconds of each workload in order that the electromyographic data corresponded with the metabolic data. Samples were centrifuged and the plasma was frozen and stored for later analysis.

Procedures for Lactate Determinations. Lactate determinations were performed enzymatically (Sigma) using a

method involving conversion of lactate to pyruvate following the introduction of lactate dehydrogenase and NAD^+. Values were obtained by comparison of samples with a series of measurements of a stock solution in the 0 - 6.6 mmol/l range. All samples were analyzed in duplicate after adding 0.1 ml of the sample to 2.9 ml of reagent solution. Samples were then measured for optical density at 340 nm. Lactate values were calculated from a least squares regression equation based on the standards from the stock solution.

Procedures for Electromyographic (EMG) Recording. The myoelectric signals were recorded with silver/silver chloride bipolar recording electrodes placed over the distal portion of the right vastus lateralis muscle at an interelectrode distance of 4 cm. A reference electrode was placed over the volar aspect of the anterior crest of the ilium. Skin resistance was reduced by abrading. The EMG signal was first introduced into a Voltage Coupler (Beckman 9878) with a frequency range of 5-2000 hertz (hz) and a maximum sensitivity of 5 microvolts (uv). The signal was then passed through a Variable Preamplifier (Beckman R411) and into a custom made bioamplifier with a frequency range of 20-1000 hz, a gain of 1000, an input impedance of ten megaohms and a common mode rejection ratio (CMMR) of greater than 100 db. The preamplifier setting was adjusted by having the subject perform a single brief isometric contraction with the pedal

crank at 90 degrees past TDC. The preamplifier setting was selected to give an output which would be near ten volts at the highest power output. EMG data sampling was initiated by a five volt Transitor Transistor Logic (TTL) pulse which was generated by breaking a photoelectric beam at Top Dead Centre (TDC) of the pedal cycle. The TDC pulse was recorded simultaneously with the EMG signal. The EMG signals were sampled at a rate of 500 hertz for the final 8 secs of each work load. The signal was amplified, low passed filtered at 1 kHz, and stored for later analysis on floppy disk using a Data General MicroNova mini computer system.

Data Analysis

Integration of EMG Data. Integration was performed over each pedal cycle (CIEMG, TDC to TDC). Integration was performed by a computer program using an algorithm based on the Trapezoid rule.

Timing and Duration of EMG Activity. The duration and timing of the EMG activity for each pedal cycle was calculated in relation to the TDC pulse. The duration and timing values were expressed as a percentage of total cycle time for each burst. A mean duration, beginning point and end point of activity for each cycle was then calculated for each workload.

Determination of Lactate Threshold

Two different methods were tested to determine the most appropriate method of estimating Tlac from the data collected.

The first method is that proposed by Beaver et al. (1985) which utilized a plot of Log-Log transformations for lactate and VO_2. The second method was that employed by Hughson, Weisiger and Swanson (1987) which used a continuous exponential plus constant model for defining a plot of lactate and VO_2. A computer program developed by Hughson compared the fit of the two models to the data by calculating the residual sum of squares and mean square error for the two models. The Log-Log model fit the present data best and was therefore employed to estimate Tlac. Tlac was taken to be the point of intersection of two regressions lines calculated for points lying on either side of a visually identified inflection point. A more complete description of the method is available in Beaver et al. (1985). Tlac was estimated using each subjects Control Trial data and due to the proximity in time of the testing sessions was assumed to be the same for the Experimental Trial. Tlac was expressed in terms of oxygen uptake in litres per minute.

Determination of Rate of Change (Slope) of IEMG vs Power Output

A post-hoc analysis was performed of the slope of the line described by the plot of CIEMG vs Power Output plot for each subject over the entire test duration. As well the slope of the line was determined for the line prior to Tlac and for the line after Tlac. The mean slope for each line was then calculated for both the Experimental and Control Conditions

and an analysis of variance was performed to determine if there was any significant difference in the rate of change in IEMG. In addition Pearson's Product-Moment Correlation was performed between lactate and CIEMG and power output and CIEMG to determine which of the two better predicted the changes in CIEMG.

Chapter 4

RESULTS

The following chapter contains a presentation and analysis of the data obtained from this investigation. The data are categorized as follows: (a) Description of subjects; (b) Analysis of cardio-respiratory data; (c) Analysis of lactate data; (d) Analysis of Integrated EMG data.

Description of Subjects

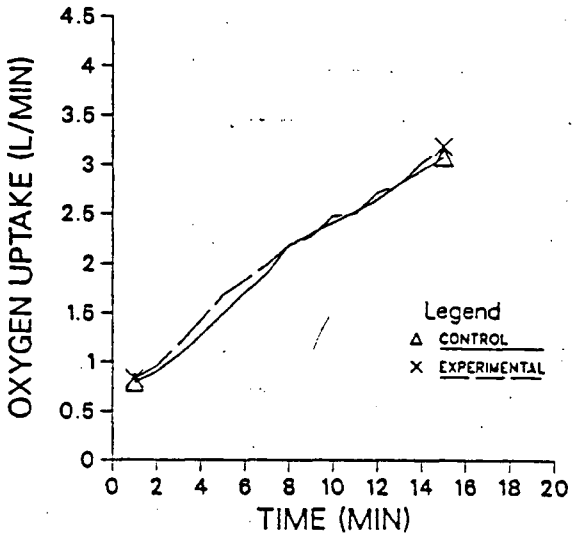
Six trained male cyclists volunteered as subjects for this study. The basic descriptive data on all subjects is included in Appendix E. The subjects' mean age, height and weight were 22 +/-4.1 years, 175.6 +/- 1.2 cm., and 66.0 +/- 5.9 kg. respectively.

Analysis of Cardio-Respiratory Data

The mean cardiorespiratory data was calculated using values up to minute 15. All subjects completed this workload in the Control trial and all except one completed it in the Experimental Trial. There was no significant difference in the mean duration of the incremental exercise tests performed in the Control and Experimental Trials ($t = 1.074$ $p = 0.308$) with only one subject (Subject 6) showing a marked difference in duration.

Control Trial Cardiorespiratory Data. Figure 1a shows the mean Control Trial response in VO_2. The typical leveling off of VO_2 at the final workloads is not evident. Similarly,

(a)



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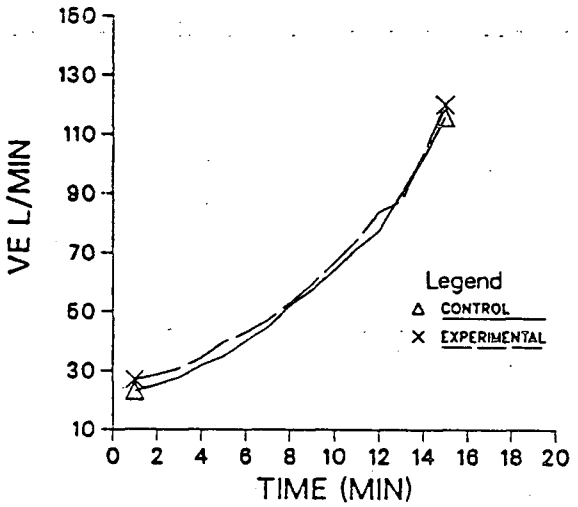

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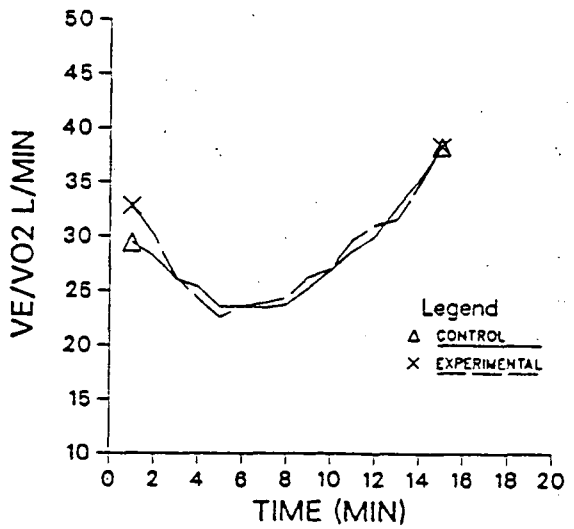


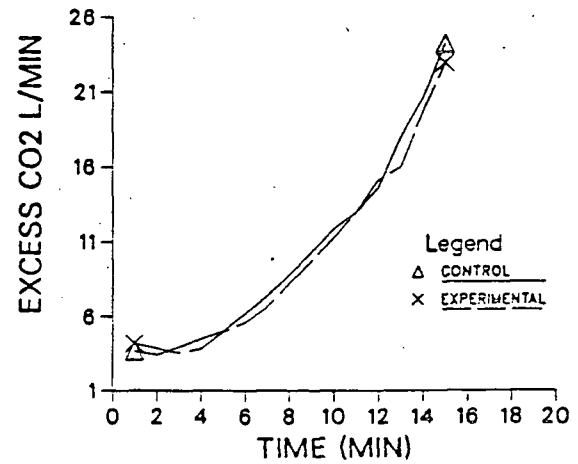


Figure 1 (a) Oxygen Uptake - Control and Experimental Trials.
(b) Ventilation - Control and Experimental Trials.
(c) V_E/VO_2 Ratio - Control and Experimental Trials.
(d) Excess CO_2 - Control and Experimental Trials.

heart rate data does not show the normal leveling off. High R.Q. ratios in (Appendix E - Table 2a, p. 113) do indicate reliance on anaerobic glycolysis at higher power outputs. However these values represent the maximums within the constraints of the incremental protocol and are likely less than subjects' absolute maximum. The mean maximal oxygen uptake value for the Control Trial was 3.15 +/- 0.52 l/min and the mean maximal Ventilation was 133.51 +/- 9.22 l/min. Figures 1b,1c, and 1d indicate typical responses to incremental exercise in $\dot{V}_E$, Excess CO_2, and $\dot{V}_E/\dot{V}O_2$.

Experimental Trial Cardiorespiratory Data. The maximal oxygen uptake and ventilation were 3.29 +/- 0.61 l/min and 127.58 +/- 18.67 l/min respectively. The plots in Figures 1a, 1b,1c, and 1d shows the mean response of the cardiorespiratory measures for the Experimental Trial were very similar to those of the Control Trial.

The results of the analysis of variance displayed in Table 1 show no significant difference in the maximal values for $\dot{V}_E$, $\dot{V}O_2$ and Excess CO_2 between Trials.

Analysis of Lactate Data

Values for lactate determination represent the mean of two spectrophotometric determinations. Mean Plasma Lactate values at rest and for each workload for both the Experimental and Control Trials are displayed in Appendix B - Table 1. All values are likely somewhat higher than the true values. This is due to the haemolysis of some samples. The haemolysis also

Table 1

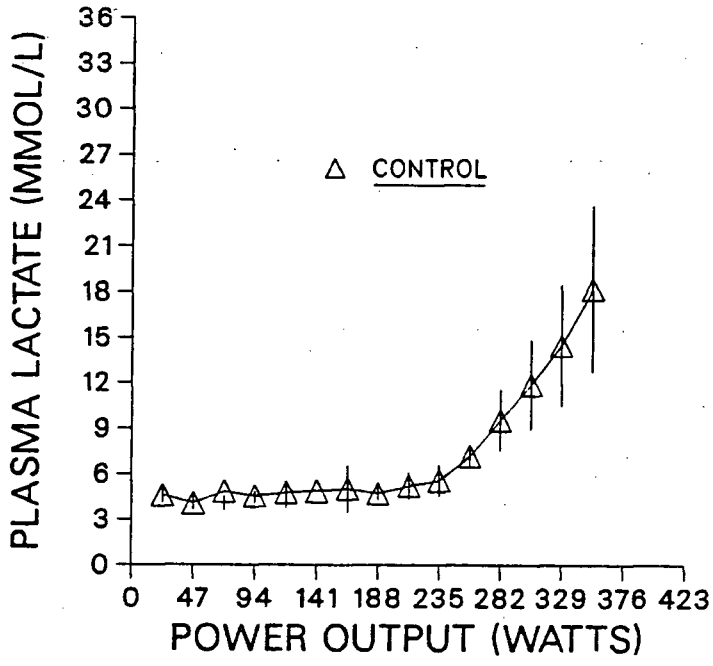
Analysis of Variance of Selected Cardiorespiratory Measures

Comparison of Cardiorespiratory Parameters Between Control and Experimental					
ANALYSIS OF VARIANCE SUMMARY TABLE					
SOURCE	SUMS OF SQUARES	MEAN SQUARES	DF	F RATIO	P
BETWEEN SUBJECTS ERROR	708.4485	141.6897	5		
WITHIN SUBJECTS					
TRIALS (CONT. vs. EXPER.)	61.3872	61.38720	1	.807	.410
ERROR	380.2730	76.05460	5		
VO2, VE, EXCESS CO2	110503.4	55251.68	2	557.280	.00001
ERROR	991.454	99.14536	10		
TRIAL x VO2, VE, CO2	57.35253	28.67627	2	.471	.638
ERROR	609.4042	60.94042	10		

explains some of the observed variability in this measure since the degree of haemolysis was not consistent. As well no correction was performed for the expected plasma water shift that occurs during exercise and the samples were not deproteinized. The spectrophotometric analysis is sensitive to the degree of haemolysis and would result in artificially high values. The lack of correction for the plasma water shift and deproteinization would have a similar effect on the lactate concentration determinations though to a lesser extent. There was no significant difference between the resting lactate values ($t=0.234$ $p>.25$) of the control and experimental trials.

