

Neuromotor Mechanisms Involved in the Recovery from Local Muscular Fatigue

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ABSTRACT

The phenomenon of over-recovery consists of a participant's maximal force levels returning to values above initial levels. The present study examined the presence and causes of over-recovery following local muscular fatigue. Fourteen males completed two fatigue protocols consisting of maximal isometric dorsiflexion contractions. Upon completion of the fatigue protocol participants' force was monitored over a 15 minute recovery period. Dorsiflexion force and surface electromyography (sEMG) from the tibialis anterior and soleus were monitored concurrently.

Following the two fatigue conditions (10 and 20% force decrement) force recovered to 100.5 and 99.5% of initial levels for each condition, respectively. Surface EMG root-mean-square amplitude and MPF exhibited changes consistent with a warm-up effect. It was concluded that over-recovery was not present in the tibialis anterior following a local muscular fatigue. However, the return in force to initial values, rather than a persistent decrement as normally observed, was mediated by the warm-up effect.

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CHAPTER 1: DEVELOPMENT OF THE PROBLEM

1.1 Introduction

Fatigue and recovery of skeletal muscle are complementary events occurring consistently in daily life. Muscular fatigue can be defined as “any exercise-induced reduction in the maximal capacity to generate force or power output” (Vollestad, 1997, p. 220). Recovery from fatigue is the period immediately following fatigue where activity has ceased, or is minimal enough to no longer cause fatigue. The mechanisms behind fatigue and recovery vary greatly depending on the type of activity, the duration and intensity of activity, and the characteristics of the muscle(s) involved, to name a few. This thesis is primarily concerned with potential neuromuscular mechanisms involved in recovery from local muscular fatigue caused by intermittent, short duration maximal isometric contractions.

Walter Kroll (1967a; 1967b; 1971a; 1971b) examined the effect of varied inter-trial rest periods and the level of maximal strength of participants on fatigue and recovery. Both studies observed a relatively new phenomenon at that time, termed “over-recovery”. Over-recovery occurs when force levels, recorded during a period of recovery after fatigue, return to levels higher than the participant’s initial (pre-fatigue) maximum. Kroll (1967b; 1971b) found over-recovery (101-118%) in both male (1967b) and female (1971b) ‘low-strength’ participants, in comparison to initial maximal voluntary contraction force levels. This phenomenon was also shown by Stull and Clarke (1971) with recovery percentages of 107% participants’ initial strength after 235 seconds of recovery from 3 minutes of a maximal, rhythmic handgrip exercise. Thus, the over-recovery phenomenon is not an isolated finding attributed to a single investigator. Stull

and Clarke (1971) offered no explanation for the over-recovery phenomenon while Kroll (1967b; 1971b) could only rule-out potential psychological factors such as motivation and perceived exertion, because low-strength participants exhibited fatigue and recovery curves similar to those of their middle- and high-strength counterparts.

1.1.1 Potential Mechanisms

Motor learning has been repeatedly shown to increase fatigue resistance as assessed by the mean force level across trials (Kroll, 1965; Gabriel & Kroll, 1991; Gabriel, Basford, & An, 2001). Thus, one potential mechanism involves a motor learning effect that is associated with massed-practice due to serial contractions within a test session (Calder & Gabriel, 2007). This would be demonstrated as an increase in neural drive to the agonist muscle group (Gabriel, Basford, & An, 2001; Rutherford & Jones, 1986) or a reduction in antagonist co-activation (Carolan & Cafarelli, 1992). In this paradigm, the contractions that occur during the recovery phase are analogous to a retention test.

An equally possible alternative explanation is based on post-activation potentiation. Post-activation potentiation (PAP) is an increase in muscle twitch force and low frequency force following the performance of brief maximal voluntary contraction (Hamada et al., 2000; Sale, 2004). PAP is hypothesized to be caused by a myosin regulatory light chain phosphorylation-mediated increase in the calcium-sensitivity of force development (Metzger, Greaser, & Moss, 1989; Persechini, Stull, & Cooke, 1985; Sweeney & Stull, 1990). Inglis and colleagues (2011) have demonstrated that the neuromuscular consequence of post-activation potentiation was a decrease in motor unit discharge rates for the same level of force under control conditions. Presumably, a

comparable summation of twitch forces with lower motor unit discharge rates was possible because their individual magnitudes were greater. The co-existence of fatigue and post-activation potentiation (Hamada et al., 2003) may support an increase in twitch force during recovery as the neural and metabolic consequences of fatigue are alleviated by rest.

A more likely explanation for over-recovery is the potentiation of high-frequency force following a warm-up protocol. Since PAP has been found to potentiate only low-frequency and twitch force it may not be directly responsible for the increase in a maximal voluntary contraction even if it is present in twitch force following a conditioning stimulus. Force increases following a warm-up protocol have been found in both animal and human muscles following a conditioning stimulus similar to PAP (Baudry & Duchateau, 2007; Bruton et al., 1996; Bruton et al., 1997). This 'warm-up' results in potentiation of high-frequency tetanic force and a greater rate of force development in both high-frequency tetanic and voluntary contractions (Baudry & Duchateau, 2007; Bruton et al., 1996; Bruton et al., 1997). The cause of force increases due to a warm-up protocol is not confirmed but it has been speculated to be caused by an increase in inorganic phosphate (P_i) buffering (Bruton et al., 1997). Following a contraction there is an increase in P_i which is a limiting factor of force production due to limited ATP hydrolysis leading to a decreased number of active cross-bridges (Potma et al., 1995). This build up of P_i depresses force production by reducing myofibrillar Ca^{2+} sensitivity (Brandt et al., 1982; Fryer et al., 1995; Kentish, 1986) and hindering the transition of cross-bridges from low-force (weak) to high-force (strong) states (Westerblad et al., 2002). An increase in P_i buffering may depress P_i to levels below the

initial contraction allowing for greater force production and/or a greater rate of force development (Bruton et al., 1997).

1.2 Purpose

The purpose of this thesis was to investigate potential mechanisms involved in the over-recovery phenomenon. Participants were administered a local muscular fatigue protocol consisting of maximal isometric contractions of the tibialis anterior on two separate test sessions, each corresponding to a different target level of percent force decrement. Recovery was assessed by additional contractions at regular intervals following the last contraction of the fatigue protocol while force and surface electromyographic (sEMG) activity were monitored concurrently throughout the entire test session.

1.3 Hypotheses

1. If over-recovery is present due to potentiation, force levels greater than baseline will be seen following a low level of fatigue (Condition 1: 10% strength decrement versus Condition 2: 20% strength decrement).
 - 1a. Potentiation effects will be evident as either an increase in force or similar force level during the recovery period, compared to baseline. The recovery force will be accompanied by a decrease in mean power frequency of the tibialis anterior sEMG signal while root-mean-square sEMG amplitude remains unchanged. Twitch contractions during the recovery period will also exhibit an increase.
2. If over-recovery is present due to an alteration in neuromotor coordination control mechanisms, force levels greater than baseline will be seen following both levels

of fatigue (Condition 1: 10% strength decrement and Condition 2: 20% strength decrement).

- 2a. Neuromotor coordination control mechanisms can involve one or more of the following alterations: (a) a decrease in soleus antagonist co-activation as measured by root-mean-square sEMG amplitude; (b) an increase in tibialis anterior agonist root-mean-square sEMG amplitude; and/or (c) an increase in extensor digitorum longus synergistic root-mean-square sEMG amplitude.

1.4 Significance of the Study

The proposed research has both theoretical and practical applications. While the study of local muscular fatigue is extensive, the need exists for a deeper understanding of recovery from fatigue. The potential neural and/or muscular mechanisms involved in recovery from local muscular fatigue must first be identified before they can be manipulated, optimized and refined for practical applications.

For example, resistive exercise is a relatively new intervention to improve functional capacity in patients suffering from multiple sclerosis (White et al., 2004). It is possible that one recommendation for therapeutic resistive exercise is to perform serial contractions until a specific percent decrement in strength has been achieved, to enhance neuromuscular adaptations and fatigue resistance due to potentiation. Alternatively, the depth of local muscular fatigue may be irrelevant for training-related adaptations in neuromotor coordination control mechanisms, which have also been shown to increase fatigue resistance. Therefore, a search for mechanisms underlying over-recovery will facilitate development of the best – and not just better – kinds of training programs.

1.5 Assumptions

1. Maximal voluntary contractions are representative of the participants' upper limit of muscular strength.
2. Participants generated maximal effort contractions throughout the duration of the fatigue protocol.
3. The sEMG interference pattern is an indirect measure of neural drive to the muscle and reflects the combined effects of motor unit recruitment and discharge rates.
4. The neuromotor coordination control mechanisms investigated: an increase in agonist and synergist muscle activation and a decrease in antagonist co-activation, are measures of muscle coordination.
5. Potentiation can be measured as an increase in twitch force while voluntary contractions exhibit as a decrease in the mean power frequency of the sEMG signal as an indirect measure of motor unit discharge rates.

1.6 Delimitations

1. This study included only right-foot dominant college age (18-25 years) male participants.
2. Only one joint action was studied which consisted of dorsiflexion at the ankle.
3. The sEMG activity of only the tibialis anterior, extensor digitorum longus, and soleus muscles of the lower leg was studied.
4. Only two mechanisms were investigated: (a) alterations in neuromotor coordination control and (b) post-activation potentiation.
5. Only isometric contractions were used.

1.7 Limitations

1. Since only right-foot dominant college age male participants were tested in the proposed study, the results may not apply to non-preferred limbs, to females, or to humans of a different age group.
2. Since only one joint action was studied, consisting of maximal isometric dorsiflexion at the ankle, the results may not apply to complex actions at multiple joints.
3. Since this study only recorded sEMG activity from the tibialis anterior, extensor digitorum longus, and soleus muscles, the role of other muscles of the lower leg was not investigated.
4. Since only two potential mechanisms were investigated, the importance of other factors involved in the over-recovery process were not assessed.
5. Since only isometric contractions were used, the results may not apply to isotonic or eccentric actions of the muscle as occur during normal human movement.

CHAPTER 2: REVIEW OF LITERATURE

2.1 Background

2.1.1 Anatomy of the Tibialis Anterior

Skeletal muscle is the composition of contractile tissues to generate movement. Skeletal muscle is composed of muscle fibres and surrounded by fascia that extends beyond the length of the muscle and becomes tendon to connect muscles to bones (Saladin, 2007, pp. 322-325). The focus of this review will be primarily on the tibialis anterior, and secondarily on the extensor digitorum longus as a synergist and the soleus as an antagonist (see Figure 1).

The tibialis anterior originates on the upper half of the lateral and anterior surfaces of the tibia. It is a single joint muscle, inserting on the medial side of the first cuneiform and the first metatarsal. The main actions of the tibialis anterior are dorsiflexion and inversion (Saladin, 2007, p. 375). It is a parallel-fibre muscle composed mainly of type I slow twitch fibres (Holmback, Porter, Downham, Andersen, & Lexell, 2003). The tibialis anterior is innervated by the deep peroneal nerve, which also innervates the extensor digitorum longus and extensor hallucis longus (see Figure 2). The extensor digitorum longus and extensor hallucis longus are synergists to the tibialis anterior during dorsiflexion. The extensor digitorum longus is a pennate muscle originating at the lateral condyle of the tibia and the upper 2/3rds of the shaft of the fibula. Its tendon crosses the ankle joint and fans to insert on the four lateral phalanges. The extensor hallucis longus originates on the middle 1/3rd of the fibula and inserts on the distal phalanx of the big toe (Saladin, 2007, p. 374).

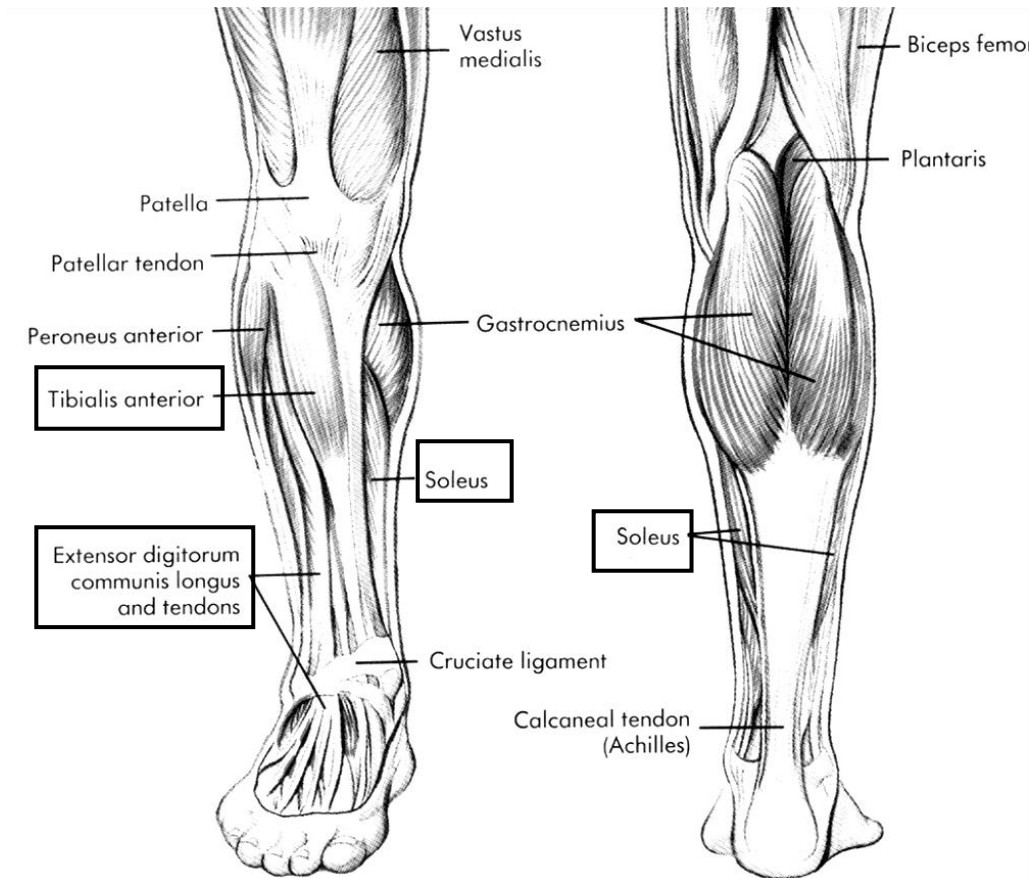


Figure 1. Anatomy of the lower leg muscles. Modified from Anthony, C.P. and Kolthoff, N.J. (1975). *Textbook of anatomy and physiology* (9th ed.). St. Louis: Mosby.

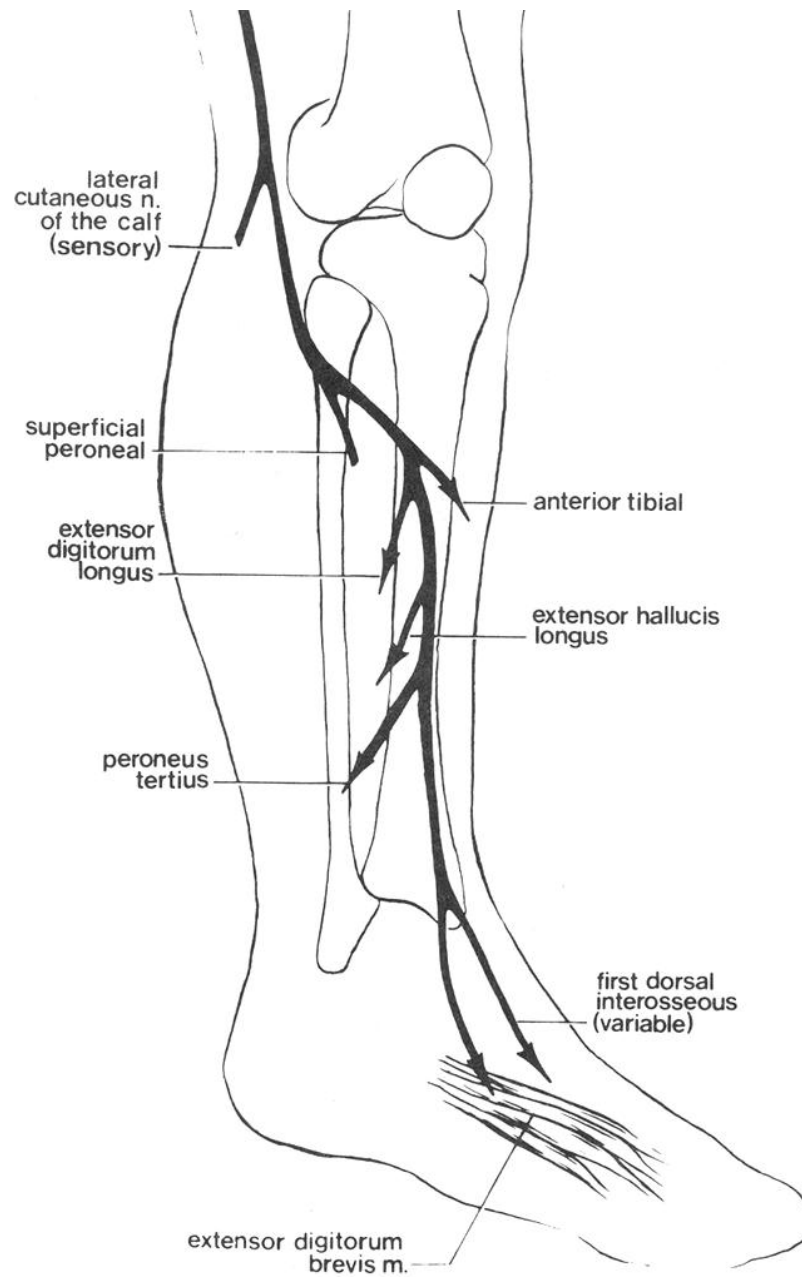


Figure 2. Innervation of the tibialis anterior and extensor digitorum longus by the deep peroneal nerve (branched from the common peroneal nerve). Johnson, E.W. (Ed.). (1980). *Practical Electromyography*. Baltimore, MD: Williams & Wilkins, Figure 9.27, page 249.

As mentioned above, the tibialis anterior is composed mainly of type I slow twitch (or slow oxidative) fibres (Holmback et al., 2003). These fibres have a rich concentration of capillaries for aerobic respiration and therefore, their primary role is to maintain steady contractions (Saladin, 2007, p. 429). Holmback et al. (2003) found that the proportion of type I fibres in the dorsiflexion muscles (tibialis anterior and extensor digitorum longus) was 76.9 and 77.8% for females and males, respectively. This agrees with Johnson and colleagues' (1973) autopsy results, which found an average of 73.1% of type I fibres in the tibialis anterior and 87.7% of type I fibres in the soleus.

Henriksson-Larsen, Lexell, and Sjostrom (1983) showed that the fibre type distribution in the tibialis anterior was not random but rather that there was a greater concentration of type II fibres deep in the tibialis anterior (30-50% compared to 12-25%) and predominantly type I fibres in the surface portion of the tibialis anterior. Similarly, Mesin, Merlo, Merletti and Orizio (2010) determined that large-diameter muscle fibres appeared to be greater in concentration deep in the tibialis anterior compared to the surface.

The action of the tibialis anterior is also influenced by the position of the ankle joint as it affects the length of the muscle fibres. As initially described by Blix (1894), the length-dependent aspect of force is related to the number of cross bridges formed in the muscle fibres as a function of the extent of overlap that is present between actin and myosin at a certain muscle length. Marsh, Sale, McComas, and Quinlan (1981) found that maximum torque from the tibialis anterior was produced at 10° of plantar flexion during both stimulation of the peroneal nerve and maximal voluntary contractions. Similar studies have found that force increases continuously as the ankle joint moves further into

plantar flexion (Frigon, Carroll, Jones, Zehr, & Collins, 2007; Maganaris, 2001). While these studies did not offer an optimum length for the tibialis anterior, one explanation presented by Marsh et al. (1981) for the inability to determine optimum length is the large series-elastic component that is present due to the length of the tibialis anterior tendon.

2.1.2 Anatomy of Skeletal Muscles

The functional unit of the neuromuscular system is the motor unit (see Figure 3), which is composed of a single alpha-motor neuron and all the muscle fibres that it innervates (Saladin, 2007, p. 412, Staudenmann, Roeleveld, Stegeman, & van Dieen, 2010). Muscle force production is dependent on the number of active motor units and the rate at which they fire, both of which are regulated by the nervous system (Broman, De Luca, & Mambrito, 1985b). The number of motor units in the tibialis anterior is estimated to be approximately 120-150 in healthy, young adults (Boe, Dalton, Harwood, Doherty, & Rice, 2009; McNeil, Doherty, Stashuk, & Rice, 2004) with an innervation ratio (number of muscle fibres per motor unit) of approximately 124 fibres (Gath & Stalberg, 1981). The average firing rate for the same population ranges from approximately 12.2 to 28 Hz, depending on the level of the contraction (10-75% maximal voluntary contraction) (Boe et al., 2009; Connelly, Rice, Roos, & Vandervoort, 1999; McNeil et al., 2004).

Motor unit size refers to the number of muscle fibres innervated by a single alpha neuron. This is termed the innervations ratio and ranges from approximately 20 to 2000 fibres per motor unit in human skeletal muscle (Buchthal & Schmalbruch, 1980; Christensen, 1959). Henneman's Size Principle states that motor units are recruited in an orderly fashion from small (low threshold) to large (high threshold) during the gradation of muscle force (Broman et al., 1985b; Feireisen, Duchateau, & Hainaut, 1997;

Henneman, 1957; Kamen, 2004). However, motor unit recruitment and firing rate act together to regulate force (De Luca et al., 1982; Kukulka & Clamann, 1981). The use of firing rate to modulate recruitment was examined by Broman et al. (1985b), who found an inhibitory effect. It was speculated that a decrease in firing rates occurred to diminish their contribution to the force output thereby causing the recruitment of new motor units to maintain the desired force level.

Recruitment and rate coding were examined by Kukulka and Clamann (1981), and De Luca et al., (1982) in small (adductor pollicis and first dorsal interosseus) and large (biceps and deltoid) muscles. Both research groups observed that motor unit recruitment was responsible for force increases until approximately 40 to 50% of maximal voluntary contraction force in the small muscle as opposed to 80 to 90% in large muscles. The percent maximal voluntary contraction force interval over which new motor units are recruited is called the “recruitment range”. After this point recruitment of new motor units ceases and firing rates alone are responsible for further force increases. The firing rate of each motor unit begins slowly and increases to achieve required force levels (see Figure 4). Erim et al. (1996) examined motor unit recruitment and firing rates in the tibialis anterior and found patterns coinciding with three distinct regions of a maximal contraction. The first region, from 10 to 20% of maximal force, includes the recruitment of lower threshold motor units and a rapid increase in firing rates as force increases. The second region consists of increased recruitment, firing rates increasing linearly with force, and the fusion of twitches for most motor units. Finally, the third region, occurring from 70% to 100% maximal force, consists of rapid increases in both lower and higher threshold motor units firing rates to achieve maximal force.

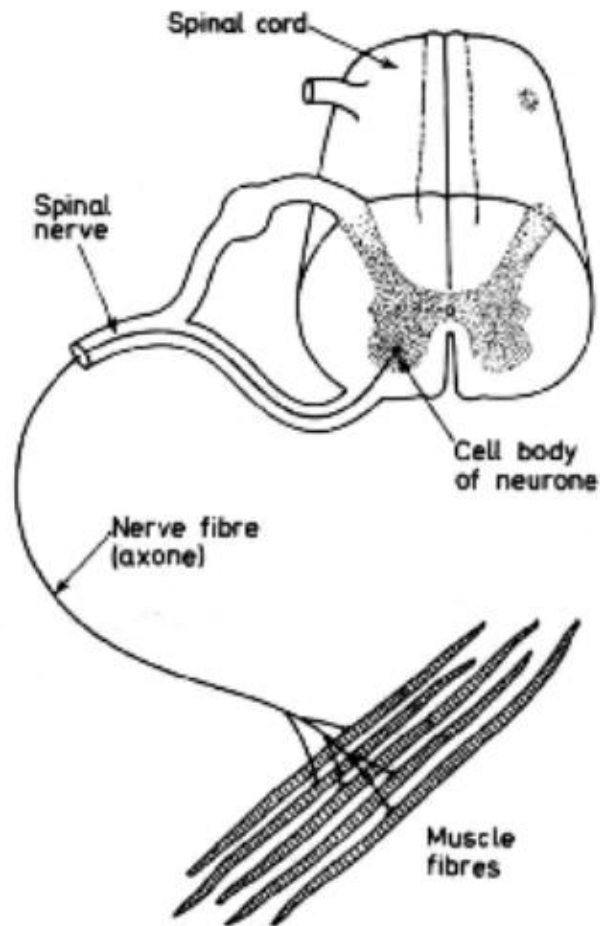


Figure 3. Anatomy of a single motor unit: one alpha motor neuron and all the muscle fibres that it innervates. Basmajian, J.V., and De Luca, C.J. (1985). *Muscles Alive: Their function revealed by electromyography* (5th ed.). Baltimore, MD: Williams & Wilkins.

Figure 1.7, page 12.

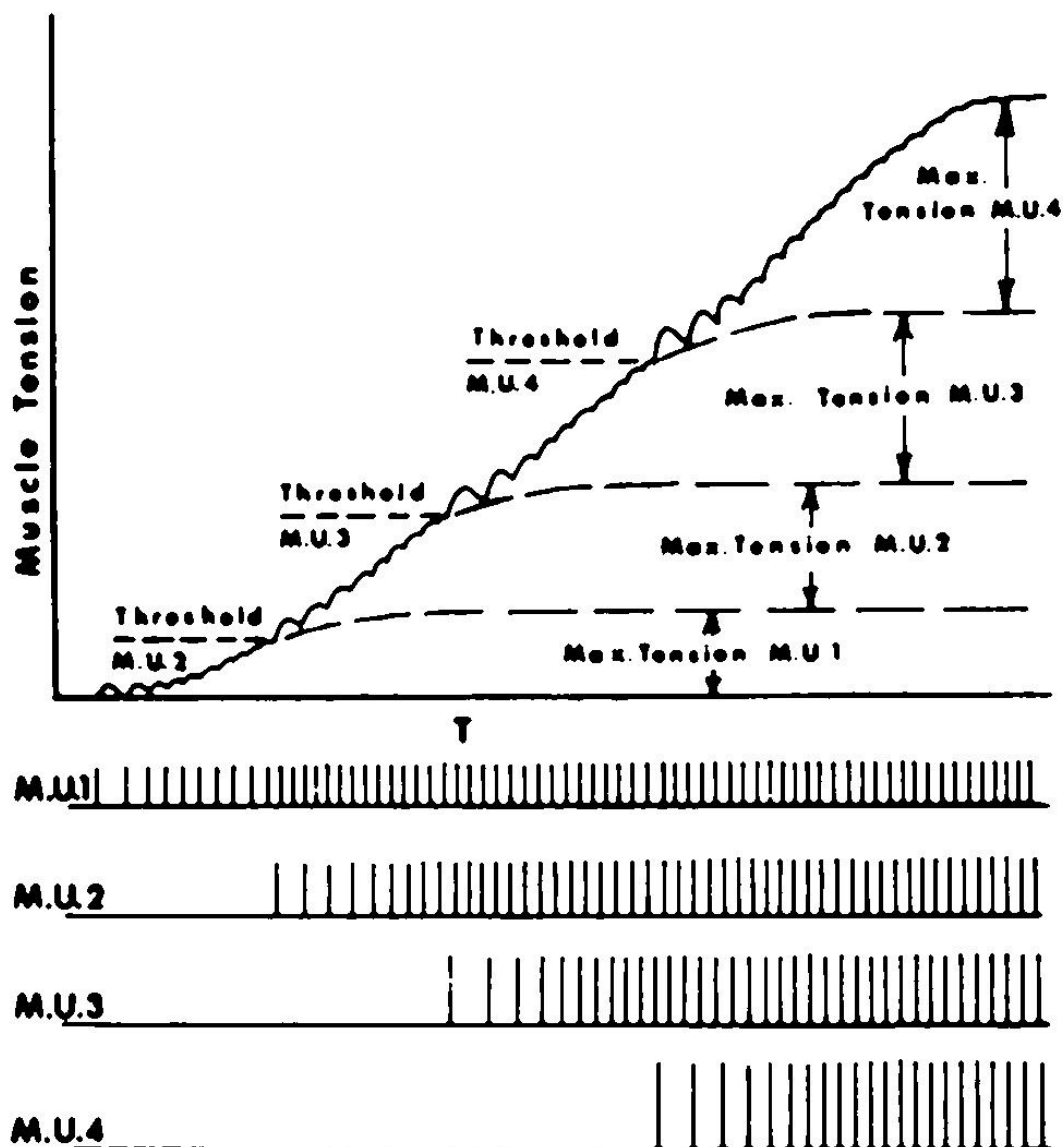


Figure 4. Firing Rates of single motor units. As muscle tension increases additional motor units are recruited and the firing rates of active motor units increase to produce the desired force level. Winter, D. (2009). *Biomechanics and Motor Control of Human Movement* (4th ed.). Hoboken, NJ: John Wiley and Sons Inc. Figure 9.3, page 227.

Muscle force is generated as a result of innervation from the nervous system. The point where the nervous system (motor neurons) and skeletal muscle (muscle fibres) converge is known as the neuromuscular junction (Saladin, 2007, pp. 412-415). If an impulse reaching the neuromuscular junction is large enough to reach the threshold for excitation, a muscle fibre action potential will propagate in either direction along the fibres innervated by that motor unit (Masuda, Miyano, & Sadoyama, 1983a). Each motor unit creates motor unit action potentials, which are composed of the individual muscle fibre action potentials (see Figure 5). The repetitive firing of these motor unit action potentials create a train which are then added together to produce the interference pattern (see Figure 6).

2.1.3 Surface Electromyography

Surface EMG (sEMG) is the application of electrodes on the skin's surface over a particular muscle, with the intention of recording the summation of electrical activity produced by motor units within the muscle (Mesin et al., 2010; Petrofsky, 1981; Staudenmann et al., 2010). The two main electrode configurations in sEMG are monopolar and bipolar. A bipolar configuration, which was used for this thesis, consists of two active electrodes placed side by side over the muscle belly. Each electrode records the muscle activity producing a differential recording by the amplifier, which reduces the common mode noise (Roeleveld, Stegeman, Vingerhoets, & Van Oosterom, 1997). A ground electrode is also used for both noise reduction and safety purposes. The ground electrode minimizes noise recorded from electrical equipment in the area, such as lights and computers. It is also used to shunt any excess electrical activity that may travel from the equipment to the human body in the event of an electrical shortage. The human body

acts like an antenna and the ground shunts electromagnetic interference to the equipment, resulting in a stable isoelectric reference for the recording electrodes (Clancy, Morin, & Merletti, 2002).

When using a bipolar electrode configuration the electrical activity of the muscle is recorded as a signal a few milliseconds apart as it passes under the two electrodes (Staudenmann et al., 2010). This time difference is affected by the distance between the two electrodes, referred to as interelectrode distance. The greater the interelectrode distance, the larger the amount of time between the two signals, and the greater the recording area. An interelectrode distance of 20 mm for bipolar electrode configurations is recommended by SENIAM (the European project on ‘Surface EMG for Non-Invasive Assessment of Muscles’) (Day, 2002; Mesin, Merletti, & Rainoldi, 2009). The ideal placement for bipolar electrodes is to have the two electrodes parallel to the direction of the muscle fibres, slightly distal to the motor point (Mesin et al., 2009). The motor point is an area of densely populated motor end plates; it can be identified electrically where the smallest amount of stimulation will produce a muscle twitch. This point is often located in the innervation zone (Masuda, Miyano, & Sadoyama, 1983b), where the nerve innervates the muscle and motor unit action potentials originate and propagate bidirectionally (Mesin et al., 2009). By placing both electrodes distal to the motor point it can be ensured that unidirectional motor unit action potentials will be recorded (Mesin et al., 2009).

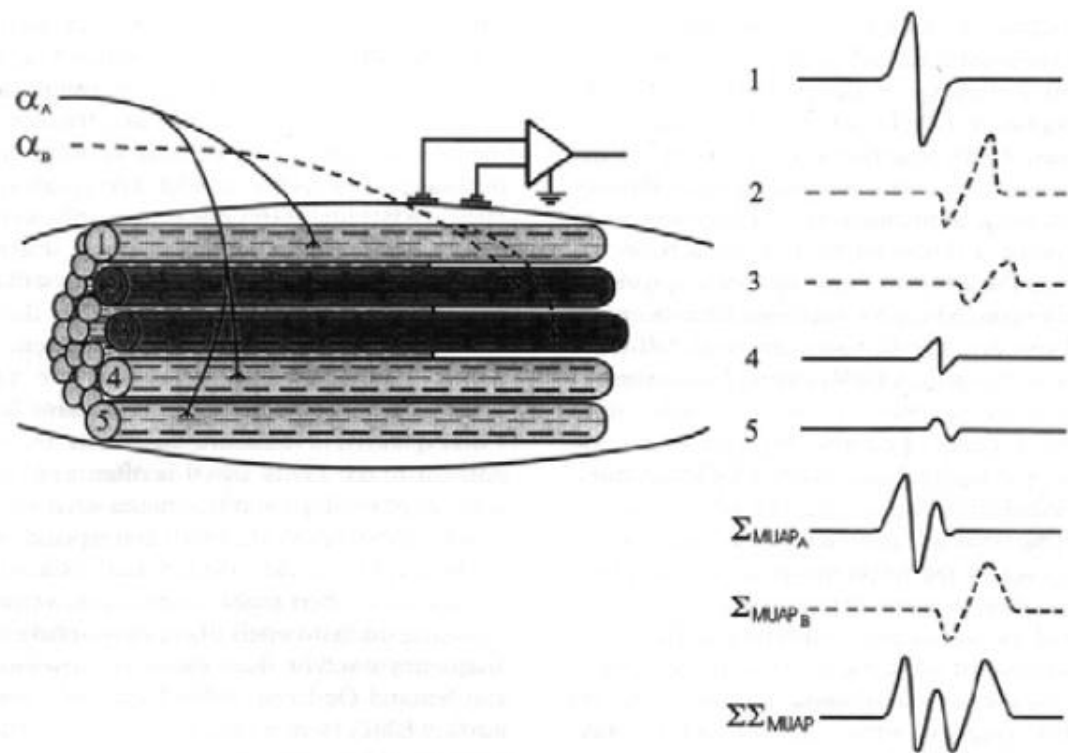


Figure 5. Surface electromyography recorded from two motor units. Motor unit A contains three muscle fibres which produce muscle fibre action potentials 1, 4, and 5; and combine to produce motor unit action potential A. Motor unit B contains two muscle fibres which produce muscle fibre action potentials 2, and 3; and combine to produce motor unit action potential B. The sum of both motor unit action potentials creates an interference pattern. Kamen, G. & Caldwell, G.E. (1996). Physiology and interpretation of the electromyogram. *Journal of Clinical Neurophysiology*, 13(5), 366-384. Figure 1, page 368.

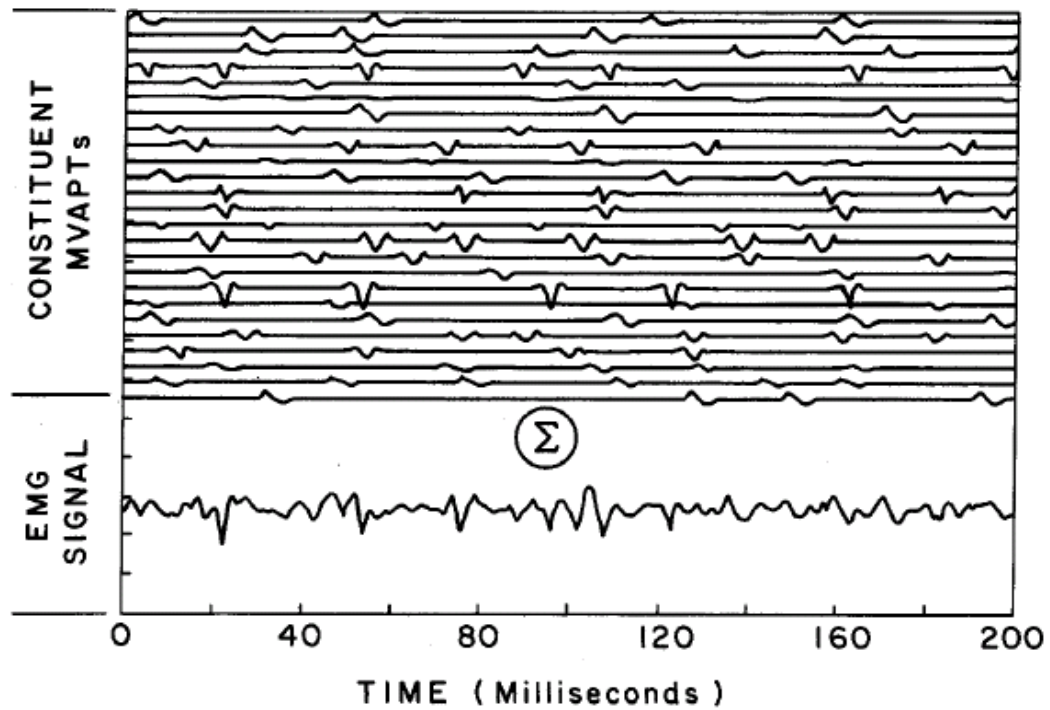


Figure 6. Motor unit action potential trains composed of the repetitive firing of multiple motor unit action potentials. The summation of trains produces an EMG signal.

