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Muscle performance during slow isokinetic speeds

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MUSCLE PERFORMANCE DURING
SLOW ISOKINETIC SPEEDS

by

Jonathan Michael Kwantes
Bachelor of Science
University of Missouri-Rolla
1999

A thesis submitted in partial fulfillment
of the requirements for the

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ABSTRACT

Muscle Performance During Slow Isokinetic Speeds

By Jonathan Kwantes

Dr. John Mercer, Examination Committee Chair
Assistant Professor of Biomechanics
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The purpose of this study was to investigate the possible mechanism responsible for the decrease in force during the 5 °/sec velocity compared to the isometric velocity. Subjects (n=9) completed MVC knee extension exercises during eccentric speeds of 5, 10, and 15 °/sec, as well as isometric contractions. Extensor force and average EMG (aEMG) of the vastus lateralis (VL) and biceps femoris (BF) were quantified at knee angles of 55° and 65°. Six ANOVA (α = 0.05) tests determined a decrease in $F_T$ ($p < 0.05$) at both knee angles and a decrease in VL aEMG ($p < 0.05$) at 55° during 5 °/sec condition compared to the isometric condition. No change in BF aEMG was observed across speeds ($p > 0.05$). It was concluded that there is a controlling mechanism which is neurological in nature which reduces $F_T$ during slow eccentric conditions.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABSTRACT</td>
<td>iii</td>
</tr>
<tr>
<td>LIST OF FIGURES</td>
<td>vi</td>
</tr>
<tr>
<td>LIST OF TABLES</td>
<td>vii</td>
</tr>
<tr>
<td>ACKNOWLEDGMENTS</td>
<td>viii</td>
</tr>
<tr>
<td>CHAPTER 1 INTRODUCTION</td>
<td>1</td>
</tr>
<tr>
<td>Muscle Function</td>
<td>1</td>
</tr>
<tr>
<td>Torque-Velocity Relationship</td>
<td>2</td>
</tr>
<tr>
<td>Purpose</td>
<td>4</td>
</tr>
<tr>
<td>Hypothesis</td>
<td>5</td>
</tr>
<tr>
<td>Limitations</td>
<td>5</td>
</tr>
<tr>
<td>Definitions</td>
<td>6</td>
</tr>
<tr>
<td>CHAPTER 2 LITERATURE REVIEW</td>
<td>8</td>
</tr>
<tr>
<td>Anatomy of Skeletal Muscle</td>
<td>8</td>
</tr>
<tr>
<td>Factors Contributing to Muscle Force</td>
<td>9</td>
</tr>
<tr>
<td>Sliding Filament Theory</td>
<td>9</td>
</tr>
<tr>
<td>The Role of ATP</td>
<td>12</td>
</tr>
<tr>
<td>Neural Action Potential to Muscle Contraction</td>
<td>12</td>
</tr>
<tr>
<td>Neural-Muscular Coordination</td>
<td>14</td>
</tr>
<tr>
<td>Muscle Contraction Types</td>
<td>17</td>
</tr>
<tr>
<td>Isometric Contractions</td>
<td>18</td>
</tr>
<tr>
<td>Concentric Contractions</td>
<td>18</td>
</tr>
<tr>
<td>Eccentric Contractions</td>
<td>19</td>
</tr>
<tr>
<td>Muscle Performance</td>
<td>19</td>
</tr>
<tr>
<td>Length-Tension Relationship</td>
<td>19</td>
</tr>
<tr>
<td>Three-Element Model</td>
<td>20</td>
</tr>
<tr>
<td>Torque-Velocity Relationship</td>
<td>23</td>
</tr>
<tr>
<td>Voluntary Contractions</td>
<td>27</td>
</tr>
<tr>
<td>Artificial Stimulation</td>
<td>28</td>
</tr>
<tr>
<td>Other Factors Contributing to Force Production</td>
<td>29</td>
</tr>
<tr>
<td>Electromyography (EMG)</td>
<td>31</td>
</tr>
<tr>
<td>Force-EMG Relationship</td>
<td>32</td>
</tr>
</tbody>
</table>

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LIST OF FIGURES

Figure 1. Theoretical Force-Velocity Relationship .............................................................. 3
Figure 2. Sliding Filament Mechanism of Muscle Contraction ............................................ 10
Figure 3. Three-Element Muscle Model ............................................................................. 21
Figure 4. Length-Tension Relationship ............................................................................ 22
Figure 5. Force-Velocity Plot reported by Kwantes et al. (2000) ....................................... 27
Figure 6. Average $F_T$ for nine subjects at knee angles of 55° and 65° ............................. 40
Figure 7. aEMG of VL for nine subjects at knee angles of 55° and 65° ............................ 41
Figure 8. aEMG of BF for nine subjects at knee angles of 55° and 65° ............................ 41
Figure 9. Subject plot of $F_T$, BF and VL aEMG at knee angles of 55° and 65° .......... 49
LIST OF TABLES

Table 1. Comparison of results to Kwantes et al. (2000). Values are percent of isometric force ................................................................. 44
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viii
CHAPTER 1

INTRODUCTION

Muscle Function

Skeletal muscle has several important functions within the human body. For example, skeletal muscle maintains posture and body position, aids in the regulation of body temperature, gives support to visceral organs and other soft tissues, and performs muscular contractions. Movement of the skeleton is allowed through the contraction of muscle and a joint structure. How and why the skeletal muscle functions the way it does is a very broad topic that the fields of biomechanics, physiology, and motor control work to understand. Based on theoretical and experimental data these fields, piece together important information that provide the understanding of human movement which exists today. Individually each of these areas provide theory and disciplines important in the understanding of human movement. However, when the information of one area is used in conjunction with the others, the complex systems which make human movement possible is better understood. The way in which these fields cooperatively work to explain, and expand, the area of human movement, or kinesiology, is of vital importance. How these three fields work together controlling muscular contractions, which produce certain performance characteristics is of particular interest for the present study.
Torque-Velocity Relationship

During contraction, skeletal muscles create a force which can be used to move a load or external force. When the force acts about an axis of rotation, (i.e., a joint), a torque is created. When the torque exerted by the muscle is greater than the torque exerted by the load, a concentric contraction occurs. If the load torque is significantly less than that of the muscle torque, the contraction will occur at a fast velocity. That is, the smaller the load torque, the faster the muscle can contract. Furthermore, the faster the muscle contracts, the less torque it can exert on the load (Komi, 1973; Kramer, Vaz, & Hakansson, 1991; Seger & Thorstensson, 2000; Westing, Cresswell, & Thorstensson, 1991; Westing, Seger, Karlson, & Ekblom, 1988). This general observation of muscle performance is known as the torque-velocity relationship. This relationship between muscle torque and movement velocity has been investigated in vitro as well as in vivo. When referring to in vitro muscle contraction, the relationship is referred to as the force-velocity. When referring to in vivo muscle contractions, the relationship is referred to as torque-velocity.

Previous research investigating the force-velocity relationship has produced both theoretical and experimental results. Figure 1 illustrates a general theoretical plot of the in vitro force-velocity relationship.

The force capability of the muscle during concentric contraction is inversely related to muscle contraction velocity (Enoka, 1994). That is, the force generated during fast movements is lower than the force generated during slow movements. However, during eccentric contractions the relationship between force and velocity is not inverse.
During eccentric contractions, previous research (Seger & Thorstensson, 2000; Westing et al., 1988) has determined that there is an initial increase in force compared to isometric contraction, then a leveling off of force production as eccentric velocity increases.

![Theoretical Force Velocity Curve](image)

Figure 1. Theoretical plot of force-velocity relationship. Contraction velocities are measured along the x-axis. Muscle force is measured along the y-axis. Isometric condition is represented with the vertical line.

Others (Evetovich, Housh, Johnson, Smith, Ebersole, & Perry, 1998; Kellis & Baltzopoulos, 1998; Westing et al., 1991) have observed a similar relationship between force and velocity using only eccentric and concentric contractions but not isometric contraction. Nevertheless, despite subtle differences in protocol between studies, the general shape of the eccentric force-velocity relationship is rather consistent between studies. In general, it is expected that eccentric force either increases or stays the same as eccentric contraction velocity increases relative to isometric contraction.
Purpose

Despite the wealth of information on the force-velocity relationship, there has been little investigation of muscle performance during slow eccentric velocities (less than 20 °/sec) (Cresswell & Pinniger, 1999; Kwantes, Rasmussen, Mercer, Hoffman, & Osternig, 2000; Pinniger Steele, Thorstensson, & Cresswell, 1999; Seger & Thorstensson, 2000). Kwantes et al. (2000) tested knee extensor muscles during isometric conditions and during eccentric speeds of 5, 10, 15 and 20 °/sec. The authors reported during slow eccentric velocities there was a deviation from the expected in vitro force-velocity curve. At the 5 °/sec eccentric speed Kwantes et al. (2000) reported a decrease in maximum force output from the isometric force value during the 5 °/sec condition. Pinniger et al. (1999) reported a similar deviation at the 5 °/sec speed when testing the plantar flexors. There are no studies which have reported that eccentric force was greater than the isometric force value during 5 °/sec velocity. Other studies which measure muscle performance eccentric during eccentric actions have tested during velocities greater than 20 °/sec (Evetovich et al., 1998; Kellis & Baltzopoulos, 1988; Westing et al., 1988; Westing et al. 1991). Kwantes et al. (2000) reported similar results to that of previous studies only when eccentric speeds were greater than 10 °/sec (Evetovich et al., 1998; Kellis & Baltzopoulos, 1998; Seger & Thorstensson, 2000; Westing et al., 1988).

Due to the lack of other studies (Cresswell & Pinniger, 1999; Pinniger et al. 1999; Kwantes et al., 2000; Seger & Thorstensson, 2000) which test muscle performance at such slow speeds, a definitive conclusion could not be formed by the authors that explained the reason for the decreased force at the 5 °/sec speed (Kwantes et al., 1999).
The present study was designed to test possible mechanisms that explain the force during the 5 °/sec conditions when compared to isometric conditions. The question central to the study was: Is the reason for this decrease in force physiological or neurological in nature? Since there is a lack of experimental evidence in this area of the force-velocity plot, an underlying neuromuscular or physiological mechanism of muscle contraction could have been concealed. Therefore the purpose of this study was to determine if the mechanism responsible for the deviation described by Kwantes et al. (2000) is neurological or physiological in nature.

Hypothesis:

The following hypotheses were implemented to determine the nature of the mechanism.

$H_0$: The mechanism responsible for the decrease in force during slow eccentric isokinetic speeds is not neurological in nature.

$H_1$: The mechanism responsible for the decrease in force during slow eccentric isokinetic speeds is neurological in nature.

Limitations

The purpose of this study was to identify the nature of the controlling mechanism responsible for the decrease in force during slow eccentric isokinetic speeds. When developing the logic for this study, it was determined that the mechanism explaining the decrease in force during slow eccentric velocities would either be a neurological or
physiological based mechanism. The possible mechanism would be either located and controlled somewhere within the nervous system or it would be located and controlled somewhere within the muscular structure. Using the techniques of electromyography, (EMG) and isokinetics, the results could be coordinated and a conclusion could be drawn if the mechanism was neurological. Listed below are some limitations identified for the present study.

