

PROPRIOCEPTIVE NEUROMUSCULAR FACILITATION OF THE WRIST  
FLEXORS

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Submitted in partial fulfillment of the requirements of the degree of  
Master of Science in Applied Health Sciences  
(Kinesiology)

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## ABSTRACT

The present study examined a wrist extension-to-flexion contraction pattern that was theorized to result in proprioceptive neuromuscular facilitation. However, the “reversal of antagonists” contraction pattern may have, alternatively, interfered with motor learning-related increases in strength. Participants ( $N=24$ ) were matched on predicted strength and randomly assigned to either the control or experimental group. Training occurred during three test sessions within a one-week period. Retention and transfer (crossed-condition) tests were administered during a fourth test session two-weeks later. Both groups exhibited comparable increases in strength (20.2%) and decreases in muscle coactivation (35.2%), which were retained and transferred. Decreases in error and variability of the torque traces were associated with parallel decreases in variability of muscle activity. The reversal of antagonists technique did not interfere with motor learning-related increases in strength and decreases in variability. However, the more complex contraction pattern failed to result in proprioceptive neuromuscular facilitation of strength.

## ACKNOWLEDEMENTS

I would like to begin by thanking my supervisor and mentor, Dr. David Gabriel. Without you, the completion of this document and project would not have happened. You have provided me with more support, guidance, man hours, and help than any student could ask for from a supervisor and words cannot express the gratitude I feel. The passion, dedication, knowledge, and also humor you bring to every meeting, chat, or email have made this process so much more enjoyable for me and have allowed me appreciate the past two years so much more... Even through those frustrating times!

Secondly, I owe a very large thank you to both Greig Inglis and Lara Green. Without the many hours both of you put into teaching me the ins and outs of data collection, I most definitely would not have made it through collecting all my data, let alone started the process. I may have in fact still been staring at the computer wondering how to make it collect the EMG signal.

Finally, without the support of my family and friends, making it through the past two years would have been a lot harder. I thank everyone who was always there when I needed to vent, laugh, cry, or even just for a hug. All of you are the reason my sanity was kept during this crazy, intense, but amazing process.

I would like to end with dedicating this document to my father. You were always my biggest supporter and always believed I would accomplish whatever I set my mind to. No matter how frustrated I was during the past two years, you were always there to tell me how proud you were of me and how you knew I would make it through whatever was challenging me. I thank you for this unconditional and unwavering support.

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## CHAPTER 1: INTRODUCTION

Herman Kabat (1952) developed a series of therapeutic resistive exercise techniques based on the work of Sherrington (1906). Sherrington (1906) described a series of experiments that explored segmental reflexes and conditions that produced facilitation and inhibition of reflex muscle contractions. Kabat (1952) utilized these findings to develop contraction patterns that would evoke spinal reflexes to augment or reinforce weak voluntary drive of muscle in patient populations (Kabat, 1947; Kabat & Knott, 1948; 1953). The particular contraction pattern under investigation is the reversal of antagonists technique; it involves a quick reversal of antagonistic musculature. In the case of isometric contractions, the Golgi tendon organs (GTOs) are used to inhibit the agonist and facilitate the antagonist (which is the target muscle) prior to recruiting it as an agonist (Kroll, 1972a).

The efficacy of the reversal of antagonists technique has been studied sporadically over the last 40 years with mixed results (Bohannon, 1985; Gabriel & Kroll, 1991; Gabriel, Basford, & An, 1997; 2001; Grabiner, 1994; Kroll, 1972a; Holt et al., 1969). The few studies that have reported increases in maximal voluntary contractions have been conducted on patient populations (Bohannon, 1985; Holt et al., 1969), which leads to the speculation that facilitation effects cannot be manifested unless there is an existing deficit in neural drive as might exist in a patient population (Bohannon, 1985; Gabriel, Basford, & An, 1997; Kroll, 1972a; 1972b).

While the results of the facilitation effects are thus far equivocal, there is direct evidence that the reversal of antagonists may interfere with the early strength gains associated with motor learning (Gabriel & Kroll, 1991; Kroll, 1972a; 1972b), which has

particular implications for rehabilitation. The reversal of antagonists technique is a more complicated contraction pattern than agonist only resistive exercise. To be effective, the reversal sequence must occur within a very specific time period (1 second) corresponding to maximal changes within the spinal cord when the GTOs are activated (Moore & Kukulka, 1991). It is therefore reasonable to suggest that the reversal of antagonists technique is a more difficult task compared to agonist only contractions (Kroll, 1972a; 1972b).

Task complexity is a concern because the reversal of antagonists is used for stroke rehabilitation in older adults (Bohannon, 1985, Dickstein et al., 1986; Duncan et al., 1998; Ernst, 1990; Lisiński, Huber, Samborski, & Witkowska, 2008) who already exhibit decrements in motor performance and learning (Shea, Park, & Braden, 2006, Voelcker-Rehage, 2008). In support, Barry, Riek, and Carson (2005) provide evidence that task complexity for isometric contractions reduces motor output in older adults (65-80 years). The authors studied a visually guided aiming task that required the generation of isometric torques about the elbow joint to targets corresponding to 30 and 50% MVC, every 30 degrees in the frontal plane. There were simple contractions that required only flexion, extension, supination, or pronation. However, the off axes contractions were more complex and involved two in combination (i.e., flexion and supination, flexion and pronation, extension and supination, extension and pronation). Compared to the younger adults (19-29 years), deficits in the rate of force development exhibited by the older adults were exacerbated by the muscle coordination requirements for the more complex contraction combinations. Thus, there is a definite need to determine if the reversal of antagonist technique interferes with the early acquisition of muscular strength through

motor learning in a young healthy population (18-27 years) before examining the technique in an older population.

### **1.1 Statement of the Problem**

The purpose of this study was to determine if the reversal of antagonists technique can result in facilitation of isometric muscle contractions or does it interfere with the acquisition of maximal strength increase through motor learning. The overarching research question being addressed was: what is the relative importance of motor learning and facilitatory techniques to the acquisition of muscle strength?

### **1.2 Experimental Approach to the Problem**

A control group performed agonist only contractions consisting of maximal isometric wrist flexion strength trials. Assessment followed a measurement schedule that has previously been demonstrated to result in increases in maximal strength due solely to motor learning (Calder & Gabriel, 2007; Kroll, 1963a). There were three days of strength testing within a one-week period. Retention and transfer tests were administered two-weeks following the last day of testing. The two-week retention and transfer tests were used to determine if motor learning had occurred (Etnier & Landers, 1998; Katak & Winstein, 2012; Kohl & Gaudagnoli, 1998; Lai & Shea, 1999; Wright & Shea, 2001). It was hypothesized that increases in maximal isometric wrist flexion strength would be due to an increase in flexor carpi radialis (FCR) surface electromyographic (sEMG) activity (Tillin et al., 2011; McGuire et al., 2014) and/or a decrease in extensor carpi radialis (ECR) sEMG coactivity (Carolan & Cafarelli, 1992; Floeter, Danielian, & Kim, 2013; Geertsen, Lundbye-Jensen, & Nielsen, 2008). At the same, alterations in the magnitude

of joint torque and sEMG should be accompanied by reductions in the variability of these measures (McGuire et al., 2014).

The experimental group performed the reversal of antagonists technique, designed to facilitate the wrist flexors during maximal isometric contractions. The contraction pattern involved a wrist extension-to-flexion sequence. The same measurement schedule as the control group was used to evaluate the potential interference of strength acquisition through motor learning (Gabriel & Kroll, 1991). In this case, maximal isometric wrist flexion strength and sEMG activity would be depressed relative to the control group (Gabriel & Kroll, 1991). Maximal isometric wrist flexion joint torque and sEMG activity would also exhibit greater variability (McGuire et al., 2014). Retention or transfer of increases in maximal isometric wrist flexion strength and sEMG activity would also be blunted in comparison to the control group (McGuire et al., 2014). It was equally possible that the reversal of antagonists technique resulted in proprioceptive neuromuscular facilitation: maximal isometric wrist flexion strength and sEMG activity would be greater than that of the control group (Grabiner, 1994; Kamimura et al., 2009). However, torque variability and sEMG variability would not be expected to be any greater than agonist only contractions (Gabriel, Basford, & An, 1997). It was equally possible for either interference or facilitation to occur, however based on review of the previous literature, as well as pilot testing performed, it was hypothesized that the experimental group would display similar trends as the control group. This would involve comparable increases in strength, accompanied by reductions in the variability of the torque and sEMG measures.

In addition to torque and sEMG activity, maximal M-waves, Hoffman (H) reflexes, and V-waves were elicited using peripheral stimulation for all participants. The maximal

M-wave was designed as a control measure and represented the recruitment of the entire motoneuron pool when the median nerve was stimulated using a maximal stimulation.

The H-reflex assessed spinal excitability at rest, while the V-wave represented changes in neural drive to the muscle during the maximal voluntary contractions.

### **1.3 Significance of the Study**

According to the American Heart Association (2012), each year approximately 795, 000 people are affected by a new or recurrent stroke in the United States (US), while an estimated 50,000 Canadians are affected by a new or current stroke (Hakin, Silver, & Hodgson, 1998). Approximately, 315,000 Canadians are living with stroke associated health complications (PHAC, 2011) and each year over 14,000 Canadians die from stroke (Statistics Canada, 2012). Worldwide, 366.93 out of every 100, 000 people have previously suffered from a stroke (Feigin et al., 2013). Stroke has typically been categorized as a disease of the elderly with the majority of people affected by a stroke being over the age of 75 years. Recently this trend has changed, with the age demographics of those suffering from a stroke or living post-stroke decreasing (Feigin et al., 2003). A recent study conducted by Feigin and colleagues (2003) reported that more young to middle-aged (20-64 years) adults were experiencing strokes. In 2010, 31% of those who suffered from a stroke, 43% of those living post-stroke, and 22% of all stroke deaths were young to middle-age adults (Feigin et al., 2003). The broadening demographic only points to a problem that is increasing.