Basmajian, J.V., and De Luca, C.J. (1985). *Muscles Alive: Their function revealed by electromyography* (5th ed.). Baltimore, MD: Williams & Wilkins, Figure 3.9, page 81.

When using sEMG there are several limitations that must be acknowledged or accounted for. One limitation with sEMG is the assumption that electrodes are recording electrical activity from only one muscle but this is not necessarily the case (De Luca & Merletti, 1988). Electrical activity recorded from nearby muscles is termed cross-talk. The factors affecting cross-talk include muscle size, subcutaneous tissue thickness, interelectrode distance, and neighbouring muscle proximity (De Luca & Merletti, 1988; Staudenmann et al., 2010). Body composition can also affect the amplitude of the sEMG signal (Mesin et al., 2010; Nordander et al., 2003; Staudenmann et al., 2010). Nordander et al. (2003) compared sEMG amplitude to skinfold thickness and found a negative correlation of $r = -0.82$. While this limitation cannot be controlled, it can be accounted for by obtaining anthropometric measurements and skin-fold thickness when possible (Nordander et al., 2003; Staudenmann et al., 2010). Impedance of the electrical signal to the electrodes is not only found through subcutaneous tissue but also through the skin itself. Dirt, dead skin cells, skin oils, soaps and lotions will all contribute to skin-electrode input impedance. The use of electrode gel assists in minimizing skin-electrode input impedance by creating salt bridging for the signal to pass easily through. Although it is advised that impedance be lower than 10 KOhm, it is most important to ensure that impedance between electrodes, and between testing sessions, is balanced for proper signal comparison (Day, 2002).

Another source of error in sEMG is related to wires that carry the signal from the electrodes to the amplifiers. Since the electrical activity travels through these wires it is crucial to keep them as short as possible to minimize resistance. Movement of the wires must also be controlled by securing them to the limb or an immovable object.

Movement artifact can also be produced at the skin-electrode interface by muscle or skin movement under the electrode (De Luca, Gilmore, Kuznetsov, & Roy, 2010). This is minimized with double sided tape to hold electrodes firmly to the skin's surface.

Electrode gel also assists in decreasing movement artifact by creating a flexible salt bridge from the skin's surface to the recessed surface of the passive electrode. This allows for the greater transfer of ions through the path of least resistance from the skin to the recording surface of the electrode.

Bandwidth filtering is used to cut out noise from extraneous electrical activity and movement. A high pass filter eliminates low frequencies, which can be caused by wire or skin movement, a low pass filter cuts out high frequencies beyond the span of muscle activity. The combination of a high and low pass filter produces a bandwidth filter (see Figure 7). De Luca et al. (2010) found that movement artifact influenced the shape of a sEMG signal until approximately 20 Hz, and therefore suggested that this be the high pass cut off for minimizing this artifact. High-pass filtering can be lowered to 5 or 10 Hz if measures are taken to minimize artifact (Day, 2002). The low pass filter level is the point at which the amplitude of noise surpasses the amplitude of the muscle activity. De Luca et al. (2010) suggests that this point is between 400 – 450 Hz.

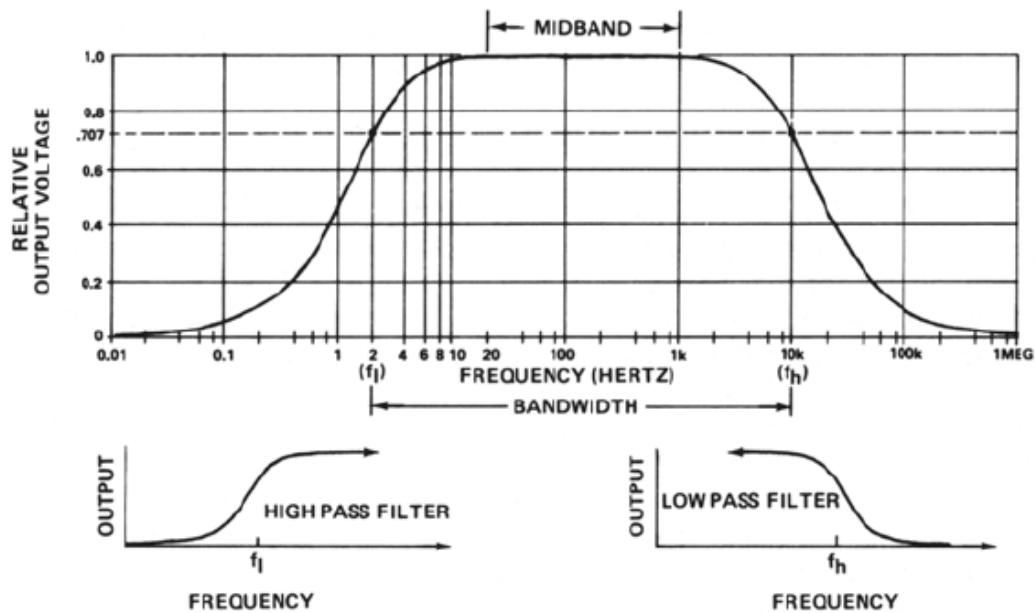


Figure 7. Bandwidth filter of surface electromyography signals composed of a high and low pass filter. Johnson, E. (1980). *Practical Electromyography*. Baltimore, MD: Williams & Wilkins. Figure 15.7, page 364.

Aside from body composition, other possible physiological factors affecting sEMG include hydration levels and skin temperature. Evetovich et al. (2002) found that sEMG patterns do not differ between states of euhydration and dehydration during isometric maximal voluntary contractions. In agreement with previous studies, they concluded that ‘impulse traffic’, muscle response, and resting membrane potential are not affected by states of dehydration compared to euhydration (Evetovich et al., 2002). Temperature also has an effect on sEMG and is controlled for by recording and managing skin temperature. Previous studies have shown an increase in the amplitude and duration of the compound muscle action potential, as well as a decrease in nerve conduction and mean power frequency, when recording sEMG at a low skin temperatures between 21.7°C and 30°C (Bigland-Ritchie, Thomas, Rice, Howarth, & Woods, 1992; Dewhurst et al., 2010; Winkel & Jergensen, 1991). To control for this factor, skin temperature is measured and recorded, and should remain constant throughout each test session and from one session to the next for each participant.

2.2 Muscular Fatigue

Muscular fatigue can be defined in two parts: central and peripheral. Central fatigue is the reduction of electrical events (neural drive) related to the formation of action potentials (Sacco, McIntyre, & Jones, 1994; St. Clair Gibson, Lamnert, & Noakes, 2001). Peripheral fatigue is the metabolic changes occurring at the muscular level which lead to a decrease in contractile processes as a result of high energy requirements (Bigland-Ritchie & Woods, 1984; Kent-Braun, 1999; Sacco et al., 1994). The potential sites of fatigue include the central nervous system, neural transmission from the central nervous system to the muscle, and the individual muscle fibres themselves (Miller et al.,

1987; Bigland-Ritchie & Woods, 1984). An all-inclusive definition from Vollestad (1997) defines fatigue as “any exercise-induced reduction in the maximal capacity to generate force or power output”.

2.2.1 Central Fatigue

The gradation of muscle force involves the recruitment of motor units and the rate at which they fire. Knight and Kamen (2001) examined the recruitment of motor units and found incomplete motor unit activation during maximal voluntary contractions. This refers to a reserve of motor units that are not fully recruited by the central nervous system during contractions. Twitch interpolation is a technique used to measure the amount of motor unit recruitment; it consists of a supramaximal electrical stimulus applied to the innervating nerve during a maximal contraction. Any increase in force demonstrated with the twitch during a maximal contraction, is due to a reserve of motor units not voluntarily activated (Shield & Zhou, 2004). During fatiguing maximal contractions, motor unit recruitment becomes maximized and the modification of firing rate becomes the main mechanism to maintain force (St. Clair Gibson et al., 2001).

2.2.2 Peripheral Fatigue

The peripheral mechanisms of fatigue are related to the decline of force through diminished high-energy phosphates and an accumulation of metabolites (Bigland-Ritchie & Woods, 1984). Over the course of an exhaustive, sustained contraction phosphocreatine (PCr) was found to decrease to 10% of resting levels within 2 minutes, simultaneous to a force decline to approximately 25%. Following the depletion of PCr, the supply of adenosine tri-phosphate (ATP) to meet energy demands was found to decrease to approximately 70% of resting levels between the third and fourth minute,

simultaneous to a force decline to 10% (Bigland-Ritchie & Woods, 1984). The decline in force due to diminished substrates is further increased due to the accumulation of metabolites, particularly in the case of isometric and static contractions where blood flow is often occluded (Bigland-Ritchie & Woods, 1984; Kent-Braun, 1999). Blood flow limitation, possibly due to a rise in intramuscular pressure (Vollestad, 1997), results in a decrease in the amount of metabolites (such as lactic acid) being removed from the muscle (Miller et al., 1987; Bigland-Ritchie & Woods, 1984; Kent-Braun, 1999; St. Clair Gibson et al., 2001). The presence of metabolites can reduce the binding affinity of calcium (Ca^{2+}) to troponin thereby decreasing excitation-contraction coupling (Bigland-Ritchie & Woods, 1984; Vollestad, 1997). Simultaneously, a decrease in Ca^{2+} released from the sarcoplasmic reticulum will also limit the binding of actin and myosin (Bigland-Ritchie & Woods, 1984; Leppik et al., 2004; Li et al., 2002; Vollestad, 1997). The formation of fewer cross-bridges between actin and myosin results in a fatigue induced decline in force (Vollestad, 1997).

2.2.3 Measuring Fatigue with Electromyography

The criterion measurements used to analyze sEMG will include root-mean-square, mean power frequency, and median power frequency. Root-mean-square (RMS) is used to characterize changes in sEMG amplitude during fatigue and can be considered a measure of voluntary drive to the muscle (Zwarts, Bleijenberg, & van Engelen, 2008). Root-mean-square amplitude is an indirect measure of motor unit recruitment, firing rate, and conduction velocity during a contraction (Bigland-Ritchie & Woods, 1984; Dimitrov, Arabadzhiev, Hogrel, & Dimitrova, 2008; Watanabe & Akima, 2010). It is important to note that RMS amplitude cannot track the recruitment or firing rate of individual motor

units but rather it reflects the active population as a whole (Vollestad, 1997). During fatiguing maximal contractions, a decrease in RMS amplitude, similar to the decline in force, may be observed as fatigue sets in (Baker et al., 1993; Dimitrov et al., 2008). This is most likely due to a decrease in firing rate, motor unit dropout, or a slowing in conduction velocity caused by an imbalance in membrane potential (Dimitrov et al., 2008; Erim et al., 1996; Watanabe & Akima, 2010).

However, during fatiguing submaximal contractions RMS amplitude remains at a stable level until the onset of fatigue, then an increase in RMS amplitude occurs (Bigland-Ritchie & Woods, 1984; Dimitrov et al., 2008; Dimitrova & Dimitrov, 2003; Watanabe & Akima, 2010). It is hypothesized that the increase in RMS amplitude during a submaximal contraction is due to an increase in the recruitment and firing rates of motor units in order to maintain the desired level of force (Bigland-Ritchie & Woods, 1984; Dimitrov et al., 2008; Dimitrova & Dimitrov, 2003; Watanabe & Akima, 2010). An increase in synchronous discharges between active motor units may also contribute to an increase in RMS amplitude (Bigland-Ritchie & Woods, 1984; Dartnall et al., 2008).

The frequency spectrum of the EMG signal is another measure of fatigue (Bigland-Ritchie & Woods, 1984; Broman, Bilotto, & De Luca, 1985a; Cornwall, Krock, & Wagner, 1994; Merletti, Knaflitz, & De Luca, 1990; Watanabe & Akima, 2010). The frequency spectrum consists of each frequency and its relative contribution to the signal. The general shape of the frequency spectrum of the sEMG interference pattern is indicative of the shape of the frequency spectrum of individual motor unit action potentials contributing to the signal. The shape of the frequency spectrum of an individual motor unit action potential is dependent on both its location (i.e. tissue filtering

effects) and conduction velocity. Changes in the frequency spectrum are measured using mean frequency (MPF) or median frequency (MDF) (Merletti et al., 1990).

During fatigue it has been shown that MPF decreases (Bigland-Ritchie & Woods, 1984; Broman et al., 1985a; Cornwall et al., 1994; Enoka & Stuart, 1992; Kupa, Roy, Kandarian & De Luca, 1995; Luttmann et al., 1996; Merletti et al., 1990; Vollestad, 1997; Watanabe & Akima, 2010; Zwarts et al., 2008) meaning that there is a greater contribution of lower frequencies to the EMG signal. Erim et al. (1996) examined the recruitment and firing rates of motor units based on their recruitment thresholds. It was found that high threshold motor units maintain lower firing rates than their low threshold counterparts in order to minimize fatigue. However, during a maximal contraction high threshold motor units will increase their firing rates to achieve the desired force even at the cost of fatiguing. Erim and colleagues (1996) concluded that high threshold motor units would fatigue earlier in a maximal contraction than low threshold motor units thereby diminishing their contribution to force and decreasing the contribution of high frequencies to the frequency spectrum.

The second explanation for the decrease in MPF during fatigue is the slowing of muscle fibre conduction velocity (Bigland-Ritchie & Woods, 1984; Broman et al., 1985a; Enoka & Stuart, 1992; Kupa et al., 1995; Merletti et al., 1990; Vollestad, 1997; Watanabe & Akima, 2010; Zwarts et al., 2008). Some of the factors contributing to the slowing of the muscle fibre conduction velocity include: increased extracellular K^+ , metabolite accumulation, decreased firing rate of motor units, decreased blood flow, decreased pH, and decreased phosphocreatine (PCr) and ATP (Broman et al., 1985a; Enoka & Stuart, 1992; Kupa et al., 1995; Merletti et al., 1990; Watanabe & Akima, 2010; Zwarts et al.,

2008). While there are slight differences in MPF changes across fatigue protocols, it is widely accepted that a decrease in MPF is present during fatigue and can therefore be used as an indicator for fatigue (Bigland-Ritchie & Woods, 1984; Broman et al., 1985a; Cornwall et al., 1994; Enoka & Stuart, 1992; Kupa et al., 1995; Merletti et al., 1990; Vollestad, 1997; Watanabe & Akima, 2010; Zwarts et al., 2008). It is not known, however, if the cause of this decrease is due to low threshold motor unit activation and high threshold motor unit drop out, the slowing of conduction velocity, or a combination of these two factors.

A contributing factor to decreased conduction velocity is the occlusion of blood flow during activity. Muscular contractions create intramuscular pressure causing blood flow occlusion, which leads to the accumulation of metabolites and decreased oxygen delivery. Blood flow occlusion always includes decreased oxygenation coupled with decreased metabolite removal. However, decreased oxygenation can also occur independently of blood flow occlusion, such as during hypoxemic conditions. Russ and Kent-Braun (2003) examined intermittent maximal contractions with and without blood flow occlusion in males and females. Without occlusion, force decrements ranged from 22 to 35% after twenty-four contractions, compared to a force decrement of approximately 80% under ischemic conditions. Russ and Kent-Braun (2003) speculated that the increase amount of fatigue was more due to a lack of oxygenation as opposed to decreased blood flow in the active muscles. Tachi and colleagues (2004) corroborated the previous results with intermittent submaximal dorsiflexion contractions to exhaustion. Blood volume and tissue oxygenation were decreased when contractions were performed with the leg above the level of the heart compared to below. This resulted in decreased

endurance and a steeper decline in mean power frequency. Lastly, both conditions were repeated with blood flow occlusion via tourniquet. This additional variable negated any difference found between the leg-up or –down conditions confirming that the increased fatigue was purely due to the decrease in blood flow and tissue oxygenation (Tachi et al., 2004).

The amount of decrease in MPF during fatiguing contractions depends on the type of contraction and force level. There is a greater decrease in MPF for static, compared to dynamic, contractions (Vollestad, 1997) and for contractions performed at a higher force level (Cornwall et al., 1994). Fibre type also affects the rate of decrease in MPF. Slow twitch muscle fibres have a lower initial MPF and exhibit a slower decrease in MPF during fatigue, compared with fast twitch fibres (Kupa et al., 1995). On average, MPF will decrease anywhere from 10-50% during a fatiguing contraction depending on the aforementioned factors (Cornwall et al., 1994; Kupa et al., 1995; Merletti et al., 1990).

Peripheral fatigue, from the peripheral nerve to the muscle, may be assessed by using electrically evoked potentials. An M-wave, or compound muscle action potential, is the massed action potential elicited by electrical stimulation of the peripheral nerve associated with the muscle of interest (Bigland-Ritchie & Woods, 1984). The M-wave displays “the effectiveness of electrical propagation across the neuromuscular junction and along the muscle surface membrane” (Bigland-Ritchie & Woods, 1984). Factors affecting the M-wave are displayed in Figure 8. The M-wave indirectly represents the excitability of the muscle membrane which decreases during fatigue due to an imbalance of sodium (Na^+) and potassium (K^+) across the membrane (Dimitrova & Dimitrov, 2002; Dimitrova & Dimitrov, 2003; Miller et al., 1987). The build-up of metabolites (such as

lactic acid) during activity also contributes to the changes in the M-wave signal (Vollestad, 1997; Zwarts et al., 2008).

The change of the M-wave with fatigue is inconsistent across studies and across methods of fatigue. A decrease in the amplitude of the M-wave is often seen if there is impairment in the membrane excitation or action potential propagation which often occurs if blood flow is occluded (Bigland-Ritchie & Woods, 1984; Dimitrov et al. 2008; Dimitrova & Dimitrov, 2002; Miller et al., 1987; Enoka & Stuart, 1992; Russ & Kent-Braun, 2003). Alternatively, it is possible for an M-wave to increase in amplitude due to decreased dispersion between action potentials, caused by presynaptic or end-plate facilitation, which results in greater temporal summation (Bigland-Ritchie & Woods, 1984; Dimitrova & Dimitrov, 2002; Feireisen, Duchateau & Hainaut, 1997). This decrease will also affect the duration and total area of the M-wave signal. The duration of the M-wave is often found to increase during fatigue due a decrease in muscle fibre conduction velocity caused mainly by metabolite accumulation and decreased Ca^{2+} release (Bigland-Ritchie, Johansson, Lippold, & Woods, 1983; Bigland-Ritchie & Woods, 1984; Kent-Braun, 1999).

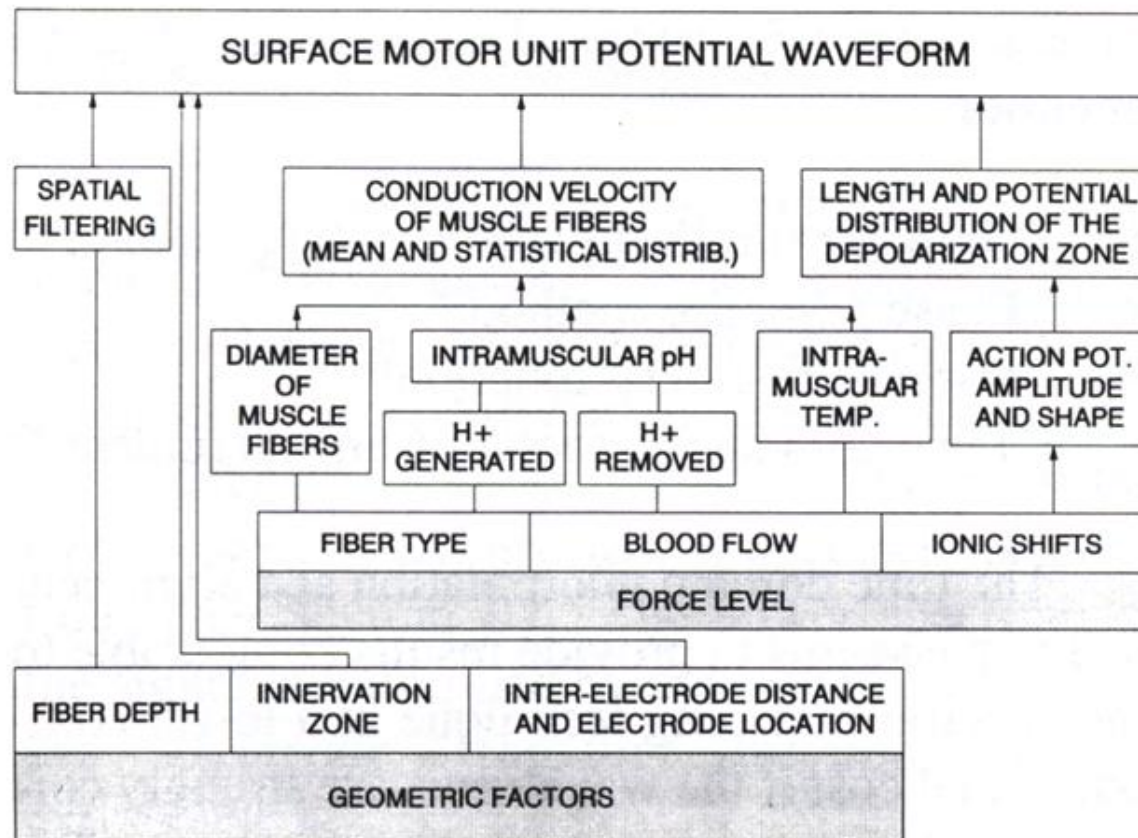


Figure 8. Factors contributing to a single motor unit potential waveform and the frequency spectrum of an individual motor unit action potential. Merletti, R., Knaflitz, M., & De Luca, C. (1992). Electrically evoked Myoelectric signals. *Critical Reviews in Biomedical Engineering*, 19(4), 293-340. Figure 11, page 314.

2.2.4 Voluntary Activation (*Twitch Interpolation*)

When inducing fatigue through voluntary contractions, physiological factors (central and peripheral fatigue) are the main cause of a decrement in force; however, psychology can play an integral role in the motivation of the participant to elicit maximal contractions to the point of fatigue or pain. One methodological control for central fatigue and motivation is the use of the twitch interpolation technique. This consists of a superimposed stimulus applied during a maximal contraction, as piloted by Merton (1954).

Central fatigue has been defined as a decrease in the voluntary activation of a muscle that is not accompanied by an equal decrease in the evocable force of that muscle (Vollestad, 1997; Zwarts et al., 2008). Twitch interpolation is therefore used for the purpose of identifying ‘incomplete motor unit activation’ (Gabriel et al., 2006; Merton, 1954). Since the goal of the superimposed twitch is to elicit maximal activation, a supramaximal stimulus is used to ensure all motor units are active (Shield & Zhou, 2004). A supramaximal stimulus can be considered the stimulus at which an increase in the CMAP (M-wave) is no longer exhibited when the stimulus voltage is increased (Kent-Braun, 1999; Kent-Braun & LeBlanc, 1996). The technique originally employed by Merton (1954) was to double the voltage required for maximal stimulus under normal conditions.

Other techniques used in previous literature include: a maximal stimulus evoked between 100-150V (Behm, Power, & Drinkwater, 2001), an increase of 25 volts (approximately 10-15%) above maximal stimulus (Kent-Braun, 1999; Kent-Braun & LeBlanc, 1996), and an increase of 15-20% above maximal stimulus (McKenzie,

Bigland-Ritchie, Gorman, & Gandevia, 1992), to identify a few. These supramaximal stimulus levels are usually used for a single or double twitch. A separate technique involves the use of a train of stimuli (also known as tetanic stimuli) usually applied at 50-100 Hz over a period of 100-500 ms or in a set train of 5-8 pulses (Behm et al., 2001; Bilodeau, 2006; Kent-Braun, 1999; Schillings et al., 2005; Shield & Zhou, 2004; Suter & Herzog, 2001; Taylor et al., 2000). Although Shield and Zhou (2004) reported that no differences exist in maximal force production between the use of 1, 2 or 5 stimuli, other studies have reported in favour of tetanic stimuli for maximal stimulation (Behm et al., 2001; Kent-Braun, 1999; Kent-Braun & LeBlanc, 1996).

The location of the stimulus application itself can prove problematic. The twitch can be applied at either the innervating nerve or directly to the motor point of the muscle. Stimulation of the motor point is less common (McKenzie et al., 1992; Schillings et al., 2005), and while it can specifically activate only the desired muscle it may not activate all of the motor units within that muscle. (Bilodeau, 2006; Shield & Zhou, 2004). Alternatively, stimulation of the innervating nerve is less painful at supramaximal levels than motor point stimulation, though not without a certain level of discomfort (Behm et al., 2001; Shield & Zhou, 2004). The tibialis anterior, specifically, is most often evoked through stimulation of the deep peroneal nerve accessible 1 cm distal to the fibular head (Connelly et al., 1999; Kent-Braun, 1999; Kent-Braun & LeBlanc, 1996) or in the medial popliteal fossa (Belanger & McComas, 1981; Suter & Herzog, 2001) (see Figure 9). The main limitation of nerve stimulation is the co-activation of antagonist muscles innervated by the same nerve. With tibialis anterior stimulation, the plantar flexor and peroneal muscles are also innervated by the deep peroneal nerve (Kent-Braun & LeBlanc, 1996;

Shield & Zhou, 2004). This can mean that supramaximal stimulation will also activate motor units of these antagonist muscles. As a methodological control for co-activation, Connelly et al. (1999) stimulated at 'maximal twitch torque', defined as the voltage level that activated the maximum amount of tibialis anterior activity before interference from antagonistic muscle was present ("a decrease in twitch torque with continued increase in voltage").

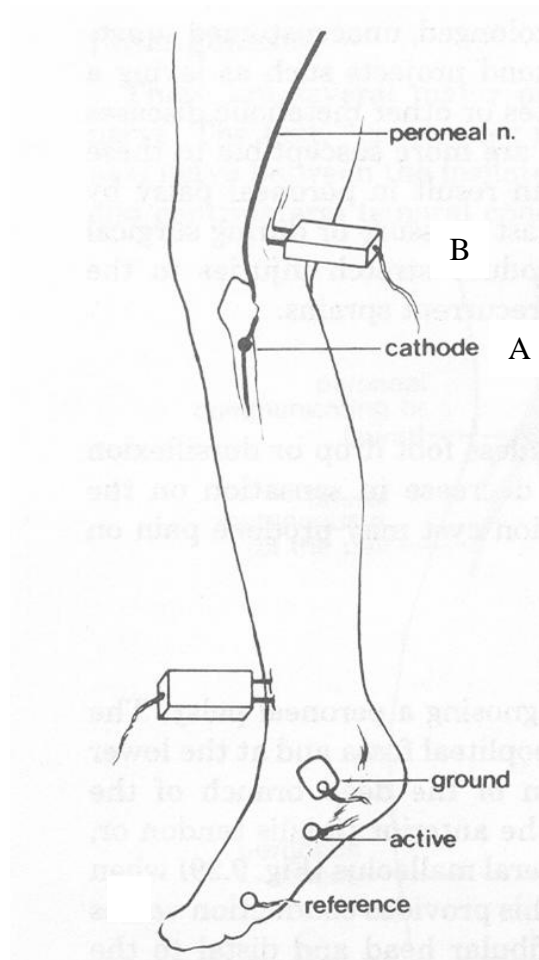


Figure 9. Stimulation locations of the peroneal nerve: 1cm distal and posterior to the fibular head (A) and medial popliteal fossa (B). Johnson, E.W. (Ed.). (1980). *Practical Electromyography*. Baltimore, MD: Williams & Wilkins. Figure 9.29, page 252.

Another requirement, and possible limitation, of twitch interpolation is the need for participants to be well motivated and practiced at the exercise to produce a ‘true’ maximal voluntary contraction (Baratta, Solomonow, Zhou, & Zhu, 1998; Enoka & Stuart, 1992). Even during an ideal contraction it is possible that minute increments in force or muscle activity will go undetected, a problem that is compounded by the variability of contractions repeated within a short time frame (Enoka & Stuart, 1992; Shield & Zhou, 2004). A final consideration may be the fact that some muscle groups may not have the ability to reach full activation during a voluntary contraction compared to other muscle groups (Enoka & Stuart, 1992). Belanger and McComas (1981) examined voluntary activation through twitch interpolation in the tibialis anterior and triceps surae. They observed lower levels of voluntary activation in the plantar flexors compared to the dorsiflexors for 14 of the 28 participants, concluding that incomplete activation was isolated to the specific muscle group. Although the technique can be difficult, twitch interpolation should fully activate all the motor units of a muscle and produce an increase in force if incomplete motor unit activation is present (Suter & Herzog, 2001).

2.2.5 Fatigue Patterns

Contraction Intensity

The patterns of force decrements produced during fatiguing contractions vary greatly depending on a number of factors, of which contraction intensity plays a major role. Fatiguing protocols consisting of maximal contractions have been shown to elicit greater fatigue in a smaller amount of time compared to submaximal contractions (Cornwall et al., 1994; Komi & Tesch, 1979). While central and peripheral fatigue are both seen in almost all contractions, it has been shown that fatigue from maximal

contractions is mainly peripheral (Bigland-Ritchie, Furbush, & Woods, 1986; Bigland-Ritchie & Woods, 1984; Taylor et al., 2000).

Increased substrate depletion, metabolite accumulation, and contractile failure have all been seen with maximal fatigue protocols (Bigland-Ritchie et al., 1986; Kent-Braun, 1999; St. Clair Gibson et al., 2001). These differences in maximal compared to submaximal contractions are evident in the sEMG signal. Root-mean-square amplitude has been shown to decrease during maximal contractions and increase during submaximal contractions. This is caused by an immediate activation of all available motor units in a maximal contraction that decline with fatigue, as opposed to the ability to increase the firing rate and the number of active motor units during a submaximal contraction to offset the decline in force (Dimitrov et al. 2008; Dimitrova & Dimitrov, 2003; Watanabe & Akima, 2010; Enoka & Stuart, 1992).

During submaximal contractions at different force levels, it has been found that higher intensity submaximal contractions will elicit steeper increases in RMS amplitude than low intensity contractions (Cornwall et al., 1994). Another measure of differences between maximal and submaximal contractions is through the frequency spectrum as measured by the MPF. Mean frequency is known to decrease during fatigue, which suggests a shift in the firing rates to lower frequencies due to a decrease in high threshold motor unit firing rates caused by a faster rate of fatigue in high threshold motor units (Erim et al., 1996). The decrease in MPF is greater and more rapid in maximal contractions and high intensity submaximal contractions compared to low intensity contractions (Cornwall et al., 1994; Enoka & Stuart, 1992).

Contraction Type

A second factor that determines the pattern of fatigue is the use of sustained versus serial contractions. Sustained (long duration) and intermittent (repeated) are the two main contraction types used. During sustained contractions a level must be maintained at a target force for either a set amount of time, or until that level can no longer be held. Sustained contractions have been linked to greater excitation-contraction coupling impairment due to metabolite accumulation from decreased blood flow (Watanabe & Akima, 2010). The occlusion of blood flow limits the removal of harmful metabolites causing a faster decline in force, and a more rapid rate of fatigue (Baker et al., 1993; Clark, 1978). Alternatively, intermittent contractions consist of repeated contractions being performed at a target force level for a specific duration with specific intertrial rest periods. Intermittent contractions have been shown to better remove metabolic substrates during contractions, which is mainly due to less blood flow occlusion compared to sustained contractions (Enoka & Stuart, 1992; Taylor et al., 2000). Baker and colleagues (1993) compared short duration sustained activity to long duration intermittent contractions and speculated that the quick recovery following sustained activity was due to the recovery of metabolic mechanisms; whereas the prolonged fatigue of intermittent activity was due to the slow recovery of impaired activation.

Chasiotis, Bergstrom, and Hultman (1987) examined energy utilization during intermittent and sustained contractions evoked by electrical stimulation. Force decrements produced from intermittent contractions were much greater than continuous contractions (50 versus 90%) and required a larger amount of total work to be performed. Both ATP and PCr were utilized in greater amounts, and at a faster rate during

intermittent contractions, which was mirrored in a higher glycolytic rate. Lactate and H^+ accumulation was also significantly higher during intermittent compared to continuous contractions. Finally, Chasiotis and colleagues (1987) concluded that not only is there a greater energy cost when performing intermittent contractions, but the cost appears to increase with contraction time. The authors speculated that this was due to a less economical state of cross-bridge cycling due to Ca^{2+} pumping being required to restore the intracellular gradient between every contraction as opposed to holding a steady state. The re-uptake of Ca^{2+} following a contraction is performed by the sarco(endo)plasmic reticulum Ca^{2+} -ATPase (SERCA). Impairment and slowing of SERCA pumps are found during repetitive activity and lead to fatigue through increased resting intracellular Ca^{2+} , reduced stores of Ca^{2+} in the Sarcoplasmic Reticulum, and limited Ca^{2+} re-uptake leading to slowed muscle relaxation (Tupling, 2004).

A factor within intermittent contraction protocols is the work to rest ratio (duty cycle) of the contractions performed. When the duty cycle increases (contractions are longer) or the cycle duration decreases (rest is shorter) there will be an increase in the amount of fatigue and the rate at which fatigue develops. An increased duty cycle will rely more on energy expenditure since there is a greater amount of work that is being performed (Clarke, 1978; Enoka & Stuart, 1992). A decrease in the cycle duration will cause an increase in fatigue due to a greater accumulation of metabolites caused by a decrease in blood flow (Clarke, 1978; Enoka & Stuart, 1992). In a study by Kroll (1967a), thirty 5-second maximal voluntary contractions were performed with rests of 5, 10 or 20 second durations. It was found that fatigue ranged from 30-40% force decrements during the 1:1 work rest ratio, compared to 20-30% force decrements during

the 1:2 and 1:4 work rest ratios. Taylor et al. (2000) found similar results when comparing duty cycles (work:rest) ranging from 50 to 86%. The 50% duty cycle (1:1 work rest ratio) produced a force decrement of 40% compared to the higher duty cycles, which produced 60% force decrements.

Muscle Composition

Independent of the type or level of contraction is the muscle fibre type composition of the muscle and its effect on the fatigue curve. Komi and Tesch (1979) compared primarily fast twitch versus slow twitch muscles, which were classified by greater than or less than 50% fast twitch fibres, respectively. It was found that fast twitch muscles had a greater initial force level, a more rapid decline in force, and lower force values at the final contraction compared to slow twitch muscles (Komi & Tesch, 1979). Furthermore, fast twitch muscles also had a greater, more rapid decrease in RMS amplitude and MPF compared to slow twitch during maximal contractions (Komi & Tesch, 1979). Rainoldi and colleagues (2008) observed a greater slowing of conduction velocity in primarily fast twitch muscles compared to slow twitch muscles during a high intensity fatiguing protocol. In agreement with this, Bigland-Ritchie et al. (1986) found that slow twitch muscles demonstrated less contractile failure when compared to fast twitch muscles during a sustained submaximal contraction.

Strength

The strength of the participant can also affect the fatigue curve. Trend analysis can be used to identify linear, quadratic, cubic and quartic components of a fatigue curve. Kroll (1967a) compared high, middle and low strength male participants, which were categorized into three equal groups depending on their initial force levels. It was found

that high and middle strength participants had similar fatigue curves with strength decrements ranging from 25-40% compared to low strength participants with force decrements ranging from 0-30%. Similar force decrements were found when the study was reproduced using female participants (Kroll, 1971a).

