THE EFFECT OF CADENCE ON AEROBIC AND ANAEROBIC CONTRIBUTIONS TO THE TOTAL ENERGY REQUIREMENTS OF CYCLING AT CONSTANT POWER OUTPUT.

By

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ABSTRACT

Steady-state cycling at constant power output can be achieved at any one of a number of cadences. Data have been published (Coast and Welch, 1985) that suggest riding at a particular cadence which minimizes oxygen uptake ($\dot{V}O_2$) aids in achieving optimal performance. This cadence has been referred to as optimal. However, optimal cadences based solely on an indicator of aerobic metabolism (e.g. $\dot{V}O_2$) fail to recognize any contributions made by anaerobic metabolism to the total energy costs of performing the work. The anaerobic contribution becomes particularly important at power outputs greater than a rider's anaerobic threshold. This study was designed to evaluate the effect of cadence on a) the contributions of the aerobic and anaerobic energy pools to the total energy cost of cycling and b) the EMG activity of five major leg muscles at constant power outputs below and above the anaerobic threshold.

Male cyclists (n=4) completed progressive, incremental maximal exercise tests at cadences of 60 and 120 rpm to determine the ventilatory threshold (T_{vent}) for each cadence. Six minute steady-state rides at power outputs 20% below and 20% above T_{vent} were subsequently performed at both experimental cadences on separate days. $\dot{V}O_2$ and excess CO_2 data were collected throughout the steady-state rides and presented as the mean value over the final three minutes of each ride. EMG data were collected during the final 10 seconds of each six minute ride and averaged to represent one mean cycle of normalized EMG activity.

Both $\dot{V}O_2$ and excess CO_2 were found to be significantly greater ($p < 0.05$) at 120 rpm than at 60 rpm. However, when these data were corrected to account for the zero-load costs of cycling at each cadence, no significant differences were found in either variable. Only one of the five muscles studied (rectus femoris) exhibited significantly greater ($p < 0.05$) integrated activity at 60 rpm.

Based on the results of this study, it was concluded that cadence does play a significant role in the aerobic and anaerobic contributions to the total energy cost of

performing work at constant power output. It was found that simply moving the legs at a higher cadence significantly ($p < 0.05$) elevated the ventilatory response thereby resulting in significant ($p < 0.05$) increases in the responses at sub-T_{vent} and supra-T_{vent} power outputs. Cadence was also found to have a significant effect on the amount of activity in the rectus femoris, but not on any other muscle studied suggesting that there may be a shift in the metabolic profile of the active muscle fibers that cannot be measured by the EMG quantification methods used in this study.

It was concluded that the increases in steady-state $\dot{V}O_2$ and excess CO_2 observed with increases in cadence were primarily due to the increase in the energy requirements of the lower limb which is moving at a higher rate. The effect of load at each cadence was not a significant factor. The absolute amount of muscular activity did not significantly change between the two cadences suggesting that the mechanisms underlying muscle activation at different cadences may be more complicated and require measures more sensitive than surface EMG.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

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Chapter 1

INTRODUCTION

The fundamental objective for any competitive cyclist is to maximize performance. At constant power outputs, maximum cycling performance is most easily achieved by minimizing the metabolic and biomechanical costs that are associated with riding a bicycle at a particular power output. While riding on the road, a cyclist is able to maintain a constant power output at a number of different cadences by simply selecting an appropriate gear. Consequently, the problem facing the rider is selection of cadence. Data have been published (Coast and Welch, 1985; Böning et al., 1984) identifying the existence of an 'optimal' cadences for a wide variety of power outputs and suggest that riding at the optimal cadence will aid in maximizing performance. These and other investigations (e.g. Seabury et al., 1977) have determined physiologically-based optimal cadences for constant power outputs ranging from as low as 100 watts to as high as 300 watts. However, absolute values of the optimum vary due to the fact that testing procedures are not consistent and subjects used for these studies usually vary in ability. For example, Coast and Welch (1985) and Böning et al. (1984) both report optimal cadences ranging from 50 rpm to 80 rpm depending on the magnitude of the power output. Hagberg et al. (1981) report an optimal cadence of 91 rpm, although the subjects preferred cadences ranged from 72 to 102 rpm, while Seabury et al. (1977) report optimal cadences never exceeding 65 rpm at power outputs ranging from zero watts to 200 watts.

The most common physiological parameter used to determine an optimal cadence while cycling at a particular power output is oxygen uptake ($\dot{V}O_2$). Coast and Welch (1985) have reported a parabolic relationship between $\dot{V}O_2$ and cadence while cycling over a wide range of constant power outputs and have defined the optimal cadence as the one which elicited the minimum $\dot{V}O_2$. However, the absolute values of the optimal cadence reported by Coast and Welch (1985) and other investigators (e.g. Seabury et al., 1977; Böning et al., 1984) are consistently below the

preferred cadences of many competitive cyclists. Many competitive cyclists report that they prefer to ride at cadences between 90 and 110 rpm, while the optimal cadences reported in the literature range from 60 to 80 rpm. This suggests that for a particular power output, competitive cyclists ride at cadences above the optimum and as a result, needlessly elevate their $\dot{V}O_2$ and heart rate. Nevertheless, competitive cyclists continue to ride at cadences above the physiological optimum, possibly to take advantage of the fact that increasing cadence while cycling at a constant power output reduces pedal forces (Patterson and Moreno, 1990), thereby reducing the peripheral input to the sensation of effort.

Effort is a result of a complex combination of peripheral and central inputs. Peripheral inputs to the sensation of effort such as muscle tension have been reported to increase as pedal resistance increases (Cafarelli, 1977). At constant power outputs, riders experience increased pedal resistances as cadences are decreased. Therefore, by increasing cadence to 90 or 100 rpm, competitive riders perceive less effort peripherally due to the decreased muscle tension. Central inputs, such as metabolic demand also contribute to a rider's sense of effort. Muscular tension combines with the rate of contraction to drive the muscle's demand for fuel. The demand placed on the cardiorespiratory system to fuel the muscle is a central factor and when combined with peripheral factors result in an overall sense of effort (Figure 1).

Increases in effort are reflected in an increase in the amount of electrical activity recorded from the muscle. Surface electromyographic techniques provide a method of measuring the amount of electrical activity present in a muscle. Efforts to try to understand the relationship between neuromuscular activity and physiological parameters during dynamic contractions are limited. Bigland-Ritchie and Woods (1974) report a linear relationship between EMG activity and oxygen uptake during dynamic contractions performed on a cycle ergometer. Reimer et al. (1989) studied the EMG activity of the vastus lateralis while cycling at constant

power output (200 watts) to determine whether the amount of activity indicated a minimum level associated with a particular cadence similar to the physiological

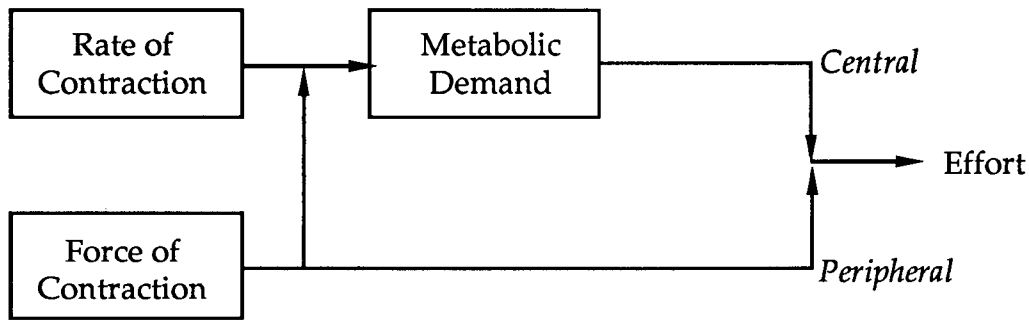

Figure 1. Schematic diagram of components that drive the sense of effort while cycling (Adapted from Cafarelli, 1977).

data reported by Coast and Welch (1985). Their results indicated no significant differences in either $\dot{V}O_2$ or integrated EMG activity at cadences ranging from 50 rpm to 100 rpm (see Appendix C). The results suggested that even though the force and rate of contraction varied, the metabolic demand did not change. In other words, the rider could choose to ride at any one of the experimental cadences and not risk any significant increases in $\dot{V}O_2$ or muscle activity.

Coast and Welch (1985) report oxygen uptake reductions accompanying increases in cadence, but only at cadences lower than the optimum. As cadence increased beyond the optimal, their data clearly demonstrated increases in oxygen uptake even though pedal resistances continued to decrease. This increase in oxygen uptake can be credited to the increased rate of contraction. In order to move the legs faster, the muscles need to contract quickly and therefore require more fuel per unit time. Circulation and respiration must increase to meet these demands and eventually the rider will fatigue when energy demands are not met. Therefore, increasing cadence to reduce peripheral fatigue does not come without its disadvantages. The degree to which the advantages gained by lowering pedal

resistances outweigh the disadvantages of increased $\dot{V}O_2$ at cadences above the optimum may play an important role in explaining why competitive riders select greater-than-optimal cadences.

Oxygen uptake is a variable that reflects a subject's capacity for deriving energy from an aerobic source. However, the aerobic energy system is only one of three energy delivery systems utilized during exercise and does not function in an isolated fashion. Rather, it operates in concert with two other systems, creatine-phosphate splitting and anaerobic glycolysis, to ensure that the supply of energy in the form of ATP is sufficient to meet the demands of exercise (MacDougall et al., 1982). It could therefore be argued that an aerobic measure such as oxygen uptake does not completely describe the effect of cadence on the total energy cost of cycling at constant power output. Instead, it only describes the aerobic contribution and does not take into account the anaerobic contribution to the total energy cost of exercise. This becomes particularly important during longer work bouts where the accumulation of anaerobic metabolism by-products could influence performance. The magnitude of the power output and the total time spent exercising must also be considered when evaluating the effects of cadence, especially when the magnitude of the power output is greater than the individual's anaerobic threshold. At power outputs higher than an individual's anaerobic threshold the contribution of the anaerobic energy pool to the total energy cost increases, thereby justifying the anaerobic energy system as an integral component in meeting the total energy requirements. To date, the effect of cadence on the contribution of the aerobic and the anaerobic energy pools to the total energy cost of cycling at constant power output has not been addressed.

Statement of the Problem

The total energy cost while cycling at constant power output is directly affected by the magnitude of the resistive forces, the rate at which the rider overcomes the resistive forces (i.e. cadence), and the total time spent exercising (i.e. volume of exercise). A complete description of the total energy cost of performing work at constant power outputs on a bicycle must identify both the central and the peripheral factors contributing to the effort. This description must not ignore the contributions of the anaerobic energy-yielding mechanisms nor the effects measured peripherally at the muscle level. The $\dot{V}O_2$ data reported by Coast and Welch (1985) is limited in that it only measures the aerobic contribution to the total energy cost of performing the work. The $\dot{V}O_2$ data reported in a number of studies (e.g. Coast and Welch 1985; Böning et al., 1984; Seabury et al., 1977) presents the contribution of the aerobic pool, but fails to quantify the contribution of the anaerobic pool. For example, $\dot{V}O_2$ data reported by Coast and Welch (1985) were measured at power outputs as high as 300 watts. Although not reported, this power output is clearly above most individuals' anaerobic threshold, yet the contribution of anaerobic energy-yielding systems were not reported.

Theoretically, the total energy cost of cycling at a particular power output is constant regardless of cadence, therefore a low $\dot{V}O_2$ does not necessarily mean that metabolic costs are minimized because the anaerobic component of energy production must increase to meet the total energy demands. This will lead to early fatigue while cycling at cadences which are assumed to be optimal, based solely on aerobic measures. To date, the effect of cadence on the contribution of aerobic and anaerobic energy pools while cycling at constant power output have not been addressed. Based on the results of Reimer et al. (1989), there also seems to be a disagreement as to whether the constant power output relationship between $\dot{V}O_2$ and cadence is parabolic or linear. Therefore, this study was designed to determine how cadence affected a) the contribution of the aerobic and the anaerobic energy

systems to the total energy cost and b) the amount of integrated muscle activity while cycling at constant power outputs above and below an individual's ventilatory threshold (T_{vent}).

Hypotheses

1. At a power output 20% below the power output at T_{vent}, it was hypothesized that there would be no significant difference between any of the dependent variables (Excess CO_2, $\dot{V}O_2$, and per-cycle integrated EMG) while cycling at cadences of 60 and 120 rpm.
2. At a power output 20% above the power output at T_{vent}, it was hypothesized that:
   a) $\dot{V}O_2$ would be significantly ($p < 0.05$) greater at a cadence of 120 rpm than at 60 rpm as shown by Coast and Welch (1985).
   b) Excess CO_2 would be greater at a cadence of 60 rpm than at 120 rpm, reflecting the additional contribution of anaerobic metabolism to maintain the overall energy cost of performing work.
   c) Per-cycle integrated EMG would be greater at 60 rpm reflecting the need for greater muscular activity to overcome the additional pedal forces.

Significance of the Study

This study addressed the importance of the anaerobic energy-yielding mechanism's contribution to the total energy cost of performing work. The role played by the anaerobic energy-yielding mechanism is especially important when the power output of the individual is greater than T_{vent}. The failure to recognize the contribution of the anaerobic component of energy production has led to an incomplete description of the effects cadence while cycling at constant power output. By addressing the role of the anaerobic component, this study represented all the contributing energy production systems and attempted to describe further the effects

of cadence while cycling at constant power outputs above and below an individual's anaerobic threshold.

Electromyographic data and $\dot{V}O_2$ data collected during this study served to confirm whether these variables were in fact greater at 120 rpm, supporting the relationship reported by Coast and Welch (1985) or whether they were not significantly ($p < 0.05$) different from each other (Reimer et al., 1989) at constant power outputs below and above an individual's T_{vent}.

Definitions of Terms

Anaerobic Threshold (AT). The power output just below that at which metabolic acidosis and the associated changes in gas exchange occur (Wasserman et al., 1973). The anaerobic threshold can be assessed invasively (blood lactate levels) or non-invasively (ventilatory parameters).

Per-Cycle Integrated Electromyogram (CIEMG). The integral (Simpson's Rule) of the root-meaned squared electromyographic (RMS EMG) signal linearly normalized to cycle time.

Progressive Incremental Maximal Exercise Protocol. A cycle ergometer protocol that elicits peak levels of exercise in subjects by progressively increasing the power output every three minutes while maintaining constant cadence.

Steady-State. A work situation where oxygen uptake equals the oxygen requirements of the tissue.

Sub T_{vent}. The power output 20% below the power output at T_{vent}.

Supra T_{vent}. The power output 20% above the power output at T_{vent}.

Top Dead Center (TDC). The position in the pedal cycle where the pedal is at the top of the cycle, oriented 90° to the horizontal.

Ventilatory Threshold (T_{vent}). The power output during the the progressive incremental exercise test where a non-linear increase in excess CO_2 occurred.

Delimitations

The muscles chosen for this study are not all of the muscles that contribute to moving the pedal around a normal pedal cycle. Muscles that were selected for this study however, represent all major muscle groups of the lower limb (i.e. hip, knee, and ankle flexors and extensors). Subjects were all well-trained, competitive cyclists with a minimum of three years racing experience. Results may have been affected by the fact that the subjects were participating as racers in different categories (skill levels).

Assumptions

Any changes in $\dot{V}O_2$ and excess CO_2 were assumed to be due to the demands of the active leg muscles alone. It will also be assumed that the EMG signal collected via surface electrodes is the signal from the desired muscle only and does not include any interfering signals from adjacent muscles.

Chapter 2

REVIEW OF LITERATURE

To provide the information necessary for a fundamental understanding of the physiological and neuromuscular aspects of cycling, this review will be divided into the following four sections: (a) energy requirements for exercise; (b) physiological response to exercise; (c) the effects of cadence and power output on exercise responses of cyclists; (d) neuromuscular response to exercise.

Energy Requirements for Exercise.

Energy for Muscular Contraction. During exercise, the principal consumers of metabolic energy from the breakdown of carbohydrates and fats are the skeletal muscles. The oxygen required for the breakdown of the carbohydrates and fats comes from the air that we breathe. The quantity of oxygen required during exercise depends on a number of factors including age, sex, level of fitness, but most importantly relies on the intensity of exercise. At rest, the rate of oxygen consumption is approximately 200 to 300 ml per minute; during maximal exercise, the rate can increase to as high as 5 - 6 L per minute (Fox, 1984).

The energy requirements of the contracting skeletal muscle are met by the delivery of the energy-rich molecule adenosine triphosphate (ATP). ATP is an immediate, usable form of energy that is formed through a series of chemical reactions that, in most cases, require oxygen.

If a muscle fiber is to sustain contractile activity, molecules of ATP must be supplied by metabolism as rapidly as they are broken down by the contractile process. Therefore, the muscle must have some way of continually forming molecules of ATP. There are three ways that a muscle fiber is able to form ATP: (1) phosphorylation of ADP by the splitting of the creatine phosphate molecule, (2) substrate phosphorylation of ADP, primarily by the glycolytic pathway, and (3) oxidative phosphorylation of ADP in the mitochondria. Phosphorylation by splitting the creatine phosphate molecule is rapid since it is a near equilibrium

reaction. It is localized to the energy consuming area in the muscle and is utilized during the first few seconds of activity. Although this system can produce ATP very quickly, it is limited by the relatively low concentrations of creatine phosphate present in the skeletal muscle. Substrate phosphorylation of ADP by the glycolytic pathway is primarily utilized in an anaerobic environment, thereby leaving the latter of the three methods, oxidative phosphorylation, to generate the majority of the ATP molecules during moderate, aerobic levels of exercise (Green, 1982).

Methods of ATP Regeneration. In order for muscular contraction to continue, chemical energy in the form of ATP must be made available in sufficient amounts. Because the muscle does not contain large amounts of stored ATP, mechanisms for ATP regeneration must exist. Green (1982) has summarized three distinct processes that are responsible for the regeneration of ATP.

i) Creatine Phosphate Splitting. The simplest and most rapid method of regenerating ATP involves the donation of a phosphate group from creatine phosphate (CP) to adenosine diphosphate (ADP) to form adenosine triphosphate (ATP) and creatine (C).

$$H^+ + CP + ADP \text{ <---(CK)---> } ATP + C \qquad (1)$$

The role of CP in ATP regeneration is particularly important in early stages of intense exercise where the depletion of stored ATP can be as high as 80%. Although the concentration of CP in the muscle is three to four times greater than ATP (Gollnick and Hermansen, 1973), the amount of CP in the muscle is still very small. Due to the low amounts of CP stored in the muscle, ATP regeneration by this process is limited. Because of this limited ability, other metabolic pathways must predominate if ATP levels are to remain normal. These other pathways include anaerobic (glycolytic) and aerobic (oxidative) metabolism.

ii) Anaerobic Glycolysis. In order for exercise to continue beyond a brief period of time, ATP must be regenerated quickly. Glycolysis involves the breakdown of carbohydrate to lactate in the absence of oxygen. Limited amounts of

ATP can be regenerated at high rates from this method, however muscular contraction cannot continue over an extended period of time due to the accumulation of lactate and H^+ ions. Lactate begins to accumulate at approximately 55-60% of an individual's maximum aerobic capacity. Accumulation has been attributed to the fact that the rate of formation of lactate exceeds the rate of its removal via oxidation in Krebs cycle metabolism or reconverted back to glucose (Brooks, 1985). The muscle cell can survive anaerobically as long as it can produce sufficient amounts of ATP and as long as the levels of lactate do not become excessive.

iii) Aerobic (Oxidative) Metabolism. Although anaerobic glycolysis can rapidly supply energy in the absence of oxygen, relatively little ATP is resynthesized in this fashion. Consequently, the majority of energy is provided by aerobic metabolism, especially if the exercise proceeds beyond two to three minutes. Aerobic metabolism begins with the formation of two moles of pyruvate (3C) from one mole of glucose (6C). The pyruvate is converted to acetyl coenzyme-A which in turn begins a cyclic metabolic pathway called the Krebs cycle. A large number of reduced coenzymes (nicotinamide adenine dinucleotide (NAD) and flavin adenine dinucleotide (FAD)) are generated, providing electrons necessary for the oxidative phosphorylation of molecules of adenosine diphosphate (ADP). The sources of carbohydrate for aerobic processes are present both in the muscle tissue as well as sources outside the muscle. Glycogen and free fatty acids stored in the muscle, glucose from the liver, and free fatty acids from adipose tissue all serve as glucose sources (Fox, 1990). In order for aerobic metabolism to contribute a significant number of ATP molecules, oxygen must be present.