1. Subjects were untrained in isokinetics, except for one orientation session prior to the test session.

2. Subjects gave maximal effort during the isometric and eccentric conditions.

3. EMG accurately represented muscle activity of the vastus lateralis and biceps femoris.

4. Subjects were tested on their left leg only.

5. Test conditions included isometric and eccentric speeds of 0, 5, 10, and 15 °/sec. There were no test conditions which included intervals speeds between 0, 5, 10, and 15 °/sec.

6. If the controlling mechanism was not determined to be physiological, it was concluded the mechanism was neurological.

Definitions

*aEMG* - average EMG; the mean EMG over a specified time period.

Concentric contraction - when muscle torque is greater than the load torque and muscle
length shortens.

**Dynamometer** - instrument used to measure force.

**Eccentric contraction** - when muscle torque is less than the load torque and muscle lengthens.

**Electromyography (EMG)** - the measurement of the electrical activity within a muscle which is caused by action potentials sent from the nervous system.

**Force** - a push or pull that tends to cause motion. SI units of N.

**In vitro** - muscle tested outside the body.

**In vivo** - muscle tested within the body.

**Isokinetics** - motion when there is no acceleration, thus the velocity is constant.

**Isometric contraction** - when muscle torque is equal to the load torque, which results in no change in whole muscle length.

**MVC** - maximal voluntary contractions; the total contraction force a subject can exert voluntarily.

**Speed** - time rate of change of distance. (See also velocity).

**Tangential force** - \( F_t \) the force component that acts parallel to and along a surface.

**Torque** - the tendency of a force to cause rotation. SI units of Nm.

**Velocity** - time rate of change of displacement. (See also speed).
CHAPTER 2

LITERATURE REVIEW

Anatomy of Skeletal Muscle

Within the human body, there are three types of muscle: skeletal muscle, smooth muscle, and cardiac muscle (Martini & Timmons, 1995). Each of these types of muscle are vital to life in their individual way. However, the performance of skeletal muscle will be the focus of this literature review. A single muscle cell is referred to as a muscle fiber. Muscle fibers range in diameter from 10 to 100 μm and may have lengths that are 20 cm long (Vander, Sherman, & Luciano, 1998) depending on the location of the fiber. Muscle fibers are bound together by connective tissue to form groups of muscles. Each skeletal muscle has three layers of connective tissue which hold it together. The most outer layer surrounds the entire muscle and is known as the epimysium. The next layer, which groups bundles of muscle fibers together, is known as the perimysium. Finally, at the cellular level, the endomysium surrounds each individual fiber and binds it to the fibers which surround it.

In addition to connective tissue structure, the muscle fiber still has further organization. Each muscle fiber has a membrane which surrounds the contents of the fiber. The sarcolemma is the membrane, and the sarcoplasm is the contents of the muscle fiber. The sarcoplasm contains hundreds of myofibrils. These myofibrils contain the
contractile elements which make contraction possible. Surrounding each myofibril is a structure known as the sarcoplasmic reticulum (SR). The SR, and structures known as transverse tubules (T-tubules), are key in initiating and coordinating the neural and muscular systems. T-tubules are sets of narrow tubules that extend into the SR. Within the myofibril, contractile elements are organized in repeating functional contractile units known as sarcomeres. Sarcomeres are the smallest functional units of the muscle fiber. Interactions between the contractile elements are what make muscle contraction possible (Martini & Timmons, 1995). The physiology of these structures and how they create force within the muscle will be described.

Factors Contributing to Muscle Force

Sliding Filament Theory

Within skeletal muscle the functional contractile unit is the sarcomere. Sarcomeres are arranged in series within a myofibril. A sarcomere is composed of two groups of overlapping contractile filaments, categorized as thick and thin filaments. The thick filaments are composed mainly of the contractile protein myosin. The thin filaments are composed mainly of the contractile protein actin, but also two regulator proteins troponin and tropomyosin. The contributions of the troponin and tropomyosin proteins to muscle contraction will be described shortly. When force is produced during a muscle shortening contraction, the thick and thin filaments slide past each other, resulting in a shortening of the sarcomere. There is no change in the length of the thick and thin filaments. This process is known as the sliding filament mechanism of muscle.
contraction (Vander et al., 1998). Figure 2 is a general illustration of how the sliding filament mechanism works. During a muscle contraction, force is produced when the myosin cross bridges pull the thin filament towards the center of the sarcomere. During a concentric contraction, this results in a shortening of the sarcomere. The actual length of the thin (actin) and thick (myosin) filaments do not change, only the degree of overlap of the filaments.

![Relaxed and Contracted Muscle Diagram](image)

Figure 2. The sliding filament mechanism of muscle contraction.
During contraction, the actin and myosin filaments are connected by projections from the myosin filaments known as cross bridges. The cross bridges extend from the surface of the myosin molecule toward the actin molecule. When the muscle contracts, the cross bridges bind to the actin molecule and move in a manner like an oar on a boat, pulling the actin filaments toward the center of the sarcomere, thus shortening the sarcomere. The cross bridges do not produce enough shortening of the sarcomere in just one stroke to contract the whole muscle body. Instead, the cross bridges perform many strokes resulting in a greater movement of the filaments. There are approximately 200 cross bridges located on each myosin filament within a sarcomere. However, not all of these bind at the same time. Depending on the stimulus, at any one instant during contraction, about half of the cross bridges are bound and producing tension on the actin filament. Different binding timings and sequences allow the muscle to contract smoothly. The frequency and timing of the cross-bridges could play an important role in classifying the tension regulating mechanism as physiological or neurological.

The repeating process of a cross bridge binding to a thin filament is known as a cross bridge cycle (Vander et al., 1998). Each cycle consists of four steps: (1) attachment of the cross bridge to a actin filament, (2) movement of the cross bridge, producing tension in the actin filament, (3) detachment of the cross bridge from the actin filament, and (4) energizing of the cross bridge so it can again attach to an actin filament and repeat the cycle, (Vander et al., 1998). Remembering that sarcomeres are arranged in series within a muscle fiber, the coordination of the shortening of several to hundreds of
sarcomeres within many muscle fibers, produce the movements associated with tasks such as walking, throwing, or jumping.

The Role of ATP

Adenosine triphosphate (ATP) and calcium (Ca) are two important chemicals which must be present for muscular contraction and relaxation to occur via cross bridge cycling. The role of calcium will be explored in the next section. For now the focus will be on the ATP molecule; one of the primary sources of energy in all cells. The hydrolysis of ATP to adenosine diphosphate (ADP) and an inorganic phosphate group (Pi) releases a large amount of energy which is stored in the terminal phosphate bond of an ATP molecule. When muscles are not stimulated to contract, the myosin cross bridges are not bound to the actin filament. However, an ATP molecule is bound to the myosin molecule. The hydrolysis of ATP to ADP by myosin energizes the cross bridges, which in turn provide the energy required for the force production during muscular contraction. The enzyme actomyosin ATPase catalyzes the hydrolysis (ATP $\rightarrow$ ADP + Pi + energy). Energy is required to allow the cross bridges to dissociate from the actin and repeat their cycle of activity. In other words the presence of ATP is necessary for the relaxation of the muscle to occur. The energy produced when ATP is hydrolyzed to ADP provides the energy for the cross bridge cycle.

Neural Action Potential to Muscle Contraction

A contraction stimulus is sent to the muscle via the central nervous system (CNS). When a neural signal is sent to a muscle, the signal in the nerve cell is traveling in the form of an action potential. An action potential is a rapid change in the resting membrane...
potential of a cell. Muscle cells have a resting membrane potential of about -70 mV. This is due to concentrations of various ions located within the cell. An action potential can essentially be described as electrical energy. This electrical energy must be changed to mechanical energy for muscular contraction via cross bridge cycling. As the action potential is reaching the end of the nerve cell and approaches the muscle, it must cross the neuromuscular junction. When the action potential reaches the neuromuscular junction, it is relayed from the nerve cell to the muscle cell membrane through the release of a neurotransmitter, known as acetylcholine (ACh), from the axon bulbs of the nerve cell. Neurotransmitters act as chemical messengers between nerve cells, or in this case, between nerve and muscle cells. ACh crosses the neuromuscular junction and binds to ACh specific receptors located on the muscle. The binding of ACh to the post-synaptic membrane causes a change in the membrane potential of the myofibril. The neural action potential has now been changed to a muscle action potential.

Once the muscle action potential is initiated, it travels very quickly over the muscle fiber membrane. The muscle action potential travels down the muscle fiber where it enters the T-tubules. The T-tubules are folds within the membrane wall which allow the muscle action potential to travel within the muscle fiber. The presence of the action potential within the muscle fiber, triggers the release of calcium ions from the sarcoplasmic reticulum into the sarcoplasm. Once in the sarcoplasm, the calcium binds to the troponin complex, located on the actin filament. When there is no action potential sent to the muscle, the tropomyosin is in a position which blocks the cycling of the cross bridges. For the cross bridges to bind, a conformational change must be performed on the
tropomyosin molecule, also located on the actin filament. This is where calcium ion is involved. The troponin complex is located on the tropomyosin molecule which twists around the actin molecule. As the calcium is released via the muscle action potential, it recognizes a calcium binding site located on the troponin molecule. When the calcium binds to the troponin, the tropomyosin undergoes a conformational change recognizing the bound calcium ion. The conformational change moves the tropomyosin into a contraction ready orientation. This orientation exposes the cross bridge binding sites located on the actin filament. Now the energized myosin can bind to the actin filament and cross bridge movement can occur.

For muscle contraction to cease and relaxation to occur, the calcium ion must be removed from its binding site on the troponin complex. As neural action potential ceases, so does muscle action potentials, and calcium is no longer released into the T-tubules. The calcium is removed from the troponin complex by an enzyme known as Ca-ATPase. This enzyme actively transports calcium against a concentration gradient into the sarcoplasmic reticulum where it can be used for future muscle contractions. The transport of calcium begins as soon as calcium is released. When the calcium is removed from the troponin, the tropomyosin complex again undergoes a conformational change resulting in blocking of actin binding sites and the muscle fiber relaxes.

**Neural-Muscular Coordination**

Nerve cells provide the electrical stimulation in the form of action potentials which initiate and control muscle contractions. Muscular and nervous systems act together to produce movement via the motor system. The foundation of movement is
built upon functional structures known as motor units. Each motor neuron, and the skeletal muscle fibers which it innervates, constitutes a motor unit. The motor unit is the smallest unit of the motor system which can be controlled. Neurologically, the motor unit is made up of a synaptic junction in the ventral root of the spinal cord, a motor axon (nerve), and a motor end plate which is located within the muscle fibers (Winter, 1979). Motor neurons are commonly called the "final common pathway" (Vander et al., 1998).

Within each muscle, there is located a finite number of motor units, and those motor units control a certain amount of contractile elements. The number of contractile elements a motor unit innervates, is dependant on the location of the muscle within the body. For example, fine movements, such as that of the eye, are controlled by motor units which innervate a few muscle fibers. Large movements, such as that performed by the vastus lateralis, are controlled by motor units which innervate many muscle fibers. Winter (1979) calculated that in a motor unit 0.1 cm² there are approximately $4 \times 10^{11}$ contractile elements controlled by it.

Motor units control the muscle fibers they innervate by relaying their action potentials from the CNS to muscle membranes. "The electrical indication is a motor unit action potential; the mechanical result is a twitch of tension" (Winter, 1979). This twitch of tension is the result of the cross bridge binding. The motor units are able to control the amount of tension generated by muscle by two methods. The first method is by increasing the rate at which the motor unit fires. That is, the motor unit sends action potentials at a faster rate. The second method is by increasing the amount of motor units that fire on the muscle fibers. That is, the more motor units which are recruited, the more muscle fibers
there are which produce tension.