Control Trial Lactate Data. Figure 2a shows the Mean Plasma Lactate rising slightly above resting values and remaining relatively constant until the tenth minute (235.5 watts) when a steady increase to a Mean Maximum Plasma Lactate of 18.21 +/- 5.46 mmol/l is reached during the fifteenth

(a)



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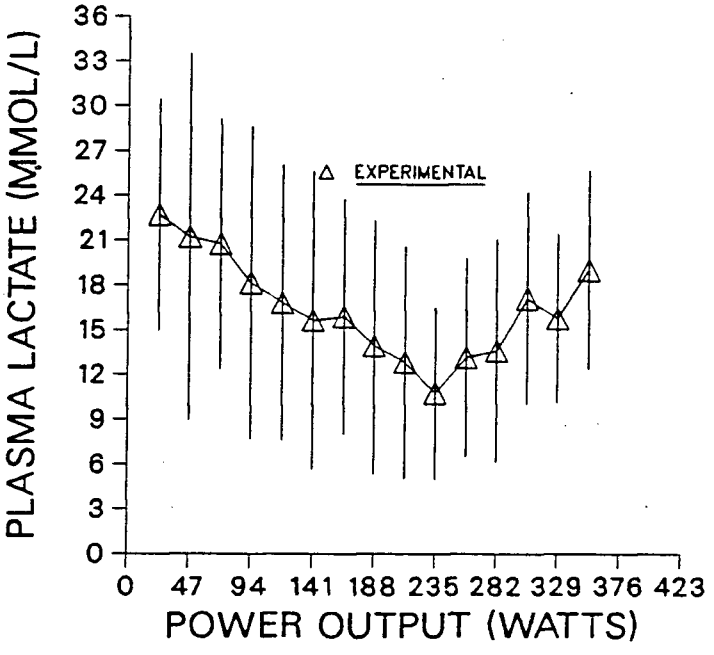


Figure 2 (a) Mean Plasma Lactate vs. Power Output Control Trial
(b) Mean Plasma Lactate vs. Power Output Experimental Trial

minute. The lactate value at the fifteenth minute is used as the maximum Mean Plasma Lactate for the Control Trial because all subjects completed that workload. The averaged data in Figure 2a is representative in direction of the individual data (See Appendix D). The complete data (Appendix E - Table 2a, p.10) shows that Subjects two and six had test durations of 18 and 16 minutes respectively and that individual subject's maximum lactates ranged from 15.21 to 27.68 mmol/l at the end of the Control Trial.

Experimental Trial Lactate Data. The plot in Figure 2b for Mean Plasma Lactate in the Experimental Trial shows a markedly different trend than the Control Trial. The arm ergometer protocol performed by subjects in the Experimental Trial significantly elevated Mean Plasma Lactate to 26.61 +/- 8.86 mmol/l prior to the commencement of the incremental exercise protocol. All subjects with one exception (Subject 2) began the incremental exercise protocol in the Experimental Trial with plasma lactate values that were greater than the maximal values reached at the end of the incremental exercise protocol in the Control Trial. The Mean Plasma Lactate values in the Experimental Trial are clearly higher than those of the Control Trial at all workloads except for the final few workloads.

The high degree of variability evident in the mean lactates of the Experimental Trial (Figure 2b) can be attributed to the variability in the response of the

individual subjects to the arm work. Because of the relatively untrained state of the arms and upper body musclature of these cylists a more variable response to exercise using these muscles is expected. The variability decreases slightly during the course of the incremental protocol but remains quite large. This demonstrates the individual variability in the response to a lactate load and its removal. The varied ability to handle elevated lactate loads is apparent in the control trial as well with the varibility increasing markedly after Tlac.

Anaerobic Threshold Determination Using Plasma Lactates

The anaerobic threshold (Tlac) was determined using the log-log model of Beaver, Wasserman and Whipp (1985) which fit the data more accurately than the continuous model proposed by Hughson, Weisiger and Swanson (1987). The threshold is expressed in terms of oxygen uptake both in absolute values and percentage of maximum achieved. Complete Tlac data for subjects is presented in Appendix E - Table 3. The mean anaerobic threshold was 2.28 +/- 0.37 l/min and is indicated by the arrow in Figure 2a. The anaerobic threshold of the Control Trial was used as the anaerobic threshold in both trials for all subjects. This was due to the altered dynamics of lactate removal and appearance when lactates are elevated prior to incremental exercise. As well an inflection point was undetectable in three of the six subjects during the Experimental Trial. The anaerobic threshold as estimated from

the mean plot of lactate and VO_2 represents a mean power output of 203.9 watts and a value which represents 72.6% of maximum achieved. The estimates of the anaerobic threshold are used in the following analysis of the EMG data.

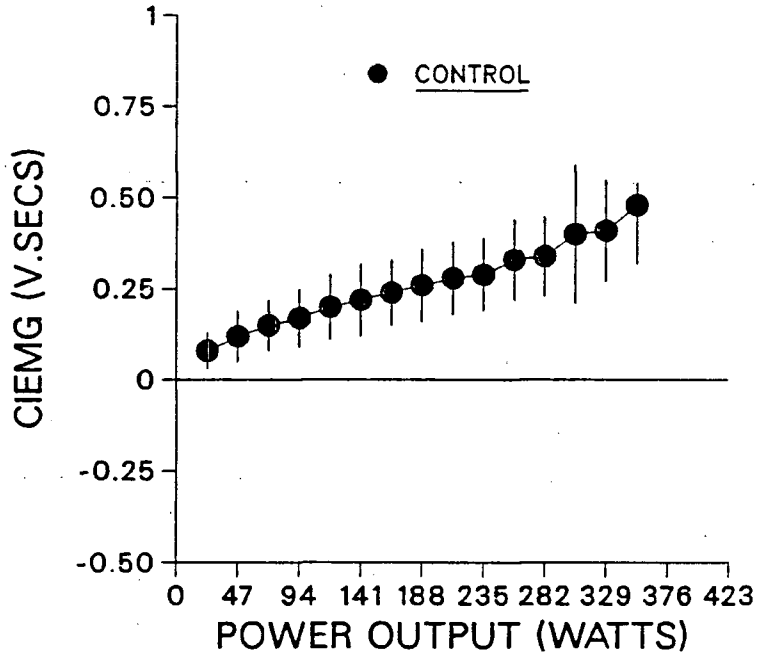
Analysis of Integrated EMG Data

The integral of the electrical activity during each pedal cycle within each sample was calculated. The integrals of each pedal cycle were then averaged to give a mean per cycle value at each workload (CIEMG).

Mean CIEMG was plotted against power output and initially examined for the existence of an inflection point which could be used to estimate the lactate threshold using the IEMG - Power method as outlined by Moritani (1980). This method requires that an inflection point be visually determined prior to deriving its exact location.

Control Trial Integrated EMG Data. An inflection point in the vicinity of Tlac could not be visually identified for any of the subjects in the Control Trial so the IEMG-Power method could not be employed. The trend of the Mean CIEMG data was therefore examined by calculating the slope of the IEMG vs. Power Output for three different sections of each plot. This method also eliminated the problem of high variability between subjects seen (Figures 3a and 3b) in the absolute CIEMG values. The slopes were calculated for both trials using sections of each plot as identified by: (a) Total Exercise Period; b) Pre-Tlac; and c) Post T-Lac. The slope values are

(a)



(b)

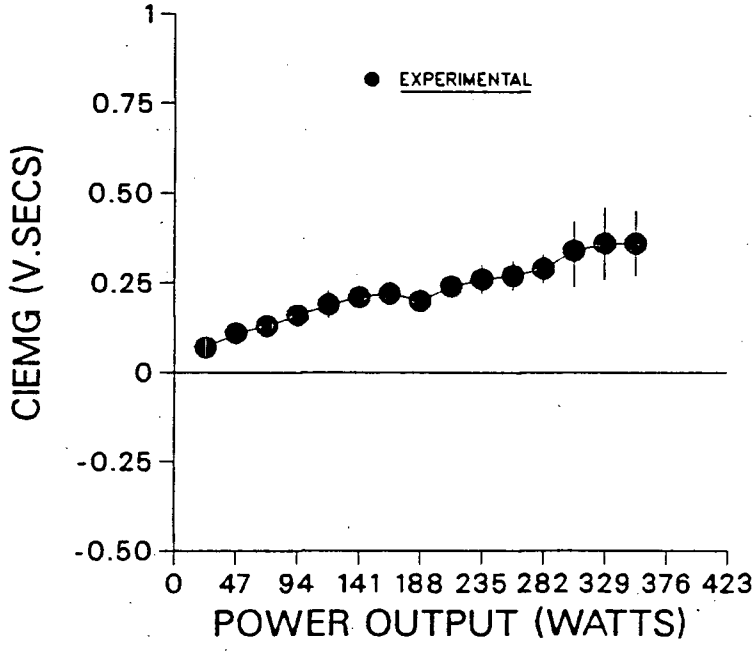


Figure 3a - CIEMG vs. Power Output - Control
Figure 3b - CIEMG vs. Power Output - Experimental

displayed in Table 2. An analysis of variance was performed to determine if any significant difference existed between the slope of the lines for these comparisons. Table 3 indicates there was no significant difference in the slopes of the IEMG vs. Power Output for any of the comparisons made.

Table 2

Slope of IEMG vs. Power Output Plot for the Total Exercise Period and Before and After the Lactate Threshold

	CONTROL TRIAL			EXPERIMENTAL TRIAL		
Subj. #	Slope Total Durat.	Slope of CIEMG Pre-Tlac	Slope of CIEMG Post-Tlac	Slope Total Durat.	Slope of CIEMG Pre-Tlac	Slope of CIEMG Post-Tlac
1	.111	.125	.098	.117	.126	.018
2	.191	.173	.091	.078	.097	.061
3	.121	.073	.429	.131	[a]	[a]
4	.121	.115	.055	.088	.102	.051
5	.082	.054	.182	.114	.118	.182
6	.221	.148	1.82	.108	.080	.229
Mean	.141	.115	.445	.106	.105	.109
S.D.	.050	.040	.631	.020	.020	.082

Note. [a] indicates that there was an insufficient number of points beyond the anaerobic threshold to determine a slope. This subject was not included in the analysis of variance.

Experimental Trial Integrated EMG Data. No identifiable inflection point existed in the vicinity of Tlac in any of the trials in the Experimental Trial. Table 3 indicates that there was no significant difference between Pre-Tlac and Post-Tlac slopes of the IEMG vs. Power plot for the Experimental Trial and that no significant difference existed between Control and Experimental Trials in Total Exercise Period slopes, Pre-Tlac or Post-Tlac slopes.

Table 3

Analysis of Variance of Slopes of IEMG vs. Power Output Plots

Comparison of Slope of IEMG vs. Power Output Between Control and Experimental					
ANALYSIS OF VARIANCE SUMMARY TABLE					
SOURCE	SUMS OF SQUARES	MEAN SQUARES	DF	F RATIO	P
BETWEEN SUBJECTS ERROR	.5207872	.1301968	4		
WITHIN SUBJECTS					
TRIALS (CONT. vs. EXPER.)	.1357441	.1357441	1	1.363	.308
ERROR	.3984209	.0996052	4		
PRE & POST TLAC & TOTAL	.1716329	.085816	2	0 .762	.498
ERROR	.9004025	.1125503	8		
TRIAL x PRE,POST,TOTAL	.1606889	.0803444	2	1.079	.385
ERROR	609.4042	60.94042	10		

The rate of increase in electrical output of the vastus lateralis was not significantly different in either trial. Further there was no significant difference in the pre- and post-Tlac comparisons for any of the subjects in either trial. The plots in Figure 3a and 3b graphically confirm these conclusions. The high degree of intra-subject varibility in the mean CIEMG and mean Plasma Lactate is evident in both measures for both trials. However examination of individual plots (Appendix E, p. 91-96) show that individual subjects data are very similar in direction for both lactate and CIEMG.

Examination of the Relationship Between Lactate and CIEMG

In order to determine the degree to which changes in plasma lactate are reflected in changes in the CIEMG Pearson's Product Moment Correlation was performed for lactate and CIEMG. In the Control Trial only five of the six subjects showed significant correlations with coefficients ranging from 0.57 to 0.97. The mean r value for the subjects showing

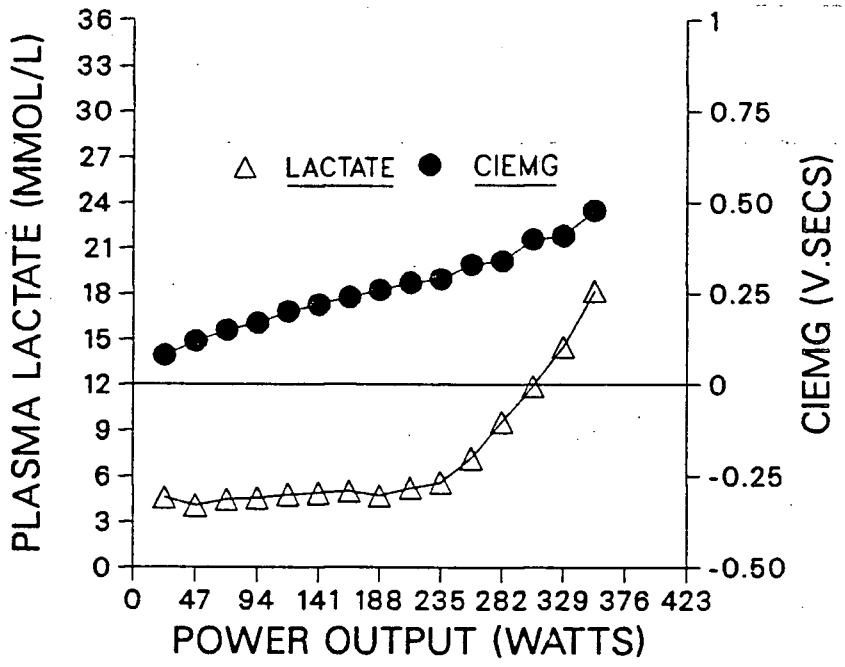
significant correlations in the Control Trial was 0.81+/-0.15.