The increase in demand for post-stroke rehabilitation over the past 20 years has been so great that there is a drive towards the development and use of robotic manipulators capable of delivering therapy (Marchal-Crespo & Reinkensmeyer, 2009).

Results developed from this thesis could help guide the type of therapeutic techniques the robotic manipulators are designed to perform. Rehabilitation engineers and clinicians are collaborating on how to integrate the neurophysiological mechanisms underlying facilitation techniques with motor learning principles to optimize functional outcomes of physical therapy (Krakauer, 2006; Lu et al., 2011; Morales et al., 2011). The data generated by this thesis will be infinitely useful in understanding the best approach for the delivery of therapeutic resistive exercise.

#### **1.4 Basic Assumptions**

1. Maximal voluntary contractions represented the upper limit of the participants' maximal strength.
2. Participants did not perform any resistive exercises involving the forearm for the duration of the study.
3. The two-week retention period was sufficient to ensure that hypertrophy and neurophysiological adaptations that occur with long-term resistive exercise had no influence upon the results.
4. Alterations in the magnitude of sEMG activity from the flexor and extensor carpi radialis were indicative of changes in neuromotor control due to motor learning.
5. The flexor and extensor carpi radialis were the main agonist and antagonist, respectively, involved in the task.

#### **1.5 Delimitations**

1. The proposed study only included university aged (18-27years) males who were right-hand dominant.

2. Only one joint, the wrist, was studied.
3. Only the extension-to-flexion contraction pattern was studied
4. This study investigated only isometric contractions.
5. The sEMG activity of only the flexor and extensor carpi radialis was studied.
6. The reversal of antagonists is the only facilitation technique that was studied.

### **1.6 Limitations**

1. Since only university aged (18-27years) males who were right-hand dominant were studied, the results may not apply to left-handed people and/or those of a different age group.
2. Since only the wrist joint was studied, the results may not apply to other joints, or complex actions at multiple joints.
3. Since the extension-to-flexion sequence is the only contraction pattern that was studied, the results may not apply to the flexion-to-extension sequence.
4. Since only isometric contractions were examined, the results may not apply to isotonic or eccentric contractions of the muscle, as occur during normal human movement.
5. Since only the sEMG activity of the FCR and ECR were studied, the role of other forearm muscles during wrist flexion was not investigated.
6. Since the reversal of antagonists is the only facilitatory technique that was studied, the results may not apply to other patterns, such as bilateral reciprocal contractions.

## **1.7 Summary**

Participants in this study performed maximal isometric contractions of the wrist extensors then immediately initiated maximal isometric contractions of the wrist flexors. The contraction pattern that participants performed is termed the “reversal of antagonists” technique and it is an integral part of therapeutic resistive exercise in stroke rehabilitation. It was theorized that proprioceptive feedback generated during wrist extension was used to augment the subsequent wrist flexion contraction. However, the more complex contraction pattern may have interfered with normal learning-related increases in strength through skill development. The continued use of the reversal of antagonists contraction pattern in rehabilitation, despite rather disquieting findings in the extant literature, was used to justify the study.

## CHAPTER 2: LITERATURE REVIEW

Participants in this study performed maximal isometric contractions of the wrist extensors then immediately initiated maximal isometric contractions of the wrist flexors. It is theorized that proprioceptive feedback generated during wrist extension is used to augment the subsequent wrist flexion contraction (Kabat, 1952; Kabat & Knott, 1948; 1953; Moore & Kukulka, 1991). However, the more complex contraction pattern may interfere with motor learning-related increases in strength through skill development (Gabriel, Basford, & An, 1997; Gabriel & Kroll, 1991). This literature review will cover topics related to the biomechanics of the wrist musculature, the sensorimotor system relative to proprioceptive feedback that facilitates and inhibits muscle contractions, and increases in strength through motor learning.

### **2.1 Anatomy of the Flexor Carpi Radialis and Extensor Carpi Radialis**

The flexor carpi radialis (FCR) muscle (Figure 1) is a pennate muscle located on the anterior aspect of the forearm, when in anatomical position. Originating on the medial epicondyle of the humerus, the FCR runs lateral to the flexor digitorum superficialis and inserts on the anterior aspect of the base of the second and third metacarpal bones (Martini & Nath, 2009). The primary functions of the FCR are to flex the wrist and act as a synergistic muscle with the extensor carpi radialis (ECR) during radial deviation (Bawa, Chalmers, Jones, Sjøgaard, & Walsh, 2000; Boles, Kannam, & Cardwell, 2000). The ECR is a muscle that acts as an antagonist muscle during wrist flexion. It originates on the lateral epicondyle of the humerus, runs laterally down the posterior aspect of the forearm, when in anatomical position, and inserts onto the lateral dorsal surface of the base of the third metacarpal bone (Figure 1) (Martini & Nath, 2009). The FCR is

innervated by the median nerve, which originates from the brachial plexus and is the only nerve to pass through the carpal tunnel, while the ECR is innervated by the radial nerve (Boles et al., 2000).

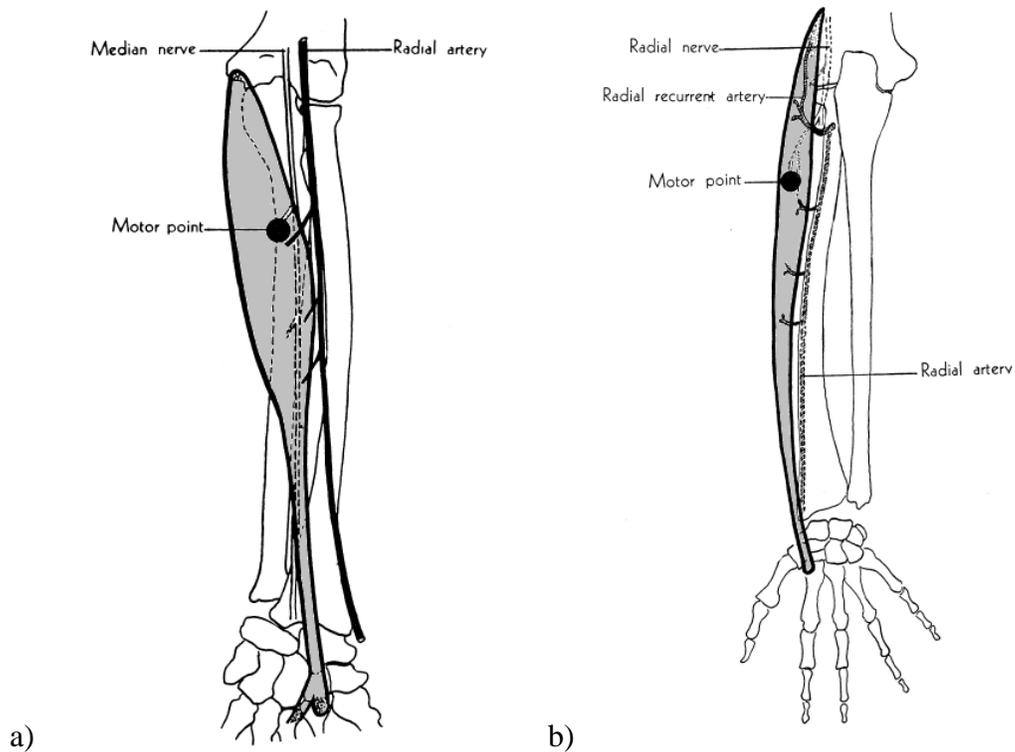


Figure 1. Anatomy of the forearm muscles with nerve innervation. a) Flexor carpi radialis muscle with median nerve innervation. b) Extensor carpi radialis with radial nerve innervation. Quiring, D.P. & Warfel, J.H. (1960). *The extremities*. Philadelphia: Lea and Febiger. Pages 32, 40.

The muscle fibres of the FCR originate from the superficial surface of a deep tendon that is shared with the pronator teres, flexor digitorum superficialis and, when present, the palmaris longus muscles (Segal, Wolf, DeCamp, Chopp, & English, 1991). The muscle fibre length of the FCR muscle is approximately  $59.8 \pm 1.5$  mm (Gonzalez, Buchanan, & Delp, 1997; Loren et al., 1996). Segal and colleagues (1991) determined that fibres inserting along the midline of the tendon are longitudinally oriented, whereas fibres that insert along the sides of the tendon are at oblique angles. The same authors also examined the median nerve, which innervates the FCR. They noted that the nerve split into two divisions: the distal division, which supplies the medial oblique muscle fibres and the proximal division, which has two branches. The medial branch of the proximal division innervates the longitudinal fibres, while the lateral branch innervates the lateral oblique muscle fibres (Segal et al., 1991).

Previous research has modeled the wrist flexor and extensor muscles and predicted that the FCR muscle performs optimally (i.e., generates peak muscle force) when the wrist is in full wrist extension (Loren et al., 1996). Loren and colleagues (1996) anticipated that when the FCR was at its longest muscle length, hence while the wrist is in full extension, it would be able to generate maximal muscle force. It was predicted that peak muscle force generated by the FCR muscle ranged from 51.2 N to 60 N (Gonzalez et al., 1997; Loren et al., 1996). It was also speculated that the flexor moment arms were the greatest when the wrist was flexed and they would decrease as the wrist moved into extension (Loren et al., 1996). Loren and colleagues (1996), as well as, Gonzalez and colleagues (1997) examined the wrist flexor and extensor muscles for their biomechanical characteristics. It was determined that, when moving from flexion to

extension the maximal moment arm of the FCR ranged from 16 mm to 17.3 mm in length and this length was reached at an angle of approximately 40° (Gonzalez et al., 1997; Loren et al., 1996). Gonzalez and colleagues (1997) also examined the physiological cross sectional area (PCSA) of the muscles and determined that the PCSA of the FCR muscle was 20 mm<sup>2</sup>.