Although it may seem logical when the frequency of firing and the number of motor units firing are increased, the more likely it is that tension can be increased, this is not always the case. During voluntary muscle contractions motor units typically fire independently of each other (Milner-Brown, Stein, & Lee, 1975). The motor units can fire in what is known as a synchronous or asynchronous pattern (Milner-Brown et al., 1975). If the motor unit firing patterns are synchronous, an increase in muscle activity is generally likely to produce a greater tension within the muscle. If the motor unit firing patterns are asynchronous, an increase in muscle activity is not likely to produce a greater tension within the muscle. The most forceful contraction occurs if the greatest number of motor units are recruited and all are firing in a synchronous pattern at a high frequency. Motor units can be trained to fire in synchronous patterns (Enoka, 1988). Strength training has been known to produce neural adaptations which can result in increases in strength. For example, Milner-Brown et al. (1975) tested the synchronization of motor units in weightlifters. They reported the use of muscles to exert large brief forces can cause an increase in synchronization of motor unit firing patterns (Milner-Brown et al., 1975).

The electrical impulses sent to a muscle can be recorded by a technique known as electromyography (EMG). The EMG represents the measurement of action potentials within muscle (Enoka, 1994). A high level of neural activity means the muscle is being stimulated above that level of what is happening during normal conditions, such as during more forceful contractions. However, a high level of neural activity can not distinguish between synchronous or asynchronous firing patterns. Although this is a caveat of using
EMG, it is still a common method for correlating force and EMG. When used correctly, EMG techniques and analysis can help uncover what is happening neurologically during muscular contractions.

Muscle Contraction Types

When skeletal muscle is stimulated to contract, there are three types of contraction that can occur: isometric, concentric, and eccentric. Each of these contractions are specific to both the internal and external conditions to which they are exposed. Each one of these contraction types will be described.

Muscle contraction exerts a force because of the binding and pulling of the cross bridges and subsequent shortening of the sarcomere. This force, in vivo, creates a torque about a joint. A torque is simply a force which tends to cause rotation. For example, through contraction, the biceps brachii muscle exerts a force on the radius via the tendon which inserts on the radius. As the muscle contracts a force is created within the muscle fiber causing the distance between its origin and insertion sites to decrease. The force created within the muscle fiber pulls on its insertion site. In this example, the force is applied to the radius. The elbow joint is a pivot point on which the forearm can rotate. The torque is created about the elbow joint, which results in flexion of the radius. Torque is the result of a force acting a distance from an axis of rotation and can be expressed as \[ T = F \cdot d \]. Where \( T \) represents the torque and has units of Newton meters (Nm); \( F \) is the force and has units of Newtons (N); \( d \) is the perpendicular distance between the line of application of the force and axis of rotation and has units of length (meters).
Isometric Contractions

Although the tendency of the muscle fiber is to shorten as it contracts, the force it exerts does not always result in movement of the whole muscle. When a muscle contracts with no visible movement, the contraction is referred to as an isometric (same length) contraction. For example, consider a biceps curl where the starting movement of the elbow is at 180° and there is no initial movement. An isometric contraction would happen when the torque exerted by the muscle is equal to the magnitude of the torque exerted by the load. If the two torque magnitudes are equal and opposite in direction, no movement about the joint will occur. Isometric contractions occur when one muscle may attempt to shorten to move a body part, but, due to resistive forces exerted by an external object, person, or an antagonistic muscle, the body part does not move (Kreighbaum & Barthels, 1996). Isometrics are very important to the present study because they represent a type of contraction when there is no contraction velocity.

Concentric Contractions

If the torque exerted by the muscle and the torque exerted by the external load are not equal, movement of the joint occurs. If the muscle exerts a torque that is greater than that of the external load, muscle shortening occurs. This type of contraction is called a concentric contraction. The flexion portion of the biceps curl is an example of concentric contraction. This is where the function of the sliding filament model of muscle contraction can be demonstrated. The concentric contraction results in movement towards the center of the sarcomere.
Eccentric Contractions

If the muscle exerts a torque which is less than that of the load, muscle lengthening occurs. This type of contraction is known as an eccentric contraction. An example of this type of contraction is the quadriceps action during the controlling of the weight during the down phase of a squat lift. An eccentric contraction is performed by the quadriceps muscle due to the weight used during the squat. If the weight was not controlled by the torque of the eccentric quadriceps contraction, the weight would drop to the ground at a faster rate. Because eccentric contractions occur under conditions different from that of concentric contraction, it is plausible there is a certain degree of muscle characteristics specific to each type of contraction.

Muscle Performance

Length-Tension Relationship

The amount of force a muscle can produce during a contraction through a range of motion is partially determined by the length of the muscle. This relationship between muscle length and force generated states that there is an optimal length ($L_o$) at which the contractile elements of the muscle can exert the greatest amount of force (Winter, 1979). As the muscle shortens or lengthens past the optimal length the force capability decreases. The shape of the length-tension relationship is generally parabolic, with the peak force occurring at resting length ($L_o$). The greatest amount of tension produced at $L_o$ is primarily the result of the optimal overlapping of thick and thin filaments within the sarcomere. At $L_o$, the thick and thin filaments overlap so the maximum amount of cross
bridges are allowed to bind. Due to this binding of the cross bridges, the maximum amount of force the contractile elements of the muscle is capable of producing occurs at $L_0$.

The angular position at which the quadriceps can produce the greatest force has been reported to occur between $40^\circ - 70^\circ$ of knee flexion (Kramer et al., 1991). James, Sacco, Hurley, and Jones (1994) tested the length-tension relationship of knee extensors and reported the greatest maximal voluntary contraction (MVC) force was achieved between $0.8 - 1.2$ (rad). This is approximately between $45^\circ - 69^\circ$ of knee flexion.

Since torque is the product of muscle force and moment arm. MVC produce similar results when referring to angular position with either force or torque. For example, Seger and Thorstensson (2000) tested subjects at isokinetic speeds of 10, 20, and 30 $^\circ$/sec and recorded torques at $60^\circ$ of knee extension. Westing et al. (1988) tested subjects at isokinetic speeds of $30^\circ$, $120^\circ$, and $270^\circ$/sec and measured torques every $10^\circ$ from $30^\circ$ to $70^\circ$ of knee extension. Aagaard and Anderson (1998) tested subjects at isokinetic speeds of $30^\circ$, $120^\circ$, and $240^\circ$/sec and measured torques at $50^\circ$ of knee extension. These torques are all located at or around the range associated with $L_0$ of an individual. Regardless of using torques or forces, $L_0$ of the quadriceps generally occurs around the same range of knee angles ($40^\circ-70^\circ$ of knee flexion).

**Three-Element Model**

In addition to connective tissue, all elements needed to exert a force are contained within the muscle sarcomere. These elements can generally be represented by a three-element model (Enoka, 1994; Winter, 1979). These elements are a parallel elastic
element (PE), a series elastic element (SE), and a contractile element (CE). The PE and SE are both elastic components and can be represented by springs. The CE represents the contractile force capabilities of the sarcomere. Figure 3 represents the organization of the three-element model within the force component model.

The PE is made up of the connective tissue which surrounds the contractile elements and functions similarly to that of an elastic band. The PE, as its name indicates, lies in parallel to the contractile elements. When the muscle is in a relaxed state, the PE is in a relaxed state and exerts no tension.

![Diagram of force component model](image)

Figure 3. A force component model including PE (parallel elastic), SE (series elastic), and the CE (contractile element).
When the muscle begins to lengthen, tension is built up within the PE. The contribution of the PE component to force production is dependant on muscle length. Figure 4 illustrates the how the PE and the CE contribute to the force-length curve. The PE is represented by the $F_{pe}$ line. This is the force produced by the parallel component. Notice as length increases, so does the force. The CE is represented as $F_c$. The force produced by the contractile element is represented by $F_c$. Notice the general parabolic shape, with its peak force occurring at $L_o$. The $F_t$ represents the total length-tension relationship as it occurs in the body. $F_t$ is the result of the total force produced by the $F_{pe}$ and $F_c$. (Generally the length-tension relationship uses force to measure tension. Although it should probably be called the length-force relationship, it is accepted to refer to it as length-tension).

![Figure 4. Length-tension relationship.](image)
The SE, CE, and PE are capable of producing forces which vary depending on factors such as joint angle, contraction type, and muscle architecture. Muscle architecture encompasses differences in muscle such as the type of fibers, organization of fibers, and whether the muscle acts over one or two joints. Therefore, identifying the position where the greatest force is produced is dependent on several factors. By comparing the same muscle groups at the same angle between conditions, the variability due to these factors is minimized.

**Torque-Velocity Relationship**

During isotonic contractions, when the muscle torque and load torque are not equal, there is a change in whole-muscle length. This results in eccentric or concentric contractions depending on the relationship between the two torques. "The torque that the muscle can exert under these conditions depends on the magnitude and direction of the rate of change in muscle length" (Enoka, 1988). That is, torque due to muscle contraction is dependant on how fast the muscle changes length, which is dependant on the velocity of the contraction. Another way to interpret muscle performance is the velocity of the contraction is dependant on the torque exerted by the load. The performance capability of muscle during different contraction velocities is known as the force-velocity relationship. Although torque and force are not the same mechanical concept, the relationship between torque due to muscle force and angular velocities is similar to that of force and velocity.

The force-velocity relationship was first established by applying electrical stimulation to animal muscle in vitro (Hill, 1938). Because Hill (1938) tested animal muscle in vitro, the relationship was first described as the force-velocity relationship.
However, when testing in vivo, the amount of force exerted by a body segment is a result of the torque produced about a joint. The torque produced about a joint is a function of muscle force and moment arm. For example, the amount of force exerted by the leg against an isokinetic dynamometer is a function of the torque about the knee.

Hill (1938) determined that the in vitro force-velocity relationship to be curvilinear. Specifically, as the speed of muscle contraction increases, the force exerted by the muscle decreases. Since Hill (1938), studies have confirmed the curvilinear relationship between the force and velocity of contraction in vivo (Froese & Houston, 1985; Komi, 1973; Kramer et al., 1991; Seger & Thorstensson, 2000; Westing et al., 1988). Subtle differences exist in the force-velocity curve from one study to another, however the general shape is rather consistent between studies. That general shape includes the force decreasing curvilinearly as velocity increases during MVC contractions. The specific, subtle differences between studies can be attributed to variations in instrumentation, subtle differences in testing protocol or the subject population.

To determine the relationship between muscle force and velocity of contraction isometric, eccentric, and concentric contractions are tested. Westing et al. (1988) tested 21 males on an isokinetic dynamometer SPARK system for maximal voluntary isometric, concentric, and eccentric contractions of the quadriceps muscle at four isokinetic speeds. Muscle performance was tested during isometric actions, as well as during concentric and eccentric contractions during speeds of 30 °/sec, 120 °/sec and 270 °/sec. Westing et al. (1988) reported eccentric torques were consistently higher than corresponding concentric peak torques at the same velocity. Although they reported the eccentric torques were
slightly higher than isometric torques at the same angle, they concluded there was no significant difference between contraction conditions. However, concentric torques were always significantly lower than isometric torques when comparing at a specific angle.