Kroll (1967a, 1971a) examined the fatigue curves of both males and females and found that high strength participants had more complex fatigue curves compared to their low strength counterparts, even though males were twice as strong as females in the high strength group. In agreement, Patton, Hinson, Arnold, and Lessard, (1978) examined fatigue curves of high and low strength males and females performing isokinetic elbow flexion. When completing maximal contractions to exhaustion, the high strength participants produced fatigue curves with linear and quadratic components and a higher amount of fatigue compared to the low strength participants whose curves exhibited a mild linear decrease. Similar to the study by Kroll (1967a; 1967b), Patton et al. (1978) observed a rapid negative decline in force in high strength male and female participants within the initial few contractions.

Kroll et al. (1980) compared power- and endurance-trained males and found that the power-trained group had more complex fatigue curves and a greater decrease in initial strength compared to endurance-trained participants. Similarly, Kanehisa, Ikegawa and Fukunaga (1996) compared weight-trained individuals to endurance runners and untrained individuals performing isokinetic knee extensions. Weight trained individuals had a significantly higher initial force than endurance runners and untrained individuals and a more rapid decline in force which is in agreement with Kroll et al. (1980). Behm and St. Pierre (1998) found conflicting results during submaximal plantar flexion

contractions in untrained and trained males. The overall strength decrement was found to be greater in the untrained group compared to the trained group; however, they performed significantly fewer contractions before exhaustion, which may account for this discrepancy.

2.2.6 Participant Motivation

It cannot be ignored that force has a psychological component especially during fatiguing voluntary contractions. Kroll (1967b) compared the fatigue and recovery following isometric contractions performed by participants of three different strength levels. Kroll (1967b) stated that the role of motivational factors could not be dismissed from the explanation of recovery rates above initial values present in the low strength group. A possible methodological control for motivation is to keep the requirements of the fatigue protocol unknown to the participants. This is done so that participants do not attempt to use or reserve strength based on the amount of time or contractions they have to complete. However, a study by Maisetti, Guevel, Legros, and Hogrel (2002) performed spectral analysis of the sEMG signal generated during fatiguing contractions with a known, pre-set contraction time compared to contractions to exhaustion with no time limit and found that knowledge of exercise duration had no influence on fatigue.

2.2.7 Fatigue Summary

The presence of fatigue in daily life is persistent as it can occur with repeated submaximal efforts at merely 10-15% of a muscle's maximal strength (Stafford & Petrofsky, 1981). The amount of fatigue and the speed with which it occurs is extremely variable and depends on a number of factors, with the type of exercise performed being the most influential. The sites of fatigue include the central nervous system, the structures

responsible for transmission from the central nervous system to the muscle, and the individual muscle fibres (Bigland-Ritchie & Woods, 1984; Miller et al., 1987). For this reason fatigue is due to both central and peripheral mechanisms.

The measurement of fatigue through sEMG involves the amplitude and frequency content of the signal. Root-mean-square amplitude is found to increase during submaximal contractions due to increasing motor unit recruitment and firing rate, to offset force decrements (Bigland-Ritchie & Woods, 1984; Dimitrov et al. 2008; Dimitrova & Dimitrov, 2003; Watanabe & Akima, 2010). This same measure decreases during maximal contractions due to fatigue of fully recruited motor units firing at maximal rates (Dimitrov et al. 2008; Dimitrova & Dimitrov, 2003; Watanabe & Akima, 2010). The frequency spectrum, as measured by MDF and MPF, is found to shift to lower frequencies consistently during fatigue (Bigland-Ritchie & Woods, 1984; Broman et al., 1985a; Cornwall et al., 1994; Enoka & Stuart, 1992; Kupa et al., 1995; Watanabe & Akima, 2010; Zwarts et al., 2008).

The alteration of the M-wave, as a measure of muscle fibre conduction velocity (Bigland-Ritchie & Woods, 1984) and membrane excitability (Bigland-Ritchie & Woods, 1984; Dimitrova & Dimitrov, 2002; Miller et al., 1987), is ambiguous in the literature. It has been reported to increase in amplitude due to reduced dispersion of action potentials (Bigland-Ritchie & Woods, 1984; Dimitrova & Dimitrov, 2002), or decrease in amplitude due to decreased membrane potential and increased metabolite accumulation (Bigland-Ritchie & Woods, 1984; Miller et al., 1987; Dimitrov et al. 2008; Dimitrova & Dimitrov, 2002). Regardless of the type of fatigue protocol, the occlusion of blood flow has been found to increase metabolite accumulation, slow muscle fibre conduction

velocity, and limit the presence of high-energy phosphates all causing fatigue (Bigland-Ritchie & Woods, 1984; Kent-Braun, 1999; Miller et al., 1987; St. Clair Gibson, 2001). Finally, the main indicator of fatigue, force, is found to decline immediately at the onset of fatigue in variable amounts depending on the exercise being performed (Vollestad, 1997).

2.3 Recovery

Recovery from fatigue begins as soon as the fatiguing effort has ceased, and can continue for hours after exercise. Recovery is generally assessed as a reference to pre-fatigue (initial) levels of performance (force) or physiological (EMG) variables. Recovery consists of the peripheral and central mechanisms diminished during fatigue, returning to their pre-fatigue states. Merton (1954) attempted to classify fatigue based on the rate of recovery stating that immediate recovery would indicate central fatigue whereas recovery requiring the return of blood flow would indicate peripheral fatigue. It is now well known that a combination of peripheral and central mechanisms, which cause fatigue, will recover depending on the type of contraction performed, the level of force, and the duration of fatigue.

2.3.1 Force Recovery

Fatigue and recovery are determined by both central and peripheral mechanisms which will alter a muscle's ability to produce force. A decline in force is the main indicator of fatigue and will occur if fatigue is present regardless of the type or level of exercise performed. It is only fitting then that a recovery of force to pre-fatigue levels be used as the main marker for recovery. The amount of decline in force from fatigue will depend greatly on the fatigue protocol used. Although it is difficult to compare protocols,

sustained contractions will generally cause a greater force deficit than intermittent contractions; and maximal contractions will generally diminish force more than submaximal contractions (Baker et al., 1993).

Baker et al. (1993) compared short (2 minute sustained maximal voluntary contractions) and long duration exercise (15-20 minute intermittent maximal voluntary contractions), and found that force declined to 35 and 42% of initial values, respectively. Although the sustained group saw a greater force decrement they recovered significantly faster in both absolute and relative force recovery (100% recovery in 10 minutes compared to 80% in 15 minutes, for short and long duration exercise respectively) (Baker et al., 1993). An increased speed of recovery from sustained exercise, as compared to intermittent, appears to be consistent across the literature (Baker et al., 1993; Clarke & Stull, 1969; Miller et al., 1987; Stull & Clarke, 1971). However, sustained exercise produces a greater force decrement during fatigue compared to intermittent exercise and may therefore appear to have a faster rate of recovery when force is presented in absolute terms. It is important to normalize force fatigue relative to initial (pre-fatigue) levels and to normalize recovery relative to the force decrement when comparing rates of recovery (Clarke & Stull, 1969).

2.3.2 Possible Phases of Recovery

It has been speculated that recovery can be split up into three distinct phases based on the speed at which certain mechanisms recover and when that recovery begins. Miller et al. (1987) concluded that the three phases of recovery included an early phase consisting of the normalization of membrane function within 5 minutes (as displayed in M-wave recovery), an intermediate phase associated with high-energy phosphate and pH

recovery (in a similar time course as force recovery), and a prolonged phase consisting of the delayed recovery of excitation-contraction coupling (as determined by neuromuscular efficiency). The concept of prolonged recovery due to excitation-contraction coupling impairment has been speculated across fatigue protocols and has been reported to last for hours after the cessation of fatiguing effort (Baker et al., 1993; Edwards, Hill, Jones, & Merton, 1977; Girard et al., 2008; Miller et al., 1987).

2.3.3 Measuring Recovery with Electromyography

The change in root-mean-square sEMG amplitude associated with fatigue is highly dependent on the type of fatiguing contraction performed. Typically, RMS amplitude is found to decrease during maximal contractions and increase during submaximal contractions to fatigue (Dimitrov et al. 2008; Dimitrova & Dimitrov, 2003; Watanabe & Akima, 2010). Findings for the recovery of RMS amplitude are more consistent, with the majority of the literature reporting close to full recovery occurring within 5 minutes after fatigue by submaximal, maximal, sustained and intermittent contractions (Baker et al., 1993; Cornwall et al., 1994; Petrofsky, 1981). The percentage of RMS amplitude recovered within one minute has been reported to be between 50 and 80% (Baker et al., 1993).

The main measurements of peripheral fatigue include contractile properties as indicated by twitch force, M-wave amplitude and duration, and MPF or MDF. These measures are related to muscle fibre conduction velocity, which is affected by metabolites, pH balance, calcium, and blood flow (Baker et al., 1993; Cornwall et al., 1994; Petrofsky, 1981; Tesch & Wright, 1983; Yates et al., 1987). As previously mentioned, a decrease in MPF and MDF is found during fatiguing exercises, regardless

of the type or force of contraction. Similarly, the recovery of the frequency spectrum is rapid regardless of the fatigue protocol. Mean power frequency and MDF have been found to fully recover within 10 minutes, with the majority of recovery occurring within 2-5 minutes after fatigue (Cornwall et al., 1994; Kuorinka, 1988; Mills, 1982; Petrofsky, 1981).

A consistent pattern of rapid recovery is also evident in the M-wave (Miller et al., 1987). Although, alterations in the amplitude and duration of the M-wave vary greatly depending on the type of contraction, the rate of recovery seems to be consistent across methodologies. Previous studies, including sustained submaximal (Miller et al., 1987), sustained maximal (Baker et al., 1993), and intermittent maximal (Baker et al., 1993; Mills, 1982) fatigue protocols, reported full recovery of the M-wave to pre-fatigue values within 10 minutes, with most recovery occurring within 5 minutes.

Many determinants of peripheral fatigue have been found to recover early and rapidly after the cessation of the fatiguing effort, contributing to the timely recovery of MPF and M-wave amplitude and duration. The restoration of blood flow appears to be a primary determinant of recovery from peripheral fatigue. After both sustained and intermittent contractions, there is a recovery of blood flow almost immediately (Baker et al., 1993; Cornwall et al., 1994; Yates, Kearney, Noland, & Felts, 1987). The removal of intramuscular metabolites (such as lactic acid and inorganic phosphate) has been found to occur within 2-10 minutes of recovery from varying fatigue protocols, mainly due to blood flow restoration (Baker et al., 1993; Cornwall et al., 1994; Tesch & Wright, 1983; Yates et al., 1987). Petrofsky (1981) found immediate recovery of muscle fibre conduction velocity within seconds after fatigue, speculated to be caused by the removal

of potassium that had accumulated in the interstitial fluid during fatigue. The simultaneous recovery of membrane pH has been reported to occur within 4 to 15 minutes (Baker et al., 1993; Cornwall et al., 1994; Miller et al., 1987). An increase in post-fatigue blood flow can also restore decreased levels of high-energy phosphates (Cornwall et al., 1994; Miller et al., 1987; Yates et al., 1987).

Sjogaard and colleagues (1988) examined the role of blood flow by comparing intermittent and sustained handgrip contractions performed from 5 to 50% of a maximal voluntary contraction. During the sustained contractions there was an initial increase in blood flow followed by a decline as the contraction persisted. Immediately after the contraction ceased there was a rapid increase in blood flow (approximately 4 times pre-contraction levels). Alternatively, during intermittent contractions blood flow appeared to mimic values obtained during the sustained contractions but then peaked during intertrial rests. These results may explain why recovery from fatigue is rapid after sustained versus intermittent contractions, which utilize blood flow to better maintain homeostasis during fatigue. This conclusion was further investigated using a cuff to occlude blood flow during activity. Pitcher and Miles (1997) had participants perform isometric intermittent contractions at 80% maximal voluntary contraction. The decline in force reached a plateau between 40 and 50% maximal after 7 minutes and remained there until completion at 15 minutes. Alternately, the same protocol was repeated with a cuff occluding blood flow and a force decrement to 18% was seen at exhaustion after 3 minutes. These findings demonstrate the role of blood flow in maintaining homeostasis to limit fatigue during an intermittent protocol.

Recovery from fatigue can last hours after exercise due to deficits in excitation-contraction coupling persisting long after the recovery of metabolites, high-energy phosphates and conduction velocity. Edwards and colleagues (1977) highlighted the role of excitation-contraction coupling on the duration of recovery when they observed that long lasting fatigue was still evident after the recovery of high-energy phosphates and electrical activity. Excitation-contraction coupling failure was further described for sustained submaximal contractions (Miller et al., 1987) and for a comparison of sustained and intermittent maximal contractions (Baker et al., 1993). Neuromuscular efficiency (force produced per unit of integrated EMG at 50% maximal voluntary contraction) was measured pre-, during, and post-fatigue from a 4 minute sustained 50% maximal voluntary contraction (Miller et al., 1987). At 15 minutes post-fatigue, when most physiological mechanisms had recovered to initial values, neuromuscular efficiency was only at 64% and only neared full recovery after 60 minutes (Miller et al., 1987). To investigate the role of excitation-contraction coupling on neuromuscular efficiency, post-tetanic potentiation of a twitch was studied and found to be only 37% of the pre-fatigue value after 15 minutes (Miller et al., 1987). Although Miller et al. (1987) speculated that excitation-contraction coupling impairment was a function of submaximal contractions due to decreased calcium release and low frequencies, similar results were also found with maximal contractions (Baker et al., 1993). In a comparison of sustained and intermittent maximal contractions, Baker et al. (1993) found that prolonged recovery from long duration intermittent contractions could be explained by persistent excitation-contraction coupling failure. Both studies (Baker et al., 1993; Miller et al., 1987) reported

that the site of lasting fatigue, and therefore delayed recovery, lies distal to the muscle membrane at the site of excitation-contraction coupling.

2.3.4 Recovery Patterns

As with fatigue, the pattern of recovery depends on the methodology used to elicit fatigue, and the characteristics of the participants. Baker et al. (1993) compared fatigue protocols of intermittent and sustained contractions. It was found that, although the intermittent protocol was 5-7 times more demanding, there was a greater decrease in force with the sustained contraction followed by a quicker return to pre-fatigue force levels. When comparing intermittent contractions at submaximal levels, Petrofsky (1981) found that high intensity fatigue (70% compared to 25 and 40% maximal voluntary contraction) experienced the greatest force decrement but also the quickest recovery. The literature appears to support a general pattern of fast recovery from greater levels of force deficits (Clarke & Stull, 1969; Cornwall et al., 1994; Petrofsky, 1981).

Similar to fatigue, recovery curves can show linear, quadratic, cubic, and quartic patterns when subjected to trend analysis. Kroll (1967b; 1971b) examined recovery curves in males and females after thirty 5-second isometric maximal wrist flexion contractions. Both intertrial rest periods and participant strength levels were accounted for in these two studies. In males, the 5-second intertrial rest condition displayed linear and quadratic components as opposed to only linear components for the 10- and 20-second rest conditions. Contradictory results were found for females, who displayed quadratic, cubic and even quartic trends across the 10- and 20-second rest conditions in the right and left hand. However, both studies demonstrated that the greater the duty cycle (work:rest ratio) the more complex the recovery curve (Kroll, 1967b; 1971b).

When examining recovery pattern on the basis of participants' strength level, a crucial consideration is brought to light. Kroll (1967b) found no differences in recovery curve patterns between different strength levels in males. In females, despite having similar fatigue patterns to males, there were significant differences in recovery patterns between groups of different strength levels. Kroll (1971b) found that the greater the initial strength level the more complex the recovery curve, which is in agreement with the fatigue curves produced by both males and females. The crucial methodological difference between males (1967b) and females (1971b) was the inclusion of the last fatigue trial in the recovery analysis for females, which produced the significant trend results. Figure 10 displays the force levels from one group of female participants during fatigue and recovery. The final trial of fatigue (T_{30}) is included in the recovery analysis. It is evident that a distinct portion of the force was recovered in the first minute following fatigue (from T_{30} to R_1). If the inclusion of the last fatigue trial had not been included, this portion of force recovered would have been over-looked.

2.3.5 Over-Recovery

Over-recovery was first noted by Kroll (1967b) as a return of force to levels higher than the initial maximum. It has since been reproduced by Kroll (1971b) in the wrist flexors and also found by Stull and Clarke (1971) in a hand-gripping exercise. The exercise protocol employed by Stull and Clarke (1971) included maximal rhythmic contractions performed at a pace of 30 per minute for 3 minutes. Force decreased by 42% of initial strength and by 235 seconds force had recovered to 107% of the initial maximal voluntary contraction. It is unclear if this was the full extent of force recovery since recovery data was only collected up to 235 seconds post-fatigue.

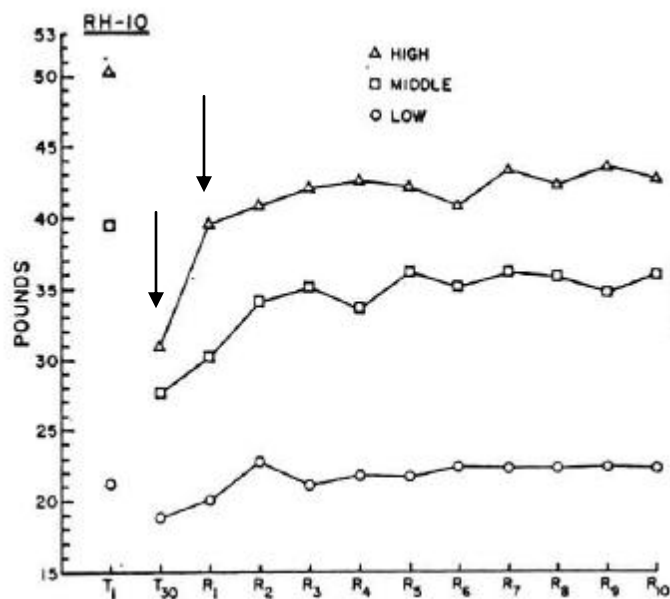


Figure 10. Force values for one group of participants (Right Hand, 10-second intertrial rest condition, females) during fatigue and recovery. The arrows indicate the final fatigue trial (T₃₀) and the initial recovery trial (R₁). The inclusion of the final fatigue trial in the force recovery analysis means that force recovered between these two times (between the two arrows) will be taken into account. Kroll, W. (1971b). Recovery patterns after local muscular fatigue for different levels of isometric strength in college age females.

American Corrective Therapy Journal, 25(5), 132-138. Figure 1, page 136.

Kroll (1967b; 1971b) found over-recovery in males and females after thirty 5-second maximal isometric contractions followed by 10 minutes of recovery. An additional factor examined by Kroll (1967b; 1971b) was the initial strength level of the participants, categorized into high-, middle-, and low-strength based on the initial force level in comparison to each other. Recovery values for males ranged from 100 to 118% in low-strength participants compared to 85 to 97% in middle- and high-strength groups combined (Kroll, 1967b). In females the recovery values ranged from 100 to 108% in low-strength participants compared to 85 to 101% in middle- and high-strength groups combined (Kroll, 1971b). Although Kroll did not speculate as to the reasons for the over-recovery of strength, he did suggest that motivation could not be dismissed as a possible explanation. Stull and Clarke (1971) similarly suggested that a ‘warm-up phenomenon’ may have increased muscle temperatures and contributed to over-recovery.

2.3.6 Postulated Mechanisms of Over-Recovery

The phenomenon of over-recovery observed by Kroll (1967b, 1971b) and Stull and Clark (1971) remains unexplained to date. Two postulated mechanisms that may contribute to over-recovery include neuromotor mechanisms and post-activation potentiation.

Neuromotor Mechanisms

Potential neuromotor mechanisms associated with over-recovery include an increase in agonist (tibialis anterior) activity, an increase in synergist (extensor digitorum longus) activity, and/or a decrease in antagonist (soleus) activity as measured by RMS amplitude. Lastly, there is the potential for synchronization of motor unit firing patterns as evidence by a decrease in mean power frequency (Gabriel et al., 2001). These

improvements in muscle coordination may be due to a motor learning effect. The role of motor learning and retention for an isometric strength task was examined by Calder and Gabriel (2007). Fifteen maximal isometric voluntary elbow flexion contractions were performed in either a set of 15 consecutive contractions or 3 sets of 5 contractions performed on three separate days. It was found that 15 contractions were sufficient for familiarization demonstrated by torque increases within the first two blocks of 5 contractions and stabilization within the third block. A set of 5 contractions was then performed 2 weeks and 3 months later with no training in between. A significant increase in force was found during both retention tests which cannot be attributed to any training effect from the initial 15 contractions. Motor learning was also seen in the sEMG activity as an increase in agonist RMS amplitude without any change in antagonist activity (Calder & Gabriel, 2007). It is possible that the serial contractions of a fatigue protocol constitute a motor learning stimulus similar to the 15 contractions used by Calder and Gabriel (2007). The brief amount of rest following the fatigue series and the contractions used to assess recovery are then analogous to retention tests. Thus, when fatigue has dissipated, the alterations in neuromotor coordination manifest themselves as over-recovery.

In support of this hypothesis, Gabriel, Basford, and An (2001) examined the role of motor learning in strength gains due to a fatigue protocol repeated after two and four weeks. Thirty maximal isometric elbow extension contractions were performed on three separate occasions, separated by two weeks. Torque increases of 8% were found from day 1 to 3, accompanied by a 24% increase in agonist (triceps) activity and a 39% increase in antagonist (biceps) activity. The increase of antagonist activity, although

contradictory to neuromotor adaptations for strength improvement, may have been due to the large increase in agonist activity and a need for joint stabilization.

Although a decrease in antagonist sEMG was not found by Gabriel et al. (2001) or Calder and Gabriel (2006), it can be speculated that a balance between agonist and antagonist co-activation is of primary importance. This concept was demonstrated by Carolan and Cafarelli (1992) following an 8-week training program of the knee extensors. An increase in maximal voluntary contraction force was found, accompanied by a decrease in hamstring (antagonist) activation, but no change in quadriceps (agonist) activation. Carolan and Cafarelli (1992) attributed the increase in knee extension strength to a “non-hypertrophic adaptation of the neuromuscular system” as demonstrated in decreased antagonist activity. This has also been shown when comparing trained and untrained individuals. Behm and St-Pierre (1998) found that the average maximal voluntary contraction of the trained individuals was 15% higher than the untrained, which may be explained by a small antagonist/agonist activity ratio (only 41% that of the untrained group). This demonstrated the ability of trained individuals to reduce antagonist co-activation. Extending these findings to a fatigue protocol, it is possible that subjects learn how to reduce antagonist co-activation while performing serial contractions. The result would then be stronger contractions during recovery.

The last neuromotor mechanism that may contribute to over-recovery is the synchronization of motor units following the fatigue protocol. Motor unit synchronization is a strength training adaptation, which allows for greater force production (Fling, Christie, and Kamen, 2009). The effect of synchronization has been shown to be most influential at higher force levels. Fling et al. (2009) found greater synchronization values

during 80% versus 30% maximal voluntary contraction force levels. Furthermore, the results suggested that even greater values might have been produced during 100% contractions.

Exercise induced motor unit synchronization has been shown to occur as a product of training status (Fling et al., 2009; Milner-Brown et al., 1975), a short-term training program (Milner-Brown et al., 1975), and even a single-session fatigue protocol (Dartnall et al., 2008). Milner-Brown and colleagues (1975) hypothesized that “synchronization of motor units might also arise from regular use of muscles to exert large, brief forces”. This hypothesis was first tested by comparing 6 maximal voluntary contractions of the first dorsal interosseus performed by weight lifters and untrained individuals. It was found that weight lifters had over 80% of motor units exhibiting high levels of synchrony as opposed to less than 20% of the untrained individuals’ motor units. This is supported by Fling and colleagues (2009) who found that trained individuals exhibited synchronization values 30% greater than untrained individuals. Secondly, Milner-Brown and colleagues (1975) also examined synchrony before and after a 6-week training program of the first dorsal interosseus. After the training program a force increase of 20% was present accompanied by a 56% increase in the synchronization ratio.

To extend the hypothesis of exercise-induced synchronization to over-recovery, the training effect would need to take effect within a single fatigue protocol. Dartnall et al. (2008) examined maximal voluntary elbow flexion contractions before and after a single-session fatigue protocol in the biceps brachii. An average of 125 contractions were performed resulting in an average force decrement of 46%. Immediately following the

fatigue protocol motor unit synchronization was found to have increased by 30% accompanied by an 11% increase in motor unit firing rates and a 100% increase in biceps activity. Although force was recorded immediately after fatigue and remained at a 46% decrement, these results suggest that increased synchronization from a single-session fatigue protocol may contribute to over-recovery.

Post-Activation Potentiation

The second potential mechanism, post-activation potentiation (PAP) is the product of intramuscular changes occurring after a conditioning stimulus such as a maximal voluntary contraction (Inglis et al., 2011; Sale, 2004). The main mechanism behind PAP is the increase in the calcium-sensitivity of the force development due to myosin regulatory light chain phosphorylation (Metzger et al., 1989; Persechini et al., 1985; Sweeney & Stull, 1990). The result of PAP is a transient increase in muscle twitch force and low frequency force (see Figure 11) (Hamada et al., 2000). Concurrent with this, is a decrease in the motor unit firing rate required to maintain a given force level due to the increase in force exerted per muscle fibre (Inglis et al., 2011; Klein, Ivanova, Rice, and Garland, 2001).

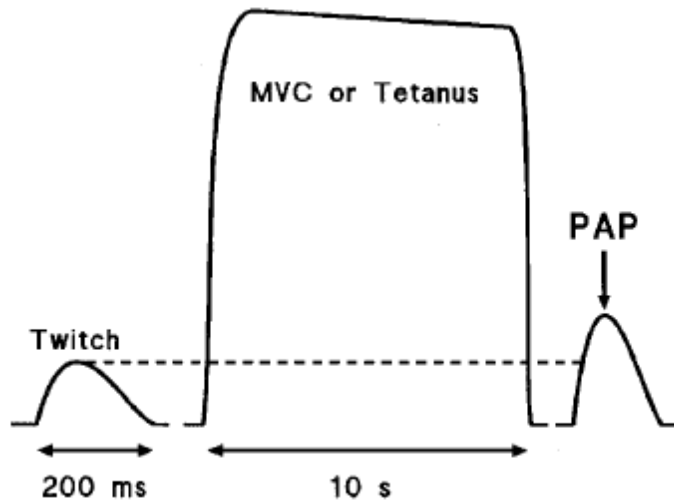


Figure 11. An example of post-activation potentiation (PAP). Twitches are evoked before and after a conditioning stimulus (maximal voluntary contraction or tetanus) resulting in increased twitch force and rate of force development. Sale, D. (2002).

Postactivation potentiation: Role in human performance. *Exercise and Sport Sciences Reviews*, 30(3), 138-143. Figure 1, page 139.

The mechanisms behind PAP have primarily been examined in mouse skeletal tissue. Gittings et al. (2011) examined the role of myosin regulatory light chain phosphorylation on twitch force potentiation after stimulation by comparing wildtype mice and myosin light chain kinase deficient mice. The initial stimulation consisted of a short potentiation protocol which resulted in a 30% twitch force increase from the wildtype group only. The second protocol consisted of fatiguing tetani which resulted in twitch force increases in both the wildtype (37%) and the kinase deficient group (14%). Lastly, the amount of phosphorylation following fatiguing stimulation was examined and found to be 3-4 fold in the wildtype group with no increase in the kinase deficient group. These results indicate that phosphorylation of the regulatory light chain is still present during moderate to severe fatigue. As well, the results of this study corroborate the hypothesis that the phosphorylation of the regulatory light chain preserves mechanical function during fatigue and increases twitch forces following conditioning stimuli. However, the potentiation of twitch forces following fatigue in the kinase deficient group may indicate that there are secondary mechanisms present during PAP. This is also supported by Rassier and colleagues (1999) who found that staircase potentiation existed in atrophied muscle deficient of regulatory light chain phosphorylation. This lends evidence to the importance of calcium on increased twitch force during staircase potentiation.

Potentiation is found to have the greatest relative effect when calcium levels are diminished. Previous literature is in agreement that regulatory light chain phosphorylation causes the greatest relative increase in twitch force when calcium levels are low (Palmer & Moore, 1989; Persechini et al., 1985; Sweeney & Stull, 1990). As

outlined by Millar and Homsher (1990), calcium is required for cross-bridges to form strong attachments. The state, or activation, of cross-bridges is primarily regulated by the binding of calcium and troponin which explains the large effect of calcium concentration on twitch force. Therefore, when calcium levels are diminished, baseline twitch force will be minimal and the *relative* amount of cross-bridges activated by regulatory light chain phosphorylation will be greater causing a greater *relative* increase in force (Sweeney & Stull, 1990).

The catch-like properties of skeletal muscle tissue shares some of the mechanisms associated with post-activation potentiation. First introduced by Burke and colleagues (1970; 1976), the catch-like property of muscle refers to the increase in force activated by high-frequency bursts spaced closely together during tetani. Catch-like trains of stimuli typically include two pulses, known as a doublet, at the beginning of a tetani, which increase the amplitude and duration of force that would be produced with a continuous-frequency train (Binder-MacLeod & Barrish, 1992; Burke et al., 1970; Ding et al., 2003).

Force augmentation, due to catch-like properties, is caused by two potential mechanisms. The first is an increase in muscle stiffness as the high-frequency pulses take up the slack in the muscle fibres' series elastic component (Binder-MacLeod & Barrish, 1992; Binder-MacLeod & Kesar, 2005; Ding et al., 2003). The second mechanism is an increase in Ca^{2+} concentration in the sarcoplasmic reticulum (Binder-MacLeod & Barrish, 1992; Binder-MacLeod & Kesar, 2005; Ding et al., 2003) which may also increase the myofibrillar Ca^{2+} sensitivity (Abbate et al., 2002). A combination of these mechanisms leads to an increased rate of tension development when the pulses are administered because there is an increased amplitude and duration of the resulting force

(Burke et al., 1970; Binder-MacLeod & Barrish, 1992; Ding et al., 2003). Catch-like properties have been thought to have the greatest effect on force in slow-twitch muscle fibres since the increase in the rate of tension development would be more pronounced (Binder-MacLeod & Barrish, 1992). The added recruitment of previously inactive motor units has been dismissed as a potential mechanism for catch-like force augmentation as firing rates have been found not to change throughout the responses (Burke et al., 1970; Burke et al., 1976; Ding et al., 2003). Since catch-like properties are most pronounced in fatigued, un-potentiated muscle (Burke et al., 1976; Ding et al., 2003) it is possible that the neuromuscular system may produce doublets during recovery contractions to contribute to over-recovery.

Finally the contribution of inorganic phosphate (P_i) to PAP is examined. The presence of P_i is known to depress isometric twitch force by causing force-producing cross-bridges to return to their inactive state (Pate & Cooke, 1989; Stienen et al., 1990). The hydrolysis of ATP to ADP during actin-myosin binding yields P_i as an end-product. Upon accumulation, “increasing concentrations of phosphate decrease the free energy of hydrolysis of MgATP” (Pate & Cooke, 1989). Millar and Homsher (1990) measured P_i during evoked isometric contractions at altered calcium concentrations. Force increases were negatively correlated with the accumulation of P_i due to a decreased number of active thin filament units. In the model presented by Millar and Homsher (1990), weak cross-bridges consisting of actomyosin (AM), ADP, and P_i are shifted to strong cross-bridges as P_i is released and AM·ADP cross-bridges remain. Furthermore, it was found that a reduction in P_i had the greatest relative and absolute effect on force production when calcium concentrations were low.

The application of these mechanisms to over-recovery was examined by Bruton and colleagues (1996) in frogs. Following a series of ten tetani to a single muscle fibre force, calcium, and P_i were measured. Tetanic force had declined by 10% following the stimuli and recovered to 109.4% after 5 minutes of recovery. This force augmentation persisted up to 15 minutes of recovery and had returned to baseline by 50 minutes. At the time of potentiation (during recovery) calcium concentration was diminished by 30% and P_i was reduced by 40% compared to pre-stimuli levels. The authors attributed the over-recovery to a 'warm-up' phenomenon due to these alterations in metabolite concentrations.

Post-activation potentiation is dependent on the maximal contraction or contractile history that is causing it (Bruton et al., 1996; Inglis et al., 2011; Smith et al., 2011). Baudry and Duchateau (2004) examine PAP in the tibialis anterior and found that the type of contraction (concentric, eccentric, and isometric) did not affect the presence of PAP in consecutive twitches. Post-activation potentiation has also been demonstrated in sport movements. Guillich and Schmidtbleicher (1996) found a significant increase in vertical jump height following a series of 3-5 maximal voluntary leg press contractions. The duration of the preliminary contraction(s) also appears to be independent from the presence of PAP. Increases in twitch forces have been found following a 5-second maximal contraction (Smith et al., 2011), 10-second maximal contraction (Inglis et al., 2011), a 5-second contraction at 75% maximal (Klein et al., 2001), and 16 consecutive 5-second maximal contractions (Hamada et al., 2003).

Smith and colleagues (2011) invoked potentiation in the triceps using a 5-second maximal voluntary contraction. The extent of PAP was examined by comparing evoked

twitches and a 10-second contraction at 25% maximal before and after the maximal voluntary contraction. Twitch torque was found to increase 217% when the muscle was in a shortened position (120° elbow flexion) and 77% when in a lengthened position (40° elbow flexion). Neuromuscular efficiency was used as a measure of PAP by dividing the muscle torque output by the sEMG RMS amplitude. Since the contraction level was held consistent at 25%, an increase in neuromuscular efficiency indicates that less neural input was required to maintain the same force level. There was a 12% increase in neuromuscular efficiency at a shortened muscle length, and no change in the lengthened muscle. Although the increase in neuromuscular efficiency was relatively low compared to the twitch force increase, the results do indicate that a maximal voluntary contraction has the ability to potentiate a voluntary contraction as well as an evoked twitch (Smith et al., 2011).

To apply PAP as a mechanism contributing to over-recovery, PAP during fatigue must be examined. Fowles and Green (2003) investigated PAP during low-frequency fatigue as caused by intermittent 30% maximal voluntary knee extension contractions. After performing contractions for 60 minutes, low-frequency twitch force had declined from 15-36% and did not recover in the 15 minute recovery period; ensuring low-frequency fatigue was in fact obtained. During the fatigue protocol, twitches had been administered at the 5, 20, 40 and 60 minute mark. Twitch force was potentiated at the 5 and 20 minute marks (17% and 13%, respectively) and displayed a 39% increase in the rate of force development of the twitch. By 40 minutes potentiation was no longer present, suggesting that fatigue began to dominate and over-take the capacity for potentiation (Fowles and Green, 2003).

Hamada and colleagues (2003) reached similar conclusions during a fatigue protocol consisting of 16 maximal voluntary knee extension contractions. Furthermore, the difference between participants with primarily type I and primarily type II fibres was examined. Twitches were administered after each of the 16 contractions and every 30 seconds during a 5 minute recovery period. For both groups, greatest PAP was elicited after the second maximal voluntary contraction, potentiating twitch force 126% in type II group and 38% in type I group. Post-activation potentiation was evident in twitches following the first 8 to 10 contractions before fatigue exhausted the capacity to potentiate. Following the 16 maximal voluntary contractions, twitch force was diminished 34% in the type II group and 17% in the type I group; voluntary force had declined 49% in the type II group and 23% in the type I group. Although the type II group exhibited the greatest potentiation, they also experienced the greatest amount of fatigue in twitch and voluntary force. These results suggest that PAP and fatigue are not mutually exclusive and can occur simultaneously although only one is prominent at a given time. Figure 12 displays the effect of PAP following a conditioning stimulus. At low frequencies, the conditioning stimulus elicits an increase in force through PAP. However, at higher frequencies the effect of the conditioning stimulus is fatigue. This demonstrates the hypothesis that, while PAP and fatigue can occur simultaneously, they can have opposing effects on force depending on the conditions (i.e. frequency). Although the TA is primarily a slow twitch muscle it has been shown to have the capacity for potentiation (O'Leary et al., 1997). As demonstrated by Hamada and colleagues (2003) type II fibres have a greater capacity for potentiation following a single conditioning stimulus, however, the recovery of PAP following fatigue was similar in both type I and II fibres.

