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Muscle Fatigue at the End of a Maximal Oxygen Consumption Test

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by

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ABSTRACT

Fatigue is a task dependent multifactorial phenomenon, and the most common tool to analyze it is the VO_{2max} test. The purpose of this study was to investigate if muscle is exhaustively taxed at the end of a VO_{2max} test. This was based on data from sEMG, heart rate, and force applied to the pedals during and immediately after a VO_{2max} test compared to an intense anaerobic test. **Methods.** 12 male participants (mean ± SD age = 28.09 ± 6.55 years, height = 176.7 ± 2.97 cm, weight = 73.31 ± 8.44 kg, and VO_{2max} = 60.93 ± 9.75 mL/kg/min) were recruited for the study to perform a VO_{2max} test and an intense anaerobic test (the control), to compare the level of muscle fatigue at the end of the VO_{2max} test. Following a load determining VO_{2max} cycling trial, exercise trials were performed in a randomized order separated by at least seven days. For the VO_{2max} tests, the participants performed a graded exercise protocol at 60 rpm on a mechanically braked cycle ergometer; the power output was set according to each subject's fitness level to

complete the test in 8 to 10 minutes. The intense anaerobic test consisted of cycling on the same ergometer at 60 rpm and 100% of the initial $Watts_{peak}$. In both tests, the participants were encouraged to pedal constantly until reaching volitional exhaustion. The root mean square (RMS), mean power frequency (MPF), and median frequency (MF) were extracted and normalized using the maximal voluntary contraction (MVC) from the selected sEMG samples. MATLAB was used in order to observe the tendencies over time for the left and right rectus femoris, vastus medialis and vastus lateralis. Force production applied to the pedals was measured using four flexiforce load sensors (Teskan, Inc. Boston, MA. USA) installed within each pedal. Heart rate (HR) was monitored during the tests via a commercial clinical electrocardiogram (GE Medical Systems, Milwaukee, WI, USA). Bilateral MVCs were measured before and immediately after each test with the knee positioned at 60 degrees of flexion prior to the MVCs. A paired t-test was used to compare means from sEMG, heart rate and force production pre-test and post-test between both group trials with significance set at $p<0.05$. **Results.** The percentage change in mean sEMG from baseline MVC values was significantly larger for the intense anaerobic tests at the end compared to the VO_{2max} test for two of the six muscles monitored. For the left rectus femoris the remaining percentage of baseline MVC was 84.53 ± 15.01% and 89.9 ± 16.74, respectively, for the intense anaerobic and the VO_{2max} trial; $F(1,5)= 8.124$, $p = 0.036$. Also, a significantly lower remaining percentage of baseline MVC was found for the anaerobic intense trial compared to the VO_{2max} test for right vastus lateralis, 81.92 ± 8.0% and 93.12 ± 4.21% respectively; $F(1,6)= 11.47$, $p = 0.015$. There was a significant remaining difference in the median sEMG frequency for three of the six muscles monitored. A lower remaining percentage was found for the

intense anaerobic test for the right vastus lateralis than for the VO_{2max} test (80.54 ± 13.1% and 93.46 ± 8.78% respectively; $F(1,6)= 7.58$, $p = 0.03$. respectively). Similarly, the right rectus femoris showed a smaller remaining percentage for the anaerobic test than for the VO_{2max} (82.5 ± 10.51% and 92.9 ± 8.85%; $F(1,5)= 12.60$, $p = 0.016$). The only muscle that showed a smaller remaining percentage for the VO_{2max} compared to the intense anaerobic test was the left vastus medialis (79.39 ± 11.26% and 88.28 ± 9.6%, respectively; $F(1,6)= 8.51$, $p = 0.027$). No statistically significant differences were found for the post-test heart rate, force production and MVC. **Conclusion.** Mean and median frequency sEMG data provided the best way to analyze muscle fatigue at the end of a maximal test. From these signals muscle fatigue at the end of a VO_{2max} test seems not to be maximal; there is a reserve of muscle fiber recruitment mainly from the slow-twitch fiber type.

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SIMBOLS / ABBREVIATIONS

<: less than

>: greater than

%: percent

± SD: standard deviation

^{o}C: Degrees Celsius

Ω: ohm

ADP: adenosine diphosphate

ATP: adenosine triphosphate

a-vO_2 difference: difference in oxygen concentration at the arterial and venous level

Ca^{2+}: calcium ion

Ca_{O2}: arterial oxygen content

cm: centimeter

CNS: central nervous system

CO_2: carbon dioxide

EKG: electrocardiography

EMG: electromyography

F_IO_2: fraction of inspired oxygen

H^+: hydrogen ion

HR: heart rate

Hz: Hertz

IEMG: integral electromyography

IRB: institutional review board

K^+: Potassium ion

km: kilometer

Mg^{2+}: Magnesium ion

mL/kg/min: milliliters O_2/kg body weight/minute

mmHg: millimeters of mercury

MVC: maximal voluntary contraction

NIR: near-infrared spectroscopy

O_2: Oxygen

PAR-Q: physical activity readiness questionnaire

PCr: phosphocreatine

Pi: inorganic phosphate

pH: hydrogen potential

Q: cardiac output

RER: respiratory exchange ratio

RF: rectus femoris

RMS: root mean square

RPE: rate of perceived exertion

RPM: revolutions per minute

sEMG: surface electromyography

SV: stroke volume

VCO_2: volume of carbon dioxide production

VE: minute ventilation

VL: vastus lateralis

VM: vastus medialis

VO_{2max}: maximal rate of oxygen consumption

CHAPTER 1

INTRODUCTION

The development of maximal physical ability has been one of the goals for athletes throughout history. In fact, the first known methodological process to increase performance during athletic events is related to the Greek culture (Koulori, 2010). This culture had sporting events that required ancient Greek athletes to train on a regular basis to develop stronger, faster, or more resistance trained muscle fibers that allowed them to compete in what were often grueling events. The winners were considered national heroes and semi-gods. Today, with the popularity of world-wide sporting events such as the summer and winter Olympic Games, soccer World Cup, world championships in athletics or the Tour de France, we have the opportunity to see elite athletes who are able to delay the fatigue process and push their bodies beyond normal human limits as they strive to achieve optimal athletic performance, win events, or break records.

Elite athletes train at the highest limits of their bodies, challenging the normal human state of physiological equilibrium or homeostasis. One reason athletes can achieve this level of performance is because of physiological adaptations that maintain muscle force production for longer periods of time while delaying muscle metabolite accumulation and acidosis. The decreasing force produced by contracting skeletal muscle during repeated contractions is a widely accepted definition of fatigue in an exercise and performance context (Fitts, 1994). Thus, highly trained athletes have physiological systems adaptations that delay the onset of fatigue, thereby improving performance.

Fatigue is one of the oldest concepts in the exercise physiology field. Brainbridge (1919) published an exercise physiology book in which he recognized that the origin of exercise-induced fatigue resides in the central nervous system. A. V. Hill´s work in the early years of the 20th century and his pioneering work on maximal aerobic capacity (VO_{2max}) built the foundation for the study and analysis of human physiological limitations (Basset & Howley, 2000). A more contemporary definition of fatigue is any exercise-induced reduction in the ability to exert muscle force or power, regardless if the task can be sustained (Bigland-Ritchie & Woods, 1984). Many physiologists believe that the fatigue response is a safety mechanism aimed at preventing injury or death during exercise (Abbis & Laursen, 2005). Reaching fatigue depends on factors within the central nervous system being able to balance motivation to continue exercise with the physiological ability to recruit motor units (Dalsgaard & Secher, 2007). Fatigue is divided into two important aspects: peripheral fatigue occurring distal to the point of nerve stimulation, and central fatigue resulting from a failure to activate the muscle voluntarily (Gandevia, 2001). Gandevia identified the contribution to central fatigue at the spinal level (motor properties; afferent signaling from muscle spindles, Golgi tendon organs and the muscles themselves) and at the supraspinal level (primarily motor cortex).

Liu and colleagues (2007) explained that the primary mechanism contributing to muscle fatigue seems to reside at sub-cortical levels of the neuromuscular system. Alternatively, Kayser (2003) mentioned that the motor cortex perceives the sense of effort, and that exercise is volitionally terminated when the sense of effort and other sensations such as pain become more intense than is tolerable. The connection between muscle fatigue during intense exercise may be explained by the accumulation of

metabolic by-products such as hydrogen ions (H^+), Pi, ADP, Mg^{2+}, or extracellular K^+, (Lamb, 2006) as well as an increase in core temperature. These changes activate group III and IV muscle afferent neurons that have an inhibitory influence on the alpha motor neurons; activation of these afferent neurons results in a reduced ability at the spinal level to recruit motor units (Gandevia, 1998; Hug *et al* 2003). Also, Thomas and Stephane (2008) reported a decrease in prefrontal cortical oxygenation just before motor failure during a progressive maximal exertion test; this supports the notion of a prefrontal cortex role in the decrease of efferent signaling to the muscles just at the end of exercise, when volitional exhaustion appears. Activation of the group III and IV muscle afferent neurons produces an inhibition at the neural command (alpha motor neurons) leading to task failure (Martin *et al.,* 2009).

More recently, an integrative view about the fatigue process has been proposed. Noakes (2008) has theorized that an anticipatory model can be used to explain how subjects, even before beginning their exercise, have already chosen a pace based on factors including their physiological capacity, the duration of the exercise period, and previous personal experience with the task. Noakes claimed that this strategy allows subjects to complete the exercise bout without a physiological failure due to energy depletion, dehydration or a dramatic increase in core temperature. This feedback mechanism between the afferent receptors and the central nervous system regulates the exercise intensity through the number of motor units recruited in the exercising limbs.

Fatigue may be explained as a task dependent multifactorial phenomenon for which the most common tool used to analyze it is the VO_{2max} test (Levine, 2008). VO_{2max} testing makes it possible to evaluate the ability of the heart and lungs to deliver oxygen to the

working muscles. According to Levine, this test represents a parametric measure of the cardiorespiratory capacity that allows athletes to develop a more compliant pericardium capable of providing an increased volume of blood to the working muscles. This increased compliance of the heart's chambers generates a higher stroke volume as explained by the Frank-Starling mechanism: the greater the stretch of the heart's chambers to accommodate blood during diastole, the more forceful the contraction and ejection of blood during systole (Levine *et al.*, 1991; Vella & Robergs, 2005). Levine explained that athletes stop exercising at VO_{2max} because of severe functional alteration at the muscle level due to convective oxygen (O_2) transport which may activate muscle afferent neurons thereby leading to the cessation of central motor and voluntary drive to continue. The relationship between O_2 transport limitation and motor performance failure during progressive maximal exercise testing is explained by Amann and Calbet (2008) as a progressive muscle de-oxygenation that may activate group III and IV muscle afferent neurons, thereby impairing peripheral ability to maintain adequate muscle contraction at exhaustion. It has also been observed that de-oxygenation of the prefrontal cortex occurs before the end of the VO_{2max} test suggesting cortical activation may modulate motor output at the point of voluntary exhaustion (Thomas & Stephane, 2008).

Skeletal muscle substantially increases energy demand from rest to exercise. Despite the large fluctuation in energy demand the concentration of muscle adenosine triphosphate (ATP) remains almost constant (Baker, McCormick, & Robergs, 2010). Muscle fatigue is traditionally related to the energy supply limitations such as glycogen and phosphocreatine (PCr) depletion or lactate accumulation during intense exercise (Sahlin, Tomkonogi, & Soderlun, 1998). A substantial increase in the concentration of

inorganic phosphate (Pi), a byproduct of excessive rates of ATP hydrolysis, may allow Pi to enter the sarcoplasmic reticulum and combine with calcium (Ca^{2+}) to form calcium phosphate. This leads to a decreased Ca^{2+} release from the sarcoplasmic reticulum and, subsequently, muscle contractile performance is adversely affected (Allen & Westerblad, 2001).

Maximal muscle fatigue appears when the ability to generate ATP through the anaerobic pathways (PCr and glycolytic systems) reaches its maximal capacity. During maximal muscle fatigue ATP concentration decreases to values just 20% below of the initial values (Spriet, *et al.*, 1987). Anaerobic capacity is defined as the maximal amount of ATP that can be resynthesized by anaerobic metabolism, that is mainly PCr hydrolysis and glycolysis (Noordhof, de Koning and Foster, 2010). Medbo and colleagues (1988) found that maximal anaerobic capacity occurs after 2 minutes of exhausting exercise at 100% or higher of VO_{2max}.

One way to analyze alterations in muscle performance, especially during muscle fatigue, is through the use of surface electromyography (sEMG) (Camic *et al.*, 2010; De Luca, 1985). This technique consists of strategically placing an electrode on the skin and over the muscle of interest. This electrode amplifies the electrical signal generated by the action potentials to produce muscle contraction. Changes in sEMG during muscle contraction can be observed and interpreted to explain the number of active motor units; motor unit force-twitch; mechanical interaction between muscle fibers; motor unit firing rate; number of detected motor units; amplitude, duration, and shape of the motor unit action potential; synchronization, or changes in the conduction velocity of muscle fibers (De Luca, 1997). From the parameters used to measure the frequency shift of the sEMG,

the mean and the median frequency are used to analyze muscle fiber type recruitment. Of these parameters, the median frequency seems to provide a reliable, consistent, and unbiased estimate of frequency spectrum modification that is related to the muscle fiber conduction velocity (De Luca, 1985, p. 201-222). The integral EMG (IEMG) and the root mean square (RMS) of the sEMG are used to analyze the extent of muscle fiber type recruitment during muscle contraction. sEMG signals have been correlated with neuromuscular fatigue, and this fatigue occurs around the ventilatory threshold; at this intensity a possible accumulation of (H^+) and potassium activate muscle afferent neurons in contracting muscles thereby generating the shift in sEMG pattern (Hug, *et al.*, 2003). Hug explained that the shift produces a decline in median frequency provoked by the enhanced recruitment of slow-firing motor units and/or a reduced recruitment of fast twitch motor units. Conversely, RMS or the IEMG of the sEMG signal during fatigue protocols shows an increase in the amplitude as a consequence of the increment in motor unit recruitment and/or increased motor unit discharge rate (MacDonald, 2008). Furthermore, Camic *et al.* (2010) defined the increment in the sEMG amplitude as being due to decrements in the pH that altered muscle contractility, and the decrement in the median frequency being due to the increased interstitial potassium (K^+). During exercise, the declining pH alters muscle contractility whereas the increasing K^+ reduces membrane excitability.

Problem Statement

Despite more than 80 years of research on the measure of VO_{2max} and the physiological determinants of this measure, a gap in the research literature remains regarding the extent to which exercised muscle is fatigued during maximal exertion

exercise. To analyze this extent, muscle fatigue during a VO_{2max} test was compared under two cycling test conditions using sEMG and custom developed force sensors placed on the pedals. Muscle fatigue developed during a VO_{2max} test will be compared to that developed during an intense anaerobic test. The muscle fatigue developed during an intense anaerobic test that produces maximal accumulated oxygen deficit, depletes PCr and, at the maximal capacity, stresses the glycolytic anaerobic pathway (Medbo, 1988, Noordhof *et al.*, 2010, Spriet *et al.* 1987). For this reason, we produced maximal muscle fatigue in 2 – 3 minutes working at intensity equivalent to 100% of previously determined peak watts ($Watts_{peak}$). Muscle fatigue was determined through sEMG activity and force applied to the pedals.

Purpose of the Study

The purpose of this study was to investigate if muscle is exhaustively taxed at the end of a VO_{2max} test. This study quantified skeletal muscle fatigue based on data from sEMG, heart rate and force applied to the pedals during and immediately after a VO_{2max} test as compared to an intense anaerobic test at 100% of peak watts at constant load and cadence (60 rpm).

Hypotheses

Three sets of hypotheses were tested; the first set compared sEMG results from the VO_{2max} and the intense anaerobic trial. The second set of hypotheses compared force production from the pre and post tests. The last hypothesis investigated heart rate responses under the two conditions. All of the hypotheses compare values acquired pre-test and immediately following the respective protocol. The post-test values for the

sEMG and force production hypotheses are normalized to the values obtained during the pre-test maximal voluntary contraction (MVC).

Hypothesis I

a. Mean sEMG frequency will be significantly higher (less fatigue) at the end of the VO_{2max} test compared to the intense anaerobic test.
b. Median sEMG frequency will be significantly higher at the end of the VO_{2max} test compared to the intense anaerobic test.
c. Root Mean Square (RMS) sEMG signals will be significantly lower at the end of the VO_{2max} test compared to the intense anaerobic test.

Rationale. The decline in mean and median sEMG frequency is caused by an enhanced recruitment of slow-firing, fatigue resistant motor neurons and a reduced recruitment of fast-twitch motor units (Hug 2003, *et al.*; Arabadzhiev, *et al.* 2010; MacDonald *et al.*, 2008). A higher recruitment of slow-firing motor neurons and a reduced recruitment of rapid firing motor units will be present as a consequence of the increased muscle fatigue during and after the intense anaerobic test compared to the VO_{2max} test (Rouffet and Hautier, 2008: Farina *et al.* 2004: Farina, 2006). Furthermore, due to an overriding central nervous system (CNS) constraint on motor unit recruitment near the end of the VO_{2max} test, peak RMS signals will be higher for the intense anaerobic test bout compared to the VO_{2max} test (Hug & Dorel, 2009; Tenant *et al.*, 2010).

Hypothesis II

a. Muscle force production, as measured by peak force applied to the pedals, will be significantly higher at the end of the anaerobic test compared to the values of the VO_{2max} test.
b. Muscle force production during maximal voluntary contractions (MVCs) post-test will be significantly higher after the VO_{2max} test compared to the MVC from the intense anaerobic test as compared to the respective initial values.

Rationale. Because of the consistently higher workload, greater ATP hydrolysis, and glycogen breakdown during the anaerobic bout of intense exercise as compared to the VO_{2max} test, there will be higher peak applied force during the intense anaerobic test (Laia *et al.*, 2009; Sahlin, Tonkonogi & Sunderlund, 1998; Krustup *et al.*, 2004). Similarly, this greater metabolic demand, and subsequently, lower muscle and blood pH will generate a greater activation signaling of group III and IV muscle afferent neurons (Amman, 2010). As a consequence the inhibitory influences on the alpha motor neurons will be higher and resulting in a greater decrease in the muscle MVC after the intense anaerobic test compared to the VO_{2max} test (Amann *et al.*, 2011; Gandevia, 2001).

Hypothesis III

Peak heart rate will be lower for the VO_{2max} compared to the intense anaerobic test.

Rationale. Because of the greater metabolic demand and subsequently lower muscle and blood pH of the intense anaerobic test, peripheral and central cues of exertion will be greater than during the VO_{2max} test. When the accumulated O_2 deficit is greater, metabolic byproduct concentration is higher causing a detrimental perturbation of the excitation-

contraction cycle in the skeletal muscle and myocardial cells as consequence of the inhibitory feedback from the central nervous system (Amann & Calbet, 2007). Additionally, because O_2 delivery depends on the cardiorespiratory system (Saltin, Calbet & Wagner, 2006) there will be a compensatory mechanism (higher heart rate) to maintain blood flow to the working muscles to satisfy O_2 needs during intense exercise.

Scope of the Study

This study was designed to quantify the extent of skeletal muscle fatigue at the end of a VO_{2max} test compared to an intense anaerobic test. Twelve men were recruited from the University of New Mexico and the surrounding communities. Potential subjects were screened prior to being included in the study. Exclusion criteria were defined as knee or ankle problems, VO_{2max} lower than 45 mL/kg/min, or any medical contraindication precluding maximal exertion cycling. The study required that each participant complete two VO_{2max} tests and one intense anaerobic test, with no more than 7 days between the second VO_{2max} test and the intense anaerobic test.

Assumptions

The following assumptions were made:

1. The participants in this study are representative of the population of adult men having a good aerobic fitness level, defined as a VO_{2max} greater than or equal to 45 mL/kg/min.
2. The health history and physical activity were honestly reported
3. All equipment was calibrated accurately before use.

4. The participants followed instructions regarding all pre-test guidelines prior to each testing session.
5. The laboratory environmental conditions during the tests in the lab were similar for all the participants.
6. Participants gave maximal effort during all tests.
7. Electrodes were placed over the same muscle area for all participants and trials.
8. Custom force transducers in the pedals accurately captured the force applied to the pedals during all trials.
9. sEMG accurately depicted RMS, mean and median frequency values from muscle activity.
10. Custom computer software accurately captured and computed the pedal force and cadence values.

Limitations

This study was subject to the following limitations:

No validity data are available for the custom LabView program used to capture pedaling force and cadence data. Because the sample consisted of healthy, aerobically-trained experienced male cyclists having a VO_{2max} no lower than 45 mL/kg/min, the results are only generalizable to this demographic. There was no direct measurement to quantify central fatigue, only the indirect measurements related to force production during pedaling.

Significance of the Study

In exercise physiology, the most common and widely method used to measure fatigue during exercise is the VO_{2max} test. Nevertheless, there are still some questions and controversies related to the level of muscle fatigue at the end of the test. Quantifying the level of muscle fatigue at the end of a VO_{2max} test will allow exercise physiologists to understand the level of muscle stress immediately prior to volitional exhaustion. If the muscle fatigue values from the VO_{2max} test are not comparable to those of an intense anaerobic test, then muscle fatigue, most likely, does not keep participants from continuing the VO_{2max} test. To our knowledge this is the first study to compare muscle fatigue at the end of a VO_{2max} test to an intense anaerobic exercise through analysis of sEMG signals, force production during pedaling, pre- and post-exercise MVC, and heart rate responses. If the anaerobic test generates lower remaining percentage values than those of the VO_{2max} test we should review the belief that a VO_{2max} test is representative of one's maximal physiological capability.