Differences in force output when measuring muscle performance during isokinetic speeds were reported by Westing et al. (1988) and Astrand and Rodahl (1986). Astrand and Rodahl (1986) used isolated in vitro muscle instead of in vivo muscle. A key difference between in vitro and in vivo muscle is the lack of a controlling nervous system in vitro. Westing et al. (1988) concluded that the findings of Astrand and Rodahl (1986) describes muscle properties in the purest sense, while their findings are more typical of a more complex nervous, muscular and skeletal system interaction. Westing et al. (1988) suggest that the reason why large increases of eccentric torque were not observed in vivo could be due to a neural tension-restricting mechanism which protects the muscle when tension develops during low-velocity contractions. Other studies have also suggested this tension-restricting mechanism (Perrine & Edgerton, 1978; Seger & Thorstensson, 2000) is present during slow eccentric speeds. Another point of interest raised by Westing et al. (1988) was the conclusion that the tension-restricting mechanism was not active during concentric contractions during the same speeds.

There is a great deal of literature investigating the force-velocity relationship during isokinetic contractions at speeds ranging between 30 °/sec and 270 °/sec (Komi, 1973; Kramer et al., 1991; Seger & Thorstensson, 2000; Westing et al., 1991; Westing et al., 1988). Instead of measuring the angle in degrees, some of the isokinetic research measures angles using radians (e.g., James et al., 1994). In any case, muscle performance
is tested the majority of the time at speeds between 30 °/sec and 270 °/sec.

Despite this large amount of literature for fast speeds, there is a limited amount of literature that test isokinetic muscle contractions at speeds slower than 30 °/sec (Cresswell & Pinniger, 1999; Kwantes et al., 2000; Pinniger et al., 1999; Seger & Thorstensson, 2000). This could be due to the fact that generally, human motion does not take place during such slow speeds, and is therefore not practically important. However, testing at slow eccentric speeds does help to identify different characteristics of muscle performance. For instance, Kwantes et al. (2000) reported a decrease in knee extensor force production at eccentric speeds of 5 °/sec from that of isometric force. Pinniger et al. (1999) reported a slight decrease in plantar flexion torque from that of isometric torque. A decrease in force during eccentric speeds compared to isometric contraction has not been previously reported.

Figure 5 is a graph of the results reported by Kwantes et al. (2000) during eccentric speeds of 5, 10, 15 and 20 °/sec. Notice the decrease in force during the 5 °/sec speed compared to the isometric condition. This decrease in force from the isometric was not previously reported for in vitro muscle studies. This decrease in force is possibly due to the tension restricting mechanism and may only be active during the 5 °/sec speed in vivo. Perhaps it is a result of some neural aspect which is only operational during voluntary contractions.
Figure 5 Force-velocity results reported from Kwantes et al. (2000) at 55° of knee flexion for eccentric speeds of 5, 10, 15, and 20/sec. 0 represents isometric contraction. (S5, S6, and S7 are individual subject plots. Av is entire group average).

Voluntary Contractions

During MVC, subjects exert as much force as possible over the determined range of motion or contraction time. When testing under MVC conditions, the researcher must be sure the subject is indeed exerting as much force as possible, and can reproduce similar force. This is a very important point when measuring muscle performance over multiple trials. The researcher(s) must create a situation in which the subject is able to produce a maximal effort. There is inherently more opportunity for the subject or researcher to act differently over multiple sessions. Examples of changes which could occur in the subject over multiple test days could be muscle soreness, muscle fatigue, a
training effect, or some psychological factors. Changes which could occur in the test environment due to the researcher could be orientation of limbs during testing, the giving of instructions of the task to be completed, or even subconscious behavior (i.e., Rosenthal effect).

Trying to account for all of these factors, plus many not mentioned, are reasons why muscle performance testing typically takes place on one day (Aagaard & Anderson, 1998; Evetovich et al., 1998; Kellis & Baltzopoulos, 1998; Kwantes et al., 2000; Seger & Thorstensson, 2000; Westing et al., 1988; Westing et al., 1991). This way the researcher can eliminate many errors that could occur with multiple test days. However, it is possible to obtain muscle performance results which are the same from multiple test days (Westing et al., 1988; Kramer et al., 1991). Westing et al. (1988) tested muscle performance of subjects on two days of testing. No significant difference was observed between Day 1 and Day 2 peak torques or angle specific torques (measured at 30°, 40°, 50°, 60° and 70°) during isometric, concentric or eccentric contractions. Although it has been shown it is possible to reproduce results from isokinetic testing, it appears to be more widely accepted to test the subject using one session. Thus, for this study subjects were tested on one day.

Artificial Stimulation

As much as the practice of MVC is used, artificial stimulation is also a common method used to measure muscle performance. Artificial stimulation of the muscle is done by stimulating a large nerve close to the muscle with electrical impulses. The electrical
impulses create a muscular contraction just as a MVC does. The difference between the two methods, is the bypassing of the CNS and directly stimulating the peripheral nervous system (PNS).

When comparing forces during MVC and artificial stimulation, it has been reported that MVC forces are not representative of the true maximum force a muscle can produce (Westing, Seger, & Thorstensson, 1990). For example, Westing et al. (1990) reported that electrical stimulation of the knee extensors can produce higher force values than MVC at the corresponding eccentric speeds. Furthermore, when electrical stimulation and MVC were conducted at the same time force magnitude was greater than the force produced using MVC or electrical stimulation individually. This is further evidence there is a neural mechanism inhibiting MVC during slow eccentric contractions. The observation of decreased force output during slow eccentric speeds compared to isometric contraction (Cresswell & Pinniger, 1999; Kwantes et al., 2000; Pinniger et al., 1999) seems to suggest this force-limiting mechanism may produce a stronger effect during slow eccentric velocities. However, no studies have presently examined why forces are lower during slow eccentric speeds compared to isometric speeds.

Other Factors Contributing to Force Production

Within a contraction velocity, the ability of a muscle to produce force is dependent on several neural and muscular properties. These properties include such features as muscle length, cross-sectional area of the muscle, the individual’s force-tension relationship, and the amount of motor unit activation. The structural organization
of sarcomeres within a muscle ultimately affects the force produced by the muscle. If a muscle is longer, the length of its muscle fibers are longer. Since sarcomeres are arranged in series within a muscle fiber, there are a greater number of contractile elements located within each muscle fiber located in the muscle. Therefore, the increase in contractile elements with muscle length allow the muscle fiber to contract in more quickly. In other words, longer muscles are capable of contracting at greater velocity, resulting in faster joint motion, than shorter muscles. Enoka (1994) states the greater number of sarcomeres in series, the greater the change in myofibril length and the greater the rate of change in length in response to a given stimulus. Thus the contraction velocity of a muscle can be represented as $\Delta V = n(\Delta v)$ (Enoka, 1994). Where $\Delta V$ represents contraction velocity of the whole muscle; where $n$ represents the number of sarcomeres in series; and where $\Delta v$ represents the rate of change in length of each sarcomere.

Another structural aspect of muscle that affects performance, is muscle cross-sectional area. The larger the cross-sectional area of the muscle, the greater amount of muscle fibers located within that sectioned area. Therefore, more contractile elements are located within a thicker muscle than a thinner muscle. The thicker muscle can not contract faster than the thinner muscle, it can, however produce more force. A long thick muscle can produce more force than a shorter thinner muscle (Enoka, 1994).

The angle at which the line of pull of the muscle fibers is known as the angle of pennation (Enoka, 1994). For example, a muscle such as the trapezius has triangular pennation, while a muscle such as the vastus lateralis has linear or strap pennation. Instead of adding sarcomeres in series to each other, pennation acts to add the number of
sarcomeres parallel to each other. For this reason, pennation increases the number of muscle fibers in a given volume, but reduces the contribution of contraction velocity to the whole muscle. For these reasons it is difficult to compare muscular performance across different muscle groups.

Most force-velocity studies compare force characteristics between the same muscle group. The majority of the literature seems focus on the knee extensors and flexors. Evetovich et al. (1998), Westing et al. (1988), Westing et al. (1990), Seger and Thorstensson (2000), and Aagaard and Anderson (1998) tested only knee extensor muscles during isokinetic speeds while Kellis and Baltzopoulos (1998) tested both knee extensor and knee flexor (hamstring). Other studies have investigated performance of elbow flexors (Griffin, Tooms, Vander Zwaag, Bertorini, & O'Toole, 1993). The reason for a large portion of the literature to focus on the knee extensors and flexors is not known. However, it can be reasonable to conclude that the muscles are easy to test and can typically exert more force than any other muscle group. Therefore, any noticeable change in force is most likely significant. Also, because knee injuries are common, a lot of research has been conducted to understand the role of knee flexor and extensor strength in preventing injuries.

Electromyography (EMG)

The most common method to quantify muscle activation is to use surface electromyography (EMG). EMG measures the amount of neural activation during a muscular contraction. The muscle contraction force is dictated partly by the amount of
neural activation and therefore can only produce a maximum contraction if all motor units are fully activated. There are factors other than the amount of motor units activated that can affect the quality of an EMG. Factors such as the number of active muscle fibers, firing rates of the muscle fibers, diameter of the muscle fibers, arrangement of the muscle fibers with respect to surface electrode, and the amount of subcutaneous tissue can all contribute to the EMG signal. These are several intrinsic factors of the muscle that De Luca (1992) warns must be understood so a clear understanding and interpretation of EMG data can be done.

Force-EMG Relationship

Using an isokinetic dynamometer and EMG, the possible controlling mechanism of muscle decrease muscle force during the 5 °/sec condition compared to isometric conditions determined. Measuring muscular performance is typically performed on dynamometers, more specifically on isokinetic dynamometers. A dynamometer is an instrument which measures force. An isokinetic dynamometer measures force during constant velocities. A researcher can determine force-velocity characteristics, torque-velocity characteristics, and length-tension characteristics by using isokinetic dynamometers. One of these dynamometers, the Kin-Com 125 E Plus, was used to explore the force-velocity characteristics of the quadriceps during isokinetic eccentric muscle contractions at isometric, eccentric and concentric speeds ranging from 0-15 °/sec.

Surface EMG is a tool frequently used when a correlation between muscle activation and muscle force (Alkner, Tesch, & Berg, 2000; De Luca, 1992; Enoka, 1994).
During MVC, EMG has been used to understand the relationship between muscle activation and muscle force (Alkner et al., 2000; De Luca, 1992; Kellis & Baltzopoulos, 1997; Komi, Kaneko, & Aura, 1987). A higher level of muscle activation is related to an increase in number of motor units firing, or to an increase in firing frequency. In either case, during concentric contractions, it has been reported that an increase in EMG corresponds with an increase in force. (Alkner et al., 2000). Alkner et al. (2000) reported a linear relationship between EMG and force within the vastus lateralis muscle during knee extension and leg press exercises.