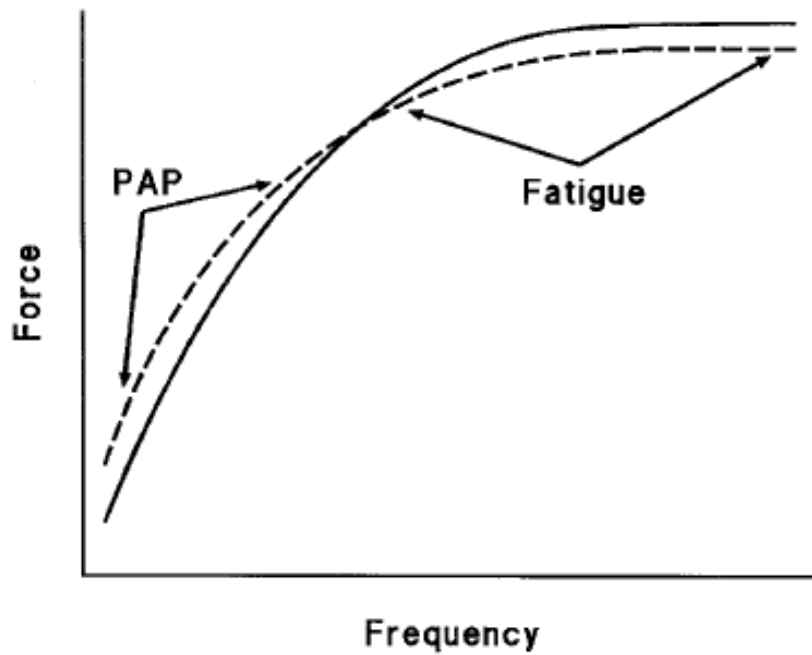


Figure 12. The effect of post-activation potentiation (PAP) on the force-frequency curve.

At low-frequencies PAP potentiates force while at high-frequencies the conditioning stimulus may actually diminish the amount of force that can be produced due to fatigue.

Sale, D. (2002). Postactivation potentiation: Role in human performance. *Exercise and Sport Sciences Reviews*, 30(3), 138-143. Figure 2, page 139.

Finally, we examine the effects of a warm-up protocol on high-frequency force. Since PAP has only been found to potentiate twitch force it may be present during, but not the primary cause of, over-recovery. The effects of a warm-up protocol consist of an increase in high-frequency tetanic force and an increase in the rate of high-frequency force development following a conditioning stimulus (Baudry & Duchateau, 2007; Bruton et al., 1997). The cause of this force increase is thought to be due to increased buffering of P_i following a conditioning stimulus, to compensate for the increase in P_i levels. Since over-recovery is the increase in high-frequency, voluntary force and not twitch force, this warm-up effect may be a more likely explanation for over-recovery than PAP.

High-frequency force increases have been seen following a single maximal voluntary contraction (Baudry & Duchateau, 2007) as well as a series of fatiguing tetanic contractions (Bruton et al., 1996; Bruton et al., 1997). Force increases have been found in a variety of muscles including *Xenopus* lumbrical muscle (Bruton et al., 1996), mouse soleus muscle (Bruton et al., 1997), human adductor pollicis muscle (Baudry & Duchateau, 2007) with the average amount of potentiation being 110% of the initial levels of tetanic force. Although the same potentiating effect was not found during voluntary contractions of the human adductor pollicis muscle, the same mechanisms may be responsible for an increased rate of force development following a conditioning stimulus (Baudry & Duchateau, 2007).

This increase in high-frequency tetanic force and rate of force development in voluntary contractions following a conditioning stimulus may contribute to the over-recovery of voluntary maximal force. One mechanism behind the phenomenon is thought to be an increase in buffering of P_i following a conditioning stimulus. During fatiguing activity the breakdown, or hydrolysis, of Creatine Phosphate (CP) and ATP causes an increase in the concentration of myoplasmic P_i (Kentish, 1986). This build up of P_i depresses force production by reducing myofibrillar Ca^{2+} sensitivity (Brandt et al., 1982; Fryer et al., 1995; Kentish, 1986) and hindering the transition of cross-bridges from low-force (weak) to high-force (strong) states (Westerblad et al., 2002). This causes a rightward shift in the pCa/tension curve as more Ca^{2+} is needed to produce the same given amount of force (see Figure 13). A second force-limiting consequence is the increase of P_i in the Sarcoplasmic Reticulum (SR) lumen which causes a reduction in the amount of Ca^{2+} available for release (Fryer et al., 1995). A decrease in the rate of SR Ca^{2+} release causes decreases in the amount of force that can be produced and the rate at which force is produced (Fryer et al., 1995).

An increase in P_i buffering may alleviate the detrimental effects of P_i accumulation during fatiguing activity. In a study by Phillips and colleagues (1993) mouse soleus and EDL fibres were bathed in metabolic fuel solutions and examined during a 10 or 50 Hz tetanic contraction. The fibres bathed in a pyruvate solution demonstrated potentiation similar to that found with the warm-up protocol at both frequencies in the soleus muscle whereas fibres bathed in glucose demonstrated no increase in tetanic force. An increase in the rate of force development, and a decreased rate of relaxation were also present following the application of pyruvate. The authors

concluded that these effects were due to the significant decrease in P_i following only the pyruvate bath, accompanied by an increase in average force produced per active cross-bridge. No potentiation was seen in the EDL muscle which was speculated to be due to the very low resting P_i levels in that particular muscle.

Since P_i has been shown to increase following fatiguing activity and potentiation has been shown to occur when P_i levels are depressed, there must be a buffering, or removal, of P_i present following activity in order for potentiation to exist. Bruton and colleagues (1997) examined potentiation of tetanic force and P_i levels following 2 conditioning stimuli in mouse soleus muscles. Potentiation (110%) was only present following the more demanding stimuli (15 tetani at 2-second intervals versus 5-second intervals). Concurrently, there was a 50% decrease in P_i following the more demanding 2-second interval stimulus and no change in the 5-second interval condition demonstrating that a certain amount of metabolic stress is required to depress P_i through buffering. While the authors concluded that P_i buffering was responsible for the decrease in P_i levels and therefore the potentiation in tetanic force, they could not specify the exact mechanism responsible for this buffering suggesting an 'inwardly directed P_i transport system' (Bruton et al., 1997). The effects of a warm-up protocol may be a contributor of over-recovery if the presence of a mild fatigue protocol produces an increase in the buffering of accumulated P_i thereby offsetting its detrimental effects to force production.

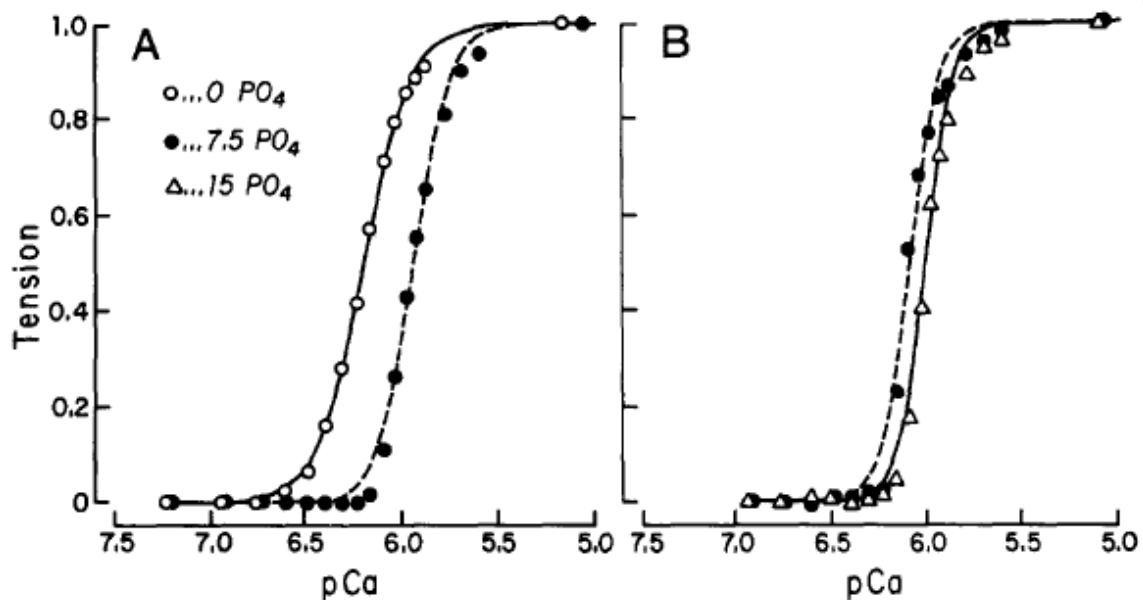


Figure 13. The pCa/tension curve in rabbit psoas muscle demonstrating the rightward shift with the introduction of greater P_i levels. The open circles are from fibre bathed in 0-phosphate saline, the closed circles are the same fibre bathed in 7.5 mM phosphate saline, and finally the open triangle is a separate fibre bathed in 15 mM phosphate saline.

Brandt, P., Cox, R., Kawai, M., & Robinson, T. (1982). Regulation and tension in skinned muscle fibres: Effect of cross-bridge kinetics on apparent Ca^{2+} sensitivity.

Journal of General Physiology, 79, 997-1016.

2.3.7 Recovery Summary

The process of recovery from fatiguing exercise is often categorized based on the speed it takes mechanisms diminished during fatigue to return to pre-fatigue values. Three distinct phases of recovery were presented by Miller et al. (1987) and defined as early, intermediate and prolonged recovery. Early recovery is said to be the return of membrane function as shown in the recovery of the M-wave (Miller et al., 1987). Rapid recovery also includes blood flow restoration removing accumulated metabolites (Baker et al., 1993; Cornwall et al., 1994; Yates et al., 1987), RMS amplitude returning to initial values (Baker et al., 1993; Cornwall et al., 1994; Petrofsky, 1981), and the increase of MPF (Cornwall et al., 1994; Kuorinka, 1988; Mills, 1982; Petrofsky, 1981). Intermediate recovery includes the balancing of intramuscular pH, the return of high-energy phosphates, and the increase of force close to pre-fatigue levels (Miller et al., 1987). However, prolonged recovery can last for hours after fatigue caused mainly by the persistent failure in excitation-contraction coupling (Miller et al., 1987). Since the amount and speed of recovery is contingent on fatigue, the restorations of mechanisms diminished during fatigue are also dependent on the many factors involved in fatigue.

The phenomenon of over-recovery not only requires a return to homeostasis but an improvement in force producing mechanism(s). The neuromotor mechanisms that may elicit over-recovery include an increase in agonist or synergist activation, a decrease in antagonist activation, or a synchronization of active motor units. These force-producing factors have been found to increase with training and may improve over the course of a fatigue protocol therefore contributing to over-recovery. In addition is the existence of post-activation potentiation (PAP) concurrent to fatigue. The phosphorylation of the

regulatory light chain may contribute to over-recovery by increasing the amount of force that is produced during actin-myosin cross-bridging for the same relative amount of calcium (Metzger et al., 1989; Persechini et al., 1985; Sweeney & Stull, 1990). A full return of force levels to initial values may be the result of PAP if accompanied by decreased RMS or MPF values. This would suggest that less neural input is being required to maintain the same initial force level (improved neuromuscular efficiency) as a product of PAP. The co-existence of PAP and fatigue has been demonstrated during a maximal fatigue protocol and may contribute to over-recovery as fatigue subsides.

CHAPTER 3: METHODS AND MATERIALS

3.1 Participants

Sample size estimation was completed according to the methods outlined for Cohen's (1988) case four formula (Appendix A) and revealed a necessary 14 participants for the current study. A total of 17 participants were collected to guard against the possibility that means and standard deviation error may be higher in the present study compared to the pilot data included in the sample size estimation. Each participant was verbally acquainted with the physical demands of testing, and was provided written informed consent to participate in the study (Appendix B) as approved by Brock University Research Ethics Board (REB#02-283). All participants were engaged in weightlifting activity at least 6 months prior to their participation in the study.

3.2 Experimental Design

3.2.1 *Experimental Apparatus*

Participants were seated in an adjustable chair with hips and knees at approximately 90°. Adjustable belts were strapped across the waist and chest to avoid extraneous movement. The participant's right foot was secured on a foot plate positioned at 20° of plantar flexion (McIntosh & Gabriel, 2011; Marsh et al., 1981; McNeil et al., 2004). A load cell (JR3 Inc., Woodland, CA) attached to the bottom of the foot plate recorded force. An adjustable metal bar (padded with 1 cm of foam) was secured above the metatarsal joints and tightened to a comfortable position to restrain the foot. The participant's left leg rested on a foot support. Participants were instructed and monitored not to use their hands, arms or upper legs to assist in the dorsiflexion contractions. An

oscilloscope (VC-6525, Hitachi, Woodbury, NY) was placed in front of the participants and traced their force during voluntary contractions (see Figure 14). This was to encourage participants to hold a steady force level throughout a contraction.

3.2.2 Evoked Potentials

Twitch contractions in the tibialis anterior were evoked through electrical stimulation of the deep peroneal nerve, via a pedal operated stimulator (Grass Telefactor S88, Astro-Med Inc., West Warwick, RI), while participants were seated in the experimental apparatus. A self-adhesive cathode (3.2 cm diameter, 879100, Axelgaard Manufacturing Co., Ltd., Fallbrook, CA) was placed on the posterior portion of the fibular head. A self-adhesive anode (5 cm diameter, CF5000, Axelgaard Manufacturing Co., Ltd., Fallbrook, CA) was placed directly in line with the cathode on the opposite side of the joint (see Figure 15). The stimulus duration was 1 ms with at least 20 seconds between each pulse (Christie, Lester, LaPierre, and Gabriel, 2004). The resulting massed action potentials (M-wave) was viewed on an oscilloscope (VC-6525, Hitachi, Woodbury, NY) while the level of stimulation was increased until there was no further increase in M-wave amplitude. The force-time curves from the twitch contraction was used to assess presence or absence muscle fatigue and potentiation. An evoked potential superimposed upon a maximal voluntary contraction is termed an “interpolated twitch” and this procedure was used to ensure that participants were generating maximal effort contractions.

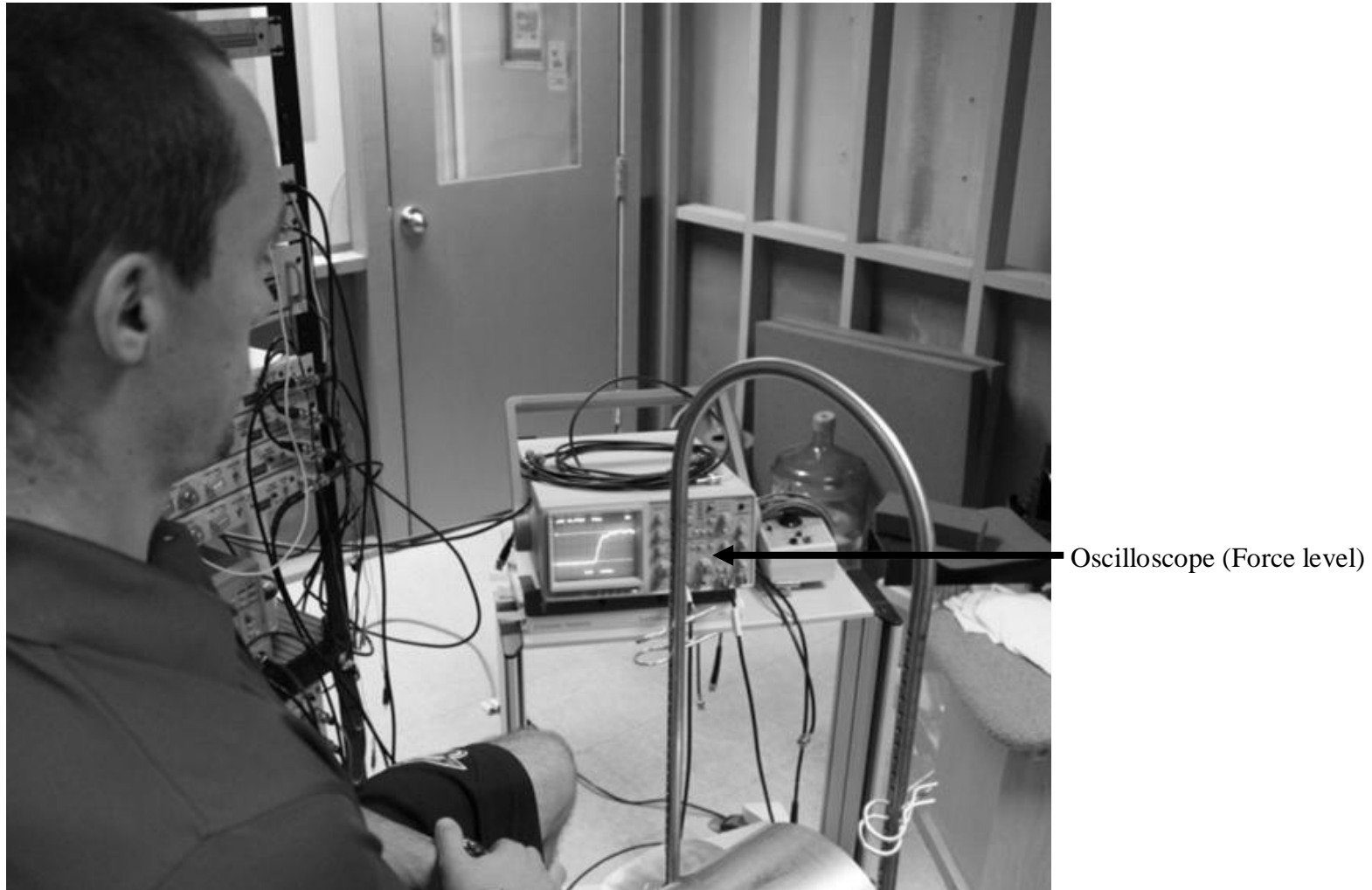


Figure 14. Experimental set-up within a Faraday cage in the Electromyographic Kinesiology Laboratory at Brock University.

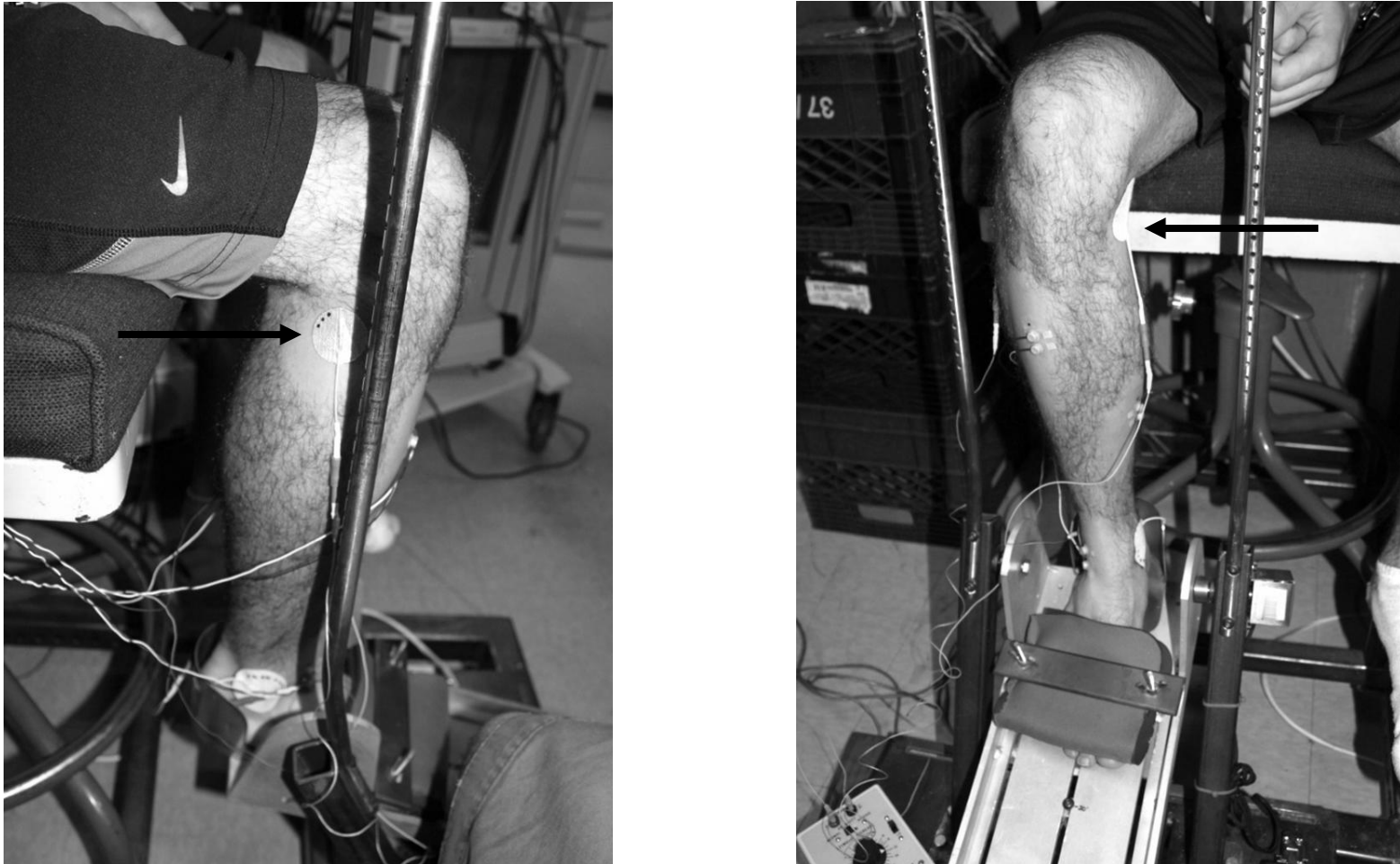


Figure 15. Experimental set-up displaying peroneal nerve stimulation. Location of cathode is posterior to the fibular head (above left) and location of anode is directly in line with the cathode on the opposite side of the joint (above right).

3.2.3 Surface Electromyographic Recordings

For each session, the participant's right leg was prepared for sEMG recordings. Small areas of the tibialis anterior, extensor digitorum longus, and soleus were shaved, abraded (NuPrep®, Weaver and Company, Aurora, CO), and cleansed with isopropyl alcohol. Skin-electrode impedance was maintained below 10 K Ω as measured by an impedance meter (Grass EZM5, Astro-Med Inc., West Warwick, RI). To ensure that changes in the sEMG were due to the experimental procedure, rather than the impedance of the electrodes, impedance was measured before and after the full protocol for a sample of participants (N=8). There was no significant difference ($p > 0.05$) in the impedance before (5.3 ± 2.8 K Ω) compared to after the protocol (5.0 ± 2.7 K Ω).

The motor point of each muscle was found using low level stimulation across the surface of the skin at a rate of 1.5 pulses per second. Bipolar electrode configurations were placed on each of the three muscles located 1 cm distal to the electrically identified motor point. Pediatric-sized electrodes (3 mm electrode diameter, F-E9M 11 mm, GRASS Technologies, Asto-Med, Inc., Warwick, RI) with an interelectrode distance of 1 cm were used to record muscle activity from the tibialis anterior, soleus and extensor digitorum longus. Electrodes were affixed with two sided tape and electrolyte gel (Signa Gel®, Parker Laboratories, Fairfield, NJ). The ground electrode for the tibialis anterior and soleus was placed on the medial malleolus. Likewise, the ground electrode for the extensor digitorum longus was placed on the lateral malleolus (see Figures 16 and 17).

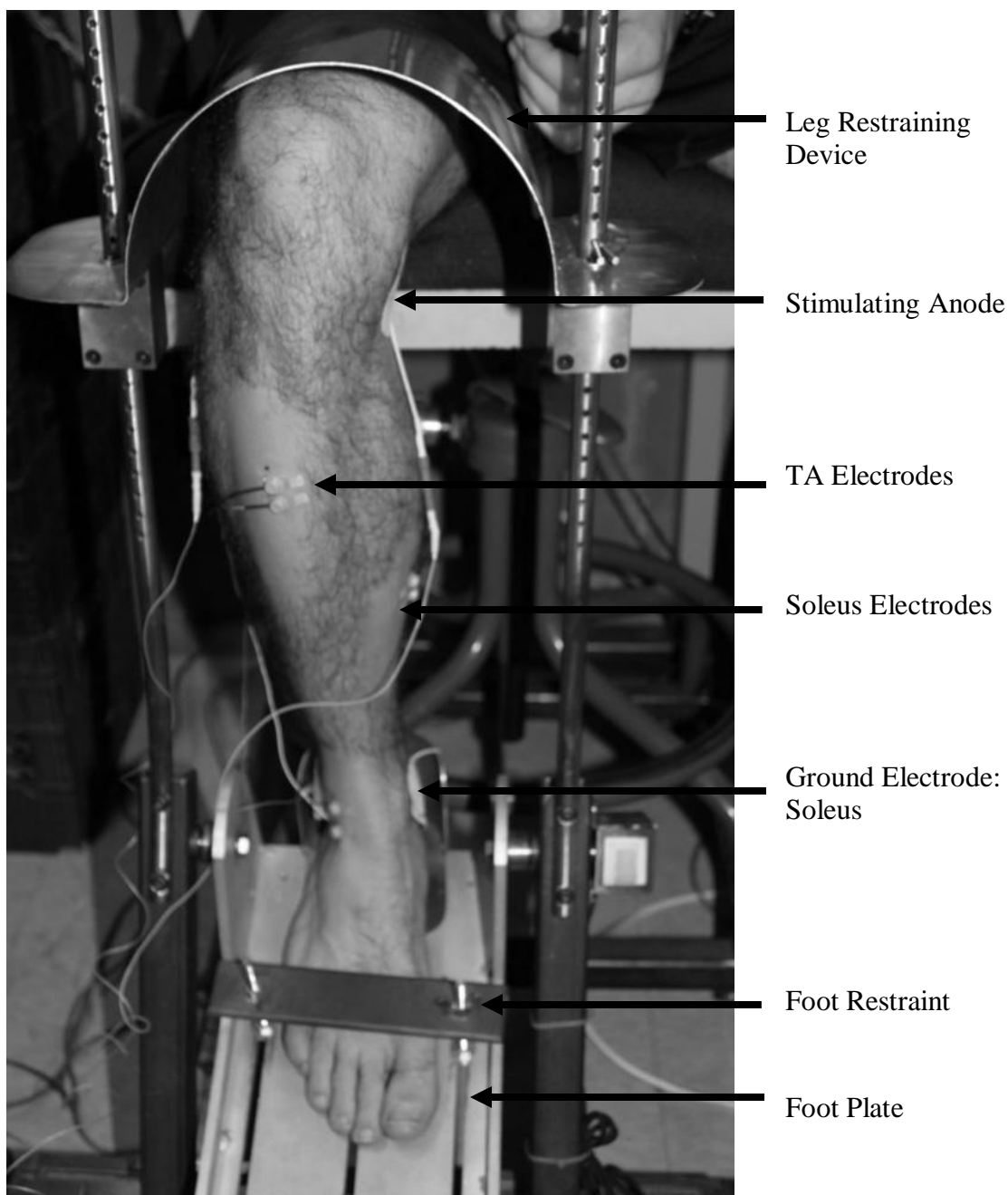


Figure 16. Experimental set up displaying EMG and force recordings (frontal view).

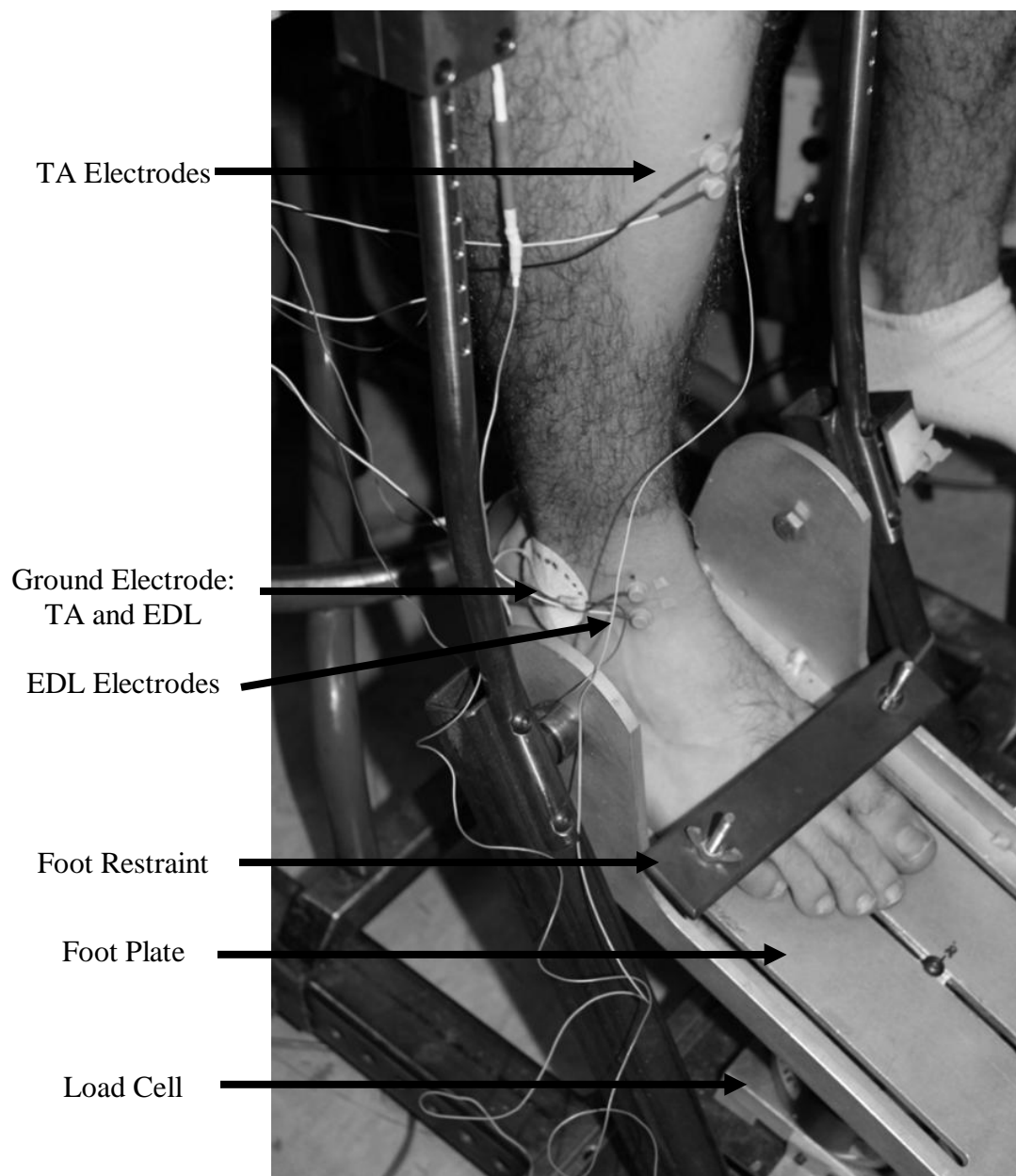


Figure 17. Experimental set up displaying EMG and force recordings (side view).

3.2.4 Measurement Schedule

The measurement schedule that was used to test participants is depicted in Figure 18. The bars denote maximal voluntary contractions whereas the spikes represent evoked potentials for muscle twitches. A combination of the two (bar and spike) denotes a maximal voluntary contraction with an interpolated twitch. In brief, there was one familiarization test session followed by two test days. Each test day corresponded to a fatigue protocol that required a different percent decrement. Condition 1 corresponds with the 10% force decrement and condition 2 with the 20% force decrement.

3.2.4.1 Familiarization

Testing took place in the Electromyographic Kinesiology Laboratory at Brock University (WH 21). Participants completed a total of three sessions, each separated by at least 24 hours. The initial session was to familiarize participants with the demands of the experiment and obtain demographic information, physical activity levels, and anthropometric measurements (APPENDIX C). The familiarization session began with a series of 3 evoked contractions. Participants then completed 15 maximal isometric dorsiflexion strength trials. Each contraction was 5 seconds in duration with a two-minute intertrial rest interval (Kooistra, de Ruiter, and de Haan, 2005; Whitley and Elliott, 1968). Voluntary contractions 5, 10, and 15 included an interpolated twitch in the middle of the trial. The familiarization session then concluded with a series of 3 evoked contractions. One goal of the familiarization session was for participants to learn how to perform maximal effort contractions of the tibialis anterior while minimizing extraneous movements that may recruit other muscles (Calder & Gabriel, 2007; McIntosh & Gabriel, 2011).

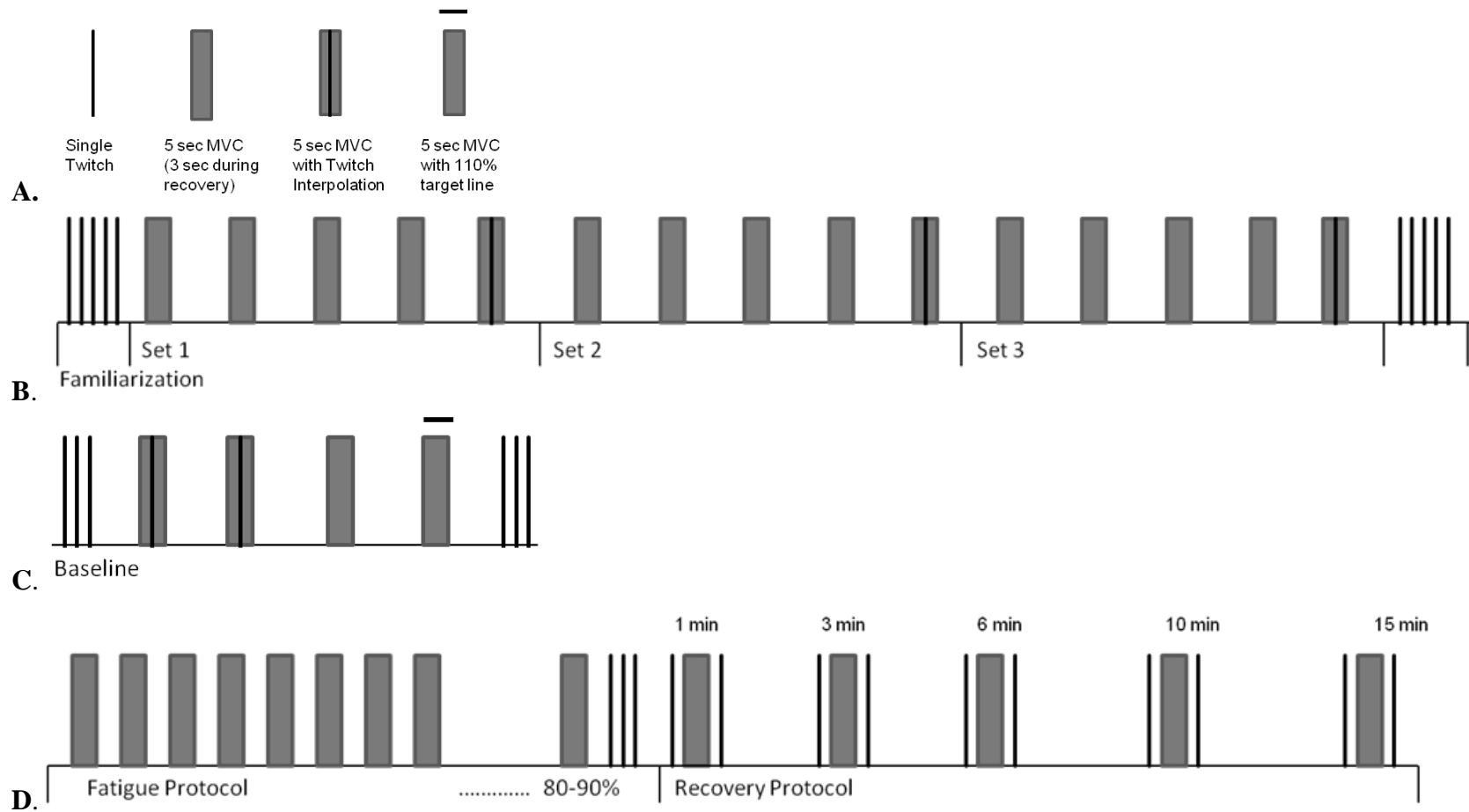


Figure 18. Experimental protocol, as denoted by the above legend (A). A series of 15 contractions were performed during the familiarization test day (B). Baseline testing (C) and the fatigue and recovery protocol (D) occurred on both test days.