ATP Regeneration During Exercise. During exercise, creatine phosphate splitting, anaerobic metabolism, and aerobic metabolism do not function in isolation of each other, but rather work in concert with each other to satisfy the energy requirements of the muscle. All three processes operate concurrently,

however the proportion of energy (ATP) delivered from each varies depending on the intensity and duration of the exercise (Green, 1982). Fox (1984) also recognizes this parallel function, referring to it as the "energy continuum concept".

During rest, most of the ATP in muscle tissue is regenerated by aerobic metabolism at a rate that matches its utilization. As exercise intensity increases, the need for ATP also increases. Immediately following an increase in exercise intensity, ATP regeneration via creatine phosphate splitting and anaerobic glycolysis occurs. As exercise continues aerobic metabolism gradually accelerates. If the energy demands of the muscle can be met by aerobic metabolism, the contributions of the other two processes are reduced, resulting in relatively stable levels of lactate, an indicator of anaerobic metabolism.

During prolonged types of low to moderate intensity exercise, the major source of ATP is the aerobic system. However, during high intensity exercise, the extent to which the aerobic processes can supply ATP to the muscle is far below its energy demands, therefore the anaerobic system becomes an extremely important source of ATP.

Physiological Response to Progressive Incremental Exercise.

Skinner and McLellan (1979) have identified three distinct phases during the progressive change from low intensity exercise to maximal exercise. Phase I primarily involves aerobic metabolism during low-intensity exercise and is characterized by linear increases in $\dot{V}O_2$, V_E , $\dot{V}CO_2$, and heart rate.

Phase II involves exercise intensities between approximately 40 % and 60 % $\dot{V}O_2$ max and is characterized by a nonlinear increase on V_E and VCO_2 plus a rise in blood lactate. During this stage, $\dot{V}O_2$ and heart rate continue to rise in a linear fashion however lactate concentration begins to increase to levels of about 2 mmol/l, approximately twice that of resting levels. Hydrogen ions (H^+) associated with the increased lactate concentrations are buffered by the bicarbonate buffering system (i.e., $H^+ + HCO_3^- <--> H_2CO_3 <--(CA)--> H_2O + CO_2$). The dissociation of

carbonic acid results in an increased production of CO_2, which consequently stimulates the respiratory center to increase $\dot{V}_E$. An increase in CO_2 production accompanied with higher $\dot{V}_E$ values result in increases in $\dot{V}CO_2$. The increase in $\dot{V}_E$ and $\dot{V}CO_2$ is greater than the increase VO_2 therefore increases in $\dot{V}_E/\dot{V}O_2$ and R are also observed. The onset of phase II corresponds to the anaerobic threshold reported by Wasserman et al. (1973).

Phase III includes exercise intensities of approximately 65 % to 90 % VO_2 max. $\dot{V}O_2$ and heart rate continue to rise linearly during this stage until near-maximal workloads are attained, at which time these variables begin to plateau. The onset of phase III is characterized by a sharp increase in blood lactate concentration resulting in a marked increase in $\dot{V}_E$ and $\dot{V}CO_2$ in an attempt to compensate for the increased lactate concentration. It has been noted that the anaerobic threshold reported by MacDougall (1978) and Green et al. (1983) appears to correspond to the onset of phase III.

Exercise Tests Used to Evaluate Oxygen Uptake. If an exercise test is to be centered on the analysis of the maximal oxygen transport function, large muscle groups must be engaged in order to best reflect the oxygen utilization of the whole body. The modes of exercise incorporated into laboratory testing procedures must therefore require the subject to use these large muscle groups. Nearly all oxygen transport research employs either treadmill running and bicycle riding as the form of exercise. Both have their advantages and disadvantages, however the cycle ergometer's advantages far outweigh its disadvantages. It's relatively inexpensive, portable, and most importantly the exercise loads can be expressed in standard units of work (kilogram-meters) and thus work comparisons can be made easily with the cycle ergometer (Astrand, 1986).

Increasing the power output of a cycle ergometer can be accomplished by increasing the resistance while maintaining a constant cadence. By increasing the power output at regular time intervals, a subject can eventually be brought to a

maximum level of physical exertion. When the increase in power output fails to elicit any further increase in oxygen consumption, the highest value attained represents the maximum oxygen consumption ($\dot{V}O_2max$). An incremental exercise test used for determining a subject's $\dot{V}O_2max$ has a number of advantages: 1) the test begins at relatively low work rates, therefore the subject is not required to exert a great amount of force or a sudden cardiorespiratory stress during the early stages of the exercise; 2) the $\dot{V}O_2max$ can be determined from a test lasting approximately 10-15 minutes in which the subject is stressed at high power outputs for only a few minutes; and 3) the $\dot{V}O_2$-power output relationship can be determined or estimated.

The $\dot{V}O_2$-work rate relationship describes how much oxygen is utilized by the exercising subject in relation to the quantity of external work performed. For mild to moderate levels of exercise, many investigators have determined this relationship to be linear. During aerobic exercise below the anaerobic threshold Wasserman and Whipp (1975) have provided evidence that suggests that the aerobic cost of cycle ergometry is approximately 10.0 to 10.5 ml $\cdot$ min^{-1} $\cdot$ watt^{-1}. Many other investigators have also supported this evidence by demonstrating a similar linear relationship between oxygen consumption and power output.

Because oxygen uptake kinetics become more complex than a simple linear relationship above the anaerobic threshold, the slope of the relationship may not necessarily be constant. If the work rate increment is large then a relatively large proportion of energy would be generated anaerobically and the slope would become more shallow. If the work rate increments are small, the test is prolonged, higher work rates are encountered, and a number of factors could affect the oxygen uptake. First, subjects often use additional muscles when performing heavy exercise. For example, a subject could begin to pull up on the handlebars which leads to unmeasured arm work. Second, at higher work rates breathing is increased non-linearly. As these high levels of breathing are reached, oxygen uptake by the

breathing muscle increases. Third, during heavy exercise there is significant lactate conversion to glycogen which requires oxygen uptake to increase in those organs in which this reaction is occurring (Wasserman et al., 1987).

The Anaerobic Threshold. Most of the ATP production used to provide energy during the early stages of a progressive incremental exercise test comes from aerobic sources (Skinner and McLellan, 1979). However, as exercise intensity increases, levels of lactate in the blood begin to rise. The power output at which blood lactate levels begin to rise significantly above normal resting levels during progressive incremental exercise has been termed the anaerobic threshold (AT) (Wasserman et al., 1973). Arguments over the terminology exist and the inflection point has also been identified as the lactate threshold (LT) as well as the onset of blood lactate accumulation (OBLA) (England et al., 1985; Mader and Heck, 1985; Jones and Ehrsam, 1982). The reason for the caution taken when the term anaerobic threshold is used lies in the meaning of the term 'anaerobic'. Some believe that anaerobic metabolism occurs because oxygen supply falls short of the demands of the muscle. Others feel that 'anaerobic' describes a metabolic pathway that does not require oxygen (glycolysis). It has also been proposed that rises in blood lactate concentrations are due to an increasing use of glycolytic pathways due to the recruitment of glycolytic muscle fibers. The formation of lactate may also be due to changes in the balance of regulatory enzymes resulting in the formation of pyruvate at a rate that is faster than it can be oxidized in the Krebs Cycle (Jones and Ehrsam, 1982).

Function and Assessment of the Anaerobic Threshold. The AT serves as an indicator of an athlete's ability to sustain exercise at a high percentage of their $\dot{V}O_2$ max. When exercise intensity exceeds this level, lactate begins to accumulate and endurance time is reduced due to a number of factors that accompany lactate accumulation such as an increased acidity in the muscle and a diminished capacity

to mobilize lipids which also corresponds to a diminished capacity to spare muscle glycogen (MacDougall, 1978).

Detecting the anaerobic threshold can be achieved a number of different ways. One method is to draw blood samples and measure the levels of lactate in the blood at the end of each workload during a progressive incremental maximal exercise test. This protocol is widely used, however there is no general consensus on the duration of the test or the rate of increase in power output. The shorter the duration of the test, the sharper the definition of the threshold (Wasserman et al., 1973). MacDougall (1978) is a critic of the short protocols based on the grounds that the measurements are too susceptible to the response variables of rapidly -increasing exercise. He advocates protocols including a duration of 4-6 minutes at each power output in order to obtain a relatively steady-state of oxygen uptake and blood lactate concentration. Protocols incrementing power output every 1-2 minutes serve as a reasonable compromise, however comparison of studies employing different testing protocols may be very difficult.

Because the appearance of lactate in the blood follows its production in the muscle, there is evidence suggesting that blood lactate concentrations do not provide an accurate estimate lactate concentrations in the muscle (Karlsson, 1971). Jordfeldt et al. (1978) published results suggesting the existence of a lactate concentration gradient between muscle and blood. They found significantly higher lactate concentrations in human muscle than in venous blood during exercise.

The concentration of lactate in the blood is dependent on the balance of production, utilization, and removal in the muscle, therefore a low blood lactate concentration could indicate either low production of lactate or a higher rate of production coupled with a higher rate of removal (Belcastro and Bonen, 1975). Unfortunately, blood lactate levels are not able to distinguish between these two possibilities.

Despite the popularity and wide acceptance of assessing the AT by an invasive technique (blood sample), changes in heart rate and a variety of ventilatory measures during progressive incremental exercise tests have also served as effective, non-invasive techniques for estimating the AT (Wasserman et al., 1973; Yoshida et al., 1981).

Relationship Between Blood Lactate Threshold and Ventilatory Threshold. During progressive incremental exercise tests, ventilation ($\dot{V}_E$) increases linearly with $\dot{V}O_2$, but this straight line gives way to a curve of increasing $\dot{V}_E$ to $\dot{V}O_2$ at high levels of exercise (Issekutz et al., 1965). It has been suggested (Wasserman and McElroy, 1964; Wasserman et al., 1973) that the onset of the anaerobic threshold can be detected using this and other ventilatory indices. They demonstrated that a disproportionate increase in ventilation during progressive exercise was associated with an increase in hydrogen ion concentration and P_{CO2} in the blood. Jones and Ehrsam (1982) report that in most situations the increased accumulation of lactate in blood plasma is accompanied by H^+, an increase in the expired volume of CO_2, and a simultaneous breakaway in ventilation. Wasserman et al. (1973) have proposed that the relationship between lactate accumulation and efflux of H^+ allows the ventilatory threshold to be used as a non-invasive indicator of the anaerobic threshold.

Some investigators (Caiozzo et al., 1982; Davis et al., 1976; Wasserman et al., 1973) have suggested that $\dot{V}_E/\dot{V}_{O2}$ offers the most sensitive measure of the lactate threshold, however others (Anderson and Rhodes, 1990) have proposed that a threshold based on excess CO_2 correlate most highly with the lactate threshold although the threshold did not represent the actual occurrence of the lactate threshold. In most situations accumulation of lactate in the blood is accompanied by the efflux of H^+ ions. The increase in CO_2 output is not caused by the increased levels of blood lactate, but rather the entry of H^+ ions into the blood according to the following equation (Jones and Ehrsam, 1982):

$$H^+ + HCO_3 <--> H_2CO_3 <--> CO_2 + H_2O \quad (2)$$
Bicarbonate Buffering System

Excess CO_2 in the lungs will appear in the time it takes the venous blood to reach the lungs. It is this association between blood lactate and H^+ ion production that allows the ventilatory threshold to be used.

Davis et al. (1976) tested the accuracy with which the ventilatory threshold predicted the lactate threshold in nine subjects and found an exceptionally close fit between the two methods (r = 0.95). Another close fit was also reported by Reinhard et al. (1979) in fifteen subjects.

The Effects of Cadence and Power Output on Exercise Responses of Cyclists.

Effects of Cadence. Many incremental exercise tests designed to measure an individual's $\dot{V}O_2$max on cycle ergometers have routinely been standardized with cadences of 50 or 60 rpm. The problem that is encountered when performing these tests at cadences of 50-60 rpm is the magnitude of the loads that accompany these lower cadences. In order to achieve adequate increments in power output at these cadences, loads are relatively high. These high resistive loads are perceived by the rider at the pedal and can often lead to premature leg fatigue resulting in a symptom-limited $\dot{V}O_2$max that doesn't reflect a true cardiovascular maximum. Differences in pedal speed have been found to affect oxygen uptake (Lollgen et al., 1980) as well as fatigue index (Jones et al., 1985). Because subjects perceive less exertion associated with higher cadences versus lower cadences while cycling at constant power output (Pandolf and Noble, 1973), it seems intuitive that if a progressive maximal exercise test were performed at a higher cadence, a higher $\dot{V}O_2$max could be attained (Moffat and Stamford, 1978).

To determine whether higher cadences could elicit higher $\dot{V}O_2$max's similar to those achieved on treadmills, McKay and Banister (1976) collected metabolic parameters from five male athletes during separate exhaustive tests performed at

four different cadences on a cycle ergometer. The results of this study showed that cadence did play a significant role in the determination $\dot{V}O_2max$. They found that cadences of 80 to 100 rpm produced maximum $\dot{V}O_2$ values, significantly higher than $\dot{V}O_2max$ values at 60 and 120 rpm, and suggested that although subjects preferred cadences of 60 to 80 rpm, cadences greater or equal to 80 rpm should be used in cycle ergometry to produce maximum cardiorespiratory function.

Although $\dot{V}O_2max$ is a relatively accurate measure of one's level of fitness, it is calculated from a test that increases power output incrementally to maximum. This type of test does not provide valuable information that can help answer the question of how best to perform at a constant work rate (constant power output), which best reflects the everyday riding conditions that most riders encounter.

The power output of the rider is the product of the force applied to overcome a resistance (i.e. pedal force) and the velocity at which the force is applied (i.e. cadence). Resistance while cycling can exist in several forms including air resistance, changes in terrain (e.g. hills), frictional resistance created between moving parts of the bicycle, and resistance at the tire-road interface. The cadence dictates the velocity at which the force is applied and can be adjusted while riding through judicious use of the gears on a bicycle.

During constant power output cycling on an ergometer, variations in cadence are accompanied by inverse changes in resistance. For a given constant power output, increasing the rider's cadence enables the rider to ride against lower resistances, thereby decreasing the force that must be applied to the pedal per revolution to overcome the resistance. This may seem a more advantageous strategy for the rider, however it costs the rider from an oxygen uptake perspective because he must move his lower limbs at an increased rate. Conversely, if the rider chooses to ride at a lower cadence, the resistance is increased to maintain the equivalent power output and consequently the pedal forces also increase. Because it becomes increasingly more difficult to pedal at lower and higher cadences, either

from a pedal force or an oxygen uptake perspective, an optimal cadence must exist between the two extremes where the effects of pedal force and oxygen uptake can be minimized. It is for this reason that information regarding the most efficient or optimal cadence is very important.

Laboratory studies using oxygen uptake as a measure of energy expenditure have indicated that while cycling on an ergometer at a constant power output the relationship between $\dot{V}O_2$ and cadence is best described by a parabolic function. The minimum of this parabolic relationship indicates the cadence at which the oxygen uptake is at its lowest level for that particular power output and has therefore been identified by some (Seabury et al., 1977; Böning et al., 1984; Coast and Welch, 1985) as the optimal cadence. These results propose that if a rider was going to choose to ride at a particular power output, it would be in that rider's best interest to ride at the optimal cadence in order to minimize energy expenditure.

Seabury et al. (1977) have offered a suggestion as to why the parabolic relationship exists while cycling at constant power output by likening the muscles of the body to a machine that is capable of a particular power output. They report that a muscle fiber has its upper limit of work that it can perform per contraction depending on the type of muscle fiber. As muscles contract slower and slower, therefore developing more tension with each contraction, more fibers are recruited to overcome the total load and the recruitment of the additional fibers generates heat. At cadences less than the optimal, more muscle fibers are recruited to overcome the load, thus heat production arising from the recruitment increases. This effect is further intensified at higher power outputs since more muscle fibers are involved and just like a machine, the heat limits the efficiency of the whole system (Seabury et al., 1977).

At cadences greater than the optimal, Seabury credits the internal viscous friction of the muscle tissue as the factor involved in decreasing the efficiency of the muscle. According to Guyton (1971), the viscous friction increases with speed of

contraction which may contribute to the decreased efficiency. Gaesser and Brooks (1975) have also suggested that at cadences greater than the optimal, more energetically inefficient types of muscle fibers are selectively recruited. At constant power output, the recruitment of less efficient muscle types is also associated with an increase in metabolic heat production. Just as before, the increase in heat production ultimately leads to a less efficient system.

Seabury et al. (1977) came to these conclusions based on a study that they conducted to observe the influence of cadence and power output on energy expenditure during cycle ergometry. They had three subjects ride for six minutes at all possible combinations of four different power outputs (0, 82, 164, & 196 watts) and eight different cadences (30, 40, 50, 60, 70, 80, 100, & 120 rpm) resulting in 32 experimental conditions. The results showed that energy expenditure ($kJ \cdot kg^{-1} \cdot min^{-1}$) varied with cadence at constant power output such that the results were best described by a parabolic function. This was true for all four power outputs including the unloaded condition (0 watts). The fact that energy expenditure increases with cadence in the unloaded condition provided evidence that simply moving the lower limbs faster to accomplish higher cadences increases the energy expenditure. Seabury et al. (1977) plotted the energy expenditure at the eight different cadences for all four power outputs on the same graph and it was clearly evident that the combinations of low cadence and high resistance greatly increases the energy expenditure for reasons explained above. Although Seabury et al. (1977) did not present the actual values for energy expenditure for the different conditions, they do provide the least squares equations of best fit. A moderate power output that is neither easy nor too difficult for most cyclists is 200 watts. Seabury et al.'s (1977) equation for the power output of 196 watts yields energy expenditures presented in Table 1.

Table 1: Effect of cadence on energy expenditure at 196 watts (Seabury et al., 1977).