Loren and colleagues (1996) and Gonzalez and colleagues (1997) also examined the wrist extensors while defining the biomechanical characteristics of the wrist muscles. Both studies divided the ECR into its two components: the extensor carpi radialis brevis (ECRB) and the extensor carpi radialis longus (ECRL). It was determined that the ECRB has a fibre length ranging from 59 mm to 70.8 ± 1.7 mm, while the ECRL fibre length ranges from 94 mm to 127.3 ± 5.6 mm. (Gonzalez et al., 1997; Loren et al., 1996). The moment arms of the ECRB and ECRL were approximately 16mm and 10mm, respectively, with the PCSAs of the two muscles roughly 27mm and 15mm, respectively (Gonzalez et al., 1997).

## **2.2 Examining Neural Activity**

### ***2.2.1 Spinal and Supraspinal Control of Motoneuron Excitability***

The motoneuron is an integral part of the motor unit, which is the functional unit of the neuromuscular system (Kabat, 1952). One motor unit is comprised of a single alpha motoneuron and all the muscle fibres that it innervates (Figure 2). Motoneurons are responsible for the excitation and/or inhibition of the muscle they are associated with. The excitability of a motoneuron is dependent on the total sum of the excitatory and inhibitory activity within the synaptic clefts (Åstrand & Rodahl, 1986). Motoneurons are controlled at two different levels: spinal and supraspinal. At the supraspinal level,

motoneurons are controlled by areas in the brain such as the cerebellum, cerebral cortex, and various nuclei (Bawa, 2002). At the spinal level, Renshaw cells, muscle spindle complexes, Golgi tendon organs, joint receptors, and cutaneous receptors control the excitability of motoneurons. Figure 3 depicts the various spinal and supraspinal mechanisms that affect the excitability of an alpha motoneuron. The following subsections review ways in which the sensorimotor system facilitates and inhibits muscle contractions at both levels.

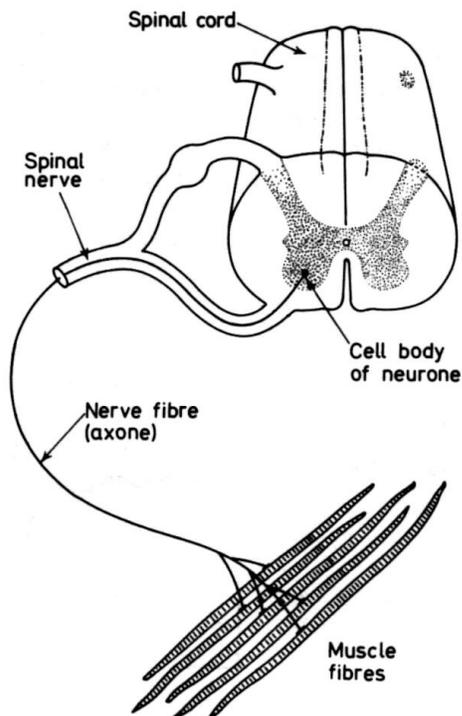


Figure 2. A motor unit consisting of an alpha motoneuron and all of the muscle fibres it innervates. Basmajian, J.V., & DeLuca, C.J. (1985). *Muscles alive: their functions revealed by electromyography*. Baltimore: Williams and Wilkins. Figure 1-7, p.12.

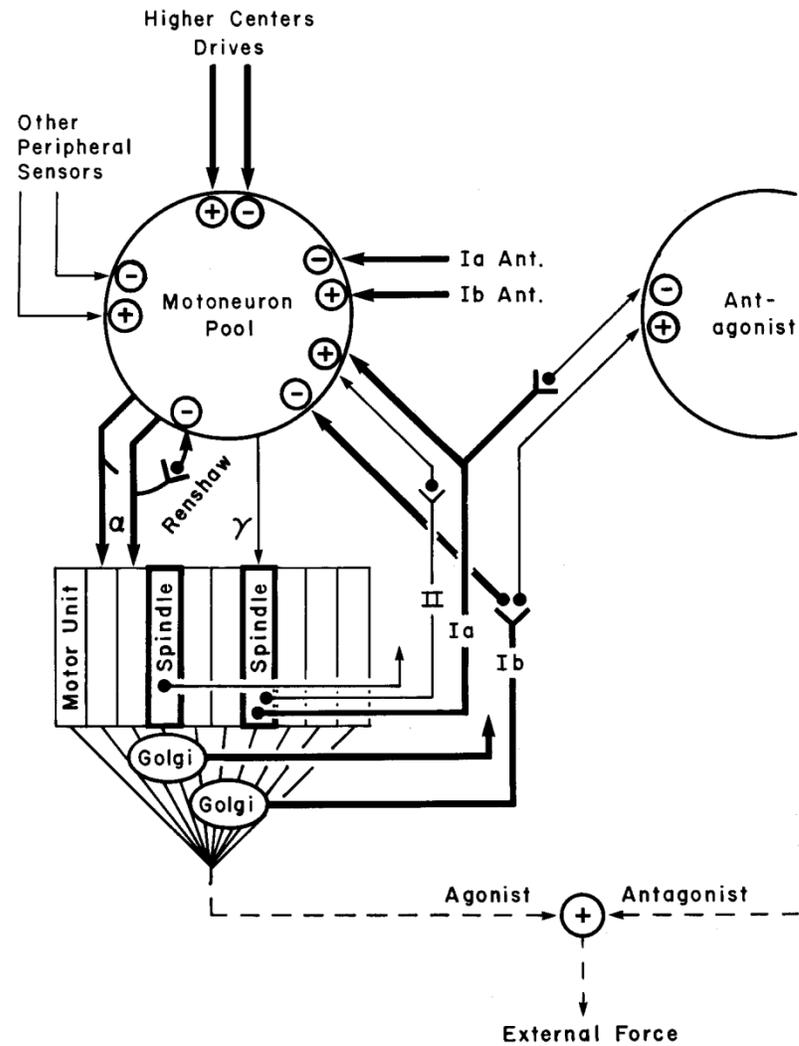


Figure 3. An alpha motoneuron and the spinal and supraspinal mechanisms that affect its excitability. Basmajian, J.V. & DeLuca, C.J. (1985). *Muscles alive: their functions revealed by electromyography*. (5<sup>th</sup> ed.). Baltimore, MD: Williams & Wilkins. Figure 5.1, page 126.

### *2.2.1.1 Spinal Control*

Therapeutic resistive exercise techniques attempt to activate sensorimotor receptors to facilitate muscle activity when there is a deficit in central nervous system (CNS) drive to the muscle (Kabat, 1952; Kabat & Knott, 1948; 1953). The type of sensory receptor activated depends on the particular technique being performed. This does not mean that a particular sensory receptor is activated in isolation. Rather, the goal is to manipulate the contraction so that it dominates the net sensory input resulting in either facilitation or inhibition (Kabat, 1952). Other receptors still contribute to the overall response. The following subsections review sensorimotor receptors that may impact the strength of an isometric contraction.

#### *Renshaw Cells*

As motoneurons traverse the spinal cord, they give off collateral branches that form excitatory synaptic connections with interneurons that are located within the ventromedial region of the spinal cord (Åstrand & Rodahl, 1986; Renshaw, 1941). These interneurons are known as Renshaw cells. Renshaw cells have inhibitory connections with the same or other alpha motoneurons and interneurons of that segmental level (Åstrand & Rodahl, 1986). Figure 4 outlines how a Renshaw cell affects motoneuron excitability.

The Ia afferent enters through the dorsal root ganglion of the spinal cord and forms an excitatory connection with the alpha motoneuron innervating the agonist muscle and an inhibitory neuron that is in connection with the alpha motoneuron innervating the antagonist muscle (Åstrand & Rodahl, 1986). Arising from the motoneuron of the agonist muscle is a recurrent collateral branch that forms an excitatory connection with a

Renshaw cell (Åstrand & Rodahl, 1986). This Renshaw cell then forms inhibitory connections with the motoneuron of the agonist and with the Ia inhibitory interneuron of the antagonist. When the Ia afferent is stimulated, the motoneuron of the agonist muscle will be stimulated causing the agonist muscle to contract and the inhibitory interneuron will be stimulated which will inhibit the antagonist muscle (Åstrand & Rodahl, 1986). As well, the collateral branch may also excite the Renshaw cells, which will quiet the activity of the agonist and the inhibitory interneuron, allowing for more activity of the antagonist (Åstrand & Rodahl, 1986). While this reflex loop does quiet the activity of the agonist muscle, it can be beneficial because it provides a mechanism to protect against overloading of the muscle as the recurrent inhibition produced by the Renshaw cells acts to limit and stabilize motoneuron firing rates (Åstrand & Rodahl, 1986; Windhorst, 1996).

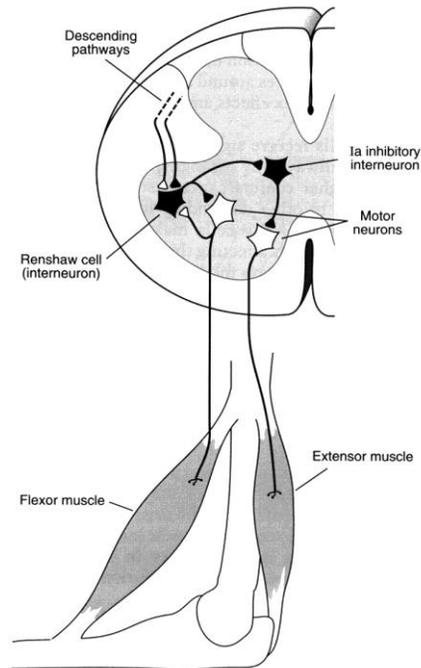


Figure 4. The pathway of a Renshaw cell. Kandel, E.R., Schwartz, J.H., & Jessell, T.M. (2000). *Principles of neural science (4<sup>th</sup> Ed.)*. New York: McGraw-Hill. Figure 36-5b, page 722.