In the Experimental Trial only three of the six subjects showed significant correlations between lactate and CIEMG. The significant r values in the Experimental Trial were all negative and ranged from -0.62 to -0.96. The mean r value for the Experimental Trial was -0.84+/-0.15. The negative value is expected in the Experimental Trial data since lactate was decreasing for the majority of the test while CIEMG increased steadily throughout. Correlation coefficients for each subject in both trials are contained in Table 4 below.

A further correlation performed between Power Output and CIEMG demonstrated highly significant correlations for all subjects in both trials. The coefficients are displayed in Table 10 above. The r values ranged from 0.92 to 0.99 and from 0.91 to 0.99 in the Control and Experimental Trials respectively. All r values except one were significant at the .00001 level. The mean r value for the Control Trial was 0.96+/-0.02 and the mean r value for the Experimental Trial was 0.95+/-0.03. The mean r^2 value for the Control and Experimental Trials was 0.90 and ranged from 0.84 to 0.98 which indicates that on average 90% of the variability in the CIEMG is explained by the variation in the power output.

In contrast the mean r^2 value for the Control Trial for lactate versus CIEMG is only 0.66. Thus only 66% of the variability in the CIEMG is explained by the varibility in the plasma lactate. It should also be noted once again that

(a)



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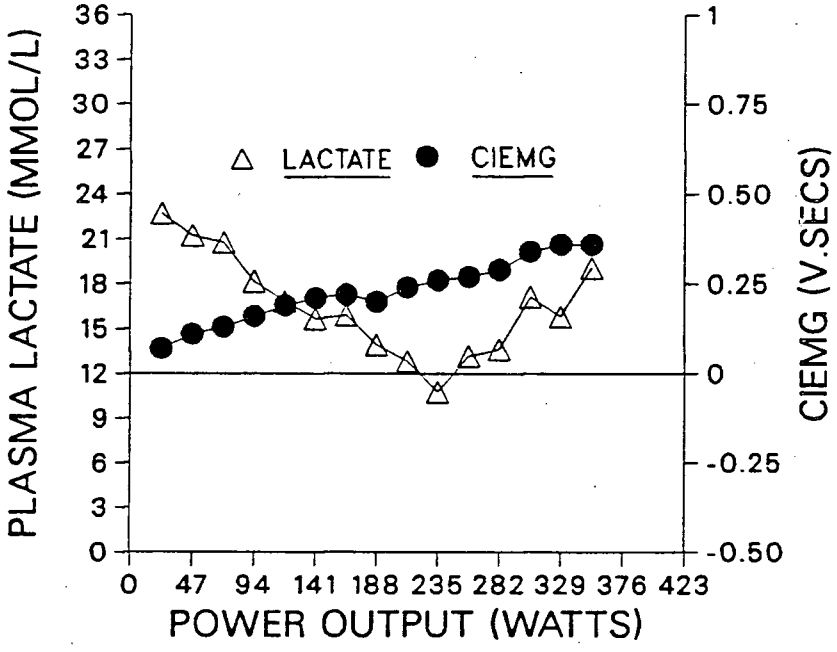


Figure 4a - Mean CIEMG and Plasma Lactate vs. Power Output Control Trial

Figure 4b - Mean CIEMG and Plasam Lactate vs. Power Output Experimental Trial

correlation was significant in five of six subjects. A similar analysis of the r values in the Experimental Trial would indicate that 71% of the variability in CIEMG is explained by the variability in plasma lactate. However the negative correlations would suggest that increasing lactate causes a decrease in CIEMG. This is clearly not the case and indicates the complete reversal of the relationship between lactate and CIEMG during the Experimental Trial.

Table 4

Correlations Coefficients for CIEMG and Power Output and CIEMG and Plasma Lactate for both Trials

POWER OUTPUT VS. CIEMG					LACTATE VS. CIEMG			
	Control		Experimental		Control		Experimental	
#	R	p	R	p	R	p	R	p
1	0.99	10-6 *	0.93	10-6 *	0.82	.0003*	-0.93	.0008*
2	0.92	10-6 *	0.97	10-6 *	0.57	.02 *	-0.62	.02 *
3	0.94	10-6 *	0.99	10-6 *	0.97	10-5 *	-0.96	.0004*
4	0.97	10-6 *	0.96	10-6 *	0.62	.05	-0.34	.41
5	0.94	10-6 *	0.95	10-6 *	0.86	.004 *	0.31	.36
6	0.96	10-6 *	0.91	.000006*	0.71	.03	-0.47	.11
x-	0.96	10-6 *	0.95	.000009*	0.76	.02	-0.50	.15
sd	0.02	n/a	0.03	.000001*	0.13	.02	0.43	.17

Note. - * indicates R value is signficantly greater than 0.

Chapter 5

DISCUSSION

The results of the investigation presented in the previous chapter will be discussed in detail here under the following subsections: (a) Cardiorespiratory and metabolic data; (b) Response of EMG to Plasma Lactate; (c) Explanation of conflicting results.

Cardiorespiratory and Metabolic Data

The cardiorespiratory and metabolic data collected on the subjects in this investigation are in general agreement with previous results.

The mean maximal oxygen uptake value reported for trained cyclists by Simon et al. (1986) was 63.8 +/- 1.3 ml/kg/min. In this investigation a somewhat lower mean value of 47.1 +/- 5.62 ml/kg/min was found. The high R.Q. ratios do indicate a heavy reliance on anaerobic glycolysis and more importantly the anaerobic or lactate threshold was clearly surpassed in all subjects for both trials. The somewhat lower than expected oxygen uptake values are inconsequential for the purposes of this study because Tlac was the point being examined for comparison. However the fact that absolute maximums were not reached may be important in light of the present results and will be considered in the final section. All other cardiorespiratory measures showed typical progressions for incremental exercise. Values were lower than could be expected from trained cyclists but commensurate with

the oxygen uptake values. All values obtained for the cardiorespiratory measures are representative of maximum under the constraints of the protocol used.

The absolute values for plasma lactate concentrations are higher than those reported by other investigators. In the Control Trial a mean maximum value (at 15 minutes) of 18.21 +/- 5.5 mmol/l was found as compared to the mean maximal value of 10.5 +/- 1.1 mmol/l reported by Simon et al. (1986). The plasma lactate determinations of the present study were not corrected for plasma water shifts known to occur during exercise and many of the samples were haemolysed. These factors both would have the effect of elevating plasma lactate concentrations which were spectrophotometrically determined.

Response of EMG to Plasma Lactate

Plasma Lactate Changes in Control Trial. The IEMG has been reported to be useful as a non-invasive estimate of the lactate threshold (Nagata et al., 1981, Moritani et al., 1984, Viitasalo et al., 1985) during incremental work. The results of the present investigation do not support the conclusions of previous work.

The trend of the changes in mean plasma lactate concentration with incremental work for the Control Trial are typical with a very clear and definite inflection point. The anaerobic threshold (Tlac) as defined for this investigation was identified and determined by computer analysis (Hughson et

lactate accumulation. Muscle lactate accumulation is related to the observed decrement in muscle pH (Keul et al., 1972 and Sahlin, 1978) and has been shown to be related to detrimental effects on the contractile apparatus (Fuchs et al., 1970, Donaldson, 1978, Schadler, 1967, Nakamura and Schwartz, 1972). These detrimental effects have been related to altered electrical characteristics which may be reflected in the EMG signal (Häkkinnen et al., 1986, Komi, 1984, Moritani, Muro and Nagata, 1986, Komi and Tesch, 1979, Viitasalo and Komi, 1978, and Tesch et al., 1983) It has been postulated that these relationships should allow for the use of the EMG in the detection of the Tlac.

The Effect of Lactate Accumulation on the CIEMG During the Control Trial. The analysis of the CIEMG data showed no identifiable inflection points when plotted against power output. This made the use of the IEMG-Power method for anaerobic threshold determination as outlined by Moritani (1980) and validated by Nagata et al. (1981) impossible since it is predicated on prior visual identification of an inflection point. This study produced results which are contrary to Moritani et al. (1984). Using incremental forearm exercise with a modified handgrip dynamometer Moritani et al. (1984) were able to show very high correlations for all five subjects between venous lactate, IEMG and MPF (0.977 to 0.857 and -0.960 to -0.862 respectively). In the present investigation correlation coefficients for venous lactate and

CIEMG were significant in only four of six subjects (0.57 to 0.97).

It is notable that Moritani did not employ his own IEMG-Power method for determination of the lactate threshold in this investigation. In fact the study by Moritani et al. (1984) used no objective criterion to determine any of the inflection points. After identifying the lactate threshold as "...an abrupt increase in lactate concentration." Moritani et al. (1984) showed significant differences in IEMG, MPF, and venous lactate between points on either side of the lactate threshold. However close examination of Moritani's data do not support the rationale used to explain his findings. The results show the lactate threshold occuring at a mean venous lactate concentration of approximately 2 mmol/l. This lactate load is less than that shown by previous investigators to be sustainable and demonstrates the possible existence of other mechanisms for the observed inflection points. Moritani (1984) contends that lactate and the accompanying decrement in pH has detrimental effects on the contractile apparatus at venous concentrations of approximately 2 mmol/l and arterial concentrations of 1.5 mmol/l. Jorfeldt (1970) examined the uptake and utilization of lactate using radioactive tracers during steady state forearm exercise at 1.634 watts after arterial lactate concentrations were artificially elevated. Subjects had their arterial lactate concentrations artificially elevated by continuous infusion and then

exercised for a period of 60 minutes. The arterial lactate concentrations after 10 minutes of exercise were 3.35 mmol/l and after 40 minutes were only 3.36 mmol/l. Measurements of pH of arterial blood taken simultaneously showed values of 7.43 and 7.47 at 10 and 40 minutes respectively. These values are in agreement with data presented in Keul et al. (1972) from a previous study by Keul et al. (1967) which showed that at arterial concentrations of 2 mmol/l pH has decreased very little to a value of approximately 7.3. The values presented by Keul et al. (1972) give somewhat higher estimates of pH both at rest and after maximal exercise than some more recent studies (Sahlin et al., 1972 and Sahlin et al., 1976). However despite differences in the absolute values the relative decrement would be expected to be similar given the highly significant negative correlations between lactate and pH (Sahlin, 1978, Keul et al., 1972). The intramuscular pH would be lower than these extracellular values but it seems unlikely that a decrement in pH can explain the observed changes in the IEMG with an arterial lactate load of only 1.5 mmol/l (Moritani ,1984). In fact to suggest that changes observed were related to metabolic alterations is questionable particularly in view of the subjective manner of determining the inflection points. The protocols of the Jorfeldt (1970) and Moritani et al. (1984) were different therefore one could expect some differences in the dynamics of lactate accumulation but the accumulation would still be expected to

have a similar effect on pH making it difficult to attribute changes in the IEMG to small changes in pH. Moritani (1984) did not report lactate values per kilogram of body weight making quantitative comparisons inconclusive.

In the present investigation the resting lactate levels of the Control Trial were more than twice venous lactate levels demonstrated by Moritani et al. (1984) at the lactate threshold. At the lactate threshold present values were more than three times Moritani's values. This is expected given the much larger muscle mass of the vastus lateralis muscle and the elevated absolute lactate values of the present investigation. However if the relationship between lactate and IEMG is to be supported it would be expected that changes previously observed in the IEMG would be clearly evidenced at these lactate loads. The lack of an inflection point in the CIEMG refutes the existence of a predictable relationship between lactate and CIEMG under the conditions of the present investigation. However the difference in results may be attributable in part to differences in methodology and the previously demonstrated relationship may exist under certain specific conditions.

To determine if a visually undetectable change was occuring an analysis of the slopes of these plots was performed using a method adapted from Viitsaalo et al. (19851). Viitsaalo reported a significant difference in slope of the IEMG-Power relationship at power outputs below and above the

arterial lactate threshold. In the present investigation the CIEMG recorded during the Control Trial increased in a near linear fashion despite a marked rapid increase in the plasma lactate concentration once the lactate threshold was reached. The slope of the CIEMG vs Power Output plot was not significantly different when compared before and after the lactate threshold for any of the subjects in the Control Trial. The results of this analysis indicate that the CIEMG shows no sensitivity to changes in blood lactate concentration during incremental exercise using the present protocol and that the use of IEMG as an indicator of the lactate threshold or anaerobic threshold is not reliable under all conditions.