Cadence (rpm)	Energy Expenditure (kJ/kg/min)
30	0.932
40	0.786
50	0.740
60	0.730
70	0.743
80	0.764
100	0.821
120	0.888

It can be clearly seen from Table 1 that the energy expenditures at 30 and 120 rpm were much greater than the energy expenditure at cadences ranging from 40 to 80 rpm. This tends to magnify the parabolic relationship. The cadences of 30 and 120 rpm are not frequently used in recreational cycling. In fact, cadences in the order of 120 rpm are only used by competitive cyclists in racing and training conditions. It would therefore seem wise to focus on the relationship over a range of cadences that is most frequently used by the majority of the cycling population (i.e. 40 - 80 rpm) (Figure 2).

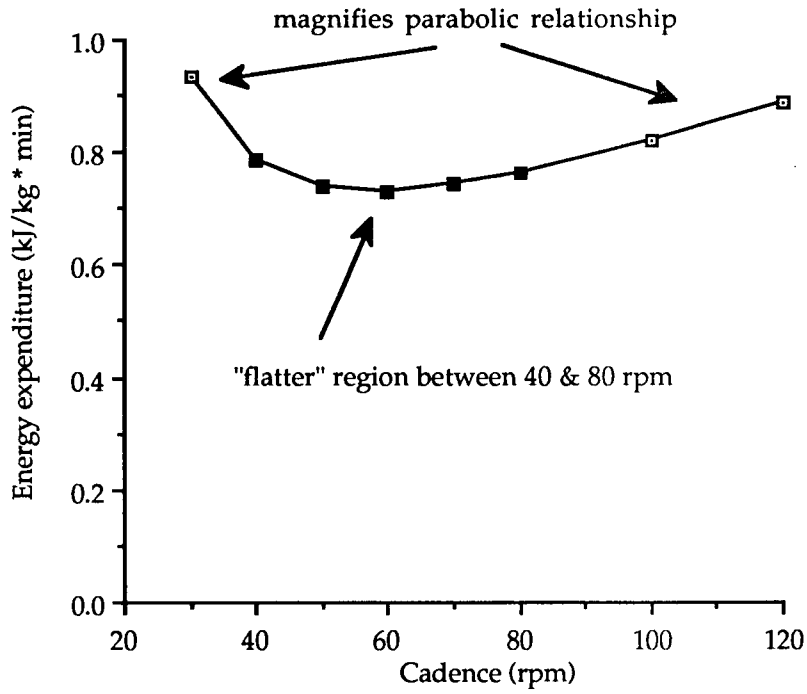

Figure 2. Energy expenditure vs. cadence at constant power output (196 watts) (Adapted from Seabury et al., 1977).

The graph shown in Figure 2 demonstrates that a "flatter" region existed over the cadences that are normally used by the majority of cyclists and how the lower and upper extremes of the cadence range tended to magnify the parabolic relationship. Based on the parabolic relationship over the full range of cadences, Seabury et al. (1977) found that the optimal cadence for cycling at 196 watts was 58 rpm. It's quite possible that if the calculation of optimal cadence was based on the data from cadences ranging from 40 to 80 rpm, an optimal cadence may not have been found due to the fact that there may be no significant difference between the energy expenditures. Therefore, the resulting graph would simply be a horizontal line indicating that while cycling at this particular power output (196 watts) an optimal cadence doesn't exist and the rider can simply choose a cadence that feels most comfortable. Reimer et al. (1989) showed a 'flat' relationship between steady-state

VO_2 and cadence as well as between integrated EMG activity of the vastus lateralis and cadence while cycling at cadences ranging from 50 to 100 rpm (see Appendix D).

Böning et al. (1984) have also studied the relationship between work load, pedal frequency, and physical fitness and have found a parabolic relationship between VO_2 and cadence at constant power output. Their value for an optimal cadence while cycling at 200 watts was approximately 65-70 rpm. They also took the work of Seabury et al. (1977) further by comparing the results from a group of well trained cyclists and a group of untrained students. Subjects from both groups cycled at four different power outputs (0, 50, 100, & 200 watts) at five different cadences (40, 60, 70, 80, & 100 rpm) resulting in 20 experimental conditions. Unlike the Seabury et al. (1977) study, the subjects in this study exercised for 15 minutes at each power output changing cadence every 3 minutes on a electronically braked ergometer. Böning et al. (1984) reported that the ergometer was one year old and only occasionally used which made the calibration error improbable. The calibration of the ergometer is always an important step that must not be overlooked for reasons already outlined earlier in this discussion (e.g. Landry et al., 1984).

The results showed that $\dot{V}O_2$ values for the untrained group were significantly higher than for the trained group and for each power output the relationship between $\dot{V}O_2$ and cadence was best approximated by quadratic regression. It was also noted that the effects of cadence on $\dot{V}O_2$ was reduced as the power output increased as demonstrated by a flattening of the regression curves. As was the case in the Seabury et al. (1977) study, the optimal cadence shifted from approximately 40 rpm at 50 watts to 70 rpm at 200 watts. The phenomenon of optimal cadence increasing with increases in power output was later confirmed in a study by Coast and Welch (1985) who found that the increase in optimal cadence with increases in power output was linear.

Coast and Welch had five subjects complete five progressive incremental maximal tests on a Monark cycle ergometer each at one of five cadences (40, 60, 80,

100, & 120 rpm). The work load was increased every three minutes until exhaustion. From the five progressive maximal tests, an oxygen cost of cycling was calculated for each rider at each cadence. The $\dot{V}O_2$ cost of riding at five different power outputs (100, 150, 200, 250, & 300 watts) were interpolated from the oxygen cost relationships. A curve of best fit was calculated for each power output and the first derivative was used to determine the optimal cadence.

The results showed that when $\dot{V}O_2$ was plotted against cadence at equivalent power outputs the curves were best described by a parabolic equation. Correlation coefficients for the curves varied between 0.86 and 0.99.

Steady-State versus Progressive Incremental Exercise. Both Böning et al. (1984) and Coast and Welch (1985) have arrived at conclusions about steady-state cycling at constant power output. However, both have derived their steady-state results based on progressive exercise procedures. In Böning et al.'s (1984) study, subjects rode at one power output, but randomly changed cadences five times (three minute intervals). In Coast and Welch's (1985) study the subjects' steady-state results were all derived from a progressive maximal test in which the power output changed every three minutes. In order for the results of these two studies to be valid, subjects must reach a steady-state in the three minute time period which in some cases may not be enough time.

Because steady-state and progressive rates of exercise are two different types of work, caution must be used when predicting one load from another. In the case of steady-state exercise, the rate of work doesn't change and the body eventually adjusts to the demands that are placed upon it. During progressive exercise the work rates are constantly changing and in the case of progressively maximal tests are constantly getting more difficult. The body only has a fixed period of time in which to reach a steady-state or equilibrium during progressive tests and in many cases this period of time is only three minutes. The validity of steady-state results derived

from progressive testing procedures is somewhat questionable and the issue must be addressed.

The research that argues the point that steady-state conclusions cannot be drawn from progressive incremental exercise protocols argues that a "steady-state" condition is not achieved within the time constraints used in many tests. Although Thompson (1977) found that the time constraints did not play a significant role in the calculation of a VO_2max, he also states in the same paper that a "steady-state" is notoriously difficult to obtain even for submaximal work. Subjects require varying periods of time to reach a steady-state level of stability and it may be that such a state is unobtainable. The oxygen consumption in a subject in steady-state is not constant and must be averaged over an appropriate time interval, and it is for this reason that Thompson (1977) has suggested that the term "steady-state" is unobtainable and may be a misnomer. In Thompson's (1977) study the maximum amount of time that a subject was required to exercise at a given power output was only two minutes. Perhaps a steady-state, in the true sense of the term is obtainable, but only during exercise protocols that incorporate longer time intervals.

Most progressive incremental maximal tests use time intervals of three minutes, however Astrand (1986) argues that steady-state conditions at submaximal work rates are not met in three minute work bouts. He recommends that steady-state conditions require an exercise period of at least five minutes. The progressive maximal exercise tests with three minute power output increments provides one with a quick and relatively accurate method to reveal a subject's peak oxygen uptake, but since steady-state conditions are not attained at changing work rates, this method does not provide reliable information to base steady-state conclusions on.

Fernandez et al. (1974) have also found that the oxygen uptake measured by a progressive maximal exercise method was not the same as that measured by a steady-state method. The study required five subjects who all had comparable $\dot{V}O_2$'s

to complete a progressive exercise protocol at 1 minute increments and a steady-state exercise protocol with 6 minute exercise bouts. These results were compared and then used to test the validity of a single exponential mathematical model. The results led to a conclusion that the oxygen consumption measured by the steady-state method was not the same as that measured by the progressive method for identical workloads. It was found that during the first three minutes of progressive exercise, the oxygen consumption may be greater than the steady-state oxygen consumption. Over the midrange of the work intensities, a constant difference between the progressive and the steady-state measurements was found.

Effects of Power Output. Based on the argument that at high power outputs exercise is not supported solely on atmospheric oxygen, but is also supplemented by ATP that is generated by anaerobic metabolism, Hansen et al. (1988) published a study testing a hypothesis that the $\dot{V}O_2$ responses to incremental work results in a nonlinear response and the pattern of the responses are dependent on the magnitude of the incremental work rate.

Their study included ten healthy men who performed six progressive maximal exercise tests at one of three different power output increments. The power output increments were 15, 30, and 60 watts $\cdot$ min^{-1}. All tests were performed at a cadence of 60 rpm. The results showed that for mild to moderate levels of exercise, the relationship between oxygen consumption and power output was linear with a slope of approximately 10.0 ml $\cdot$ min^{-1} $\cdot$ watt^{-1}.

For heavy or exhaustive work that is typical of the later portions of a progressive maximal exercise test, the same linear relationships did not exist. For the lower half (i.e. mild to moderate exercise levels) of the oxygen uptake vs. power output relationship, the slopes for the 15 and 30 watts $\cdot$ min^{-1} conditions were equal, however the slope for the 60 watts $\cdot$ min^{-1} condition was significantly lower from the other two conditions. Over the upper half of the relationship (i.e. heavy,

exhaustive levels of exercise), the slopes of all three conditions were significantly different from each other (Table 2).

Table 2. Mean slope (watts/min ± 1 SE) of upper and lower halves of $\dot{V}O_2$-power output relationship (Hansen et al., 1988).

Increment	Lower Half	Upper Half
15 watts/min	9.9 ± 0.21	12.45 ± 0.21 *
30 watts/min	9.9 ± 0.21	10.49 ± 0.15 *
60 watts/min	8.4 ± 0.25 •	8.70 ± 0.25 •

• sig. difference between power output increments
* sig. difference between lower and upper half increments

Hansen et al. (1988) offered three explanations as to why the slope of the upper half of the relationships were significantly different. First, the lower slope seen in the 60 watts · min^{-1} condition probably resulted from the major energy contribution of anaerobic glycolysis over a very short period of time. In other words, the majority of the muscle's need for ATP was met through the production of ATP during anaerobic glycolysis ultimately resulting in high state of oxygen deficit. Second, the fact that the slope of the upper half of the 15 watts · min^{-1} condition was significantly higher suggests a higher energy cost per watt of heavy exercise than per watt of mild exercise. Third, the near linear relationship in the 30 watts · min^{-1} condition was more than likely a result of a balance between the two extremes described above.

Regardless of the work rate increment used in the Hansen et al. (1988) study, the $\dot{V}O_2max$ was not significantly affected. This is also illustrated in the number of different protocols that are now in existence for determining maximal oxygen consumption, all reporting similar values. For example, the "Scandinavian" protocol increments the power 50 watts for men and 33 watts for women, each increment being maintained for 4 to 6 minutes (Jones and Campbell, 1982). However, increments of this size may mean that less fit subjects complete only two

or three increments. The solution for less fit subjects is to carry out an exercise protocol that has lower power increments. As a result, a number of studies have been done to determine if the power increments have a significant effect on the $\dot{V}O_2max$.

Davis et al. (1982) have completed one of the more complete studies observing the effects of power increment on a number of aerobic parameters. They had twelve males and two females perform four progressive maximum tests on a cycle ergometer to their limits of tolerance. Each of the four tests was characterized by a different power increment. The power increments were 20, 30, 50, and 100 watts $\cdot$ min^{-1}. Each of the four tests were performed at a cadence of 60 rpm. The results showed that the $\dot{V}O_2max$ values calculated in each of the four conditions were not significantly different. Maximal ventilations for the four progressive maximal tests were also not significantly different from each other, however the maximum heart rate in the 100 watts $\cdot$ min^{-1} condition was significantly lower than those found for the other three tests. This result was found to be somewhat obscure by the investigators due to the fact that the $\dot{V}O_2$ and the ventilation in the study were the same for all four conditions and that it had been found by Linnarsson (1974) that the heart rate dynamics are faster than $\dot{V}O_2$ and ventilation dynamics. If this was true, then the heart rate should have been greater than the other three conditions.

A possible explanation for the higher heart rate at the higher power increment has been described by Rowell (1974). Based on the data presented in Davis et al.'s (1982) paper, the durations of the progressive maximal tests varied (Table 4). Rowell describes that during steady-state exercise, the heart rate tends to increase slightly, possibly due to an increase in body temperature. According to Rowell, this "cardiovascular drift" may have occurred during the longer tests (ie 20 watts $\cdot$ min^{-1}), but not during the shorter test (100 watts $\cdot$ min^{-1}). This may have

been the case in this study due to the fact that the time spent exercising at 20 watts · min^{-1} was over 350% longer than that for the 100 watts · min^{-1} condition.

Table 3: Duration of incremental tests used by Davis et al. (1982).

Increment	Duration of Test
20 watts/min	12.25 min.
30 watts/min	8.60 min.
50 watts/min	5.36 min.
100 watts/min	3.36 min.

Thompson (1977) observed the time sequence in which the power increments are applied in a series of tests where eight subjects rode a cycle ergometer that was incremented to a power output of 120 watts. The first test was completed in five minutes with power output being incremented 15 watts every 30 seconds. The duration of the second test was six minutes with power output being incremented 30 watts every minute. The third test also had a power increment of 30 watts, however this test incremented the power output every 90 seconds. The fourth and final test incremented power output 30 watts every two minutes.

Regression lines for each of the four conditions were calculated. Correlation coefficients of no less than 0.98 were obtained. The $\dot{V}O_2$ values at a heart rate of 180 beats · min^{-1}, which Thompson claimed was very near the heart rate at $\dot{V}O_2max$, were extrapolated and no significant difference was found between any of the values. The one minute increment condition yielded the highest $\dot{V}O_2$ value and for this reason was used as the power increment of choice for the remainder of the study. Subjects for this study ranged in age from 18 to 42 years and although the extrapolated $\dot{V}O_2max$ values were low relative to other reported values, the results still support the linear relationship reported by Davis et al. (1982).

In contrast to the evidence that $\dot{V}O_2max$ is not affected by work rate increment, Buchfuhrer et al. (1983) completed a study that had subjects complete

progressive maximal exercise tests at three different work rates. The work rates were identical to the ones used by Hansen et al. (1988); 15, 30, and 60 watts · min^{-1}. Whereas Hansen et al. (1988) found no significant difference between VO_2max values at these three different work rate increments, Buchfuhrer et al. (1983) did. They demonstrated that with a one minute incremental test, $\dot{V}O_2max$ varied with the size of the increment. The highest $\dot{V}O_2max$ was achieved during the tests employing intermediate work rate increments (i.e., 30 watts · min^{-1}). They also concluded that subjects of different fitness seem to have similar "stress" from the tests of similar duration rather than of similar incremental size.

It appears that the main difference between the studies of Hansen et al. (1988) and Buchfuhrer et al. (1983) is the way in which the work rate increments were applied. In the Hansen et al. (1988) study work rate was constantly changing ultimately creating work rate increments of 15, 30, and 60 watts · min^{-1}. This type of loading protocol is called a "ramp" technique and is only possible with electronically braked cycle ergometers. It is felt that this method has the advantage of providing an infinitely gentle progression of work load and no sudden increments to disturb the subject's equilibrium (deVries, 1980). It did not specifically state that Buchfuhrer et al. (1983) used a ramp loading technique, therefore it can only be assumed that the work rate was incremented every minute even though an electronically braked ergometer was used.

Neuromuscular Response to Cycling Exercise.

The Surface EMG Signal. Basmajian (1978) defines the EMG signal as "... the electrical manifestation of the neuromuscular activation associated with a contracting muscle." The EMG signal is a very complex signal, affected by the type of muscle fiber studied, the electrical characteristics of the tissue surrounding the muscle, and the location of the electrodes with respect to the muscle. The EMG activity of a muscle has been used (a) to study the relationship between force, velocity and the amount of activity in human muscles (Bigland and Lippold, 1954), (b) as a measure of muscular force (Philipson and Larsson, 1988), (c) to correlate muscular activity with oxygen uptake (Bigland-Ritchie and Woods, 1974 & 1976) and (d) as a means of describing the timing and activity patterns of muscles during cycling (e.g. Mohr et al., 1981; Houtz and Fischer, 1959; Gregor et al., 1982).

Relationship Between EMG Activity, Force, and Oxygen Uptake. The relationship between oxygen uptake and exerted muscle force has been demonstrated by many physiologists to be linear. This relationship is the basis for many progressive incremental exercise protocols. However, during submaximal isometric contractions, the relationship between muscle tension and EMG is not as clear. Kuroda et al., (1970) found that although the relationship was linear for most submaximal isometric contractions, with tensions above 90% maximal voluntary contraction (MVC) the EMG rose exponentially.

During dynamic contractions, Bigland and Lippold (1954) found a linear relationship between the integrated EMG of the gastrocnemius muscle and force for submaximal isotonic contractions. Bigland-Ritchie and Woods (1974) carried this research further to include oxygen uptake and reported that the integrated EMG could be used as a relative measure of the combined number and stimulus frequency of active muscle fibers. They also proposed that under suitable precautions, the EMG measurements could be compared with the rate of oxygen

uptake to obtain information about changes in the energy expenditure per unit of muscle activity.

EMG During Cycling Exercise. Houtz and Fischer (1959) were one of the first groups to analyze the EMG patterns of a number of muscles in the lower limb while cycling. They studied 14 muscles in all and concluded that the muscles contracted in a pattern which was orderly and coordinated. The activity patterns of the muscles were highly reproducible within and between subjects. As the resistance was increased the activity patterns remained unchanged, however the electrical activity intensified in all muscles. They also identified the tensor fascia latae, sartorius, quadriceps femoris, and tibialis anterior as the primary muscles involved in cycling.

Mohr et al. (1981) have also studied the EMG of the lower limb muscles of competitive and noncompetitive cyclists while cycling with and without toe-clips as well as cycling in the seated and standing positions. The amplitude of the EMG signals increased with increasing workloads and no real differences in the timing of the muscle activity were found between the competitive and the noncompetitive cyclists.

The number of investigations studying the effects of cadence on the EMG patterns at constant power outputs are limited. Goto et al. (1975) monitored EMG activity from four muscles (vastus lateralis, gluteus maximus, gastrocnemius, and tibialis anterior) at different cadences and reported a linear relationship between cadence and the integrated EMG of the vastus lateralis and the gastrocnemius, while the gluteus maximus and the tibialis anterior displayed curvilinear relationships.

Chapter 3

PROCEDURES

Subjects.