Definition of Terms

The terms in this study have been operationally defined as follows:

Adenosine triphosphate (ATP) The high-energy phosphate compound from which the body derives its energy.

Alpha motor neurons The largest neurons in the spinal cord. Their myelinated axons exit the spinal cord by the ventral roots and travel in peripheral nerves to innervate muscle fibers.

Central fatigue The failure to activate muscle voluntarily due to diminished central nervous system activation.

Fatigue Any exercise-induced reduction in the ability to exert muscle force or power, regardless if the task can be sustained.

Frank-Starling mechanism The mechanism by which an increased amount of blood in the ventricle of the heart causes a stronger ventricular contraction thereby increasing the amount of blood ejected.

Group III muscle afferent neurons Sensory receptors that increase the activation of the cerebral cortex in proportion to noxious stimuli (muscle stretch, touch, and contraction).

Group IV muscle afferent neurons Peripheral afferent nerve endings that respond to extracellular substances such as metabolic byproducts like pH, H^+, K^+ produced during fatiguing exercises.

Maximal aerobic capacity (VO_{2max}) The maximal rate of oxygen consumption by the body during maximal exertion.

Mean sEMG frequency Mathematical average of the spectral curve derived from surface electromyography.

Median sEMG frequency Divides the power density spectrum in two halves.

Muscle afferent neurons Peripheral afferent nerve endings that provide feedback from the locomotor muscles to the spinal cord and brain.

Near infrared spectroscopy A technique following physical principles of absorption of light used to measure hemoglobin saturation at the brain or muscle level.

Peripheral fatigue Reduced ability to exert muscle force or power due to factors within the cell or occurring distal to the point of nerve stimulation.

Prefrontal cortex Area of the brain involved in the processing fatigue-related feedback and/or adjustment of the descending command for the ongoing task.

Root Mean Square (RMS) RMS is a statistical technique used to determine the area under the rectified sEMG curve used to measure the amount of muscle fiber activity or recruitment during a giving task.

Sarcoplasmic reticulum A system of tubules that is associated with the myofibrils and stores of calcium for muscle contraction.

Stroke volume The amount of blood ejected from the left ventricle during a single contraction; the difference between the end-diastolic volume and the end systolic volume.

Surface electromyography (sEMG) A non-invasive technique to monitor and record the development of the myoelectrical signals. These signals are created by physiological alterations at the muscle fiber level. sEMG signals generate force development information related to the number of active motor units, motor unit fiber type, and motor unit firing rate.

CHAPTER 2

LITERATURE REVIEW

Definition of Fatigue and Early Efforts

Exercise-induced fatigue is a multifaceted phenomenon; it is task dependent and based on exercise mode, intensity, duration, environmental conditions, fitness level, health status, and nutrition. Fatigue has been investigated for more than 80 years but there are still gaps in our understanding of its determinants. New theories about the fatigue process have been developed. Although these theories include metabolic acidosis, potassium concentration [K^+], and oxygen availability, none have been fully accepted by the scientific community. The difficulty evaluating all possible variables in a single experiment led to this disagreement.

This review of literature adds additional data interpretation and commentary on the different aspects that may contribute to the fatigue process during incremental exercise thereby establishing the peak values seen at the end of the test for such measures as heart rate, ventilation and VO_2.

Exercise – Induced Fatigue

In an effort to understand the mechanisms underlying exercise – induced fatigue and limitations of the maximal rate of oxygen consumption (VO_{2max}), investigations into the contributory roles of central and peripheral factors have been reported. This section highlights these factors and how their independent and collective influences contribute to our current understanding of the cessation of maximal exertion aerobic exercise.

Multiple Levels of Fatigue

The most common method to evaluate physiological function, and, indirectly, the processes of fatigue is the incremental exercise test to exhaustion. This test measures VO_{2max}. Exercise physiologists use the VO_{2max} test to establish the lactate threshold, which represents the exercise intensity at which athletes' musculature starts relying more on the phosphate and glycolytic pathways to produce the ATP needed to produce muscle contraction. The test traditionally lasts between 8-12 minutes (Yoon, Kravitz, and Robergs, 2007). Among the variables often measured during the test are electromyography (EMG) signals, anaerobic threshold, exercise mode specific parameters (e.g. running speed, cycling power, cycling cadence), metabolic parameters and cardiorespiratory variables. In addition, such measures are also obtained with alterations in the fraction of inspired oxygen (F_IO_2) such as in hyperoxia, or hypoxic conditions. The researcher can also analyze the effects of heat during an incremental exercise test to establish the effects of hyperthermia on the increased central nervous system fatigue that produces alterations in muscle contraction during exercise.

In general, fatigue is any exercise-induced reduction in the ability to exert muscle force or power, regardless of the ability to sustain the task (Bigland-Ritchie, 1984). The term "fatigue" is used during voluntary exercise and is influenced by the contracting peripheral muscles, the respiratory muscles, arterial oxygen saturation, muscle perfusion, organs regulating fuel, metabolic and ionic homeostasis (McKeena & Hargreaves, 2008). A complete review of the physiological factors that contribute to the fatigue process during exercise performance is presented by Hargreaves (2008) who explains this process from the perspective of energy availability, calcium release from and reuptake into the

sarcoplasmic reticulum, alterations in oxygen partial pressure during hypoxia or hyperoxia, as well as the adverse effect that hyperthermia has on central motor drive to produce muscle contraction.

Central Fatigue

Central fatigue, which is defined by Gandevia (2001) as a progressive reduction in voluntary activation of muscle during exercise, is one of the biggest challenges to measure in humans. With advancements in technology, it is now possible to measure changes to the central nervous system during incremental exercise testing to determine what factors are controlling the continuation or cessation of exercise. Near-infrared spectroscopy (NIR) is a technique that works through absorption of light of different wavelengths by the tissues. Compounds such as hemoglobin change their absorption of light depending on the oxygenation status, thus detecting changes in cerebral oxygenation at specific areas of the brain (Owen-Reece, Smith, Elwell & Goldstone 1999). Thomas and Stephane (2008) tested the hypothesis that cerebral oxygenation would significantly drop before a failure in motor performance in highly trained cyclists. Cerebral oxygenation at the prefrontal cortex area increased from warm up to the second ventilatory threshold, and then decreased until the end of the test. At the muscle level, muscle oxygenation dropped from the beginning of the test to exhaustion, and during recovery cerebral oxygenation was higher than resting values. The authors concluded that there is a significant cerebral deoxygenation at the prefrontal cortex just before voluntary exhaustion, compared to the start of the exercise, causing a reduction in efferent signaling initiating the end of the exercise. Similarly, during acute hypoxia, cortical deoxygenation is faster than observed in a normoxic condition. The resting cerebral oxygenation was

lower during hypoxia than normoxia producing an accelerated inhibitory effect on effector signaling that decreases power output (Subudhi, Miramon, Granger, & Roach, 2009).

Psychological Factors

During exercise, physical fatigue is regulated by the motor cortex, balancing the inhibitory and excitatory processes. According to Noakes (2012), there is an anticipatory/feedforward system where the brain is receiving signals from all sensory inputs in order to sense the actual fuel state, core body temperature, and hydration status necessary to recruit the proper number of muscle fibers required by the impending task. This recruitment is regulated by biological, emotional, and mental fatigue as well as sleep factors in an anticipated manner even before the start of exercise.

The emotional aspect of fatigue could be analyzed by the rating of perceived exertion (RPE) created and validated by Borg (Borg, 1970). The RPE is a tool to associate the psychological aspect with the physiological variables during incremental or constant exercise. Joseph *et al.* (2008) analyzed the relationship among RPE increment, power output and covered distance. In the study, 10 well-trained recreational –level male cyclists participated. The experiment was conducted in two parts. In part 1, the cyclists performed three time trials of 2.5, 5.0 and 10.0 km in a random order. In part 2, the participants performed three experimental 5-km time trials (control and 2 hypoxic). The RPE was obtained at each 10% increment of the total distance in each time trial. The authors found that RPE increases according to the covered distance, irrespective of external factors, in this case the hypoxic conditions.

Motivation

Some authors question if peripheral or central fatigue causes exhaustion during incremental exercise testing. They support the hypothesis that exercise tolerance is determined by the perception of effort and motivation (Marcora and Staiano 2010; Trent, *et al*. 2008; Noakes, St. Clair Gibson and Lambert, 2005). Nevertheless, there is no disagreement that motivation to continue is a strong component during maximal exertion exercise testing. However, the motivation element has not been fully accepted as the most dominant factor for a person voluntarily ending the maximal effort task. Arguments against the motivation model are based on the difficulty in developing good research models to separate motivation for exercising at maximal intensities from the role of decreasing motor unit recruitment (Weir, Beck, Cramer & Housh, 2006).

One theory of why athletes stop exercising at VO_{2max} is based on the severe functional alteration at the muscle level due to convective O_2 transport; this may activate group III and IV muscle afferent neurons leading to the ending of voluntary contraction (Levine, 2008). Noakes (2008) outlined newer arguments against how VO_{2max} testing has been conducted by exercise physiologists and health professionals. Noakes argued that it is not possible to use a test to establish maximal capacity if the workload is externally predetermined. Noakes suggested that the subject should choose the work intensity during the test so the motor cortex determines the number of motor units to recruit for every muscle contraction.

Noakes, during the 1996 J.B. Wolffe Memorial Lecture, argued that the historical model developed by A. V. Hill about the limitation from the cardiovascular system to supply oxygen to the working muscles during maximal exercise is unproven (Noakes,

1997). Noakes explained that the oxygen consumption plateau phenomenon during incremental exercise until voluntary fatigue has failed to show scientific evidence, as the plateau is not present in all subjects. Instead, Noakes proposed a model regulated by neural, and chemical regulators to prevent organ failure or death during exercise, in health or disease condition in different environments. Basset and Howley (1997) critized Noakes' viewpoint for the lack of evidence to support his argument and concluded that the VO_{2max} is limited by the circulatory and/or the respiratory system, and added that the VO_2 plateau is not the main evidence for the cardiorespiratory limitation. Later, Noakes explained that if the brain is considered an unimportant factor during the test, then the cardiovascular system is assumed to be the limiting factor of a VO_{2max} test. Instead, Noakes proposed what he called the ¨central governor model¨. This is basically a feedback mechanism in which the brain receives constant information from the body during exercise to regulate energy and motor unit recruitment to avoid catastrophic events resulting from unanticipated loss of energy or a dramatic increase in core body temperature (Noakes, 2008). This theory has been criticized for the lack of background research or data with scientific validity to support it. For example, Brink-Elfegoun, Kaijser, Gustafsson, and Ekblom (2007) found that working at either $VO_{2max,}$ or 10% higher than VO_{2max} produced similar cardiac output (24.7 ± 3.2 and 25 ± 3.8 L/min, respectively), and heart rate (190 ± 5 and 191 ± 5 bpm, respectively); however, blood pressure was different for both work rates, (mean blood pressure 108 ± 1, and 117 ± 15mmHg, respectively. They concluded that the limitation of central circulation during the maximal work was not explained by a central nervous system inhibition.

With advancements in technology, it may now be possible to implement more sophisticated studies and analyze how the oxygenation and deoxygenation of the brain and working muscles are changing during an incremental exercise test. With the use of NIR it is possible to study the relationship between O_2 transport limitation and motor performance failure due to the inability to stimulate muscle fibers to generate movement during a progressive maximal exertion exercise test. This is explained by Thomas and Stephane (2008) as progressive muscle deoxygenation that may be directly responsible for an impaired peripheral ability to maintain adequate muscle contraction at exhaustion due to activation of group III and IV muscle afferent neurons. At the central level, a decrease in the prefrontal cortical oxygenation just before motor performance failure was observed during progressive maximal cycling exercise. Similar results were reported by Peltonen, et al. (2009) during incremental exercise in hypoxic and normoxic conditions. They found cerebral blood flow velocity to be similar in hypoxia and normoxia. But in hypoxia a greater cerebral deoxygenation was observed at a given work rate compared to normoxia. Interestingly, muscle deoxygenation was similar in both conditions, suggesting a tight regulation for muscle perfusion and oxygen delivery.

Peripheral Fatigue

Peripheral fatigue is, according to Taylor and Gandevia (2007), a reduction in force production occurring at or distal to the neuromuscular junction due to maximal or submaximal exercise. This peripheral fatigue can be demonstrated by a fall in the force produced by peripheral nerve stimulation.

Incremental exercise testing is also a good tool for evaluating changes in metabolic parameters due to training during hyperoxia compared with normoxia. During a six-week

exercise program nine participants were trained, in a random order, with high-intensity interval training in normoxia (room air equal to 21% O_2), and a six-week exercise program in hyperoxia (60% O_2) with an interval of six weeks of detraining between both programs. The results showed that even when in a hyperoxic environment, training power output was higher for approximately eight percent of power output compared to normoxic environment; endurance performance, VO_{2max}, or mitochondrial enzyme activity were not different for normoxia at the end of the six-week exercise program (Perry, Talantan, Helgenhauser, & Spriet, 2007).

On the other hand, Gore, et al. (1996) observed an increased arterial oxygen desaturation at as low as 580 meters above sea level in sea-level residents who were trained cyclists (VO_{2max} = 77 ± 1 mL/kg/min) compared with untrained cyclists (VO_{2max} = 51 ± 3 mL/kg/min). This suggests that athletes are more sensitive to a decrease in the partial pressure of inspired oxygen (P_{IO2}) because of a widened partial pressure gradient between the alveolar and arterial oxygen.

EMG Signals and Fatigue During Incremental Exercise

Surface EMG (sEMG) is a non-invasive method to study muscle fatigue. Three applications for the sEMG signals are the most important as an indicator of: (1) the initiation of the muscle activation, (2) the force generated by a muscle, and (3) the extent of fatigue occurring within a muscle (De Luca, 1997). Changes in the sEMG signals, i.e. slower action potential propagation velocity, are explained as altered metabolic states in the muscle; among these are a decrease in muscle pH and an increase in extracellular [K^+] (Shushakov, Stubbe, Peuckert, Endeward, & Maassen, 2007).

The most commonly analyzed sEMG data are the root mean square, mean power frequency, and median power frequency. Root mean square represents the signal power, or the amount of muscle fiber recruitment during a given task. The mean frequency represents the mathematical average of the spectral curve, and the median frequency divides the spectral signal into two equal parts, and is an indicator of muscle fatigue (Hug & Dorel, 2009). The median frequency is, according to De Luca (1997), the most reliable parameter to analyze muscle fatigue because it is less sensitive to static noise. Furthermore, in most cases, it is more sensitive to the biochemical and physiological changes that occur during isometric muscle contractions than is mean frequency, root mean square, or the integral EMG signal. Normalization of the sEMG data (percentage of its maximal MVC value from the cycle ergometer pre-test) is important to account for the influence of electrode placement relative to the innervation zone during an incremental exercise (Malek *et al*., 2006). Ventilatory threshold and sEMG signals have been correlated as corresponding parameters to detect fatigue during incremental exercise trials on a cycle ergometer (Hug *et al.,* 2003; Graef *et al*., 2008).

sEMG has been shown to be a useful tool to measure muscle fiber conduction velocity during cycling exercise and has been used to assess progressive motor unit recruitment during dynamic exercise (Farina *et al*., 2004; MacDonald, Farina & Marcora, 2008). During the non-fatigue state the motor unit recruitment in contracting muscle occurs in a random order and with independence from muscle activity in the same muscle to produce smooth movements. In contrast, when the muscle is reaching the fatigue state there is an increase in the motor unit synchronicity and dependence from different muscle fibers to maintain force production until volitional exhaustion (Naik, Kumar, Yadav,

Wheeler, & Arjunan, 2009). sEMG is also useful to measure the physical work capacity at the fatigue threshold, defined as the highest power output that can be sustained without evidence of neuromuscular fatigue (Camic *et al.*, 2010). A moderate correlation (r = 0.75) between ventilatory threshold (the deflection point where ventilation increases abruptly as consequence of CO_2 accumulation), and the sEMG fatigue threshold (the deflection point where the sEMG increases abruptly as consequence of an increase in motor unit recruitment).

Cardiovascular System and Fatigue

The first theory about the limitation of VO_{2max} relates to the cardiovascular system as outlined by Hill and Lupton's pioneering study in 1923. Physiological factors such as pulmonary diffusing capacity, cardiac output, oxygen carrying capacity, or peripheral oxygen diffusion have been reported as being limiting factors for VO_{2max} (Bassett & Howley, 2000). These factors have been recognized and analyzed during normoxia and under conditions such as decreased/increased oxygen availability, heat, and dehydration.

During the incremental exercise, reinfusion of red blood cells into athletes has produced a 5% increase in the VO_{2max} values at 24 hours and 7 days after the infusion. This increase in VO_{2max} remained after 16 weeks and allowed the athletes to train at a higher than normal intensity during the 16-week period (Buick *et al.* 1980). Buick and colleagues suggested that the transport of the oxygen to the muscles is a limiting factor of aerobic capacity.

Another way to increase oxygen availability is by increasing P_{IO2}. This is done by having participants breathe hyperoxic air. For example, in a study by Perry and

colleagues (2007), nine participants exercised in hyperoxic conditions (60% O_2) for six weeks three days/week, using high intensity interval training. The interval training involved four minutes of exercise at approximately 90% VO_{2max} with two minutes of recovery, for 10 repetitions. The authors reported an increase in skeletal muscle oxidative enzymes, VO_{2max}, and endurance performance, but this improvement was not different than training in normoxic conditions (21% O_2 concentration) (Perry *et al.*, 2007).

Similar results were obtained by Ekblom, Huot, Stein, and Thorstensson (1975). Their subjects exercised under hyperoxic (F_IO_2 = 0.50%) conditions during submaximal (30% VO_{2max} and 70% VO_{2max}) and maximal intensities; a 12.5% increase in the VO_{2max} value was observed and attributed to a 7.4% increase in arterial oxygen content (Ca_{O2}). No change in the maximal cardiac output was reported (Ekblom *et al.*, 1975). Indeed, when Ca_{O2} was reduced by carbon monoxide, maximal cardiac output was reduced by 6.1%.

According to Calbet *et al.* (2002), reduction in VO_{2max} during acute severe hypoxia is due a reduction of P_{IO2}, leading to a detrimental pulmonary gas exchange, and the subsequent reduction of maximal cardiac output. The authors also agreed that O_2 delivery is the limiting factor of VO_{2max} in normoxia and during hypoxia. During chronic adaptation to hypoxia a VO_{2max} increase is not substantially higher, since cardiac output is lower, and the redistribution of blood to the non-contracting muscles impaired the increment in VO_{2max} (Calbet *et al.*, 2003).

Heat stress is reported to produce a reduction in VO_{2max} mainly by reducing cardiac output and mean arterial pressure. These reductions decrease skeletal muscle blood flow,

oxygen delivery, and oxygen uptake when athletes started exercising at 1 °C higher core temperature compared to starting at their normal core temperature (approximately 37 °C) (Gonzalez-Alonso, & Calbet, 2003). Also, heat stress was shown to produce a higher muscle lactate accumulation and hydrolysis of ATP and ADP even when glycogen is not depleted. During prolonged pedaling exercise at approximately 60% of VO_{2max} 7 euhydrated, endurance-trained cyclists performed 2 cycling exercise trials in the heat (35 °C; 40-50% relative humidity) separated by one week. The first trial was in dehydration conditions (0.8 L of fluids) until volitional exhaustion. During the second trial water was provided (~4.3 L of fluids) for the same period of time as during trial 1 (135 ± 4 minutes); esophageal temperature was kept at 38 °C. The results showed that under conditions of dehydration and hyperthermia (39 ± 0.3 °C esophageal temperature), blood flow to active muscles decreased by approximately 13% in the dehydration trial compared to the control trial. This decreased blood flow to exercising muscles accounted for approximately two-thirds of the reduction in cardiac output (Gonzalez-Alonso, Calbet, & Nielsen, 1988). During submaximal exercise the heart increases stroke volume before heart rate. During dehydration, according to Ekblom (2006), the heart is not able to maintain stroke volume because of the reduced plasma volume; as a result, the declining ventricular filling volume becomes a critical factor that speeds up the fatigue process.

These changes in blood flow during heat stress, dehydration, and hypoxia are regulated by a neural mechanism that activates the α-adrenoreceptors in response to increments in the intracellular [Ca^{2+}]. This ionic change produces contraction of vascular muscle, increasing total peripheral resistance, thereby increasing cardiac output. The

increase in extracellular [K^+] also promotes smooth muscle hyperpolarization to produce a decrease in blood flow. At the same time, working skeletal muscles increase blood flow via the increase in stimulation of the β-adrenergic receptors to decrease local vascular resistance. These receptors are stimulated by the increase in temperature, [H^+], interstitial [K^+], [lactate] and other by-products of muscle metabolism that promote local vasodilation and an increased muscle blood flow (Thomas & Segal, 2004).

During incremental exercise testing, cardiac limitation is specifically related to ventricular dilation, compliance characteristics of the myocardium, and pericardial mechanical strain (Levine *et al.,* 1991). Levine and associates suggest that endurance athletes have a more efficient heart because of the increase in ventricular compliance that alters the heart's pressure-volume relationship. As a result, end diastolic volume and stroke volume increase generating a larger volume of blood flowing to exercising skeletal muscle. These athletes have developed adaptations in the contractile capacity of the cardiomyocyte by increasing the degree and the rates of shortening during systole and elongation during diastole, and by the improved ability to generate force (Wisløff, Ellingsen, & Kemi, 2009).