### *Muscle Spindles*

Motoneuron excitability is also controlled by muscle spindle complexes. Muscle spindles are stretch receptors that relay information to the spinal cord and brain regarding the current muscle length and changes in muscle length (Kandel, Schwartz, & Jessell, 2000; Macefield, 2005; Proske, 1997). These small, elongated structures are anchored in parallel to the contractile extrafusal muscle fibres (Emonet-Dénand, Hunt, & Laporte, 1988; Kandel et al., 2000; Proske, 1997), therefore when a movement occurs that increases muscle length, the muscle spindle also stretches (Macefield, 2005). Edin and

Vallbo (1990) examined various muscle afferents and their responses when participants performed a slowly increasing isometric contraction, with a period of steadiness followed by a quick relaxation. The authors found that in the majority of muscle spindles examined, the discharge rates increased at the beginning of the contraction and then plateaued early while torque continued to increase (Edin & Vallbo, 1990). It was also noted that when subjects relaxed, the muscle spindles that increased in discharge rate displayed one of two reactions. They either displayed a short-lasting burst of accelerated discharge or a prompt cessation of the discharge when the tension began to fall. These results demonstrate that muscle spindles are also active during static contractions or contractions where no physical movement of the limb occurs.

Muscle spindles consist of a connective tissue capsule that contains a group of muscle fibres termed intrafusal fibres (Emonet-Dénand et al., 1988; Lephart, Pincivero, Giraldo, & Fu, 1997; Proske, 1997). The ends of the spindle are contractile, while the central regions lack myofibrils and are non-contractile (Silverthorn, 2009). The contractile ends of the spindle are innervated by gamma motoneurons, and the central regions are wrapped by sensory nerve endings that are stimulated by muscle stretch (Emonet-Dénand et al., 1988; Lephart, Pincivero, Giraldo, & Fu, 1997; Proske, 1997). There are two types of intrafusal fibres within a muscle spindle: nuclear bag fibres and nuclear chain fibres (Emonet-Dénand et al., 1988). Nuclear bag fibres are relatively long (6-10mm) and thick (20µm in diameter), while nuclear chain fibres tend to be shorter (3-4mm) and thinner (12µm in diameter) (Emonet-Dénand et al., 1988; Proske, 1997). Bag fibres can be further subdivided into two types of fibres: bag<sub>1</sub> and bag<sub>2</sub> (Emonet-Dénand et al., 1988; Proske, 1997). The arrangement of a muscle spindle can be seen in Figure 5.

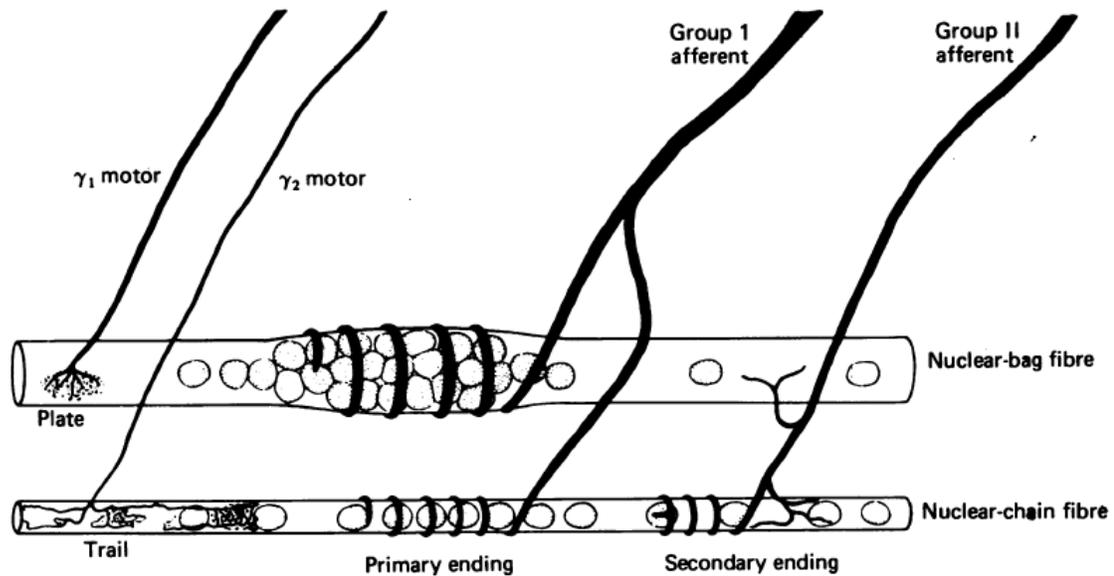


Figure 5. A simplified diagram of a muscle spindle being innervated by type Ia and II afferents, as well as gamma innervation. Matthews, P.B.C. (1981). Evolving view on the internal operation and functional role of the muscle spindle. *Journal of Physiology*, 320: 1-30. Figure 1, page 4.

The muscle spindle relays information to the alpha motoneuron via the Ia afferent and gamma pathways (Kandel et al., 2000). The large Ia afferent (primary afferent endings) enters the capsule of the muscle spindle and branches repeatedly with each unmyelinated terminal region wrapping around the nucleated portion of each intrafusal fibre (Macefield, 2005; Proske, 1997). With the Ia afferent, a smaller group II fibre (secondary afferent endings) also enters the muscle spindle capsule and has branches that terminate on primarily bag<sub>2</sub> and chain fibres (Macefield, 2005; Proske, 1997). While both afferent endings act as stretch receptors, the primary afferent endings have a higher

dynamic sensitivity (Macefield, 2005), which is usually ascribed to the bag<sub>1</sub> fibres and their terminals (Emont-Dénand et al., 1988). This means that the primary endings are more sensitive to smaller changes in length than the secondary endings (Fallon & Macefield, 2007). Secondary afferent endings (Group II fibres) are normally responsible for signaling absolute muscle length (Fallon & Macefield, 2005). The intrafusal fibres of the muscle spindles also receive motor innervation from either gamma ( $\gamma$ ) or fusimotor neurons (Macefield, 2005; Proske, 1997). It is generally agreed upon that bag<sub>1</sub> fibres are innervated by dynamic ( $\gamma_D$ ) axons, while static ( $\gamma_S$ ) innervation is slightly disagreed upon (Proske, 1997). Although it has been suggested that  $\gamma_S$ -axons tend to predominately innervate bag<sub>2</sub> and chain fibres (Proske, 1997).

While at rest, the central region of the spindle is stretched enough to activate the sensory fibres, thus creating a tonic activation of the muscle (Harris, 1984; Silverthorn, 2009). The gamma motoneurons play a crucial role in adjusting the sensitivity of the muscle spindle so it is always active no matter the length of the muscle (Silverthorn, 2009). When a gamma motoneuron fires, the ends of the fibres contract and shorten which lengthens the central region and maintains the stretch on the sensory neurons (Silverthorn, 2009). Gamma motoneurons are typically activated at the same time that the alpha motoneurons of the muscle fire (Lephart et al., 1997). This coactivation occurs to prevent the central region from becoming too relaxed and losing the tonic activation that occurs in the muscle (Lephart et al., 1997).

### *Golgi Tendon Organs*

A third mechanism controlling motoneuron excitability are the Golgi tendon organs (GTOs). Golgi tendon organs are found at the junction of tendons and muscles

and are placed in series with muscle fibres (Kandel et al., 2000; Macefield, 2005). These proprioceptors respond primarily to the tension a muscle develops during an isometric contraction and cause a relaxation reflex (Gregory & Proske, 1979; Guissard & Duchateau, 2006; Lephart et al., 1997; Schoultz & Swett, 1972). During stretching, it has been noted that GTOs reduce motor neuron excitability and they are known to be more responsive to the force of a contraction, rather than to the mechanical tension of a passive stretch (Fallon & Macefield, 2007; Guissard & Duchateau, 2006; Macefield, 2005). Edin and Vallbo (1990) studied various muscle afferents and their responses to isometric contractions. The authors demonstrated that during isometric contractions, GTOs produced a sustained increase of impulse rate and that the discharge rate was closely related to the active torque produced (Edin & Vallbo, 1990). Guissard and Duchateau (2006) also discussed GTOs and stated that GTOs appear to only be activated during large-amplitude stretching and that they are a contributor to the postsynaptic inhibition of the motor neuron pool that is occurring during the large-amplitude stretches.

Golgi tendon organs are innervated by a single group Ib axon, which enters the capsule of the GTO and branches into many unmyelinated endings that wrap around and between collagen fibres (Figure 6) (Fallon & Macefield, 2007; Kandel et al., 2000). During an isometric contraction, the tendon of a muscle acts as an elastic component, which pulls the collagen fibres within the GTO tight, thus pinching the sensory endings of the Ib axon, increasing its firing rate (Kandel et al., 2000; Silverthorn, 2009). The increased firing rate of the Ib axon will excite the Ib inhibitory interneuron within the spinal cord, thus causing inhibition of the alpha motoneurons innervating the muscle, resulting in a decreased contraction level or ending the contraction altogether

(Silverthorn, 2009). Essentially, the GTOs are in place as a mechanism to prevent excessive contraction that may injure the muscle (Lephart et al., 1997).