3.2.4.2 Baseline Testing

Three twitch contractions were evoked immediately before baseline maximal isometric strength testing. Participants then completed a series of four baseline maximal voluntary contractions prior to the fatigue and recovery protocols. Each contraction was 5-seconds in duration with inter-trial rests of 2 minutes. The force of the contractions was presented in real time to the participant on an oscilloscope (TDS-460A, Tektronix, Beaverton, OR). The peroneal nerve was stimulated during the middle 3 seconds of the first two contractions to evoke interpolated twitches. A third contraction was then performed without any interference. After the first three contractions, a target line was presented on the oscilloscope (TDS-460A, Tektronix, Beaverton, OR) at 110% of the highest force achieved during the previous contractions. If the participant was able to reach this line another contraction was performed with a target line at 110% of the participant's previous maximum, if the participant did not reach this line their previous force level was taken as their maximal voluntary contraction (Baratta et al., 1998). Three twitch contractions were then evoked upon completion of the last baseline maximal isometric strength testing trial.

3.2.4.3 Fatigue and Recovery Testing

Five minutes after baseline testing, participants completed a fatigue protocol to either a 10 or 20% decrement in strength, each corresponding to a separate test session. The order of the two testing sessions was balanced over participants across days to minimize the possibility of "carry-over" effects due to the previous test session. Participants were unaware of the force decrement required for the fatigue protocols. This was to ensure that maximal effort was exerted without a decline to purposefully shorten

the duration of activity (Stokes and Dalton, 1991). Both protocols consisted of a series of 5-second maximal voluntary contractions separated by 10-seconds of rest. The work-to-rest ratio was controlled by a digital recording that instructed participants when to contract and relax. Participants received no verbal encouragement. However, feedback was given to ensure that participants isolated the tibialis anterior during isometric dorsiflexion, and that the force of the each contraction reached a steady plateau as observed on an oscilloscope (TDS-460A, Tektronix, Beaverton, OR) placed in front of them. Serial isometric contractions were continued until participants had achieved the target strength decrement (10% for condition 1, 20% for condition 2) for three consecutive contractions (Stokes and Dalton, 1991). Three evoked contractions were then elicited immediately following the last contraction of the fatigue protocol.

Assessment of recovery consisted of five maximal effort isometric dorsiflexion contractions performed at 1, 3, 6, 10, and 15 minutes after the last contraction of the fatigue protocol was completed. The contractions were three seconds in duration. One twitch was elicited immediately (approximately 3 seconds) before and after each recovery contraction. Lastly, skin temperature was recorded from the location of the tibialis anterior electrodes immediately prior to, and following, the full session on each testing day.

3.3 Signal Processing

The signal-to-noise ratio for data collection was maintained between 20 and 40 dB. The sEMG signals were amplified (Grass P511, Astro-Med, Inc., Warwick, RI) to maximize the resolution of the 16-bit analogue-to-digital converter (DI-205, DATAQ Instruments, Akron, OH). The sEMG signals were then band-passed filtered (3-1000 Hz)

prior to digitization at 2000 Hz (WinDaq Acquisition, DATAQ Instruments, Akron, OH). The force signal from the JR3 load cell (JR3 Inc., Woodland, CA) were low-passed (15 Hz, 3 dB) using a 4th order Butterworth digital filter, off-line in MATLAB (The Mathworks Inc., Natick, MA).

3.4 Data Reduction

Sample force and sEMG signals during a maximal voluntary contraction from a representative pilot subject are presented in Figure 19. The criterion measures were calculated from a 500 ms window immediately before the middle of each contraction. For contractions with an interpolated twitch, the window is 500 ms immediately before the stimulus pulse. The criterion measures include: mean force, and root-mean-square (RMS) amplitude, and mean power frequency (MPF) of the sEMG signal from tibialis anterior, extensor digitorum longus, and soleus. Algorithms were used to calculate these measures in MATLAB, as detailed in Appendix D. In the case of maximal voluntary contractions with an interpolated twitch, there were two data windows. Peak force was obtained and compared 250 ms before and after the stimulus pulse, as depicted in Figure 20. The criterion measures extracted from the force-time curve produced by twitch contractions are depicted in Figure 21, which shows how contraction and relaxation times were defined.

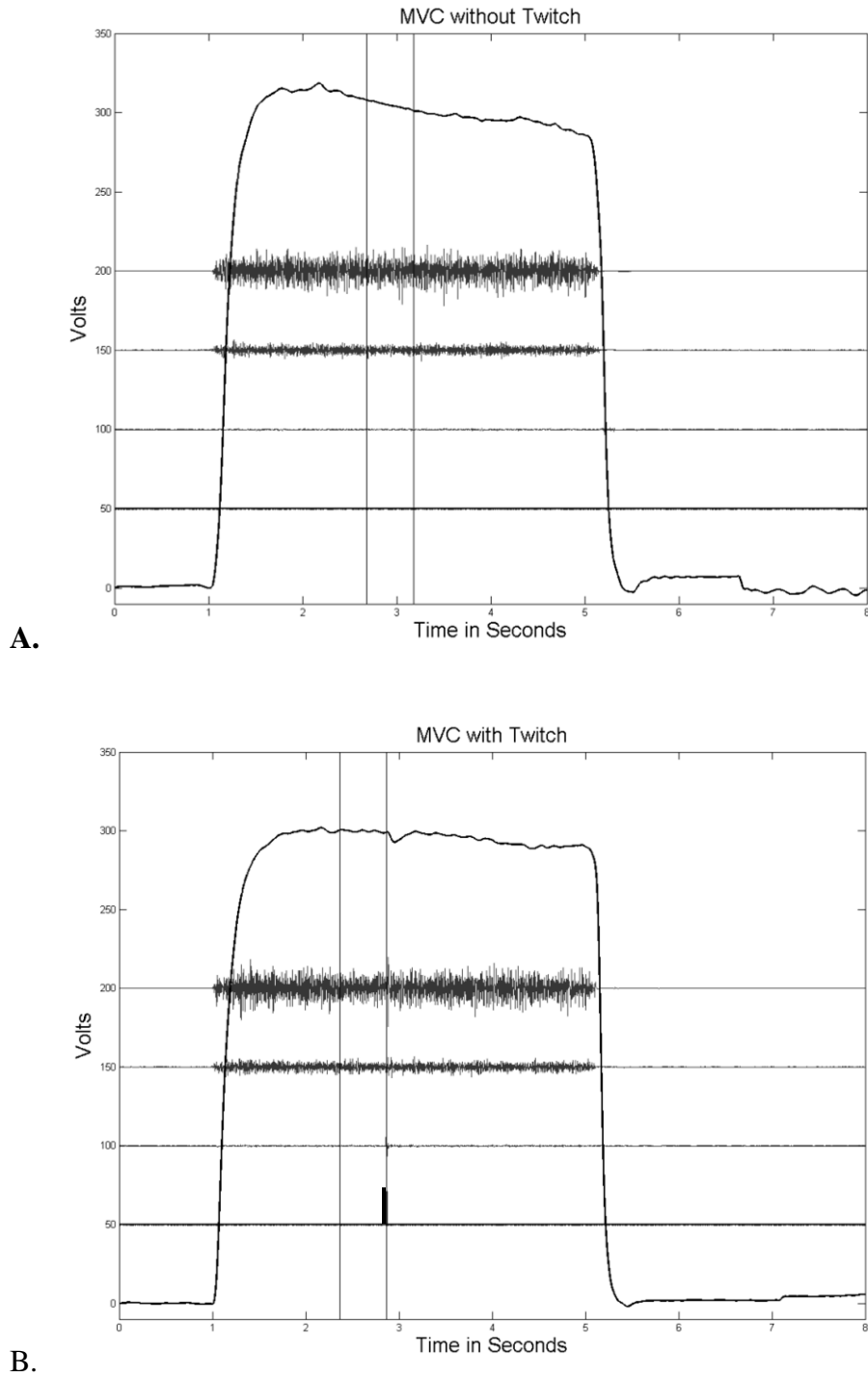


Figure 19. Criterion measures (force, RMS, MPF) as extracted from a 500ms window occurring immediately before the middle of the contraction (A) or immediately before a stimulus pulse, if applicable (B).

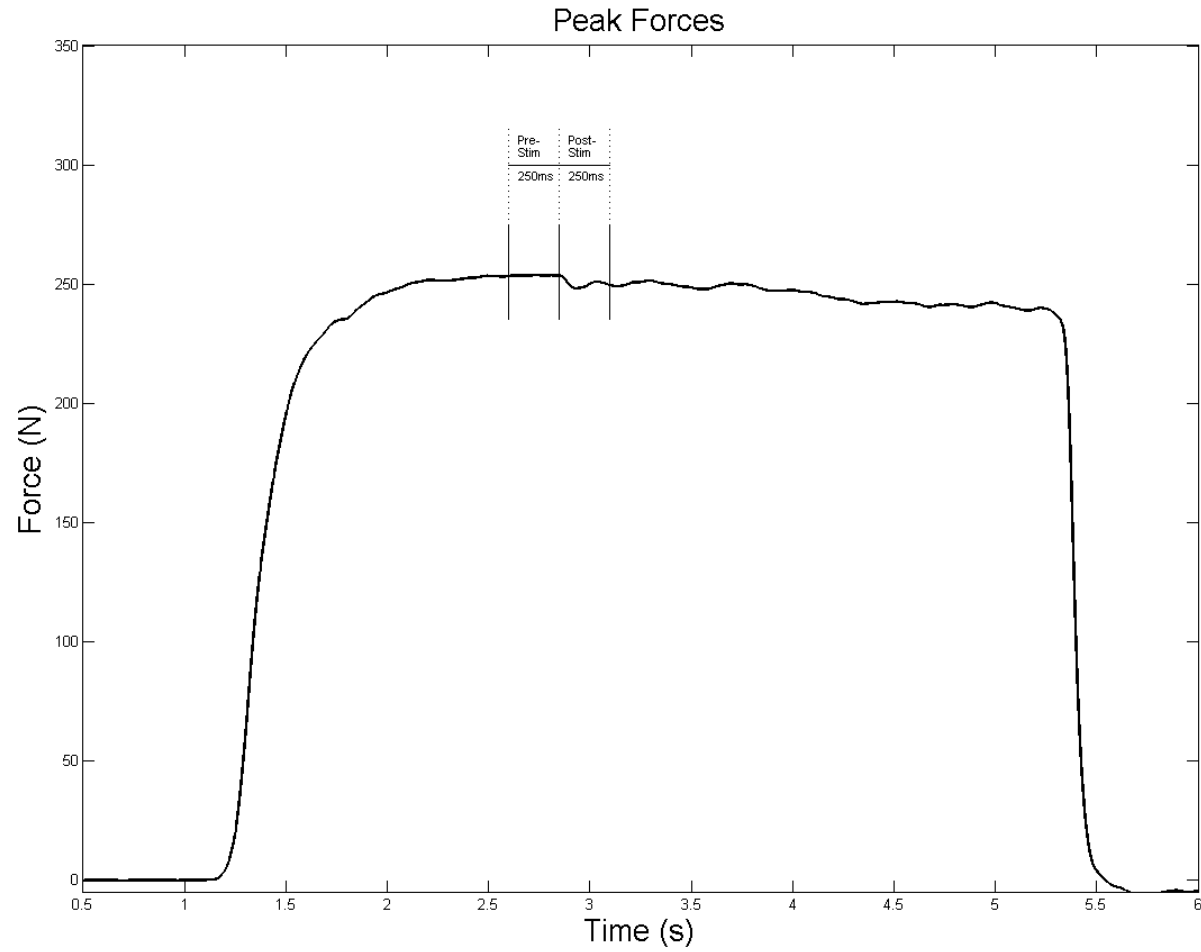


Figure 20. Criterion measures (peak force occurring 250ms immediately before and after the occurrence of a twitch) as extracted from a maximal voluntary contraction with twitch interpolation.

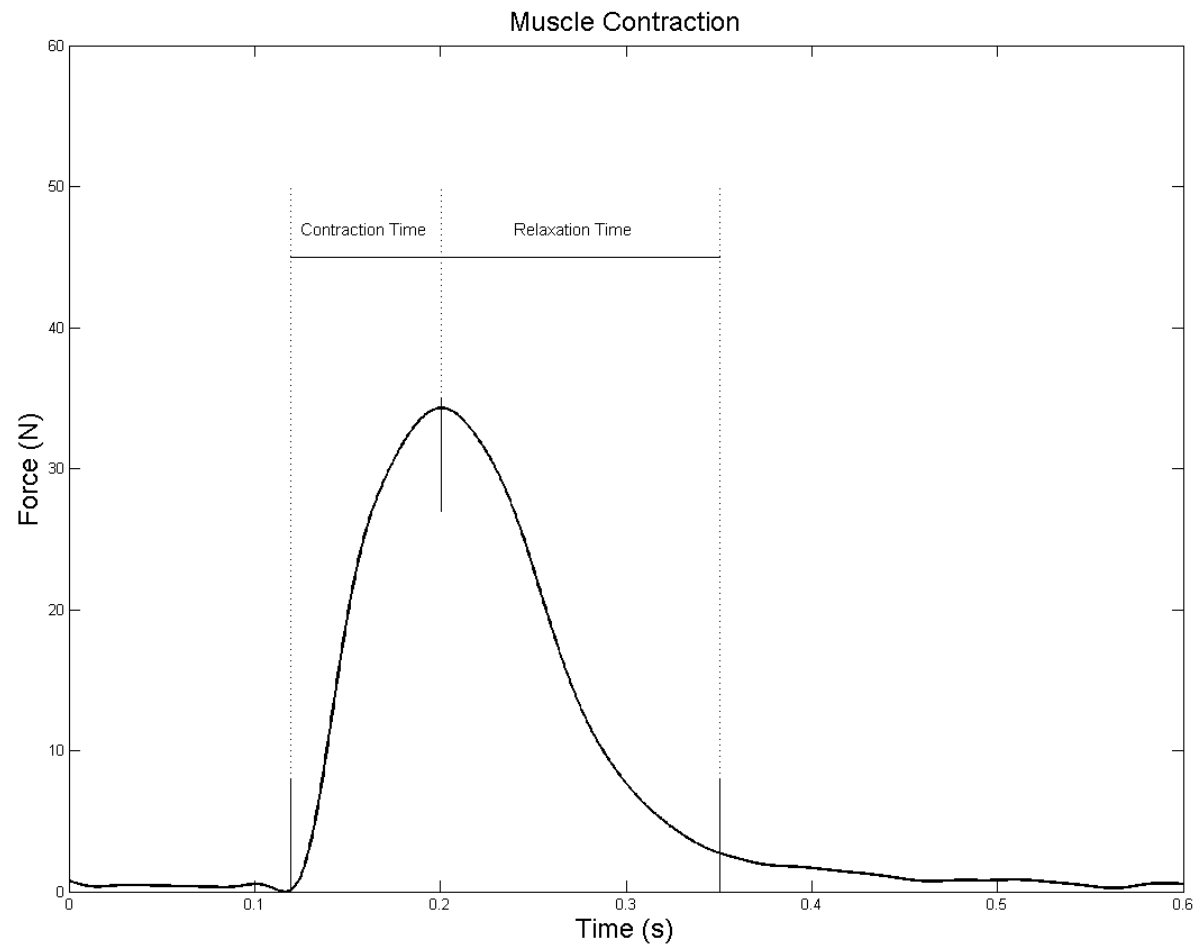


Figure 21. Criterion measures (contraction and relaxation time) as extracted from the force-time curve produced by a twitch contraction.

3.5 Statistical Analysis

All ANOVA statistics were performed using SAS[®] Software (SAS Institute Inc., Cary, NC). All orthogonal polynomials were performed using SYSTAT[®] Software (Systat Software, Inc., Chicago, IL).

3.5.1 Reliability

All criterion measures obtained during baseline testing were subjected to reliability analysis, which involved a consideration of both the consistency of scores within subjects and the consistency of means across test days. The consistency of scores within subjects were evaluated using the intraclass correlational analysis of variance (ANOVA) technique. A fully nested ANOVA model with two dimensions (Days and Subjects) was required. The repeated measurements (Trials) on subjects within each test day constitute a “Within-Cells” replication of measures (Feldt & McKee, 1958). The error term in this model is the Within-Cells error variance. The mean squares from this ANOVA model were used to calculate the intraclass correlation coefficient (Appendix E). The stability of the means across test days were assessed by a complimentary two factor ANOVA with main effects for Days and Subjects but Days were not nested within Subjects. Thus, there was a Day-by-Subjects interaction term while the Within-Cells remained as the error term (Feldt & McKee, 1958). Sample tables are presented for the consistency (Table 1) and reliability (Table 2) of mean force.

Table 1. Sample nested analysis of variance table for the consistency of mean force across the four baseline trials repeated on two days.

	df	SS	MS	F	P
Subjects	13	350073.82	26928.76	82.71	<.0001
Days (Subjects)	14	11743.53	838.82	2.58	0.0039
Within Cells	84	27348.92	325.58	--	--

Table 2. Sample nested analysis of variance table for the reliability of mean force across the four baseline trials repeated on two days.

	df	SS	MS	F	P
Days	1	3065.40	3065.40	9.42	0.0029
Subjects	13	350073.82	26928.76	82.71	<.0001
Days \times Subjects	13	8678.13	667.55	2.05	0.0260
Within Cells	84	27348.92	325.58	--	--

3.5.2 *Fatigue and recovery*

A three-factor repeated measures ANOVA was used to analyze the first three trials of the fatigue series, the last three trials of the fatigue series, and the last three trials of the recovery series across two conditions (i.e., fatigue conditions). Thus there were 2 conditions consisting of 3 blocks each, which each included 3 trials for a $2 \times 3 \times 3$ ANOVA. A sample table is presented to illustrate the statistical model for mean force (Table 3). The inclusion of the first and last three trials of fatigue are analogous to a manipulation check to determine if there was a significant difference between the two target levels of strength decrement (10 versus 20%). The inclusion of recovery trials was used to address the main thesis questions: that is, does the phenomenon of over-recovery exist, and if evident, does it depend on the level of fatigue? Since the focus of this study was on the first order interactions a trend analysis was performed on the resulting means using orthogonal polynomials for the linear and quadratic components.

To assess neuromuscular coordination between the TA and soleus activity a co-activation ratio was determined using the sEMG RMS amplitude. The co-activation ratio was calculated by dividing the TA RMS amplitude by the soleus RMS amplitude. An increase in this ratio means that there was less relative co-activation achieved either by an increase in TA RMS amplitude or a decrease in soleus RMS amplitude compared to their baseline values. A repeated measures ANOVA was used to compare the co-activation ratio across the three blocks in the fatigue and recovery protocol.

Table 3. Sample three factor repeated measures analysis of variance for mean force across fatigue and recovery.

	df	SS	MS	F	P
Subjects (S)	13	--	--	--	--
Conditions (C)	1	11249.05	11249.05	11.445	0.005
Error (S × C)	13	12777.49	982.88		
Blocks (B)	2	87935.31	43967.65	54.314	0.000
Error (S × B)	26	21047.21	809.51		
Trials (T)	2	3077.83	1538.92	18.231	0.000
Error (S × T)	26	2194.69	84.41		
C × B	2	1282.52	641.26	5.303	0.012
Error (S × C × B)	26	3143.99	120.92		
C × T	2	607.86	303.93	3.829	0.035
Error (S × C × T)	26	2063.67	79.37		
B × T	4	2494.02	623.50	5.508	0.001
Error (S × B × T)	52	5886.27	113.20		
C × B × T	4	216.61	54.15	0.674	0.613
Error (S × C × B × T)	52	4177.30	80.33		

3.5.3 Twitch force during fatigue and recovery

Twitch force and duration were analyzed using a three-factor ($2 \times 4 \times 3$) repeated measure ANOVA similar to Table 3 presented for mean force with the addition of an extra degree of freedom for blocks. Groups of 3 trials were performed for 4 blocks (baseline, pre-fatigue, post-fatigue, recovery) which were performed for 2 conditions by each participant. The first two blocks of trials (baseline and pre-fatigue) were compared to evaluate any alteration in muscle properties due to baseline strength assessment before the fatigue series. The comparison of the middle two blocks (pre-fatigue and post-fatigue) was used to further assess fatigue effects by comparing the last three twitches evoked during baseline testing to those elicited upon completion of the fatigue series, on both test days (i.e., fatigue conditions). Finally, potentiation effects were evaluated by comparing the last three twitches evoked during baseline testing (pre-fatigue) to those elicited immediately prior to the last three maximal isometric contractions completed during recovery testing, across test days (i.e., fatigue conditions). A trend analysis using orthogonal polynomials was again used to determine the linear and quadratic trends for each of the first order interactions.

CHAPTER 4: RESULTS

4.1 Participant Characteristics

The participants' (n=14) physical characteristics, including means and standard deviations, are presented in Table 4. Skin temperature was recorded from the location of the TA electrodes immediately prior to each session following preparation (pre-temperature) and following the completion of recovery during clean up (post-temperature). Temperature significantly ($p < 0.05$) increased an average of 0.7°C from beginning to end of a session.

4.2 Data Screening

All data was screened for outliers for each variable from the 4 baseline contractions performed on each test day. Histograms were plotted for each variable. Outliers were determined to be variables located outside of 3 standard deviations from the variable mean. There were no outliers for mean force, TA RMS, TA MPF or soleus RMS. There was 1 outlier for soleus MPF with the mean of baseline contractions on day 1 falling just below 3 standard deviations.

Of the original 17 participants two did not fully complete the fatigue protocol and were therefore excluded. A third participant was excluded due to equipment failure during one session leaving a total of 14 participants. Of these 14, two participants exhibited only plantar flexion, as opposed to dorsiflexion, during the evoked twitches and therefore their twitch data was excluded. Therefore, for all voluntary contraction measures (mean force, sEMG RMS, and sEMG MPF) the data from 14 participants were

used. For all twitch measures (peak force, contraction time, half relaxation time, and rate of force development) the data from 12 participants was used.

The EDL data was excluded from the analysis for the sake of simplicity in reporting the results. The sEMG RMS amplitude and MPF of the EDL were very similar to that of the TA and provided no additional insight to explaining the results for dorsiflexion force.

Table 4. Means (M) and standard deviations (SD) for the physical characteristics of the present study's participants.

Physical Characteristic	M \pm SD
Age (years)	22.14 \pm 2.18
Height (cm)	180.71 \pm 7.61
Weight (kg)	79.08 \pm 12.55
Calf Circumference (cm)	38.43 \pm 3.28
Leg Length (cm)	38.14 \pm 2.13
Foot Length (cm)	25.93 \pm 1.54
Forefoot Length (cm)	15.14 \pm 1.05
Skin Pre-temperature ($^{\circ}$ C)	29.93 \pm 0.73
Skin Post-temperature ($^{\circ}$ C)	30.61 \pm 0.93

4.3 Statistical Assumptions

Prior to completing a repeated measures ANOVA the statistical assumptions associated with it were examined. Violation of any of the following four assumptions will

result in a lower estimate of reliability (Kroll, 1962). A nested ANOVA was used to assess the reliability and consistency of all measures across the initial trials on each test day.

The first assumption is that all data from each measure follows a normal distribution. To be considered normally distributed the data from each measure on each day should exhibit skewness values less than 2 and kurtosis values less than 3. All data fell within the upper limit for skewness however, there were two exceptions exceeding the limit for kurtosis. The sEMG RMS of the soleus (6.1), as well as the MPF of the soleus (4.7) were greater than 3 for kurtosis on day 1. However, Glass, Peckhman, and Sanders (1972) have indicated that the ANOVA results are robust to violations of normality, even with kurtosis values greater than that observed in the present study.

The second and third assumptions are equal population sizes (balanced cells) and independence of the observations, respectively. All participants completed both days of the procedure supplying equal population sizes across the two conditions (days), therefore this assumption is met. In contrast, since each participant completed both days the individual observations are not independent but correlated. This assumption is therefore replaced by the requirement for equal variances or homogeneity of variance across days (Keppel, 1973). To assess this assumption an F_{max} test can be used using the standard deviation (σ) from each day (j) (Kirk, 1995). The equation is as follows:

$$F_{max} = \frac{\sigma_{j \text{ largest}}^2}{\sigma_{j \text{ smallest}}^2}$$

The F_{max} critical value are given based on degrees of freedom p and $n-1$, where p is the number of variances (2 days) and n is the number of observations within each

treatment (4 trials). For degrees of freedom 2 and 3, at an alpha level of 0.05 the F_{\max} critical value is 15.4. To accept the hypothesis of homogeneity the F_{\max} must be lesser than the corresponding critical value. All F_{\max} values calculated from the first two days were less than 15.4 therefore the assumption of equal variances is met.

4.4 Reliability and Consistency of Measures

Four maximal voluntary contractions and the initial three twitches from each test day were used as baseline trials to determine the reliability and consistency of measures. These data were obtained prior to the administration of the fatigue protocols. Recall that the intraclass correlation coefficient (ICC) is a measure of consistency, i.e., how consistent a subject is at reproducing their own score. Stability was then assessed using a complementary ANOVA model to determine if the group means remained unchanged. Both stability and consistency are part of reliability analysis.

4.4.1 Force

Mean force across the two days showed a significant ($p < 0.05$) increase from 246.19 ± 61.35 N on day 1 to 256.65 ± 57.06 N on day 2 (Table 5). Although statistically significant the increase of 4.3% may be deemed trivial (Mcintosh & Gabriel, 2012). The ICC for mean force was 0.97 suggesting that this is a highly consistent measure.

4.4.2 Surface Electromyographic Activity

There was a significant ($p < 0.01$) increase in TA sEMG root-mean-square (RMS) amplitude from 318.89 ± 191.4 μ V on day 1 to 357.29 ± 205.5 μ V on day 2, amounting to 12% (Table 5). The resulting ICC was 0.80 suggesting good consistency (Merletti et al., 1995). Thus, although there was a slight change in TA sEMG RMS across days, the

consistency of scores offset the decreased stability. Similarly, there was also a slight increase (3.1%; $p < 0.05$) in TA mean power frequency (MPF) from 128.32 ± 23.5 Hz on day 1 to 132.35 ± 26.3 Hz on day 2. The ICC was 0.93, indicating excellent consistency (Merletti et al., 1995).

The sEMG RMS for the soleus was not statistically significant ($p > 0.05$) across the two days, indicating that the means were stable across days. However, the ICC value of 0.67 was lower than expected. Further inspection of the raw scores revealed that the data was highly kurtotic (6.1), indicating that the observations were within a very narrow range. Thus, the scores were more homogeneous for this measure, which is known to artificially deflate the ICC in the same manner that a limited range of scores decreases the Pearson correlation coefficient. Homogeneous scores is also evident in a lower true score variance compared to the other measures (Bilodeau et al., 1994). Based on these diagnostics, the ICC value for TA sEMG RMS is deemed to be good for inclusion in a discussion of the results.

There was an increase (5.3%; $p > 0.05$) in soleus MPF from 68.90 ± 20.6 Hz on day 1 to 72.54 ± 9.7 Hz on day 2. The majority of the error in this measure was associated with the day-to-day variance (68%), followed the trial-to-trial variance (36.2%), resulting in a negative true score variance (-4.1). The latter of which is theoretically impossible and tends to occur when the number of subjects is low and the range of scores is limited (Carlson & Kroll, 1970). While the means were different across the two test sessions, scatter plots of the data also revealed a narrow range of scores on one day versus a broad range of scores on another, indicating a measurement

problem. As a result, this measure is unsuitable for inclusion in a discussion of the results.

4.4.3 Twitch Contraction Measures

Reliability analysis was performed on peak force (PF), force contraction time (CT), force half relaxation time (HRT), and rate of force development (RFD) for the initial three evoked twitch contractions across the two test days (Table 6). There was an 11% decrease ($p < 0.01$) in PF from 25.20 ± 2.53 N on day1 to 22.41 ± 1.83 N on day 2. Though statically significant, the magnitude of this change was minor and was compensated for by excellent consistency as reflected by an ICC value of 0.93. The CT and RFD also exhibited small but statistically significant (p 's < 0.01) decreases of 3.8 and 7.2%, respectively. There was no significant ($p > 0.05$) change in HRT across the two days. The consistency of these measures ranged from good to excellent with ICC values of 0.69 for CT, 0.89 for HRT, 0.94 for RFD (Merletti et al., 1995). The twitch contraction measures are, therefore, all deemed to have good reliability.

Table 5. Analysis of variance of baseline contractions for reliability and consistency of measures.

	Force (N)	TA RMS (μ V)	TA MPF (Hz)	SOL RMS (μ V)	SOL MPF (Hz)
Test Day	M \pm SD	M \pm SD	M \pm SD	M \pm SD	M \pm SD
1	246.19 \pm 8.20	318.89 \pm 191.4	128.32 \pm 23.5	16.02 \pm 6.8	68.90 \pm 20.6
2	256.65 \pm 7.62	357.29 \pm 205.5	132.35 \pm 26.3	15.05 \pm 7.7	72.54 \pm 9.7
Difference of Means (Percent Change)	10.46** (4.3%)	38.40** (12.0%)	4.03* (3.1%)	-0.97 (6.1%)	3.64 (5.3%)
($\sigma_{e_1}^2 - Trials$)	325.6 (8.8%)	0.003 (6.9%)	80.5 (12.3%)	12.6 (23.1%)	95.1 (36.2%)
($\sigma_{e_2}^2 - Days$)	128.3 (3.5%)	0.013 (30.3%)	57.8 (8.8%)	19.3 (35.3%)	178.8 (68.0%)
($\sigma_{true}^2 - True$)	3261.2 (87.8%)	0.026 (62.8%)	516.2 (78.9%)	22.7 (41.6%)	-10.8 (-4.1%)
Grand Mean	251.42 N	338.1 μ V	130.33 Hz	15.54 μ V	70.72 Hz
SEM	18.04 N	53.49 μ V	8.97 Hz	3.55 μ V	9.75 Hz
R	0.97	0.80	0.93	0.67	-0.12

Significant difference between days, * = $p < 0.05$, ** = $p < 0.01$.

Table 6. Analysis of variance of baseline twitches for reliability and consistency of twitch measures.

	Peak Force (N)	Contraction Time (ms)	Half Relaxation Time (ms)	Rate of Force Development (N/s)
Test Day	M ± SD	M ± SD	M ± SD	M ± SD
1	25.20 ± 2.53	8.58 ± 0.18	8.08 ± 0.36	511.64 ± 280.04
2	22.41 ± 1.83	8.25 ± 0.15	7.81 ± 0.39	474.76 ± 206.10
Difference of Means	-2.79**	-0.33	-0.27	-36.88
(Percent Change)	(11.1 %)	(3.8%)	(3.3%)	(7.2%)
($\sigma_{e_1}^2 - Trials$)	1.35 (0.7%)	4.02 (4.0%)	45.6 (8.6%)	379.14 (0.6%)
($\sigma_{e_2}^2 - Days$)	25.1 (13.5%)	44.7 (44.5%)	83.5 (15.7%)	7029.7 (10.9%)
($\sigma_{true}^2 - True$)	160.1 (85.8%)	51.6 (51.4%)	403.7 (75.8%)	56728.1 (88.4%)
Grand Mean	23.8 N	8.4 ms	7.94 ms	493.2 N/s
SEM	1.16 N	0.20 ms	0.68 ms	19.5 N/s
R	0.93	0.69	0.89	0.94

4.5 Fatigue and Recovery of Voluntary Contractions

There were two conditions, 10% (condition 1) and 20% (condition 2) fatigue. Within each fatigue condition, there were three blocks of three trials: the first three trials of the fatigue series, the last three trials of the fatigue series, and the last three trials of the contractions used to assess recovery. The statistical model was a $2 \times 3 \times 3$ (Conditions \times Blocks \times Trials) repeated measured ANOVA, to determine if there was a significant difference in the level of fatigue and if over-recovery was present in relation to the level of fatigue. Thus, the main focus of the analysis was Conditions \times Blocks interaction term. However, the other first order interaction terms (Conditions \times Trials and Blocks \times Trials) will be elaborated on if a significant effect was present. In the case of the present study the second order interaction (Conditions \times Blocks \times Trials) was non-significant for all measures. Orthogonal polynomials were then used for trend analysis of the means.

4.5.1 Force

There was a significant difference in force (10.5 N) between conditions ($F_{(1,13)} = 11.45$, $p < 0.01$) when collapsed across blocks and trials. This main effect was produced by a slight offset that existed between conditions at Block 1, and greater level fatigue required for Condition 2 at Block 2 (see Figure 22). Collapsed across conditions, there was a significant difference in blocks ($F_{(2,26)} = 54.31$, $p < 0.01$) that followed a quadratic trend accounting for 99.99% of the variance ($p < 0.01$). There was a decrease in force from pre-fatigue to post-fatigue followed by a significant increase in force during recovery regardless of condition. As expected for serial contractions, the overall main effect for trials was significant ($F_{(2,26)} = 18.23$, $p < 0.01$). Inspection of the means in Figure 22 reveals a significant Conditions \times Blocks interaction term ($F_{(2,26)} = 5.303$, $p <$

0.05). The two conditions therefore differed with respect the quadratic trend component in means across blocks, which accounted for 96.5% of the variance. The average amount of fatigue from Block 1 to Block 2 in Condition 1 (10% condition) was 14.5%, which was followed by an average recovery to 100.5% of pre-fatigue force level at Block 3. In contrast, the participants exhibited an average strength decrement of 19.6% between Blocks 1 and 2 in Condition 2 (20% condition), with an average recovery to 99.5% of at Block 3. The return of force levels to pre-fatigue values (99.5 and 100.5%) includes 16 of the 28 protocols showing over-recovery ranging from 101-115% and 12 of the 28 protocols showing prolonged force decrements ranging from 80-99% pre-fatigue values (see Figures 23 and 24).

The second-order interaction terms for Trials were significant but are relatively unimportant comparisons. For example, the Conditions \times Trials interaction term ($F_{(2,26)} = 3.83, p < 0.05$) indicated that serial isometric contractions associated with a greater level of fatigue exhibited a more pronounced decline from one trial to the next, as occurred in Condition 1 versus Condition 2 (see Figure 25). Likewise it is not surprising that the pattern of trials across blocks are different, resulting in a significant Blocks \times Trials interaction term ($F_{(4,52)} = 5.51, p < 0.01$). Block 1 represents the first three trials of the fatigue pattern, so there is a progressive decline in force that was more pronounced (see Figure 26). It is common to observe a plateau in trial scores towards the end of a fatigue series as seen in Block 2. Similarly, Block 3 has the expected plateau in trial scores towards end of recovery period. Nevertheless, the pattern of means for trials within blocks, collapsed across conditions illustrates why the linear trend component for the main Trials effect accounted 91% of the variance.