The subjects for this study were four experienced male cyclists. Each rider completed an informed consent form. All riders were actively involved in racing and were familiar with the wide varieties of load and cadence combinations that were required for this study.

Progressive Incremental Maximal Exercise Protocol.

All subjects completed two maximal exercise tests on a Monark cycle ergometer. Each test was performed at one of two cadences (60 and 120 rpm). It has been shown that the rate of increase in power output affects the rate of increase in physiological measures such as $\dot{V}O_2$ and heart rate (Astrand, 1986 (pg. 302)). Therefore, in order to achieve the same responses that were recorded by Coast and Welch (1985), a three minute loading protocol was used in this study. Power output was increased 32 watts every three minutes. A three minute loading protocol was used in order to best achieve a balance between the desire to achieve steady-state at each load increment as well as preventing a prolonged exercise test. Prolonging the period of time spent at each increment would help insure steady-state conditions, however the test would become too long, introducing fatigue factors, resulting in a symptom limited $\dot{V}O_2max$. Power output was corrected by 9% to take into account the friction in the transmission, particularly the chain (Astrand, 1986). The test proceeded until the subject could not complete the full three minutes at a particular power output. The greatest $\dot{V}O_2$ value achieved during the final three minute work bout was considered as the peak $\dot{V}O_2$. Cadence was monitored and displayed on a liquid crystal display mounted on the handlebars and the subject was required to maintain the cadence within $\pm$ 2 rpm.

Sub-T_{vent} and Supra-T_{vent} Power Output Protocols.

All subjects performed a series of randomized six-minute submaximal steady-state rides at each of two cadences (60 and 120 rpm) at two different power outputs determined from the maximal exercise protocols. The power outputs were normalized to each individual's T_{vent}. The magnitude of the power outputs equalled the power outputs at $\pm$ 20 % of the T_{vent}. The two rides at 60 rpm and the two rides at 120 rpm were completed on separate days. All subjects pedalled for two minutes at a power output less than 100 watts which served as warm-up and an opportunity for the subjects to become accustomed to maintaining the required cadence. Following the warm-up, each subject completed the sub-T_{vent} ride and rested until heart rate fell to at least 80 beats per minute. Following the rest period the supra-T_{vent} ride was performed. Subjects were also required to maintain the cadence within $\pm$ 2 rpm during these tests.

Data Collection

Oxygen Uptake and Excess CO_2. Beckman gas analyzers were used to measure the gas fractions in the expired air collected via conventional open-circuit spirometry. Oxygen uptake ($\dot{V}O_2$) and excess CO_2 samples were collected every 15 seconds and averaged across each minute during both the incremental maximal tests as well as the steady-state rides. The $\dot{V}O_2$ provided a measure of the aerobic contribution to the total energy cost of incremental and steady-state cycling. Excess CO_2 values offered an indirect measure of the anaerobic contribution to the total energy cost of performing the work during steady-state and incremental cycling.

EMG. The root-mean-square (RMS) EMG activity was collected from the rectus femoris (RF), vastus lateralis (VL), biceps femoris (BF), tibialis anterior (TA), and medial gastrocnemius (MG). The RF represents the hip flexor/knee extensor group, the VL represents the knee extensor group, the BF represents the hip extensor/knee flexor group, the TA represents the dorsiflexor group, and the MG represents the plantarflexor/knee flexor group. All five muscles are superficial

muscles which will ensure accurate EMG collection. The site of electrode application followed methods reported by Delagi and Perotto (1980). The skin above the muscle was thoroughly cleaned and coated with a thin layer of electrode jelly before the electrodes were applied to aid in better conductance.

A Therapeutics Unlimited, Inc.© Electromyograph System Model 544 was used to record surface EMG signals from the muscles. Silver-silver chloride electrodes combined with a solid state preamplifier potted in a plastic enclosure was fixed to the skin above each muscle. The gain of each preamplifier was 35 ± 10% and each was supplied with isolated ± 2.5 V DC regulated power. The preamplified myoelectric signals were amplified within ± 10 volts by the EMG-544 Amplifier/Processor modules supplied with the EMG recording system. The amplified signal was high pass filtered at a cutoff frequency of 20 Hz. The low-pass averaging time constant of the root mean squared output was 2.5 msec.

The EMG recording was triggered by a 5 volt pulse indicating top dead center (TDC). The RMS signals were sampled through a 12-bit analog-to-digital converter at a rate of 900 Hz (180 Hz per muscle) during the last 10 seconds of the last minute of the sub-T_{vent} and supra-T_{vent} tests.

Data Analysis

Normalization and Integration of EMG Data. One of the most common methods used to evaluate an EMG signal is integration. The correct interpretation of integration is purely mathematical, and means "area under the curve." After the raw signal has been filtered, rectified, and normalized, integration can be performed. All RMS EMG data were normalized and averaged to one cycle (i.e. from TDC to TDC). The integration of the RMS signal was performed using Simpson's Rule and presented as a per cycle, averaged, normalized integrated EMG (CIEMG) value. The raw RMS EMG data were normalized linearly in time in order to obtain information regarding the timing of the muscular activity.

$$\text{Area} = (S/3) \times [(F + L) + 4(\Sigma E) + 2(\Sigma O)] \quad (3)$$

where S = time interval between ordinates.
F = value of first ordinate
L = value of last ordinate
ΣE = sum of even ordinates
ΣO = sum of odd ordinates

All EMG data, regardless of cadence, were time-normalized to one pedal cycle. Each pedal cycle was represented by 20 ordinate values therefore, the relative time interval between ordinate values was 5% of the total pedal cycle. Normalizing EMG data to obtain a consistent time base allowed the comparison of EMG timing patterns across cadences because the relative time bases were the equivalent. Without normalization procedures this comparison could not have been performed because the time spent during a pedal cycle at 60 rpm was twice that at 120 rpm. The difference in the real time spent during a pedal cycle at the two cadences results in two different S values in the Simpson's Rule formula. Consequently, the area under two similar EMG curves collected at the 60 and 120 rpm is very different due to the different time intervals between ordinate values. This effect is graphically represented in Figure 4. Figure 3 represents actual data collected from the rectus femoris. The two curves in Figure 3a and Figure 3b are very similar however when integrated result in two very different values, primarily due to the different time bases present at the two different cadences. Therefore, graphical data that appears to be similar upon visual inspection, does not correspond to similar integrated values. This must always be considered when analyzing and comparing EMG data in this fashion. Just as two similar curves can yield two different IEMG values, two very different curves can also yield two similar integrated values for the same time-base reasons explained above. This effect is graphically described in Figure 4.

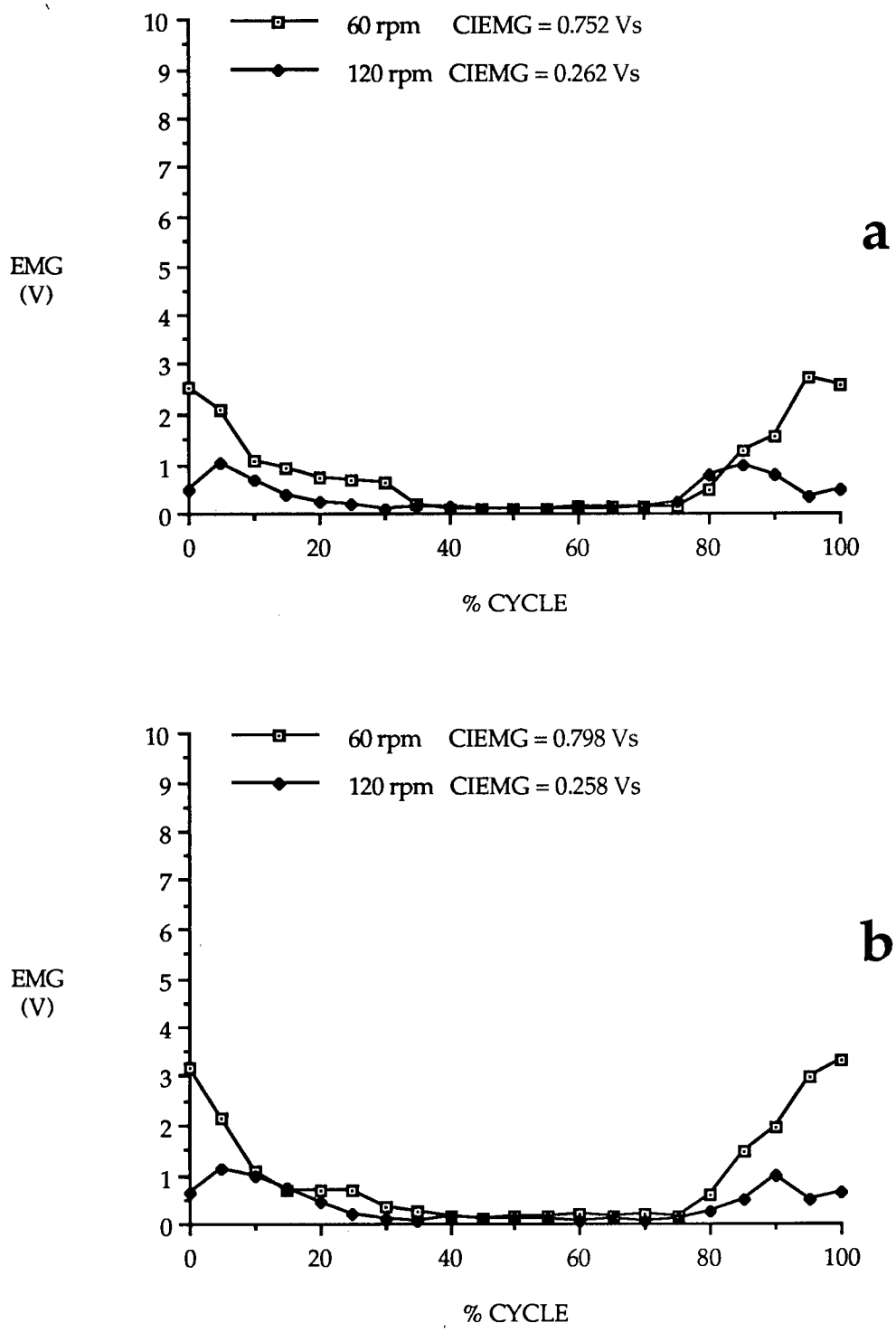

Figure 3. Mean CIEMG recorded from the rectus femoris at (a) sub-T_{vent} and (b) supra-T_{vent} power outputs.

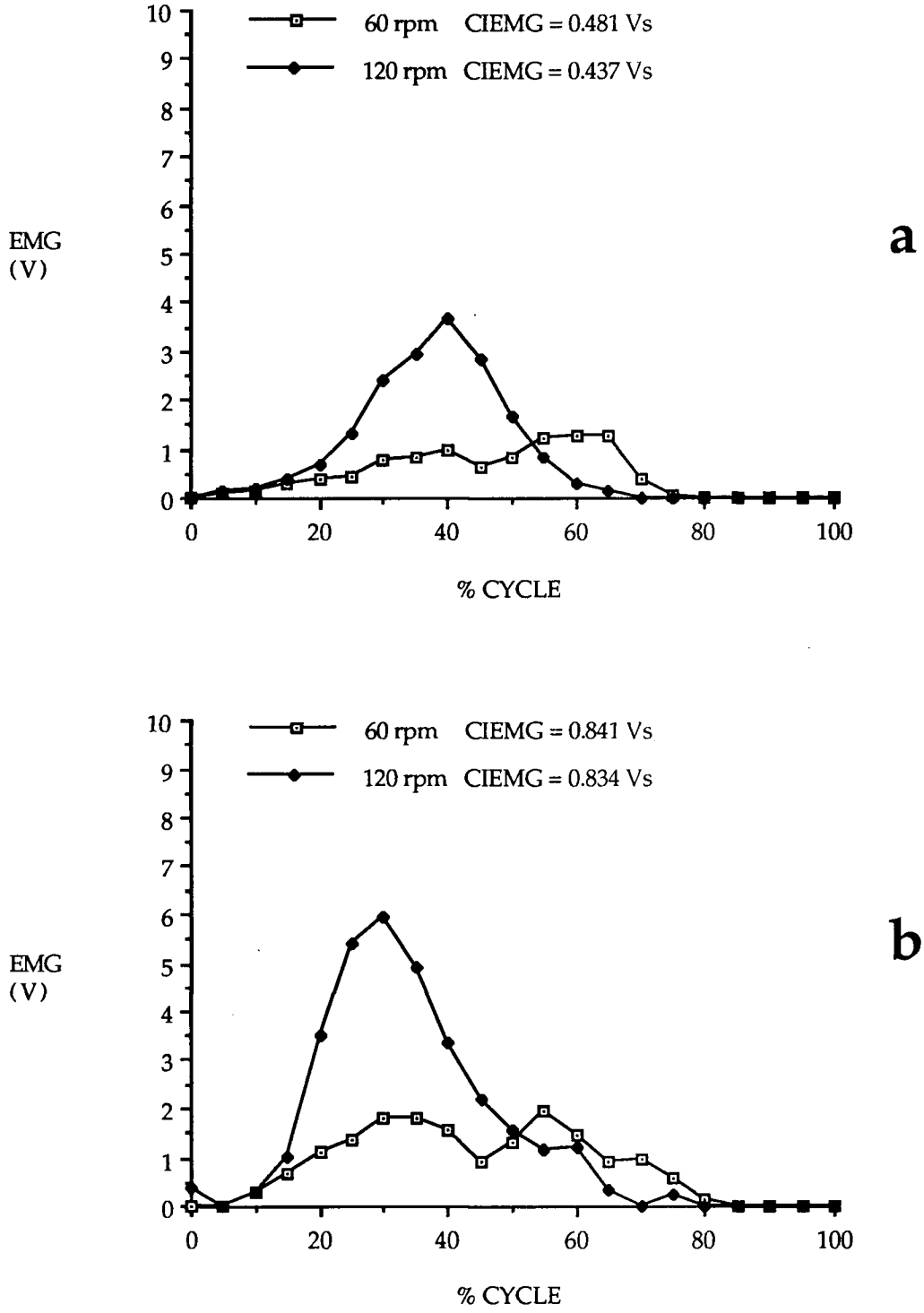

Figure 4. Mean CIEMG recorded from the (a) biceps femoris and (b) medial gastrocnemius at sub-Tvent power outputs.

Calculation of Power Output at Ventilatory Threshold. The ventilatory threshold (T_{vent}) was determined by using a computer algorithm based on an algorithm by Orr et al. (1982) that modelled the excess CO_2 data using a pair of simple linear regressions. All possible data combinations were fitted to this model and the combination that resulted in the smallest sum of residual squares was retained. The point of intersection between the two linear regressions was identified as the T_{vent}. The power output at T_{vent} was determined and following this determination, power outputs 20% below and 20% above the power output at T_{vent} were calculated.

Steady-State Ventilatory Data. Steady-state $\dot{V}O_2$ and excess CO_2 values were calculated as the mean value for the last three minutes of the six minute steady-state ride. The first three minutes of the six minute test allowed the riders to reach steady-state and ensured that the mean of the final three minutes would represent a steady-state average value.

Correction of Ventilatory Data. Oxygen uptake and excess CO_2 data were corrected to account for the difference in rates of muscular contraction and energy production due exclusively to cadence. In the case of the oxygen uptake data collected during the incremental maximal test, a linear regression was fitted to the data to determine the oxygen uptake at a power output of zero watts. An exponential curve was fitted to the excess CO_2 data collected from the same trials order to determine the excess CO_2 at a power output of zero watts. The value for the zero-load cycling was subtracted from all subsequent corresponding ventilatory values and the results were referred to as either corrected $\dot{V}O_2$ or corrected excess CO_2 data.

Statistical Analysis. A t-test for correlated samples was used to determine if peak $\dot{V}O_2$, peak excess CO_2, steady-state $\dot{V}O_2$, steady-state excess CO_2, or steady-state CIEMG were affected by cadence. The level of significance for all tests was $p < 0.05$.

Chapter 3

RESULTS

Description of Subjects.

Four trained male cyclists served as subjects for this study. The subjects' descriptive data is included in Appendix A. The mean age, height, and weight were 21 ± 3.2 years, 173.7 ± 9.1 cm., and 75.4 ± 7.1 kg. respectively.

Analysis of EMG Data

The per-cycle integrated EMG (CIEMG) value is directly affected by the magnitude of the pedalling resistance as well as the pedalling velocity or cadence. Holding power output constant at the sub-T_{vent} power output and varying cadence resulted in CIEMG activity that was greater at 60 rpm than at 120 rpm. The only muscle of the five muscles studied that displayed significantly greater CIEMG activity at 60 rpm was the rectus femoris ($t=3.455$, $p=0.041$). Although not statistically significant, the same trend was evident in the other four muscles. At the supra-T_{vent} power output, the rectus femoris was also the only muscle that showed significantly greater CIEMG at 60 rpm ($t=6.32$, $p=0.008$). All other muscles showed greater CIEMG at 60 rpm, however the differences were not statistically significant (Table 4).

Before interpreting these data, it is important to note that the real time base differs between the two curves on each graph. Each curve represents one pedal cycle of muscle activity, however one pedal cycle at 60 rpm takes twice as much time to complete than a pedal cycle at 120 rpm. Therefore in relative terms the graphs represent the same amount of time (i.e. one pedal cycle), but there is a profound difference in real time. In an attempt to account for this difference, all CIEMG values collected at 120 rpm were also doubled to represent the amount of activity during the same amount of absolute time and listed inTable 5. Statistical analysis of

these data also resulted in no significant differences between the CIEMG at the two cadences at both sub-T_{vent} and supra-T_{vent} power outputs.

Table 4. Mean per-cycle integrated EMG (CIEMG) (Vs).

MUSCLE	60 rpm (<T_{vent})	60 rpm (>T_{vent})	120 rpm (<T_{vent})	120 rpm (>T_{vent})
B. Femoris	0.481	0.944	0.437	0.385
M. Gastroc.	0.841	1.038	0.834	0.599
R. Femoris	0.752	0.798	0.262	0.258
T. Anterior	0.491	0.556	0.334	0.173
V. Lateralis	1.024	1.210	0.562	0.853

Table 5. Mean time-adjusted per-cycle integrated EMG (CIEMG) (Vs).

MUSCLE	60 rpm (<T_{vent})	60 rpm (>T_{vent})	120 rpm (<T_{vent})	120 rpm (>T_{vent})
B. Femoris	0.962	1.888	1.748	1.540
M. Gastroc.	1.682	2.076	3.336	2.396
R. Femoris	1.504	1.596	1.048	1.032
T. Anterior	0.982	1.112	1.336	0.692
V. Lateralis	2.048	2.420	2.248	3.412

Although the differences between the CIEMG at the two cadences at both power outputs were not significant, the timing of the activity did change such that the position in the pedal cycle where the majority of muscular activity occurred were different depending of the cadence. As cadence increased, the peak muscular activity occurred earlier in the pedal cycle. The biceps femoris and the medial gastrocnemius muscles were the best examples of this timing change (Table 6).

The effect of power output on the CIEMG and the timing of the CIEMG was less prominent than the effect of cadence. The CIEMG profiles for all five muscles demonstrated only subtle differences (Table 6).

Table 6. Position in pedal cycle where peak EMG activity occurred.