As mentioned earlier, it is generally accepted that eccentric muscle contractions can generate a higher torque compared to that of concentric muscle contractions (Westing et al., 1990). However, it has been reported that muscle activity is greater during concentric compared with eccentric contractions (Kellis & Baltzopoulos, 1998; Westing et al., 1990). This is most likely due to the greater contribution of elastic components to overall muscle force during eccentric contractions. In contrast, force produced during concentric contractions is mainly due to the contractile elements of the muscle. Because the contractile elements require a higher motor unit activation, this results in higher EMG activity. Thus the force-EMG relationship during concentric contractions can be generally stated as force increases, EMG activity increases. The exact degree of the relationship between EMG and force varies between muscles. For example, Alkner et al. (2000) reported a linear relationship between force and EMG in the vastus lateralis, while the rectus femoris did not produce a linear force-EMG relationship. This makes the vastus lateralis an ideal muscle for testing the force-EMG relationship.
CHAPTER 3

METHODOLOGY AND DATA DESCRIPTION

Subjects

Nine college age individuals (age = 25.4 ± 2.9 yrs; height = 160.5 ± 21.5 cm; mass = 79.1 ± 11.4 kg), free of any previous knee injury, volunteered for participation in this investigation. This study was conducted in the Sports Injury Research Center at the University of Nevada, Las Vegas and was approved by the Human Subjects Board. All subjects gave informed consent prior to participation in this investigation (Appendix A).

Instrumentation

Tangential force (F_T) was measured using an isokinetic dynamometer (Kin Com 125 A Plus, Chattecx Corp., Chattanooga TN). The dynamometer was calibrated following manufacturer's instructions prior to testing. Vastus lateralis (VL) and biceps femoris (BF) muscle activity levels were measured using a electromyography (EMG) unit (Noraxon USA, Inc., Scottsdale AZ). The EMG and F_T data were recorded concurrently. Synchronization of signals was completed by using a magnetic switch that was set to close and send a square wave signal to the EMG record at specific, known angular positions. F_T data were recorded at 100 Hz and EMG data were recorded at 500 Hz.
Procedure

Subjects were recruited to test muscle performance characteristics during slow isokinetic conditions during eccentric contractions of the knee extensors. Each subject completed one orientation and one testing session separated by two to seven days. All orientation exercises and test exercises were completed using the left leg. Both sessions were completed with the subject seated with the thigh oriented horizontally and their back against the backrest. The angle created between the seat and the backrest was about 90°. The lateral condyle of the tibia was aligned with the rotational axis of the dynamometer and the subject was secured to the seat using velcro straps. Anatomical position of the knee angle was set to zero degrees, with flexion angles positive. An ankle cuff was attached and secured to the distal aspect of the tibia. Additional straps were placed across the anterior aspect of the thighs as well as a seat belt across the waist of the subject. These straps functioned to help isolate the knee extensor group, as well to keep the subject from moving in the chair. During test conditions, subjects were instructed to keep their arms folded across their chest.

The orientation session was incorporated into this study so all subjects would be familiar with isometric and eccentric isokinetic exercises. Use of the Kin Com for exercise is something most people are not familiar with, therefore this orientation session was used as a learning period to help ensure voluntary maximal contraction would be performed on the test day. The orientation session included isokinetic exercises which were both eccentric (negative speeds) and concentric (positive speeds) in nature. The first group of exercises included two sets of six repetitions, with each knee flexion and
extension actions being concentric in nature for agonist muscles. That is, the subject performed a concentric quadriceps contraction followed by a concentric hamstring contraction. Contraction speeds were 60 °/sec in both directions. The second group of exercises included two sets of six repetitions each which were concentric/eccentric in nature. The subject performed a concentric quadriceps contraction followed by an eccentric quadriceps contraction. Contraction speeds were 60 °/sec and 30 °/sec respectively. The third group of exercises included two sets of six repetitions each which were concentric/eccentric in nature. The subject performed a concentric quadriceps contraction followed by an eccentric quadriceps contraction. Contraction speeds were 30 °/sec and 15 °/sec respectively. There was a rest period of one minute between the different groups of exercises. There was 45 seconds of rest between each set of exercises within each group. Including rest periods, the training session lasted approximately 10 minutes.

During the test day, each subject performed a warm-up protocol consisting of concentric and eccentric contractions of the knee extensors similar to that of the orientation session. After the warm-up protocol the subject was allowed as much rest time as needed before testing. The maximal voluntary knee extensor strength was measured isometrically and at eccentric isokinetic speeds of 5 °/sec, 10 °/sec and 15 °/sec. The isometric test was always performed before the isokinetic tests, with the order of the eccentric speeds counterbalanced. The subject performed two 5 second maximal isometric knee extension tests at both 55° and 65°. The eccentric exercise test protocol consisted of three repetitions at each angular velocity.
After the warm up, bipolar surface electrodes were used to record EMG of the VL and BF. The site for electrode placement was determined by visually inspecting the muscle during a voluntary contraction and identifying the muscle belly. This site was then shaved of hair and the skin was aggressively cleaned with alcohol wipes. The type of electrodes used, contained two electrodes of fixed distance on one adhesive strip. The electrodes used had a fixed distance and were 2 cm apart, as measured from center to center, and were placed in line with the general direction of the muscle fibers. Depending on the subject, the body landmark used for the ground electrode placement was the lateral condyle of the tibia or the head of the fibula.

Data Reduction

The 3 second interval of the 5 second isometric contraction which produced the highest average force was determined to be the maximum voluntary isometric force. \( F_T \) was analyzed at the positions of 55° and 65° by averaging force values across specific ranges. \( F_T \) at 55° was calculated as the average force between 54° and 56°; \( F_T \) at 65° was calculated as the average force between 64° and 66°. \( F_T \) of each subject was represented as the mean \( F_T \) across three repetitions at each eccentric speed.

EMG Procedures

Muscle activity was sampled and converted to digital form at a rate of 500 Hz. EMG data were processed by removing the DC bias followed by full-wave rectification. Average EMG (aEMG) across 50° - 60° and 60° - 70° of knee flexion were calculated.
for knee positions of 55° and 65° respectively.

Study Design

The study was a one-way within subjects design with the factor speed having five levels. The levels of speed included 0 °/sec, 5 °/sec, 10 °/sec, and 15 °/sec (measured at 55° and 65° of knee extension). Mean tangential force ($F_t$) produced during a maximum voluntary contraction (MVC) were analyzed using a one-way ANOVA for $F_t$ at 55° [knee angle (55°)] × 4 [speed(5, 10, 15, and 0 °/sec)]; and $F_t$ at 65° [knee angle (65°)] × 4 [speed(5, 10, 15, and 0 °/sec)] 65°. aEMG of the VL were analyzed using a one-way ANOVA for aEMG at 55° [knee angle (55°)] × 4 [speed(5, 10, 15, and 0 °/sec)]; and aEMG at 65° [knee angle (65°)] × 4 [speed(5, 10, 15, and 0 °/sec)] 65°. aEMG of the BF were analyzed using a one-way ANOVA for aEMG at 55° [knee angle (55°)] × 4 [speed(5, 10, 15, and 0 °/sec)]; and aEMG at 65° [knee angle (65°)] × 4 [speed(5, 10, 15, and 0 °/sec)] 65°. Planned comparisons were made after each ANOVA for $F_t$, aEMG of the VL, and aEMG of the BF were completed. Each of the planned comparisons test compared the data of the 0 °/sec condition to the data of each of the eccentric conditions (5 °/sec, 10 °/sec, 15 °/sec).
CHAPTER 4

DATA ANALYSIS

Purpose

The purpose of the present study was to determine if the mechanism responsible for the decrease in force during the 5 °/sec compared to isometric conditions as described by Kwantes et al. (2000) is neurological or physiological in nature. F_T and EMG data collected during isometric and slow eccentric velocities was used to provide evidence that the controlling mechanism was neurological in nature.

Results

Individual data sets are located in Appendix D. At 55° of knee flexion, F_T was different across speeds (Figure 6, F (3, 24) = 14.7, p<0.001). Using planned comparisons F_T during 0 °/sec was greater than F_T during 5 °/sec (F (1,8) = 43.4, p<0.001). Using planned comparisons F_T during 0 °/sec was greater than F_T during 10 °/sec (F (1,8) = 25.6, p = 0.001). Using planned comparisons F_T during 0 °/sec was greater than F_T during 15 °/sec (F (1,8) = 16.6, p = 0.004).

At 65° of knee flexion, F_T was different across speeds (Figure 6, F (3, 24) = 15.1, p<0.001). Using planned comparisons F_T during 0 °/sec was greater than F_T during 5 °/sec (F (1,8) = 28.7, p = 0.001). Using planned comparisons F_T during 0 °/sec was
greater than $F_T$ during 10 °/sec ($F (1,8) = 49.9, p<0.001$). Using planned comparisons $F_T$ during 0 °/sec was greater than $F_T$ measured during 15 °/sec ($F (1,8) = 10.6, p = 0.012$).

At 55° of knee flexion, aEMG of the VL was different across speeds (Figure 7. $F (3,24) = 4.3, p = 0.014$). Using planned comparisons aEMG during 0 °/sec was greater than aEMG during 5 °/sec ($F (1,8) = 14.6, p = 0.005$). Using planned comparisons aEMG during 0 °/sec was greater than aEMG during 10 °/sec ($F (1,8) = 10.7, p = 0.011$). Using planned comparisons aEMG during 0 °/sec was greater than aEMG during 15 °/sec ($F (1,8) = 6.8, p = 0.031$).

At 65° of knee flexion, aEMG of the VL was not different across speeds (Figure 7, $F (3.24) = 0.99, p = 0.416$). At 55° of knee flexion, aEMG of the BF was not different across speeds (Figure 8). ($F (3,24) = 0.65, p = 0.592$). At 65° of knee flexion, aEMG of the BF was not different across speeds (Figure 8, $F (3,24) = 0.42, p = 0.741$).

**Figure 6.** Average $F_T$ results for nine subjects during MVC at knee positions of 55° and 65°. (* : indicates significant decrease in force compared to isometric at 55° and 65°).
Figure 7. aEMG of the VL for nine subjects during MVC at knee positions of 55° and 65°. (*: Indicates a significant decrease in EMG activity during eccentric velocities compared to the isometric condition at 55°).

Figure 8. aEMG of the BF for nine subjects during MVC at knee positions of 55° and 65°. No change in EMG activity was observed for BF across speeds.
CHAPTER 5

DISCUSSION, CONCLUSIONS, AND RECOMMENDATIONS

Force Results

Introduction

The force-velocity curve is a characteristic of muscle performance that has been investigated in vitro and in vivo. There is a wealth of studies which have explored the force-velocity relationship during velocities of 30 °/sec or greater (Evetovich et al., 1998; Kellis & Baltzopoulos, 1998; Perrine & Edgerton, 1978; Westing et al., 1988; Westing et al., 1991). Recent studies have tested muscle performance during velocities less than 30 °/sec (Kwantes et al., 2000; Pinniger et al., 1999) and have observed results not previously reported. For example, it was reported that when testing a muscle during slow eccentric conditions, there was a decrease in force during the 5 °/sec condition compared to the isometric condition. That is, a higher force was produced during isometric contractions compared to the 5 °/sec condition. Seger & Thorstensson (2000) have hypothesized that there is a tension regulating mechanism active primarily during slow eccentric maximal voluntary knee extensor actions. Therefore, the purpose of the present study was to investigate a possible mechanism controlling the decrease in force. The studies which test muscle performance during slow eccentric velocities, have not recorded EMG concurrent with force data. The present study recorded EMG concurrent
with force data in order to better understand what is happening during the 5 °/sec eccentric velocity condition.