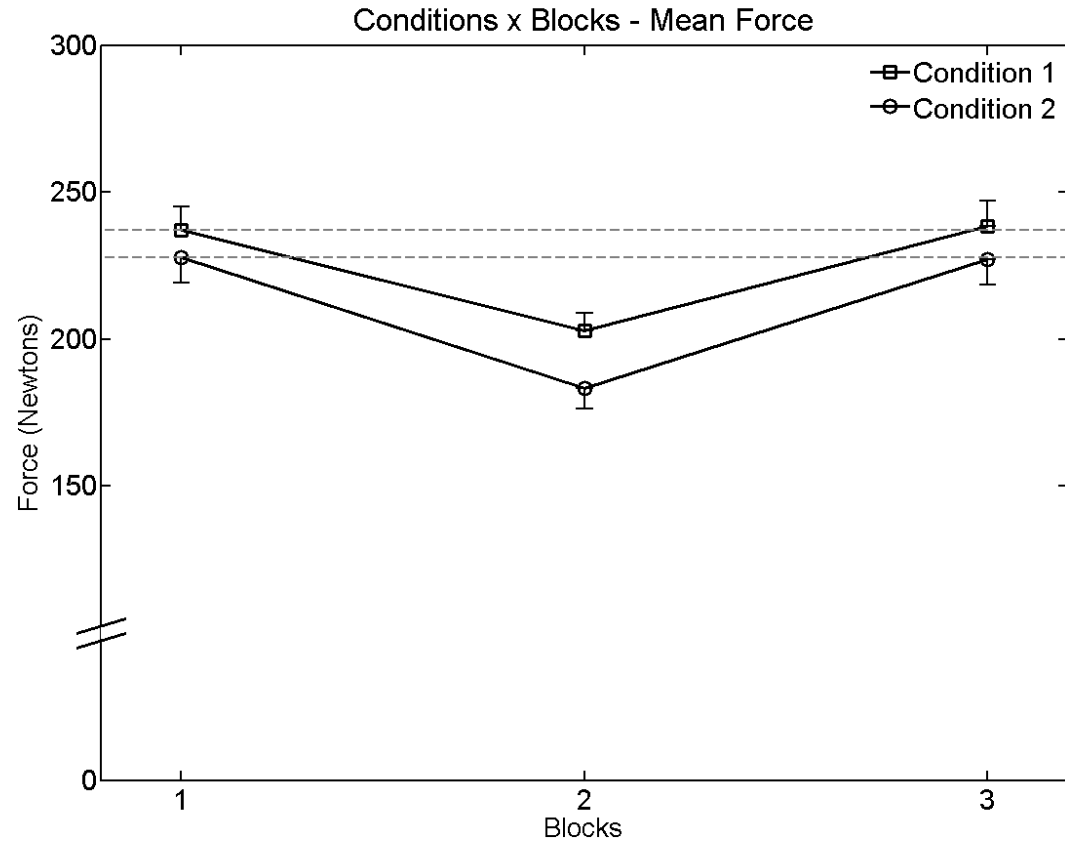


Figure 22. Means (squares for condition 1 and circles for condition 2) and standard errors (vertical bars) for the Conditions \times Blocks interaction for mean force. The dotted lines represent the baseline force level for condition 1 (upper line) and condition 2 (lower line).

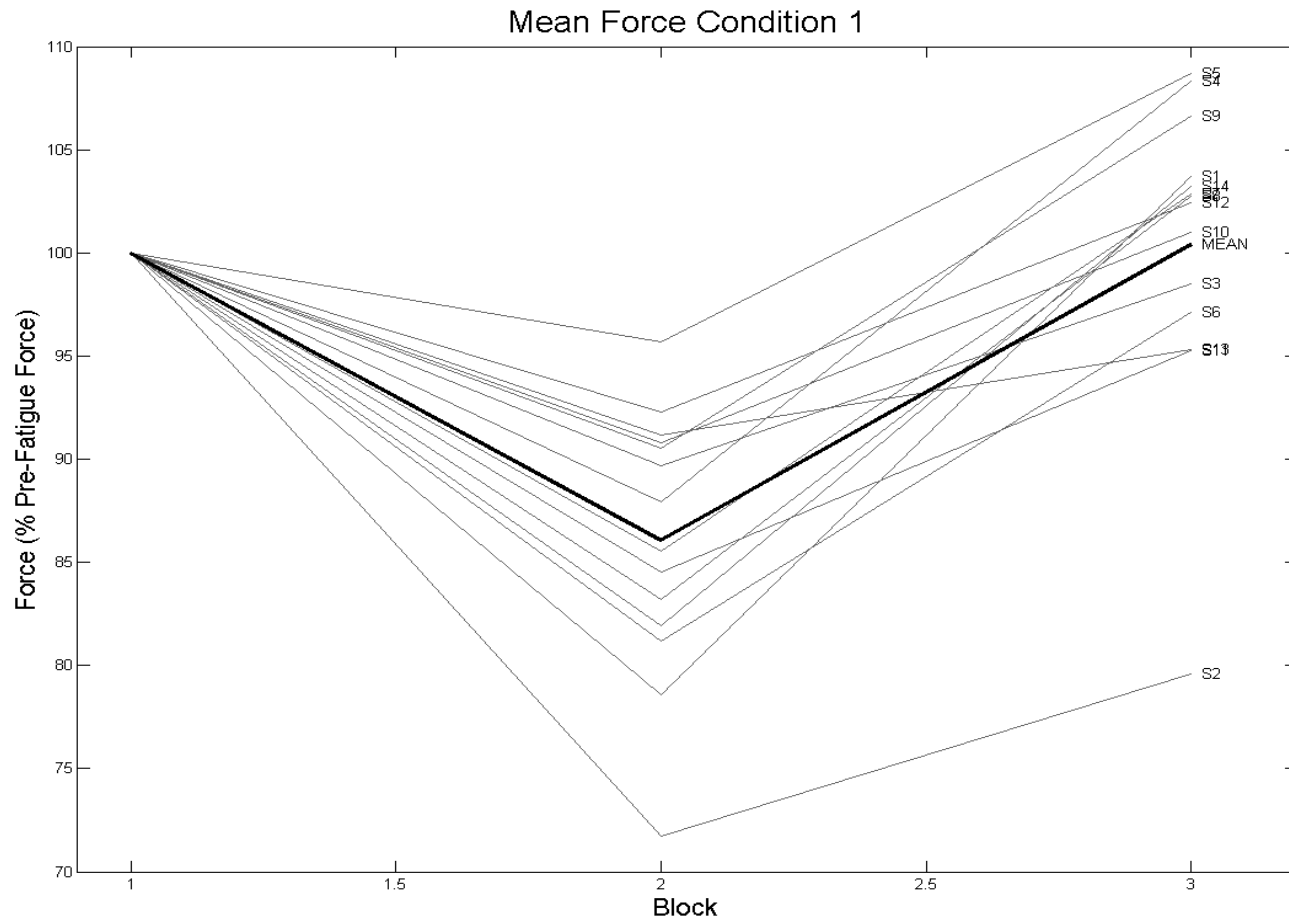


Figure 23. Individual force traces (N=14) for condition 1 (10% force decrement) mean force relative to participants' initial levels.

Mean of participants shown as darker line.

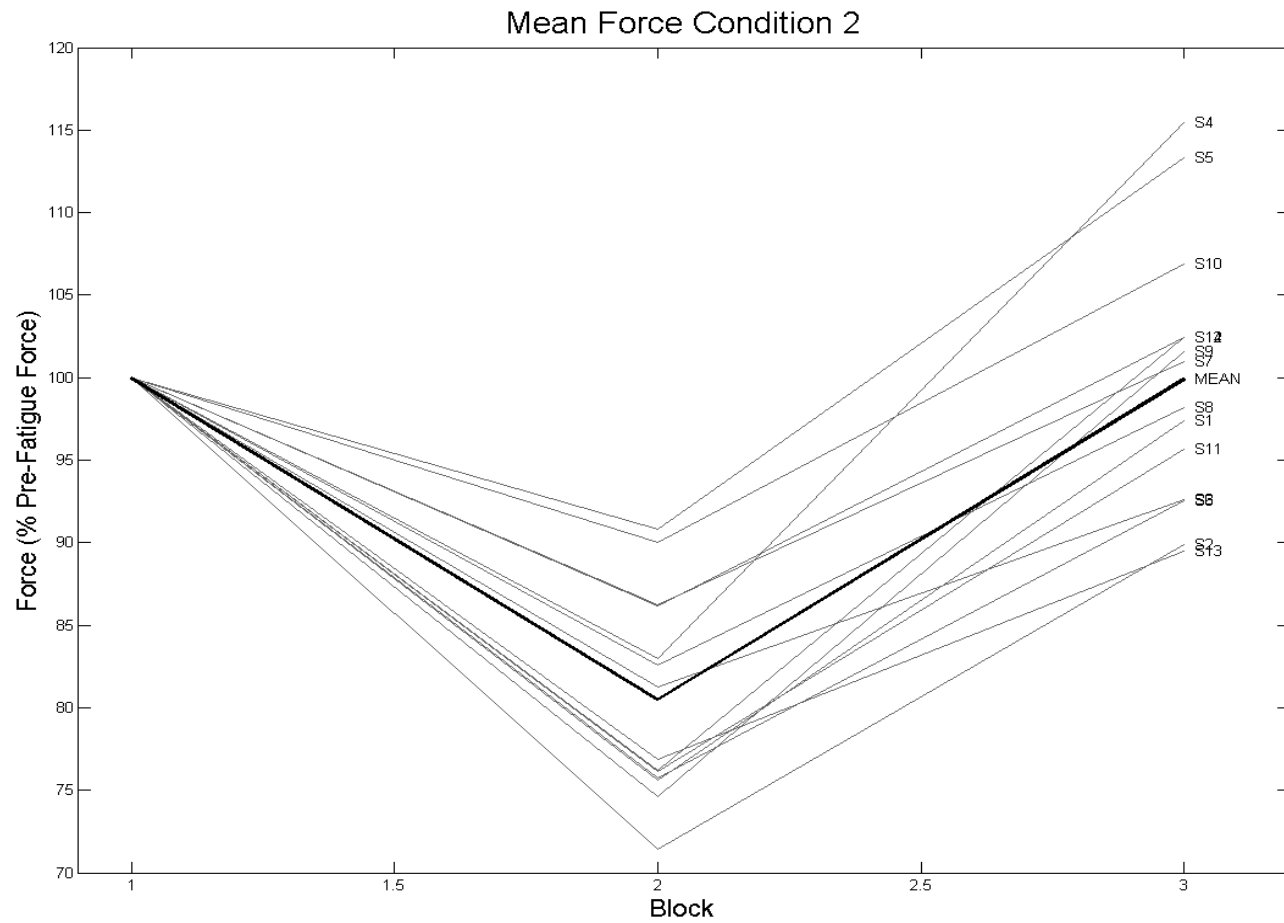


Figure 24. Individual force traces (N=14) for condition 2 (20% force decrement) mean force relative to participants' initial levels.

Mean of participants shown as darker line.

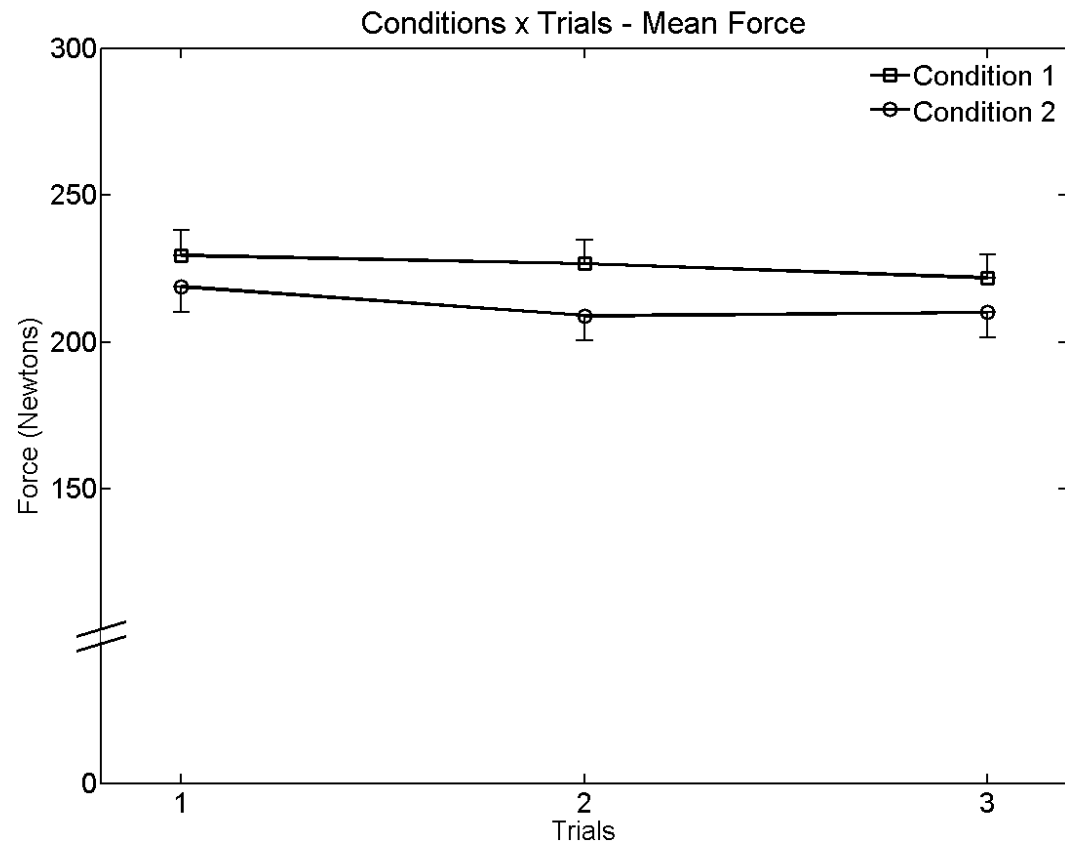


Figure 25. Means (squares for condition 1 and circles for condition 2) and standard errors (vertical bars) for the Conditions \times Trials interaction for mean force.

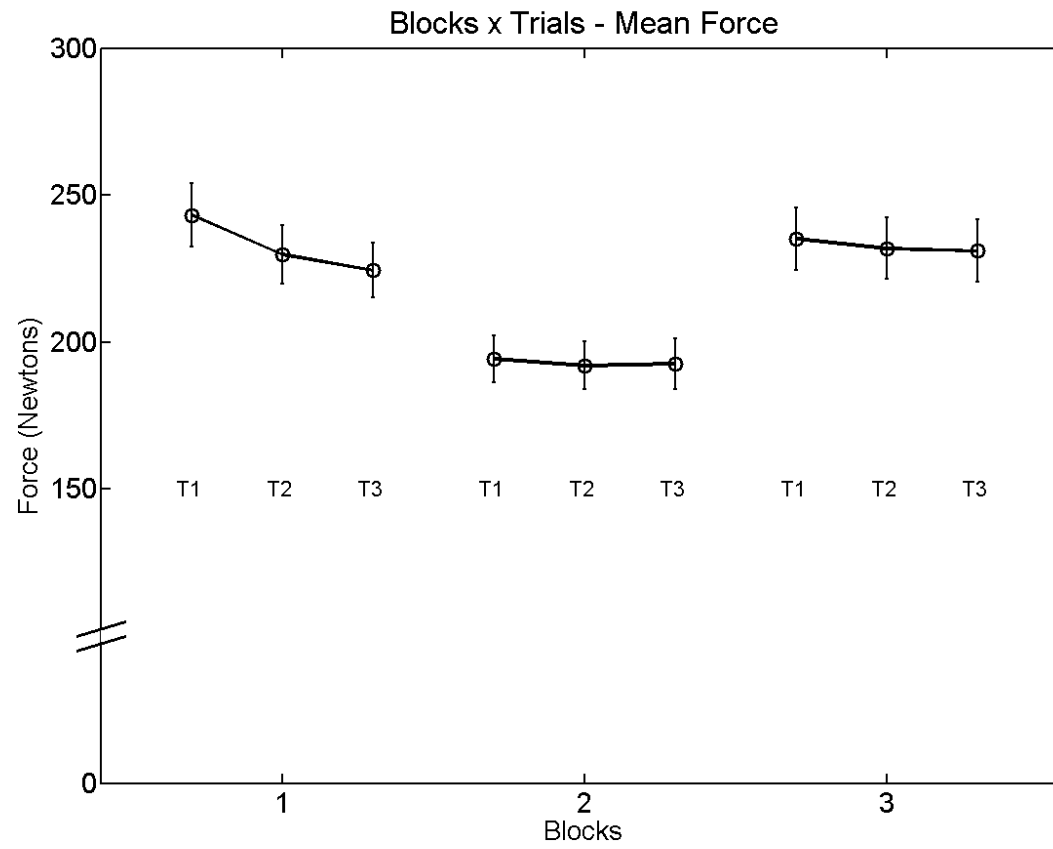


Figure 26. Means (circles) and standard errors (vertical bars) for the Blocks \times Trials interaction for mean force.

4.5.2 *Tibialis Anterior sEMG Root-Mean-Square Amplitude*

The tibialis anterior (TA) surface electromyographic (sEMG) root-mean-square (RMS) amplitude had a significant conditions main effect ($F_{(1,13)} = 4.89, p < 0.05$). Figure 27 illustrates that the mean sEMG RMS amplitude for Condition 1 (0.21 ± 0.06 mV) was greater than that for Condition 2 (0.19 ± 0.08 mV), which is consistent with the mean force results presented above. The experimental design required less fatigue for Condition 1 with the expectation that there would be enhanced recovery in comparison to Condition 2. These effects would therefore manifest themselves in an overall greater magnitude of TA sEMG activity.

The TA sEMG RMS amplitude had a significant Blocks main effect ($F_{(2,26)} = 18.53, p < 0.01$) with significant linear ($p < 0.01$) and quadratic ($p < 0.01$) components (see Figure 28). The linear component accounted for 23.7% of variance; there was a significant decrease in TA sEMG RMS amplitude from Block 1 (pre-fatigue) to Block 3 (recovery) amounting to 0.019 mV. Thus, TA muscle electrical activity did not fully recover to pre-fatigue levels. The quadratic component accounted for 74.6% in means across blocks; there was a decrease in sEMG RMS amplitude from Block 1 (pre-fatigue) to Block 2 (fatigue) of 0.038 mV (17.2%). The reduction in sEMG is consistent with the amount of fatigue as measured by mean force which, averaged across conditions, was 17.1% fatigue. Tibialis anterior sEMG RMS amplitude then increased 0.019 mV (8.6%) from Block 2 (fatigue) to Block 3 (recovery), returning to only 91.4% of the initial level, completing the quadratic component. There were no significant main effects for Trials or any of the interaction terms (p 's > 0.05).

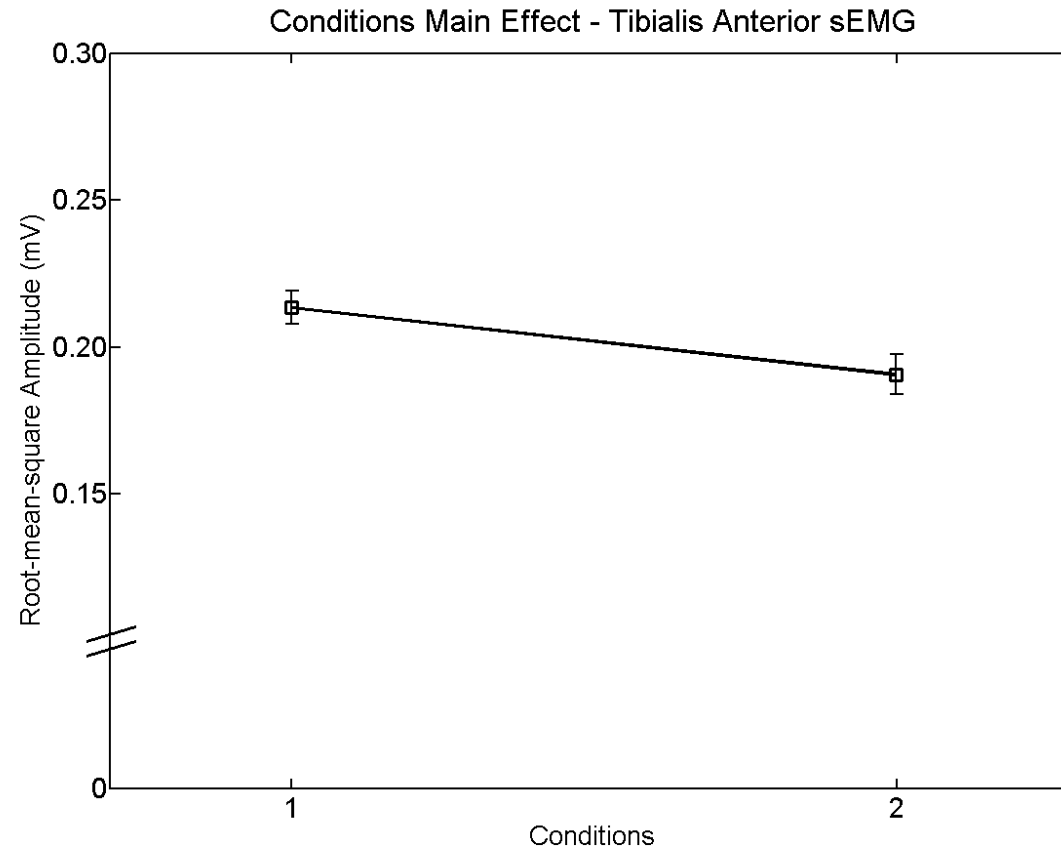


Figure 27. Means (squares) and standard errors (vertical bars) for the Conditions main effect for TA sEMG RMS amplitude.

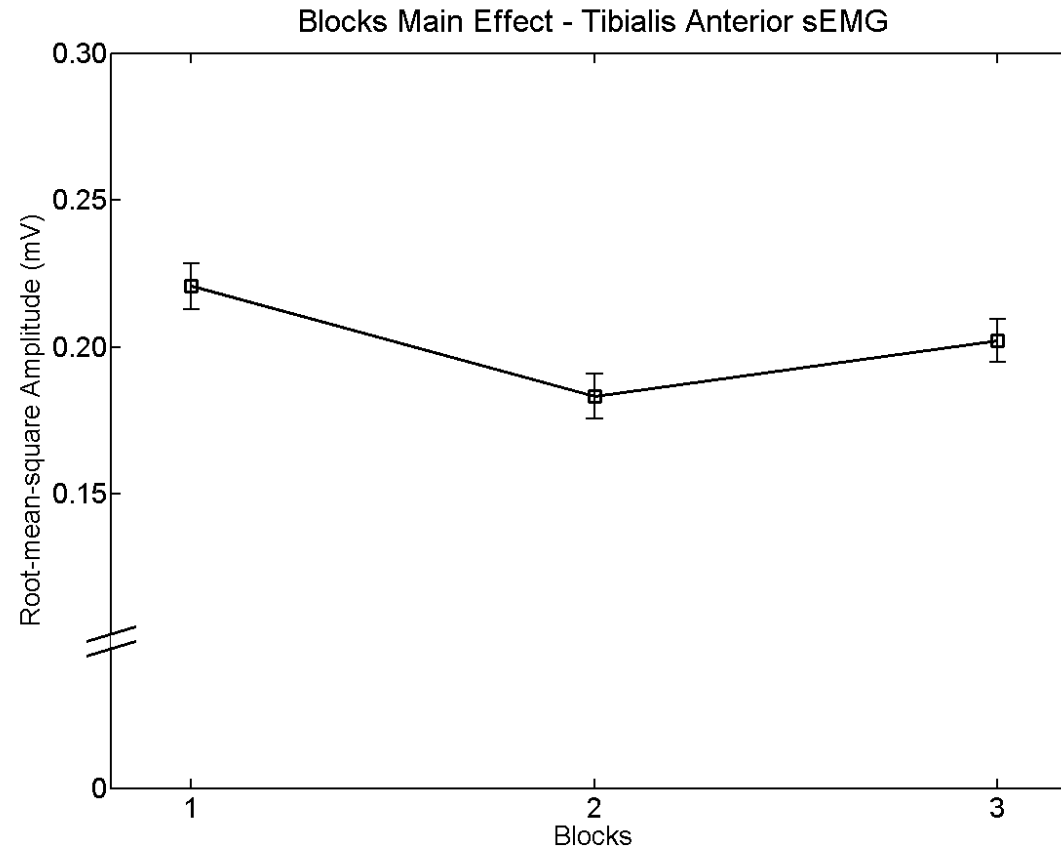


Figure 28. Means (squares) and standard errors (vertical bars) for the Blocks main effect for TA sEMG RMS amplitude.

4.5.3 *Tibialis Anterior sEMG Mean Power Frequency*

The Blocks main effect was significant ($F_{(2,26)} = 46.26, p < 0.01$) as the means followed ($p < 0.01$) linear and quadratic ($p < 0.01$) trend components (see Figure 29). The linear component accounted for 56.5% of variance in means. Tibialis anterior mean power frequency (MPF) increased 18.02 Hz (14.3%) from Block 1 to Block 3. The quadratic component accounted for 43.5% of the variance in TA MPF means. Tibialis MPF decreased 4.68 Hz (3.7%) from Block 1 to Block 2 prior to a 22.7 Hz (18%) increase between Blocks 2 and 3. The Blocks \times Trials interaction term was significant ($F_{(4,52)} = 2.71, p < 0.05$), with a significant ($p < 0.05$) linear component accounting for 44.8% of the variance in means. As illustrated in Figure 30, the linear slopes of the three blocks of trials are different: Block 1 has the steepest negative slope, followed by a relatively stable set of trials for Block 2, and finally a slight negative slope across the three trials in Block 3. There were no other significant main effects or interaction terms (p 's > 0.05).

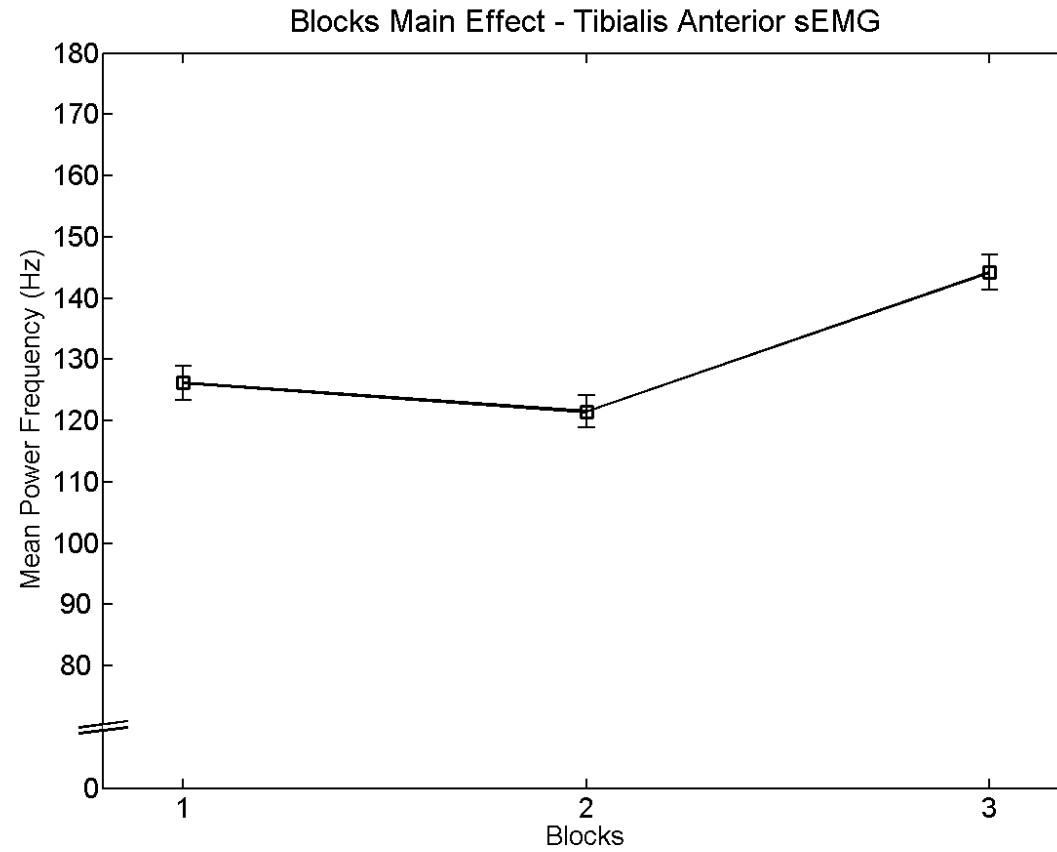


Figure 29. Means (squares) and standard errors (vertical bars) for the Blocks main effect for TA mean power frequency.

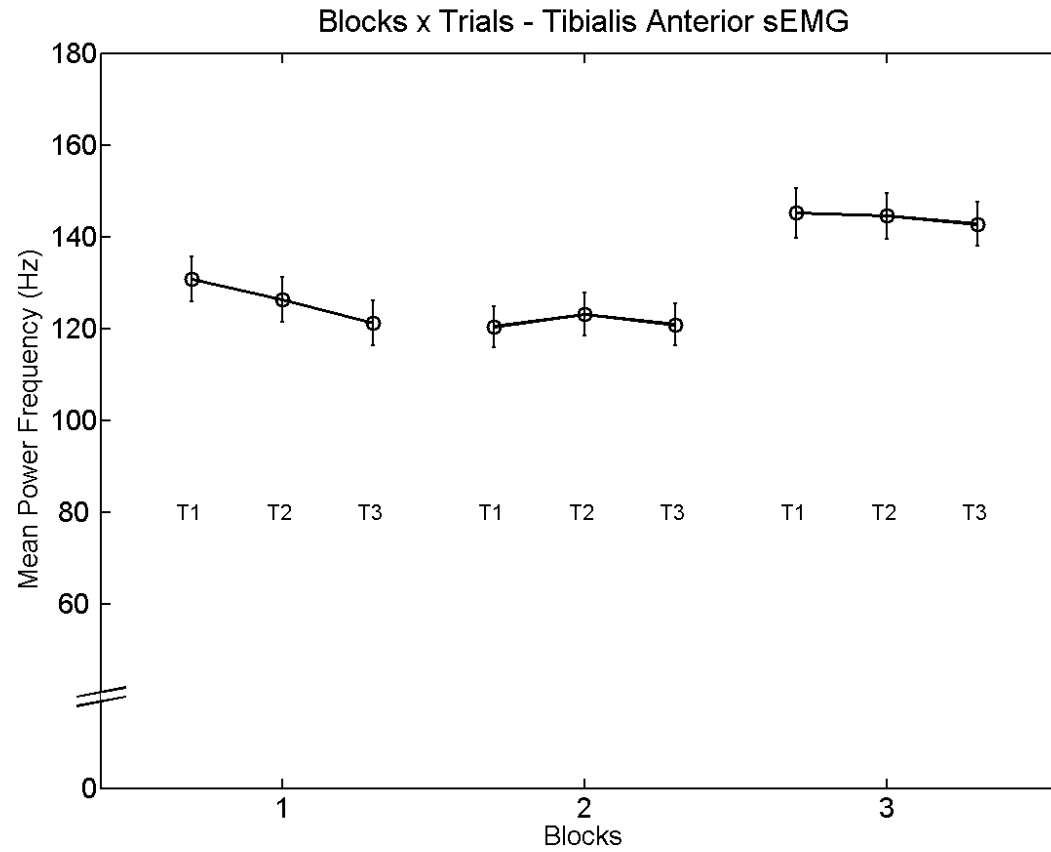


Figure 30. Means (circles) and standard error (vertical bars) for the Blocks \times Trials interaction for TA mean power frequency.

4.5.4 Soleus sEMG Root-Mean-Square Amplitude

It is obvious from an inspection of Figure 31 that there was a significant Blocks main effect ($F_{(2,26)} = 8.49, p < 0.01$), and that the pattern of means followed a linear decrement. The resulting linear trend component accounted for 89.3% of the variance in means ($p < 0.05$). Averaged across conditions, there was a 2.28 μV (15.7%) reduction in soleus sEMG amplitude across the three blocks. Figure 32 shows that pattern of decrement across blocks was different between the two conditions, resulting in a significant Conditions \times Blocks interaction term ($F_{(2,26)} = 4.15, p < 0.05$). The 2.93 μV (19.2%) decrease from Block 1 to 3 in Condition 1 was significantly greater than the 1.65 μV (12%) decrease in Condition 2. There were no other significant main effects or interaction terms (p 's > 0.05).

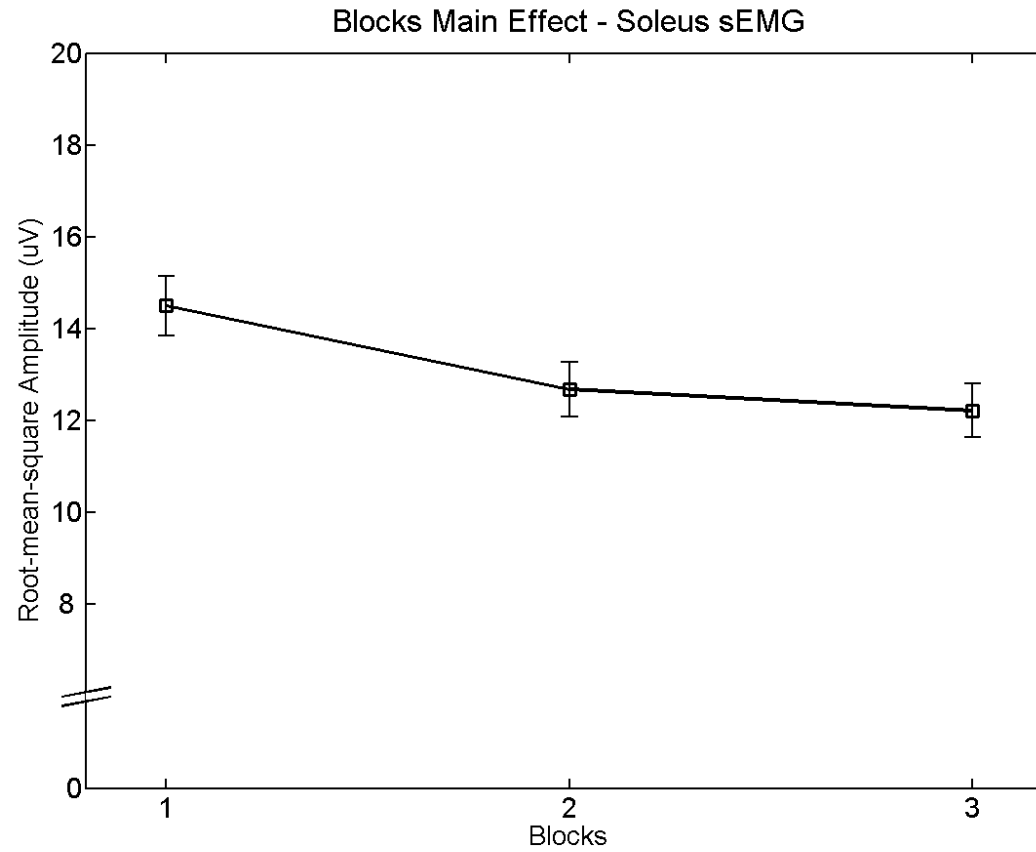


Figure 31. Means (squares) and standard errors (vertical bars) for the Blocks main effect for soleus sEMG RMS amplitude, in microvolts (μV).

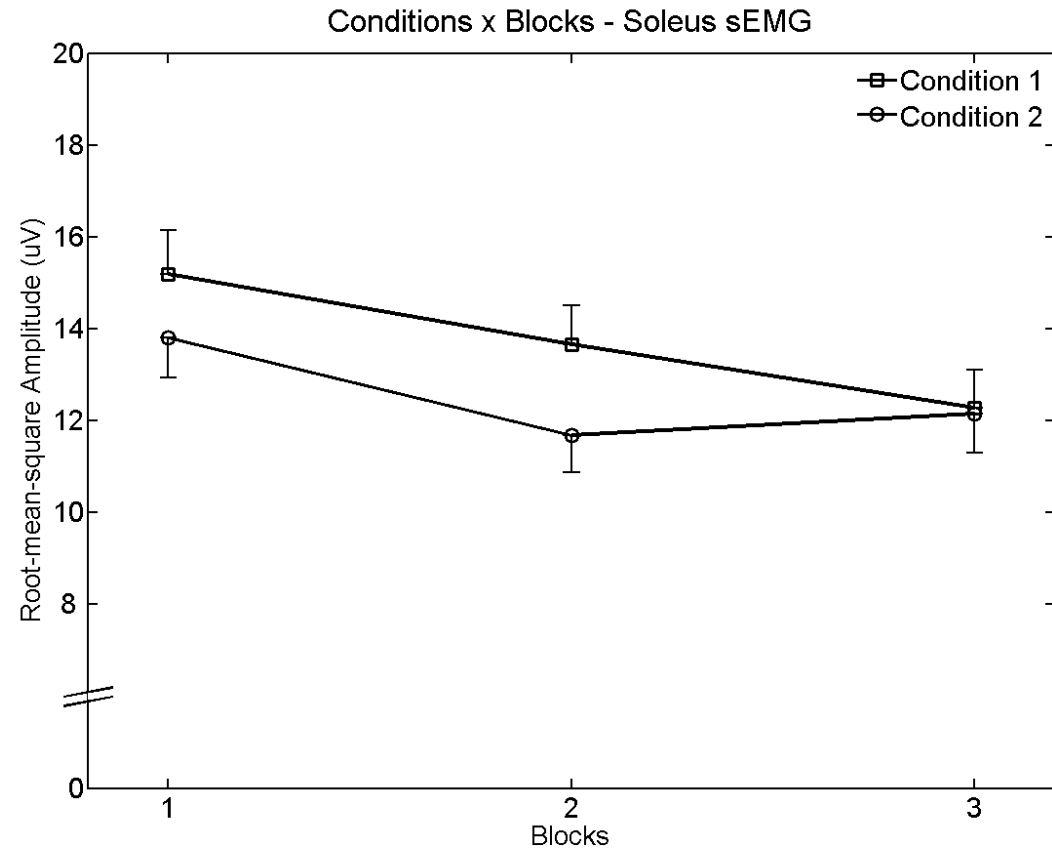


Figure 32. Means (squares for condition 1 and circles for condition 2) and standard errors (vertical bars) for the Conditions \times Blocks interaction for soleus sEMG RMS amplitude, in microvolts (μV).