MUSCLE	60 rpm ($<T_{vent}$)	60 rpm ($>T_{vent}$)	120 rpm ($<T_{vent}$)	120 rpm ($>T_{vent}$)
B. Femoris	60%	60%	40%	40%
M. Gastroc.	55%	55%	30%	30%
R. Femoris	95%	100%	85%	90%
T. Anterior	90%	90%	85%	85%
V. Lateralis	100%	100%	10%	5%

Analysis of $\dot{V}O_2$ Data.

Figure 6 shows the mean uncorrected and corrected $\dot{V}O_2$ response to the progressive incremental exercise protocol at the two experimental cadences. Error bars represent ± one standard deviation. The corrected $\dot{V}O_2$ data was obtained by subtracting the correction factor from each of the data points. The correction factor was calculated by determining the y-intercept from the least-squares line of best fit for each set of data. The mean uncorrected peak $\dot{V}O_2$ values (60 rpm = 3.71 l/min, 120 rpm = 3.83 l/min) were not significantly different between the two cadences ($t = 0.866$, $p = 0.420$), however when the corrected peak $\dot{V}O_2$ values (60 rpm = 3.51 l/min, 120 rpm = 2.80 l/min) were compared the difference was significant ($t = 3.900$, $p = 0.008$).

The correction of the $\dot{V}O_2$ data also resulted in a reversal in relative magnitude. In the uncorrected $\dot{V}O_2$ data, 120 rpm consistently elicited higher $\dot{V}O_2$ values than 60 rpm. When the data were corrected, 60 rpm elicited higher $\dot{V}O_2$ values than 120 rpm due to the larger correction factor at 120 rpm. The mean

correction value at 60 rpm (0.20 l/min) was significantly lower than the correction value at 120 rpm (1.02 l/min) ($t = 5.565$, $p = 0.001$).

Analysis of Excess CO_2 Data.

Figure 7 shows the values of the mean corrected and uncorrected excess CO_2 during the progressive incremental exercise protocol at the two experimental cadences. Error bars represent ± one standard deviation. The corrected excess CO_2 data were obtained by a method similar to the one used for the VO_2 data except the line of best fit did not follow a linear model. Rather, the data was fitted to an exponential function and the y-intercept was determined. The mean uncorrected peak excess CO_2 values (60 rpm = 19.74 ml/kg/min, 120 rpm = 18.84 ml/kg/min) were not significantly different between the two cadences ($t = 0.538$, $p = 0.610$). When the corrected peak excess CO_2 values (60 rpm = 18.44 ml/kg/min, 120 rpm = 14.96 ml/kg/min) were compared the difference remained non-significant ($t = 2.345$, $p = 0.057$).

The mean correction value at 60 rpm (1.29 ml/kg/min) differed significantly from the correction value at 120 rpm (3.88 ml/kg/min) ($t = 6.432$, $p = 0.001$). The large difference between the two correction values however, did not result in a relative reversal in magnitude in the incremental data as was the case in the VO_2 data. Instead, correcting the data brought the two lines closer together, essentially describing the same response to the incremental protocol regardless of cadence.

Analysis of Steady-State Data.

Figure 8 summarizes the effects of cadence on steady-state $\dot{V}O_2$ and excess CO_2 while riding at a sub-T_{vent} and supra-T_{vent} power outputs. Each bar represents the mean $\dot{V}O_2$ and excess CO_2 calculated over the final three minutes of the six minute steady-state ride. Analysis of these data revealed that cadence did have a significant effect on both the uncorrected steady-state $\dot{V}O_2$ and the uncorrected steady-state excess CO_2. In all four cases, 120 rpm elicited a greater uncorrected steady-state value

than 60 rpm. Applying the correction factor to these data led to some interesting results. In all four cases, correcting the data resulted in non-significant differences. Correcting the steady-state VO_2 data also led to a slight relative magnitude reversal similar to the reversal found during the progressive incremental tests. Correcting the steady-state excess CO_2 data did not lead to a relative magnitude reversal. This reflected the progressive incremental test results however, where the correction factor resulted in similar excess CO_2 values from the two cadences during the incremental protocol, the corrected steady-state excess CO_2 at 60 rpm was still less than its counterpart at 120 rpm.

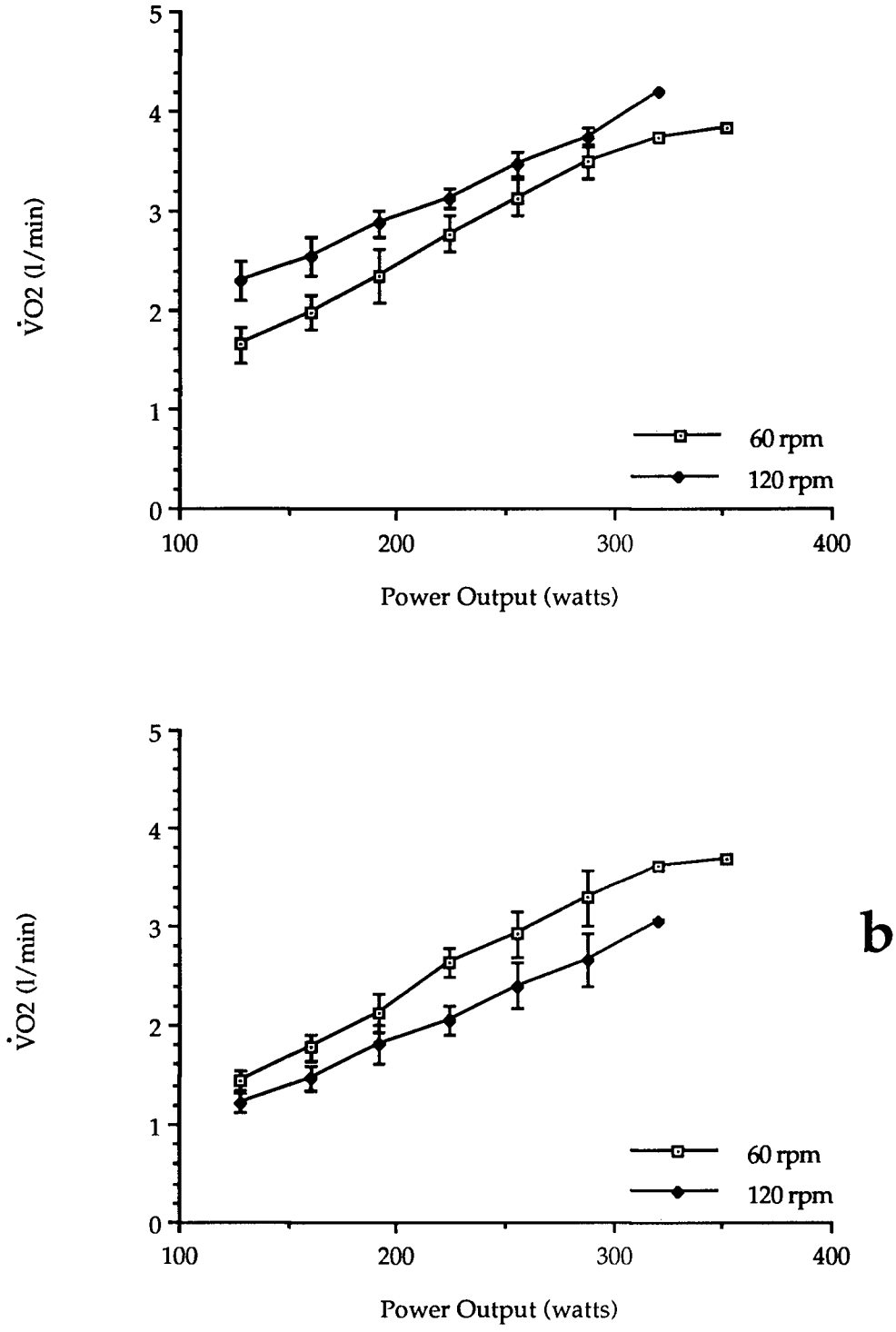

Figure 5. (a) Uncorrected and (b) corrected mean $\dot{V}O_2$ (±1 SD) response during progressive incremental maximal exercise protocol.

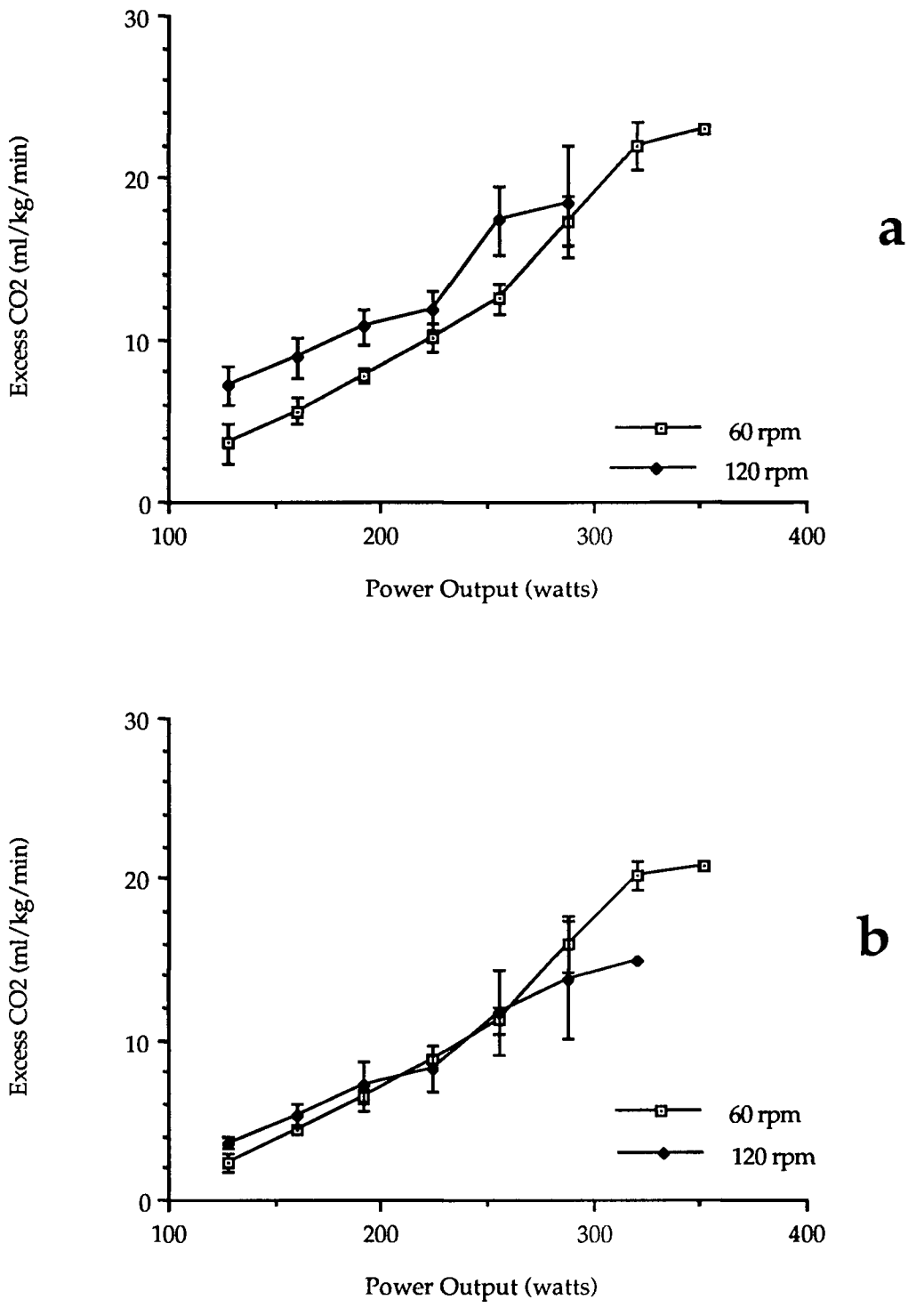

Figure 6. (a) Uncorrected and (b) corrected mean excess CO_2 (±1 SD) response during progressive incremental maximal exercise protocol.

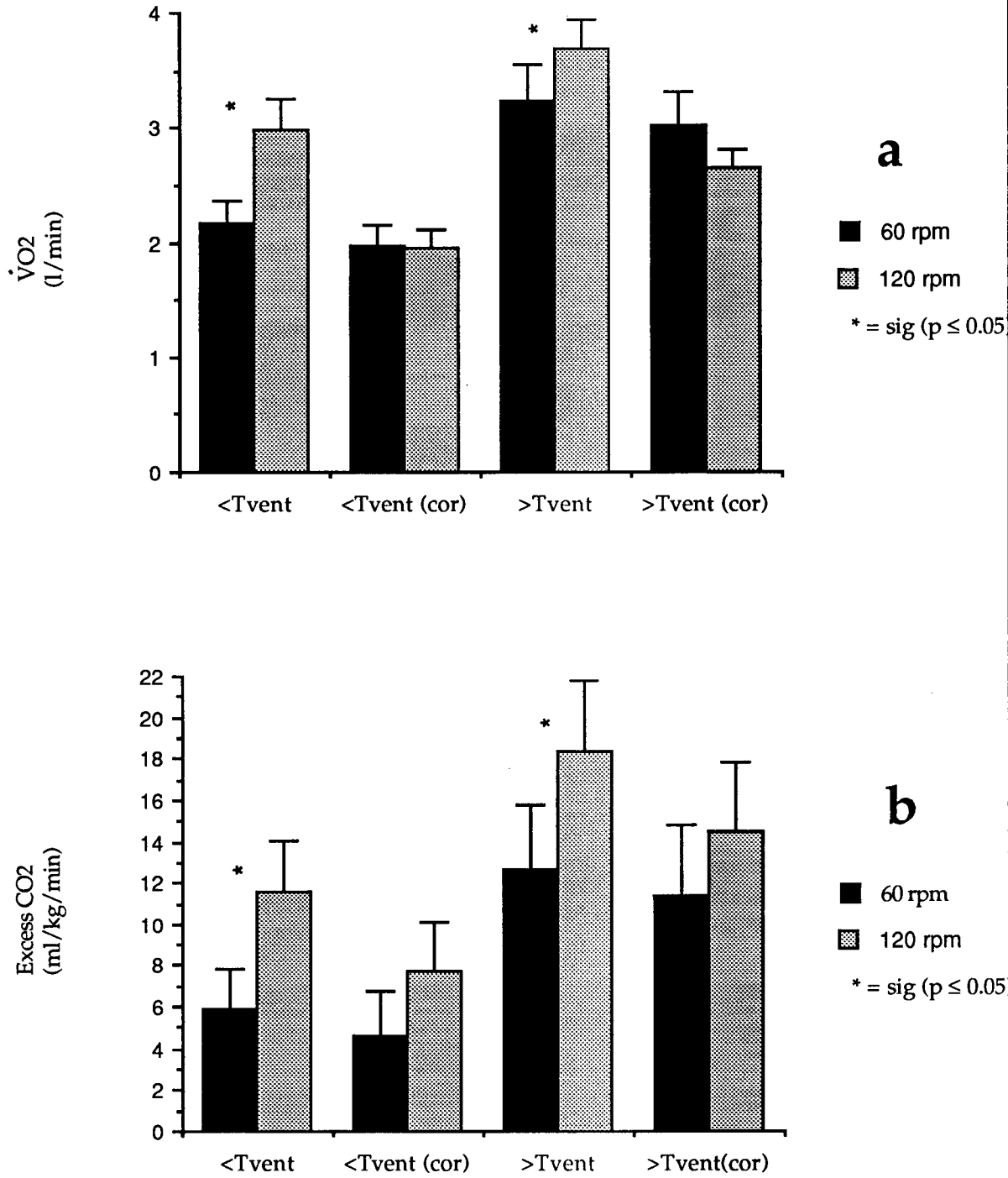

Figure 7. (a) Mean steady-state $\dot{V}O_2$ (± 1 SD) (corrected and uncorrected) and (b) mean steady-state excess CO_2 (± 1 SD) (corrected and uncorrected) responses while cycling at sub-T_{vent} and supra-T_{vent} power outputs.

Chapter 5

DISCUSSION

Overview

The purpose of this study was to determine if cadence had an effect on the contributions of the aerobic and anaerobic energy pools while cycling at constant power outputs below and above the anaerobic threshold. Peak VO_2 and peak excess CO_2 values were not significantly different between cadences and the values for these variables agreed with values reported in the literature (e.g. Pivarnik et al., 1988). The anaerobic threshold was identified in this study as the point during incremental exercise where excess CO_2 rose non-linearly. This point was referred to as the ventilatory threshold (T_{vent}) and was calculated objectively by a computer program based on an algorithm by Orr et al. (1982).

The results showed that steady-state VO_2 and excess CO_2 were significantly greater at 120 rpm than at 60 rpm while cycling at power outputs below and above T_{vent}. However, correcting these data to account for the zero-load cost of cycling at different cadences resulted in non-significant differences between cadences for both variables suggesting that moving the legs faster at the higher cadence was responsible for the significantly higher $\dot{V}O_2$ and excess CO_2 values.

The changes seen in the ventilatory parameters during steady-state cycling are driven principally by the amount of activity present in the muscles of the legs. EMG activity recorded from five major cycling muscles in the leg showed no significant differences between cadences in four of five muscles at both sub-T_{vent} and supra-T_{vent} power outputs despite the significant differences seen in the ventilatory parameters. Only the rectus femoris showed significantly greater amounts of activity at 60 rpm.

The following discussion will first describe the responses of the leg muscles during steady-state cycling at sub-T_{vent} and supra-T_{vent} power outputs. Following

the review of muscle activity will be a discussion of the ventilatory responses and a short summary devoted to a synthesis of these two responses.

Electromyographic Response of Selected Muscles During Bicycle Pedalling.

Timing of CIEMG. Many attempts have been made to describe the activity patterns of a number of muscles contributing to the action of pedalling a bicycle. Houtz and Fischer (1959) studied the activity of fourteen muscles for three subjects riding a cycle ergometer to attempt to correlate surface electromyographic data with joint range data. Mohr et al. (1981) collected EMG activity from six muscles in the lower extremity while manipulating variables such as power output, cadence, and riding position. Finally Gregor et al. (1982) reported the EMG activity profiles of eight lower extremity muscles collected from ten elite male cyclists riding on an ergometer that closely resembled a conventional racing bicycle. All of these studies report EMG patterns that closely resembled the patterns demonstrated in this study.

Biceps Femoris. Houtz and Fischer (1959) report the activity of the biceps femoris to be short in duration, building to a peak and rapidly diminishing. Although not reported, the position in the pedal cycle where the activity occurred was the middle third of the cycle, between approximately 35% and 70% cycle time. The data collected in the present study tends to support the results of Houtz and Fischer (1959). Contrary to these data, Mohr et al. (1981) report activity in the biceps femoris beginning approximately 45° before top-dead-center and not ending until 45° past bottom-dead-center. This would correspond to approximately 85% to 65% cycle time leaving only 25% of the cycle without any major activity. Gregor et al. (1982) report moderate to high levels of activity in the biceps femoris throughout the entire pedal cycle. The biceps femoris is two-joint muscle, responsible for both knee flexion and hip extension. While cycling, hip extension during the power phase (0% to 50% cycle time), while knee flexion occurs during the recovery phase (50% to 100% cycle time). This could explain why Gregor et al. (1982) report a certain

degree of activity throughout the cycle. The ergometer used in this study was equipped with a set of clipless pedals which may have contributed to minor shifts in the location of activity within the pedal cycle. Mohr et al. (1981) report little if any differences in the timing of biceps femoris activity between pedals with and pedals without toe-straps, however the cadences and power outputs used in their study were different than those used in this study.