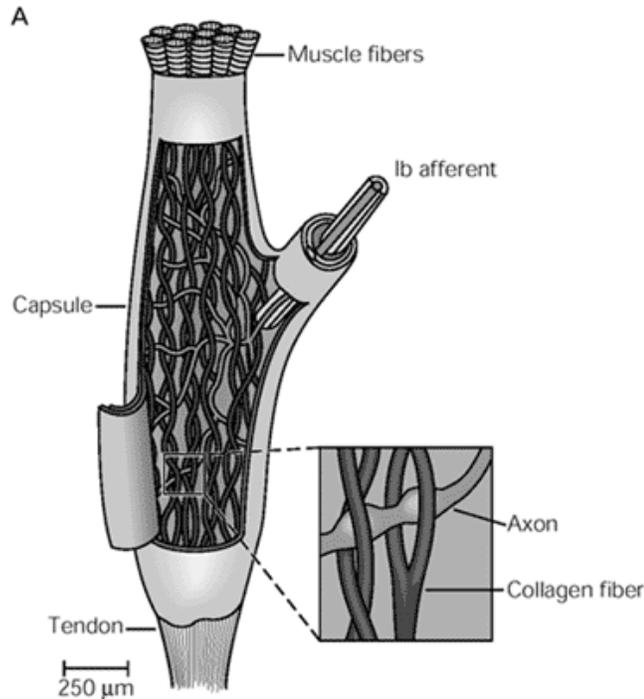


Figure 6. Anatomy of a Golgi tendon organ. Kandel, E.R., Schwartz, J.H., & Jessell, T.M. (1995). *Essentials of neural science and behaviour*. Stanford, Connecticut: Appleton and Lange. Figure 27-6c, page 507.

### *Joint and Cutaneous Receptors*

Joint and cutaneous receptors are generally ignored when considering muscle force output during maximal voluntary contractions. During a fast maximal voluntary isometric contraction there is still joint compression (Amis, Dowson, & Wright, 1980)

and indentation of the skin of the limb secured within the strength testing device (Johansson & Westling, 1984), in addition to muscle and tendon lengthening (Maganaris & Baltzopoulos, 1999). Moreover, since these receptors have been shown to adapt with gradation of muscle force (Ashby, Hilton-Brown, & Stålberg, 1986), it is important to acknowledge their potential contribution (Johansson, 1991; Solomonow, 2006; Stubbs et al., 1998; Zimny & Wink, 1991).

Joint receptors are mechanoreceptors located at joints that recognize force applied across a joint (Figure 7) (Macefield, 2005). These receptors are primarily responsible for relaying information regarding the movement of a joint, acting as joint limit detectors, and acting as joint pain receptors or nociceptors (Macefield, 2005; Proske et al., 1988). Proske and colleagues (1988) review evidence that indicates that joint receptors most likely do not play a role in recognizing joint position, except perhaps towards the extreme ranges of motion of the joint. Joint receptors form a negative feedback loop with the Ib inhibitory interneuron (Kandel, Shwartz, & Jessell, 1995). When activated, the joint receptor will send signals to the Ib inhibitory interneuron, which will inhibit the agonist muscle, thus preventing the joint from moving outside of its normal range of motion (Kandel et al., 1995).

Cutaneous receptors are found within the layers of skin (Figure 7) (Kandel et al., 1995). These receptors are primarily responsible for detecting movement of the skin, movement of the hair on the skin, or injury to the skin's surface (Kandel et al., 1995; Macefield, 2005; Sulka & Rees, 1997). According to Macefield (2005) there are four different kinds of cutaneous afferents found in glabrous skin (hairless) and five classes from the hairy parts of skin. The four afferents found in glabrous skin are categorized into

fast-adapting (FA) and slow-adapting (SA) (Macefield, 2005). Within each category there is a type I (FAI and SAI) and type II (FAII and SAII) afferent. FAI afferents are generally activated by discrete stimuli, in a small-well defined area and are particularly sensitive to light stroking across the skin. While, FAII afferents are sensitive to brisk mechanical transients such as vigorous blowing across the skin or tapping across the area (Macefield, 2005). SAI afferents have high dynamic sensitivity to indentation of the skin, whereas SAII afferents respond to forces applied normally to the skin, as well as laterally stretching of the skin. Within the hairy parts of the skin, two of the afferents found are hair units and field units (Macefield, 2005). Hair units respond to movements of the individual hairs or air puffs over the skin, whereas field units respond to actual skin contact. The intensity of the applied stimulus, will determine whether just the affected area or muscle contracts or the entire limb contracts to move away from the stimulus (Kandel et al., 1995).

In addition to detecting movement associated with the skin or hairs on the skin, cutaneous receptors are also responsible for detecting pain or injury to the skin's surface (Sluka & Rees, 1997). If the receptors detect a painful stimulus, there is a resulting increase in the primary afferents supplying the region the stimulus has been applied (Sluka & Rees, 1997). The cutaneous receptors that detect painful stimuli are called nociceptors, which are unencapsulated receptors that convey information to the CNS via two types of afferents: A-delta afferents and C-fibres (Sluka & Rees, 1997). A-delta afferents are thinly, myelinated axons that when stimulated a pricking pain is felt, whereas C-fibres are unmyelinated axons that produce a burning pain in addition to a tingling or tapping sensation when stimulated (Ochoa & Torebjörk, 1983). Once a

noxious stimulus has been detected on the skin's surface, nociceptors are activated, increasing the activity of all cutaneous afferents, as well as the input to the dorsal horn of the spinal cord (Sluka & Rees, 1997). The increase activity to the dorsal horn results in increased excitability of the central neurons within the spinal cord (Sluka & Rees, 1997), which will result in contraction of the affected area or muscle, or even contraction of the entire limb segment (Kandel et al., 1995).

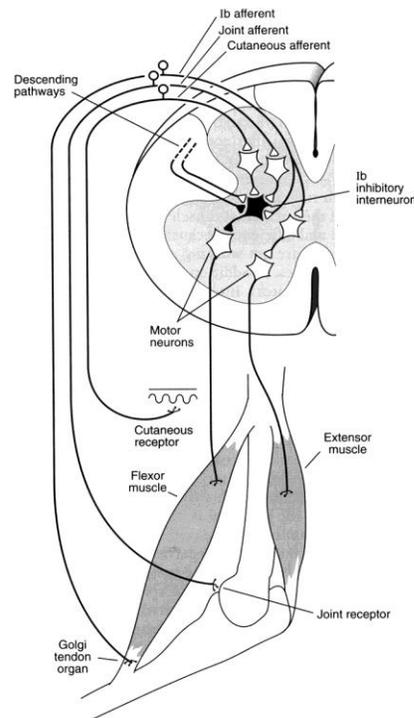


Figure 7. Summary of the receptors and afferents involved in the spinal control of motoneuron excitability. Kandel, E.R., Schwartz, J.H., & Jessell, T.M. (2000). *Principles of neural science* (4<sup>th</sup> Ed.). New York: McGraw-Hill. Figure 36-7a, page 724.

### *2.2.1.2 Supraspinal Control*

This subsection will focus on CNS control of the agonist-antagonist alpha motoneuron pool. Lamarre and colleagues (1978) demonstrated that after complete upper limb deafferentation, the inhibition of the antagonist muscle continued to precede the activation of the agonist muscle. It was concluded that these results proved that the stimulus creating the inhibitory reflex seen in the antagonist originated in the CNS, not from muscle spindles or Golgi tendon organs in the contracting agonist muscle. In agreement, Humphrey and Reed (1983) examined how monkeys learned to control the position of the wrist and demonstrated the existence of two motor control systems that are partially independent of one another. The first system related to the reciprocal activation of the antagonist muscles and the second was organized for the coactivation of the antagonist muscles (Humphrey & Reed, 1983).

Frysinger and colleagues (1984) trained monkeys to perform two different contraction patterns, one that produced reciprocal activation of the antagonist, while the other required coactivation of the antagonist. The authors demonstrated that cerebellar Purkinje fibres play a role in reciprocal activation and coactivation of the antagonist muscles, selecting neural pathways that alternate between the two types of contractions (Frysinger et al., 1984). Similarly, Bourbonnais and colleagues (1986) demonstrated that discharges within the monkey cerebellar cortex increase and are tightly correlated with velocity of stretch and muscle length, suggesting that the cerebellar cortex plays a role in monitoring muscle changes (Bourbonnais et al., 1986).

De Luca and Mambrito (1987) examined myoelectric (ME) activity of various motor units from the thumb muscles in humans. The authors reported findings that extend

the primate work of Humphrey and Reed (1983) who suggested that there were two basic command systems: reciprocal innervation and coactivation of antagonist muscles. De Luca and Mambrito (1987) suggested that the corticomotoneuronal and rubromotoneuronal cells have connections to motor units in both the flexor and extensor muscle groups. These connections exert a “common drive” to both muscle groups at the same time to execute one of three of motor plans: (1) the extend command which inhibits the flexor motoneuron pool, (2) the flex command which inhibits the extensor motoneuron pool, and (3) the coactivate command activates both flexor and extensor motoneuron pools. Common drive was demonstrated in the firing rates of motor units of the agonist and antagonist muscles controlling the thumb during voluntary isometric contractions. The firing rates of agonist and antagonist muscles were shown to be highly correlated during flexion, extension, and coactivation tasks, suggesting that the nervous system controls the motoneuron pools in a uniform fashion, not individual motor units (De Luca & Mambrito, 1987).

### ***2.2.2 The Neurophysiological Basis of PNF***

The strength of a muscle contraction depends on the number of motor units activated, which is ultimately a function of the level of excitation within the anterior horn. Proprioceptive neuromuscular facilitation techniques are designed to activate the segmental reflex system during voluntary muscle contractions (Kabat, 1952). It is theorized that proprioceptors can be used to provide facilitatory feedback to the anterior horn to augment weak CNS drive of the motoneuron pool in patient populations (Kabat, 1952; Kabat and Knott, 1948; 1953). The central and peripheral motor system can then be combined to recruit additional motor units and increase the strength of muscle

contractions. The mechanism for sensorimotor integration of neural inputs is the subliminal fringe anterior horn cell (Harris, 1984).