Efficiency

During pedaling there is an important aspect that may be beneficial or detrimental for those that are cycling; this aspect is efficiency. Efficiency can be defined as the ratio of work generated to the total metabolic energy cost, and two of the most influential variables are the cadence and work rate (Ettema & Wuttudal, 2009). Cycling efficiency not only involves kinetics, but also aspects of the muscle related to the agonist-antagonist activation or frictional resistances of joints, tendons or ligaments (Minneti, 2010). The

delta efficiency equation seems to be the most appropriate method to calculate efficiency where contractile properties of the muscle produce the variation in efficiency (Gaesser & Brooks, 1975) (equation 1).

Equation 1) $$Delta\ Efficiency = \frac{Delta\ work\ accomplished}{Delta\ energy\ expended} = \frac{\Delta W}{\Delta E} \times 100$$

The slow-twitch muscles are more efficient compared to the fast-twitch muscles, and this may explain the decrement in efficiency with the increase in cadence and work rate (Gaesser & Brooks, 1975). In this way, working at low (40-60 rpm) to moderate (60-80 rpm) cadences will produce the highest values in efficiency, especially in older cyclists (Morgensen, Bagger, Pedersen, Ferstrom, & Salhin, 2006).

Conclusions

The most common and widely used method to measure fatigue during exercise is the VO_{2max} test. The main purpose of this test is to develop progressive fatigue and analyze different variables that have been classified as being either from the central or peripheral systems. Some researchers support the idea that oxygen availability is the limiting factor to maintaining exercise. Another group supports the idea about a limitation at the heart, where factors such as stroke volume and myocardial compliance are limiting factors at a certain level of exercise intensity. Also, researchers are studying the level of importance that the central nervous system has at the end of the test, mainly by studying the energy depletion process or cerebral deoxygenation during intense exercise. In addition, it is possible to study fatigue and the relationship to interstitial $[K^+]$, metabolic acidosis, or elevated core temperature, pre-exercise, during, at the end and post-exercise. The question, "what is the limiting factor for volitional exhaustion during

exercise?" is still without a concrete answer. One of the confounding factors is that the human body works as an integral unit; it is affected as an entity. When any system is modified or altered, the other systems will also be affected somehow.

Fatigue is task-dependent and during certain activities one system can be more affected than other(s) thereby speeding up the fatigue process. With the technology currently available, it is now possible to develop more complex and complete research designs that allow analysis of multiple variables at the same time and during different conditions; the primary outcome is the ability to generate a more complete idea of how the fatigue process manifests at different systems and in response to different stimuli. The central nervous system, working muscles, participant motivation, as well as cognitive and volitional factors are important in the study and understanding of the exertional limitations to exercise testing.

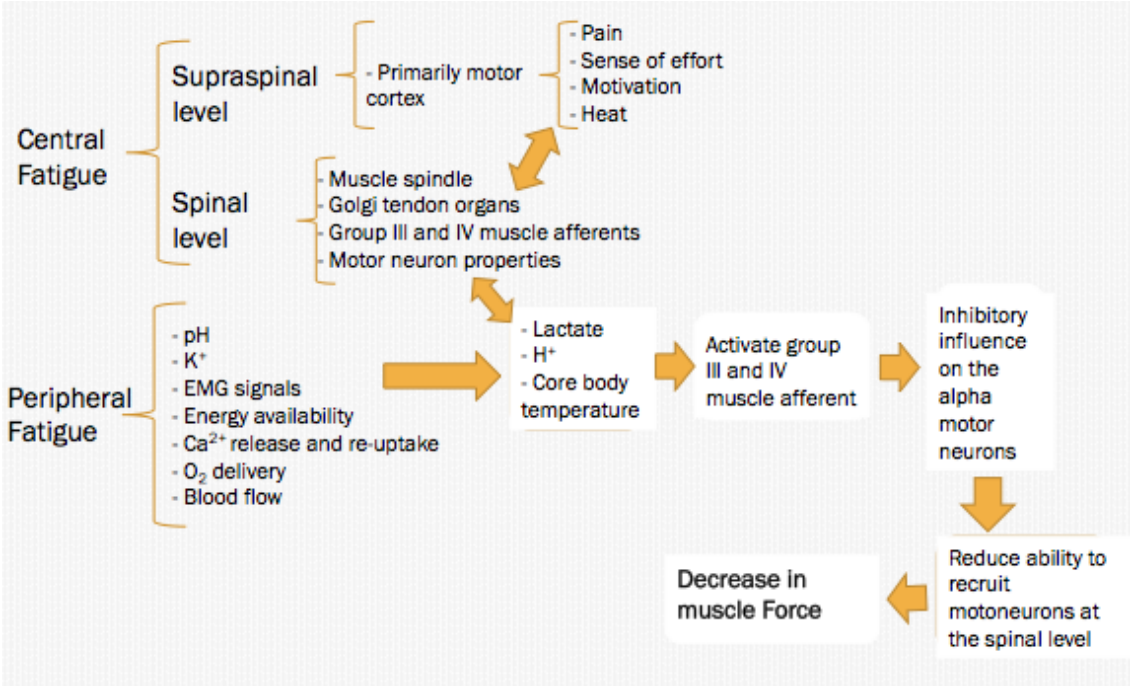


Figure 1. Factors that affect peripheral and central fatigue

From Figure 1 we can appreciate the different factors of the fatigue process until voluntary exhaustion during incremental exercise leading to voluntary exhaustion. Central fatigue is divided into spinal and supraspinal fatigue; supraspinal level is related to the primarily motor cortex. It is in charge of aspects such as pain, sense of effort, motivation, and perception of heat. The spinal level contribution to central fatigue is related to reflex systems and/or a biofeedback mechanism to maintain in a safe condition the body. This reflex system depends on receptors that are sending information about force production, muscle stretching, mechanical or biochemical changes in the muscle. These changes could increase the inhibitory influence on the alpha motor neurons, reducing the ability to recruit motor neurons, and finally generating a decrease in force production. Peripheral fatigue is related with events that happen at the muscle level and are related to changes in muscle metabolites, muscle electrical activity, and/or core temperature. Also, peripheral fatigue is related in how these factors generate the signaling to communicate and the responses that occur during the fatigue process. The level of fatigue depends heavily on the kind of test performed, incremental or continuous, and at maximal or submaximal intensity.

CHAPTER 3

METHODS

For the present study two tests were employed; one was the VO_{2max} test, and the other the intense anaerobic test. The former will generate information about the degree of peripheral fatigue at the end of the test. The latter will be used as the control test for the dependent variables (muscle force, heart rate, mean and median sEMG frequency, and sEMG RMS) at the end of an exercise condition known to fully tax the anaerobic energy systems. An intense anaerobic bout of around two or three minutes is, according to Medbo *et al.*, (1988), a reliable test to produce maximal muscle fatigue because it depletes the phosphocreatine (PCr) system and the anaerobic glycolytic pathway, generating maximal concentrations of inorganic phosphate (P_I) and both muscle and blood lactate. This anaerobic bout also results in the lowest blood pH values and largest decreases in force production as well as median frequency sEMG signals (Medbo, & Tabata, 1989: Baker, McCormick, & Robergs, 2010: Sahlin, Tonkonogi, & Söderlund 1998). In this way, the intense anaerobic test will serve as the criterion against which to evaluate the extent of muscle fatigue at the end of the incremental exercise to volitional exhaustion (VO_{2max} test).

This chapter is organized into five sections. Specifically, these are: a) setting, b) participants, c) procedures, d) research design, and e) statistical analysis.

Setting

All tests were performed in the Exercise Physiology Laboratory at the University of New Mexico. A single stationary cycle ergometer (Monark 825E Ergomedic, Varberg,

Sweden) was used for all cycling trials. To insure sEMG and force production wires were similarly extended over all trials, the footprint of the stationary bike was marked by tape on the floor.

Participants

For this study 12 healthy male participants were recruited. Prior to participation and after having given written informed consent and authorization for the use and disclosure of protected health information, the men were screened for cardiovascular and musculoskeletal diseases using a medical history questionnaire and the Physical Activity Readiness Questionnaire (PAR-Q) (Heyward, 2006). Inclusion criteria consisted of age (20 to 40 years), a VO_{2max} equal to or greater than 45 mL/kg/min and current involvement in aerobic training at least 3 days per week during the last year. Exclusion criteria included a risk stratification level of "moderate" (asymptomatic and two or more positive risk factors for cardiovascular disease) or "high" (symptomatic of or having known cardiopulmonary or metabolic disease) according to the guidelines of the American College of Sport Medicine (American College of Sport Medicine, 2010). Also, candidates with knee and/or ankle issues or lower body muscle problems that could affect the data output during the study were excluded. The study was reviewed and approved by the IRB committee from the University of New Mexico.

Procedures

Each participant performed three cycling trials. Each trial involved the calibration of all equipment and the collection of anthropometric data and metabolic gases. Before each trial, standing height (custom built stadiometer) and weight (SECA 884, Hamburg,

Germany) were measured for each participant; for this, the men were barefoot and wearing minimal clothes. Values were recorded in cm and kg, respectively.

The metabolic gas analyzers were calibrated with known concentrations of medically grade N_2, O_2, and CO_2 at the start of each test session. The flow turbine was calibrated using a 3-L syringe. Seat height was set according to the participant's leg length with the greater trochanter of the participant's femur used as the reference for seat height. After the participant's data were entered into the computer and the calibration completed, a mouthpiece connected to the Hans Rudolph breathing valve and integrated into the analyzer's mixing bag was placed into the participant's mouth. A nose clip was placed over the participant's nose to occlude the nostrils. Two minutes of resting basal O_2, and CO_2 were collected before the pedaling started. Breath-by-breath data were programmatically recorded for the duration of the test. Expired gases were analyzed via a custom software program (LabView, National Instruments, Austin, TX).

Heart rate (HR) was monitored during the tests via a commercial clinical electrocardiogram (GE Medical Systems, Wisconsin, USA) with a 5-electrode configuration (right leg, left leg, right arm, left arm, V5). The sites of electrode placement were prepped by shaving the chest hair as necessary and then removing debris and body oil by wiping the areas with an alcohol prep pad. All electrodes were firmly and properly attached to the participant's skin to help prevent EKG artifact during the tests. Heart rate was saved for later analysis using a GPS-enabled trainer (Garmin, Forerunner 305, Olathe, KC, USA).

VO_{2max} test

Two VO_{2max} tests were administered during the study. The first one was completed the day the participant was included into the study and served to familiarize the participant with the bike, equipment for metabolic gas collection during the tests (mouthpiece, headset), and cycling cadence of 60 rpm. At this time, participants were randomized for their subsequent testing sequence. The first VO_{2max} test also provided an initial determination of maximal aerobic capacity; the second test was used to compare the data with the intense anaerobic test.

For the VO_{2max} tests, the participants performed a graded exercise protocol on a mechanically braked cycle ergometer (Monark 825E Ergomedic, Varberg, Sweden); the power output was set to complete the test in 8 to 10 minutes according to each subject's fitness level. The same protocol was used for both VO_{2max} tests.

After two minutes of resting basal data collection with the participant seated on the bike, he started to pedal at 60 RPM against 13% of his previously determined $Watts_{peak}$. The first stage lasted two minutes; thereafter, workload was manually adjusted by adding 11% of the participant's $Watts_{peak}$ every minute until volitional exhaustion. A cadence of 60 RPM was maintained throughout the test. After the test was completed, the workload was decreased to one kg. The participant cooled down until his HR was less than 120 beats/min. Subsequently, all electrodes were removed and discarded. The first VO_{2max} test to obtain the $Watts_{peak}$ was performed 24 hours before the first of the randomized tests (either the second VO_{2max} test or the intense anaerobic test). At least 7 days separated the second VO_{2max} trial from the intense anaerobic test.

The data collected were then stored and exported into a Microsoft Excel (Office 2011 for Macintosh, Cupertino CA, USA) file. To remove variability from the breath-by-breath data, curves were preprocessed and smoothed by means of an 11-breath average. Thus, breath averaging was also applied to the oxygen consumption (VO_2), carbon dioxide production (VCO_2), minute ventilation (VE), and respiratory exchange ratio (RER). Heart rate (HR) was recorded at the end of every minute. sEMG and force production were measured during the entire test as described later. VO_{2max} was established as a RER = 1.0 and the highest 11-breath average and leveling of oxygen consumption despite an increase of workload.

Intense Anaerobic Test

According to the randomization of protocols, this test was scheduled for a different session at least 7 days before or after the second VO_{2max} test. The intense anaerobic test consisted of cycling on the same ergometer at 60 rpm and 100% of the initial $Watts_{peak}$. Expired gases were collected in the same manner as during the VO_{2max} test. During this intense anaerobic test, the participants were encouraged to pedal constantly until reaching volitional exhaustion.

Surface Electromyography (sEMG)

In order to lower skin impedance, the skin was prepared by shaving to remove leg hair and swabbing the area with an alcohol prep pad. Three pairs of silver surface gel electrodes (Vermed ECG Sensor Electrodes, Bellow Falls, VT USA) were placed on both legs at the midpoint of the muscle belly of the rectus femoris (RF), vastus medialis (VM), and vastus lateralis (VL). The ground electrode was placed on the medial condyle of tibia of the dominant leg. The Telemyo 2400T G2 system (Noraxon Inc., Scottdale Arizona)

was utilized to acquire the sEMG signals at a rate of 2500 Hz. Six bipolar channels were used at a sampling frequency of 1500 Hz. sEMG signals were recorded by telemetry throughout the test. During the tests an elastic netting (Walgreens, USA) was placed on both legs to reduce cable motion and associated noise in the sEMG signals. Raw data segments were obtained with a custom made program MATLAB 7.9 (Mathworks, Massacusetts, USA). The root mean square (RMS), mean power frequency (MPF), and median frequency (MF) were extracted from the selected sEMG samples using MATLAB in order to observe the tendencies over time (Figures 2, 3, and 4). Values were normalized to the pre-test maximal voluntary contraction (MVC) of the respective leg. Pre-test MVC was used as the 100% value for the sEMG signals, and from those values, every segment analyzed was compared with the reference value to observe tendencies for the RMS and for the mean and median signals.

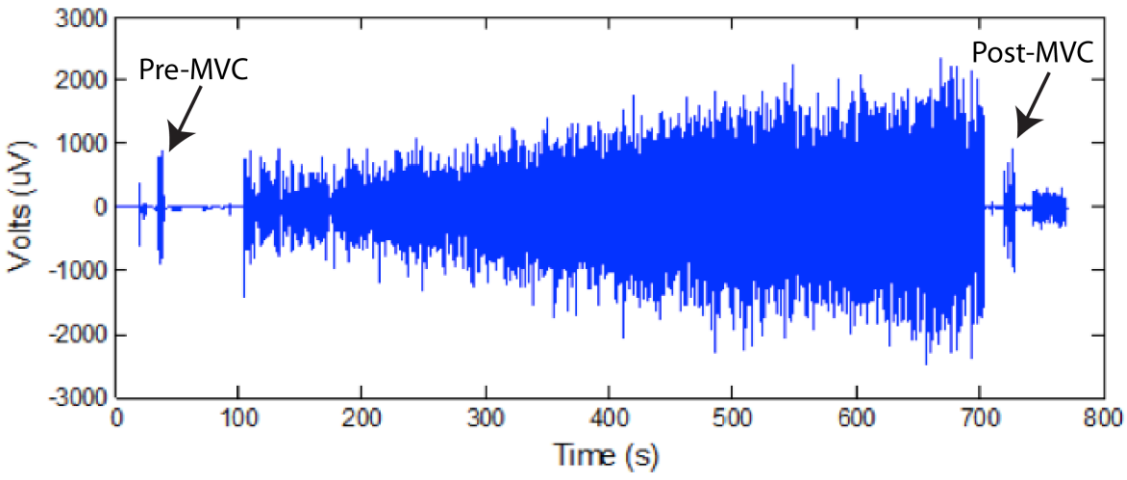


Figure 2. sEMG raw data from the VO_{2max} test from a single representative participant. Maximal voluntary contraction pre and post test are shown in the figure.

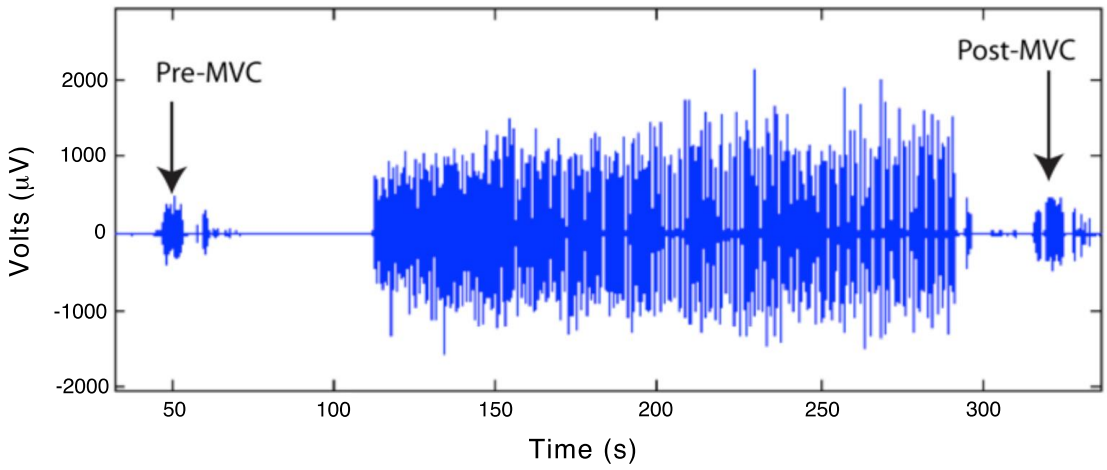


Figure 3. sEMG raw data from the intense anaerobic test from a single representative participant. MVC pre and post are shown in the figure.

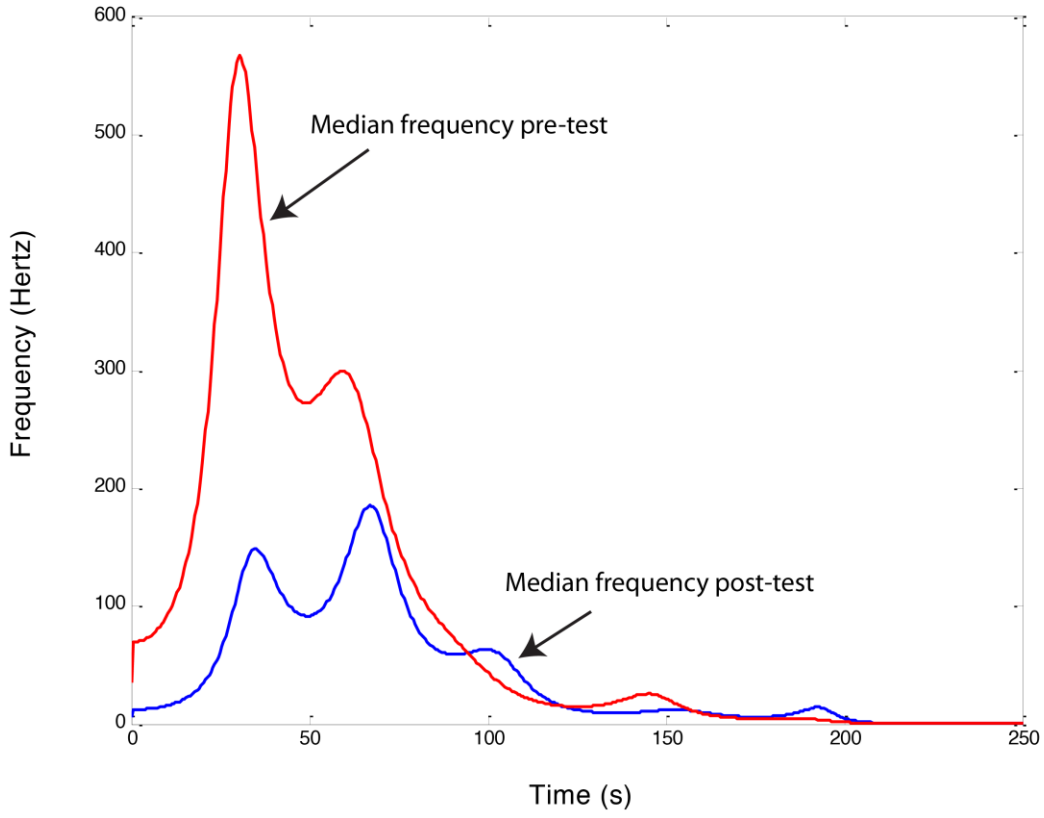


Figure 4. Frequency spectrum values pre and post MVC from a representative participant.

The RMS value of the sEMG signal was calculated over a running average window of one cycle of the specified fundamental frequency as follows in equation 2.

Equation 2) $$RMS\big(f(t)\big) = \sqrt{\frac{1}{T}\int_{t-T}^{t} f(t)^2}$$

where: f(t) represents the raw sEMG at time point t, and T is 1/(fundamental frequency).

IEMG is a time domain method which in mathematical terms is the area under the rectified sEMG curve; it was computed utilizing the following equation:

Equation 3) $$IEMG[X(t)] = \int_0^t |X(t)|\mathrm{dt},$$

where: X(t) represents the raw sEMG at time point t.

To rectify the sEMG, the absolute value of the raw sEMG data at time point t was derived. This gave the fully rectified sEMG values. Then the trapezoidal rule was used to compute the definite integral of the rectified sEMG as shown in equation 4.

Equation 4) $$\mathrm{IEMG} = \int_a^b \mathrm{f(x)\,dx} \approx (\mathrm{b-a})\frac{f(a)-f(b)}{2}$$

where a and b are the limits of the integral.

The frequency domain parameter, mean power frequency (MPF), was computed from the power density spectrum of the sEMG ($S_{sEMG}(f)$) which describes how the power of a signal x(t) is distributed over the different frequencies. This indicator of the central frequency value is described by the Harmonic Hjorth (Van Hese, Philips, De Konink, Van de Walle, & Lemahieu, 2001) parameters as shown in equation 5.