Plasma Lactate Changes in Experimental Trial. The relationship between lactate and IEMG was tested further by altering the normal dynamics of lactate utilization and production. The trend of the changes in mean plasma lactate concentrations of the Experimental Trial show a clear and marked alteration in the dynamics of lactate production and utilization as compared to the Control Trial. The steady decrease in lactate concentration from the initially high value indicates preferential utilization of the lactate produced prior to incremental exercise. As expected this steady decrease continues until the lactate threshold is reached when a sudden increase occurs. At all workloads for all subjects the plasma lactate concentration of the Experimental Trial was greater than those of the Control

Trial. Karlsson et al. (1975) demonstrated that lactate levels were elevated in non-exercising muscle following exhaustive short term exercise by other muscle groups and concluded that:

> ...muscles which have been engaged in heavy exercise exhibit a definite influence on the metabolic situation in other "non-exercised" muscles which will limit their performance. (p. 766)

These investigators showed a decrease in performance time when lactate was elevated prior to an exercise bout. They attributed this decrease in performace to "...one or more local metabolic factors..." and identified phosphagen depletion or lactate accumulation as possible candidates. This would support the premise that the increased concentration of lactate produced through the high intensity arm protocol of the present experiment would have a detrimental effect on the vastus lateralis during cycling. However a significant difference in performance time was not found. The present investigation utilized an incremental exercise protocol following the elevation of plasma lactate. This would make the presence of lactate less detrimental initially and could explain the lack of a statistical difference in mean test duration between the two trials. Recent research has shown clearly that lactate is an important oxidative substrate during exercise (Eldridge, et al., 1974 and Issekutz, 1984). Further the utilization of lactate as a substrate in oxidation is increased with increasing exercise intensity. This

utilization is clearly reflected in the steady decrease in plasma lactate concentration seen in the Experimental Trial. Despite the use of lactate as a substrate, production or appearance of lactate eventually exceeds utilization or disappearance and accumulation occurs (Wasserman, 1986, and Wasserman et al., 1986). This is evidenced by the increase in the plasma lactate seen around Tlac of the Experimental Trial. Despite the considerable utilization of lactate the concentration of the lactate was higher in the Experimental Trial than in the Control Trial for all subjects throughout the exercise period it would be expected that the proposed detrimental effects of increased lactate concentration would have been accelerated in the Experimental Trial. The sensitivity of the CIEMG to changes in plasma lactate should be clearly evidenced under these circumstances if previous research is to be supported.

The Effect of Lactate Accumulation on the CIEMG During the Experimental Trial. During the Experimental Trial a near linear increase, very similar to the Control Trial was again demonstrated in the CIEMG. This trend continued during periods of both decreasing and increasing plasma lactate concentrations. The CIEMG did not respond as predicted in the Control Trial and was also impervious to the clearly altered lactate dynamics of the Experimental Trial. In fact the nature of the relationship between lactate and CIEMG was entirely reversed during the Experimental Trial with

significant negative correlation coefficients in only three of the six subjects (-0.62 to -0.97). The negative correlations in the Experimental Trial are not unexpected. The rationale used to support the existence of a relationship between lactate and IEMG points to decreased pH as a factor in the observed change in IEMG. In the early submaximal loads of the Experimental Trial a decrease in pH would not be expected because there is net lactate utilization and therefore no net proton accumulation. However the lack of a consistent relationship for all subjects in the Experimental Trial indicates that changes in lactate do not affect the CIEMG in a predictable or consistent manner. This demonstrates an insensitivity of the EMG signal to changes in the plasma lactate concentration during incremental exercise protocol of the present investigation. It is important to note that the CIEMG of the Experimental Trial responded almost identically to the CIEMG of the Control Trial. The metabolic changes that occur with an increased lactate load would not be altered in absolute terms from those of the Control Trial however they would be accelerated and the sudden increase in lactate accumulation (Tlac) would be expected to occur earlier than the Tlac of the Control Trial. A subjective comparison of the Experimental and Control Trial (Appendix E) plots indicates that Tlac did occur earlier in the Experimental Trial than in the Control Trial in five of the six subjects. In all instances Tlac was shifted one workload to the left in

the Experimental Trial. This demonstrates that the lactate accumulation and therefore the decrement in pH would have begun earlier in the Experimental Trial. Previous results (Moritani, 1980, Nagata et al., 1981, Moritani et al., 1984, and Viitasalo et al., 1985) would suggest that the IEMG (CIEMG) should have been sensitive to this shift.

Explanation of Conflicting Results

The rationale provided by previous investigators for the relationship between the IEMG and plasma lactate has been that the increase in lactate causes a decrement in muscle pH which in turn impairs some of the contracting fibres through several possible mechanisms. The muscle compensates for this deficit in contractility by recruiting more motor units or by increasing the firing frequency of already recruited fibres. As the power output increases an increasing number of the recruited fibres are fast glycolytic fibres. These fibres produce greater amounts of lactate and have greater peak amplitudes and firing frequency. It is suggested that this would result in an increase in the IEMG greater than that attributed to the increasing power output resulting in the observed inflection point. The theoretical logic is sound and is supported by previous work which has demonstrated a relationship between alterations in the IEMG of a working muscle during exercise and changes in the metabolic environment.

The data of the present investigation can also be

supported by recent research which would suggest that the relationship between IEMG and lactate during incremental exercise is conincidental (Bourbrit, 1983, Mills and Edwards, 1983, Komi, 1984, and Helal et al., 1987). Previous research suggesting that IEMG can be used as an estimate of the lactate threshold is based upon research which indicates a relationship between lactate accumulation and changes in the amplitude and frequency spectrum of a working muscle (Lindstrom et al., 1970, Mortimer et al., 1970, Viitasalo and Komi, 1978, Komi and Tesch, 1979, Tesch et al., 1983, Komi, 1984 and Moritani et al., 1986). This is further supported by research which demonstrates that muscles which are higher in FG fibres and therefore produce greater amounts of lactate show a greater decrease in the center frequency than muscles of predominantly SO fibres (Komi and Tesch, 1979, Tesch et al., 1983). This relationship is well substantiated.

The conflicting data on the nature of the relationship between IEMG and power output in the absence of fatigue must be considered. A number of investigations have demonstrated non-linear relationships. All previous studies examining the postulated IEMG - lactate relationship have reported high correlations between these two variables. None of these studies have reported correlations between power output and IEMG. In the present investigation significant correlations between lactate and CIEMG were demonstrated for five subjects (Control). However in both trials all subjects data showed

power output to be more highly correlated with the changes in CIEMG than was lactate. It is possible that previously observed inflection points in the IEMG during incremental work are simply the result of a non-linear relationship between CIEMG and power output which has been previously demonstrated in the absence of lactate accumulation. The existence of higher correlations between IEMG and power output does not eliminate the possibility of a relationship between lactate and IEMG. However it suggests the relationship may be conincidental and is clearly not causal under all conditions.

Comparison of previous results with present results must also consider methodological differences. There were two major differences between the present investigation and those that employed incremental cycling exercise protocols in previous studies. They were: (1) Cadence (2) Subject's cycling experience. Power output was used as measure of intensity in all investigations. Power output on a cycle ergometer is a product of the cadence and the applied force. Equivalent power outputs may be obtained by varying either of these two factors. There is some data which indicates (Coast and Welch, 1985, Boning et al., 1987) that equivalent power outputs at different cadences do not elicit equivalent metabolic responses. Coast and Welch (1985) reported a parabolic relationship between oxygen uptake and increasing cadence at equivalent power outputs during incremental exercise. These researchers did not monitor EMG but it is

possible the altered metabolic response would be reflected in the EMG signal. It is possible to speculate that a muscle's recruitment pattern/strategy would be entirely different with a light load and high cadence when compared to a heavy load and low cadence. The present investigation used higher cadence (90 rpm) than any of the previous investigations which demonstrated a relationship between IEMG inflection points and lactate inflections points. As well none of the previous studies monitored cadence or used it as a criterion for ending the exercise test. Helal et al. (1987) used a cadence of 80 rpm and reported the inflection points in only five of their nine subjects. It is possible then that the lactate - IEMG relationship may only exist under certain conditions with a particular type of muscle recruitment strategy dictated by a certain force and cadence combination. It is possible to speculate that the inflection point may disappear at higher cadences used in the present investigation and as seems to be occurring in the investigation by Helal et al. (1987). The influence of the varying cadence on recruitment during incremental exercise has not been examined and would provide data to confirm or refute such speculation.

Also the use of a drop in cadence of only one rpm as the criterion for stopping the test seems to have not allowed subjects ot attain their true maximum. This is evident in both the oxygen uptake values and the heart rate data neither of which showed the typical leveling off. It is possible that

CIEMG inflection points may have become apparent if true maximum had been reached. However this does not alter the present conclusion that CIEMG does not respond to increased lactate concentration following under the conditions of this investigation. The present investigation also differed in that it was the only study to employ experienced trained cyclists. It is possible that such athletes could have developed a different recruitment strategy than their untrained counterparts during incremental cycling exercise. Such differences may account in part for the present conflicting results but no evidence is available at this point.

The use of IEMG to estimate the lactate threshold implies that the IEMG is sufficiently sensitive to and driven by changes in lactate concentration and the subsequent decrement in pH. The use of the IEMG to predict the lactate threshold would require that the relatively small changes in pH at the point of the threshold cause alterations in the EMG signal immediately. Data are available which indicate a drop in pH of only 0.5 units from rest to maximal exercise. The lactate levels at Tlac are significantly greater than submaximal levels but less than the maximal levels that are attained at the point of exhaustion. A commensurate decrement in pH would be expected at Tlac. The data of Keul et al. (1972) indicate that at the mean lactate levels of Tlac in the Control Trial (7.18 +/-0.74 mmol/l) of the present experiment a decrement

of approximately 0.1 units in pH from the resting pH of the arterial blood could be expected. Further a decrement of only 0.05 pH units would be expected in the pH from the pH value one minute prior to Tlac. These small decrements in pH may have an effect on the electrical characteristics of the working muscle. It seems unlikely that this small decrement would be reflected in the EMG given the global nature of the surface electromyogram as a measurement tool.

The results of the present experiment clearly do not support the suggestion of such a high degree of sensitivity. In fact these results suggest that changes in lactate concentration are poorly related to changes in CIEMG (IEMG) of the muscle during incremental exercise and show no measureable changes at the intensity represented by the lactate threshold. This conclusion is supported by recent research which shows a an uncoupling of the proposed lactate - IEMG relationship (Bourbrit, 1983, Mills and Edwards, 1983, Komi, 1984, and Helal et al., 1987). The time course for recovery of increased lactate concentration has been demonstrated to be drastically different than the time course for the recovery of the amplitude and frequency characteristics of the EMG signal. Investigations examining this subject have noted that lactate may require anywhere from several minutes to a few hours to return to normal while amplitude and mean power frequency have been shown to recover almost immediately upon the cessation of exercise (Edwards et al., 1972, Karlsson et al., 1975, Broman,

1977, Petrofsky, 1980, Komi, 1984). Bourbrit (1983) showed similar results but in addition showed that the frequeny spectrum had returned to normal even while lactate concentration was still increasing. Mills and Edwards (1983) supported further the lack of a causal relationship between increased lactate concentration and change in the IEMG of a muscle. They showed a greater frequency shift in subjects with myophophorylase deficiency who produce absolutely no lactate during exercise than in normal controls. This would indicate the existence of some other mechanism or mechanisms responsible for the altered EMG signals.

In the present investigation the increase in CIEMG was nearly linear and showed highly significant correlations with power output for all subjects in both conditions. In all subjects the correlation between power output and CIEMG was greater than the correlation between lactate and CIEMG. The changes in CIEMG were predicted better by changes in power output than by changes in lactate. The suggested relationship between IEMG and lactate is based upon the effects lactate accumulation has on pH and the subsequent effects it has on the contractile apparatus. The use of the surface electromyogram and the IEMG to determine the lactate threshold suggests that the effect of increased lactate accumulation and decreased pH has an immediate measureable effect on the recruitment and firing frequency of the working muscle at the level of the lactate threshold. However a majority of the

evidence in this area reports effects on the IEMG which are related to metabolic changes only at or near maximal levels. While the lactate threshold represents a "point of no return" for sustained performance the muscle is still capable of maintaining or even increasing developed tension for a considerable period of time beyond this point.

The relationship between muscle lactate and alterations in the IEMG at the lactate threshold of a working muscle appear to be fortuitous. A change in the IEMG of a muscle as recorded by surface electrodes requires there be: (a) a change in the number of motor units recruited; (b) a change in the firing frequency of already recruited fibres or newly recruited fibres (Bigland and Lippold, 1954, and Milner-Brown et al., 1975). Changes in these parameters have been shown to be related to changes in lactate concentration and to several other factors. However these parameters may also be altered simply by changes in recruitment patterns within a group of muscles performing a complex movement. Consideration of this possibility is important given the summative nature of the surface electromyogram. Procedural difficulties inherent in the use of surface electromyogram must also be considered. The characteristic high inter-individual variability makes grouping of EMG data for analysis difficult. Individual subject data is often presented as characteristic making valid generalizable conclusions impossible. Further the problems in examining data for inflection points without employing an

objective criterion and then performing correlations on the identified points introduces greater possibility of error and makes subsequent conclusions questionable.

On the other hand given the differences in methodology discussed above it is possible that the relationship does exist under certain conditions. The issue of the effect of varying cadence on the recruitment pattern during incremental exercise is important and must be resolved to make any conclusive statements concerning the effect of a changing metabolic environment on the electromyographic signal. The evidence to date in combination with the present results would suggest that while a relationship between lactate and IEMG seems to exist this relationship may be the result of the two variables responding independently to some other factor or factors.