Discussion of Force Results

The results of the present study are similar to other studies in that forces were lower during slow eccentric velocities compared to isometric velocities (Kwantes et al., 2000; Pinniger et al., 1999). Figure 6 (page 41) illustrates the force-velocity curve produced from the average F<sub>r</sub> of nine subjects. The decrease in F<sub>r</sub> compared to isometric contraction during the 5 °/sec eccentric contraction was the primary interest for the present study. F<sub>r</sub> dropped significantly during the 5 °/sec eccentric contraction compared to isometric contraction. These results are consistent with those reported by Kwantes et al. (2000) and Pinniger et al. (1999). It is difficult to compare actual force values in this study with those of Pinniger et al. (1999), because actual data points were not reported in Pinniger's abstract. However, when the forces reported by Kwantes et al. (2000) were normalized to percentages, very similar percent decreases in force during the 5 °/sec occurred between studies. Table 1 reports the average percent isometric force across speeds for Kwantes et al. (2000) at 35°, 45° and 55°, as well as the average percent isometric force at 55° and 65° observed in the present study. The percentages of isometric force reported across speeds are very similar between the two studies. Kwantes et al. (2000) reported a force of about 65 % of isometric during the 5 °/sec condition at knee angles of 35° and 45°, and a force of 58 % of isometric during the 5 °/sec condition at a knee angle of 55°. The results of the current study indicate forces of about 65 % of isometric forces during the 5 °/sec condition at both knee angles (55° and 65°). Overall,
there is relative consistent percent decreases in force across speeds at each angle between studies.

Because the decrease in force during the 5 °/sec condition compared to the isometric condition of this force-velocity curve occurred at 55° and 65° of knee flexion, the decrease in force does not seem to be angle specific. Further evidence that the decrease force is not related to knee position is that Kwantes et al. (2000) also reported the decrease in force during the 5 °/sec condition compared to the isometric condition at knee angles of 35°, 45°, and 55°.

Table 1. Forces normalized to % of maximum isometric for present study at angles of 55° and 65°. (*Reproduced results from Kwantes et al. (2000) at angles of 35°, 45°, and 55°).

<table>
<thead>
<tr>
<th>Study (angle)</th>
<th>0 °/sec</th>
<th>5 °/sec</th>
<th>10 °/sec</th>
<th>15 °/sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>*Kwantes (2000) 35°</td>
<td>100</td>
<td>66</td>
<td>76</td>
<td>77</td>
</tr>
<tr>
<td>*Kwantes (2000) 45°</td>
<td>100</td>
<td>64</td>
<td>75</td>
<td>81</td>
</tr>
<tr>
<td>*Kwantes (2000) 55°</td>
<td>100</td>
<td>58</td>
<td>69</td>
<td>76</td>
</tr>
<tr>
<td>Present (2001) 55°</td>
<td>100</td>
<td>67</td>
<td>73</td>
<td>73</td>
</tr>
<tr>
<td>Present (2001) 65°</td>
<td>100</td>
<td>65</td>
<td>65</td>
<td>72</td>
</tr>
</tbody>
</table>

The decrease in force during the 5 °/sec condition compared to the isometric condition does not appear to be muscle specific. Pinniger et al. (1999) reported similar findings for plantar flexion exercises to that of Kwantes et al. (2000) who reported on the knee extensors. Even though the size, fiber type, and line of pull of the knee extensors and plantar flexors are different, both muscle groups elicited similar decreases in force
during the 5 °/sec compared to the isometric. Further testing at slow eccentric speeds of other muscle groups (e.g., knee flexors, elbow extensors and flexors) could provide further evidence the decrease in force is not specific to a knee extensors and plantar flexors.

A common bond among studies reporting the decrease in force, is this drop in force during the 5 °/sec condition compared to the isometric condition only seems to appear during MVC. Previous studies have reported that when the muscle is activated by electrical stimulation, no decrease in force during the 5 °/sec condition compared to the isometric condition is reported (Pinniger et al., 1999; Seger & Thorstensson, 2000). Pinniger et al. (1999) tested the plantar flexors during MVC, submaximal electrical activations, and submaximal voluntary activations. Only during the MVC was the drop in force during the 5 °/sec condition compared to the isometric condition observed. Submaximal electrical stimulation produced significantly higher forces during 5 °/sec eccentric contractions when compared to the isometric forces (Pinniger et al., 1999). Seger & Thorstensson (2000) reported significant increases in force between eccentric forces during 10 °/sec compared to isometric forces during submaximal stimulations of 70%, 50%, and 30% of MVC. Seger and Thorstensson (2000) did not test subjects at 5 °/sec, however, they did report that during MVC no significant difference was observed between isometric forces and eccentric forces at 10 °/sec.

The results of these two studies (Pinniger et al., 1999; Seger & Thorstensson, 2000) support the hypothesis that the mechanism responsible for a decrease in force during slow eccentric velocities is only active during MVC. This would mean the
possible mechanism is active only when there is a conscious effort to exert maximally during slow eccentric velocities. During submaximal conditions, when forces are normalized there is no decrease in force during the 5 °/sec condition compared to the isometric condition (Pinniger et al., 1999; Seger & Thorstensson, 2000). The critical point to be made is the decrease in force during the 5 °/sec condition compared to the isometric condition is active only during maximal efforts. This could be the result of the mechanism being active only during MVC. Because the controlling mechanism appears to be active only during a MVC, it may be that a motor neuron in CNS responsible for MVC in an agonist muscle during shares a common pathway with the motor neuron controlling the contracting muscle. The shared neural pathway may the mechanism that causes an inhibition of muscle force during the 5 °/sec eccentric condition compared to the isometric condition.

EMG Results

Introduction

The purpose of collecting EMG data was to test whether the decrease in force during the 5 °/sec condition compared to the isometric condition is neurologic in nature. There are no studies known to the author that have analyzed EMG during eccentric velocities of less than 20 °/sec. Therefore, EMG results from this study can only be compared to EMG results during higher contraction velocities.

Discussion of EMG Results

EMG of the VL and BF muscles were recorded during slow eccentric maximal
effort knee extensor actions. aEMG were analyzed at two knee positions (55° and 65°) for each muscle during eccentric action of the knee extensors. At the 55° knee position, there was a significant difference (p = 0.005) in aEMG of the VL during the 5 °/sec compared to the aEMG of the isometric. When aEMG values were normalized to the isometric condition, the aEMG values decreased an average of about 24% from the aEMG during the 5 °/sec compared to the aEMG of the isometric condition for the entire group (n=9). Group VL aEMG values decreased during the 5 °/sec to 65.79 μV compared to the aEMG of the isometric condition of 84.73 μV. At the 65° knee position, there was no difference in aEMG of the VL during the 5 °/sec compared to the aEMG of the isometric condition.

At both knee positions, no difference was observed in BF aEMG between speeds. The entire subject population produced BF aEMG values during all eccentric conditions of about 15 % of BF aEMG during a maximum isometric conditions. The BF aEMG values were referenced to a MVC isometric contraction of the BF recorded at the end of the test session. This value is small in comparison to percent of activation (about 65 %–75%) of the VL during eccentric exercises.

Comparing changes in force and changes in EMG across speeds, it seems that as force decreased during the 5 °/sec compared to the isometric, aEMG of the VL decreased concurrently by about 24% compared to the isometric. There was about a 35% decrease in force during the 5 °/sec speed, so it appears there is a significant loss of muscle activity. This reduction in muscle activity may explain the decrease in force. aEMG of the BF does not seem to elicit a co-contraction response during slow eccentric action of
the leg extensors. It was considered there might be a level of co-contraction of the knee flexors during the slow speeds, as a type of protective mechanism during slow speeds to prevent excessive shear forces on the knee joint. However, the BF aEMG remained consistent across speeds. It was considered previously to the study that an increase in BF aEMG during the 5 °/sec speed would lead to an increase in knee flexor force resulting in less knee extensor force and subsequently reducing knee shear forces. Since BF muscle activity remained constant and at such a low level of EMG under MVC that hypothesis is rejected. It seems that activation of the BF cannot explain the decrease in force during the 5 °/sec speed compared to the isometric.

It seems that there was a relationship between force and VL aEMG levels. It appears there was decreased muscle activity of the VL concurrent with the decrease in $F_T$ during the 5 °/sec speeds. Figure 9 illustrates a typical $F_T$ and VL and BF aEMG for a single subject for each speed tested. This subject’s data represent the group response across speeds. The subject’s variables plotted in Figure 9 suggest a correlation between force and VL aEMG during the 5 °/sec speed. Notice the decrease in $F_T$ during the 5 °/sec velocity, as well as a decrease in VL aEMG during the 5 °/sec. The single subject $F_T$ decreased by 34 % while VL aEMG decreased by 35 % during the 5 °/sec condition. BF aEMG was not different during any of the eccentric speeds compared to isometric conditions. The group data is similar to the data reported for the single subject in Figure 9. The group $F_T$ and VL aEMG data suggest that muscle activity is inhibited during the 5 °/sec velocity causing a decrease of VL aEMG corresponding to the decrease in $F_T$. That is, when $F_T$ decreases, so does VL aEMG. Additionally, when $F_T$ increases, so does VL aEMG.
aEMG. The possible correlation between force and aEMG only seems to appear at the 55° knee position, because at 65° there was no difference in VL aEMG across speeds.

**EMG-Force Discussion**

The similarity between force and EMG response across eccentric velocities reported in the present study offers evidence of a neural mechanism which regulates force output of the agonist muscles. Despite this apparent relationship, the similarity between force and EMG has been shown to be a complex one. Komi, Linnamo, Silventoinen, and Sillanpaa (2000) tested elbow flexors during speeds of 0, 1, 2, 3, and 4 rad/sec concentrically and eccentrically. Komi et al. (2000) reported an increase in force during concentric action across speeds as well as an increase in muscle activity. During eccentric action Komi et al. (2000) reported no change in force across speeds with a decrease in muscle activity.

![Figure 9. Subject 8 F, VL and BF aEMG results. F decreased by 34 %, VL aEMG decreased by 35 %, and BF aEMG remained unchanged during the 5 °/sec velocity compared to isometric conditions.](image-url)

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Westing et al. (1991) tested the knee extensors during concentric and eccentric loading during speeds of 45, 90, 180, and 360 °/sec. Westing et al. (1991) reported no change in force or muscle activity across speeds. During concentric actions they reported an increase in torque, but with a decrease in muscle activity of the knee extensors. Westing et al. (1991) concluded that these results could be the result of a mechanism causing a reduction in neural drive. They further conclude that such a mechanism is likely to be a result of several afferent and efferent signals sent from joint receptors and Golgi tendon organs (Westing et al., 1991). During eccentric actions, Westing et al. (1991) state that there would be a need to inhibit certain afferent signals sent from the muscle spindle to the agonist muscle due to its sudden and complete stretch. This inhibitory signals could cause the decrease in force during the 5 °/sec condition compared to isometric conditions.

Kellis and Baltzopoulos (1998) tested the knee extensors during eccentric and concentric exercises at speeds of 30, 90, 120, and 150. They reported that muscle activity of the agonist muscles does not increase during all isokinetic conditions (Kellis & Baltzopoulos, 1998). Although they did not report statistical forces across speeds, Kellis and Baltzopoulos (1991) stated that the CNS is not fully activated under certain MVC conditions. They concluded that a neural inhibitory mechanism preventing increases in force during slow speeds of concentric and eccentric MVC may be responsible for the results they reported.

system which is partitioned into three compartments; a high-level controller (HLC), a low-level controller (LLC) and sensory receptors. The HLC is essentially the CNS which generates commands for the muscle to carry out. The LLC receives the command from the HLC and generates an appropriate sequence of muscle actions known as the motor program. Sensory receptors provide afferent feedback during which, the muscle and LLC can modify the motor program. Sensory receptors can also provide feedback to the HLC, so the overall command may change.