Equation 5) $$MPF = \frac{\int_0^f f S_{sEMG}(f)df}{\int_0^f S_{sEMG}(f)df}$$

Moreover, the median frequency (MF) indicator was used to compute the frequency value where the area described by the S_{sEMG} (f) curve was split into two exact halves as follows in equation 6.

Equation 6) $$\int_0^{MF} S_{sEMG}(f)df = \int_{MF}^{f} S_{sEMG(f)df}$$

Force Production Measurement

Force production applied to the pedals was measured using four flexiforce load sensors (Teskan, Inc. Boston, USA) within each pedal. For the mechanical coupling, the sensors were covered with small pads and placed between a metal plate and a plastic plate. The plates were bound to the pedals with screws (Figure 5).

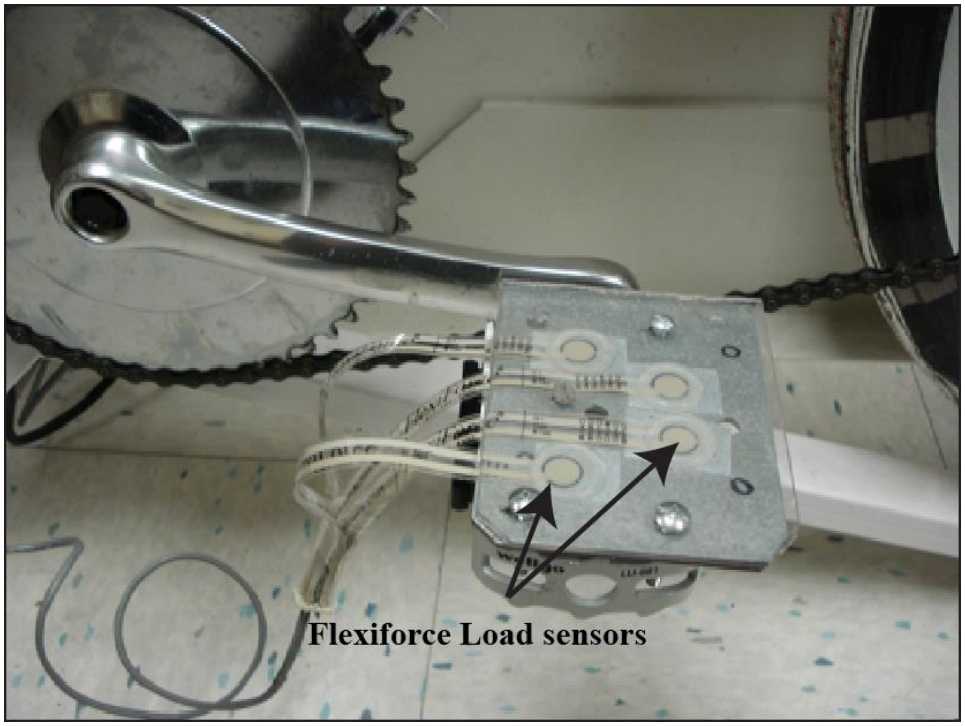


Figure 5. Flexiforce load sensor positioning on the pedals.

Each load sensor consisted of a piezoresistive device in which the resistance is inversely proportional to the force applied. In the unloaded state the force sensor has a resistance that is very high in Mohms (MΩ). When a force is applied to the sensor, this resistance decreases and is measured in kΩ. The inverse force-resistance relationship is described by equation 7.

Equation 7) $F = \alpha \frac{1}{R}$

where α is a constant of proportionality. Conductance (G) in ohms was related to resistance (R) in accordance with equation 8.

Equation 8) $G = \frac{1}{R}$

Thus, a linear force-conductance relationship with a constant of proportionality β was then given by equation 9.

Equation 9) $F = \beta G$

An amplifier-driver circuit was designed to convert the conductance from the sensors to a quantifiable voltage in a linear relationship; this voltage signal was transmitted through a data acquisition card to a custom LabView (NI LabView 2009, Austin USA) program.

The overall transfer function was determined through the multiplication of a scaling constant β, a reference voltage (V_{ref}), a resistance gain (R_{gain}) and the sum of all four conductance sensors (G_1 through G_4).

Equation 10) $F = \beta V_{ref} R_{gain} (G_1 + G_2 + G_3 + G_4)$

Verification of equation 10 was performed during the calibration process by applying five known weights (0, 20, 40, 60, and 80 kg) to the pedals and capturing the voltage output from each weight (sensor output *vs* force applied). A linear regression formula was derived from the five data points and used to calibrate the force production and obtain the scaling constant β.

To calculate the percentage remaining from pre-trial MVC the following equation vas used:

Equation 11) % of pre-test percentage remaining = (end of test value / pre-test value)*100

Maximal Voluntary Contraction

Bilateral maximal voluntary contractions (MVC) were measured before and immediately after each test. A car jack was used to stabilize the pedal crank arm. The knee was positioned at 60 degrees of flexion prior to the MVCs. Knee angle was determined via goniometry with the hip and ankle serving as reference points. Once the knee angle was determined and the leg placed in position, the participant generated a 5-sec MVC for each leg. The side sequence of MVCs was randomized.

Research Design

This is a quasi-experimental, pre-test post-test design to assess maximal muscular exertion requirements for a VO_{2max} test as compared to an intense anaerobic test.

Statistical analysis

A priori power estimation was performed using a freely available computer program (GPower, version 3.1.1, Heinrich-Heine-Universitat, Düsseldorf, Germany). To obtain a power value of 0.80 with alpha set at 0.05 and an effect size of 0.5, a total sample size of seven participants was required. To accommodate participant attrition during the study or less sensitive sEMG data, 12 subjects were recruited. Processed data were saved and imported into a graphic program (Prism, GraphPad 6.0, Software, San Diego, CA) for graphical representation.

A paired *t*-test was used to compare average values in sEMG, heart rate and force production pre-test and post-test between both trials. Data were analyzed using the software package SPSS (SPSS, 16.0, Chicago IL, USA) with significance set at $p<0.05$. Normality was tested using the Kolmogorov-Smirnov test; the dependent variables were normally distributed ($p>0.05$). The Levene's test was used for equality of the means ($p<0.05$).

CHAPTER 4

RESULTS

The results of this study are presented in the following sections: (a) descriptive characteristics of the sample, (b) sEMG signals during both tests (mean, median, and RMS) between the two tests, (c) heart rate behavior during both tests, (d) comparison of pedal force production between the two tests, and (e) maximal voluntary contraction pre- and post-test.

Descriptive Characteristics of the Sample

During the study two participants voluntarily withdrew due to time conflicts, thereby affecting the final number of participants. The physical characteristics of the sample are presented in Table 1.

Table 1. Characteristics of sample (N=10)

Variable	Mean ± SD	Minimum	Maximum
Age (years)	28.09 ± 6.55	22	39
Height (cm)	176.7 ± 2.97	173	182
Weight (kg)	73.31 ± 8.44	63	91.8
VO_{2max} (mL/kg/min)	60.93 ± 9.75	47.6	78.6

sEMG Signals During Both Tests

sEMG signals were normalized to the values from the pre-test isometric MVC. Pre-test MVC values were considered to be 100% and used as the reference values to

obtain the sEMG signal of the remaining percentage value of every post-test MVC. Data in this section represent the remaining percentage of the maximum. On occasion sEMG signal was lost during data collection. Consequently the degrees of freedom vary.

Mean Power Frequency Signals

Mean sEMG remaining percentage of pre-test MCV for the six muscles is shown in Table 2. There was a significant difference in the mean power frequency (MPF) remaining percentage of pre-test MCV for two of the six muscles monitored. The remaining percentage of pre-trial MVC of the left rectus femoris revealed that, at the end of the anaerobic test trial, it reached a lower remaining percentage of pre-trial MVC (84.53 ± 15.01%) than did the VO_{2max} test (89.66 ± 16.74%), $F(1,5)= 8.124$, $p = 0.036$. For the right vastus lateralis, a similar difference during post-testing was evident with the lower remaining percentage resulting from the intense anaerobic test as compared to the VO_{2max} test (81.92 ± 8.00% and 93.12 ± 4.21%, respectively); $F(1,6)= 11.47$, $p = 0.015$.

Table 2. Mean sEMG values by muscle and test

	VO_{2max}			Intense Anaerobic Test				
Muscle	pre-test MVC (Hz)	end of test (Hz)	% of pre-test value	pre-test MVC (Hz)	end of test (Hz)	% of pre-test value	% remaining comparison	p-value
Rectus Femoris								
Right	66.8 ± 7.8	58.6 ± 3.3	88.4 ± 7.8	70.1 ± 11.2	60.9 ± 5.2	88.9 ± 18.2	F(1,5)= 0.006	.94
Left	71.1 ± 5.0	63.4 ± 9.5	89.9 ± 16.7	71.9 ± 8.4	60.6 ± 12.2	84.5 ± 15.0	F(1,5)= 8.124	.03*
Vastus Lateralis								
Right	66.5 ± 7.7	59.7 ± 7.8	93.1 ± 4.2	72.9 ± 8.2	59.4 ± 6.9	81.9 ± 8.0	F(1,6)= 11.47	.01*
Left	65.6 ± 6.5	58.4 ± 5.3	87.3 ± 8.3	68.6 ± 6.1	58.3 ± 8.0	84.8 ± 6.2	F(1,5)= 1.57	.26
Vastus Medialis								
Right	67.8 ± 6.9	63.2 ± 7.1	94.3 ± 4.8	72.8 ± 20.6	64.56± 19.5	88.5 ± 7.3	F(1,5)= 2.99	.14
Left	64.9 ± 5.6	59.2 ± 5.3	93.5 ± 8.7	66.6 ± 4.9	60.8 ± 3.6	91.5 ± 4.5	F(1,5)= 0.185	.68

* = Significant difference, p<0. 05

% of pre-test = (End of test value / pre-test value)*100

Remaining % comparison made between % pre-test value VO_{2max} and % pre-test value intense anaerobic test

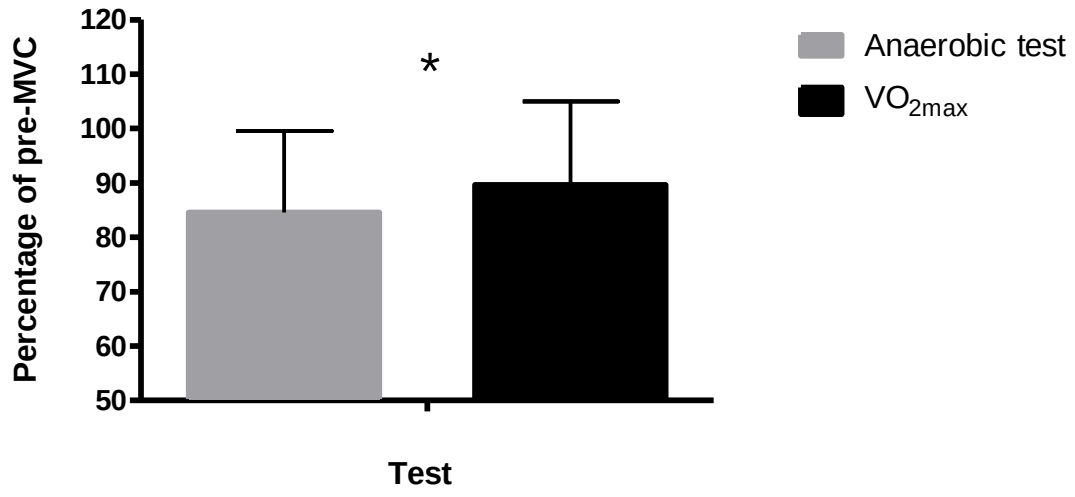


Figure 6. Mean frequency sEMG remaining percentage of pre-trial MVC in left rectus femoris. Asterisk indicates post-trial significant differences between both tests; p < . 05.

Remaining percentage (± SD) of baseline MVC mean frequency for the left rectus femoris during both tests is shown in Figure 6Figure 6. There were significant post-trials differences between the VO_{2max} test as compared to the anaerobic intense test. There were no differences noted for the right rectus femoris (Figure 7).

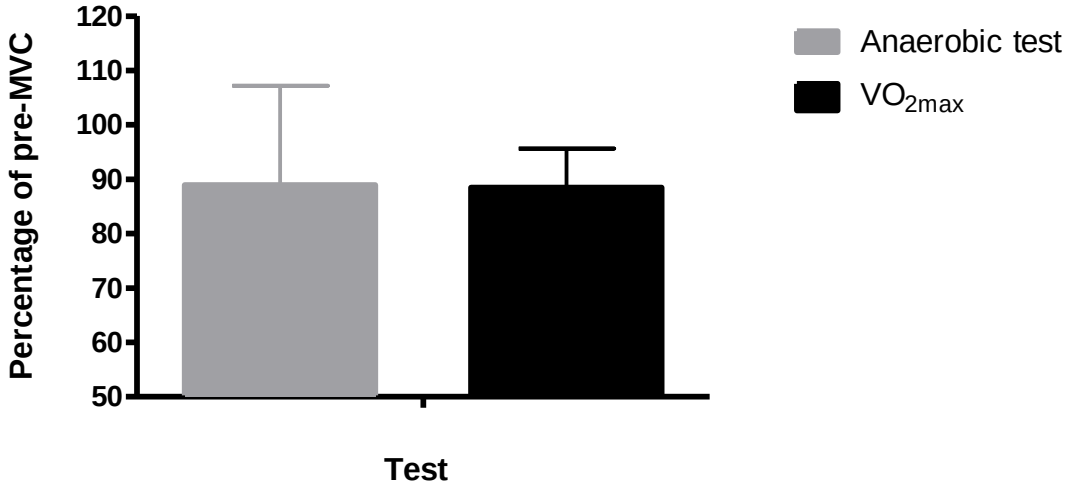


Figure 7. Mean frequency sEMG remaining percentage of pre-trial MVC in right rectus femoris.

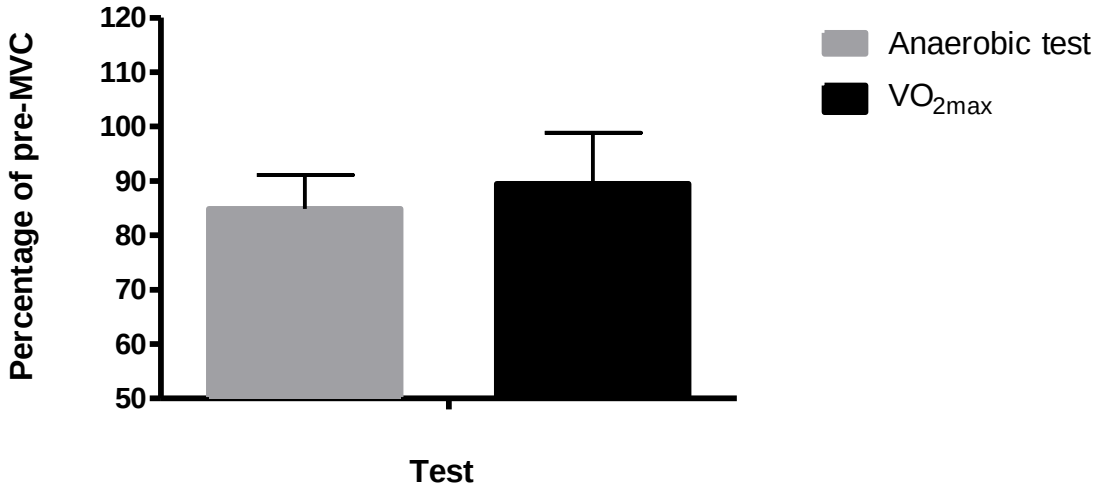


Figure 8. Mean frequency sEMG remaining percentage of pre-trial MVC in left vastus lateralis.

Changes in mean frequency for the left vastus lateralis (Figure 8) and right vastus lateralis (Figure 9) indicate that the only significant difference occurred between the VO_{2max} test and the intense anaerobic test on the right side.

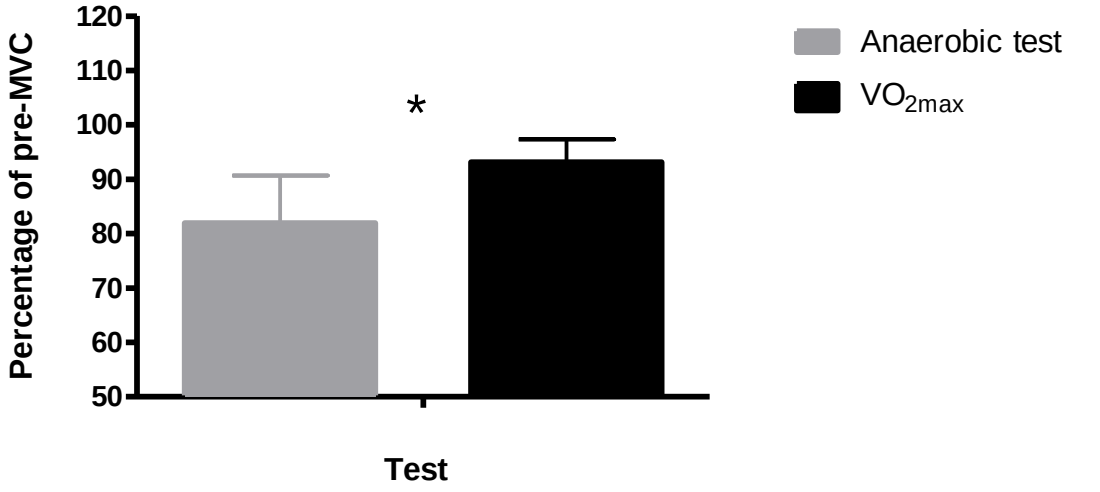


Figure 9. Mean frequency sEMG remaining percentage of pre-trial MVC in right vastus lateralis. Asterisk indicates post-trial significant differences between both tests; p < . 05.

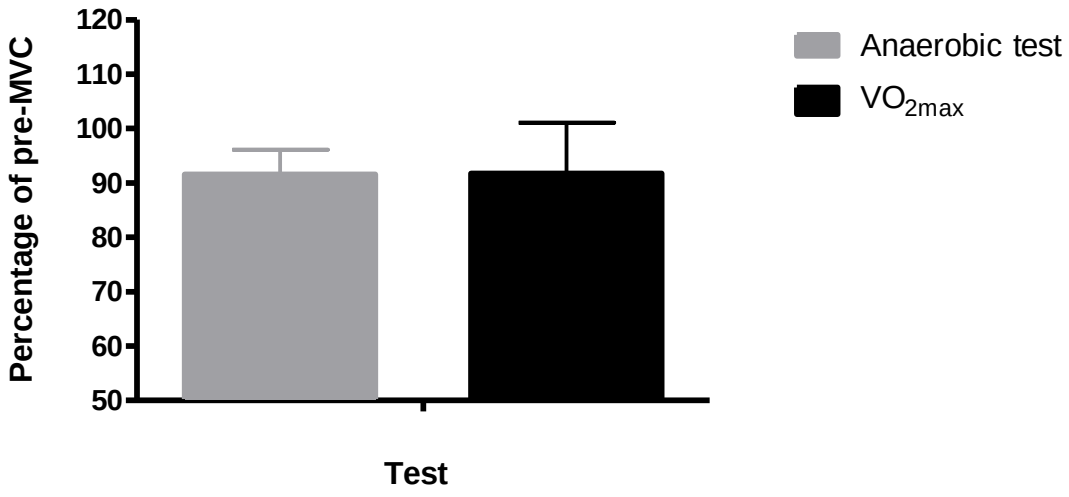


Figure 10. Mean frequency sEMG remaining percentage of pre-trial MVC in left vastus medialis

Changes of baseline MVC mean frequency for the left vastus medialis during both tests is shown in Figure 10, and for the right vastus medialis in Figure 11. There were no significant differences on either side between the VO_{2max} test and the intense anaerobic test.

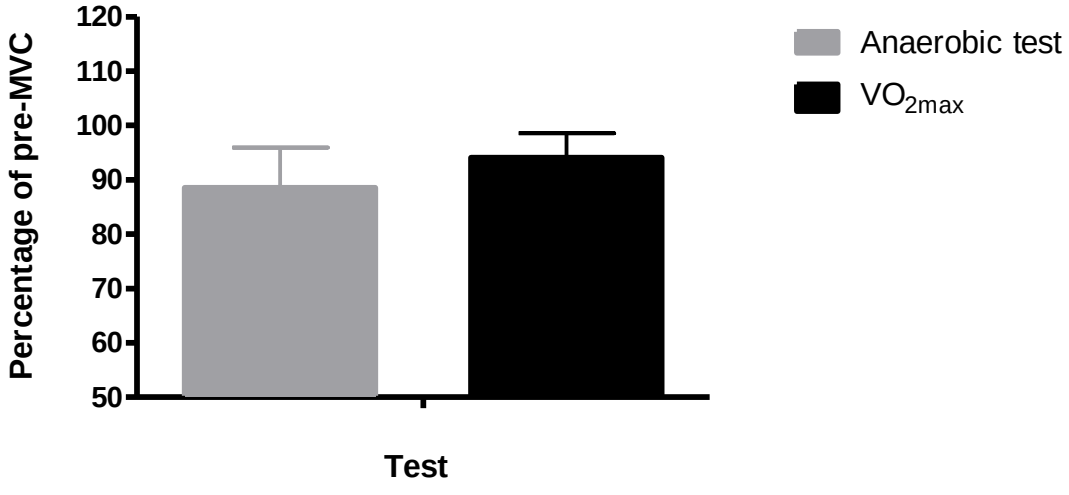


Figure 11. Mean freequency sEMG remaining percentage of pre-trial MVC in right vastus medialis.