Rectus Femoris and Vastus Lateralis. The timing of the rectus femoris and the vastus lateralis muscles are virtually identical therefore they will be discussed in the same section. All literature reporting electromyographic activity while cycling include these muscles because they are primary cycling muscles responsible for generating the power necessary to maintain cadences under high loads. The typical pattern of EMG activity for these two muscles is activity beginning approximately 10 - 20% prior to top-dead-center (80 - 90% cycle time) and ending at 40% into the pedal cycle. The EMG patterns of both the rectus femoris and the vastus lateralis in this study reflect these typical patterns.

Tibialis Anterior. The timing pattern of the activity of the tibialis anterior reported in the literature varies little. Activity in the tibialis anterior begins approximately 75% into the pedal cycle and ends approximately 25% after top-dead-center. The muscle is mainly involved in dorsiflexion at the ankle which is the action during the top half of the pedal cycle. The activity from the tibialis anterior collected in this study generally followed this timing pattern as well.

Medial Gastrocnemius. The activity collected from the medial gastrocnemius in this study closely resembles the activity reported by Gregor et al. (1982). A large burst of activity beginning approximately 20% into the pedal cycle with the majority of the activity not ending until 75% cycle time. Mohr et al. (1981) also report this long duration of activity. The medial gastrocnemius is involved in the motion at the knee as well as the ankle and plays a key role in the emphasis placed on plantarflexion employed by cyclists to improve force application to the

pedal. Data collected in this study demonstrated a burst of activity present in the medial gastrocnemius during the plantarflexion between 25% and 50% of the cycle and the second burst present during the knee flexion occurring throughout the recovery stage of the pedal cycle (50% to 100% cycle time).

The Effect of Power Output on CIEMG. No significant differences in the amount of integrated electromyographic activity of any of the five muscles were found between riding at a power output below the T_{vent} and above the T_{vent} when either of the two cadences was held constant. Although the amount of activity was not significantly different at 60 rpm, the CIEMG was on average, 31% greater at the supra-T_{vent} power output than at the sub-T_{vent} power output. This result supports the linear relationship between power output and IEMG reported by Bigland and Lippold (1957) and reflects the need for a greater amount of muscular activity to overcome the higher forces accompanying the larger power output.

The CIEMG at 120 rpm did not increase with increases in power output. Only the vastus lateralis demonstrated greater CIEMG values at the higher power output. The CIEMG of the other four muscles was, on average, 22% lower at the supra-T_{vent} power output. This result was interesting because it opposed the well documented linear relationship between IEMG and power output. The failure to demonstrate an increase in CIEMG at the supra-T_{vent} power output could be attributed to the small sample size at 120 rpm. Due to functional difficulties experienced during the data collection, EMG data from two subjects were lost, leaving a sample size of n=2 for the 120 rpm conditions. The effects of the low sample size was especially evident in the case of the tibialis anterior where the CIEMG collected from one of the two subjects was extremely low thereby reducing the overall mean for that condition. If it were not for the low sample size, CIEMG values at the higher power output at 120 rpm may have shown the same relationship demonstrated by the 60 rpm data.

The Effect of Cadence on CIEMG. The effects of cadence on the CIEMG varied depending on the muscle studied as well as the power output of the rider. It

appeared as though the major adjustment made by the rider while cycling at a power output less than or greater than ventilatory threshold was to lengthen the duration of activity, or in the case of equal activity durations, reducing sudden bursts of activity while riding at the lower cadence of 60 rpm. It must also be remembered that the EMG data have been time-normalized so that regardless of the cadence, the timing patterns of the EMG are the same (i.e. when the muscle turns on and turns off). However when integrating the data to determine how much activity is present, the time base is different because it takes twice as long to complete a pedal cycle at 60 rpm than it does at 120 rpm.

The results of this study showed that of the five muscles studied, only one (rectus femoris) displayed significantly more integrated activity at 60 rpm as opposed to 120 rpm. However, considerable differences in the duration and magnitude of the activity were evident.

For example, at the sub-T_{vent} power output, the biceps femoris exhibited a large burst of activity at 40% of the pedal cycle at 120 rpm. While riding at a higher cadence the forces that must be exerted at the pedal are less, therefore one would assume less activity at the higher cadences based on the linear force-EMG relationship (Bigland and Lippold, 1954). At 60 rpm, the rider activates the biceps femoris for a longer period of time, resulting in the same amount of CIEMG activity even though the magnitude and length of activity are different from those at 120 rpm. This same effect was demonstrated in the sub-T_{vent} profiles of the tibialis anterior and the vastus lateralis. At power outputs above the ventilatory threshold, similar effects were also evident in these muscles.

Non-significant differences in the CIEMG activity at the two cadences are supported by results reported by Reimer et al. (1989). Given that the pedal resistances are higher at lower cadences, increased levels of muscle activity would be expected at these cadences however, the results of this study demonstrated otherwise. It is possible that there may be a change in recruitment patterns that

allow the rider to maintain a lower cadence without significantly altering the amount of electrical activity in the muscle. A second reason as to why the differences between the CIEMG at the two cadences were not significantly different lies in the variability in the CIEMG measure. Measures were taken to ensure that the inherent variability of the EMG signal was minimized. Every effort was made to keep surface electrode locations consistent between trials, however small variations in electrode positions can result in changes in the EMG signal (Basmajian, 1978). Maintaining the same electrode positions was particularly difficult for the biceps femoris.

Variability also accompanies the data analysis procedure used to describe the data. Describing the data as a per-cycle integrated EMG signal measure may not be the most appropriate measure in terms of reducing variability. In order to test whether a different measure could reduce variability, the EMG data were also represented as percentages of the peak EMG activity within the pedal cycle. When these data were analyzed, no significant differences were found.

Patterson and Moreno (1990) report increases of only 3% and 13% in the resultant force on the pedals averaged over one cycle as cadence decreased from 120 rpm to 60 rpm while riding at 100 watts and 200 watts respectively. These findings may also offer an explanation as to why a cadence effect was found only in the rectus femoris and not in any of the other muscles. If a one-fold decrease in cadence results in such small increases in resultant pedal force, it is possible that the muscles of the leg may all make minor adjustments in activation to account for the small increase in pedal resistances.

Aerobic Contributions to Total Energy Cost of Cycling.

Aerobic Contributions During Incremental Exercise. The mean peak $\dot{V}O_2$ values in this study were 3.71 l/min at 60 rpm and 3.83 l/min at 120 rpm. The difference between these two values was not significant. The mean peak $\dot{V}O_2$

values in this study were slightly lower than values reported by Pivarnik et al. (1988). They reported peak $\dot{V}O_2$ values of 3.96 l/min at 50 rpm and 4.08 l/min at 90 rpm. The difference between these values was also non-significant. The slight difference between these values and the values in this study could be attributed to the different cadences used in the Pivarnik et al. (1988) study as well as the difference in ability of the subjects.

When the data were corrected to remove the calculated zero-load cost of riding at each cadence, the peak VO_2 value at 60 rpm was significantly greater than the corrected peak VO_2 at 120 rpm. Correcting the VO_2 data also resulted in a relative magnitude change such that the corrected VO_2 values at 60 rpm were consistently greater than their counterparts at 120 rpm. This change was due to a larger correction factor at 120 rpm.

It is important to consider how unloaded cycling affects physiological variables such as $\dot{V}O_2$. Seabury et al. (1977) reported energy expenditure (kJ/kg·min) values for unloaded cycling at cadences ranging from 30 rpm to 120 rpm. The data best fit a parabolic function with the energy expenditure increasing as cadence increased. Wells et al. (1986) have calculated the internal mechanical work required to simply raise and lower the legs during cycle ergometry at three different cadences and report mean internal work rates of 11.5, 20, and 62 watts at cadences of 30, 60, and 90 rpm respectively. Increasing the cadence by 50% from 60 rpm to 90 rpm resulted in an 310% increase in the internal work rate. This value would be even higher if the cadence was increased to 120 rpm such as was done in this study.

The results of these studies, one physiological in nature and the other mechanical, demonstrate that the work performed just to move the lower limbs at a prescribed rate consumes energy and the amount of work and consequently energy demand increases as the cadence increases. Therefore, when a rider is riding on a cycle ergometer that is set at 200 watts, the rider is not only met with the challenge of fuelling his muscles to overcome the resistive forces of the ergometer, but also must

fuel the muscles to move the lower limbs at the required cadence. By correcting the $\dot{V}O_2$ and excess CO_2 data, any differences observed in the corrected data were due exclusively to changes imposed by the external power output.

Aerobic Contributions During Steady-State Cycling Below and Above the T_{vent}.

Published data describing the aerobic contributions to the total energy demand of performing steady-state cycling exercise at varying power outputs is very limited. To date, only one study has specifically addressed this question. In 1981, Hagberg et al. published $\dot{V}O_2$ data collected from a group of seven road-racing cyclists while riding at cadences above and below preferred cadences at a power output requiring 80% of their maximal oxygen uptake. The results published by Hagberg et al. (1981) showed that riding at a cadence below the preferred cadence elicited $\dot{V}O_2$ values that were, on average, 3.5% greater than at the preferred cadence. At a cadence that was greater than the preferred cadence, the $\dot{V}O_2$ was an average of 11.3% greater than the $\dot{V}O_2$ at the preferred cadence. Hagberg et al. (1981) also collected data during unloaded cycling and reported significant increases in $\dot{V}O_2$ as cadence increased from 60 to 120 rpm.

The results of the Hagberg et al. (1981) study combined with the results of this study serve to show that the aerobic contribution to the total energy cost of cycling at constant power output is directly related to the cadence. As cadence increases, physiological variables describing the aerobic contribution (i.e. $\dot{V}O_2$) also increase despite the fact that pedal forces are decreasing (Figure 8). Therefore, the mechanism responsible for the significant increases seen in the $\dot{V}O_2$ data cannot be attributed mechanically to pedal forces, but rather to the fact that simply moving the legs more frequently requires the delivery of more energy to the working muscles. However, Hagberg et al. (1981) report that the relationship between $\dot{V}O_2$ and cadence even during unloaded cycling is more complex than simply moving the legs faster. They credit this to an exponential increase in $\dot{V}O_2$ observed with increasing cadences. The exponential increase could not be confirmed by the data collected in

this study due to the limited number of cadences used, however it appears as though more data is necessary in order to determine the exact interaction between cadence and $\dot{V}O_2$.

Anaerobic Contributions to the Total Energy Cost of Cycling.

Anaerobic Contributions During Incremental Exercise. The mean uncorrected peak excess CO_2 values in this study were 19.74 ml/kg/min at 60 rpm and 18.84 ml/kg/min at 120 rpm. The difference between these two values was not significant and correcting the peak excess CO_2 values did not result in any significant differences either. Pivarnik et al. (1988) did not report excess CO_2 values however using the formula for determining excess CO_2 [$\dot{V}CO_2$ - (RQ X $\dot{V}O_2$)] the peak excess CO_2 was 24.10 ml/kg/min at 50 rpm and 22.57 ml/kg/min at 90 rpm.

Correcting the excess CO_2 values also resulted in a small relative magnitude shift, but not as dramatic as the $\dot{V}O_2$. As was the case in the aerobic contribution data, the excess CO_2 correction factor for 120 rpm was significantly greater than the correction factor at 60 rpm. The higher excess CO_2 production during unloaded cycling at 120 rpm showed that while cycling at higher cadences, a greater contribution is made by the anaerobic metabolism.

Anaerobic Contributions During Steady-State Cycling Below and Above the T_{vent}. As was the case with the aerobic data, the amount of published data describing the anaerobic contributions to the total energy cost of cycling at constant power outputs is limited. The results of this study showed that while cycling at constant power outputs below and above the T_{vent}, the excess CO_2 at 120 rpm was significantly greater than at 60 rpm (Figure 8). These results are supported by Buchanan and Weltman (1985) report significantly greater blood lactate concentrations associated with 120 rpm than with 60 or 90 rpm while cycling at submaximal power outputs. Lollgen et al. (1980) and Hughes et al. (1982) also report increases in blood lactate concentration with increases in cadence beyond 60 rpm.

The cause of the elevated blood lactate concentrations at the higher cadences is still not certain. Lactate production, linked with excess CO_2 production, are indicators of anaerobic metabolism. Blood lactate levels can be affected by factors such as hypoxia, recruitment of fast-twitch type muscle fibers, as well as the rate of efflux from the muscle (Buchanan and Weltman, 1980). As cadences are increased to levels as high as 120 rpm it is possible that recruitment changes may occur that could contribute to the increased anaerobic contribution. In addition to recruitment changes, the fact that the legs are moving much faster increases the "pump-like" action of the legs which might contribute to the increased rate of efflux of lactate.

The increase in excess CO_2 due to the increase in cadence and therefore rate of leg movement, was also confirmed by observing the effect of correcting the data for zero-load cycling. The corrected, or "load-dependent" excess CO_2 at the two cadences used in this study were not significantly different, whereas the uncorrected excess CO_2 data were significantly different at the two cadences. Therefore, the result of moving the legs faster to maintain a higher cadence could be responsible for elevating the excess CO_2 production.

The original hypothesis of this study was that the summed aerobic and anaerobic contributions to the total energy cost would be constant regardless of cadence at constant power output. If this held true then an elevated aerobic contribution must be accompanied with a decreased anaerobic contribution in order to maintain a constant total energy cost. This was not found to be the case at either of the two cadences studied. In both cases, aerobic and anaerobic contributions were greater at 120 rpm.

The zero-load cost of cycling results of this and other studies (e.g. Hagberg et al., 1981) have demonstrated that riding at two different cadences such as 60 and 120 rpm is very different. Higher cadences in the order of 120 rpm have been found to significantly elevate aerobic and anaerobic metabolism. Therefore, when physiological data collected during loaded cycling at different cadences are analyzed,

the physiological data associated with the higher cadence is elevated. Removal of the calculated zero-load cost of cycling at each cadence resulted in VO_2 data that were lower at 120 rpm than at 60 rpm when they were originally greater. Lower VO_2 values at the higher cadences support the perception of effort concept proposed by Cafarelli (1977) in that higher cadences are coupled with lower pedal forces. The lower pedal forces correspond with a lower peripheral sense of effort and a lower $\dot{V}O_2$ value.

Removal of the calculated zero-load cost of cycling at each cadence did not result in lower excess CO_2 values at 120 rpm as was the case with the VO_2 data. This was due to the fact that the 120 rpm correction factor for excess CO_2 was only three times greater than the 60 rpm correction factor, whereas the difference for the VO_2 correction factors was the order of five (Appendix A). The fact that the excess CO_2 at 120 rpm is greater than at 60 rpm despited the lower pedal forces at 120 rpm contradicts Cafarelli's perception of effort concept. This result was not expected, however one possible explanation as to why this occurred could be an abnormally low excess CO_2 correction factor at 120 rpm. Further investigation to confirm this assumption is necessary.

Chapter 6

CONCLUSIONS AND RECOMMENDATIONS

Summary. This study was designed to observe the effect of cadence variations on the contributions of aerobic metabolism and anaerobic metabolism to the total energy costs of cycling at constant power outputs below and above the ventilatory threshold. The ventilatory threshold (T_{vent}) was determined by a computer program designed to fit a pair of simple linear regression to all possible combinations of excess CO2 data collected from four male subjects during progressive incremental maximal tests performed at 60 and 120 rpm. The combination that yielded the smallest sum of residual squares was retained and the point where the two lines intersected was found to be the T_{vent}. All subjects subsequently completed steady-state rides performed at 60 and 120 rpm at constant power outputs 20% below and 20% above the T_{vent}. Oxygen uptake, excess CO_2, and EMG data were simultaneously collected during all steady-state trials. All results were analyzed at a significance level of $p \leq 0.05$ to determine differences between means.

The following section will summarize the results of this study by addressing each of the hypotheses individually.

1. It was hypothesized that variations in cadence while cycling at the sub-T_{vent} power output would not significantly change steady-state $\dot{V}O_2$, excess CO_2, or CIEMG activity. The results of this study proved otherwise demonstrating a significantly greater steady-state $\dot{V}O_2$ and excess CO_2 at 120 rpm when compared to those at 60 rpm. Subtracting the calculated zero-load cost of cycling at each cadence resulted in no significant differences between cadences in either the steady-state VO_2 or the steady-state excess CO_2. Changing cadence from 60 rpm to 120 rpm resulted in a significantly lower CIEMG in only one of the five muscles studied (rectus femoris).

2. It was also hypothesized that cycling at steady-state at the supra-T_{vent} power output would result in a significantly greater VO_2 at a cadence of 120 rpm than at 60 rpm; excess CO_2 would be significantly greater at 60 rpm than at 120 rpm reflecting the increase in anaerobic metabolism to meet the equivalent total energy requirements in the presence of a reduced aerobic metabolism, and the CIEMG would be greater at 60 rpm than at 120 rpm due to the additional pedal forces required at lower cadences at constant power output.

The steady-state $\dot{V}O_2$ at 120 rpm was significantly greater than that at 60 rpm. However, excess CO_2 was not found to be significantly greater at 60 rpm, in fact, it was found to be significantly greater at 120 rpm. Correcting of these data also resulted in no significant differences between cadences. The rectus femoris was found to be the only muscle that demonstrated significantly greater CIEMG activity at 60 rpm.

Significant differences in uncorrected steady-state VO_2 and excess CO_2 values combined with the non-significant differences in the corresponding corrected data clearly demonstrate that moving the legs faster plays a significant role in the ventilatory changes observed in this study. It was also found that although the aerobic contribution to the total energy cost of cycling at constant power outputs below and above T_{vent} were predominant, increasing cadence resulted in a greater relative increase in the anaerobic contribution to the total energy cost.

The non-significant changes observed in the CIEMG of four of the five muscles studied served to show that the loads imposed on the rider at 60 rpm were not sufficient to adequately affect the peripheral input to the overall effort of the exercise. Decreasing cadence to 40 rpm may be required before any significant effects on the peripheral system can be observed.

Conclusions.

Based on the results of this study, the following conclusions were made.

1. It was hypothesized that at a sub-T_{vent} power output no significant difference would be found between any of the dependent variables while cycling at 60 and 120 rpm. Corrected ventilatory data supported this hypothesis confirming the data presented by Reimer et al. (1989) (Appendix C). Significant differences were found using uncorrected ventilatory data suggesting that the rate of leg movement is a factor in the increased $\dot{V}O_2$ and excess CO_2 at 120 rpm.

2. (a) At a supra-T_{vent} power output it was hypothesized that the VO_2 at 120 rpm would be significantly greater than at 60 rpm. This hypothesis was also supported confirming the results presented by Coast and Welch (1985), Böning et al. (1984), and Seabury et al., (1977).

Correcting the $\dot{V}O_2$ data resulted in non-significance confirming the thought that the rate of leg movement was a factor in this study.

(b) It was also hypothesized at this power output that the excess CO_2 would be greater at 60 rpm than at 120 rpm reflecting the additional contribution of the anaerobic energy system to meet the total energy cost in the presence of a reduced $\dot{V}O_2$. This hypothesis was not supported. Correcting the data resulted in non-significance, suggesting that the confounding factor of moving the legs at different velocities played a role in the initial calculations.