Figure 8 illustrates that subliminal fringe anterior horn cells are high threshold motoneurons that receive only a small number of synaptic connections from afferent terminals (Decker, 1962; Guyton, 1976; Ruch et al., 1963). When afferent fibres enter the dorsal root of the spinal cord they break into many branches, which form synaptic connections with many postsynaptic cells (Decker, 1962; Guyton, 1976; Ruch et al., 1963). If the “right” number of branches synapse onto an individual motoneuron and are all activated at the same time, the motoneuron will reach threshold and send action potentials down the efferent fibre connected to it (Denny-Brown & Sherrington, 1928; Lloyd, 1945; Sherrington, 1931). In some cases, the afferent fibre only makes a few connections with a motoneuron, and therefore when stimulated does not excite that motoneuron, but may facilitate it by lowering its recruitment threshold. Motoneurons of this kind are said to be in the “subliminal fringe” and are only at a “facilitated level” of excitation (Lloyd, 1945; Sherrington, 1931). A subliminal fringe motoneuron may also receive synaptic connections from neighboring afferent fibres. If the direct afferent connections and enough neighboring afferent connections are active at the same time, the motoneuron can receive a sufficient amount of stimulation to bring it past a facilitated level into full excitation (Denny-Brown & Sherrington, 1928; Lloyd, 1945).

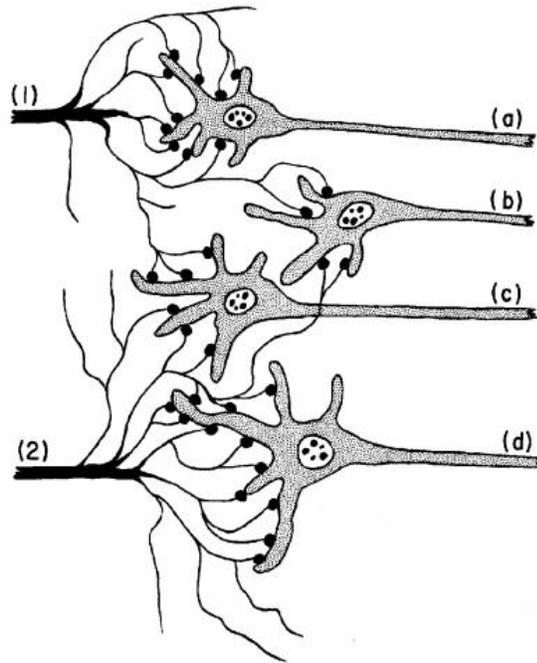


Figure 8. An example of the subliminal fringe. Diagram depicts two afferent fibres synapsing onto two motoneurons with collateral branches from each fibre synapsing onto a motoneuron in the subliminal fringe. Guyton, A.C. (1976). *Textbook of medicine physiology*. Philadelphia, PA: W.B. Saunders Company. Figure 47-4, page 629.

### *2.2.2.1 Static Contractions*

During maximal isometric contractions, it is theorized that autogenic inhibition due to the GTOs is the main inherent reflex that dominates the voluntary response (Laporte & Lloyd, 1952; Sharman, Cresswell, & Riek, 2006). While previous subsections have described how other proprioceptors may be involved, together the sum total is one of inhibition of the agonist as a protective mechanism. In support, Etnyre and Kinugasa (2002) and Moore and Kukulka (1991) have reported depression in the excitability of the agonist motoneuron pool following isometric contractions. It is therefore assumed that the motoneuron pool of the antagonist is in a facilitated state (Kabat, 1952; Morris & Sharpe, 1993). If at this moment, the force direction changes so that the antagonist is used as an agonist, the voluntary neural drive combines with the facilitating proprioceptive feedback to bring subliminal fringe anterior horns to threshold and increase the strength of the muscle contraction. However, concurrent proprioceptive facilitation has never been demonstrated in a definitive way. Rather, it has been inferred from the surface electromyogram and investigation of coactivity levels (Bazzucchi et al., 2006; Solomonow et al., 1988). It is also very difficult to separate segmental and supraspinal control of antagonist coactivation (Geertsen, Jensen, & Nielsen, 2008).

## **2.3 Resistance Training**

### ***2.3.1 Neuro-motor Aspects of Strength***

#### *2.3.1.1 Muscle Coordination*

It is well documented that during the early phases of strength training there is a marked increase in strength that occurs in the absence of muscle hypertrophy (Duchateau

& Enoka, 2002; Gabriel, Kamen, & Frost 2006). In 1963(a), Kroll demonstrated an increase in strength after three consecutive days of testing, and termed this the “quick jumps in strength phenomenon”. The strength of the participants continued to increase over the 3-week and 3-month retention periods without any resistive exercise in between test sessions. Since the time-interval between retention tests would allow for the dissipation of any physiological adaptations (i.e., detraining), it has been argued that neural and/or learning mechanisms are responsible for the early strength gains. Kroll (1974) later hypothesized that the quick jump in strength phenomenon may be due to improved neuromotor coordination control mechanisms involving the agonist and antagonist muscle groups.

Since the early work of Kroll (1963a; 1974) a number of investigators have continued to examine the role motor learning may play during the early phases of learning. Smith (1974) studied two different contraction schedules (massed versus distributed) and determined that contraction schedule did not play a role in strength acquisition. However, he did conclude that the strength-learning curves were quite similar to motor-learning curves typically seen during skill acquisition. More recent work on learning-related increases in strength has shown that there is a reduction in antagonist coactivation (Carolan & Cafarelli, 1992) and/or an increase in agonist muscle activation (Calder & Gabriel, 2007). In a companion study, McGuire and colleagues (2014) have demonstrated that early increases in strength are associated with alternating decreases and increases in antagonistic coactivity as the nervous system learns to balance competing factors related force output and joint stability. For example, it is possible to increase maximal muscle force by a reduction in antagonist coactivation (Carolan & Cafferelli

1992). However, if there is an increase in force output directly from agonist muscle itself, then a compensatory increase in antagonist coactivation would be required to provide joint stability (Bazzucchi et al. 2006; Solomonow et al. 1988).

### *2.3.1.2 Muscle Activation*

In addition to alterations in agonist-antagonist coordination, resistive exercise results in neural adaptations that increase muscle activation. The increase in neural drive to the muscles arise from both spinal and supraspinal control centers and it is very difficult to disentangle adaptations in the two (Geertsen, Jensen, & Nielsen, 2008). Furthermore, the time-course of these neural adaptations is uncertain as they are generally assessed in response to a progressive resistive exercise (PRE) program lasting six-weeks or longer, when the tasks are well-learned (Duchateau & Enoka, 2002; Gabriel, Kamen, & Frost, 2006).

The V-wave is typically used to measure the level of central drive from the excited motoneuron pool during a maximal contraction. A supramaximal stimulus is applied to the peripheral nerve as would occur during an interpolated twitch to produce an M-wave in the middle of the contraction. The descending drive to the muscle counteracts the antidromic propagation of action potentials produced by stimulation of the peripheral nerve, which causes collision within the sensorimotor reflex loop associated with the H-reflex. The reappearance of the H-reflex during the maximal voluntary contraction is termed the V-wave (Aagaard et al., 2002; Sale et al., 1983), and its magnitude is proportional to the amount of central drive to the muscle (El Bouse, Gabriel, & Tokuno, 2012). It is commonly said that H-reflex “piggy backs” off of the descending drive to the muscle. In contrast, when the H-reflex is elicited without a

voluntary contraction, it is thought to reflect alterations in spinal excitability while acknowledging the potential for presynaptic inhibition (Misiaszek, 2003; Zher, 2002).

Progressive resistive exercise (PRE) has been documented to increase V-wave amplitude, which has been interpreted as an increase in voluntary activation of the muscle (Aagaard et al., 2002; Sale et al., 1983; Vila-Chã et al., 2012). In 1983, Sale and colleagues examined the effects of weight training on two forms of the V-wave ( $V_1$  and  $V_2$ ). Both forms of the V-wave were observed to be potentiated during maximal voluntary contractions. Aagaard et al. (2002) and Vila-Chã et al. (2012) used the V-wave, M-wave, and H-reflex to isolate spinal and supraspinal adaptations associated with PRE. Aagaard and colleagues (2002) examined a 14-week resistive exercise program for the leg muscles. During a ramp increase in isometric force from 0 to 100% MVC, H-reflex amplitude increased 20% while the V-wave increased by 50%. However, resting H-reflex and  $M_{max}$  remained unchanged. These adaptations were associated with a 23 to 50% increase in MVC with training. Similarly, Vila-Chã and colleagues (2012) found that after a 3-week heavy strength training program, there was a significant increase  $V/M_{max}$ , whereas the resting  $H_{max}/M_{max}$  ratio remained unchanged.

Studies utilizing the V-wave are consistent with transcutaneous magnetic stimulation (TMS) of the motor cortex following resistive exercise. Griffin and Cafarelli (2007) showed that training-related increases in sEMG were associated with an increase in the magnitude of motor evoked potentials from the exercised muscle but the M-wave remained unchanged. Taken together, the findings presented, thus far, show that training-related increases in muscle activation may arise from both spinal and supraspinal sources.

Changes in muscle activation ultimately affect motor unit activity patterns. A number of authors have demonstrated that training-related early increases in strength following resistive exercise are caused by an increase in motor unit discharge rate (Christie & Kamen, 2010; Kamen & Knight, 2004; Leong et al., 1999; Patten, Kamen, & Rowland, 2001). Age-related differences in motor unit discharge rate following resistive exercise have also been examined. Similar to younger adults, older adults who engage in resistive exercise training have higher motor unit discharge rates than those who are not active (Leong et al., 1999). However, older adults exhibit lower motor unit discharge rates than younger adults during maximal isometric contractions, suggesting that older adults may have an impaired ability to fully recruit the surviving motor units (Clarke & Fielding, 2012; Connelly et al., 1999; Kamen, Sison, Du, & Patten, 1995; Patten & Kamen, 2000). Evidence to the contrary was recently reported by Christie and Kamen (2010), who reported no differences between young and older adults with respect to the central activation ratio, as assessed by supramaximal 50Hz stimulation of the peripheral nerve during maximal voluntary contractions. This technique is however not without its methodological challenges that affect its interpretation (Behm, Power, & Drinkwater, 2001).