Median Frequency Signal

Median frequency sEMG remaining percentage of pre-trial MVC data for the six muscles are shown in Table 3. There was a significant remaining percentage difference in the median sEMG frequency for three of the six muscles monitored. The right vastus lateralis retained less of its baseline. The remaining percentage was smaller (80.54 ± 12.02%) for the anaerobic test than for the VO_{2max} test (93.46 ± 8.78%); $F(1,6)= 7.58$, $p= 0.03$. Similarly, for the right rectus femoris the remaining percentage differences were smaller, hence retaining less of its baseline values (82.5 ± 10.51%) for the intense anaerobic test than for the VO_{2max} test (92.9 ± 8.85%); $F(1,5)= 12.60$, $p = 0.016$. Percentage change differences were also found to be significant for the left vastus medialis; however, for this muscle, the VO_{2max} test produced a smaller remaining percentage than did the intense anaerobic test (79.39 ± 11.26% and 88.28 ± 8.77%, respectively); $F(1,6)= 8.517$, $p = 0.027$.

Table 3. Median sEMG values by muscle and test

	VO_{2max}			Intense Anaerobic test				
Muscle	pre-test MVC (Hz)	end of test (Hz)	% of pre-test value	pre-test MVC (Hz)	end of test (Hz)	% of pre-test value	% remaining comparison	p-value
Rectus Femoris								
Right	58.6 ± 7.8	54.0 ± 3.1	92.9 ± 8.8	65.2 ± 7.6	53.6 ± 7.9	82.5 ± 10.5	F(1,5)= 12.6	.01*
Left	64.2 ± 6.3	57.2 ± 9.6	89.6 ± 15.4	71.8 ± 8.48	60.6 ± 12.2	86.7 ± 15.4	F(1,5)= 1.1	.33
Vastus Lateralis								
Right	58.7 ± 8.0	52.0 ± 9.1	93.4 ± 8.7	65.1 ± 8.44	52.2 ± 9.3	80.5 ± 13.1	F(1,6)= 7.8	.03*
Left	58.9 ± 6.2	52.1 ± 6.0	89.0 ± 10.9	63.2 ± 5.9	51.6 ± 7.4	81.6 ± 7.9	F(1,6)= 3.9	.09
Vastus Medialis								
Right	58.0 ± 8.4	55.2 ± 7.6	95.8 ± 10.7	64.6 ± 22.0	56.5 ± 20.0	87.5 ± 9.3	F(1,6)= 4.7	.73
Left	65.9 ± 18.2	51.0 ± 8.7	79.3 ± 11.2	58.4 ± 6.2	51.2 ± 4.4	88.2 ± 9.6	F(1,6)= 8.51	.02*

* = Significant difference, $p<0.05$

% of pre-test = (End of test value / pre-test value)*100

Remaining % comparison made between % pre-test value VO_{2max} and % pre-test value intense anaerobic test

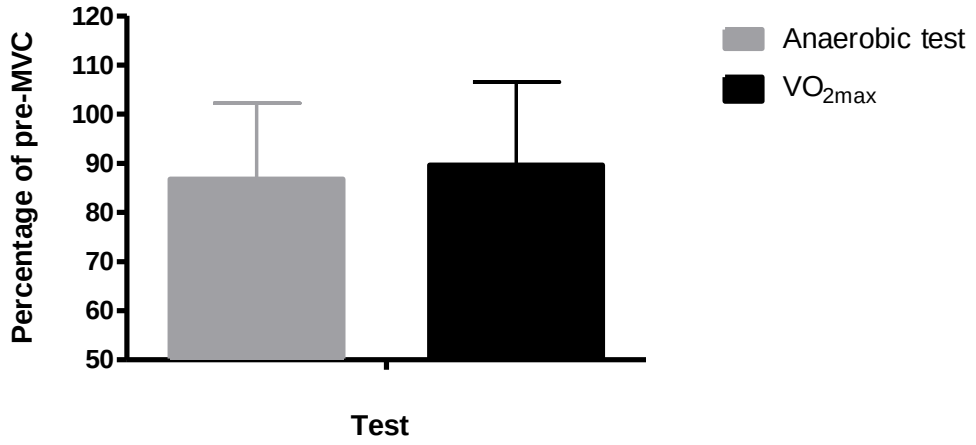


Figure 12. Median frequency sEMG remaining percentage of pre-trial MVC in left rectus femoris.

Remaining percentage for the left rectus femoris median frequency (Figure 12) and for the right rectus femoris (Figure 13) indicate that the only significant difference occurred for the right side rectus femoris. No differences were found for the left side of the rectus femoris.

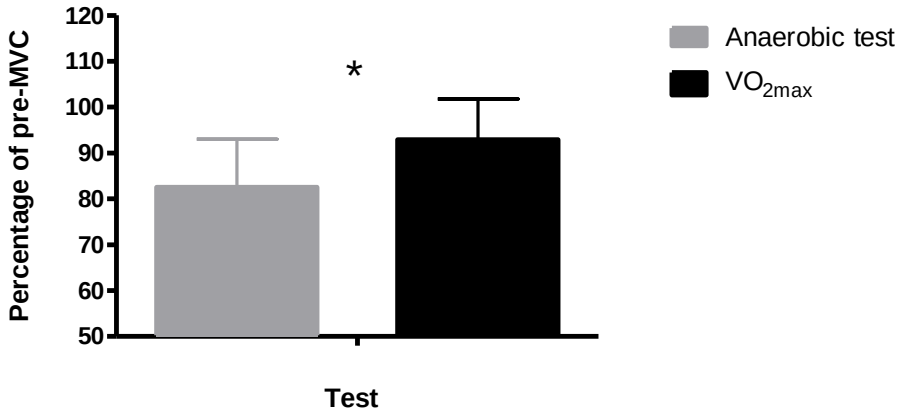
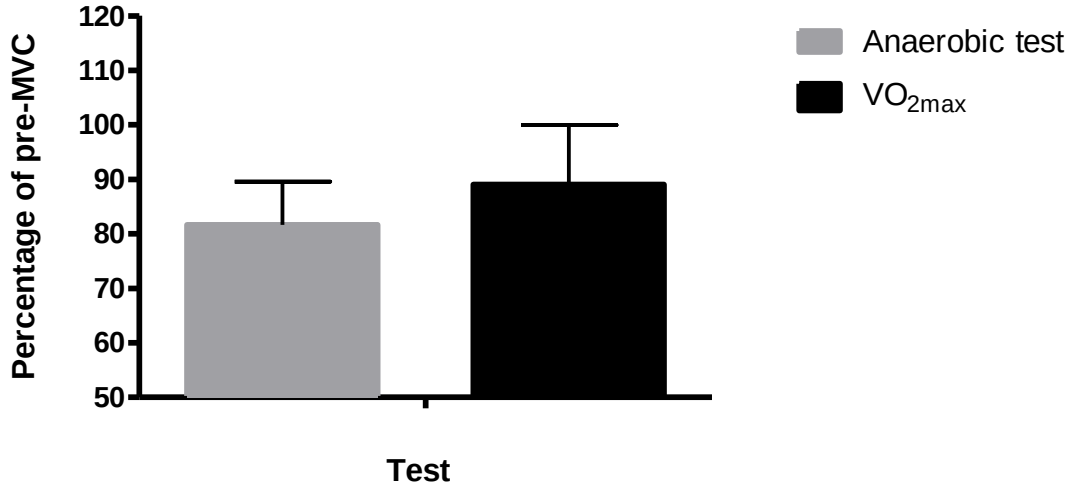


Figure 13. Median frequency sEMG remaining percentage of pre-trial MVC in right rectus femoris. Asterisk indicates post-trial significant differences between both tests; p < 05.



05.

Figure 14. Median frequency sEMG remaining percentage of pre-trial MVC in left vastus lateralis

Remaining percentage of pre-trial MVC (± SD) median frequency for the left vastus lateralis (Figure 14) and right vastus lateralis (Figure 15) indicate that the only significant difference occurred between the VO_{2max} test and the intense anaerobic test on the right side.

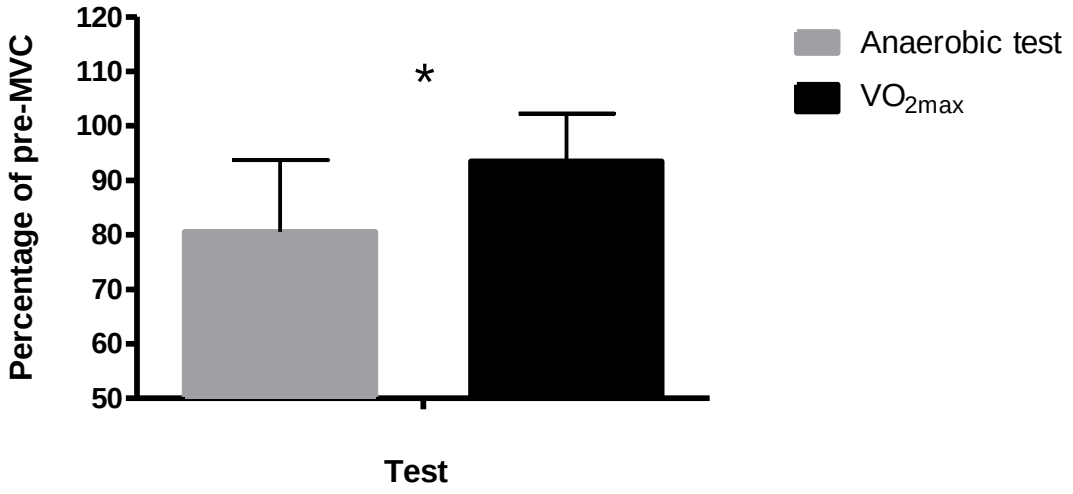
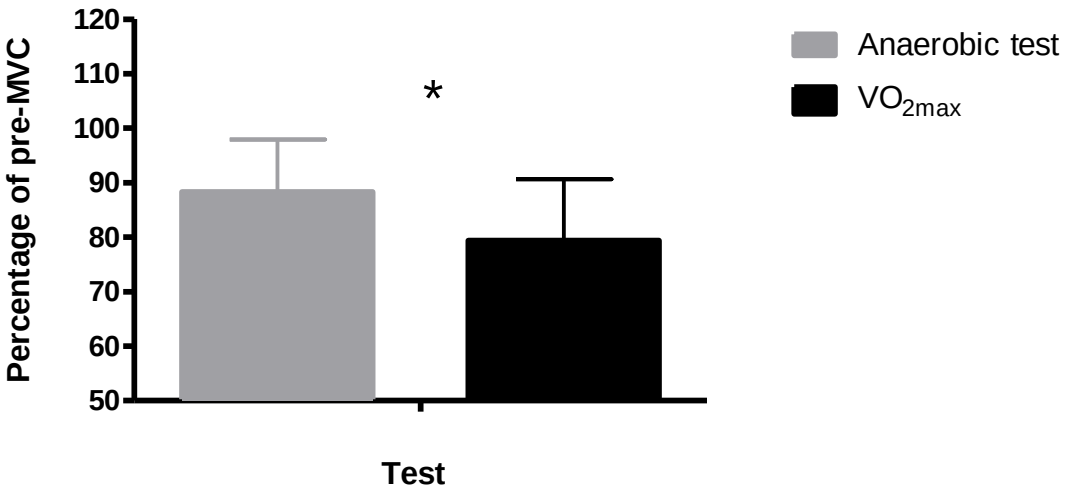


Figure 15. Median frequency sEMG remaining percentage of pre-trial MVC in right vastus lateralis. Asterisk indicates post-trial significant differences between both tests; p < 05.



05.

Figure 16. Median frequency sEMG remaining percentage of pre-trial MVC in left vastus medialis. Asterisk indicates post-trial significant differences between both tests; p < . 05.

Remaining percentage (± SD) of baseline MVC median frequency for the left vastus medialis during both tests was significantly different between the VO_{2max} test compared to the intense anaerobic test (Figure 16). There were no differences noted for

the right vastus medialis between the VO_{2max} test and the intense anaerobic test (Figure 17).

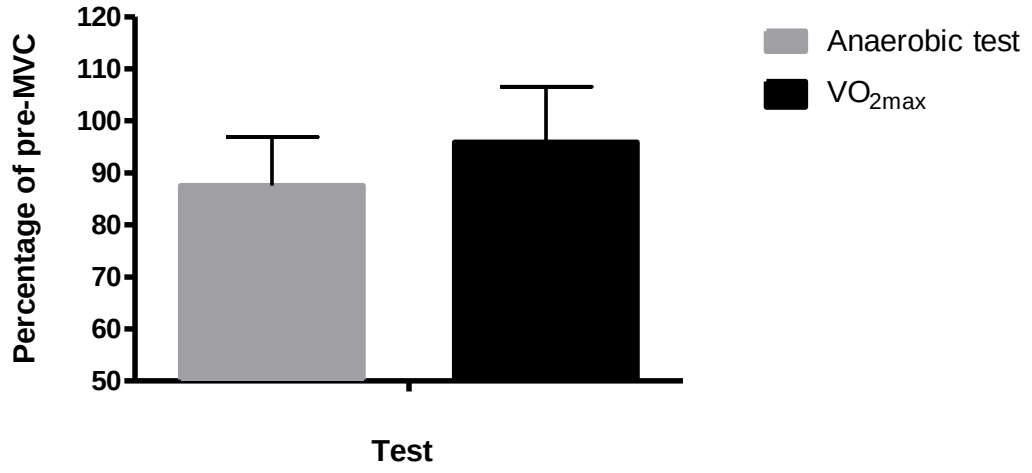


Figure 17. Median frequency sEMG remaining percentage of pre-trial MVC in right vastus medialis.

Root Mean Square (RMS) signal

No significant post-trial differences were found for the sEMG RMS for the left rectus femoris 238.29 ± 114.29% (Figure 18) or the right rectus femoris 250.50 ± 120.16% from pre-trial MVC (Figure 19). RMS showed an increment from pre-trial MVC for both tests and legs; however, these increments were not significantly different at the end of both tests for either rectus femoris.

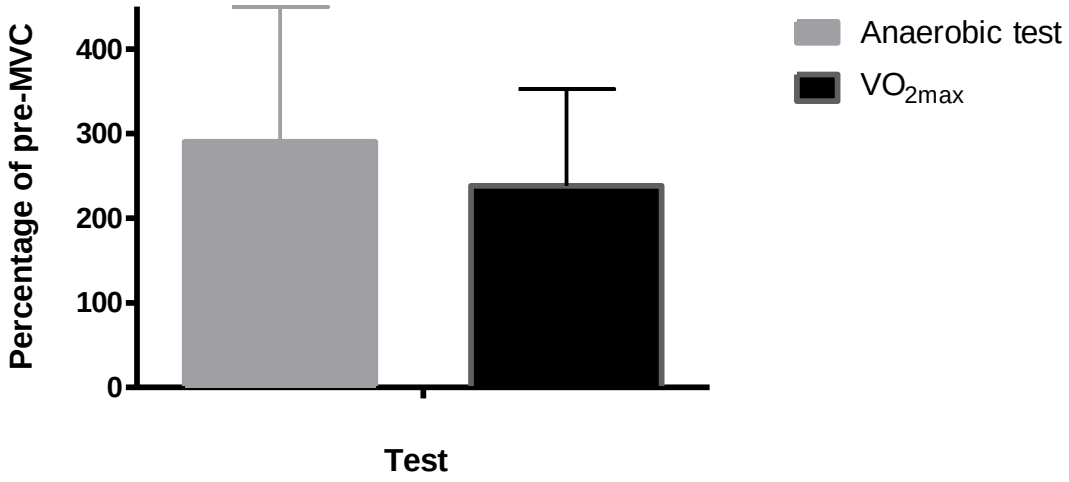


Figure 18. RMS remaining percentage of pre-trial MVC in left rectus femoris.

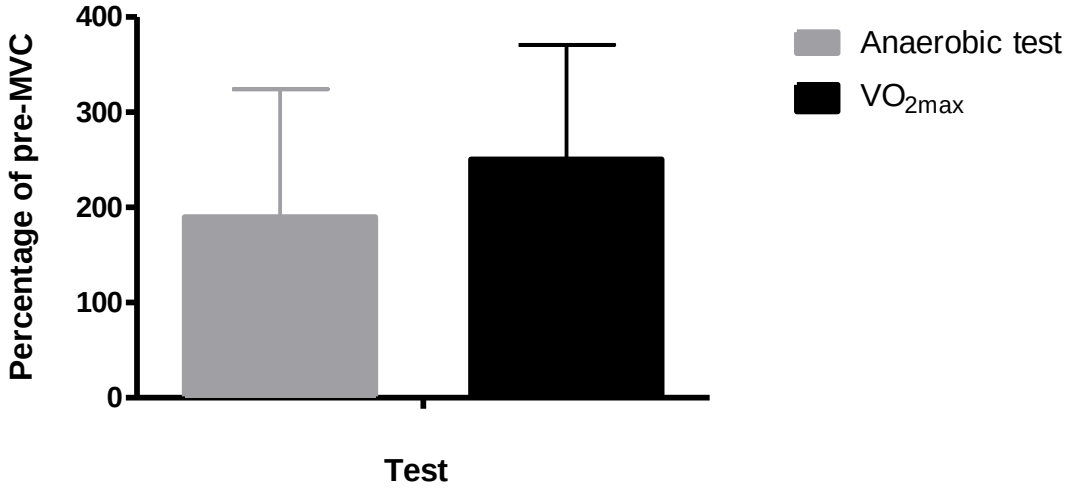


Figure 19. RMS remaining percentage of pre-trial MVC in right rectus femoris

No significant post-trial differences were found for the sEMG RMS for the left vastus lateralis (Figure 20) or right vastus lateralis (Figure 21).

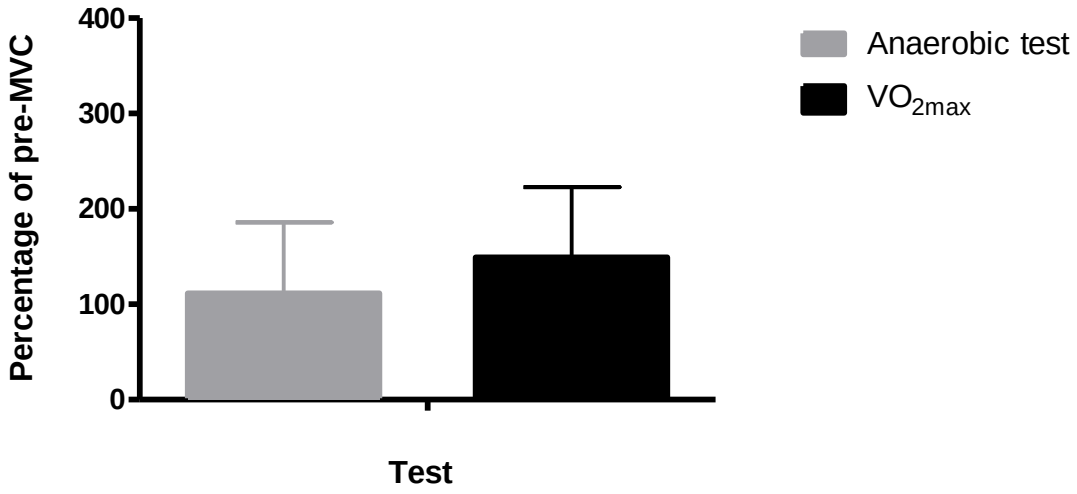


Figure 20. RMS remaining percentage of pre-trial MVC in left vastus lateralis

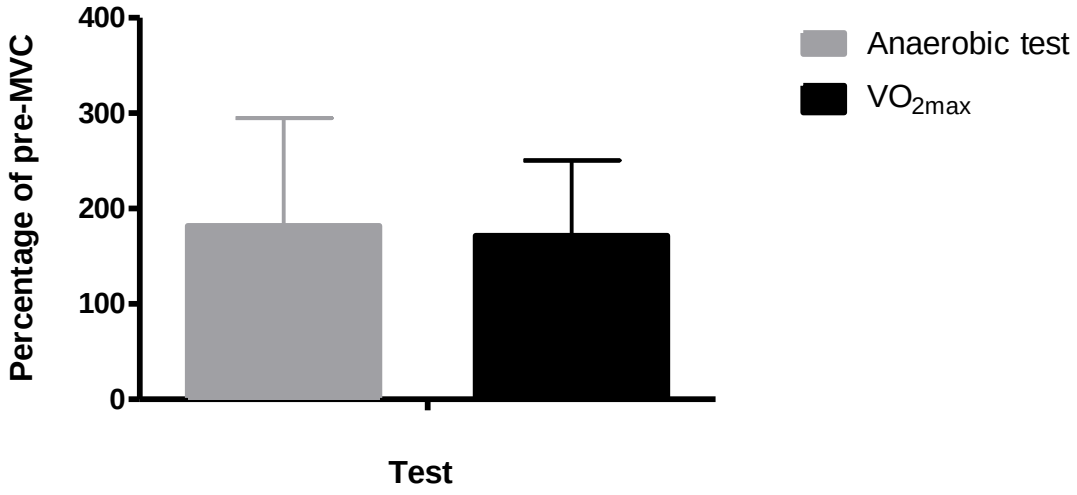


Figure 21. RMS remaining percentage of pre-trial MVC in right vastus lateralis.

No significant post-trial differences were found for the sEMG RMS for the left vastus medialis (Figure 22) or right vastus lateralis (Figure 23). The RMS increment for both legs in the vastus medialis muscle was not different.