4.5.5 Co-activation Ratio

The co-activation ratio was calculated by dividing the TA RMS amplitude by the soleus RMS amplitude for a measure of the coordination between the two opposing muscles. An increase in the ratio means that there was an increase in TA RMS amplitude or a decrease in soleus RMS amplitude relative to initial measures and each other. Only the Blocks main effect was significant ($F_{(2,26)} = 10.72, p < 0.01$). There was a significant ($p < 0.01$) linear component accounting for 27.2% of the variance and a significant ($p < 0.01$) quadratic component accounting for 55.24% of the variance (see Figure 33). The Block 1 (pre-fatigue) ratio was 17.43 which decreased slightly during fatigue to 16.16 in Block 2 (post-fatigue) and then increased in Block 3 (recovery) to 19.16. The large change between Block 1 and 3 accounted for the significant linear component while the slight decrease occurring from Block 1 to Block 2 followed by a large increase to Block 3 accounted for the quadratic component. The increase in the co-activation ratio during the recovery block was due to a greater recovery of TA RMS amplitude compared to soleus RMS amplitude, although neither muscle fully recovered to pre-fatigue values.

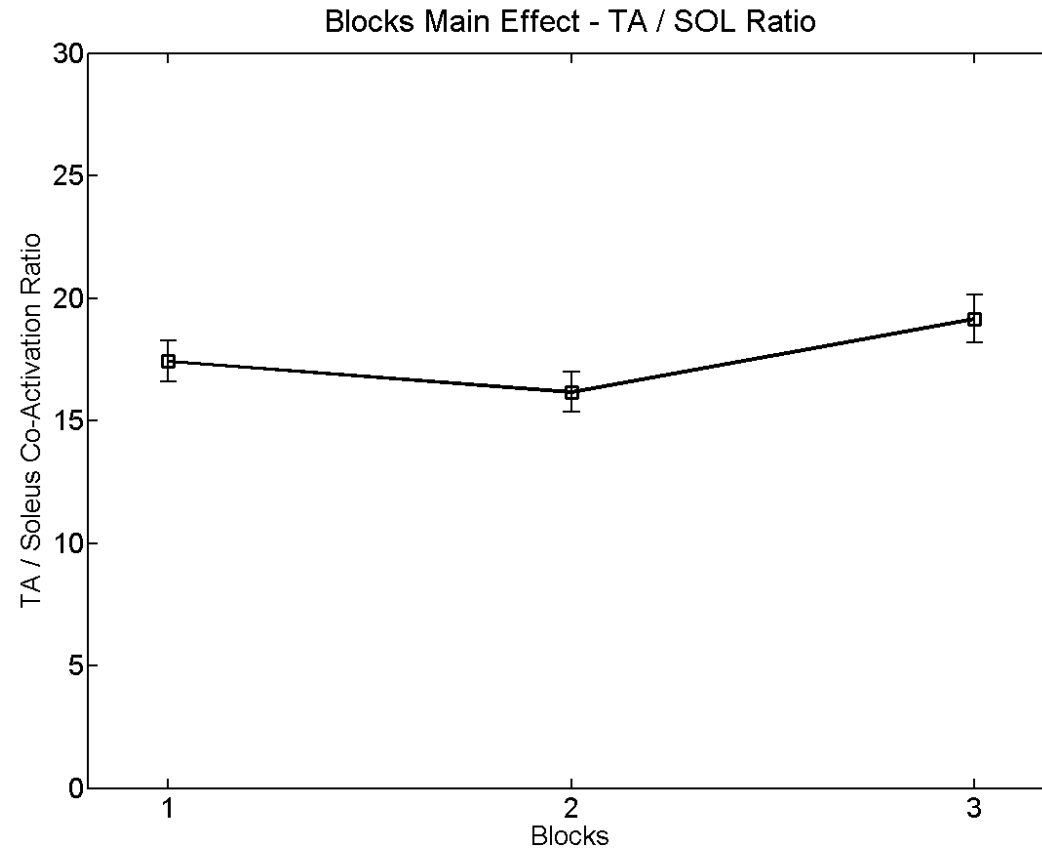


Figure 33. Means (squares) and standard errors (vertical bars) for the Blocks main effect for TA/SOL co-activation ratio.

4.6 Fatigue and Recovery of Twitches

A three-factor repeated measures ANOVA was performed similar to that of the voluntary contractions as detailed in section 4.5, but with the addition of one block. The statistical model was therefore a $2 \times 4 \times 3$ (Conditions \times Blocks \times Trials) repeated measures ANOVA. The additional block (Block 1) includes three twitches performed at the beginning of each session, to determine if there was any change in twitch properties following the four baseline voluntary contractions. The second block (Block 2) is the pre-fatigue (post-baseline contractions) twitches, the third block (Block 3) is the post-fatigue twitches, and the fourth block (Block 4) is the recovery twitches occurring prior to each voluntary contraction at minutes 6, 10, and 15 post-fatigue. Trend analysis was performed on the resulting means using orthogonal polynomials for the linear, quadratic, cubic, and quartic components. For the sake of brevity, the results will focus on peak force as all other measures extracted from the force time curve were highly correlated ($p < 0.01$) with peak force and exhibited the identical pattern of change during the fatigue and recovery protocols.

4.6.1 Peak Force

There was a significant Blocks main effect ($F_{(3,33)} = 72.72$, $p < 0.01$). Inspection of Figure 34 shows an obvious quadratic distribution of means across blocks accounting for 98.2% of the variance. Collapsed across groups, there was an increase in peak force after baseline contractions, it remained elevated (potentiated) after the fatigue series, and then returned to baseline following recovery. The Trials main effect was significant ($F_{(2,22)} = 86.51$, $p < 0.01$). The pattern of means depicted in Figure 35, collapsed across conditions and blocks exhibited a significant linear decrease of 4.4 N (13.3%) that

accounted for 98.5% of the variance ($p < 0.01$) linear and quadratic components. This to be expected as there is an exponential return towards baseline when twitches are initially potentiated.

Peak force further had a significant Conditions \times Blocks interaction term ($F_{(3,33)} = 3.82$, $p < 0.05$). Figure 36 illustrated that the two conditions were significantly different with respect to the quadratic pattern of means across blocks, which accounted 93.6% of the variance ($p < 0.05$). The interaction was present because of “minor” changes across the two conditions. There was a steeper increase in peak force from Block 1 to Block 2 in Condition 1 (13.1 N) compared to Condition 2 (10.1 N). Similarly, there was a steeper decrease in peak force from Block 3 to Block 4 in Condition 1 (13.5 N) compared to Condition 2 (10.1 N). Peak forces in Blocks 2 and 3 were comparable ($\Delta 1$ N) across the two conditions. It is again, not surprising that the Blocks \times Trials interaction was significant ($F_{(6,66)} = 30.34$, $p < 0.01$). Figure 37 shows that the pattern of means within blocks changed slope three times after Block 1, resulting in a significant cubic component accounting for 75% of the variance ($p < 0.01$).

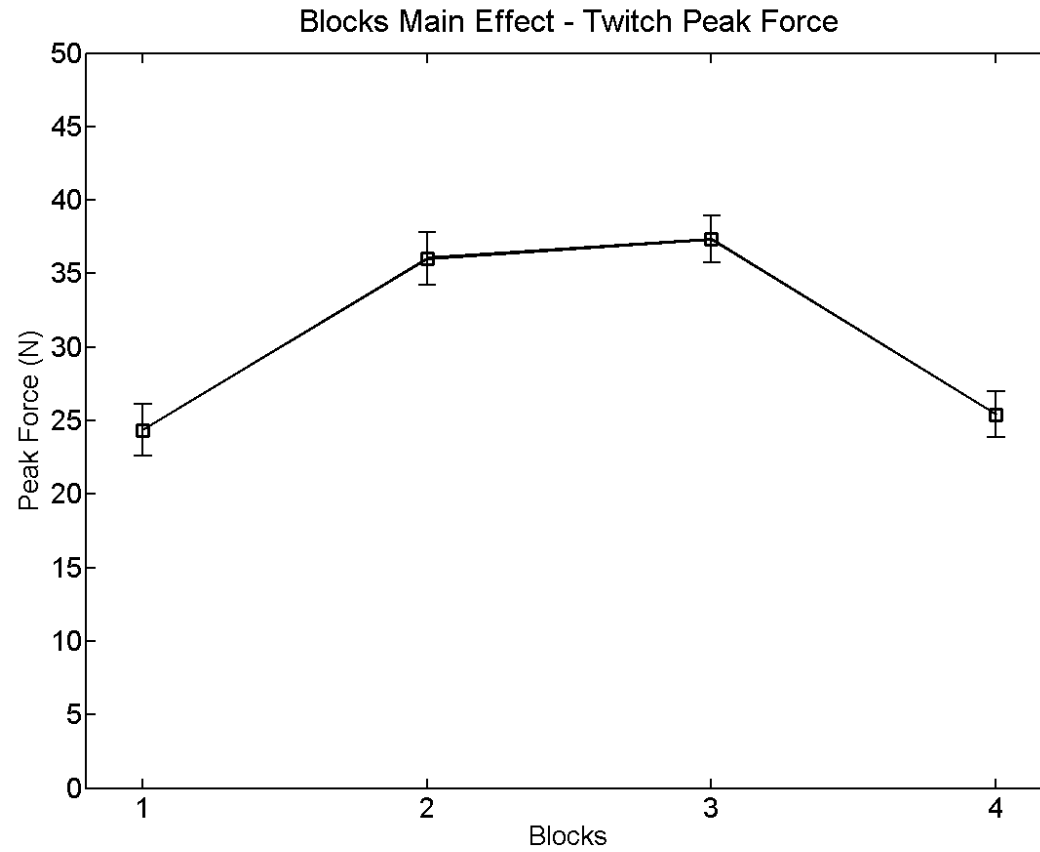


Figure 34. Means (squares) and standard errors (vertical bars) for the Blocks main effect for twitch peak force.

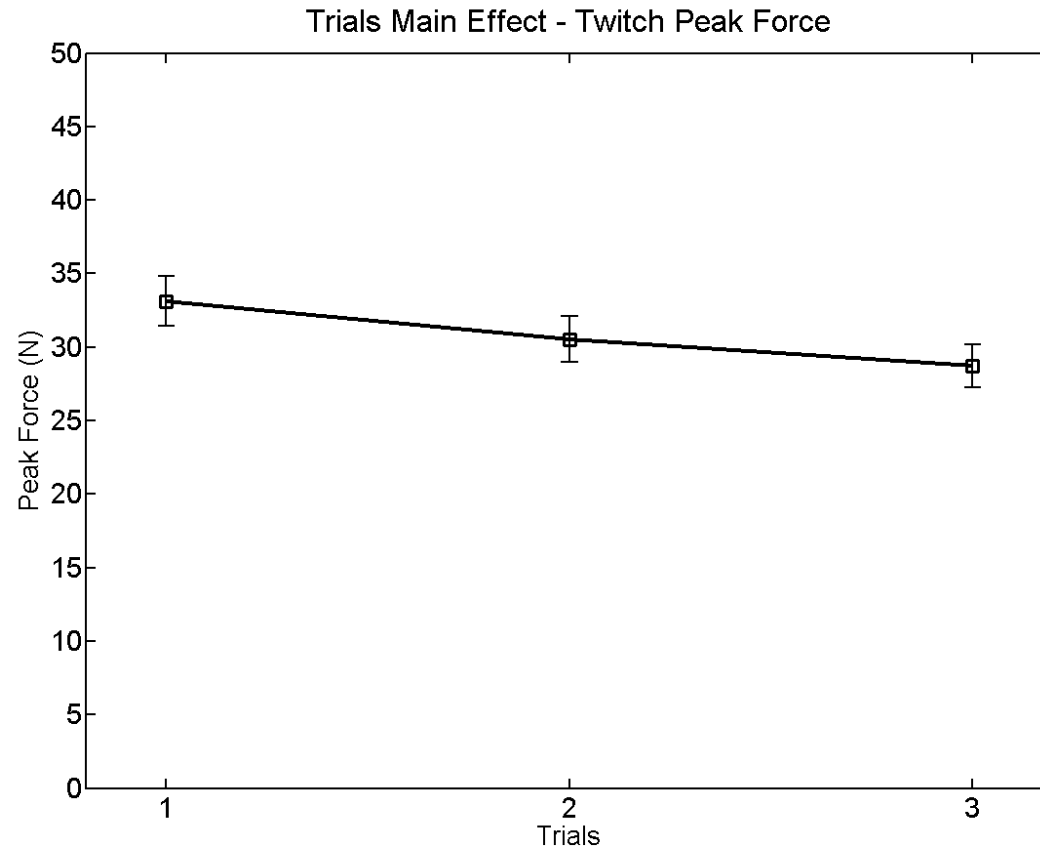


Figure 35. Means (squares) and standard errors (vertical bars) for the Trials main effect for twitch peak force.

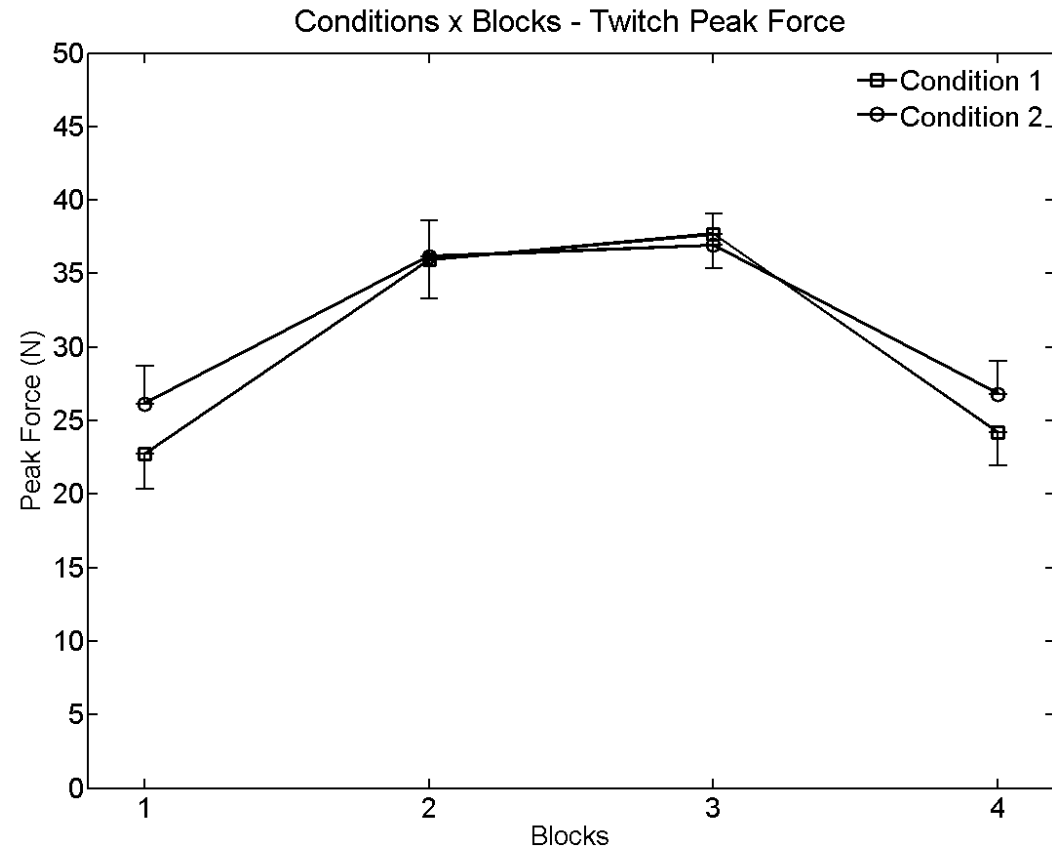


Figure 36. Means (squares for condition 1 and circles for condition 2) and standard errors (vertical bars) for the Conditions \times Blocks interaction for twitch peak force.

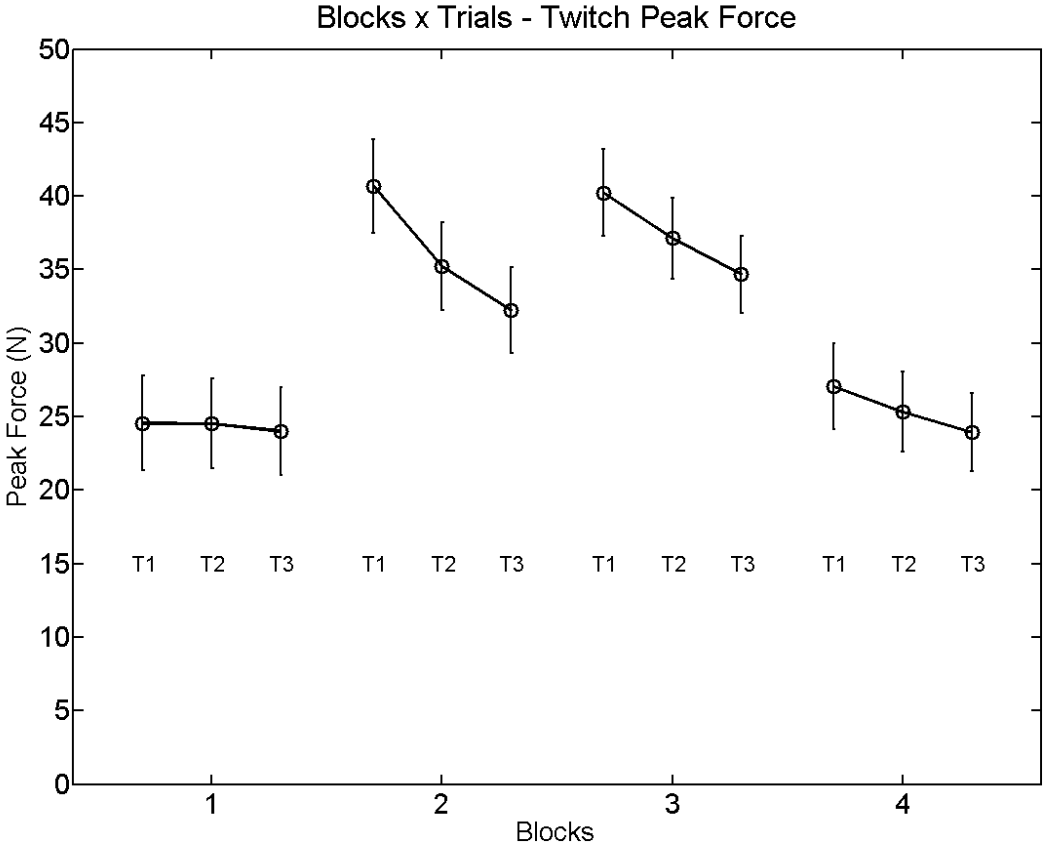


Figure 37. Means (circles) and standard errors (vertical bars) for the Blocks \times Trials interaction for twitch peak force.

CHAPTER 5: DISCUSSION

The purpose of this study was to investigate the potential mechanisms involved in the over-recovery phenomenon. Participants were administered a local muscular fatigue protocol consisting of maximal isometric dorsiflexion contractions on two separate test sessions, each corresponding to a different target level of percent force decrement. Recovery was assessed by additional contractions at regular intervals following the last contraction of the fatigue protocol. Force and surface electromyographic (sEMG) activity were monitored concurrently throughout the entire test session. It was hypothesized that over-recovery would be due to either potentiation or an alteration in neuromotor coordination control. We found that force levels returned to an average of 100.5% and 99.5% initial force levels following 10% fatigue and 20% fatigue, respectively. This means that force was not found to over-recover, but rather return to baseline levels. However, over-recovery of force was displayed by some participants following both fatigue conditions. Tibialis anterior and soleus RMS amplitude remained depressed following fatigue as opposed to MPF of the tibialis anterior, which over-recovered to a level above its initial values. The results will be discussed in greater detail in the following section.

5.1 Baseline

The following values reported are taken from the four baseline maximal voluntary contractions and the three baseline twitches. All values from the present study are reported as the grand mean \pm standard deviation. Similarly, values from the literature are reported as mean \pm standard deviation, or in some cases standard error (SE).

5.1.1 Force

Maximal isometric dorsiflexion force was 251.42 ± 18.04 N. These values are similar in magnitude to what has been previously reported for college-age males: 225.9 ± 43.5 N (McIntosh & Gabriel, 2012), 269 ± 61 N (Lenhardt et al., 2009), 251 ± 8 N (Patten & Kamen, 2000), and 262 ± 19 N (Kent-Braun & Ng, 1999). Thus, the maximal isometric dorsiflexion strength of participants in the current work is comparable to those in other studies.

5.1.2 Surface Electromyography

The RMS amplitude of the TA sEMG activity was 338.1 ± 53.5 μ V and the MPF was 130.3 ± 8.97 Hz. Previous studies in this laboratory observed similar values. Lenhardt et al., (2009) reported RMS amplitude of 524 ± 360 μ V and MPF of 115 ± 27 Hz. The RMS amplitude and MPF values obtained by McIntosh and Gabriel (2012) were 203.4 ± 95.8 μ V and 121.1 ± 28.8 Hz, respectively. Other literature consisting of maximal dorsiflexion contractions observed comparable results: McKay et al. (1996) reported a RMS amplitude of 371 ± 48 (SE) μ V while the approximated values extracted from a graph by Cioni et al. (1994) for RMS amplitude was 550 μ V and MPF was 125 Hz. The sEMG activity of the soleus in the present study was 15.54 ± 3.6 μ V for RMS amplitude and 70.7 ± 9.8 Hz for MPF. Holtermann et al. (2005) observed RMS amplitude values of 26.3 μ V for soleus co-activation during maximal dorsiflexion. Geertsen et al. (2008) reported that the soleus RMS amplitude was 6.45% of TA, which can be approximated at 18.9 μ V based on their TA RMS amplitude of 292.5 μ V. In general, the participants in the current study exhibited sEMG activity values that are similar to those reported by other investigators.

5.1.3 Twitch Measures

The means and standard deviations for the twitch measures recorded during baseline were as follows: peak twitch force was 23.8 ± 1.2 N, contraction time was 84.1 ± 2.0 ms, half relaxation time was 79.4 ± 6.8 ms, and the rate of force development was 493.2 ± 19.5 N/s. A review of related literature revealed comparable contraction and half relaxation time values. However, because peak force and the peak rate of force development are sometimes reported in N·m, comparable values were limited. Nevertheless, Inglis et al. (2011) reported a peak force of 18.4 ± 1.8 N for the evoked twitch contraction in the TA while Kent-Braun et al. (2002) observed 20.5 ± 5.2 N (mean \pm SE). Comparable values for contraction and half relaxation time have been reported by Belanger et al. (1983) and Connelly et al. (1999). Belanger et al. (1983) found a contraction time of 80.8 ± 13 ms and a half relaxation time of 93.6 ± 25 ms. Connelly et al. (1999) observed a contraction time of 98.5 ± 6.9 ms and a half relaxation time 86.5 ± 7 ms. The muscle contractile properties for the TA in participants in the current work are therefore similar to those observed in other studies.

5.2 Fatigue and Recovery

All measures taken from the voluntary contractions decreased during the fatigue protocol in both conditions. Force decreased by 14.5% and 19.6% during conditions 1 and 2, respectively. The predetermined force decrements of 10 and 20% were determined by taking a visual average of participants' baseline maximal voluntary contractions and subtracting 10 or 20% from this. The decrement of 14.5% in the 10% condition and 19.6% in the 20% condition did not match the target levels since participants were required to complete three contractions below the fatigue level to ensure it wasn't a single

failed contraction. In the 10% condition the three contractions elicited a greater amount of fatigue compared to the 20% condition where fatigue had appeared to plateau. Tibialis anterior sEMG RMS amplitude decreased during conditions 1 and 2 (16.2% and 17.7%, respectively) as did MPF but not to the same degree (3.3% and 4.2%, respectively). Soleus sEMG RMS amplitude also decreased during conditions 1 and 2 (10.1% and 15.3%, respectively).

Evaluation of recovery following fatigue consisted of the three contractions at 6, 10, and 15 minutes following the end of the fatigue protocol. Force increased during recovery exceeding or returning to initial force levels at 100.5% and 99.5% following conditions 1 and 2, respectively. Force did not “over-recover” as expected. Rather, the condition means returned to initial levels following the fatigue protocols. However, this does mean that of the 28 protocols some showed an over-recovery of force while force remained diminished for others. Over-recovery did not appear to be dependent on the depth of fatigue since over-recovery was seen in 9 of the 14 condition 1 (10% decrement) sessions and 7 of the 14 condition 2 (20% decrement) sessions.

Tibialis anterior sEMG RMS amplitude increased following fatigue but did not fully recover within the 15 minutes, remaining at only 88.6% and 95% of initial levels following conditions 1 and 2, respectively. Tibialis anterior MPF increased following fatigue, recovering to 113.8% and 114.8% following conditions 1 and 2, respectively. Soleus sEMG RMS was not consistent across the two conditions. Following fatigue in condition 1, soleus RMS continued to decline from 89.9% to 80.8% of initial levels. Following fatigue in condition 2, soleus RMS recovered slightly from 84.7% to 88% of initial levels.

5.2.1 The Amount of Fatigue

The changes in force and sEMG with fatigue are highly dependent upon the protocol used to elicit fatigue. The force level of contraction(s), the number of contractions, and the length of time a contraction is held for will all greatly affect fatigue measures. This makes it difficult to compare fatigue measures within the literature; however, our values appear to be within a normal range and consistent with studies using similar protocols. In the present study, contractions were performed until a decline in force of 10 and 20% were reached to elicit fatigue, which was accompanied by an average decrease in TA RMS amplitude of 17%. This value is in range of previous values reported in the literature during similar protocols. Following a 16-20% decline in force, Gabriel and colleagues (2001) found that agonist sEMG RMS amplitude had decreased 10%. The decline in agonist sEMG amplitude has been found to be slightly greater ranging from 22-34% following fatigue protocols with a 30-50% decline in force (Baker et al., 1993; Behm & St. Pierre, 1998; Patikas et al., 2002). It was found that soleus sEMG RMS amplitude decreased 12.7% during fatigue. This is almost identical to the 12% decrease in antagonist activity seen by Patikas and colleagues (2002) following a fatigue protocol consisting of maximal, intermittent contractions of the plantar flexors. During fatigue TA MPF decreased an average of 3.8% in accordance with Gabriel and colleagues (2001) who found a 2.7-6% decrease in agonist MPF following a similar fatigue protocol with a 16-20% decline in force.

5.2.2 The Amount of Recovery

Recovery was measured across 15 minutes using 3 second maximal voluntary contractions at minutes 6, 10, and 15. Force levels returned to pre-fatigue values (99.5%

and 100.5% initial) while RMS amplitude recovered to approximately 92% and 85% of initial values in the TA and soleus, respectively. Mean power frequency recovered to values greater than pre-fatigue in the TA (114% initial).

Over-recovery in force has been demonstrated before (Kroll, 1967b; Kroll, 1971b; Stull & Clarke, 1971) however force often is found to remain diminished for a period of time following fatigue. Although literature is readily available regarding the recovery of force (or lack thereof) from a fatigue protocol it is difficult to compare due to the large differences in methodology. The amount of recovery reported in the literature varies greatly due to the differences in fatigue protocols, especially in the case of sustained contractions where force recovery is highly dependent on blood flow return following contraction induced occlusion (Cornwall et al., 1994; Edwards et al., 1977; Miller et al., 1987; Petrofsky et al., 1981; Pitcher & Miles, 1997; Sjogaard et al., 1988). Literature utilizing an intermittent, high intensity fatigue protocol similar to the present study has mainly shown that force values recover to only 80-90% of initial maximum after 10-15 minutes of recovery following intermittent fatigue protocols (Baker et al., 1993; Behm & St. Pierre, 1998; Jakobi et al., 2000). This prolonged decrement is most likely due to the greater amount of fatigue induced during these studies (30-60% force decline) compared to the present study (10 and 20% decline). It is not surprising that the amount of recovery occurring within a minimal amount of time (10-15 minutes) is highly dependent on the post-fatigue force value (Stull & Clarke, 1971).

Root-mean-square amplitude of the sEMG exhibiting a decrease during a fatiguing protocol is found to recover to normal or near-normal values within 5-10 minutes of recovery (Baker et al., 1993; Behm & St. Pierre, 1998). The sEMG RMS

amplitude of the TA recovered to near-normal values (92%) while the soleus sEMG RMS amplitude recovered only to 85%. Mean power frequency has been found to fully recover within 10 minutes following fatigue, with the majority of recovery occurring within 2-5 minutes (Cornwall et al., 1994; Kuorinka, 1988; Mills, 1982; Petrofsky, 1981). In fact, MPF increased (i.e., “over-recovered”) in agreement with previously published values, ranging from 101 to 121%, due to a warm up effect as detailed later (Hara, 1980; Hedayatpour et al., 2008; Kuorinka, 1988; Van der Hoeven et al., 1993; Zwarts et al., 1987).

5.2.3 Hypothesis I: Potentiation

The hypothesis of potentiation is not accepted as the cause of over-recovery; however, it was present throughout the fatigue and recovery session. Had potentiation contributed to over-recovery, force would have either returned to initial levels or been enhanced while TA MPF decreased without a change in sEMG RMS amplitude. The decrease in MPF associated with potentiation is caused by a decrease in motor unit firing rate due to the greater amount of force being exerted per muscle fibre (Inglis et al., 2011; Klein et al., 2001). The results suggest that potentiation does not explain the recovery in force because MPF actually increased while there was a slight decrease in sEMG RMS amplitude during recovery as compared to pre-fatigue.

Although the presence of potentiation has been seen following fatigue (Hamada et al., 2003) and in high-frequency force (Bruton et al., 1996), it is mainly seen in low frequency twitch force following a single conditioning contraction (Hamada et al., 2000). As well, the time course of the protocol may not have been ideal for potentiation to occur. In a review of PAP literature, MacIntosh and colleagues (2012) found that PAP dissipates

over 4-6 minutes following a conditioning stimulus of varying types. Therefore, the fact that recovery was measured from 6-15 minutes post-fatigue may have already allowed for any PAP to dissipate. Potentiation was present following fatigue and during recovery, as evidenced by an increase in twitch peak force as compared to baseline. However, this potentiation was not a factor during maximal voluntary contractions.

5.2.4 Hypothesis II: Neuromotor Coordination

It was hypothesized that neuromotor coordination contributing to force over-recovery would be evident as an increase in agonist or synergist activation and/or a decrease in antagonist co-activation (Carolan & Cafarelli, 1992; Gabriel, Basford, & An, 2001; Rutherford & Jones, 1986). It is possible that adaptations in neuromotor coordination may have contributed to a return or “recovery” of force to pre-fatigue levels following fatigue. Both TA and soleus sEMG RMS amplitude decreased during fatigue in both conditions. While TA sEMG RMS amplitude recovered following fatigue, it remained diminished as compared to pre-fatigue. Soleus sEMG RMS amplitude recovered slightly during condition 2 however it also remained diminished as compared to pre-fatigue. However, soleus sEMG RMS amplitude in condition 1 continued to decline during recovery. The greater decline in soleus activity compared to tibialis anterior activity during the recovery period may have contributed to force recovery through decreased antagonist co-activation.

A co-activation ratio can be calculated to further evaluate the coordination between agonist and antagonist muscle activation by dividing the TA sEMG RMS amplitude by the soleus sEMG RMS amplitude. An increase in the ratio means that there was greater agonist activity compared to antagonist activity, meaning that there was a

decrease in antagonist co-activation. The ratio was averaged across participants and across conditions, as there was no significant difference between conditions. There was a significant increase from 17.43 in Block 1 (pre-fatigue) to 19.16 in Block 3 (recovery). This indicates that there was a greater amount of TA activation compared to soleus during recovery compared to pre-fatigue. Thus, one interpretation is that there was less co-activation opposing the agonist force.

Although the antagonist activation was minimal (13 μV , overall average during fatigue and recovery) compared to the agonist (202 μV), co-activation may still have an effect on force due to the size of the plantar flexor muscles. Surface EMG measures are collected from a single location and understood to be representative of the activity being produced across the whole muscle, or group of muscles (Staudenmann et al., 2010). The dorsiflexors are a small muscle group compared to the plantar flexors. Wickiewicz and colleagues (1983) compared the physiologic cross-sectional area (CSA) of the lower limb and found that the CSA ratio of the dorsiflexors to plantar flexors was 0.14. Thus, a large amount of activation was distributed over a small CSA, counterbalanced by a small amount of activation distributed over a large CSA. When CSA is taken into account, the contribution of antagonist activity to force production may be underestimated based solely on the magnitude of sEMG. A decrease in antagonist co-activation, therefore, cannot be ruled-out as contributor to force during recovery.

5.2.5 Warm-Up Phenomenon

The most plausible explanation for the recovery of force to initial levels following a fatigue protocol may be a phenomenon termed the ‘warm-up effect’. This phenomenon

refers to the peripheral effects associated with the warming of a muscle during activity or through passive heating (Van der Hoeven & Lange, 1994).

The warm-up effect has been shown to increase maximal voluntary contractions (Stewart et al., 2003; Van der Hoeven & Lange, 1994) and squat jump height (Stewart et al., 2003). While force did not “over-recover” in the present study, the average response demonstrated that it returned to initial values. This is noteworthy because strength decrements (80-90% MVC) typically persist 10 to 15 minutes into the recovery period (Baker et al., 1993; Behm & St. Pierre, 1998; Jakobi et al., 2000). However, if individual responses are considered, 16 out of 28 curves actually exhibited between 101 and 115% force over-recovery, regardless of the condition (10 versus 20% fatigue). Moreover, the effects of warm-up on surface EMG are consistently reported in the literature as a decrease in RMS amplitude accompanied by an increase in MPF as was observed in the current work (Hara, 1980; Hedayatpour et al., 2008; Kuorinka, 1988; Mills, 1982; Stewart et al., 2003; Van der Hoeven & Lange, 1994; Van der Hoeven et al., 1993; Zwarts et al., 1987).

Further support for warm-up effect is given by an increase in skin or muscle temperature. Skin temperature was measured in the present study at the beginning and end of each session at the location of the TA surface electrodes. Temperature measured at the end of the session may however be an underestimation of the change because it was recorded after completion of the last recovery contraction, approximately 17-20 minutes post-fatigue. Nevertheless, a significant ($p < 0.05$) increase of 0.7°C was seen, averaged across participants and conditions. Although only skin temperature was recorded, it can

be considered representative of muscle and 'near nerve' temperature changes (Rutkove, 2001).

Physiologically, the changes associated with warm-up occur both at the nerve and muscle with similar outcomes (Rutkove, 2001). An increase in temperature causes increased opening and closing of sodium (Na^+) and potassium (k^+) channels resulting in increased nerve conduction velocity (NCV), as well as decreased duration and amplitude of motor unit action potential (MUAP) (Rutkove, 2001). At the muscular level, an increase in temperature causes increased Na^+K^+ ATPase activity resulting in an increase in muscle fiber conduction velocity (MFCV), as well as a decrease in the duration and amplitude of compound muscle action potentials (CMAP) (Rutkove, 2001; Winkel & Jorgensen, 1991). Although MFCV was not recorded in the present study the changes in sEMG activity are in accordance with previous literature, which correlated sEMG changes with MFCV increases. Mean or median power frequency and MFCV have been shown to have a positive linear relationship (Van der Hoeven & Lange, 1994; Van der Hoeven et al., 1993; Zwarts et al., 1987).