Motor unit synchronization is described as multiple motor units firing simultaneously or near-simultaneously more often than normal (Kamen & Roy, 2000). In 1975, Milner-Brown and colleagues examined motor unit synchronization in weight-lifters and concluded that motor unit synchronization was found to be greater in the weight-lifters than in the control group. Since then, the techniques used to examine motor unit synchronization have undergone re-evaluation (Kamen & Roy, 2000; Semmler &

Nordstrom, 1999). Fling and colleagues (2009) reported that, while their experimental evidence was a “bit equivocal”, synchronization levels tend to be greater at higher force levels (80% MVC) and in strength-trained than untrained individuals. Furthermore, motor unit synchronization levels are similar among young and older adults (Kamen & Roy, 2000).

Motor units have been shown to fire at relatively consistent intervals regardless of the firing rate, whereas doublet firing is a phenomenon seen when a motor unit fires twice in a very short interval of time (Garland & Griffin, 1999). This phenomenon has been demonstrated to be prevalent during the onset of a muscle contraction (Van Cutsem et al., 1998). Van Cutsem and colleagues (1998) showed that resistive exercise not only increased maximum isometric strength but there also was an increase in both the maximal rate force development and in the frequency of doublets.

### ***2.3.2 Reversal of Antagonists Technique***

Herman Kabat (1952) developed a series of therapeutic resistive exercise techniques based on the work of Sherrington (1906). Sherrington (1906) described a series of experiments that explored segmental reflexes and conditions that produced facilitation and inhibition of reflex muscle contractions. Kabat (1952) utilized these findings to develop contraction patterns that would evoke spinal reflexes to augment or reinforce weak voluntary drive of muscle in patient populations (Kabat 1947; Kabat & Knott, 1948; 1953). The particular contraction pattern under investigation is the reversal of antagonists technique; it involves a quick reversal of antagonistic musculature. In the case of isometric contractions, the GTO's are used to inhibit the agonist and facilitate the antagonist (which is the target muscle) prior to recruiting it as an agonist (Kroll, 1972a).

The reversal of antagonists technique has been studied sporadically over the last 40 years with mixed results. Kroll (1972a) examined the reversal of antagonists during maximal isometric contractions of the elbow flexors and extensors in college-age females and failed to show any increases in strength. Kroll (1972a) further noted that there was a “tendency” for the contraction to be stronger when it occurred first rather than following an isometric contraction of the antagonist. The two additional studies of the elbow flexors and extensors during a reversal of antagonists on a similar population failed to observe any evidence of facilitated maximal isometric strength or increased sEMG activity (Gabriel & Kroll, 1991; Gabriel et al., 1997). The few studies that have reported increases in maximal voluntary contractions have been conducted on patient populations (Bohannon, 1985; Holt et al., 1969), which leads to the speculation that facilitation effects cannot be manifested unless there is an existing deficit in neural drive (Kroll, 1972a; 1972b) as might exist in an older adult population (Connelly et al., 1999; Kamen, Sison, Du, & Patten, 1995; Patten & Kamen, 2000).

Two studies have shown that the reversal of antagonists technique does not result in an increase in maximum strength but does enhance the rate of torque development of the subsequent contraction. Grabiner (1994) showed the intensity (percent MVC) of the conditioning isometric contraction of the antagonist was directly proportional to sEMG activity and the rate of torque development during the subsequent “isokinetic” contraction of the agonist. In partial agreement, Gabriel and colleagues (2001) also reported an increase in the maximal rate of isometric elbow extension torque development during the reversal of antagonists. However, the lack of increase in sEMG activity led the authors to suggest the effects were primarily biomechanical in nature. The

conditioning contraction of the flexors altered extensors so they were at optimal length just prior to being recruited as an agonist, or there was storage and reutilization of elastic energy in the extensor tendons.

While the results of the facilitation effects are thus far equivocal, there is direct evidence that the reversal of antagonists may interfere with the early strength gains associated with motor learning, which has particular implications for rehabilitation. In two separate studies Kroll (1972a; 1972b) showed that when complex contraction patterns of antagonistic muscle groups were performed on the same day, training-related increases in strength due to motor learning were suppressed. Gabriel and Kroll (1991) then tested the specific hypothesis that the reversal of antagonists may interfere with early strength gains due to motor learning. Untrained college-age females were assessed for muscle strength and endurance using a measurement schedule that would allow for detraining effects of any potential physiological adaptations. There were five test sessions at two-week intervals with a cross-over of conditions on the last day of testing, and no resistive exercise of any type was allowed for the duration of the study. There were five baseline maximal isometric contractions followed by a 30-trial fatigue series. A control group performed flexion only contractions while an experimental group performed the extension-to-flexion contraction pattern.

The control group maintained baseline maximal isometric elbow flexion strength and sEMG across the four days of testing, then strength and sEMG decreased when required to perform the extension-to-flexion contraction pattern. Conversely, the experimental group exhibited a progressive decrease in maximal isometric elbow flexion strength and sEMG, but rebounded when allowed to perform flexion only contractions on

the last day. The results for the mean of the 30-trial fatigue series were similar except the control group also exhibited a “quick jump” in fatigue resistance. While biceps brachii sEMG increased there was no concomitant decrease in triceps brachii sEMG (coactivation) as would be predicted by the proposed neuromotor coordination control hypothesis (Kroll, 1974). Gabriel and colleagues (1997) later reproduced the Gabriel and Kroll (1991) study but focused on the elbow flexion-to-extension maximal isometric contraction pattern. Since elbow flexion strength is “generally” greater than extension strength in females (Kroll, 1972a; 1972b), it was hypothesized the extensors would be susceptible to facilitation effects. In this case, the control and experimental groups exhibited a “quick jumps” in both strength and fatigue resistance without any evidence of facilitation. Furthermore, learning-related strength gains were associated with an increase in sEMG activity from both the agonist and antagonist muscle groups.

The data on whether or not the reversal of antagonists interferes with early strength gains through motor learning is thus far equivocal. However, the reversal of antagonists technique is a more complicated contraction pattern than agonist only resistive exercise. To be effective, the reversal sequence must occur within a very specific time period (1 second) corresponding to maximal changes within the spinal cord when the GTOs are activated (Moore & Kukulka, 1991). It is therefore reasonable to suggest that the reversal of antagonists is a more difficult task compared to agonist only contractions (Kroll, 1972a; 1972b).

Task complexity is a concern because the reversals of antagonists is used for stroke rehabilitation in older adults (Bohannon, 1985, Dickstein et al., 1986; Duncan et al., 1998; Ernst, 1990; Lisiński et al., 2008) who already exhibit decrements in motor

performance and learning (Shea, Park & Braden, 2006, Voelcker-Rehage, 2008). In support, Barry, Riek, and Carson (2005) provide evidence that task complexity for isometric contractions reduces motor output in older adults (65-80 years). The authors studied a visually guided aiming task that required the generation of isometric torques about the elbow joint to targets corresponding to 30 and 50% MVC, every 30 degrees in the frontal plane. There were simple contractions that required only flexion, extension, supination, or pronation. However, the off axes contractions were more complex and involved two in combination (i.e., flexion and supination, flexion and pronation, extension and supination, extension and pronation). Compared to the younger adults (19-29 years), deficits in the rate of force development exhibited by the older adults were exacerbated by the muscle coordination requirements for the more complex contraction combinations.

## **2.4 Measuring Sensorimotor Responses**

### ***2.4.1 M-wave***

Electrical stimulation of the peripheral nerve excites the motoneuron pool, resulting in a massed action potential travelling directly to the muscle causing an involuntary contraction (Christie, Inglis, Boucher, & Gabriel, 2005; Frigon, Carroll, Jones, Zehr, & Collins, 2007). The electrical events associated with the evoked muscular contraction can be monitored with surface electrodes. A massed muscle action potential is recorded that is termed the “M-wave”. As the stimulation level increases, the amplitude of the M-wave will increase until the maximum M-wave ( $M_{max}$ ) is reached. The  $M_{max}$  is the point at which the amplitude of the M-wave ceases to increase further even as the stimulation level continues to increase (Tucker & Tüker, 2007). It is generally accepted

that the  $M_{\max}$  represents the activity of the whole motor neuron pool being recruited (Crone, Johnsen, Hultborn, & Ørsnes, 1999; Frigon et al., 2007; Scaglioni, Narici, Maffiuletti, Pensini, & Martin, 2003).

The amplitude of the  $M_{\max}$  can be affected by several factors such as length of experiment (Crone et al., 1999), electrode placement (Bromberg & Spiegelberg, 1997; Van Dijk, van der Kamp, van Hilten, & van Someren, 1994), age of the participants (Scaglioni et al., 2003), and position of the joint or muscle length (Cresswell, Loscher, & Thorstensson, 1995; Frigon et al., 2007; Kim, Date, Park, Choi, & Lee, 2005). However, it is not affected by changes at the level of the spinal cord (Christie et al., 2005; Frigon et al., 2007). It is generally accepted that the  $M_{\max}$  does not fluctuate significantly across days (Christie et al., 2005) and remains stable as long as recording conditions are unchanged and muscular fatigue is absent (Crone et al., 1999). Crone and colleagues (1999) examined the  $M_{\max}$  in the soleus and found that during a 2-hour long experiment, the amplitude of the  $M_{\max}$  decreased by 38% from beginning to end because of repeated stimulation of the peripheral nerve over the long period of time. These findings suggest that the length of a testing session should be taken into consideration when planning an experiment that requires obtaining  $M_{\max}$ . As well, the length of time should be taken into consideration if other reflexes (such as the H-reflex or V-wave) are being recorded and normalized to the values of the  $M_{\max}$  (Crone et al., 1999).