Enoka's (1988) schematic can offer evidence of the tension regulating mechanism within the neural pathway of muscle control. It was shown that the decrease in force during the 5°/sec condition compared to the isometric condition only occurs during MVC as compared to submaximal and artificially stimulated contractions. Previous studies (Kellis & Baltzopoulos, 1998; Seger & Thorstensson, 2000; Westing et al., 1991) have proposed that a tension regulating mechanism is active during certain high loading voluntary conditions, particularly under slow eccentric and concentric speeds when MVC are performed. Somewhere along Enoka's schematic, the signal is sent via a motor pathway which is not traveled by the signal during the submaximal or artificially stimulated conditions. The controlling mechanism could be located where several complex pathways of afferent and efferent signals converge. The controlling mechanism may be activated when pathways of the HLC, LLC, and sensory receptors are all active. When these three pathways are activated at the same time, the signals sent from them could converge at the LLC and subsequently activate the controlling mechanism. Both sensory and motor information meet at the LLC from the sensory receptors and HLC.
respectively. The mechanism could be located within the LLC where the motor program is initiated and changed depending on conditions. Certain conditions could produce efferent and afferent signals sent to the LLC activating a controlling mechanism. The activation of the tension regulating mechanism during slow eccentric conditions would explain the observation of reduction muscle activation during 5 °/sec speeds compared to isometric conditions. The reduced muscle activation observed in the current study may be responsible for the decrease in force.

Conclusions

In conclusion, forces are lower during slow eccentric velocities compared to isometric actions. It was observed that VL aEMG are lower during slow eccentric velocities compared to isometric actions at 55°. It was also observed that BF aEMG was not different across test conditions. The decrease in muscle activity of the agonist muscle observed during the 5 °/sec suggests a decrease in the amount of neural input to the agonist muscle. The decrease in muscle activity may be related to a decreased motor unit recruitment. A decrease in the number of motor units may decrease the total number of muscle fibers which contribute to maximal force. The controlling mechanism seems to decrease neural input to the agonist muscle, which results in the decrease in force during the 5 °/sec condition when compared to the isometric condition. Because the decrease in neural input occurs within the nervous system, it is concluded that the controlling mechanism is neurological in nature and responsible for this action. Thus, the research hypothesis is accepted and the null hypothesis is rejected.
Accepted - $H_1$: The mechanism responsible for the decrease in force during slow eccentric isokinetic speeds is neurological in nature.

Rejected - $H_0$: The mechanism responsible for the decrease in force during slow eccentric isokinetic speeds is not neurological in nature.

It is not known how the controlling mechanism inhibits the muscle activity of the agonist muscle during MVC during slow eccentric isokinetic speeds. It may be possible that the controlling mechanism is activated during slow eccentric conditions via efferent signals sent from the CNS and afferent signals sent from muscle and joint sensory organs. Due to high shear forces about the knee generated during MVC slow eccentric contractions, joint receptors (mechanoreceptors) could provide feedback to motor neurons which control the knee extensors. The sensory information feeding back to the motor neurons could be a type of protective mechanism which would not allow the shear forces on the knee, produced under slow MVC eccentric conditions, to approach injurious limits. When the motor neurons receive afferent signals from the mechanoreceptors that the knee is approaching too high of force limits, the motor neurons could recognize this and decrease the amount of electrical signals sent to agonist muscles. The decrease of electrical activity within the agonist muscle, which was observed in the present study, could result in a decrease in the number of motor units activated, causing lower force output. The lower force output, in turn, would protect the knee from excessive shear forces. Thus the knee, and ultimately the body, is prevented from injury.

Although mechanoreceptors could play a role in decreased neural input to the agonist muscle during slow MVC eccentric conditions, it is more likely the controlling
mechanism utilizes simultaneous afferent signals sent from proprioceptive structures including Golgi tendon organs (GTO) and muscle spindles, as well as mechanoreceptors. Both structures could have a significant impact on the amount of force a muscle produces. The muscle spindles signal changes in length, while the GTO signal changes in tension of the muscle. Both of these structures provide afferent feedback about the mechanical state of the muscle to the motor system. Depending on how the controlling mechanism interprets the afferent signals, the correct response to contraction conditions can be performed on the motor neurons and the contracting muscle. If an amount of tension is applied to the knee extensors, and the GTO recognizes the tension to be too high, negative feedback detected by the controlling mechanism can decrease the tension by inhibiting neural input to the knee extensors.

For further studies, recommendation would be made to test different muscle groups at eccentric speeds less than 20 °/sec while simultaneously recording muscle activity. In addition to slow eccentric speeds, it would be recommended to test at concentric speeds less than 20 °/sec. Although this study could not directly identify or locate the controlling mechanism, the results of the present study provide empirical evidence that a neural tension regulating mechanism is active under slow eccentric conditions. Finding the exact location of the controlling mechanism will most likely be discovered by future research which would probably involve invasive procedures.

Limitations

One of the limitations of this study, as well as others studying MVC during one
test session is the effect of fatigue. All studies which test the force-velocity curve, must deal with this issue. Unless there is a very large amount of contractions during the test session, fatigue can be avoided by giving the subject plenty of rest between maximal contractions. This study examined only healthy college age subjects who were previously untrained in isokinetics. It could be possible that the neural controlling mechanism could be altered if regular training at slow eccentric isokinetic velocities. It is known during the first weeks of training, most of the strength increases are a result of neural adaptations (Enoka, 1988). Therefore, if a subject was trained regularly at eccentric velocities of less than 20°, it could be reasonable to expect no decrease in force during the 5 °/sec condition when compared to isometric conditions.

Another limitation to this study is that it remains to be seen if the decrease in force occurs at 5 °/sec because that is the velocity selected, or perhaps the decrease really occurs at 7 °/sec. Due to differences in individuals, it would be difficult to find exactly where the decrease in force occurs. Individuals with a higher percentage of fast twitch muscle fibers could likely show a decrease in force during a different velocity when compared to individuals with a higher percentage of slow twitch muscle fibers.

There is more information left to understand about this controlling mechanism which only seems to be active during slow eccentric speeds. Further and more extensive testing at slow eccentric velocities with concurrent EMG recording could provide a better understanding of where the controlling mechanism is located, and why it is activated during slow eccentric speeds.
APPENDIX A

INFORMED CONSENT FORM
University of Nevada Las Vegas
Informed Consent Statement

MUSCLE PERFORMANCE DURING
SLOW ISOKINETIC SPEEDS

Information
You are invited to participate in a research study investigating the characteristics of the knee extensor and flexor muscles. If there is a previous or current injury to the lower extremity, please notify test investigator or assistant.

Procedures
You will be asked to report to the Sports Injury Research Center for two sessions, which are held on different days. The first session will last approximately 30 minutes. The second session will last approximately one hour. The second session will take place no less than 2 days and no more than 7 days after the first session.

The procedures of the study will be clearly explained to you upon your arrival at the Sports Injury Research Center. Following your consent to participate in the study you will be placed on a knee extensor machine. The specific procedures are as follows:

The first session is a training/orientation session. The purpose of the first session is to let you practice eccentric and concentric exercises. Since the type of exercise you will be asked to complete may take some getting used to, some practice repetitions at a light intensity will be performed to help get the feel for the exercise. You will be asked to complete a series of knee flexion/extension exercises on the knee extensor machine. The knee extensor machine functions similarly to a knee extension device you would find in a weight room. Each set of knee flexion/extension exercises will be completed at different speeds. The machine you will be tested on, controls the speed of movement.

The second session will consist of testing your eccentric and concentric strength. There will be a brief warm up period prior to any testing. These exercises are similar to which were performed during the training session a few days earlier. An electromyography (EMG) unit will be used to measure muscle activity. EMG signals are sent to the processing unit by electrode leads, which are attached to the surface of the skin surrounding the quadriceps and hamstring muscles. The electrodes are used to detect muscle activity. The skin, where the electrodes are to be attached, must be shaved and then cleaned with alcohol. These electrodes are similar to the
type that is used to record EKG, as well as those used in heart rate monitors. The use of the electrodes is a noninvasive procedure.

Following the warm up and EMG setup, you will be asked to complete a series of maximal voluntary effort exercises during knee flexion and extension. The speed of the machine will vary between trials at 5, 10, and 15 °/sec. For each speed, you will be asked to complete 3 trials during eccentric contractions. There will be a total of 9 maximal effort contractions at these speeds. Two maximum isometric contractions will be completed prior to the eccentric and concentric contractions. The isometric contractions will take place at two different knee angles. You will be given at least 3 minutes between sets in order to minimize fatigue during these knee exercises.

Benefits of Participation
Upon completion of this study, you will have gained increase knowledge of your own flexion/extension ability with eccentric exercises. You will also have an increased understanding of the benefits for participating in a research project.

Risks
For the experiment, the risk to the participant is minimal. You will be performing a series of exercises similar to exercises of many strength training programs. You will have a “termination switch” in your hand during the test. If you feel any discomfort during the test, you can push the switch and the exercise will stop immediately. The test investigator and assistants will be monitoring you during the test. If any other problems should arise the test investigator will address the situation. You may experience some soreness in the leg muscles for 24 to 48 hours depending on the your physical activity level. Stretching after the exercise session may help alleviate some of the soreness. You may also experience some skin irritation due to the shaving and cleaning of the area.

Contact
If you have any questions at any time about the study, or if you experience adverse effects as a result of participation in this study, you may contact the researcher, Jon Kwantes at 895-4494 or 697-2160. For questions about your rights as a research subject you may contact the UNLV office of Sponsored Programs at 895-1357.

Participation
Your participation in this study is voluntary, you may refuse to participate in this study or in any part of this study, and you may withdraw at any time, without prejudice to your relations with UNLV. If you withdraw, you should understand
that your data would be destroyed. You may ask any questions about this study prior to its beginning or at any time during the investigation.

**Confidentiality**
No reference will be made in written or oral materials, which could link you to this study. All data will be placed in secure storage in Motor Control Laboratory.

**Consent**
I have read and understand the above information. I agree to participate in this study.

Subject’s Signature   Date

Researcher’s Signature   Date
APPENDIX B

SUBJECT QUESTIONNAIRE
SUBJECT QUESTIONNAIRE
MUSCLE PERFORMANCE DURING SLOW ISOKINETIC SPEEDS

to be completed by participant

Name ____________________________________________

Are you familiar with eccentric muscle contractions? ____________

Are you familiar with isokinetic muscle training? ________________

Do you currently, or have you recently experienced pain (including surgery) in the knee, leg, or thigh which could limit your ability to complete the specified tasks?