Chapter 6

SUMMARY, CONCLUSIONS, FINDINGS, AND RECOMMENDATIONS

Summary

This investigation was designed to test the hypothesis that changes in plasma lactate concentration drive changes in the electroymyographic profile of working muscle during incremental exercise.

The hypothesis was tested by altering the dynamics of lactate production and utilization of the vastus lateralis during incremental cycle ergometer work and comparing the changes recorded in the IEMG of the same muscle. The anaerobic or lactate threshold was determined for each subject using a log-log transformation of a lactate vs oxygen uptake plot. The lactate threshold was not detectable using the IEMG Power method as presented by previous researchers. Subsequently an analysis of the slope of the IEMG vs oxygen uptake plots was performed before and after the lactate threshold for both conditions. Correlations were determined between lactate concentration and the CIEMG and between power output and CIEMG.

The data were collected on six male cyclists (22.0 +/- 4.1 years). Each subject performed two incremental exercise trials on a cycle ergometer. The protocol consisted of 4 minutes of unloaded cycling followed by linear increases of

23.5 watts per minute at 90 revolutions per minute. The tests were ended when the cyclist deviated from the prescribed cadence for more than five seconds. The same protocol was used for the Experimental Trials but was preceded by two bouts of high intensity arm work followed by five minutes of rest.

Findings

The mean Tlac as determined using log-log plots of the lactate vs oxygen uptake data was found to occur at 77.7 +/- 9.50% of maximum achieved. An analysis of the slope of the CIEMG vs Power Output plots demonstrated that there was no significant difference in the slope of the line before and after the lactate threshold in either condition. Further it was demonstrated that there was a steady increase in CIEMG despite decreasing concentrations of lactate from initially elevated levels in the Experimental Trial.

The changes in CIEMG were significantly correlated with changes in power output in all subjects in both trials. The changes in CIEMG were significantly correlated with changes in lactate in only four of the six subjects in the Control Trial and the correlations were less significant than those demonstrated for power output in all cases. In the Experimental Trial only three of the six subjects showed significant correlations between lactate and CIEMG. In addition the significant correlations were all negative indicating a completely opposite relationship in the Experimental Trial.

Conclusions

The following conclusions were made under the conditions of this investigation.

1. It was hypothesized that a strong positive correlation would be seen between the percentage of maximal oxygen uptake at which an inflection point in blood lactate occured and the percentage of maximal oxygen uptake at which an inflection point in CIEMG occured in the Control Condition. This hypothesis was not supported as no detectable inflection points in CIEMG were found in any subjects in the Control Condition. It is concluded that under the conditions of this investigation the CIEMG is not a valid estimator of the lactate threshold during incremental exercise.

2. In the Experimental Condition it was hypothesized that the inflection point would be shifted to the left in the lactate vs. power output plot. Further the CIEMG vs. power output plot would show a similar shift in the inflection point. This hypothesis was not supported as no inflection point was identified in CIEMG for any of the subjects during the Experimental Condition. A subjective evaluation of the lactate data indicated that Tlac may have been shifted to the left with no observable effect on the CIEMG. It can be concluded that the CIEMG does not respond differently to increased levels of plasma lactate during incremental exercise.

3. The CIEMG is highly correlated with and predicted by

power output.

4. These findings are supported by research which indicates that the relationship between increasing lactate concentrations and IEMG is not valid under all conditions.

Recommendations

The present study utilized well trained cyclists as subjects. It is possible that trained cyclists have developed an ability to compensate for metabolic changes through altered recruitment patterns and reduce the effects such changes might have on the recorded electromyogram signal. At present there is no evidence to support this hypothesis.

The present investigation sampled the electroymyographic signal at a rate of 500 hz. This rate is slower than has been previously used. It would not be expected to eliminate the previously observed relationship between lactate and EMG but could result in reduced resolution of the expected inflection points.

The previous validation of the IEMG-Power method for the non-invasive determination of the anaerobic or lactate threshold is not supported by the present research. The present research would suggest that the previously demonstrated relationship between the IEMG and lactate is fortuitous. The theoretical rationale for the existence of the relationship suggests a sensitivity in the EMG which is unlikely given the nature of the the electromyogram and is not

supported a considerable body of previous work.

The present results cast doubt regarding the sensitivity and reliability of the IEMG as an indicator of the metabolic state of the muscle at the level of Tlac.

The relationship could be examined further. It would be of interest to examine the postulated relationship by:

1. Comparing the IEMG and lactate concentration changes of a group of untrained subjects with those of the trained subjects.

2. Increasing the intracellular lactate directly of the muscle being monitored to determine the effect on the IEMG during incremental exercise.

3. Monitor as many of the muscles as possible involved in cycling to determine if recruitment patterns between trained and untrained cyclists differ during incremental exercise.

4. Validate the IEMG - Power method of determining Tlac or anaerobic threshold by monitoring only IEMG and then predicting maximal performance limits during long term exercise.

5. Examining the power spectrum of the present data.

6. Performing measures of pH to be support lactate data.

7. Use NMR spectroscopy to determine intramuscular changes in pH and their relationship to observed changes in the recorded EMG signal.

APPENDIX A - COMPLETE CARDIORESPIRATORY DATA

TABLE 1 - Oxygen Uptake (l/min) - Control Trial

OXYGEN UPTAKE L/MIN - CONTROL TRIAL

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	1.070	1.093	1.218	1.425	1.518	1.818	1.953	2.233	2.619	2.759	3.084	3.480	3.815	3.983	4.142	3.940		
2	0.850	1.015	1.093	1.407	1.676	1.838	1.860	2.181	2.215	2.462	2.402	2.476	2.747	2.720	3.021	2.888	3.350	3.257
3	0.720	0.843	1.076	1.291	1.732	1.874	2.111	2.309	2.245	2.374	2.420	2.504	2.539	2.714	2.729			
4	0.738	0.835	1.099	1.259	1.483	1.637	1.840	1.965	1.885	2.037	2.076	2.238	2.469	2.738	2.791	2.840		
5	0.647	0.834	0.986	1.172	1.351	1.650	2.030	2.317	2.511	2.409	2.792	2.818	2.933	3.075	3.265			
6	0.717	0.778	0.939	1.036	1.175	1.404	1.644	2.098	2.350	2.431	2.394	2.351	2.375	2.432	2.541	2.592		
MEAN	0.790	0.900	1.069	1.265	1.489	1.704	1.906	2.184	2.304	2.412	2.528	2.645	2.813	2.944	3.082	3.065	3.350	3.257
S.D.	0.139	0.113	0.089	0.134	0.188	0.162	0.150	0.123	0.235	0.210	0.324	0.414	0.484	0.501	0.526	0.518		

TABLE 2 - Oxygen Uptake (ml/min) - Experimental Trial

OXYGEN UPTAKE ML/MIN - CONTROL TRIAL

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	14.93	15.25	16.98	19.87	21.17	25.36	27.24	31.14	36.52	38.47	43.01	48.54	53.21	55.55	57.77	54.95		
2	11.02	13.17	14.18	18.25	21.74	23.84	24.13	28.29	28.73	37.93	31.15	32.11	35.63	35.28	39.19	37.45	43.45	42.25
3	10.77	12.60	16.09	19.29	25.88	28.01	31.56	34.52	33.55	35.49	36.17	37.42	37.95	40.57	40.79			
4	12.77	14.45	19.01	21.79	25.65	28.32	31.83	34.00	32.62	35.24	35.92	38.72	42.71	47.38	48.29	49.10		
5	10.48	13.49	15.95	18.97	21.86	26.70	32.84	37.49	40.63	38.98	45.18	45.60	47.46	49.76	52.84			
6	11.60	12.58	15.18	16.75	19.00	22.70	26.58	33.93	37.99	39.31	38.71	38.02	38.39	39.33	41.08	41.91		
MEAN	11.93	13.59	16.23	19.15	22.55	25.82	29.03	33.23	35.01	37.57	38.36	40.07	42.56	44.65	46.66	45.85	43.45	42.25
S.D.	1.53	0.97	1.51	1.53	2.46	2.07	3.21	2.88	3.87	1.62	4.67	5.46	6.11	6.89	6.90	6.71		

TABLE 3 - Oxygen Uptake (l/min) - Control Trial

OXYGEN UPTAKE L/MIN - EXPERIMENTAL TRIAL

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	0.865	1.031	1.197	1.287	1.546	1.733	1.774	2.018	2.104	2.296	2.369	2.637	2.863	3.030	3.158			
2	0.668	0.955	1.141	1.375	1.652	1.677	1.739	2.019	2.121	2.385	2.405	2.867	3.059	3.479	3.774	4.026	4.233	4.407
3	0.634	0.664	1.037	1.145	1.264	1.503	1.734	1.878	2.015	2.186	2.171	2.240	2.348	2.464	2.393			
4	0.783	0.853	1.101	1.295	1.501	1.609	1.890	1.968	2.019	2.105	2.156	2.309	2.523	2.732	3.014	3.198		
5	0.845	1.002	1.203	1.548	1.864	2.077	2.256	2.491	2.619	2.841	3.043	3.160	3.246	3.390	3.590			
6	1.158	1.243	1.394	1.650	1.892	2.254	2.420	2.577	2.755	2.765	3.058	2.903	3.089					
MEAN	0.826	0.958	1.179	1.383	1.620	1.809	1.969	2.159	2.272	2.430	2.534	2.686	2.855	3.019	3.186	3.612	4.233	4.407
S.D.	0.171	0.176	0.112	0.169	0.216	0.267	0.270	0.271	0.299	0.279	0.377	0.329	0.321	0.385	0.483	0.414		

TABLE 4 - Oxygen Uptake (ml/min) - Experimental Trial

OXYGEN UPTAKE ML/MIN - EXPERIMENTAL TRIAL

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	12.26	14.62	16.97	18.25	21.92	24.57	25.14	28.61	29.83	32.56	33.58	37.38	40.58	42.95	44.77			
2	8.78	12.54	14.99	18.07	21.70	22.03	22.85	26.54	27.87	31.35	31.61	37.68	40.20	45.72	49.59	52.90	55.62	57.91
3	9.48	9.92	15.49	17.10	18.88	22.45	25.89	28.05	33.55	32.65	32.42	33.46	35.06	36.81	35.74			
4	13.50	14.75	19.05	22.40	25.97	27.84	32.69	34.05	34.93	36.42	37.30	39.94	43.65	47.20	52.15	55.33		
5	13.63	16.17	19.41	24.97	30.06	33.50	36.39	40.18	42.24	45.82	49.08	50.97	52.35	54.68	57.90			
6	18.67	20.04	22.46	26.59	30.49	36.33	39.00	41.53	44.40	44.56	49.28	46.78	49.78					
MEAN	12.72	14.67	18.06	21.23	24.81	27.79	30.33	33.16	35.47	37.73	38.87	41.04	43.60	45.47	48.03	54.12	55.62	57.91
S.D.	3.242	3.114	2.562	4.000	4.780	5.960	6.630	6.490	6.620	6.410	8.210	6.550	6.460	5.818	7.463	1.215		

TABLE 5 - Ventilation (l/min) - Control Trial

	CONTROL TRIAL - $\dot{V}_E$																	
SUBJECT	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	27.90	28.17	30.82	35.45	36.95	42.12	46.16	51.26	55.55	58.22	63.16	72.10	89.12	104.8	128.1	148.1		
2	32.58	28.95	35.20	40.13	45.86	50.00	59.74	61.26	67.02	69.97	74.66	77.57	82.88	88.00	96.29	116.6	123.64	142.45
3	23.73	26.48	29.51	32.57	38.27	42.82	47.39	55.58	58.14	64.91	77.53	83.66	94.26	106.2	125.8			
4	23.49	28.84	31.88	36.62	39.42	44.65	50.62	57.16	65.09	72.03	81.03	93.62	107.3	117.0	131.7			
5	18.31	21.75	24.12	28.64	30.62	36.08	44.50	53.60	63.55	73.96	84.39	89.15	108.7	121.6	131.8			
6	19.98	25.04	28.44	27.86	34.57	35.31	40.93	50.20	57.01	61.97	66.04	77.94	90.12	105.8	121.2			
MEAN	24.33	26.83	29.95	33.55	37.62	41.83	48.22	54.84	61.06	66.84	74.47	82.34	95.36	107.2	121.5	132.4	123.64	142.45
S.D.	4.22	2.44	2.78	4.35	3.91	4.58	5.78	4.25	5.65	7.81	9.33	12.68	13.3	14.5	13.8	16.0		

TABLE 6 - Ventilation (l/min) - Experimental Trial

	EXPERIMENTAL TRIAL - $\dot{V}_E$																	
SUBJECT	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	24.94	26.61	28.06	28.42	32.43	36.56	37.48	42.60	48.67	54.96	61.29	69.12	77.07	92.09	112.1			
2	20.92	26.51	27.56	33.58	39.94	44.70	48.51	55.32	59.55	66.38	71.23	80.40	87.11	101.6	116.0	130.9	144.7	158.69
3	26.15	23.79	30.37	34.37	36.76	39.14	46.25	50.93	57.33	61.20	66.78	74.86	84.16	99.07	110.1			
4	21.92	24.39	27.89	30.89	34.87	38.96	45.09	51.02	56.98	64.98	71.98	80.99	92.01	107.1	118.7	129.6		
5	26.01	27.95	29.65	33.45	39.55	43.57	49.36	53.21	63.53	70.57	78.29	88.81	98.13	118.4	144.5			
6	42.19	43.44	42.30	45.27	45.95	52.30	55.45	56.84	64.63	71.30	82.77	96.74	110.3					
MEAN	27.02	28.78	30.97	34.33	38.25	42.54	47.02	51.65	58.44	64.89	72.06	81.82	91.46	103.7	120.3	130.2	144.7	158.69
S.D.	7.07	6.71	5.17	5.29	6.36	6.23	5.76	6.55	6.88	8.61	11.26	13.16	7.12	8.82	12.46	0.625		