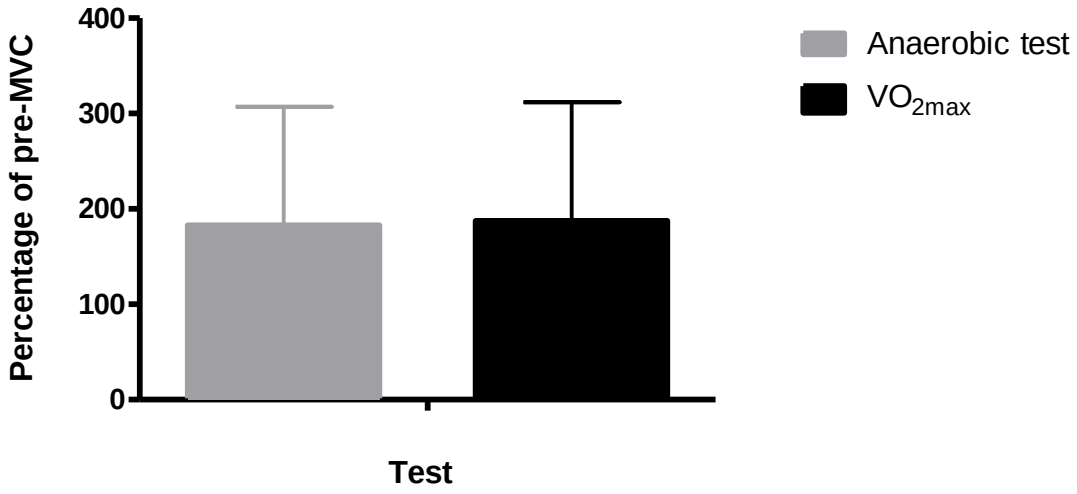


Figure 22. RMS remaining percentage of pre-trial MVC in left vastus medialis.

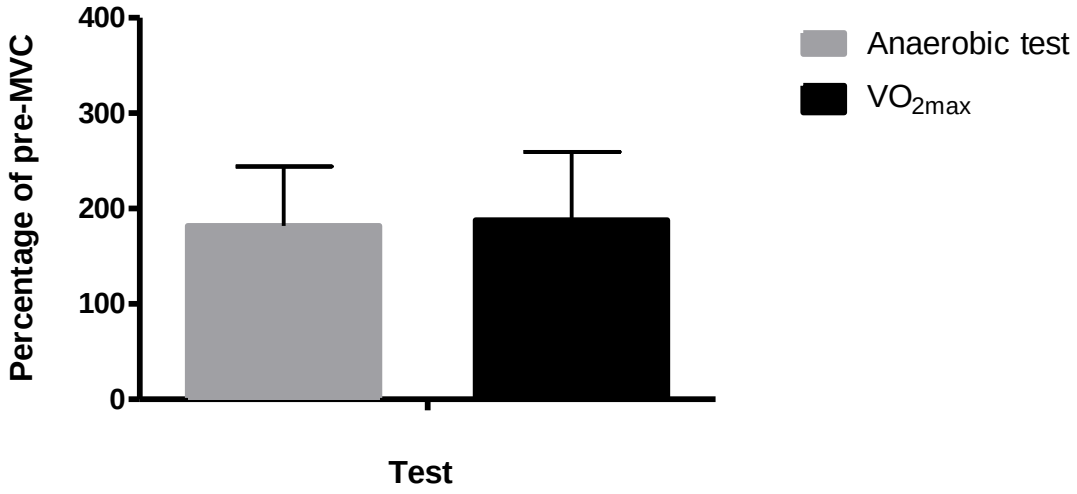


Figure 23. RMS remaining percentage of pre-trial MVC in rigth vastus medialis.

Heart Rate Behavior During Both Tests

The maximal heart rate attained at the end of the test was not significantly different between the two trials ($F(1,6) = 0.511$, $p = 0.51$). Although not significant, the

VO_{2max} test produced a higher maximal heart rate (173 ± 9 bpm) than did the anaerobic intense trial (170 ± 9 bpm).

Comparison of pedal force production between the two test

Maximal force production during pedaling (Figure 24 and Figure 25) was not significantly different ($F(1,7) = 0.830$, $p = 0.393$) at the end of the test for the left leg when comparing the VO_{2max} test and the intense anaerobic test (50.34 ± 28.95 kg and 119.0 ± 176.39 kg, respectively). Likewise, force production was similar for the right leg ($F(1,4) = 0.511$, $p = 0.514$) for the VO_{2max} test and intense anaerobic trial (44.93 ± 20.55 and 66.68 ± 48.70 kg, respectively).

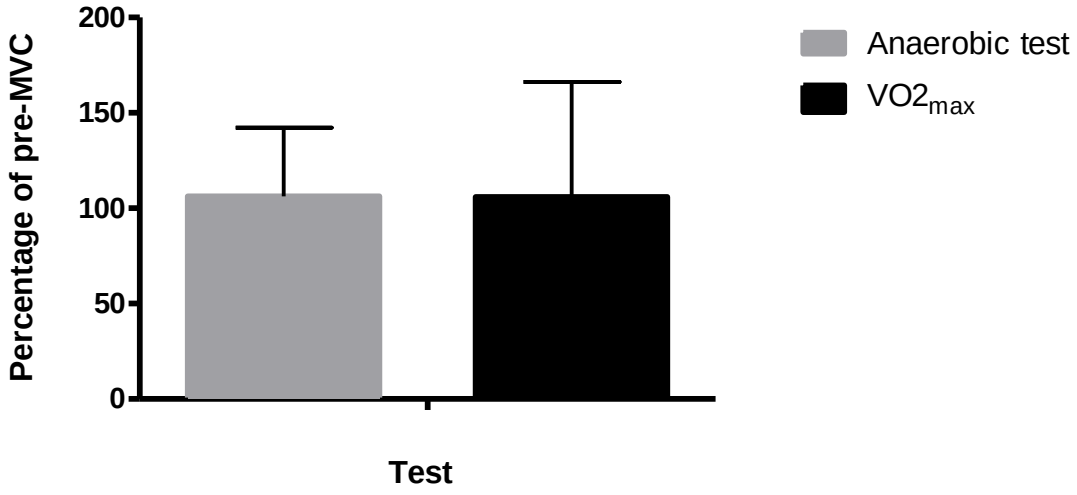


Figure 24. Force production remaining percentage of pre-trial MVC in the left leg.

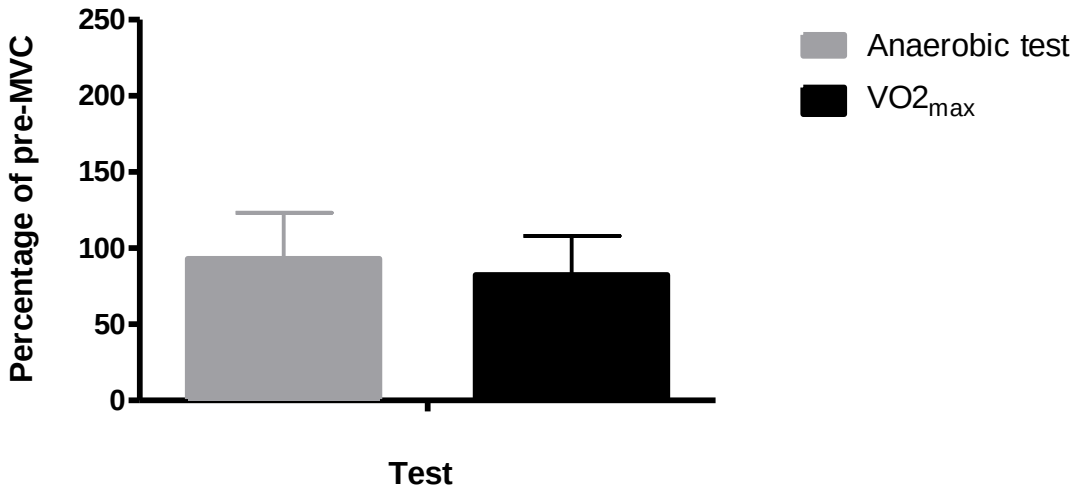


Figure 25. Force production remaining percentage of pre-trial MVC in the right leg.

Maximal Voluntary Contraction Pre and Post Test

There were no significant differences between trials for post-test maximal voluntary contraction. For the left leg ($F(1,7)= 1.44$, $p = 0.269$), the mean MVCs were 42.36 ± 19.7 kg and 50.16 ± 39.95 kg for the VO_{2max} and intense anaerobic trials, respectively. For the right leg, the mean values were 41.54 ± 18.39 kg and 50.18 ± 39.95 kg, respectively, for the VO_{2max} and the intense anaerobic trials (Figure 26 and Figure 27, respectively).

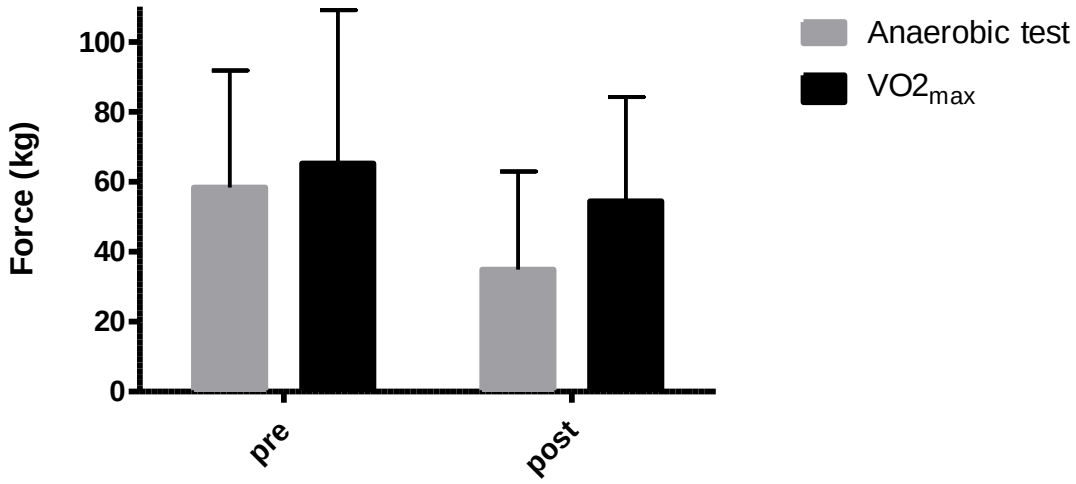


Figure 26. Pre and post test MVC values for left leg.

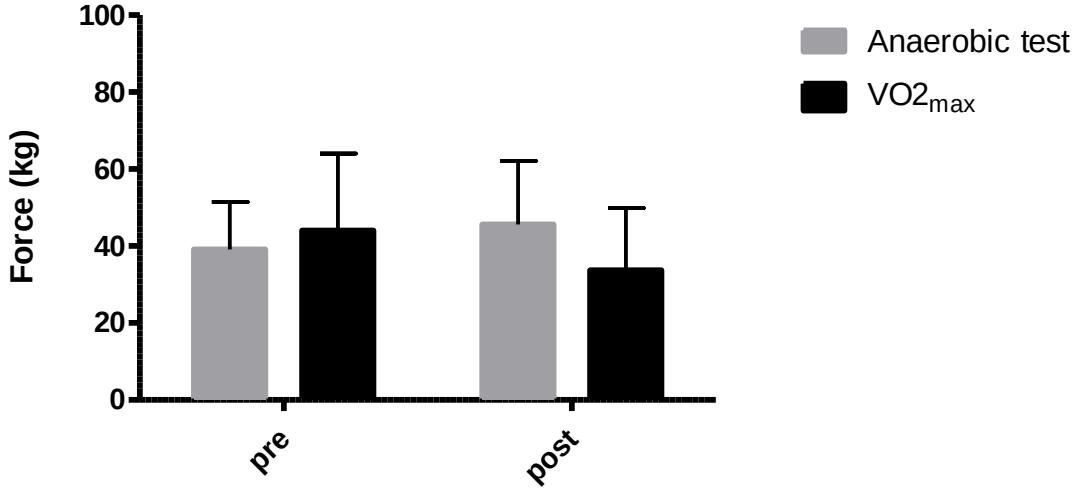


Figure 27. Pre and post test MVC values for right leg.

CHAPTER 5

DISCUSION

The discussion of the results is presented in the following sections: (1) rationale, (2) comparison of sEMG signals (mean, median frequency, and RMS) between the two tests, (3) heart rate values following both tests, (4) comparison in pedal force production between the two tests, (5) maximal voluntary contractions pre and post test, and (6) summary.

Rationale

The progressive maximal exertion test until volitional exhaustion has been used in the exercise physiology field as a tool to measure the subject's cardiovascular and muscular maximal capacity (Hawkins, Raven, Snell, Stray-Gundersen, & Levine, 2007). Currently, there is an increased interest to explore new factors that can affect the decision of the subjects to stop exercising at a certain point during the test. In our study we analyzed the level of muscle fatigue at the end of the VO_{2max} test as one possible factor influencing the decision to stop exercising at the end of the test. Groups of researchers are proposing that the central nervous system has a primarily role in the decision to continue exercising (Peltonen *et al.*, 2009; Kayser, 2003; Racinais, Girard, Micallef, & Perrey, 2007; Marcora, & Staiano, 2010); others are exploring the influence of the muscles in the biofeedback process with the central nervous system (Amman *et al.*, 2006; Amman *et al.*, 2011; Gandevia, 1998). We were not able to measure any factor related to the central nervous system in a direct way. The closest parameter was the force production at the pedals during the tests.

Comparison of sEMG signal data following both tests

sEMG has some advantages as an indicator in the fatigue process during intense exercise. Specifically, these are to produce information in real time; non-invasively capture data about the muscle fiber conduction velocity, frequency domain (mean and median power frequency); and monitor motor unit recruitment (RMS and IEMG). Usually, sEMG signals are normalized against an isometric MVC (IMVC), with the mean of the first minute of the workout, or with cycling torque velocity (Rouffet, & Hautier, 2008). For this study, the IMVC was used to normalize the sEMG signals for the tests. Muscle fatigue at the end of the VO_{2max} test was measured through the amplitude and frequency domains of the sEMG signals. The frequency spectrum provides insight into the type of muscle fibers that are being activated to accomplish the task. The frequency is measured through the mean and median power frequency values from the sEMG. According to De Luca (1985), frequency-domain indices represent a more reliable value, because these measurements are less sensitive to noise and are better able to explain the physiological and biochemical changes that occur during muscle contractions.

In the last two decades, the interest in understanding the phenomenon of fatigue during exercise has increased considerably. Even though fatigue is considered a multifactorial scenario, scientists have classified fatigue as being centrally or peripherally developed. Energy depletions, ionic, Pi, lactate, or H^+ concentrations may be considered as peripheral indicators of fatigue because all of them occur at the muscle level. Also, stroke volume, end-diastolic volume, end-systolic volume, oxygen partial pressure, and hemoglobin saturation may be considered factors that affect maximal capacity to do exercise.

Central fatigue is associated with factors related to the neuromuscular junction, motor cortex, afferent/efferent pathways, synapse processes and/or neurotransmitters. The psychological variable is considered a very important aspect to determine the voluntary exhaustion process and must be include as a qualitative assessment during fatigue studies.

Comparison of sEMG Signals

Cycling is often used as the modality for analyzing the amplitude and frequency signals. The combination of RMS amplitude domain, along with mean and median frequency domains estimate the degree of muscle activation and the development of muscle fatigue during incremental or continuous intense exercise (Farina, 2008). Knee extensor muscles are used as predictors of the muscle fatigue and prediction of coordination when fatigue is reaching a specific muscle group (Camata *et al.* 2011).

Mean Frequency

sEMG post-test mean frequency differences between the VO_{2max} and the intense anaerobic test were found for the left rectus femoris and for the right vastus lateralis. Conduction velocity at the end of both tests was slower for the intense anaerobic test as shown by the lower remaining percentage of pre-MVC. The mean frequency signal was not different for the vastus medialis muscle in both legs. According to Naik *et al.* (2009), during low-level muscle voluntary contraction mean and median frequency sEMG signals drop in a similar pattern but the coefficient of variability remains the same. Our Hypothesis 1 (a) about mean frequency reaching a lower significant remaining value at the end of the intense anaerobic test compared to the VO_{2max} test was supported for the left rectus femoris and the right vastus lateralis. Nevertheless, for the vastus medialis the

remaining percentage value was similar for both tests. From mean frequency results we found a muscle coordination effect; this is how the muscles work together in terms of relative timing and magnitude of their contractions (Wakeling, J.M., Blake, O.M., & Chan, H.K. 2010) to produce maximal power output at the end of both tests as observed by the differences in different muscles from each leg.

Median Frequency

sEMG median frequency detected post-test differences in three out of six muscles monitored. The median signal showed the same pattern as the mean frequency with slower conduction velocity at the end of the intense anaerobic exercise compared to the VO_{2max} test for the rectus femoris and vastus lateralis; this was the case only in the right leg. The left vastus medialis showed a slower conduction velocity at the end of the VO_{2max} test compared to the anaerobic test. Camata *et al*. (2011) found that when using supramaximal intensity the median frequency showed a significant decrement first for the rectus femoris, followed by the vastus lateralis and finally the vastus medialis. Our results were similar to Camata and colleagues' as we found this decrement pattern in median frequency at the end of the VO_{2max} test and the intense anaerobic test.

Post-trial differences were most consistently observed for the vastus lateralis and rectus femoris; we reported significant sEMG differences for both muscles for the right leg. This supports earlier explanations as to why, during pedaling trials, the vastus lateralis (Hug *et al.*, 2003; Farina *et al.*, 2004) and rectus femoris are the most commonly monitored muscles during fatigue studies (Hug & Dorel, 2009).

For the present study, it was proposed in hypothesis set I, that the mean and median frequency sEMG signals would be slower at the end of the intense anaerobic test as consequence of the enhanced recruitment of slow-firing fatigue resistant motor units, and a reduced recruitment of rapid-firing fatigable motor units. These hypotheses were confirmed in four out of five muscles monitored. Significant higher remaining percentage pre-trial MVC values were found for the VO_{2max} test compared to the intense anaerobic test for all but median frequency sEMG left of the vastus medialis. Carpes, Rossato, Faria, and Mota (2007a) found that during an incremental VO_{2max} test of 25 Watts every minute there was a reduction of peak crank torque asymmetry. They concluded that pedaling asymmetry decreases and tends to be non-statistically different at the highest exercise intensity of the VO_{2peak}. However, those authors only measured pedal force as the variable to identify asymmetry; they did not compare muscle coordination. From our finding we observed that at maximal power output, pedaling symmetry occurs. It is necessary to have a more complete analysis of muscle activity by adding the muscle fiber recruitment tendency and muscle coordination throughout sEMG analysis.

In regard to the RMS sEMG, no significant differences were found for any of the muscles studied. This is opposite of what was found by MacDonald, Farina, and Marcora, (2008). They reported significant RMS differences for the vastus medialis and vastus lateralis but no significant differences in the frequency domain. In contrast to our study, MacDonald and colleagues used 80% peak power over six minutes to induce significant muscle fatigue with a 28% reduction in peak power during 25-s maximal cycle effort. They found changes only for the RMS signal and not the median power frequency. Camata et al. (2011), using a supramaximal test at 110% of VO_{2peak}, found that after 50%

of total exercise time the rectus femoris RMS activity was significantly higher compared to the initial 5 seconds RMS (normalized RMS). The RMS activity for vastus lateralis and vastus medialis increased significantly just after the 75% of the total exercise time. Indeed, sEMG signal median frequency reached a lower value for the rectus femoris than for the vastus lateralis and vastus medialis. The authors concluded that the rectus femoris, compared to the vastus lateralis and vastus medialis, is the first muscle to exhibit fatigue as supported by the activity increment of the RMS signal at 50% of the exercise duration and the slower signal of the median frequency. In their study, the vastus lateralis exhibited a higher degree of fatigue than that did the vastus medialis. We found that the rectus femoris and the vastus lateralis exhibited a higher extent of muscle fatigue at the end of the tests than did the vastus medialis, as seen by the more frequently observed significant differences from pre-trial MVC.

In our study, the mean frequency signal sEMG showed significant post-trial differences for the left rectus femoris (Figure 6) and for the right vastus lateralis (Figure 9). The intense anaerobic test showed a significantly lower value at the end of the test compared to the VO_{2max} test. For the median frequency sEMG, significant post-trial differences were found for the right rectus femoris, right vastus lateralis, and left vastus medialis. The only muscle that reached a significantly lower median value for the VO_{2max} test compared to the anaerobic intense test was the left vastus medialis. This muscle behavior during cycling is explained by muscle coordination, where the fibers from different muscles are recruited in different timing and relative magnitude to generate maximal power output at the end of a maximal fatiguing test (Wakeling, Blake, & Chan, 2010). Indeed, people have lateral preferences that influence bilateral asymmetry; these

bilateral differences for cycling power may range from 5% to 20% (Carpes *et al.*, 2010). Peak crank torque asymmetry decreases during the highest exercise intensity (Carpes *et al.*, 2007a), but from our data, muscle coordination seems to play a more important role at the maximal intensity.

Significant differences were observed more often for the rectus femoris and at the vastus lateralis. This may be explained in part because the rectus femoris has more fast-twitch muscle fiber type than does the vastus lateralis, and vastus medialis (Leirdal, & Ettema, 2009). These significant differences found at the end of both tests suggests that the traditional VO_{2max} test, widely used in the exercise science field, does not fully tax muscle capacity at the end of the test. In this study, the intense anaerobic test was used as the control test, reaching maximal muscle fatigue, as seen by the greater decrement in the mean and median frequency power sEMG values. The only muscle that showed a significant median power frequency decrement for the VO_{2max} test was the left vastus medialis; this is a smaller muscle, recruited mainly at the last degrees of knee extension, and with more slow-twitch muscle fiber type when compared to the rectus femoris and vastus lateralis.

RMS Comparisons

RMS showed no significant differences at the end of the two tests. This is in opposition to previous studies using RMS to analyze muscle fatigue during incremental exercise (MacDonald, Farina, & Marcora, 2008). Nevertheless, there were higher post-test RMS sEMG values for both tests in our study, which is a classical behavior for sEMG amplitude compared to initial values (Dimitrov, Arabadzhiev, Hogrel, &

Dimitrova, 2008). Our hypothesis related to RMS sEMG signals was not supported by our findings; no muscle showed any significant difference at the end of both tests.