_________________________________________________________________

_________________________________________________________________

to be completed by principal investigator

Age ___________ Weight _______________ Height ______

Order of exercises ____________________________________________

Comments for researcher:

_________________________________________________________________
Name _______________________ Subject ________________
Age ____________ Height _______ Weight ________
Orientation Day ____________
Test Day ____________

Order of exercises

Isometric c

Eccentric c

<table>
<thead>
<tr>
<th>Speed</th>
<th>Start Angle</th>
<th>Stop Angle</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>80</td>
<td>40</td>
</tr>
<tr>
<td>10</td>
<td>90</td>
<td>20</td>
</tr>
<tr>
<td>15</td>
<td>90</td>
<td>20</td>
</tr>
</tbody>
</table>

Magnet Placement

1st Magnet __________

2nd Magnet __________
APPENDIX D

INDIVIDUAL SUBJECT DATA RECORDS
Subject 1

<table>
<thead>
<tr>
<th>S1 at 55</th>
<th></th>
<th>S1 at 65</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>VL (μV)</td>
<td>BF (μV)</td>
<td>Force (N)</td>
<td>VL (μV)</td>
</tr>
<tr>
<td>0 °/sec</td>
<td>149.82</td>
<td>17.25</td>
<td>914.50</td>
</tr>
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<td>100.73</td>
<td>13.05</td>
<td>455.55</td>
</tr>
<tr>
<td>-10 °/sec</td>
<td>116.24</td>
<td>16.03</td>
<td>438.63</td>
</tr>
<tr>
<td>-15 °/sec</td>
<td>114.52</td>
<td>15.87</td>
<td>472.24</td>
</tr>
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</table>

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Subject 2

<table>
<thead>
<tr>
<th>Subject 2 at 55</th>
<th>VL (µV)</th>
<th>BF (µV)</th>
<th>Force (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 °/sec</td>
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<td>23.10</td>
<td>1269.02</td>
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<td>110.00</td>
<td>26.80</td>
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<td>-15 °/sec</td>
<td>152.65</td>
<td>28.71</td>
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<table>
<thead>
<tr>
<th>Subject 2 at 65</th>
<th>VL (µV)</th>
<th>BF (µV)</th>
<th>Force (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 °/sec</td>
<td>131.36</td>
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<td>-5 °/sec</td>
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<td>-15 °/sec</td>
<td>154.87</td>
<td>31.35</td>
<td>1410.41</td>
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![Graph showing force and EMG activity for S2 at 55 degrees and S2 at 65 degrees](image-url)
Subject 3

<table>
<thead>
<tr>
<th>S3 at 55</th>
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<tbody>
<tr>
<td>VL (µV)</td>
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</tr>
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<td>-10 °/sec</td>
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</tr>
<tr>
<td>-15 °/sec</td>
<td>23.90</td>
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</table>

S3 at 55 deg

S3 at 65 deg

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Subject 4

<table>
<thead>
<tr>
<th></th>
<th>S4 at 55</th>
<th></th>
<th>S4 at 65</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>VL (µV)</td>
<td>BF (µV)</td>
<td>Force (N)</td>
</tr>
<tr>
<td>0 °/sec</td>
<td>69.21</td>
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<td>957.87</td>
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<td>8.02</td>
<td>553.41</td>
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<td>-10 °/sec</td>
<td>61.84</td>
<td>11.35</td>
<td>816.51</td>
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<td>-15 °/sec</td>
<td>34.86</td>
<td>7.51</td>
<td>482.59</td>
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</tbody>
</table>
Subject 5

### Subject 5 at 55 deg

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<thead>
<tr>
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<th>BF (μV)</th>
<th>Force</th>
</tr>
</thead>
<tbody>
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<td>4.68</td>
<td>630.30</td>
</tr>
<tr>
<td>-5</td>
<td>75.95</td>
<td>4.38</td>
<td>589.59</td>
</tr>
<tr>
<td>-10</td>
<td>74.17</td>
<td>5.03</td>
<td>527.79</td>
</tr>
<tr>
<td>-15</td>
<td>88.43</td>
<td>5.36</td>
<td>663.36</td>
</tr>
</tbody>
</table>

### Subject 5 at 65 deg

<table>
<thead>
<tr>
<th>Velocity (°/sec)</th>
<th>VL (μV)</th>
<th>BF (μV)</th>
<th>Force</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>62.96</td>
<td>4.38</td>
<td>820.28</td>
</tr>
<tr>
<td>-5</td>
<td>75.15</td>
<td>4.83</td>
<td>666.91</td>
</tr>
<tr>
<td>-10</td>
<td>85.13</td>
<td>5.29</td>
<td>640.77</td>
</tr>
<tr>
<td>-15</td>
<td>88.62</td>
<td>5.36</td>
<td>713.32</td>
</tr>
</tbody>
</table>

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Subject 6

<table>
<thead>
<tr>
<th></th>
<th>VL (µV)</th>
<th>BF (µV)</th>
<th>Force (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 °/sec</td>
<td>45.19</td>
<td>8.66</td>
<td>684.32</td>
</tr>
<tr>
<td>-5 °/sec</td>
<td>33.47</td>
<td>7.93</td>
<td>324.31</td>
</tr>
<tr>
<td>-10 °/sec</td>
<td>39.33</td>
<td>8.53</td>
<td>387.00</td>
</tr>
<tr>
<td>-15 °/sec</td>
<td>48.62</td>
<td>8.85</td>
<td>510.71</td>
</tr>
</tbody>
</table>

S6 at 55 deg

<table>
<thead>
<tr>
<th></th>
<th>VL (µV)</th>
<th>BF (µV)</th>
<th>Force (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 °/sec</td>
<td>42.63</td>
<td>8.06</td>
<td>679.36</td>
</tr>
<tr>
<td>-5 °/sec</td>
<td>38.39</td>
<td>8.55</td>
<td>403.78</td>
</tr>
<tr>
<td>-10 °/sec</td>
<td>37.57</td>
<td>8.41</td>
<td>291.60</td>
</tr>
<tr>
<td>-15 °/sec</td>
<td>47.05</td>
<td>8.04</td>
<td>387.21</td>
</tr>
</tbody>
</table>

S6 at 55 deg

S6 at 65 deg

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Subject 7

<table>
<thead>
<tr>
<th>Velocity (deg/sec)</th>
<th>S7 at 55 deg</th>
<th>S7 at 65 deg</th>
</tr>
</thead>
</table>

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Subject 8

<table>
<thead>
<tr>
<th>V L (μV)</th>
<th>B F (μV)</th>
<th>Force (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 °/sec</td>
<td>48.83</td>
<td>6.13</td>
</tr>
<tr>
<td>-5 °/sec</td>
<td>32.12</td>
<td>3.33</td>
</tr>
<tr>
<td>-10 °/sec</td>
<td>50.83</td>
<td>6.50</td>
</tr>
<tr>
<td>-15 °/sec</td>
<td>41.82</td>
<td>5.52</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>V L (μV)</th>
<th>B F (μV)</th>
<th>Force (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 °/sec</td>
<td>55.76</td>
<td>6.30</td>
</tr>
<tr>
<td>-5 °/sec</td>
<td>38.77</td>
<td>3.70</td>
</tr>
<tr>
<td>-10 °/sec</td>
<td>46.71</td>
<td>6.08</td>
</tr>
<tr>
<td>-15 °/sec</td>
<td>52.38</td>
<td>6.15</td>
</tr>
</tbody>
</table>

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Subject 9

<table>
<thead>
<tr>
<th>Velocity (°/sec)</th>
<th>VL (µV)</th>
<th>BF (µV)</th>
<th>Force (N)</th>
<th>Velocity (°/sec)</th>
<th>VL (µV)</th>
<th>BF (µV)</th>
<th>Force (N)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0°/sec</td>
<td>46.25</td>
<td>3.13</td>
<td>589.96</td>
<td>0°/sec</td>
<td>57.20</td>
<td>5.44</td>
<td>696.00</td>
</tr>
<tr>
<td>-5°/sec</td>
<td>34.52</td>
<td>2.59</td>
<td>448.92</td>
<td>-5°/sec</td>
<td>29.74</td>
<td>2.68</td>
<td>485.85</td>
</tr>
<tr>
<td>-10°/sec</td>
<td>40.66</td>
<td>3.05</td>
<td>497.14</td>
<td>-10°/sec</td>
<td>26.59</td>
<td>2.86</td>
<td>381.59</td>
</tr>
<tr>
<td>-15°/sec</td>
<td>25.21</td>
<td>2.12</td>
<td>368.41</td>
<td>-15°/sec</td>
<td>21.47</td>
<td>2.23</td>
<td>385.81</td>
</tr>
</tbody>
</table>

**S9 at 55 deg**

![Graph showing force and EMG activity as a function of velocity for S9 at 55 deg.]

**S9 at 65 deg**

![Graph showing force and EMG activity as a function of velocity for S9 at 65 deg.]

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APPENDIX E

TABLES OF GROUP AVERAGES FOR $F_T$ AND aEMG
Mean and standard deviations for $F_T$, VL aEMG, and BF aEMG of the nine subjects at 55°.

<table>
<thead>
<tr>
<th>Dep. Measure</th>
<th>0 (°/sec)</th>
<th>-5 (°/sec)</th>
<th>-10 (°/sec)</th>
<th>-15 (°/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$F_T$ (N)</td>
<td>843 ± 215.8</td>
<td>559.2 ± 184</td>
<td>610.6 ± 207.4</td>
<td>613.7 ± 251</td>
</tr>
<tr>
<td>VL aEMG (µV)</td>
<td>84.7 ± 41.1</td>
<td>65.8 ± 38.4</td>
<td>65.5 ± 32.5</td>
<td>65.9 ± 44.3</td>
</tr>
<tr>
<td>BF a EMG (µV)</td>
<td>9.8 ± 6.4</td>
<td>9.5 ± 8.4</td>
<td>10.5 ± 7.2</td>
<td>9.3 ± 8.2</td>
</tr>
</tbody>
</table>

Mean and standard deviations for $F_T$, VL aEMG, and BF aEMG of the nine subjects at 65°.

<table>
<thead>
<tr>
<th>Dep. Measure</th>
<th>0 (°/sec)</th>
<th>-5 (°/sec)</th>
<th>-10 (°/sec)</th>
<th>-15 (°/sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$F_T$ (N)</td>
<td>877.4 ± 184</td>
<td>573.6 ± 236</td>
<td>575.5 ± 196.8</td>
<td>646.5 ± 325</td>
</tr>
<tr>
<td>VL aEMG (µV)</td>
<td>68.9 ± 32</td>
<td>60.6 ± 31.9</td>
<td>60.1 ± 34.1</td>
<td>67.6 ± 45.9</td>
</tr>
<tr>
<td>BF a EMG (µV)</td>
<td>9.1 ± 6.7</td>
<td>9.2 ± 7.9</td>
<td>9.6 ± 7.2</td>
<td>9.8 ± 9</td>
</tr>
</tbody>
</table>
DATE: March 12, 2001

TO: Jonathan M. Kwantes
    Kinesiology
    MS 3034

FROM: Dr. Jack Young, Chair
    UNLV Biomedical Sciences Institutional Review Board

RE: Status of Human Subject Protocol Entitled:
    "Muscle Performance During Slow Isokinetic Speeds"

OPRS# 504s0101-225

This memorandum is official notification that the UNLV Biomedical Sciences Institutional Review Board approved the protocol for the project listed above and work on the project may proceed. This approval is effective from the date of this notification and will continue for a period of one year.

Should the use of human subjects described in this protocol continue beyond a year from the approval date, it will be necessary to request an extension.

If you have any questions or require any assistance, please contact the Office for the Protection of Research Subjects at 895-2794.

cc: OPRS File
REFERENCES


78


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motor units: Possible roles of exercise and supraspinal reflexes. 
*Electroencephalography and Clinical Neurophysiology.* 38, 245-254.


VITA

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