TABLE 7 - Excess CO_2 (l/min) - Control Trial

CONTROL TRIAL - EXCESS CO_2

SUBJECT	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	5.80	6.39	6.58	6.60	6.74	7.67	7.92	9.13	10.92	11.19	11.73	16.48	23.79	25.8	29.53	27.26		
2	2.80	3.05	1.17	1.42	2.23	3.10	4.85	6.28	6.76	7.40	8.42	9.75	9.62	10.59	12.31	14.74	18.23	19.98
3	3.90	4.03	4.22	4.96	6.86	8.83	9.75	11.82	11.75	13.35	15.48	16.64	18.87	22.02	26.56			
4	4.36	1.99	5.72	6.56	7.58	8.47	9.85	10.98	11.85	13.3	13.59	15.43	18.56	22.75	24.79	27.79		
5	3.36	3.55	3.99	4.90	5.36	5.81	8.42	9.64	12.79	16.44	17.6	17.98	23.28	25.69	31.90			
6	1.67	1.43	2.01	2.52	1.40	3.11	3.41	4.62	7.66	9.51	10.86	11.28	13.99	17.03	20.49	24.46		
MEAN	3.65	3.41	3.95	4.49	5.03	6.16	7.37	8.75	10.29	11.87	12.95	14.59	18.02	20.65	24.26	23.56	18.23	19.98
S.D.	1.29	1.60	1.91	1.93	2.38	2.36	2.42	2.53	2.26	2.92	3.02	3.01	4.98	5.36	6.49	5.25		

TABLE 8 - Excess CO_2 - Experimental Trial

EXPERIMENTAL TRIAL - EXCESS CO2

SUBJECT	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
EXPERIMENTAL TRIAL																		
1	2.99	3.03	2.92	2.25	3.17	3.74	3.83	5.38	6.54	8.08	9.28	12.40	15.84	17.97	23.36			
2	1.98	2.29	2.01	3.25	3.97	3.98	5.26	6.03	7.40	8.07	9.66	11.64	13.22	15.71	19.38	23.54	27.17	29.39
3	3.55	2.51	2.29	3.50	3.70	4.11	5.65	6.89	8.52	9.53	11.39	13.52	16.55	21.25	23.34			
4	5.21	5.36	5.80	6.46	7.68	8.42	9.56	10.78	11.94	13.51	13.81	16.01	18.96	23.05	25.83	28.01		
5	3.94	2.99	2.97	1.88	3.61	4.34	5.74	7.24	8.46	10.46	12.20	13.38	15.38	20.45	23.03			
6	7.35	6.94	5.29	5.37	5.47	7.70	8.99	9.76	12.69	15.14	18.02	21.35	23.83					
MEAN	4.17	3.85	3.55	3.80	4.60	5.38	6.51	7.60	9.25	10.79	12.39	14.72	17.29	19.69	22.99	25.78	27.17	29.39
S.D.	1.72	1.71	1.46	1.65	1.70	2.09	2.25	2.13	2.48	2.92	3.22	3.57	3.69	2.57	2.07	2.24		

TABLE 9 - $\dot{V}_E/\dot{V}O_2$ - Control Trial

CONTROL TRIAL - VE/VO2

SUBJECT	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	26.07	25.77	25.30	24.88	24.34	23.17	23.64	22.96	21.21	21.10	20.48	20.72	23.36	26.30	30.92	37.58		
2	34.16	32.10	26.49	25.02	23.94	24.95	26.88	27.39	27.66	27.22	29.13	30.15	28.24	30.47	29.13	33.34	34.81	37.96
3	32.96	31.41	27.43	25.23	22.10	22.85	22.45	24.07	25.90	27.34	32.04	33.41	37.12	39.11	46.08			
4	28.55	28.13	26.24	25.32	24.69	24.08	24.27	25.76	30.32	31.95	34.70	36.21	37.92	39.20	41.91	46.38		
5	28.30	26.08	24.46	24.44	22.66	21.87	21.92	23.13	25.31	30.70	30.23	31.64	37.06	39.56	40.36			
6	26.51	25.68	26.67	27.45	23.71	24.62	21.48	19.51	21.36	23.45	25.89	28.09	32.82	37.06	41.65	46.75		
MEAN	29.43	28.20	26.10	25.39	23.57	23.59	23.44	23.80	25.29	26.96	28.74	30.04	32.75	35.28	38.34	41.01	34.81	37.96
S.D.	3.07	2.65	0.96	0.97	0.91	1.07	1.81	2.46	3.25	3.78	4.57	4.88	5.37	5.09	6.16	5.75		

TABLE 10 - $\dot{V}_E/\dot{V}O_2$ - Experimental Trial

EXPERIMENTAL - VE/VO2

SUBJECT	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	28.83	25.81	23.44	22.08	20.98	21.10	21.13	21.11	23.13	23.94	25.87	26.21	26.92	30.39	35.51			
2	31.32	27.76	24.15	24.42	24.18	26.65	27.90	27.40	28.08	27.83	29.62	28.04	28.48	29.20	30.73	32.51	34.18	36.01
3	41.25	35.83	29.29	30.02	28.03	26.04	26.67	27.12	28.45	28.00	30.76	33.42	35.84	40.21	46.01			
4	27.99	28.59	25.33	23.85	23.23	24.21	23.86	25.92	28.22	30.87	33.39	35.08	36.47	39.21	39.37	40.53		
5	30.78	27.89	24.65	21.61	21.22	20.98	21.88	21.36	24.26	24.84	25.73	28.10	30.23	34.93	40.24			
6	36.43	34.95	30.34	23.93	23.20	22.91	22.06	23.46	25.79	27.07	33.32	35.69						
MEAN	32.77	30.14	26.20	24.32	23.47	23.65	23.91	24.40	26.32	27.09	29.78	31.09	31.59	34.79	38.33	36.52	34.18	36.009
S.D.	4.65	3.82	2.64	2.75	2.33	2.21	2.54	2.57	2.08	2.26	3.12	3.75	3.88	4.46	5.09	4.01		

APPENDIX B - PLASMA LACTATE DATA

TABLE 1 - Plasma Lactate Concentrations (mmol/l) at Each Workload - Control Trial

PLASMA LACTATE CONCENTRATIONS AT EACH WORKLOAD
CONTROL TRIAL

#	REST	ARM	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	5.18	*****	5.32	3.60	3.86	4.36	3.06	****	4.09	4.71	6.30	4.68	7.82	9.56	14.08	16.45	18.81	*END*		
2	3.67	*****	4.24	4.69	5.14	5.62	4.52	4.84	3.99	5.33	4.41	4.73	5.70	5.39	6.05	6.73	7.88	8.64	11.47	15.2
3	4.26	*****	****	****	4.07	****	5.44	****	5.19	****	6.41	****	7.69	9.35	12.25	14.66	18.96	*END*		
4	5.39	*****	4.25	****	4.21	****	****	****	8.21	****	4.65	7.20	7.09	10.88	12.99	17.27	22.10	*END*		
5	3.91	*****	4.90	3.93	5.29	3.71	5.03	4.90	3.58	4.72	4.09	4.82	7.01	10.50	14.04	17.38	23.31	27.68	*END*	
6	5.39	*****	4.25	****	4.21	****	5.87	****	5.01	4.13	5.45	6.46	7.77	11.74	*END*					
MEAN	4.63		4.59	4.07	4.48	4.56	4.78	4.87	5.01	4.72	5.22	5.58	7.18	9.57	11.88	14.50	18.21	18.16	11.47	15.21
S.D.	0.71		0.44	0.46	0.38	0.79	0.97	0.03	1.54	0.42	0.90	1.05	0.74	2.03	2.99	4.00	5.46	9.52		

TABLE 2 - Plasma Lactate Concentrations (mmol/l) at Each Workload - Experimental Trial

PLASMA LACTATE CONCENTRATIONS AT EACH WORKLOAD
EXPERIMENTAL TRIAL

#	REST	ARM	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
1	5.54	23.48	23.72	*****	19.91	*****	7.89	*****	10.67	*****	5.09	4.71	5.99	5.36	*END*					
2	3.01	10.34	9.67	8.95	7.99	7.69	7.15	5.69	5.62	5.54	5.02	5.05	5.64	5.56	6.64	7.89	9.69	13.0	17.1	20.1
3	3.40	35.43	29.91	*****	28.42	*****	26.82	*****	23.91	*****	19.42	15.88	17.70	16.73	20.35	*END*				
4	5.01	29.37	23.72	*****	19.91	*****	*****	*****	15.57	*****	*****	*****	16.05	13.71	17.27	20.51	24.34	*END*		
5	3.23	24.18	16.45	*****	14.75	*****	13.62	*****	11.32	*****	10.36	9.19	9.62	13.03	13.50	19.11	23.21	*END*		
6	6.64	36.84	32.68	33.51	33.51	28.62	28.66	25.62	28.28	22.33	24.31	19.08	24.01	27.37	27.99					
MEAN	4.47	26.61	22.69	21.23	20.75	18.16	16.83	15.66	15.90	13.94	12.84	10.78	13.17	13.63	17.15	15.84	19.08	13.02	17.16	20.07
S.D.	1.35	8.86	7.77	12.28	8.39	10.47	9.21	9.97	7.87	8.41	7.78	5.78	6.67	7.44	7.09	5.65	6.65			

APPENDIX C - ELECTROMYOGRAPHIC DATA

TABLE 1 - Cycle Integrated EMG (CIEMG) for Each Workload - Control Trial *

CONTROL TRIAL
CYCLE INTEGRATED EMG (CIEMG) FOR EACH WORKLOAD

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1	0.03	0.08	0.11	0.14	0.17	0.19	0.21	0.23	0.26	0.28	0.33	0.36	0.37	0.41	0.43		
2	0.19	0.27	0.30	0.34	0.39	0.43	0.42	0.46	0.46	0.47	0.53	0.52	0.78	0.57	0.63	0.63	0.64
3	0.06	0.09	0.11	0.13	0.14	0.16	0.18	0.19	0.2	0.19	0.22	0.24	0.27	0.34	0.42		
4	0.07	0.10	0.12	0.15	0.18	0.19	0.2	0.22	0.25	0.28	0.3	0.3	0.29	0.29	0.31		
5	0.06	0.07	0.08	0.09	0.11	0.12	0.14	0.15	0.16	0.18	0.21	0.21	0.21	0.23	0.36		
6	0.08	0.12	0.16	0.19	0.22	0.22	0.29	0.31	0.35	0.35	0.41	0.42	0.45	0.59	0.75		
MEAN	0.08	0.12	0.15	0.17	0.20	0.22	0.24	0.26	0.28	0.29	0.33	0.34	0.40	0.41	0.48	0.63	0.6
S.D.	0.05	0.07	0.07	0.08	0.09	0.10	0.09	0.10	0.10	0.10	0.11	0.11	0.19	0.14	0.16	0	0

TABLE 2 - Cycle Integrated EMG (CIEMG) - Experimental Trial *

EXPERIMENTAL TRIAL
CYCLE INTEGRATED EMG (CIEMG) FOR EACH WORKLOAD

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1	0.08	0.15	0.20	0.21	0.26	0.24	0.25	0.16	0.29	0.32	0.34	0.36	0.39	0.38	0.37	0.42	
2	0.06	0.09	0.12	0.15	0.16	0.17	0.19	0.19	0.21	0.21	0.23	0.25	0.26	0.29			
3	0.05	0.09	0.11	0.13	0.15	0.18	0.20	0.20	0.24	0.27	0.29						
4	0.06	0.08	0.10	0.12	0.16	0.18	0.17	0.18	0.20	0.21	0.23	0.23	0.24	0.23	0.25		
5	0.07	0.11	0.12	0.17	0.23	0.26	0.26	0.24	0.26	0.31	0.29	0.29	0.36	0.39	0.47		
6	0.11	0.13	0.14	0.17	0.19	0.20	0.22	0.23	0.25	0.26	0.26	0.30	0.45	0.51			
MEAN	0.07	0.11	0.13	0.16	0.19	0.21	0.22	0.2	0.24	0.26	0.27	0.29	0.34	0.36	0.36	0.42	
S.D.	0.02	0.02	0.03	0.03	0.04	0.03	0.03	0.03	0.03	0.04	0.04	0.04	0.08	0.10	0.09		