Heart Rate

Post-trial heart rate was not statistically different between the two tests. A possible explanation is that heart rate is not a sensitive measurement of acute adaptation to the two trial protocols. If the Fick (1870) equation: (VO_2 = Q x a-vO_2 difference, and Q = SV * HR) is used, then, SV may be a more sensitive parameter for analyzing differences produced by fatigue during intense exercise. SV during exercise initially rises linearly, reaching a plateau at around the 40-50% of the VO_{2max} for sedentary and athletes; then at the end of the exercise, a decrement in the SV value is observed when heart rate is at its maximal rate (Vella & Robergs, 2005). When Q is compromised during intense exercise, there is a SV decrement that is, in part, overcome by increments in heart rate. Heart rate was not a sensitive indicator of fatigue during intense exercise. In our study, although not significant, the VO_{2max} test produced a higher maximal heart rate (173 ± 9 bpm) than did the intense anaerobic trial (170 ± 9 bpm). These results do not support hypothesis III that indicated the VO_{2max} test would produce a lower peak heart rate. It appears that if there are no alterations in convective O_2 delivery, such as in a hypoxic environment (Amman & Calbet, 2007) or during severe heat stress (Gonzalez-Alonso & Calbet, 2003), maximal heart rate is similar and independent of the protocol used to generate maximal voluntary exhaustion.

Pedal Force

Force generated while pedaling depends on various factors such as pedaling rate, muscle coordination, workload output, and cycling efficiency. In the present study we found no significant differences in the force production at the pedals when comparing both legs at the end of the two tests. Our hypothesis II relating to pedal force applied to the pedals was not supported by the data. We found no significant differences in decrement from pre-trial MVC at the end of the VO_{2max} and the intense anaerobic tests. These results are opposite to the sEMG results for which significant post-trial differences were found for some muscles. However, this can be explained by the fact that at the beginning of the tests pedaling asymmetry has been shown to be significant and progressive. Furthermore, when the workload is increased there is an increment of pedaling symmetry (Carpes *et al* 2010; Carpes *et al*. 2007). The fatigue process has been reported to produce a shift in muscle coordination patterns due to inhibitory pathways that result in recruitment symmetry of muscle fibers at the end of the two tests (Carpes, Mota, & Faria, 2010). Indeed, according to Amman and Calbet (2007), MVC or force production was similar for both tests because the intense anaerobic test had a duration less than 4 minutes for all participants, thereby relying mainly in the anaerobic system to sustain the force production and MVC post-trial.

Our cadence was set at 60 rpm for all participants. Foss and Hallen (2004) found in elite road cyclists that 60 rpm was the most economical cadence at low workloads (0, 50, and 125 W); at intermediate workloads (200 and 270 W), the most economical cadence was 60 to 80 rpm. At the highest workload (350 W) the most economical cadence was 80 rpm for all subjects. Even though 60 rpm is reported to be the most

efficient pedaling rate, young cyclists preferred to pedal at higher cadences (Sacheti *et al.*, 2010). Cadence set at 60 rpm for our study contributed greater economy during low workloads. At this cadence there is a preferred recruitment of type I muscle fiber type, generating a more efficient relationship between O_2 consumption and ATP formation. However, during the highest workload, pedaling at 60 rpm may have missed being the most economical cadence to recruit type II muscle fiber type.

Recommendations for future research

sEMG frequency spectrum signals in our study were the most relevant parameters when analyzing muscle fatigue processes. When comparing median and mean frequency sEMG signals, the median was a more robust signal during our cycling trials because it produced more significant differences, probably because was less affected by the noise produced by the action potentials. From the muscles analyzed during pedaling, the vastus lateralis and rectus femoris provided greater differential sEMG information on the fatigue process. Although the vastus medialis also provided useful information, significant differences between trials were less frequent. Consequently, future cycling studies may benefit from use of median sEMG analysis of the vastus lateralis and rectus femoris.

The RMS sEMG signals in our study were not statistically different between the VO_{2max} and the intense anaerobic tests because the noise generated from the sEMG signals was large, for which the probability of finding significant differences for this parameter decreases. Our recommendation is to have at least 12 participants for these RMS studies, to minimize the magnitude of the standard deviation.

Sixty rpm was selected as the cadence to perform the VO_{2max} and the anaerobic tests as it has been widely reported as the most efficient speed, however, athletes commonly self-select higher cadences. Allowing the participants to be tested at their preferred cadence may produce more individualized sEMG signals (Emanuele & Denoth, 2011). Future studies may benefit from analyzing sEMG signals, force production, and MVC at 70, 80, and 90 rpm as a higher cadence may produce different results than those found in the present study.

To our knowledge, this is the first study to simultaneously analyze pedal force and bilateral sEMG to compare two different maximal exertion protocols to induce muscle fatigue. From these measurements it is possible to evaluate pedaling asymmetry and its tendency over time through the analysis of pedal force production. At the same time, muscle coordination between legs and among different muscle groups could be established through the analysis of bilateral sEMG signals during incremental or constant workload trials. These analyses give a more complete evaluation of the bilateral fatigue process during cycling.

Measurement of some intramuscular biochemical parameters such as $[H^+]$, Pi, lactate, or other metabolic by-products during fatigue-inducing protocols will contribute to a better understanding of the muscle fatigue process, and how energy stores are used and depleted during maximal effort.

Summary

Our results indicate that the intense anaerobic test induced more fatigue than did the VO_{2max} test. This is evident based on the greater decrement in the frequency values

(mean and median sEMG signals) for the rectus femoris and the vastus lateralis following the intense anaerobic trial. Our hypotheses related to mean and median frequency sEMG signals were supported by the data. We found significantly larger decrement percentage values for the intense anaerobic test compared to the VO_{2max} test in 4 out of 5 muscles, suggesting a reserve of muscle fiber recruitment not used to prevent a possible injury or a catastrophic event (Noakes, 2005).

Force production, heart rate and MVC data were similar at the end of both tests, suggesting that these variables are not sensitive enough to detect fatigue-related differences between our intense anaerobic and VO_{2max} cycling trial. From our study we observed that sEMG signals are a good tool to quantify muscle fatigue. Mean and median frequencies could be used to analyze the effect of fatigue during different protocols and to observe possible differences according to the kind of exercise or test. The sEMG frequency spectrum signals enabled us to find differences in the fatigue level of knee extensors muscles at the end of two exhaustion tests. The mean and median sEMG signals obtained imply that muscle exhaustion does not fully occur during a VO_{2max} test. The intense anaerobic test produced more muscle fatigue as evidenced by significantly slower signals at the end of the test compared to the VO_{2max} test.

Combining bilateral pedal force production and sEMG signals allows analysis of factors such as the pedaling asymmetry observed across the workload spectrum. As the point of maximal exertion is neared, asymmetry gives way to symmetry and there is no difference at the maximal exertion load. Also, because muscle recruitment is not similar among muscle groups and between legs, it is very important to analyze muscle coordination using bilateral sEMG monitoring.

REFERENCES

Abbis, C., & Laursen, P. (2005). Models to explain fatigue during prolonged endurance ciclyng. *Sport Medicine,* 35(10), 865-898.

Allen, D.G., & Westerblad, H. (2001). Role of phosphate and calcium stores in muscle fatigue. *Journal of Physiology,* 536(3), 657-665.

Amann, M., Eldridge, M., Lovering, A., Stickland, M., Pegelow, D., & Demspsey, J. (2006). Arterial oxygenation influences central motor output and exercise performance via effects on peripheral locomotor muscle fatigue in humans. *Journal of physiology,* 575(3):937-952.

Amann, M., & Calbet, J. (2007). Convective oxygen transport and fatigue. *Journal of Applied Physiology,* 104, 861-870.

Amann, M., & Kayser, B. (2008). Nervous system function during exercise in hypoxia. *High Altitude Medicine & Biology*, 10(2),149-164.

Amann, M. (2010). Exercise-induced metabolic perturbations: all roads lead to Rome. *Experimental Physiology-Viewpoints,* 95(7):765-766.

Amann, M., Blain, G., Proctor, L., Sebranek, J., Pegelow, D., & Dempsey, J. (2011). Implications of group III and IV muscle afferents for high-intensity endurance exercise performance in humans. *Journal of Physiology,* 589(21), 5299-5309.

American College of Sport Medicine (2010). ACSM's Guidelines for Exercise Testing and Prescription. 6th edition, Lippinkott Williams & Wilkins, Baltimore, USA.

Arabadzhiev, T., Dimitrov, V., Dimitrova, N., & Dimitrov, G. (2010). Interpretation of EMG integral or RMS and estimates of "neuromuscular efficiency" can be misleading in fatiguing contraction. *Journal of Electromyography and Kinesiology*, 20, 223-232.

Baker, J., McCormick, M., & Robergs, R. (2010). Interaction among skeletal muscle metabolic energy systems during intense exercise. *Journal of Nutrition and Metabolism,* 2010, 1-13.

Bassett, D.R., & Howley, E.T. (1997) Maximal oxygen uptake: "classical" versus "contemporary" viewpoints. *Medicine science in sport and exercise,* 29(5):591-603.

Basmajian J.V., & De Luca C.J. (1985). Muscles alive (5th ed., pp. 205–222). Williams and Wilkins, Baltimore, MD.

Bassett, D., & Howley, E. (2000). Limiting factors for maximum oxygen uptake and determinants of endurance performance. *Medicine and Science in Sport and Exercise,* 32(1), 70-84.

Berg, U., Ekblom, B., & Astrand P. (2000). Limiting factors for maximal oxygen uptake ´clasical´ versus ´contemporary´ viewpoints. *Medicine Science in Sport and Exercise,* 32:70-84.

Bigland-Ritchie, B., & Woods, J.J. (1984). Changes in muscle contractile properties and neural control during human muscular fatigue. *Muscle Nerve,* 7(9): 691-699.

Borg, G. (1970). Perceived exertion as an indicator of somatic stress. *Scandinavian Journal of Rehabilitation Medicine*, 2(2)92:98.

Brainbridge, F.A. *Physiology of Muscular Exercise*. London: Longmans, 1919.

Brink-Elfegoun, T., Kaijser, L., Gustafsson, T., & Ekblom B. (2007). Maximal oxygen uptake is not limited by a central nervous system governor. *Journal of applied physiology,* 102:781- 786.

Buick, F., Gledhill, N., Froese, A., Spriet, L., Meyers, E. (1980). Effect of induced erythrocythemia on aerobic work capacity. *Journal of Applied Physiology,* 48(4):636-642.

Calbet, J., Boushel, R., Radegran, G., Sondergaard, H., Wagner, P., & Saltin, B. (2002). Determinants for maximal oxygen uptake in severe acute hypoxia. *American Journal of Physiology Regulatory and Integral Comprehensive Physiology*, 284:291-303.

Calbet, J., Boushel, R., Radegran, G., Sondergaard, H., Wagner, P., & Saltin, B. (2003). Why is VO_{2max} after altitude acclimatization still reduced despite normalization of arterial O2 content? *American Journal of Physiology Regulatory and Integral Comprehensive Physiology*, 284:304-3161.

Camata, T., Altimari, L., Bortolotti, H., Dantas, J., Fontes, E., Smirmaul, B., Okano, A., Chacon-Mikahil., & Moraes, A. (2011). Electromiographic activity and rate

of muscle fatigue of the quadriceps femoris during cycling exercise in the severe domain. *Journal of Strength and Conditioning Research,* 25(9):2537-2543.

Camic, C., Housh, T., Johnson, G., Hendrix, R., Zuniga, J., Mielke M., Smidt, R. (2010). An EMG frequency-based test for estimating the neuromuscular fatigue threshold during cycling ergometry. *European Journal of Applied Physiology,* 108, 337-345.

Carpes, F., Mota. C., & Faria, I. (2010). On the bilateral asymmetry during running and cycling – a review considering leg preference. *Physical Therapy in Sport,* 11:136-142.

Carpes, F.P., Rossato, M., Faria, I.E., & Mota, C.B. (2007). Bilateral pedaling asymmetry during a simulated 40-km cycling time trial. *Journal of Sports Medicine and Physical Fitness,* 47(1), 51-57.

Dalsgaard, M.K., & Secher, N.H. (2007). The brain at work: a cerebral metabolic manifestation of central fatigue? *Journal of Neuroscience Research*, 85, 3334-3339.

De Luca, C. (1985). Control properties of motor unit. *Journal of Experimental Biology*, 115, 125-136.

De Luca, C. (1997). The use of surface electromyography in biomechanics. *Journal of Applied Biomechanics*, 13,135-163.

Dimitrov, G.V., Trabadzhiev, T.I., Hogrel, J.Y., & Dimitrova, N.A. (2008). Simulation analysis of interference EMG during fatiguing voluntary contractions. Part II – Changes in amplitude and spectral characteristics. *Journal of Electromyography and Kinesiology,* 18:35-43.

Ekblom, B., Huot, R., Stein, E., & Thorstensson, T. (1975). Effect of changes in arterial oxygen content on circulation and physical performance. *Journal of Applied Physiology*, 39(1):71-75.

Ekblom, B. (2006). Stroke volume and the endurance athlete. *Scandinavian Journal of Medicine & Science in Sports*, 16:70-71.

Emanuele, H., & Denoth, J. (2011). Power-cadence relationship in endurance cycling. *European Journal of Applied Physiology,* 107, 1-11.

Ettema, G., & Wuttudal, H. (2009). Efficiency in cycling: a review. *European Journal of Applied Physiology,* 106:1-14.

Farina, D., Macaluso, A., Ferguson, R., & De Vito, G. (2004). Effect of power, pedal rate, and force on average muscle fiber conduction velocity during cycling. *Journal of Applied Physiology*, 97:2035-2041.

Farina, D. (2006). Interpretation of the surface electromyogram in dynamic contractions. *Exercise and Sport Sciences Reviews,* 34(3):121-127.

Farina, D. (2008). Counterpoint: Spectral properties of the surface EMG do not provide information about motor unit recruitment and muscle fiber type. *Journal of Applied Physiology,* 105:1673-1674.

Fick, A. (1870). Uber die messung des blutquantums in den hertzvent rikeln. *Sitzberg Physik Med Ges Wurzburg,* 36.

Fitts, RH. (1994). Cellular mechanisms of muscle fatigue. *Physiological Review,* 74(1), 49-81.

Foss, O., & Hallen, J. (2004). The most economical cadence increases with increasing workload. *European Journal of Applied Physiology,* 92:443-451.

Gaesser, GA., & Brooks, GA. (1975). Muscular efficiency during steady-rate exercise: effects of speed and work rate. *Journal of Applied Physiology,* 38(6):1132-1139.

Gandevia, SC (1998). Neural control in human muscle fatigue: changes in muscle afferents, moto neurons and moto cortical drive. *Acta Physiological Scandinavian,* 162, 275-283.

Gandevia, SC (2001). Spinal and supraspinal factors in human muscle fatigue. *Physiological Review,* 81(4),1726-1771.

Gore, C., Hahn, A., Scroop,G., Watson, D., Norton, K., Wood, R., Campbell D., & Emonson, D.(1996). Increased arterial desaturation in trained cyclists during maximal exercise at 580 m altitude. *Journal of Applied Physiology,* 80(6):2204-2210.

Gonzalez-Alonso, J., Calbet, J., & Nielsen, B. (1988). Muscle blood flow is reduced with dehydration during prolonged exercise in humans. *Journal of Physiology,* 513(3):895-905.

Gonzalez-Alonso, J., & Calbet, L. (2003). Reductions in systemic and skeletal muscle blood flow and oxygen delivery limit maximal aerobic capacity in humans. *Circulation,* 107:824-830.

Graef, J., Smith, A., Kendall, K., Walter, A., Moon, J., Lockwood, C., Beck, T., Cramer, J., & Stout, R. (2008). The relationship among endurance performance measures

as estimated from VO_{2PEAK}, Ventilatory threshold, and electromyographic fatigue threshold: a relationship design. *Dynamic Medicine*, 7:1-15.

Hargreaves, M. (2008). Fatigue mechanisms determining exercise performance: integrative physiology is system biology. *Journal of Applied Physiology*, 104:1541-1542.

Hawkins, M., Raven, P., Snell, P., Stray-Gundersen, J., Levine, B., (2007). Maximal oxygen uptake as a parametric measure of cardiorespiratory capacity. *Medicine Science in Sport and Exercise*, 39(1):103-107.

Heyward, VH. (2006). Advanced fitness assessment and exercise prescription. (5th ed). Champaign, IL: Human Kinetics.

Hill, A.V., & Lupton, H. (1923). Muscle exercise, lactic acid, and the supply and utilization of oxygen. *Journal of Medicine*, 168:445-456.

Hug, F., Faucher M., Kipson, N., & James, Y. (2003). EMG signs of neuromuscular fatigue related to the ventilatory threshold during cycling exercise. *Clinical Physiology and Functional Imaging*, 23(4), 208-214.

Hug, F., & Dorel, S. (2009). Electromyographic analysis of pedaling: A review. *Journal of Electromyography and Kinesiology*, 19:182-198.

Joseph, T., Johnson, B., Battista, R., Wright, G., Dodge, C., Porcari, J., Koning, K., & Foster, C. (2008). Perception of fatigue during simulated competition. *Psychobiology and Behavioral Strategies*, 40(2):381-386.

Kayser, B. (2003). Exercise starts and end in the brain. *European Journal of Applied Physiology,* 90, 411-419.

Koulouri, C. (2010). From antiquity to Olympic revival: Sports and Greek national historiography (nineteenth-twentieth centuries). *International Journal of History in Sport*, 27(12), 2014-2052.

Krustup, P., Soderlun, K., Mohr, M., Bangsbo, J. (2004). The slow component of oxygen uptake during intense, submaximal exercise in man is associated with additional fibre recruitment. *European Journal of Physiology,* 447:855-866.

Laia, F.M., Perez-Gomez, J., Nordsbirg, N., & Bangsbo, J. (2009). *Scandinavian Journal of Medicine and Science in Sports,* 1-11.

Lamb, G. (2006). Point:counterpoint: Lactic acid accumulation is an advantage/disadvantage during muscle activity. *Journal of Applied Physiology,* 100:1410-1414.

Levine, B., Lane, L., Buckey, J., Friedman, D., & Blomqvist, G., (1991). Left ventricular-volume and frank-Starling relation in endurance athletes. *Journal of the American Heart Association,* 84:1016-1023.

Levine, BD. (2008). VO_{2max}: what do we know, and what do we still to know? *Journal of Physiology*, 586(1), 25-34.

Levine, B., Lane, LD., Buckey, JC., Friedamn, DB., & Blomqvist, CG. (1991). Left ventricular pressure-volume and Frank-Starling relations in endurance athletes.

Implications for orthostatic tolerance and exercise performance. *Circulation,* 84*:*1016-1023.

Leirdal, S., & Ettema, E. (2011). The relationship between cadence, pedaling technique and gross efficiency in cycling. *European Journal of Applied Physiology,* 11:1914-1923.

Liu, J., Lewandowski, B., Karakasis, C., Yao, B.,. Siemionow, V., Saghal, V., & Yue, G. (2007). Shifting of activation center in the brain during muscle fatigue: An explanation of minimal central fatigue?. *Neuroimage,* 35*:*299-307.

MacDonald, J., Farina, D., & Marcora, S. (2008). Response of electromyografic variables during incremental and fatiguing cycling. *Medicine Science in Sport and Exercise,* 40(2), 335-344.

Malek, M., Coburn, J., Weir, J., Beck, T., & Housh, T. (2006). The effects of inervation zone on electromyographic amplitude and mean power frequency during incremental cycle ergometry. *Journal of Neuroscience Methods,* 155:126-133.

Marcora, S., & Staiano, W. (2010). The limit to exercise tolerance in humans: mind over muscle? *European journal of Applied Physiology,* 10;1418-1426.

Martin, V., Dousset, E., Laurin, J., Gondin, J., Gautier, M., & Decherchi, P. (2009). Group III and IV muscle afferent discharge patterns after repeated lengthening and shortening actions. *Muscle Nerve,* 40, 827-837.

McKeena, M.J., & Hargreaves, M. (2008). Resolving fatigue mechanisms determining exercise performance: integrative physiology at its finest. *Journal of Applied Physiology*, 104:286-287.

Medbo, J.I., Mohn, A.C., Tabata, I., Bahr, R., Vaage, O., Sejersted, & O.M. (1988). Anaerobic capacity determined by maximal accumulated O_2 deficit. *Journal of Applied Physiology*, 63(1):50-60.

Medbo, J.L., & Tabata, I. (1989). Relative importance of aerobic and anaerobic energy release during short-lasting exhausting bicycle exercise. *Journal of Applied Physiology*, 67(5):1881-1886.

Minetti, A. (2011). Bioenergetics and biomechanics of cylcling: the role of 'internal work'. *European Journal of Applied Physiology*, 111:323-329.

Morgensen, M., Bagger, M., Pedersen, P.K., Ferstrom, M., & Sahlin, K. (2006). Cycling efficiency in humans is related to low UCP3 content and to type I fibres but not to mitocondrial efficiency. *Journal of Physiology*, 571(3):669-681.

Naik, G., Kumar, D., Yadav, V., Wheeler K., & Arjunan, S. (2009). Testing of motor unit synchronization model for localized muscle fatigue. 31st Annual International Conference of the IEEE EMBS

Noakes, T.D. (1997). 1996 J.B. Wolffe memorial lecture. Challenging beliefs: ex Africa semper aliquid novi. *Medicine and Science in Sport and Exercise*, 29(5):571-590.