(c) With regards to the muscle activity, it was hypothesized that the CIEMG would be greater at 60 rpm reflecting the need for greater muscular activity to overcome the increased pedal forces at the lower cadence. This hypothesis was confirmed in only one of the five muscles studied, the rectus femoris. Other muscles did show increases in the CIEMG at the lower cadence however, they were not significant.

Recommendations.

1. In all studies with low sample sizes there is a danger of arriving at conclusions that are not warranted. Future studies on this topic should employ a larger sample size. A higher sample rate for the collection of EMG data could allow collection of raw EMG data and a better indicator of the changes in muscle activation. This was one of the inherent limitations of the equipment used in this study and could not be avoided.

2. The ventilatory responses during unloaded cycling were extrapolated in this study. Future work should include data collection at a power output of zero watts to correctly calculate the zero-load cost of cycling at different cadences. Muscle lactate data would also be a much better indicator of anaerobic contributions to the total energy cost of performing work than a non-invasive measure such as excess CO_2.

3. Finally, to completely understand how the rider responds to changes in cadence, a measurement of pedal forces at a wider range of cadences are required. These measurements require force transducers mounted in a pedal. These data could account for changes seen in muscle activity as well as offer reasons as to why the ventilatory data responds as it does.

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APPENDIX A - INDIVIDUAL PHYSIOLOGICAL DATA

Table 1. Age, Height and Weight of Subjects.

Subject	Age (yrs.)	Height (cm.)	Weight (kg.)
1	20	183.7	83.3
2	20	164.3	71.6
3	19	167.8	67.6
4	26	178.9	79.1
Mean	21	173.7	75.4
Stand. Dev.	3	9.1	7.1

Table 2. Individual $\dot{V}O_2$ (l/min) correction factors.

Subject	60 rpm	120 rpm
1	0.14	1.13
2	0.13	0.83
3	0.00	0.92
4	0.54	1.22
Mean	0.20	1.03
Stand. Dev.	0.23	0.18

Table 3. Individual excess CO_2 (ml/kg/min) correction factors.

Subject	60 rpm	120 rpm
1	1.31	3.56
2	2.22	3.50
3	0.61	4.40
4	1.05	4.06
Mean	1.30	3.88
Stand. Dev.	0.68	0.43

Table 4. Individual uncorrected $\dot{V}O_2$ data (l/min) during progressive incremental maximum exercise protocol performed at 60 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	**256**	**288**	**320**	**352**
1	1.70	1.85	2.11	2.69	2.99	3.34	3.74	
2	1.52	1.89	2.14	2.59	2.95	3.34	3.74	3.82
3	1.50	1.96	2.40	2.77	3.27	3.68		
4	1.89	2.21	2.69	3.02	3.29	3.59		
Mean	1.65	1.98	2.34	2.77	3.13	3.49	3.74	3.82
Stand. Dev.	0.18	0.16	0.27	0.18	0.18	0.17	0.00	0.00

Table 5. Individual uncorrected $\dot{V}O_2$ data (l/min) during progressive incremental maximum exercise protocol performed at 120 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	**256**	**288**	**320**	**352**
1	2.43	2.65	2.78	3.14	3.51	3.81	4.19	
2	2.11	2.37	2.82	3.02	3.44	3.65		
3	2.13	2.36	2.83	3.08	3.33	3.63		
4	2.50	2.77	3.07	3.24	3.62	3.84		
Mean	2.29	2.54	2.88	3.12	3.48	3.73	4.19	0.00
Stand. Dev.	0.20	0.21	0.13	0.09	0.12	0.11	0.00	0.00

Table 6. Individual corrected $\dot{V}O_2$ data (l/min) during progressive incremental maximum exercise protocol performed at 60 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	**256**	**288**	**320**	**352**
1	1.56	1.71	1.97	2.55	2.85	3.20	3.60	
2	1.39	1.76	2.01	2.46	2.82	3.21	3.61	3.69
3	1.50	1.96	2.40	2.77	3.27	3.68		
4	1.35	1.67	2.15	2.48	2.75	3.05		
Mean	1.45	1.78	2.13	2.57	2.92	3.29	3.61	3.69
Stand. Dev.	0.10	0.13	0.19	0.14	0.24	0.27	0.01	0.00

Table 7. Individual corrected $\dot{V}O_2$ data (l/min) during progressive incremental maximum exercise protocol performed at 120 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	**256**	**288**	**320**	**352**
1	1.30	1.52	1.65	2.01	2.38	2.68	3.06	
2	1.28	1.54	1.99	2.19	2.61	2.82		
3	1.21	1.44	1.91	2.16	2.41	2.71		
4	1.28	1.55	1.85	2.02	2.40	2.62		
Mean	1.27	1.51	1.85	2.10	2.45	2.71	3.06	0.00
Stand. Dev.	0.04	0.05	0.15	0.09	0.11	0.08	0.00	0.00

Table 8. Individual uncorrected excess CO_2 data (ml/kg/min) during progressive incremental maximum exercise protocol performed at 60 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	256	**288**	**320**	**352**
1	3.57	5.71	7.43	10.41	12.03	16.10	20.92	
2	5.36	6.70	8.25	11.04	13.70	18.03	22.98	22.94
3	2.44	4.68	7.36	9.00	12.78	19.14		
4	3.01	5.41	7.86	9.94	11.54	15.94		
Mean	3.60	5.63	7.73	10.10	12.51	17.30	21.95	22.94
Stand. Dev.	1.26	0.84	0.41	0.86	0.94	1.55	1.46	0.00

Table 9. Individual uncorrected excess CO_2 data (ml/kg/min) during progressive incremental maximum exercise protocol performed at 120 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	256	**288**	**320**	**352**
1	6.70	7.86	9.12	9.99	12.61	12.71	18.51	
2	5.81	8.00	11.66	12.30	17.45	19.73		
3	8.35	10.52	11.12	12.76	14.85	20.28		
4	7.86	9.28	11.18	12.25	16.36	16.83		
Mean	7.18	8.91	10.77	11.83	15.32	17.39	18.51	0.00
Stand. Dev.	1.15	1.25	1.13	1.24	2.10	3.47	0.00	0.00

Table 10. Individual corrected excess CO_2 data (l/min) during progressive incremental maximum exercise protocol performed at 60 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	**256**	**288**	**320**	**352**
1	2.26	4.40	6.12	9.10	10.72	14.79	19.61	
2	3.14	4.48	6.03	8.82	11.48	15.81	20.76	20.72
3	1.83	4.07	6.75	8.39	12.17	18.53		
4	1.96	4.36	6.81	8.89	10.49	14.89		
Mean	2.30	4.33	6.43	8.80	11.22	16.01	20.19	20.72
Stand. Dev.	0.59	0.18	0.41	0.30	0.76	1.74	0.81	0.00

Table 11. Individual corrected excess CO_2 data (l/min) during progressive incremental maximum exercise protocol performed at 120 rpm.

Subject	Power Output (watts)							
	128	**160**	**192**	**224**	**256**	**288**	**320**	**352**
1	3.14	4.30	5.56	6.43	9.05	9.15	14.95	
2	2.31	4.50	8.16	8.80	13.95	16.23		
3	3.95	6.12	6.72	8.36	10.45	15.88		
4	3.80	5.22	7.12	8.19	12.30	12.77		
Mean	3.30	5.04	6.89	7.95	11.44	13.51	14.95	0.00
Stand. Dev.	0.75	0.82	1.07	1.04	2.14	3.30	0.00	0.00

Table 12. Steady-state $\dot{V}O_2$ (l/min) while cycling at 60 rpm at sub T_{vent} and supra-T_{vent} power outputs.

Subject	<T_{vent} uncorrected	<T_{vent} corrected	>T_{vent} uncorrected	>T_{vent} corrected
1	2.31	2.17	3.46	3.32
2	1.95	1.82	2.80	2.67
3	2.07	2.07	3.14	3.14
4	2.43	1.89	3.49	2.95
Mean	2.19	1.99	3.22	3.02
Stand. Dev.	0.22	0.16	0.32	0.28

Table 13. Steady-state $\dot{V}O_2$ (l/min) while cycling at 120 rpm at sub T_{vent} and supra-T_{vent} power outputs.

Subject	<T_{vent} uncorrected	<T_{vent} corrected	>T_{vent} uncorrected	>T_{vent} corrected
1	3.18	2.05	3.94	2.81
2	2.86	2.03	3.81	2.98
3	2.67	1.75	3.41	2.49
4	3.17	1.95	3.80	2.58
Mean	2.97	1.95	3.74	2.72
Stand. Dev.	0.25	0.14	0.23	0.22

Table 14. Steady-state excess CO_2 (ml/kg/min) while cycling at 60 rpm at sub T_{vent} and supra-T_{vent} power outputs.

Subject	$<T_{vent}$ uncorrected	$<T_{vent}$ corrected	$>T_{vent}$ uncorrected	$>T_{vent}$ corrected
1	3.23	1.92	8.71	7.40
2	6.28	4.06	11.81	9.59
3	7.52	6.91	15.33	14.72
4	6.84	5.79	14.84	13.79
Mean	5.97	4.67	12.67	11.38
Stand. Dev.	1.89	2.18	3.07	3.46

Table 15. Steady-state excess CO_2 (ml/kg/min) while cycling at 120 rpm at sub T_{vent} and supra-T_{vent} power outputs.

Subject	$<T_{vent}$ uncorrected	$<T_{vent}$ corrected	$>T_{vent}$ uncorrected	$>T_{vent}$ corrected
1	7.96	4.40	14.18	10.62
2	13.37	9.87	21.72	18.22
3	12.12	7.72	19.93	15.53
4	12.53	8.47	17.40	13.34
Mean	11.50	7.62	18.31	14.43
Stand. Dev.	2.41	2.32	3.27	3.23

APPENDIX B - INDIVIDUAL EMG DATA

Table 1. Normalized RMS EMG (V) recorded from biceps femoris at 60 rpm at sub T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	0.000	0.000	0.000	0.000	0.000	0.000
5	0.000	0.000	0.000	0.320	0.080	0.160
10	0.000	0.000	0.000	0.495	0.124	0.248
15	0.000	0.391	0.250	0.479	0.280	0.209
20	0.158	0.331	0.275	0.777	0.385	0.271
25	0.186	0.454	0.294	0.886	0.455	0.308
30	0.227	1.140	0.350	1.434	0.788	0.591
35	0.410	0.915	0.602	1.449	0.844	0.454
40	0.524	0.370	1.393	1.646	0.983	0.631
45	0.428	0.331	0.611	1.129	0.625	0.356
50	0.926	0.000	1.424	0.988	0.834	0.599
55	2.188	0.000	1.053	1.566	1.202	0.926
60	2.170	0.000	0.992	2.034	1.299	1.013
65	2.297	0.000	0.709	2.038	1.261	1.091
70	0.530	0.000	0.427	0.647	0.401	0.282
75	0.192	0.000	0.000	0.000	0.048	0.096
80	0.000	0.000	0.000	0.000	0.000	0.000
85	0.000	0.000	0.000	0.000	0.000	0.000
90	0.000	0.000	0.000	0.000	0.000	0.000
95	0.000	0.000	0.000	0.000	0.000	0.000
100	0.000	0.000	0.000	0.000	0.000	0.000
INT EMG	**0.532**	**0.201**	**0.397**	**0.793**	**0.481**	**0.249**

Table 2. Normalized RMS EMG (V) recorded from biceps femoris at 60 rpm at supra T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	0	0	0	0	0.000	0.000
5	0	0	0	0	0.000	0.000
10	0	0.225	0	0.925	0.288	0.438
15	0	0.33	0.587	0.947	0.466	0.401
20	0.38	0.373	0.982	1.169	0.726	0.411
25	0.426	0.327	0.782	1.503	0.760	0.533
30	0.634	0.794	0.799	1.246	0.868	0.263
35	1.096	1.698	1.819	1.287	1.475	0.340
40	1.436	0.566	2.587	2.888	1.869	1.071
45	2.219	0.653	0.835	1.605	1.328	0.723
50	2.424	0.27	1.661	2.231	1.647	0.973
55	5.057	0.55	1.33	1.725	2.166	1.989
60	7.584	0.546	2.377	1.554	3.015	3.137
65	6.62	0.225	2.159	1.364	2.592	2.800
70	3.856	0.233	1.303	0.847	1.560	1.592
75	0.943	0	0.485	0	0.357	0.453
80	0	0	0	0	0.000	0.000
85	0	0	0	0	0.000	0.000
90	0	0	0	0	0.000	0.000
95	0	0	0	0	0.000	0.000
100	0	0	0	0	0.000	0.000
INT EMG	**1.638**	**0.353**	**0.858**	**0.926**	**0.944**	**0.529**

Table 3. Normalized RMS EMG (V) recorded from biceps femoris at 120 rpm at sub T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	0	0	0.000	0.000
5	0	0.287	0.144	0.203
10	0	0.383	0.192	0.271
15	0	0.81	0.405	0.573
20	0	1.398	0.699	0.989
25	0.802	1.804	1.303	0.709
30	2.247	2.583	2.415	0.238
35	3.586	2.303	2.945	0.907
40	3.373	3.965	3.669	0.419
45	2.233	3.432	2.833	0.848
50	0.94	2.416	1.678	1.044
55	0.313	1.373	0.843	0.750
60	0	0.604	0.302	0.427
65	0	0.317	0.159	0.224
70	0	0	0.000	0.000
75	0	0	0.000	0.000
80	0	0	0.000	0.000
85	0	0	0.000	0.000
90	0	0	0.000	0.000
95	0	0	0.000	0.000
100	0	0	0.000	0.000
INT EMG	**0.340**	**0.533**	0.437	**0.136**

Table 4. Normalized RMS EMG (V) recorded from biceps femoris at 120 rpm at supra T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	0	0	0.000	0.000
5	0	0	0.000	0.000
10	0	0	0.000	0.000
15	0	0	0.000	0.000
20	0	1.072	0.536	0.758
25	0.699	1.416	1.058	0.507
30	2.312	2.182	2.247	0.092
35	2.757	2.598	2.678	0.112
40	3.85	3.842	3.846	0.006
45	2.576	2.833	2.705	0.182
50	1.022	2.425	1.724	0.992
55	0	1.574	0.787	1.113
60	0	0.55	0.275	0.389
65	0	0	0.000	0.000
70	0	0	0.000	0.000
75	0	0	0.000	0.000
80	0	0	0.000	0.000
85	0	0	0.000	0.000
90	0	0	0.000	0.000
95	0	0	0.000	0.000
100	0	0	0.000	0.000
INT EMG	**0.321**	**0.448**	**0.385**	**0.090**

Table 5. Normalized RMS EMG (V) recorded from medial gastrocnemius at 60 rpm at sub-T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	0	0	0	0	0.000	0.000
5	0	0	0	0	0.000	0.000
10	0.516	0.197	0.5472	0	0.315	0.263
15	1.09	0.407	0.7836	0.485	0.691	0.311
20	1.709	0.811	1.4636	0.542	1.131	0.546
25	1.005	1.688	1.1416	1.555	1.347	0.326
30	1.231	2.679	0.9228	2.395	1.807	0.860
35	0.959	2.009	1.9096	2.339	1.804	0.593
40	1.068	1.476	2.204	1.591	1.585	0.470
45	1.353	0	0.856	1.57	0.945	0.697
50	2.542	0.585	0.9168	1.279	1.331	0.856
55	3.154	1.84	0.762	2.007	1.941	0.979
60	3.275	0.225	0.8984	1.388	1.447	1.309
65	1.977	0	0.326	1.323	0.907	0.909
70	2.747	0	0.342	0.731	0.955	1.231
75	2.387	0	0	0	0.597	1.194
80	0.529	0	0	0	0.132	0.265
85	0	0	0	0	0.000	0.000
90	0	0	0	0	0.000	0.000
95	0	0	0	0	0.000	0.000
100	0	0	0	0	0.000	0.000
INT EMG	**1.251**	**0.597**	**0.630**	**0.885**	**0.841**	**0.303**

Table 6. Normalized RMS EMG (V) recorded from medial gastrocnemius at 60 rpm at supra T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	0	0	0	0	0.000	0.000
5	0.625	0.247	0	0	0.218	0.295
10	0.598	0.258	0.523	0.784	0.541	0.218
15	0.913	0.443	1.197	0.693	0.812	0.321
20	1.448	0.428	2.669	1.908	1.613	0.937
25	1.801	0.834	1.226	3.342	1.801	1.102
30	1.085	2.308	1.266	2.816	1.869	0.830
35	0.952	3.782	1.22	2.436	2.098	1.295
40	0.81	0.708	1.905	1.664	1.272	0.602
45	2.016	0.361	0.696	1.524	1.149	0.757
50	5.014	0.823	0.625	2.364	2.207	2.027
55	3.811	1.609	1.146	3.096	2.416	1.248
60	3.397	0.261	0.968	2.493	1.780	1.425
65	1.804	0	2.113	2.128	1.511	1.019
70	1.22	0	0.552	0.682	0.614	0.501
75	1.704	0	0.306	0	0.503	0.814
80	0.715	0	0	0	0.179	0.358
85	0	0	0	0	0.000	0.000
90	0	0	0	0	0.000	0.000
95	0	0	0	0	0.000	0.000
100	0	0	0	0	0.000	0.000
INT EMG	**1.387**	**0.646**	**0.812**	**1.308**	**1.038**	**0.365**

Table 7. Normalized RMS EMG (V) recorded from medial gastrocnemius at 120 rpm at sub-T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	0	0.827	0.414	0.585
5	0	0	0.000	0.000
10	0	0.585	0.293	0.414
15	0.353	1.661	1.007	0.925
20	2.829	4.167	3.498	0.946
25	5.041	5.819	5.430	0.550
30	4.847	7.094	5.971	1.589
35	3.975	5.851	4.913	1.327
40	2.444	4.247	3.346	1.275
45	1.463	2.942	2.203	1.046
50	0.96	2.16	1.560	0.849
55	1.015	1.32	1.168	0.216
60	1.414	1.005	1.210	0.289
65	0.367	0.342	0.355	0.018
70	0	0	0.000	0.000
75	0	0.517	0.259	0.366
80	0.404	0.757	0.581	0.250
85	0.397	0.524	0.461	0.090
90	0.377	0.64	0.509	0.186
95	0.477	0.561	0.519	0.059
100	0.212	0.854	0.533	0.454
INT EMG	**0.659**	**1.009**	0.834	**0.248**

Table 8. Normalized RMS EMG (V) recorded from medial gastrocnemius at 120 rpm at supra T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	0	0	0.000	0.000
5	0	0	0.000	0.000
10	0	0	0.000	0.000
15	0	1.359	0.680	0.961
20	1.421	1.679	1.550	0.182
25	2.137	3.459	2.798	0.935
30	5.663	4.84	5.252	0.582
35	4.248	5.558	4.903	0.926
40	2.487	4.007	3.247	1.075
45	0.934	3.295	2.115	1.669
50	0	2.422	1.211	1.713
55	0	1.268	0.634	0.897
60	2.535	1.255	1.895	0.905
65	0.554	0	0.277	0.392
70	0	0	0.000	0.000
75	0	0	0.000	0.000
80	0	0	0.000	0.000
85	0	0	0.000	0.000
90	0	0	0.000	0.000
95	0	0	0.000	0.000
100	0	0	0.000	0.000
INT EMG	**0.464**	**0.734**	**0.599**	**0.191**