APPENDIX D - INDIVIDUAL SUBJECT PLOTS OF CIEMG AND PLASMA LACTATE FOR BOTH TRIALS

Figure 1 - Control Trial Subject 1

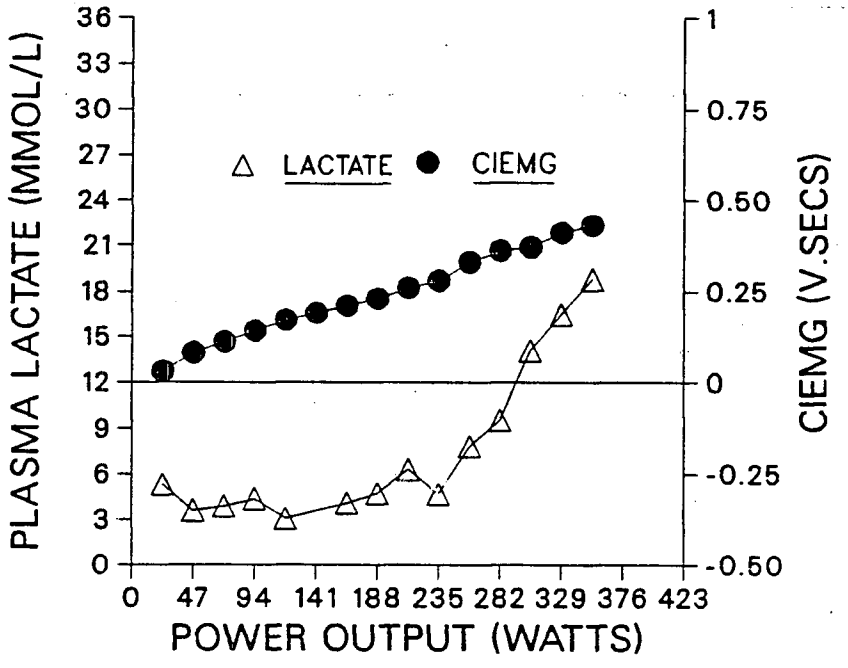


Figure 2 - Experimental Trial Subject 1

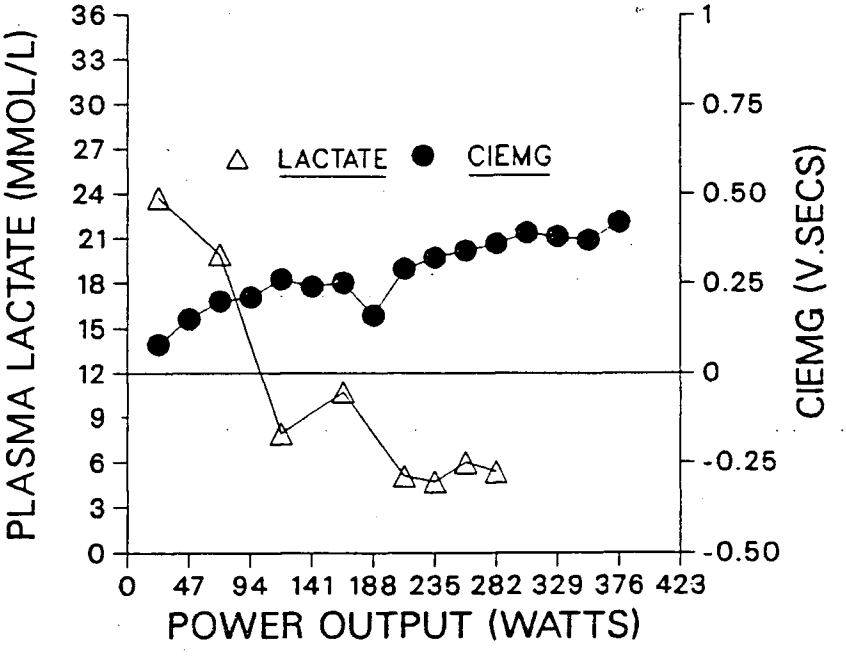


Figure 3 - Control Trial Subject 2

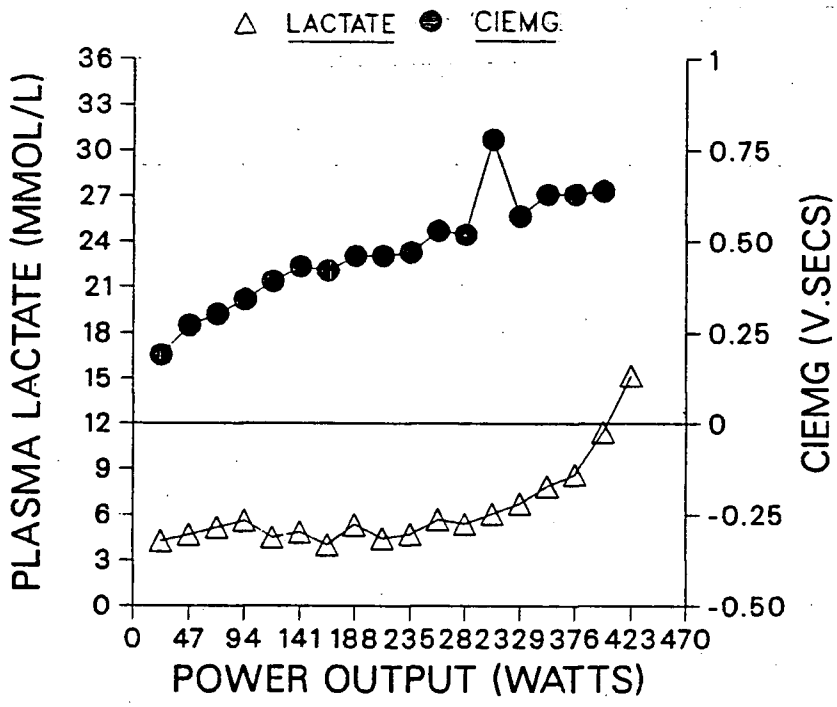


Figure 4 - Experimental Trial Subject 2

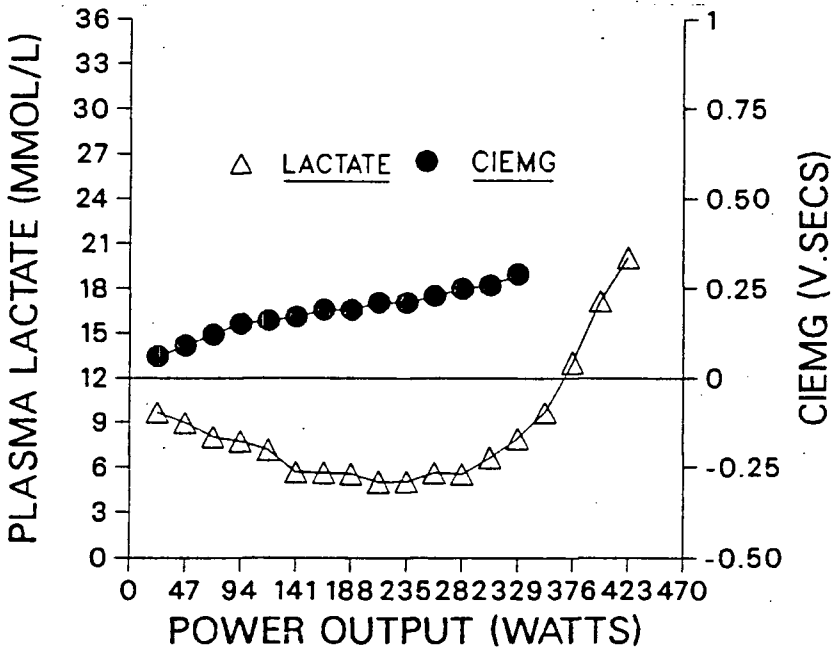


Figure 5 - Control Trial Subject 3

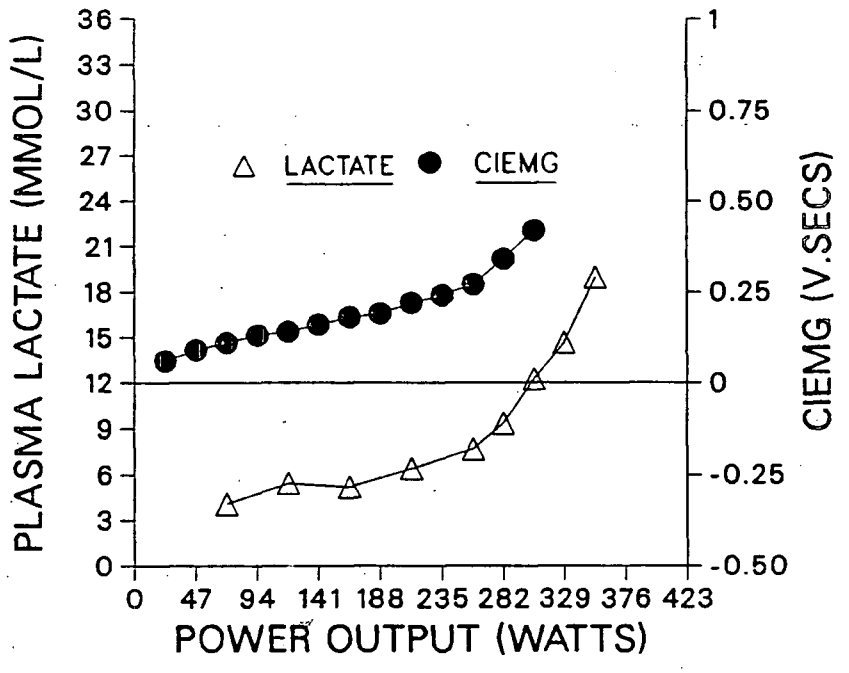


Figure 6 - Experimental Trial Subject 3

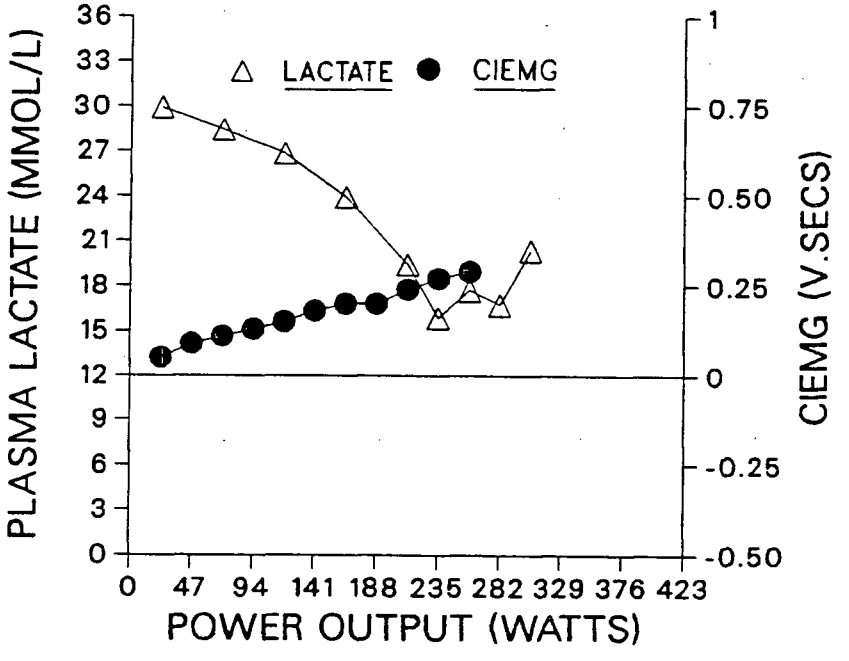


Figure 7 - Control Trial Subject 4

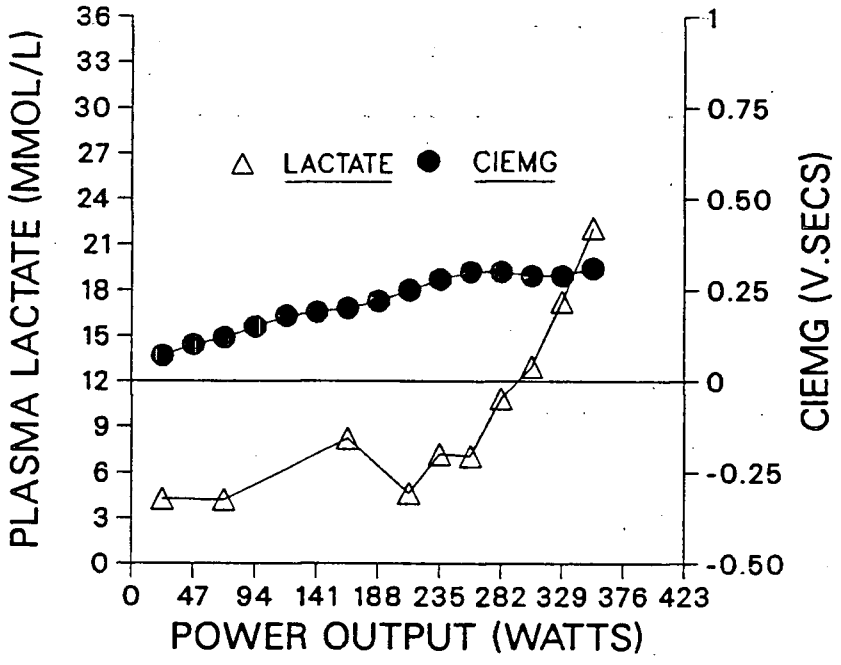


Figure 8 - Experimental Trial Subject 4

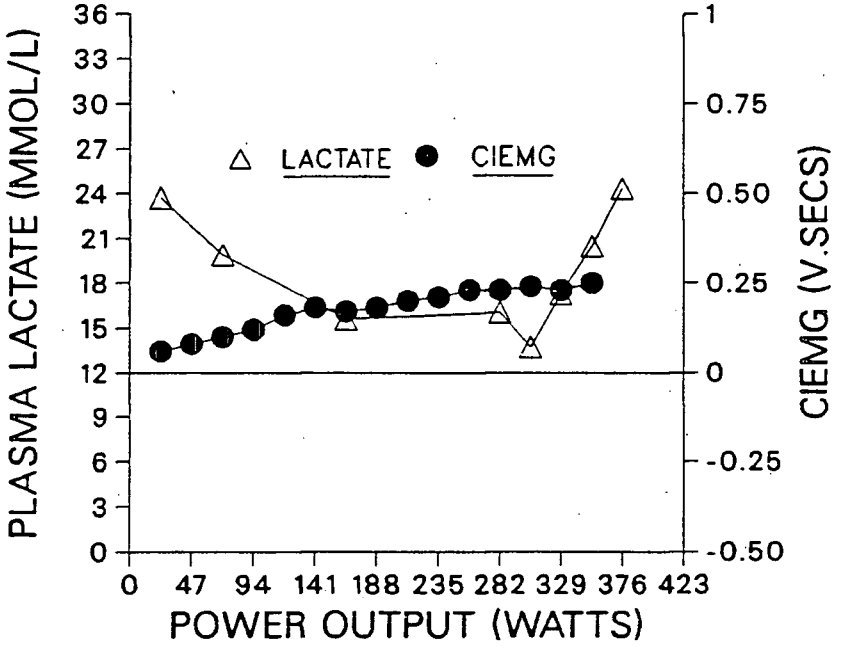


Figure 9 - Control Trial Subject:5

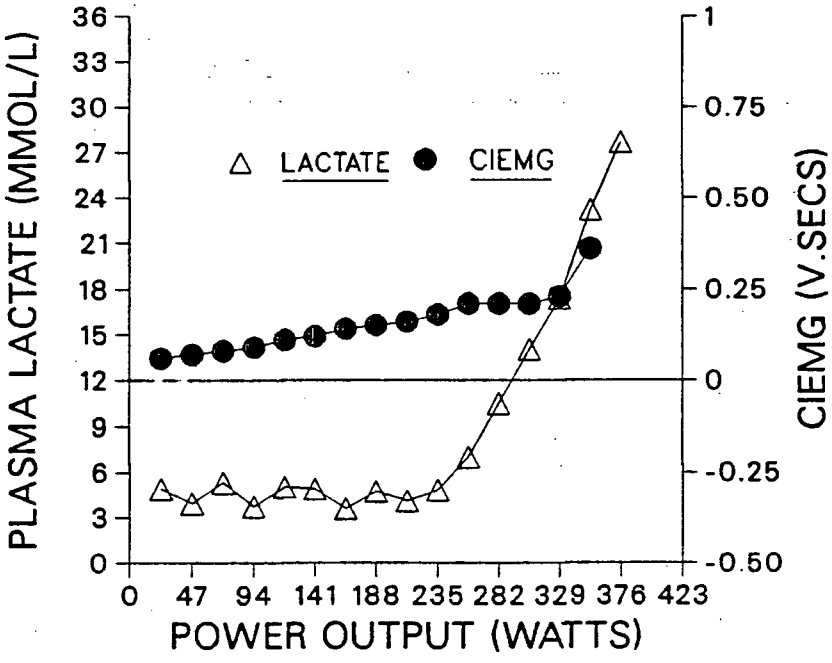


Figure 10 - Experimental Trial Subject 5

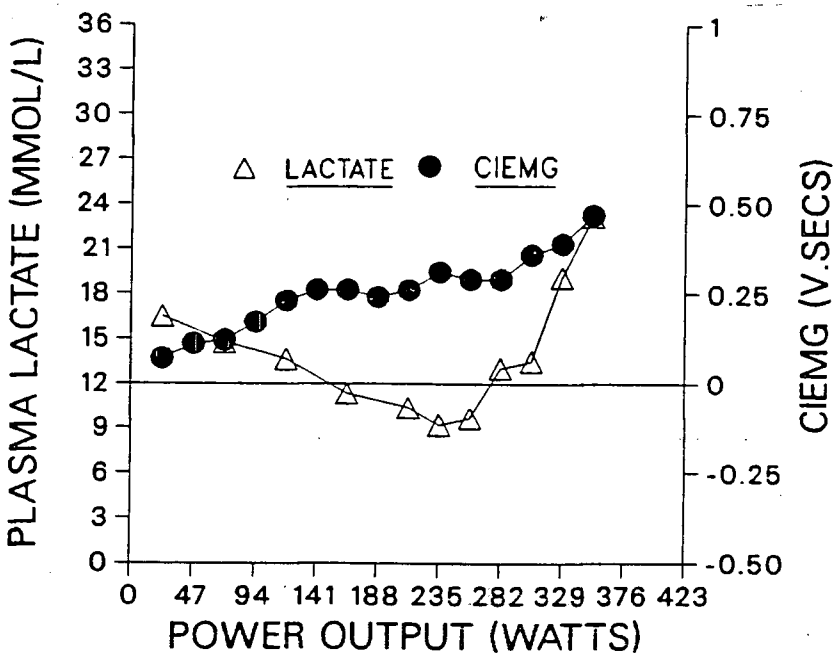


Figure 11 - Control Trial Subject.6

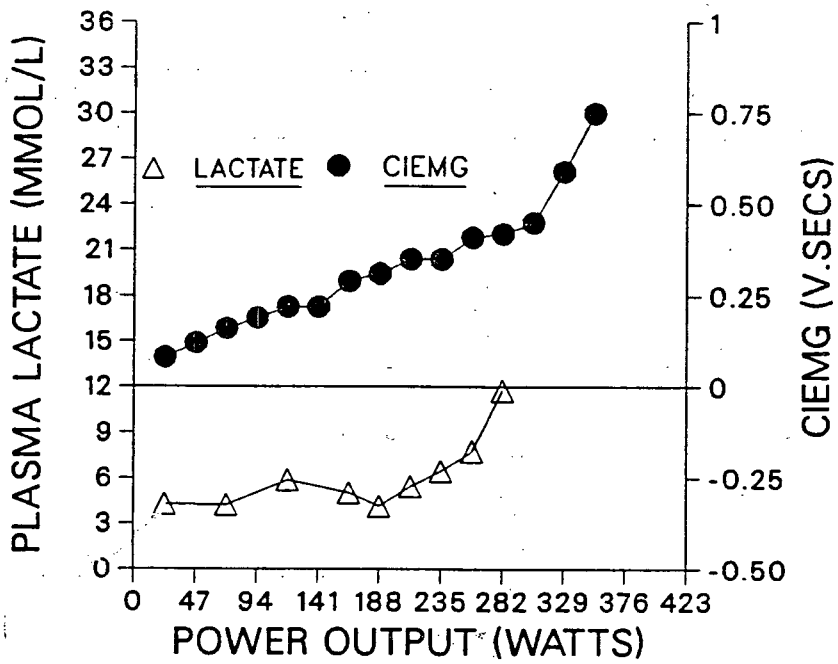
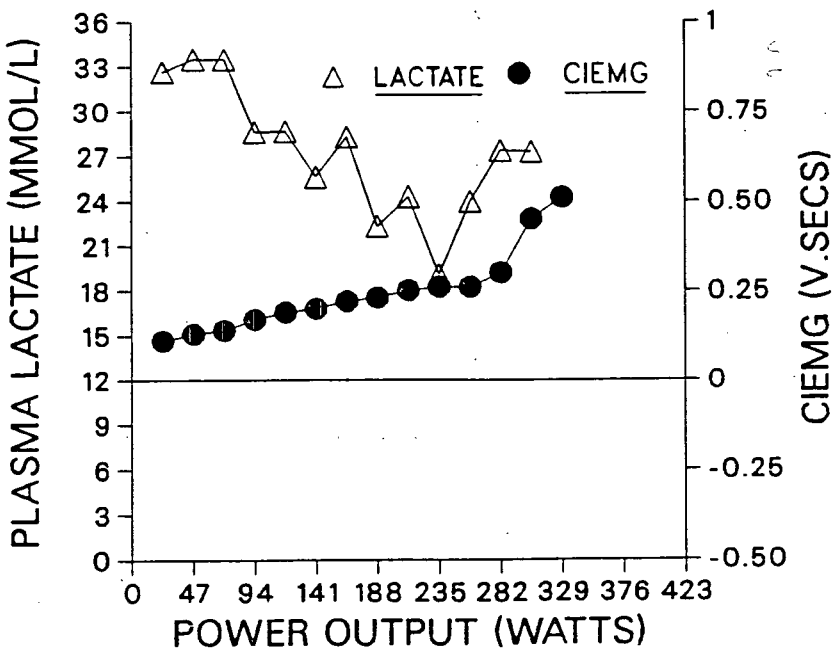


Figure 12 - Experimental Trial Subject 6



APPENDIX E - SELECTED DATA SUMMARY TABLES

TABLE 1

Age, Height, and Weight of Subjects

SUBJECT	AGE(YRS)	HEIGHT(CM)	WEIGHT(KG)
1	19	174.0	70.54
2	25	174.0	76.10
3	29	176.3	66.95
4	23	176.7	58.40
5	18	175.4	62.00
6	18	177.0	62.05
MEAN	22	175.6	66.01
RANGE	18-29	174.0 -176.7	62.00-76.10
S.D.	4.1	1.212	5.957

Table 2a

Selected Individual Maximum Cardiorespiratory Values of Control Trial

CARDIORESPIRATORY DATA SUMMARY MAXIMUM VALUES - CONTROL TRIAL							
SUBJECT	TEST DURATION (MINS)	MAXIMAL OXYGEN UPTAKE		$\dot{V}_E$	EXCESS CO_2	$\dot{V}_E/\dot{V}O_2$	R.Q.
		(L/MIN)	(ML/KG)	(L/MIN)	(L/MIN)	(L/MIN)	
1	16	4.14	54.95	148.10	27.26	37.58	1.19
2	19	3.34	43.28	142.45	25.05	37.97	1.20
3	15	2.73	40.79	125.80	26.56	46.08	1.35
4	16	2.84	49.10	131.70	27.79	46.38	1.26
5	15	3.27	52.84	131.80	31.90	40.36	1.30
6	15	2.59	41.91	121.20	24.46	46.75	1.28
MEAN	16	3.15	47.01	133.51	27.17	42.52	1.26
S.D.	1	0.52	5.62	9.22	2.41	3.98	0.06

Table 2b

Selected Individual Maximum Cardiorespiratory Values of Experimental Trial