Noakes, T.D., St. Clair Gibson, A., & Lambert, E.V. (2005). From catastrophe to complexity: a novel model of integrative neural regulation of effort and fatigue during

exercise in humans: summary and conclusions. *British Journal of Sport Medicine,* 29:120-124.

Noakes, T.D. (2008). Testing for maximal oxygen consumption has produced a brainless model of human exercise performance. *British Journal of Sport Medicine,* 42:551-555.

Noakes, T.D. (2012). Fatigue is a brain-derived emotion that regulates the exercise behavior to ensure the protection of whole body homeostasis. *Frontiers in Physiology,* 3(82):1-13.

Noordhof, D.A., de Koning, J.J., & Foster, C. (2010). The maximal accumulated oxygen deficit method. A valid and reliable measure of anaerobic capacity? *Sports Medicine,* 40(4), 285-302.

Owen-Reece, H., Smith, M., Elwell, C.E., & Goldstone, J.C. (1999). Near Infrared Spectroscopy. *British Journal of Anesthesia*, *82*(3): *418*-426.

Peltonen, J., Paterson, D., Shoemaker, K., DeLorey, D., duManoir, G., Petrella, R., & Kowalchuk, J. (2009). Cerebral and muscle deoxygenation, hypoxic ventilarory chemosensitivity and cerebrovascular responsiveness during incremental exercise. *Respiratory Physiology & Neurobiology,* 169:24-35.

Perry, C., Talantan, J., Helgenhauser, G., & Spriet, L. (2007). The effects of training in hyperoxia vs normoxia on skeletal muscle enzyme acivities and exercise performance. *Journal of Applied Physiology,* 102:1022-1027.

Racinais, S., Girard, O., Micallef, JP., & Perrey, S. (2007). Failed excitability of spinal motoneurons induced by prolonged running exercise. *Journal of Neurophysiology,* 97:596-603.

Rouffet, DM., & Hautier, CA. (2008). EMG normalization to study muscle activation in cycling. *Journal of Electromyiography and Kinesiology,* 18:866-878.

Sacchetti, M., Lenti, M., Scotto, A., De Vito, G. (2010). Different effect of cadence on cycling efficiency between young and older cyclists. *Medicine and Science in Sport and Exercise,* 42(11):2128-2133.

Sahlin, K., Tonkonogi, M., & Söderlund, K. (1998). Energy supply and muscle fatigue in humans. *Acta Physiological Scandinavian,* 162, 261-266.

Saltin, B., Calbet, J., & Wagner, P. (2006). Point: in health and in normoxic environment, Vo_{2max} is limited primarily by cardiac output and locomotor muscle blood flow. *Journal of Applied Physiology*, 100, 774-778.

Shushakov, V., Stubbe, C., Peuckert, A., Endeward, V., & Maassen, N. (2007). The relationship between plasma potassium, muscle excitability and fatigue during voluntary exercise in humans. *Experimental Physiology,* 92(4):705-715.

Spriet, L., Soderlund, K., Bergstrom, M., & Hultman, E. (1987). Anaerobic energy release in skeletal muscle during electrical stimulation in men. *Journal of Applied Physiology*, 62(2), 611-615.

Subudhi, A., Miramon, B., Granger, M., & Roach, R., (2009). Frontal and motor cortex oxygenation during maximal exercise in normmoxia and hypoxia. *Journal of Applied Physiology,* 106:1153-1158.

Taylor, J., & Gandevia, S. (2004). Noninvasive stimulation of the human corticospinal tract. *Journal of Applied Physiology,* 96:1496-1503.

Taylor, J., & Gandevia, S. (2007). A comparison of central aspects of fatigue in submaximal and maximal voluntary contraction. *Journal of Applied Physiology,* 104:542-550.

Tenan, M., McMurray, R., Blackburn, BT., McGrath, M., & Leppert, K. (2010). *Journal of Electromyography and Kinesiology,* 19:1-8

Thomas, G., & Segal, S. (2004). Neural control of muscle blood flow during exercise. *Journal of Applied Physiology,* 97:731-738.

Thomas, R., & Stephane, P. (2008). Prefrontal cortex oxygenation and neuromuscular responses to exhaustive exercise. *European Journal of Applied Physiology*, 1*02*, 153-163.

Trent, J., Johnson, B., Battista, R., Wright, G., Dodge, C., Porcari, J., Koning, L., & Foster, C. (2008). Perception of fatigue during simulated competition. *Medicine and Science in Sport and Exercise,* 40*(2)*;381-386.

Van Hese, P., Philips, W., De Koninck. J., Van de Walle, R., & Lemahieu, I. (2001). Automatic Detection of Sleep Stages Using the EEG. *Proceedings of the 23rd*

Annual EMBS International Conference, October 25-28, Istanbul, Turkey, 2001, *pp.* 1944-19.

Vella, CA., & Robergs, RA., (2005). A review of the stroke volume response to upright exercise in healthy subjects. *British Journal of Sports Medicine*, 39(1):190-195.

Wakeling, J.M., Blake, O.M., & Chan, H.K. (2010). Muscle coordination Is key to the power output and mechanical efficiency of limb movements. *The Journal of Experimenatl Biology,* 213. 487492.

Weir, J., Beck, T., Cramer, J., & Housh, T. (2006). Is fatigue all in your head? A critical review of the central governor model. *British Journal of Sports Medicine*, 40*;573-586.*

Wisløff, U., Ellingsen, O, & Kemi, O. (2009). High-intensity interval training to maximize cardiac benefits of exercise training? *Exercise and Sport Science Review,* 37(3):139-146.

Yoon BK, Kravitz L, Robergs RA. (2007). VO_{2max}, protocol duration, and the VO_2 plateau. *Medicine and Science in Sports and Exercise.* 39(7):1186-92.

Yue, G. (2007). Shifting of activation center in the brain during muscle fatigue: An explanation of minimal central fatigue? *NeuroImage*, 35, 299-307.

APPENDIX A

UNIVERSITY OF NEW MEXICO HEALTH SCIENCES CENTER
HIPAA[1] AUTHORIZATION TO USE AND DISCLOSE
PROTECTED HEALTH INFORMATION FOR RESEARCH PURPOSES

Title of Study: Musle Fatigue at the End of a Maximal Oxygen Consumption Test

Principal Investigator:	**Robert Robergs, PhD**
UNMHSC Department:	**Health Exercise & Sport Sciences**
Mailing Address:	**rrobergs@unm.edu**
Co-Investigators:	**Gustavo Sierra**

Sponsor: N/A

1. **What is the purpose of this form?** You have been asked to take part in a research study. The consent forn for this study describes your participation, and that information still applies. This extra form is required by the federal Health Insurance Portability and Accountability Act (HIPAA). The purpose of this form is to get your permission (authorization) to use health information about you that is created by or used in connection with this research.

2. **What if I don't want my personal health information (PHI) to be used in this research study?** You do not have to give this permission. Your decision not to sign this form will not change your ability to get healt care outside of this research study. However, if you do not sign, then you will not be allowed to participate i the study.

3. **What PHI am I allowing to be used for this research?** The information that may be used includes: Healt history and current health status, height, weight, age, gender, blood pressure, heart rate at rest and durign exercise. Maximal oxygen consumption capacity, maximal capacity to exercise during intense exercise with oxygen debt, the ability to produce and re-use lactate accumulation during exercise, and information of side effects (adverse events) you may experience.

4. **Where will researchers go to find my PHI?** We may ask to see your personal information in records at hospitals, clinics or doctor's offices where you may have received care in the past, including but not limited facilities in the UNM health care system.

5. **Who will be allowed to use my information for this research and why?** The researchers named above ar their staff will be allowed to see and use your health information for this research study. It may be used to check on your progress during the study, or analyze it along with information from other study participants. Sometimes research information is shared with collaborators or other institutions. Your records may also be reviewed by representatives of the research sponsor or funding agency, the Food and Drug Administration (FDA) to check for quality, safety or effectiveness, or the Human Research Review Committee (HRRC) for the purposes of oversight and subject safety and compliance with human research regulations.

6. **Will my information be used in any other way?** Your information used under this permission may be subject to re-disclosure outside of the research study and be no longer protected under certain circumstances such as required reporting of abuse or neglect, required reporting for law enforcement purposes, and for health oversight activities and public health purposes.

[1] HIPAA is the Health Insurance Portability and Accountability Act of 1996, a federal law related to privacy of health information.

7. **What if I change my mind after I give this permission?** You can change your mind and withdraw this permission at any time by sending a written notice to the Principal Investigator at the mailing address listed at the top of this form to inform the researcher of your decision. If you withdraw this permission, the researcher may only use and share your information that has already been collected for this study. No additional health information about you will be collected by or given to the researcher for the purposes of this study.

8. **What are the privacy protections for my PHI used in this research study?** HIPAA regulations apply to personal health information in the records of health care providers and other groups that share such information. There are some differences in how these regulations apply to research, as opposed to regular health care. One difference is that you may not be able to look at your own records that relate to this research study. These records may include your medical record, which you may not be able to look at until the study is over. The HIPAA privacy protections may no longer apply once your PHI has been shared with others who may be involved in this research.

9. **How long does this permission allow my PHI to be used?** If you decide to be in this research study, your permission to access and use your health information in this study may not expire, unless you revoke or cancel it. Otherwise, we will use your information as long as it is needed for the duration of the study.

I am the research participant or the personal representative authorized to act on behalf of the participant. By signing this form, I am giving permission for my personal health information to be used in research as described above. I will be given a copy of this authorization form after I have signed it.

_____	_____	_____
Name of Research Subject	Signature of Subject/Legal Representative	Date

_____
Describe authority of legal representative

_____	_____	_____
Name of Person Obtaining Authorization	Signature	Date

Version: 10/20/10
HRRC# 10-449

APPENDIX B

The University of New Mexico Health Sciences Center
Consent to Participate in Research

Muscle Fatigue at the End of a Maximal Oxygen Consumption Test

Introduction

You are being asked to participate in a research study that is being done by Ann Gibson Ph.D., who is the Principal Investigator and Gustavo Sierra Ph.D. Candidate, from the Department of Health Exercise and Sport Sciences. This research is studying the level of muscle fatigue at the end of a maximal oxygen consumption test in 12 healthy male subjects.

You are being asked to participate in this study because of your status as a subject with a good aerobic capacity, a regular training status of at least 3 times per week for more than 20 minutes per session. 12 people will take part in this study at the University of New Mexico.

This form will explain the research study, and will also explain the possible risks as well as the possible benefits to you. We encourage you to talk with your family and friends before you decide to take part in this research study. If you have any questions, please ask one of the study investigators.

What will happen if I decide to participate?

If you agree to participate, the following things will happen:

Procedures

If you decide to participate, you will be asked to report to the Exercise Physiology Laboratories (EPL) located at the basement of the Johnson gym on 3 separate occasions for testing that will take approximately 1 to 1.5 hours each.

For the first testing session, you will be asked to complete a health history questionnaire and you will perform one maximal oxygen consumption (VO_2max) test, which consists of a maximal exercise bout on a stationary bike where the time to complete the test is between 8 and 10 minutes according to your fitness level. Electrodes will be placed on your chest to record your heart rate during the test. You will be asked to wear a nose clip and breathe through a plastic breathing valve attached to hoses to allow monitoring of the air you exhale. Prior to the start of the test, you will warm-up for 10 minutes pedaling at 70 revolutions per minute. You will then rest for 5 minutes. Before you start the protocol, you will be required to rest for two minutes to obtain basal data. After these two minutes, the test will start, and you will start pedaling at 80 rpm. The test will be terminated when either (1) you have reached exhaustion, or (2) there is no further increase in O_2 consumption even with an increase in the workload. After the test is completed, the workload will automatically be lowered to 50 Watts, and you will cool down until your heart rate is lower than 120 beats/min.

For the second and third session, you will perform in a random order either a second VO_{2max} test (following the same protocol as above), or a 3-minutes all out anaerobic test. These tests will be separated by at least seven days. The anaerobic test will consist of pedaling for 3-minutes at an intensity of 110% of your maximal workload from the test completed during the first session. During these sessions a catheter will be placed in your antecubital vein that is in your arm, to drawn blood every minute during the first 5 minutes, and every 30 seconds thereafter during the VO_{2max} test, for blood lactate determination. During the 3-minutes anaerobic test, blood will be drawn every 30 seconds. Also, during these sessions some surface electrodes will be placed on your skin over different muscles of the legs to record muscle fiber activity during the tests. Finally, added sensors will be placed at the base of each pedal to measure force production during the test.

How long will I be in this study?

Participation in this study will take a total of 4.5 hours over a period of of 3 sessions of 1.5 hours each.

What are the risks or side effects of being in this study?

- Possible side effects of maximal exertion include brief feelings of nausea, lightheadedness, muscle cramps, or dizziness after completion of exercise. In addition, you may have discomfort associated with cycling to maximal exertion while breathing through a mouthpiece. In people who are highly endurance-trained, exercise testing with gradually increasing workloads to the point of fatigue have a very minimal risk of death (<0.005%) and complications of the heart (<0.001%). All personnel conducting the test are trained in cardiac life support, and are competent in exercise testing procedures. Furthermore, all research team members have previous exercise testing experience and are aware of the signs and symptoms that are associated with possible adverse reactions during exercise.

- There are risks of stress, emotional distress, inconvenience and possible loss of privacy and confidentiality associated with participating in a research study.

For more information about risks and side effects, ask your study doctor.

What are the benefits to being in this study?

Results from the VO_{2max} and the 3minutes anaerobic test will give you information concerning your aerobic and anaerobic capacity. Furthermore, an estimation of your muscle fiber recruitment ability will be generated that will give a more complete knowledge about your ability to generate muscle contraction and force production at maximal efforts. This study will help to the scientific knowledge

about the level of muscle fatigue at the end of a VO_{2max} and have a better idea about what factors cause a premature end to exercise during incremental exercise.

What other choices do I have if I do not want to be in this study?

The only alternative is to not participate in this study.

How will my information be kept confidential?

We will take measures to protect your privacy and the security of all your personal information, but we cannot guarantee confidentiality of all study data.

Information contained in your study records is used by study staff and, in some cases it will be shared with the sponsor of the study. The University of New Mexico Health Sciences Center Human Research Review Committee (HRRC) that oversees human subject research, will be permitted to access your records. There may be times when we are required by law to share your information. However, your name will not be used in any published reports about this study. A copy of this consent form will be kept in your medical record.

What are the costs of taking part in this study?

You will not be charged for any of the study's procedures.

What will happen if I am injured or become sick because I took part in this study?

If you are injured or become sick as a result of this study, UNMHSC will provide you with emergency treatment, at your cost. No commitment is made by the University of New Mexico Health Sciences Center (UNMHSC) to provide free medical care or money for injuries to participants in this study.

In the event that you have an injury or illness that is caused by your participation in this study, reimbursement for all related costs of care will be sought from your insurer, managed care plan, or other benefits program. If you do not have insurance, you may be responsible for these costs. You will also be responsible for any associated co-payments or deductibles required by your insurance.

It is important for you to tell your study doctor immediately if you have been injured or become sick because of taking part in this study. If you have any questions about these issues, or believe that you have been treated carelessly in the study, please contact the Human Research Review Committee

(HRRC) at the University of New Mexico Health Sciences Center, Albuquerque, New Mexico 87131 (505) 272-1129 for more information.

Will I be paid for taking part in this study?

There will be no financial compensation for your participation in this study.

How will I know if you learn something new that may change my mind about participating?

You will be informed of any significant new findings that become available during the course of the study, such as changes in the risks or benefits resulting from participating in the research or new alternatives to participation that might change your mind about participating.

Can I stop being in the study once I begin?

Your participation in this study is completely voluntary. You have the right to choose not to participate or to withdraw your participation at any point in this study without affecting your future health care or other services to which you are entitled.

Whom can I call with questions or complaints about this study?

If you have any questions, concerns or complaints at any time about the research study, Ann Gibson, PhD, or her associates will be glad to answer them at (505) 277-2658, Monday Through Friday 8:00am – 5:00pm. If you would like to speak with someone other than the research team in regards to any complaints you have about the study, you may call the UNM IRB at (505) 272-1129.

Whom can I call with questions about my rights as a research subject?

If you have questions regarding your rights as a research subject, you may call the UNM IRB at (505) 272-1129. The IRB is a group of people from UNM and the community who provide independent oversight of safety and ethical issues related to research involving human subjects. For more information, you may also access the IRB website at http://hsc.unm.edu/som/research/HRRC/maincampusirbhome.shtml.

CONSENT
You are making a decision whether to participate in this study. Your signature below indicates that you read the information provided (or the information was read to you). By signing this consent form, you are not waiving any of your legal rights as a research subject.

I have had an opportunity to ask questions and all questions have been answered to my satisfaction. By signing this consent form, I agree to participate in this study. A copy of this consent form will be provided to you.

Name of Adult Subject (print)	Signature of Adult Subject	Date

INVESTIGATOR SIGNATURE
I have explained the research to the subject or his/her legal representative and answered all of his/her questions. I believe that he/she understands the information described in this consent form and freely consents to participate.

Name of Investigator/ Research Team Member (type or print)

(Signature of Investigator/ Research Team Member)	Date

APPENDIX

C

Physical Activity Readiness Questionnaire (PAR-Q)

YES	NO	
___	___	1. Have you ever been diagnosed with a heart condition?
___	___	2. In the past month, have you had chest pain when you were not doing physical activity?
___	___	3. Do you feel pain in your chest when you do physical activity?
___	___	4. Do you lose balance because of dizziness or do you ever lose consciousness?
___	___	5. Have you ever been diagnosed with abnormal lung function and/or exercise-induced bronchoconstriction?
___	___	6. Are you currently taking any medications? If so, what?
___	___	7. Do you know of any reason why you should not do physical activity?

NOTE: 1. This questionnaire applies only to those 15 to 69 years of age.

2. If you have temporary illness, such as a fever or cold, or are not feeling well at this time, you may wish to postpone the proposed activity.

3. If your health changes so that you then answer yes to any of the above questions, inform the study investigator.

I have read, understood, and completed this questionnaire.

Signature_____ Date_____

Witness_____ Date_____

APPENDIX D

HEALTH HISTORY QUESTIONNAIRE

Name _____ D.O.B / / Date / /

Age yrs_____ Height cm _____ Weight kg_____ Gender _____ Ethnicity_____

MEDICIAL HISTORY QUESTIONNAIRE

Section A

1. When was the last time you had a physical examination?

2. If you are allergic to any medications, foods, or other substances, please name them.

3. If you have been told that you have any chronic or serious illnesses, please name them.

Section B

During the past 12 months

1. Has a physician prescribed any form of medication for you?	Yes No
2. Has your weight fluctuated more than a few pounds?	Yes No
3. Did you attempt to bring about this weight change through diet or exercise?	Yes No
4. Have you experienced any faintness, light-headedness, or blackouts?	Yes No
5. Have you occasionally had trouble sleeping?	Yes No
6. Have you experienced any blurred vision?	Yes No

7. Have you experienced chronic morning cough? Yes No

8. Have you experienced any temporary change in your speech pattern, such as slurring or loss of speech? Yes No

9. Have you felt unusually nervous or anxious for no apparent reason? Yes No

10. Have you experienced unusual heartbeats such as skipped bests or palpations? Yes No

11. Have you experienced periods in which your heart felt as though it were racing for no apparent reason? Yes No

At present

1. Do you experience shortness or loss of breath while walking with others your own age? Yes No

2. Do you experience sudden tingling, numbness, or loss of feeling in your arms, hands, leg, feet, or face? Yes No

3. Have you ever noticed that your hands or feet sometimes feel cooler than other parts of your body? Yes No

4. Do you experience swelling of your feet and ankles? Yes No

5. Do you get pains or cramps in your legs? Yes No

6. Do you experience any pain or discomfort in your chest? Yes No

7. Do you experience any pressure or heaviness in your chest? Yes No

8. Have you ever been told that your blood pressure was abnormal? Yes No

9. Have you ever been told that your serum cholesterol or triglyceride level was high? Yes No

10. Do you have diabetes? Yes No

If yes, how is it controlled?

□ Dietary means □ Insulin injection

□ Oral medication □ Uncontrolled

11. How often would you characterize your stress level as being high? Yes No

□ Occasionally □ Frequently □ Constantly

12. Have you ever been told that you have any of the following illness? Yes No

□ Myocardial infarction □ Arteriosclerosis □ Heart disease □ Thyroid disease

□ Coronary thrombosis □ Rheumatic heart □ Heart attack □ Heart valve disease

□ Coronary occlusion □ Heat failure □ Heart murmer

□ Heart block □ Aneurysm □ Angina

13. Have you ever had any of the following medical procedures? Yes No

□ Heart surgery □ Pacemaker implant

□ Cardiac catheterization □ Defibrillator

□ Coronary angioplasty □ Heart transplantation

Section C

Has any member of your immediate family been treated for or suspected to have any of these conditions? Please identify their relationship to you (father, mother, sister, brother, etc.).

A. Diabetes

B. Heart disease

C. Stroke

D. High blood pressure

Section D

Smoking habits

1. Have you ever smoked cigarettes, cigars, or a pipe? Yes No

2. Do you smoke presently? Yes No

Cigarettes a day

Cigars a day

Pipefuls a day

3. At what age did you start smoking? ____years

4. If you have quit smoking, when did you quit?

PHYSICAL ACTIVITY/EXERCISE QUESTIONARE

1. Do you exercise vigorously on a regular basis? Yes No

2. What activities do you engage in on a regular basis?

3. If you walk, run, or jog, what is the average number of miles you cover each workout?

_____ Miles

4. How many minutes on the average is each of your exercise workouts?

5. How many workouts a week you participate in on average?

6. Do you train weight-lifting? Yes No

7. How well trained are you?

8. How many workouts a week do you participate weight-lifting in on average?