Table 9. Normalized RMS EMG (V) recorded from rectus femoris at 60 rpm at sub T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	3.128	1.547	2.468	2.9288	2.518	0.704
5	3.202	1.099	1.1724	2.9524	2.106	1.126
10	1.272	0.752	0.7004	1.566	1.073	0.418
15	1.245	0.313	0.43	1.786	0.944	0.698
20	1.268	0.563	0.568	0.4392	0.710	0.377
25	0.818	0.891	0.618	0.4532	0.695	0.198
30	0.88	0.953	0.4236	0.324	0.645	0.317
35	0	0	0	0	0.000	0.000
40	0	0	0	0	0.000	0.000
45	0	0	0	0	0.000	0.000
50	0	0	0	0	0.000	0.000
55	0	0	0	0	0.000	0.000
60	0	0	0	0	0.000	0.000
65	0	0	0	0	0.000	0.000
70	0	0	0	0	0.000	0.000
75	0	0	0	0	0.000	0.000
80	0.269	0.616	0	0.9088	0.448	0.397
85	1.799	1.674	0.3128	1.378	1.291	0.676
90	1.177	1.335	1.8712	1.8624	1.561	0.359
95	3.485	2.71	2.284	2.4432	2.731	0.533
100	2.658	1.954	2.4652	3.3056	2.596	0.559
INT EMG	**0.964**	**0.646**	**0.523**	**0.877**	**0.752**	**0.203**

Table 10. Normalized RMS EMG (V) recorded from rectus femoris at 60 rpm at supra T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	2.987	3.645	3.677	2.3236	3.158	0.641
5	2.165	2.086	2.323	2.0276	2.150	0.128
10	1.097	0.932	1.006	1.2132	1.062	0.121
15	0.908	0.637	0.691	0.5904	0.707	0.140
20	1.037	0.796	0.618	0.3412	0.698	0.293
25	0.809	0.98	0.706	0.2684	0.691	0.303
30	0.493	0.355	0	0	0.212	0.251
35	0.413	0	0	0	0.103	0.207
40	0	0	0	0	0.000	0.000
45	0	0	0	0	0.000	0.000
50	0	0	0	0	0.000	0.000
55	0	0	0	0	0.000	0.000
60	0	0	0	0	0.000	0.000
65	0	0	0	0	0.000	0.000
70	0	0	0	0	0.000	0.000
75	0	0	0	0	0.000	0.000
80	0.393	1.168	0	0.5372	0.525	0.485
85	1.111	1.689	1.773	1.2044	1.444	0.335
90	1.344	3.221	1.851	1.4548	1.968	0.863
95	4.414	3.224	3.053	1.2912	2.996	1.287
100	2.779	3.843	3.956	2.7428	3.330	0.659
INT EMG	**0.898**	**0.917**	**0.814**	0.563	**0.798**	**0.163**

Table 11. Normalized RMS EMG (V) recorded from rectus femoris at 120 rpm at sub T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	0.621	1.139	0.880	0.366
5	0.733	2.366	1.550	1.155
10	0.818	1.322	1.070	0.356
15	0.552	0.567	0.560	0.011
20	0.36	0.409	0.385	0.035
25	0	0.334	0.167	0.236
30	0	0	0.000	0.000
35	0	0	0.000	0.000
40	0	0	0.000	0.000
45	0	0	0.000	0.000
50	0	0	0.000	0.000
55	0	0	0.000	0.000
60	0	0	0.000	0.000
65	0	0	0.000	0.000
70	0	0.294	0.147	0.208
75	0.277	0.577	0.427	0.212
80	0.844	2.102	1.473	0.890
85	1.076	2.671	1.874	1.128
90	0.845	2.145	1.495	0.919
95	0.522	0.614	0.568	0.065
100	0.609	1.095	0.852	0.344
INT EMG	**0.163**	**0.361**	**0.262**	**0.140**

Table 12. Normalized RMS EMG (V) recorded from rectus femoris at 120 rpm at supra T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	0.796	1.49	1.143	0.491
5	1.191	2.111	1.651	0.651
10	1.767	1.368	1.568	0.282
15	1.411	1.008	1.210	0.285
20	0.976	0.517	0.747	0.325
25	0.412	0.257	0.335	0.110
30	0	0.123	0.062	0.087
35	0	0	0.000	0.000
40	0	0	0.000	0.000
45	0	0	0.000	0.000
50	0	0	0.000	0.000
55	0	0	0.000	0.000
60	0	0	0.000	0.000
65	0	0	0.000	0.000
70	0	0	0.000	0.000
75	0	0	0.000	0.000
80	0.241	0.394	0.318	0.108
85	0.568	1.215	0.892	0.457
90	1.248	2.421	1.835	0.829
95	0.883	0.747	0.815	0.096
100	0.821	1.621	1.221	0.566
INT EMG	**0.233**	**0.284**	**0.258**	**0.036**

Table 13. Normalized RMS EMG (V) recorded from tibialis anterior at 60 rpm at sub T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	0.274	0.240	0.367	2.034	0.729	0.872
5	0.000	0.000	0.000	0.000	0.000	0.000
10	0.000	0.000	0.000	0.000	0.000	0.000
15	0.000	0.000	0.000	0.000	0.000	0.000
20	0.000	0.000	0.000	0.000	0.000	0.000
25	0.000	0.000	0.000	0.000	0.000	0.000
30	0.000	0.000	0.000	0.000	0.000	0.000
35	0.000	0.000	0.000	0.000	0.000	0.000
40	0.000	0.000	0.000	0.000	0.000	0.000
45	0.000	0.000	0.000	0.000	0.000	0.000
50	1.167	0.263	0.000	0.000	0.358	0.554
55	1.748	0.971	0.000	0.644	0.841	0.727
60	1.486	0.807	0.000	1.309	0.901	0.666
65	1.382	0.408	0.000	1.043	0.708	0.621
70	1.490	0.000	0.000	1.439	0.732	0.846
75	1.561	0.227	0.000	1.885	0.918	0.943
80	1.156	0.310	0.241	1.620	0.832	0.670
85	1.775	0.843	0.735	1.322	1.169	0.478
90	1.964	0.922	1.538	1.361	1.446	0.432
95	0.978	0.785	0.983	2.166	1.228	0.632
100	0.288	0.259	0.365	1.890	0.701	0.794
INT EMG	**0.749**	**0.301**	**0.186**	**0.729**	**0.491**	**0.290**

Table 14. Normalized RMS EMG (V) recorded from tibialis anterior at 60 rpm at supra T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Mean	SD
0	0.374	0.329	0.686	0.463	0.194
5	0	0	0	0.000	0.000
10	0	0	0	0.000	0.000
15	0	0	0	0.000	0.000
20	0	0	0	0.000	0.000
25	0	0	0	0.000	0.000
30	0	0	0	0.000	0.000
35	0	0	0	0.000	0.000
40	0	0	0	0.000	0.000
45	0	0	0	0.000	0.000
50	1.357	0.586	0	0.648	0.681
55	1.853	1.229	0	1.027	0.943
60	1.706	1.027	0.484	1.072	0.612
65	1.11	0.683	0.657	0.817	0.254
70	1.451	0.573	0.534	0.853	0.519
75	0.975	0.637	0.571	0.728	0.217
80	1.205	1.556	0.444	1.068	0.568
85	1.822	1.302	0.916	1.347	0.455
90	2.935	1.445	1.302	1.894	0.904
95	1.302	1.664	1.213	1.393	0.239
100	0.411	0.363	0.721	0.498	0.194
INT EMG	**0.774**	**0.553**	**0.340**	**0.556**	**0.217**

Table 15. Normalized RMS EMG (V) recorded from tibialis anterior at 120 rpm at sub T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	0.654	0.626	0.640	0.020
5	0.312	0	0.156	0.221
10	0	0	0.000	0.000
15	0	0	0.000	0.000
20	0	0	0.000	0.000
25	0	0	0.000	0.000
30	0	0	0.000	0.000
35	0.291	0	0.146	0.206
40	0.339	0.84	0.590	0.354
45	0	2.509	1.255	1.774
50	0	0.583	0.292	0.412
55	0	0	0.000	0.000
60	0	0	0.000	0.000
65	0	0	0.000	0.000
70	0	0	0.000	0.000
75	0	2.272	1.136	1.607
80	1.858	3.589	2.724	1.224
85	2.251	3.221	2.736	0.686
90	1.532	2.858	2.195	0.938
95	0.987	1.754	1.371	0.542
100	0.662	0.633	0.648	0.021
INT EMG	**0.201**	**0.467**	**0.334**	**0.188**

Table 16. Normalized RMS EMG (V) recorded from tibialis anterior at 120 rpm at supra T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	1.662	0.207	0.935	1.029
5	0.468	0.098	0.283	0.262
10	0	0.072	0.036	0.051
15	0	0	0.000	0.000
20	0	0	0.000	0.000
25	0.308	0	0.154	0.218
30	0.31	0	0.155	0.219
35	0.251	0	0.126	0.177
40	0	0.118	0.059	0.083
45	0	0.362	0.181	0.256
50	0	0.143	0.072	0.101
55	0	0	0.000	0.000
60	0.349	0	0.175	0.247
65	0	0	0.000	0.000
70	0	0	0.000	0.000
75	0	0.053	0.027	0.037
80	0.429	0.4	0.415	0.021
85	2.469	0.709	1.589	1.245
90	2.166	0.595	1.381	1.111
95	2.01	0.408	1.209	1.133
100	1.686	0.218	0.952	1.038
INT EMG	**0.266**	**0.080**	**0.173**	**0.131**

Table 17. Normalized RMS EMG (V) recorded from vastus lateralis at 60 rpm at sub T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	5.196	1.726	2.5636	1.5376	2.756	1.687
5	3.502	2.567	2.4772	3.1232	2.917	0.483
10	3.68	1.858	2.1636	3.1216	2.706	0.844
15	2.328	1.646	2.0292	2.948	2.238	0.550
20	2.578	2.407	2.4996	2.942	2.607	0.234
25	2.668	2.76	2.342	2.6128	2.596	0.180
30	1.839	2.651	2.6768	2.2064	2.343	0.400
35	0.489	0	0.3552	0.9968	0.460	0.413
40	0	0	0	0	0.000	0.000
45	0	0	0	0	0.000	0.000
50	0	0	0	0	0.000	0.000
55	0	0	0	0	0.000	0.000
60	0	0	0	0	0.000	0.000
65	0	0	0	0	0.000	0.000
70	0	0	0	0	0.000	0.000
75	0	0	0	0	0.000	0.000
80	0	0	0	0	0.000	0.000
85	0	0	0	0	0.000	0.000
90	0	0.269	0.2152	0	0.121	0.141
95	4.562	1.047	1.5696	0	1.795	1.957
100	5.22	2.29	2.8624	1.4568	2.957	1.615
INT EMG	**1.349**	**0.843**	**0.929**	**0.973**	**1.024**	**0.224**

Table 18. Normalized RMS EMG (V) recorded from vastus lateralis at 60 rpm at supra T_{vent}.

% cycle	Subj #1	Subj #2	Subj #3	Subj #4	Mean	SD
0	4.87	4.783	5.839	3.032	4.631	1.169
5	2.77	5.121	5.047	2.786	3.931	1.332
10	2.212	4.121	3.53	2.054	2.979	1.009
15	1.844	2.873	2.889	1.614	2.305	0.672
20	3.236	2.895	3.839	1.503	2.868	0.990
25	2.033	2.541	4.837	0.896	2.577	1.656
30	1.792	1.308	2.266	0.935	1.575	0.579
35	0.697	0.157	1.002	0.628	0.621	0.349
40	0	0	0	0	0.000	0.000
45	0	0	0	0	0.000	0.000
50	0	0	0	0	0.000	0.000
55	0	0	0	0	0.000	0.000
60	0	0	0	0	0.000	0.000
65	0	0	0	0	0.000	0.000
70	0	0	0	0	0.000	0.000
75	0	0	0	0	0.000	0.000
80	0	0	0	0	0.000	0.000
85	0	0.175	0	0	0.044	0.088
90	0.769	0.461	0	0	0.308	0.377
95	4.159	2.737	2.618	0.462	2.494	1.525
100	5.328	4.145	5.344	3.201	4.505	1.035
INT EMG	**1.206**	**1.351**	**1.604**	**0.681**	**1.210**	**0.389**

Table 19. Normalized RMS EMG (V) recorded from vastus lateralis at 120 rpm at sub T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	3.555	5.068	4.312	1.070
5	2.937	3.354	3.146	0.295
10	3.467	7.262	5.365	2.683
15	2.983	5.321	4.152	1.653
20	2.043	4.479	3.261	1.723
25	0	1.586	0.793	1.121
30	0	0.553	0.277	0.391
35	0	0	0.000	0.000
40	0	0	0.000	0.000
45	0	0	0.000	0.000
50	0	0	0.000	0.000
55	0	0	0.000	0.000
60	0	0	0.000	0.000
65	0	0	0.000	0.000
70	0	0	0.000	0.000
75	0	0	0.000	0.000
80	0	0	0.000	0.000
85	0	0	0.000	0.000
90	1.238	0	0.619	0.875
95	2.177	1.39	1.784	0.556
100	3.514	5.661	4.588	1.518
INT EMG	**0.441**	**0.682**	**0.562**	**0.171**

Table 20. Normalized RMS EMG (V) recorded from vastus lateralis at 120 rpm at supra T_{vent}.

% cycle	Subj #3	Subj #4	Mean	SD
0	3.221	6.354	4.788	2.215
5	7	8.533	7.767	1.084
10	5.37	5.561	5.466	0.135
15	5.577	6.714	6.146	0.804
20	3.405	6.261	4.833	2.019
25	1.701	2.489	2.095	0.557
30	0	1.168	0.584	0.826
35	0	0	0.000	0.000
40	0	0	0.000	0.000
45	0	0	0.000	0.000
50	0	0	0.000	0.000
55	0	0	0.000	0.000
60	0	0	0.000	0.000
65	0	0	0.000	0.000
70	0	0	0.000	0.000
75	0	0	0.000	0.000
80	0	0	0.000	0.000
85	0	0	0.000	0.000
90	0.8	0	0.400	0.566
95	2.014	1.184	1.599	0.587
100	3.222	6.042	4.632	1.994
INT EMG	**0.756**	**0.950**	**0.853**	**0.137**

APPENDIX C - PAPER PRESENTED AT XII ISB CONGRESS, 1989.

EFFECT OF CADENCE ON THE EMG OF THE VASTUS LATERALIS AND OXYGEN UPTAKE DURING STEADY-STATE SUBMAXIMAL CYCLING.

Brad Reimer, Dave Sanderson, and Kevin Seburn.

INTRODUCTION

Few studies have reported how cadence and load changes at constant power output affect the integrated electromyographic (IEMG) activity of the leg muscles, particularly the vastus lateralis. Goto et al. (1975) reported that at equivalent power outputs the IEMG activity of the vastus lateralis increased in a stepwise fashion. Bigland-Ritchie and Woods (1974) found that a linear relationship existed between $\dot{V}O_2$ and IEMG activity during contractions of human quadriceps.

A number of studies have determined that during cycling there exists an optimal pedal rate somewhere in the range of 60 to 100 rpm (e.g. Coast and Welch, 1985). This relationship has been derived from data collected during a number of incremental exercise tests at different cadences and then plotting $\dot{V}O_2$ and cadence for equivalent power outputs. It would seem reasonable to conclude that there would be a similar parabolic relationship between the IEMG from the leg muscles and cadence. However, this has not been shown. Thus, the purpose of this experiment was to determine whether activation of the vastus lateralis muscle indicated a minimum level associated with a particular cadence during cycling at a constant power output.

METHODS

Subjects (n=6) completed four successive rides, in random order, on a Monark bicycle ergometer at each of four cadences 50, 60, 80, and 100 rpm. The resistance was adjusted to maintain a power output of 200 watts for each ride. Subjects used the output of a handlebar-mounted tachometer to maintain the cadence within 1 rpm of the required cadence. The rider remained seated on the bike and rested between rides until his heart rate was below 80 beats/minute (at least 10 minutes).

$\dot{V}O_2$ was collected at 15-second intervals using the standard open-circuit method with a Beckman gas analyzer. The VO_2 for each ride was computed as an average of these values over the last five minutes of the ride. The EMG from the right vastus lateralis was recorded via bipolar surface electrodes placed 2 cm. apart over the lateral aspect of the thigh, approximately 10 cm. proximal to the patella, The amplified EMG signals were sampled through a 12-bit analog-to-digital converter at 500 Hz for a minimum of eight consecutive pedaling cycles. The signal was rectified, integrated, and then normalized for each cycle. An average per cycle EMG (CIEMG) was then computed from these normalized data. All values were averaged across subjects and analysis of variance for repeated measures over one factor (either CIEMG or $\dot{V}O_2$) was used to determine the effect of cadence. The chosen level of significance was $p \leq 0.05$.

RESULTS AND DISCUSSION

The results of this study are summarized in Table 1. There was little change in either CIEMG or $\dot{V}O_2$ in spite of the wide variation in cadence. Analysis of variance showed no significant difference at each condition for either the $\dot{V}O_2$ ($F_{3,15} = 2.083$, $p = 0.144$) or the CIEMG ($F_{3,15} = 2.0$, $p = 0.156$).

The $\dot{V}O_2$ and CIEMG data paralleled each other confirming that $\dot{V}O_2$ and muscle activation are linked. However, neither variable showed the parabolic trend found by Coast and Welch (1985) and Böning et al. (1984). One explanation may lie in the narrow range of pedal rates used for this experiment. For example, Coast and Welch (1985) used pedal rates that ranged from 40 to 120 rpm. There is a tendency of the $\dot{V}O_2$ at 40 rpm and 120 rpm to exaggerate the parabolic effect by shifting the ends of the curve upwards. Thus, the present data may be consistent with a flatter portion in the range of 50 to 100 rpm.

Another explanation may be that there is a fundamental difference in cardiovascular system response to cycling at a constant load versus an incremental load. If this were the case a cyclist performing a series of increments and then maintaining at a submaximal level, say 200 watts, would have a different steady-state $\dot{V}O_2$ than if he had ridden at that 200 watt level. This implies that the metabolic cost of cycling at 200 watts is very much affected by the manner in which the rider achieved that power output. The magnitude of difference in response of the cardiovascular system would likely be exasperated at loads greater than anaerobic threshold. Thus, the concept of minimal cadence based on $\dot{V}O_2$ may well be protocol specific. Clearly, further examinations of the differences in the response to incremental loads versus steady-state loads is warranted.

Table 1. The mean and standard deviations for the per-cycle integrated EMG (CIEMG) and the $\dot{V}O_2$. The CIEMG is in arbitrary units because of the need to normalize each pedal duration.

cadence (rpm)	50	60	80	100
$\dot{V}O_2$ (l/min)	2.46 ± 0.23	2.48 ± 0.35	2.36 ± 0.27	2.52 ± 0.27
CIEMG (arb)	0.60 ± 0.13	0.57 ± 0.13	0.52 ± 0.17	0.54 ± 0.17

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