CARDIORESPIRATORY DATA SUMMARY MAXIMUM VALUES - EXPERIMENTAL TRIAL							
SUBJECT	TEST DURATION (MINS)	MAXIMAL OXYGEN UPTAKE		$\dot{V}_E$	EXCESS CO_2	$\dot{V}_E/\dot{V}O_2$	R.Q.
		(L/MIN)	(ML/KG)	(L/MIN)	(L/MIN)	(L/MIN)	
1	15	3.16	44.77	112.10	23.36	35.51	1.22
2	18	4.41	57.91	158.69	29.39	36.01	1.21
3	14	2.46	36.81	110.10	23.34	46.01	1.28
4	15	3.01	52.15	129.60	28.01	40.53	1.27
5	15	3.59	57.90	144.50	23.03	40.24	1.10
6	13	3.09	49.78	110.30	23.83	35.69	1.18
MEAN	15	3.29	49.89	127.58	25.16	38.99	1.21
S.D.	1.5	0.61	7.43	18.67	2.55	3.77	0.06

Table 3
Anaerobic Threshold from Log-Log Plot of IEMG vs. VO_2

Subj. #	1	2	3	4	5	6	$\bar{X}$	S.D.
A.T. l/min	2.75	2.48	2.32	1.97	2.51	1.64	2.28	0.37
A.T. watts	235.5	235.5	188.0	188.0	211.5	164.5	203.9	26.2
% Maximum	66.4	74.3	85.1	69.4	76.8	63.4	72.6	7.2

Table 4a

Heart Rate Data - Control Trial

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1	105	101	120	126	120	127	127	138	137	154	160	169	174	178	182	185	
2	80	86	93	104	112	118	120	119	137	138	141	155	162	162	162	177	176
3	86	94	103	105	120	131	131	142	149	158	168	174	178	182	189		
4	63	77	89	88	98	107	121	128	143	149	163	170	178	185	185	187	
5	92	100	102	105	115	123	133	149	154	165	171	179	184	188	192		
6	100	112	100	121	121	140	142	158	166	172	180	185	189	194	196		
X	87.7	112	101	108	114	124	129	139	148	156	164	172	189	182	196	183	176
S.D.	13.8	11.2	9.79	12.4	7.97	10.3	7.51	12.9	10.2	10.9	12.0	9.35	8.44	10.0	11.0	4.32	0

Table 4b

Heart Rate Data - Experimental Trial

#	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17
1	112	115	117	121	124	130	137	143	150	158	161	166	172	176	180		
2	69	81	88	97	102	108	112	120	122	130	138	146	155	161	168	172	177
3	110	116	114	131	132	142	147	156	161	166	170	178	178	187	187		
4	120	125	129	140	140	152	156	163	165	174	178	182	185	187	187		
5	115	118	118	125	132	135	142	144	158	163	170	174	180	187	189	192	
6	129	131	134	138	150	153	163	170	178	185	194	196	199				
X	109	131	117	125	130	137	143	149	156	163	169	174	199	180		182	177
S.D	19.0	15.9	14.6	14.3	14.9	15.3	16.2	16.3	17.3	17.0	17.0	15.3	13.3	10.2	7.73	10	0

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