5.2.6 Alternative Hypotheses

Although the hypothesis of the warm-up effect explains the changes in sEMG in the present study there are alternative hypotheses that may concurrently have contributed to the full recovery of force. Although it is not possible to measure with sEMG, the presence (or increase) of doublets or triplets during the recovery period may have increased the rate of force development and mean force. Catch-like properties refers to the presence of doublets or triplets usually present at the onset of a contraction causing increased rate of force development through increased sarcoplasmic Ca^{2+} concentration

and increased stiffness of the series elastic component of the muscle (Binder-MacLeod & Barrish, 1992; Binder-MacLeod & Kesar, 2005; Ding et al., 2003). The catch-like properties of a muscle are most evident when skeletal muscle is fatigued and unpotentiated (Burke et al., 1976; Ding et al., 2003). The presence of catch-like properties has also been found to be enhanced following ballistic training in the dorsiflexor muscles. Following a period of training there was an increased presence of doublets (motor units located within 5 ms) from 5% to 33%, occurring both at the onset and throughout voluntary contractions (Van Cutsem, Duchateau & Hainaut, 1998).

Alternatively, motoneuron facilitation may have contributed to the recovery of force. The repeated stimulation of motoneurons has been shown to cause an increase in the resting potential bringing it closer to threshold for activation (Lagasse & Roy, 1989; McPherson et al., 2008; Schwindt & Crill, 1980). This can lead to a decrease in the afterhyperpolarization time, an increase in the amplitude of the action potential and a decrease in the rate of activation (MacDonell et al., 2010). The conditioning stimuli that have shown to invoke such a response include functional electrical stimulation (FES) training for hours (Lagasse & Roy, 1989; Vodovnik et al., 1988), repeated isometric contractions (Samii et al., 1996), voltage clamp stimulation (Schwindt & Crill, 1980; Lee & Heckman, 2000), and tonic vibration during voluntary contractions (MacDonell et al., 2010; McPherson et al., 2008).

Motoneuron facilitation is thought to arise from persistent inward currents (PICs). This refers to the increase in membrane potential by an inward currents associated with calcium and/or sodium ions (Crill, 1996; Lee & Heckman, 2000; Schwindt & Crill, 1980). The increased cell membrane potential following a conditioning stimulus results in

the failure of the voltage-gated calcium and sodium channels to inactivate (Heckman et al., 2008). Persistent inward currents then result in a plateau potential wherein the motoneuron can self-sustain its firing. Under these conditions, the response to synaptic input is amplified (Collins, Burke & Gandevia, 2001; Fuglevand et al., 2006).

Plateau potentials are typically tracked during low-level contractions through the identification of a single motor unit using indwelling EMG (Fuglevand et al., 2006; Gorassini et al., 2002; Walton et al., 2002). Experimentally, the sustained membrane depolarization associated with PICs has been shown to result in self-sustained motor unit firing, decreased motor unit recruitment thresholds, and force increases continuing after the removal of the stimuli (Collins et al., 2001; Collins et al., 2002; Gorassini et al., 2002; Walton et al., 2002). These changes are expected due to the increased excitation of motoneurons for the same amount of synaptic input (Collins, Burke & Gandevia, 2001; Fuglevand et al., 2006).

For any given “submaximal” force level, there will more active motor units but lower motor unit firing rates (Collins et al., 2002). In the sEMG signal, an increase in amplitude is expected given the larger number of active units. For maximal contractions, the recruitment threshold is a non-issue since all motor units are recruited, and there is evidence that PICs have little impact on firing rates during the force gradation process up to a maximal contraction (Oya, Riek & Cresswell, 2009). Thus, although the data from the present study do not support motoneuron facilitation it may be an underlying process contributing to the recovery in force that warrants further investigation.

5.3 Summary and Conclusion

The purpose of this study was to investigate the potential mechanisms involved in the over-recovery phenomenon. Participants completed a fatigue protocol to a specific force decrement followed by a 15 minute recovery period. Force was measured as an indicator of participants' fatigue and recovery, sEMG was measured to assess the contribution of the neuromuscular system. Lastly, evoked twitches were administered throughout the session to track involuntary neuromuscular processes and provide a measure of potentiation.

Following the fatigue protocols force returned to pre-fatigue levels. This result was accompanied by an increase in the TA MPF, and a decrease in the TA and soleus RMS amplitude as compared to pre-fatigue values. These results indicate that the recovery of force was facilitated by the warm-up effect due to the fatigue protocol resulting in an increase in MFCV as evidenced by an increase in MPF and a decrease in RMS amplitude. Furthermore, although neither TA nor soleus RMS amplitude fully recovered following fatigue the amount of TA RMS amplitude recovery was greater than that of the soleus. This means that the co-activation ratio of TA to soleus activity increased which may have contributed to force recovery through less antagonist co-activation. Lastly, the hypothesis of potentiation was dismissed as a factor in the force response since previous findings determined that firing rates decrease with the presence of potentiation and the results of the present study found an increase in MPF.

5.4 Future Directions

Future directions for the present study include clarification of the mechanisms underlying the increase in MPF seen during recovery, which may be purely biophysical

in nature or of neuromuscular origin. Muscle fibre conduction velocity and muscle temperature should be monitored concurrently to examine the contribution of the warm-up effect to recovery. The alternate explanation behind the increase in MPF is an increase in motor unit firing rates and/or an increased recruitment of high-threshold motor units as they recover. This can be measured through the simultaneous use of indwelling (needle) and surface electrodes. An additional benefit to the use of indwelling EMG is the ability to identify the potential presence of catch like properties during recovery. Since the force increase caused by catch like properties is due to doublets, this mechanism can be identified, or dismissed, through the examination of indwelling EMG signals.

Finally, it was found that over-recovery, or recovery to baseline, appeared to be participant-dependent as opposed to fatigue-dependent. The 10% condition had 9 participants display over-recovery while the 20% condition had 7 participants over-recover. This indicates that the depth of fatigue was most likely not the mediating factor. Future work should determine why some participants return to, or exceed baseline strength following a fatigue protocol, and others do not. To this end, the recovery period should be extended up to 20 minutes or more, as some subjects may take longer to recuperate from the fatigue bout.

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APPENDIX A

Sample Size Estimation

Sample size was determined using Cohen's (1998) case four formula. This required the *a priori* establishment of the following values:

Level of significance (α)

The appropriate power (β)

The mean (\bar{x}) and standard deviation (σ) of the criterion measure

The intraclass coefficient (R) measure of the criterion measure (see Appendix E)

The effect size (ES) which is deemed important or non-trivial by the investigator

To balance the risk of Type I and Type II errors, Cohen (1969) suggests using a 4:1 ratio.

To satisfy this ratio α of 0.05 and β of 0.20 are selected, where power is 0.80 (1- β).

Force obtained from pilot data (N=6) had a mean of 405.294 N and a standard deviation of 85.156 N. An 8% over-recovery force is considered non-trivial by the investigator as that was average effect-size previously observed by Kroll (1967b, 1971b). Therefore, the effect size based on 8% of the mean is 32.424 N. The intraclass coefficient measure obtained from pilot data was R=0.8843.

$$d_4 = \frac{ES}{\sigma} = 0.381$$

$$d = \frac{d_4}{\sqrt{1-R}} = 1.12$$

Linear interpolation is needed to estimate sample size from Cohen's (1988) sample size tables. Where $x_1=1.0$ with a table value of $y_1=17$ and $x_2=1.2$ with a table value of $y_2=12$ calculates sample size to be 14 participants.

$$N = y_2 + \frac{x_2 - d}{x_2 - x_1} \cdot (y_1 - y_2) = 14$$

APPENDIX B**Informed Consent****INFORMED CONSENT DOCUMENT**

Title of Project: Investigation of muscle electrical activity and force

Principle Investigator: David A. Gabriel, Ph.D., FACSM
Professor Biomechanics
Department of Kinesiology
Brock University
Phone: 905-688-5550 ext. 4362
E-mail: dgabriel@arnie.pec.brocku.ca

Lara Green, BKin
Faculty of Applied Health Sciences
Brock University
Email: lara.green@brocku.ca
Phone: 905-688-5550 ext. 3965 (WH21 Electromyographic
Kinesiology Lab)

This study has been reviewed and approved by the Brock Research Ethics Board (#02-283). The Brock Research Ethics Board requires written informed consent from participants prior to participation in a research study so that they can know the nature and risks of participation and can decide to participate or not to participate in a free and informed manner. You are asked to read the following material to ensure that you are informed of the nature of this research study and how you will participate in it if you consent to do so. Signing this form will indicate that you have been so informed and that you give you consent.

Introduction

You are being asked to participate in research being conducted by David A. Gabriel, Ph.D. The on-going research program is focused on the relationship between skeletal muscle force and the electrical activity that it generates. The electrical signal of skeletal muscle is measured from the skin surface, similar to electrocardiography (EKG) which measures the electrical activity of cardiac (heart) muscle. The skeletal muscle electrical signal is termed, electromyography (EMG). The signal can then be analyzed in a number of different ways, and each method has an impact on the EMG-to-force relationship. The long-term aspect of this project involves a continual search for better signal processing techniques on the computer, not changes in the measurement procedures.

You will come to the Electromyographic Kinesiology Laboratory (WH21). There will be three sessions with no less than 24 hours, but no more than one-month between each session. Each session will last approximately three hours. You are requested not to start any new physical activity during the interval between each session. These sessions will be scheduled for the convenience of you and the investigator.

Plan and Procedure

David A. Gabriel, Ph.D. and/or his surrogate will conduct all testing. You will begin with a familiarization session consisting of the following. You will be seated in a chair with hips and knees at approximately 90° and belts strapped across the waist and chest to avoid extraneous movement. Your right foot will be placed in a foot plate with an adjustable bar across the toes. You will be familiarized with the equipment and the maximal isometric voluntary contraction (MVC) protocol by completing a series of 5-second contractions. You will be asked to perform contractions isolating your tibialis anterior slowly increasing the level of the contraction to maximal. No more than 20 contractions will be performed on this day.

The following procedures will take place during each session. First, the right leg will be prepared for exercise testing. Small areas of the tibialis anterior, extensor digitorum longus, and soleus will be shaved, lightly abraded and cleansed with alcohol. These areas correspond to the location of the electrodes that will be taped to the skin surface. The electrodes will measure the electrical activity of leg muscles, similar to the more familiar electrocardiogram that measures the electrical activity of the cardiac (heart) muscles. These areas will be located 1cm distal to the motor point (neuromuscular junction) of each muscle. This point will be found by using a low level of repeated electrical stimulation to the skin's surface.

You will perform maximal effort isometric (same length) contractions until you experience a decrement in strength of the muscle. The contractions will be 5 seconds in duration at 10-second intervals. Because we require a specific level of fatigue, the actual number of contractions will vary between individuals. Previous experience suggests that up to 75 contractions may be required. Strength measurements will be taken while seated on a chair designed to isolate the action of the dorsiflexion muscles. Adjustable straps on the chair will ensure stability and minimize extraneous movements. A load cell will be attached to the foot plate under the ball of the foot. The leg not being tested will rest on a foot support. It is important not to hold your breath while strength testing.

A recovery period will immediately follow the fatigue protocol. You will be required to perform a recovery test at minutes 1, 3, 6, 10, and 15 following fatigue (for a total of 5 tests). Each test will include 1 electrical stimulation while the leg is at rest, followed by a 3-second MVC, and finally 3 more electrical stimulations while the leg is at rest, each separated by 1-2 seconds. This will end the session. Before you leave the Electromyographic Kinesiology Laboratory, we will mark the location of the electrodes with a non-toxic pen. You will be required to maintain these marks for the duration of the study.

Before electrode placement, the skin surface will be shaved, lightly abraded, and cleansed with alcohol to limit the at the skin-electrode impedance. Bipolar surface electrodes will record voluntary muscle activity at the skin surface of the tibialis anterior, extensor digitorum longus, and soleus. The positions of the electrode will be marked with endelible ink to ensure the consistency of the placement across test days. The participants will be further instructed to maintain the markings between sessions.

Risks and Discomforts

It is not possible to predict all possible risks or discomforts that volunteer participants may experience in any research study. Based upon previous experience, the present investigator anticipates no major risks or discomforts will occur in the present project.

1. Participants sometimes experience mild discomfort when the skin is gently cleaned and rubbed with a mild abrasive in preparation for electrode placement. On occasion, some subjects may experience skin irritation associated with the placement of the electrodes. This is usually very mild and goes away in a few hours, or a day.
2. There may be discomfort related to the delayed onset of muscle soreness associated with isometric contractions of the arm muscles. If muscle soreness does occur, it is usually very mild and should dissipate within 72 hours.
3. Risk of Burn. There is a very minimal risk of burn due to electrical stimulation. This will be controlled through precautions regarding length and intensity of the stimulation, quality of contact with the skin, and limited reuse of electrodes as per the manufacturer's instructions.
4. Maximal effort isometric contractions are associated with an increase in blood pressure. You must make sure that you do NOT hold your breath during maximal exertions. If you have received medical clearance and/or are already physically active, the risks are minimal.

Voluntary Participation

Participation in this study is voluntary. Refusal to participate will NOT result in loss of access to any services or programs at Brock University to which you are entitled. You will inform the investigator, David A. Gabriel, Ph.D., of your intention to withdrawal prior to removing yourself from this study.

Discontinuation of Participation

Participation in this research study may be discontinued under the following circumstances. The investigator, David A. Gabriel, Ph.D., may discontinue your involvement in the study at any time if it is felt to be in your best interest, if I you not comply with study requirements, or if the study is stopped. You will be informed of any changes in the nature of the study or in the procedures described if they occur. It is important to remember that you are free to terminate your participation at any time, for any reason.

Potential Benefits

Participants will receive no direct benefits from participating in this study. However, participants should know that their willingness to serve as a subject for this experiment will help a Brock University researcher and other scientists develop new theories of exercise that will benefit individuals in the future.

Costs and Compensation

The cost of the test and procedures are free. You will not receive any form of compensation for your participation in this study.

Confidentiality

Although data from this study will be published, confidentiality of information concerning all participants will be maintained. All data will be coded without personal reference to you. Any personal information related to you will be kept in a locked office, to which only the investigator has access. Four investigators will have access to the data, however, names of participants or material identifying participants will not be released without written permission except as such release is required by law.

Persons to Contact with Questions

The investigator will be available to answer any questions concerning this research, now or in the future. You may contact the investigator, David A. Gabriel, Ph.D., by telephone during office hours at (905) 688-5550 extension 4362, or by email at dgabriel@arnie.pec.brocku.ca. Also, if questions arise about your rights as a research subject, you may contact the Office of Research Services at (905) 688-5550 extension 3035. If you wish to speak with someone not involved in the study, please call the Chair of the Department of Physical Education and Kinesiology at (905) 688-5550 ext. 4538.

Consent to Participate

Certify that you have read all the above, asked questions and received answers concerning areas you did not understand, and have received satisfactory answers to these questions. Furthermore, you have completed the PAR-Q questionnaire indicating that you are physically able to participate. You willingly give consent for participation in this study. (A copy of the consent form will be given to you).

Name of Participant (Please Print): _____

Signature of Participant

Date (day/month/year)

In addition to the considerations described in this document, the investigator fully intends to conduct all procedures with the subject's best interest uppermost in mind, to insure the subject's safety and comfort.

I have fully explained the procedures of this study to the above volunteer. I believe that the person signing this form understands what is involved in this study and voluntarily agrees to participate.

Date (day/month/year)

David A. Gabriel, Ph.D., FACSM
Professor Biomechanics
Department of Kinesiology

OR

Lara Green, BKin
Faculty of Applied Health Sciences

Information Letter

Title of Project: Neuromotor mechanisms involved in the recovery from local muscular fatigue

Principle Investigator: David A. Gabriel, Ph.D., FACSM
 Professor Biomechanics
 Department of Kinesiology
 Brock University
 Phone: 905-688-5550 ext. 4362
 E-mail: dgabriel@arnie.pec.brocku.ca

Lara Green, BKin
 Faculty of Applied Health Sciences
 Brock University
 Email: lara.green@brocku.ca
 Phone: 905-688-5550 ext. 3965 (WH21 Electromyographic
 Kinesiology Lab)

The following letter and consent form describe a study that I wish to conduct, with you as a participant. I am a professor of biomechanics within the Department of Kinesiology. My research interests pertain to measuring muscle electrical activity from the skin surface during maximal effort contractions. I am trying to understand what information is contained in the electrical signal generated by muscle contractions. The main purpose is to advance the use of non-invasive measures of muscle function. The alternative involves inserting a needle into the muscle to measure its electrical activity.

There will be three sessions with no less than 24 hours, but no more than one-month between each session. Each session will last approximately three hours. You are requested not to start any new physical activity during the interval between each session. The test sessions will be scheduled at the convenience of you and the investigator. If for any reason you decide to cancel a test session, I would appreciate advance notice and the opportunity to reschedule the test session. It is important to remember that you are free to withdrawal consent at any time without loss of access to any services or programs at Brock University to which you are entitled.

If you agree to participate in the study, you will be asked to complete the PAR-Q questionnaire. Your responses will determine you if you are physically fit and can participate in a study that requires rigorous physical exertion. To participate in this study you will need to wear shorts. Changing rooms are conveniently located on the first floor of the Physical Education Complex. During the first session, we need to take some preliminary measurements: age, height, weight, length, and circumference, of the leg being tested.

Familiarization Session

You will be seated in a chair with hips and knees at approximately 90° and belts strapped across the waist and chest to avoid extraneous movement. Your right foot will be placed in a foot plate with an adjustable bar across the toes. You will be familiarized with the equipment and the maximal isometric voluntary contraction (MVC) protocol by completing a series of 5-second contractions. You will be asked to perform contractions isolating your tibialis anterior slowly increasing the level of the contraction to maximal. No more than 20 contractions will be performed on this day.

The following procedures will take place during each session:

1. First, the right leg will be prepared for exercise testing. Small areas of the tibialis anterior, extensor digitorum longus, and soleus will be shaved, lightly abraded and cleansed with alcohol. These areas correspond to the location of the electrodes that will be taped to the skin surface. The electrodes will measure the electrical activity of leg muscles, similar to the more familiar electrocardiogram that measures the electrical activity of the cardiac (heart) muscles. These areas will be located 1cm distal to the motor point (neuromuscular junction) of each muscle. This point will be found by using a low level of repeated electrical stimulation to the skin's surface.
2. Subject will sit in a testing chair with their lower right leg secured in a jig designed to isolate dorsiflexion during an isometric contraction. Five electrical stimulations are then administered to the deep peroneal nerve behind the knee to evoke maximal M-wave responses. Each stimulation is separated with 1-2 seconds of rest. You will then perform 5 maximal isometric voluntary contractions 5-seconds in duration, each separated by 2-minutes of rest. Upon completion of these 5 contractions, 5 more maximal M-wave responses will be evoked. There will be a 5 minute rest before the next protocol.
3. You will perform maximal effort isometric (same length) contractions until you experience a decrement in strength of the muscle. The contractions will be 5 seconds in duration at 10-second intervals. Because we require a specific level of fatigue, the actual number of contractions will vary between individuals. Previous experience suggests that up to 75 contractions may be required. Strength measurements will be taken while seated in a chair designed to isolate the action of the dorsiflexion muscles. Adjustable straps on the chair will ensure stability and minimize extraneous movements. A load cell will be attached to the foot plate under the ball of the foot. The leg not being tested will rest on a foot support. It is important not to hold your breath while strength testing.
4. A recovery period will immediately follow the fatigue protocol. You will be required to perform a recovery test at minutes 1, 3, 6, 10, and 15 following fatigue (for a total of 5 tests). Each test will include 1 electrical stimulation while the leg is at rest, followed by a 3-second MVC, and finally 3 more electrical stimulations while the leg is at rest, each separated by 1-2 seconds. This will end the session. Before you leave the Electromyographic Kinesiology Laboratory, we will mark the location of the electrodes with a non-toxic pen. You will be required to maintain these marks for the duration of the study.

Recording Voluntary Muscle Activity. Before electrode placement, the skin surface will be shaved, lightly abraded, and cleansed with alcohol to limit the at the skin-electrode impedance. Bipolar surface electrodes will record voluntary muscle activity at the skin surface of the tibialis anterior, extensor digitorum longus, and soleus. The electrodes will be placed in line with the muscle fibres and away from the motor point. The positions of the electrode will be marked with endelible ink to ensure the consistency of the placement across test days. The participants will be further instructed to maintain the markings between sessions.

Participation in this study is not risk-free. As part of the informed consent process you have to be made aware that the following side effects are possible:

1. Skin irritation. Skin irritation may result from mildly abrading the skin, cleaning the skin with alcohol, then applying surface electromyographic (sEMG) recording

electrodes with electrolyte gel. Washing the electrolyte gel from skin surface and applying skin lotion immediately after the test session can minimize the irritation.

2. Muscle soreness. It is possible that you might experience slight muscle soreness within 48 hours of the test. If soreness does occur, it will be very mild and dissipate within 72 hours.
3. Risk of Burn. There is a very minimal risk of burn due to electrical stimulation. This will be controlled through precautions regarding length and intensity of the stimulation, quality of contact with the skin, and limited reuse of electrodes as per the manufacturer's instructions.
4. Systemic stress due to maximal exertion. Maximal effort contractions are associated with an increase in blood pressure. You must make sure that you do NOT hold your breath during maximal exertions. If you have received medical clearance and/or are already physically active, the risks are minimal. *Understand that should any of these side effects occur, you are free to withdrawal from the study because of them. It is important to remember that you are free to withdrawal consent at any time without loss of access to any services or programs at Brock University to which you are entitled. The researchers' first priority as an investigator is to maintain the emotional, psychological, and physical health of those participating in the study.*

Aside from your name, address, and a limited number of physical measurements, the majority of the data collected in this study will be in the form of electronic signals stored on computer hard disk and on CD-ROM. The data generated by this study will only be used for educational and research purposes. The results will be presented at professional meetings, and then publish in scientific journals. To ensure anonymity, personal information will be coded and stored in a locked office to which only the investigator has access. The names of the participants or material identifying participants will not be released without written permission except as such release is required by law. I will maintain the data and associated records for the duration of my academic career, or until such time they are no longer useful to me.

This study has been reviewed and received approval from the Brock University Research Ethics Board (#02-283). Should you have any questions or concerns about your involvement in the study, you may contact the Office of Research Services at 905-688-5550 (extension 3035).

Your written consent is needed to participate in the study. To indicate your consent, please complete the enclosed CONSENT FORM and return to the investigator before entering the study. Thank you for your time and consideration in this matter.

Best Wishes,

David A. Gabriel, Ph.D., FACSM

Ethics Clearance



Brock University
 Research Ethics Board
 Tel: 905-688-5550 ext. 3035
 Email: reb@brocku.ca

Certificate of Ethics Clearance for Human Participant Research

DATE: June 6, 2011

PRINCIPAL INVESTIGATOR: GABRIEL, David A. - Physical Education & Kinesiology

FILE: 02-283 - GABRIEL

TYPE: Faculty Research STUDENT:
 SUPERVISOR:

TITLE: Analysis of Surface Electromyographic Spike Activity

ETHICS CLEARANCE GRANTED

Type of Clearance: MODIFICATION Expiry Date: 5/31/2012

The Brock University Research Ethics Board has reviewed the above named research proposal and considers the procedures, as described by the applicant, to conform to the University's ethical standards and the Tri-Council Policy Statement. Clearance granted from **6/6/2011** to **5/31/2012**.

The Tri-Council Policy Statement requires that ongoing research be monitored by, at a minimum, an annual report. Should your project extend beyond the expiry date, you are required to submit a Renewal form before **5/31/2012**. Continued clearance is contingent on timely submission of reports.

To comply with the Tri-Council Policy Statement, you must also submit a final report upon completion of your project. All report forms can be found on the Research Ethics web page at <http://www.brocku.ca/research/policies-and-forms/research-forms>.

In addition, throughout your research, you must report promptly to the REB:

- a) Changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
- b) All adverse and/or unanticipated experiences or events that may have real or potential unfavourable implications for participants;
- c) New information that may adversely affect the safety of the participants or the conduct of the study;
- d) Any changes in your source of funding or new funding to a previously unfunded project.

We wish you success with your research.

Approved:

Michelle McGinn, Chair
 Research Ethics Board (REB)

Note: Brock University is accountable for the research carried out in its own jurisdiction or under its auspices and may refuse certain research even though the REB has found it ethically acceptable.

If research participants are in the care of a health facility, at a school, or other institution or community organization, it is the responsibility of the Principal Investigator to ensure that the ethical guidelines and clearance of those facilities or institutions are obtained and filed with the REB prior to the initiation of research at that site.

APPENDIX C**Demographic Information and Physical Activity Levels**

Age: _____

Weight: _____

Height: _____

University Major: _____

How many times a week do you weight train? _____

How many hours per week do you weight train? _____

What percentage of time weight training do you spend training:

Upper body: _____

Lower body: _____

How long have you been weight training (please circle):

0-3 months 4-6 months 7-12 months 1-5 years more than 5 years

How many times per week do you do physical activity, other than weight training?

Other than weight training, what other physical activity are you participating in?

Anthropometric Measurements

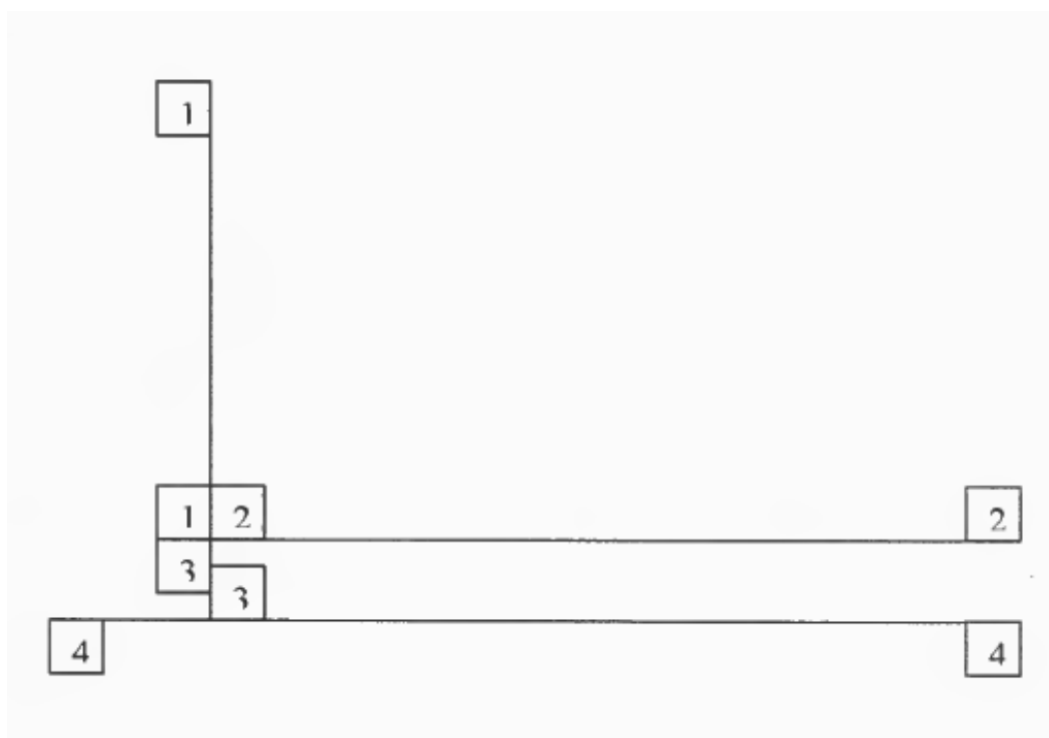
Fibular head to lateral malleolus: _____

Medial malleolus to first metatarsalphalangeal joint: _____

Medial malleolus to base of foot: _____

Foot length (Calcaneus to end of first metatarsal): _____

Calf circumference (at the widest point): _____



Data Collection Sheet

Name _____ Subject # _____ Gender _____

Age	Weight (kg)	Height (cm)

Letter of Invitation: _____ Consent Form: _____ PAR-Q: _____ PA
Questionnaire: _____**Anthropometric Measurements:****Circumferences (cm):**

1. Calf circumference (widest): _____

Lengths (cm):

Fibula head to lateral malleolus: _____

Medial malleolus to 1st metatarsophalangeal joint: _____

Medial malleolus to base of foot: _____

Foot Length (Calc to end of first): _____

Familiarization Day

Date: _____ Time: _____ USE DATA (New Participant): Y / N

Motor Points: TA _____ EDL _____ Soleus _____

	Impedance (Ohms)			Temperature	Amplification		
	TA	EDL	Soleus		TA	EDL	Soleus
PRE							
POST							

MVC Protocol:**3 Twitches (Bipolar):**

1. _____ 2. _____ 3. _____

Set 1: 5 Contractions

1. _____ 2. _____ 3. _____ 4. _____ 5. _____ (twitch)

Set 2: 5 Contractions

1. _____ 2. _____ 3. _____ 4. _____ 5. _____ (twitch)

Set 3: 5 Contractions

1. _____ 2. _____ 3. _____ 4. _____ 5. _____ (twitch)

3 Twitches (Bipolar):

1. _____ 2. _____ 3. _____

Day:____ Date: _____ Time: _____

Condition: 1. 5-Second MVCs to 10%

Motor Points: TA_____ EDL_____ Soleus _____

	Impedance (Ohms)			Temperature	Amplification		
	TA	EDL	Soleus		TA	EDL	Soleus
PRE							
POST							

Bipolar Twitches: 1____ 2____ 3____

5 Trials to Determine MVC:

Trial	MVC Force	
1		Twitch interpolation
2		Twitch interpolation
3		Normal MVC
4		10% increase
5		10% increase (if target reached)

3 Twitches: 1____ 2____ 3____

MVC Force: _____ 90% _____ 80% _____

1	11	21	31	41	51
2	12	22	32	42	52
3	13	23	33	43	53
4	14	24	34	44	54
5	15	25	35	45	55
6	16	26	36	46	56
7	17	27	37	47	57
8	18	28	38	48	58
9	19	29	39	49	59
10	20	30	40	50	60

Number of Contractions (total): _____ MVC Force % at Failure: **10%**

3 Twitches: 1____ 2____ 3____

RECOVERY: Twitch - 3-second MVC - Twitch

Minute	MVC Force
1	
3	
6	
10	
15	

Day: _____ Date: _____ Time: _____

Condition: 2. 5-Second MVCs to 20%

Motor Points: TA _____ EDL _____ Soleus _____

	Impedance (Ohms)			Temperature	Amplification		
	TA	EDL	Soleus		TA	EDL	Soleus
PRE							
POST							

Bipolar Twitches: 1 _____ 2 _____ 3 _____

5 Trials to Determine MVC:

Trial	MVC Force	
1		Twitch interpolation
2		Twitch interpolation
3		Normal MVC
4		10% increase
5		10% increase (if target reached)

3 Twitches: 1 _____ 2 _____ 3 _____

MVC Force: _____ 90% _____ 80% _____

1	11	21	31	41	51	61
2	12	22	32	42	52	62
3	13	23	33	43	53	63
4	14	24	34	44	54	64
5	15	25	35	45	55	65
6	16	26	36	46	56	66
7	17	27	37	47	57	67
8	18	28	38	48	58	68
9	19	29	39	49	59	69
10	20	30	40	50	60	70

Number of Contractions (total): _____ MVC Force % at Failure: 20%

3 Twitches: 1 _____ 2 _____ 3 _____

RECOVERY: Twitch - 3-second MVC - Twitch

Minute	MVC Force
1	
3	
6	
10	
15	

APPENDIX D

Criterion Measures Algorithms

The three criterion measures taken from the sEMG signal include root-mean-square (RMS), mean power frequency (MPF), and median power frequency (MDF). Each measure will be taken from a 500 ms window in the middle of the sEMG signal of the tibialis anterior, extensor digitorum longus, and soleus.

Root-mean-square (RMS) is a measure of the amplitude of the sEMG signal. Each data point of the sEMG signal is first squared (to remove any negatives), these squares will be summed together and finally the square root is taken to offset the effect of squaring the values (Gabriel & Kamen, 2010). The equation is as follows:

$$RMS = \sqrt{\frac{1}{N} \sum_{i=1}^N x_i^2}$$

Mean and median power frequency (MPF and MDF, respectively) will be taken as a measure of the sEMG frequency spectrum. Mean power frequency is the average frequency of the power spectrum. The equation is as follows:

$$MPF = \frac{\sum_{i=1}^M f_i P_i}{\sum_{i=1}^M P_i}$$

Median power frequency is the frequency at which the power spectrum is divided equally into two parts. The equation is as follows:

$$MDF = \frac{1}{2} \sum_{i=1}^M P_i$$

Where P_i is the i th line of the power spectrum and M is the highest frequency in the spectrum (Farina & Merletti, 2000).

APPENDIX E

Reliability (ICC)

To determine the reliability of the criterion measures that will be used, an intraclass correlation coefficient was calculated using Baseline contractions. Maximal voluntary contractions were compared for each participant across 2 days consisting of 4 trials each, for a total of 8 contractions per participant.

A two-way repeated measure ANOVA will be used. The mean squares from this ANOVA will compose the error terms of the intraclass correlation coefficient, which consists of a ratio between the true error and the total error of a single measure.

$$R = \frac{\sigma_t^2}{\sigma_t^2 + \sigma_e^2}$$

Further breaking down the ratio compares the true score variance (σ_{true}^2) to the sum of the true score variance and the error further separated into error attributed to day ($\sigma_{e_1}^2$) and trial ($\sigma_{e_2}^2$) sources (Christie, Inglis, Boucher, and Gabriel, 2005).

$$R = \frac{\sigma_{true}^2}{\sigma_{true}^2 + \frac{\sigma_{e_1}^2}{a} + \frac{\sigma_{e_2}^2}{a \cdot n}}$$

As determined by the ANOVA, the true error is the within subject Mean Squares (MSs), and the total error additionally accounting for the between subjects Mean Squares across days (MSd) and trials (MSt).

$$R = \frac{\frac{(MSs - MSd)}{(day * trial)}}{\left(\frac{MSs - MSd}{(day * trial)}\right) + \left(\frac{MSd - MSt}{day}\right) + \left(\frac{MSt}{(day * trial)}\right)}$$

Table 1. Intraclass correlation analysis of variance for maximal voluntary contractions performed during baseline for force, root mean square (RMS), mean power frequency (MPF), and median power frequency (MDF). Below are the mean squares (MS), variance components (percent accounted for), means, standard error of measurement (SEM), and the resultant intraclass correlation coefficients (R) calculated according to Gabriel and McIntosh (2011).

Source	df	Force	RMS	MPF	MDF
Subjects	5	77182.50	0.0555	2055.42	2467.75
Days (Subjects)	6	8929.60	0.0087	373.02	628.25
Within Cell	36				
$(\sigma_{e_1}^2 - Trials)$		617.95 (5.5%)	0.0048 (41.2%)	96.97 (25.8%)	103.97 (22.4%)
$(\sigma_{e_2}^2 - Days)$		2077.91 (18.5%)	0.0010 (8.5%)	69.01 (18.3%)	131.07 (28.2%)
$(\sigma_{true}^2 - True)$		8531.61 (76%)	0.0059 (50.3%)	210.3 (55.9%)	229.94 (49.4%)
Grand Mean		390.86 N	0.268 mV	112.27 Hz	93.88 Hz
SEM		103.36 N	0.126 mV	24.10 Hz	31.79 Hz
R		0.88	0.84	0.82	0.75

Table 2. Analysis of variance for maximal voluntary contractions performed during baseline testing. Below are the means, standard deviations, percent change, variance components, and the associated F-ratios for force, root mean square (RMS), mean power frequency (MPF), and median power frequency (MDF).

		Force (N)	RMS (mV)	MPF (Hz)	MDF (Hz)
Test Day		M ± SD	M ± SD	M ± SD	M ± SD
1		376.42 ± 111.3	0.256 ± 0.07	112.90 ± 17.9	95.83 ± 20.9
2		405.29 ± 85.2	0.279 ± 0.13	111.64 ± 19.4	91.92 ± 20.4
Percent Change		28.87 (7.7%)	0.023 (9.0%)	1.26 (1.1%)	3.91 (4.1%)
ANOVA <i>F</i> -Ratios	df				
Days	1	16.19*	1.38	0.20	1.77
Subjects	5	124.90*	11.59*	21.2*	23.73*
Days x Subjects	5	14.10*	1.91	4.58*	6.90*
Within Cells	36				

* Significant at the 0.01 probability level

Percent change = (Test Day 2 – Test Day 1 / Test Day 1) x 100