The location of the recording electrode is also of importance when trying to obtain  $M_{\max}$ . Some would argue that the recording electrode should be placed over the motor point to obtain the largest amplitude  $M_{\max}$ , but according to Bromberg and Spiegelberg (1997) and Van Dijk and colleagues (1994), recording over the motor point

is not always the most accurate location. Van Dijk and colleagues (1994) studied the thenar and hypotenar muscles and determined that only 62% of the time, the maximum amplitude response was recorded over the motor point and the rest of the time it was recorded 1cm away from the motor point. Similarly, Bromberg and Spiegelberg (1997) mapped out a 1cm x 1cm grid and found that adjustments in electrode placement even less than 1cm could result in large changes in the amplitude. The authors also concluded that slight changes in the reference electrode placement could also have an effect on the shape of the waveform produced (Bromberg & Spiegelberg, 1997).

As mentioned, age also has an effect on  $M_{\max}$  amplitude (Scaglioni et al., 2003). Scaglioni and colleagues determined that an elderly population (mean age 73 years) presented with amplitudes for  $M_{\max}$  that were 57% lower than a younger population (mean age 25 years). These results demonstrate that as age increases, the amplitude of the  $M_{\max}$  decreases, therefore age should be taken into account when examining  $M_{\max}$  data and comparing it among different populations.

#### ***2.4.2 H-reflex***

The Hoffman reflex (H-reflex) is an evoked monosynaptic reflex observed when a peripheral nerve is stimulated using a low-level electrical stimulation (Brinkworth, Tuncer, Tucker, Jaberzadeh, & Türker, 2007; Stein & Thompson, 2006; Zehr, 2002). Considered a measure of spinal excitability (Aagaard, Simonsen, Anderson, Magnusson, & Dyhre-Poulsen, 2002), the H-reflex has been used to study changes in neural activity during resistive exercise (Aagaard et al., 2002; Vila-Chã, Falla, Velhote-Correia, & Farina, 2012) and neuromuscular disorders (Upton, Sica, & McComas, 1972).

When the peripheral nerve is stimulated using a low intensity current, action potentials are elicited in the axons of the sensory Ia afferents specifically because of their low threshold, large axon diameter (Aagaard et al., 2002; Brooke et al., 1997; Zehr, 2002). These action potentials propagate to the spinal cord, where they give rise to excitatory postsynaptic potentials, which in turn elicit action potentials which travel from the alpha motoneurons to the muscle and are recorded as an H-reflex (Figure 9) (Aagaard et al., 2002).

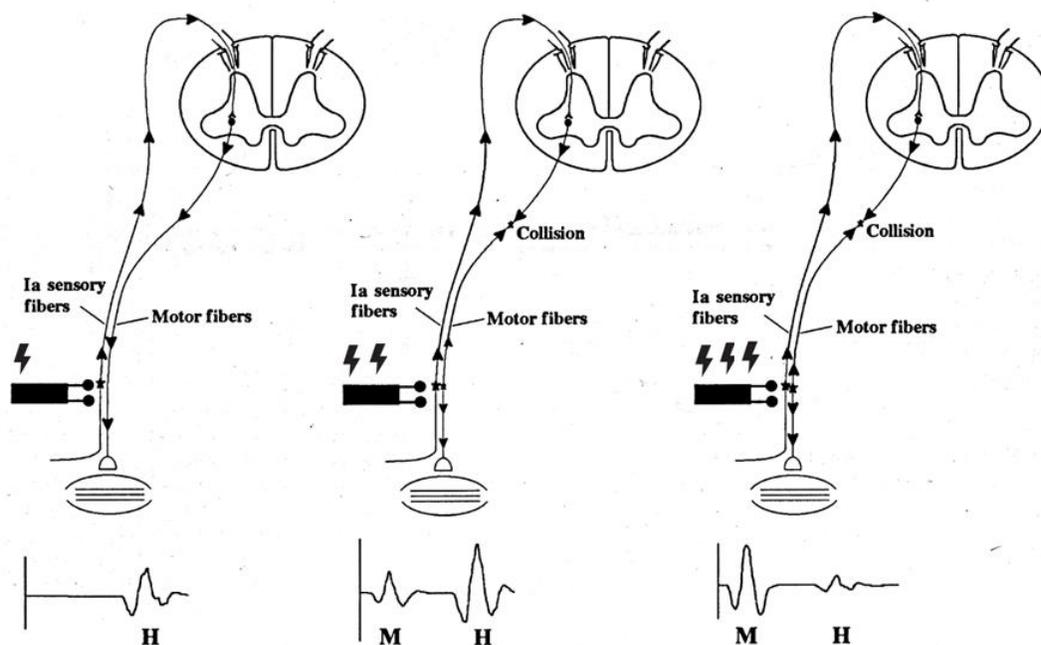


Figure 9. Pathways excited when a peripheral nerve is stimulated to evoke an H-reflex and/or M-wave. Picture displays increasing level of stimulation from left to right.

Adapted from Preston, D.C. & Shapiro, B.E. (1998). *Electromyography and neuromuscular disorders: clinical-electrophysiologic correlations*. Boston: Butterworth-Heinemann. Figure 4-8, p.52.

The H-reflex not only reflects spinal excitability, but can also reflect the level of presynaptic inhibition of the Ia afferent synapses (Hultborn et al., 1987). According to Aagaard and colleagues (2002) at a particular level of stimulus, an increase in H-reflex amplitude could represent an increase in the excitability of the alpha motoneuron and/or a decrease in the level of presynaptic inhibition. Presynaptic inhibition occurs through the action of an inhibitory interneuron that acts on the Ia afferent terminals (Zehr, 2002). This action leads to a decrease in the neurotransmitter release, as well as a reduction in motoneuron depolarization induced by Ia activity (Zehr, 2002). According to Zehr (2002), presynaptic inhibition has been shown to alter the afferent signal that evokes an H-reflex, which can lead to different patterns of motoneuron excitability. In discussing the H-reflex and presynaptic inhibition, Zehr (2002) also discusses a few of the factors that can affect presynaptic inhibition. He states that these factors include, but are not limited to, descending supraspinal commands and afferent feedback from peripheral receptors such as muscle spindles, GTOs, or cutaneous receptors. To control for these factors, it is important to ensure participant posture is maintained throughout the test sessions, as well as that the intention of the participant remains the same.

The H-reflex is commonly measured in relation to the  $M_{\max}$  (Brinkworth et al., 2007; Zehr, 2002). An important methodological control is to use a stimulation intensity that is calibrated as a percentage of  $M_{\max}$ . A fixed percentage between 5 and 20% of  $M_{\max}$  is used to minimize collision and gauge the number of motoneurons that are involved. For example, if a stimulation level that produced an M-wave that is 20% of  $M_{\max}$  is used, it is assumed that there are a fixed number of motoneurons (20% of the motoneuron pool) that are being recruited each time the peripheral nerve is stimulated (Hugon, 1973). This

allows researchers to conclude that alterations in H-reflex amplitude are due to changes in spinal excitability or presynaptic inhibition, not stimulus intensity.

The H-reflex is a highly variable reflex that is known to fluctuate greatly from trial to trial (Brinkworth et al., 2007; Christie, Kamen, Boucher, Inglis, & Gabriel, 2010). According to Brinkworth and colleagues (2007), the size of the H-reflex is a function of three factors: 1) the precision of the stimulus intensity; 2) the excitability of the entire H-reflex arch; and 3) the accuracy of the recording. To help minimize variability, the placement of the electrodes and position of the participant should remain consistent throughout test sessions (Zehr, 2002), stimulation levels should remain constant (Zehr, 2002), testing sessions should occur at the same time each day (Guette, Gondin, & Martin, 2005), and the H-reflex should be normalized to a percentage of the  $M_{\max}$  to ensure consistency among participants (Brinkworth et al., 2007). Factors such as age (Scaglioni et al., 2003) and caffeine intake (Walton, Kalmar, & Cafarelli, 2003) should also be taken into account.

### ***2.4.3 V-wave***

The V-wave is typically used to measure the level of central drive from the excited motoneuron pool during a maximal contraction (Aagaard et al., 2002; Sale, MacDougall, Upton, & McComas, 1983; Vila-Chã et al., 2012). The V-wave uses the same reflex-arc as the H-reflex but has been termed the V-wave to indicate its presence during volitional activity but absence during rest (Aagaard et al., 2002). Supramaximal nerve stimulation of the peripheral nerve, similar to that used to evoke an M-wave, during maximal voluntary contraction will elicit a V-wave (Aagaard et al., 2002; 2003; Vila-Chã et al., 2012). Efferent motor action potentials generated during voluntary contractions

collide with antidromic motor action potentials evoked by supramaximal peripheral nerve stimulation, allowing the evoked H-reflex response to pass to the muscle (see Figure 10) (Aagaard et al., 2002; Upton, McComas, & Sica, 1971; Vila-Chã et al., 2012). Therefore, the V-wave can be used as an indication of the magnitude of antidromic clearing, and thereby reflect the frequency and number of efferent impulses in the alpha motoneuron axons during voluntary muscle activation (Aagaard et al., 2002). The supramaximal stimulation of the peripheral nerve results in both large and small motoneurons being recruited (Aagaard et al., 2002).

Several factors have been known to affect the amplitude of the V-wave, as well as its latency. Upton and colleagues (1971) studied the potentiation of late responses (V-waves) in muscles during contractions and found that as the electrodes were moved proximally along the muscle, the latency of the V-wave decreased, suggesting involvement of the spinal cord in the production of the V-wave. As well, the V-wave is affected by the same factors involved in the H-reflex: (1) changes in motor neuron responsiveness, (2) synaptic transmission efficacy at Ia afferent terminals, and/or (3) postsynaptic inhibition (Vila-Chã et al., 2012).

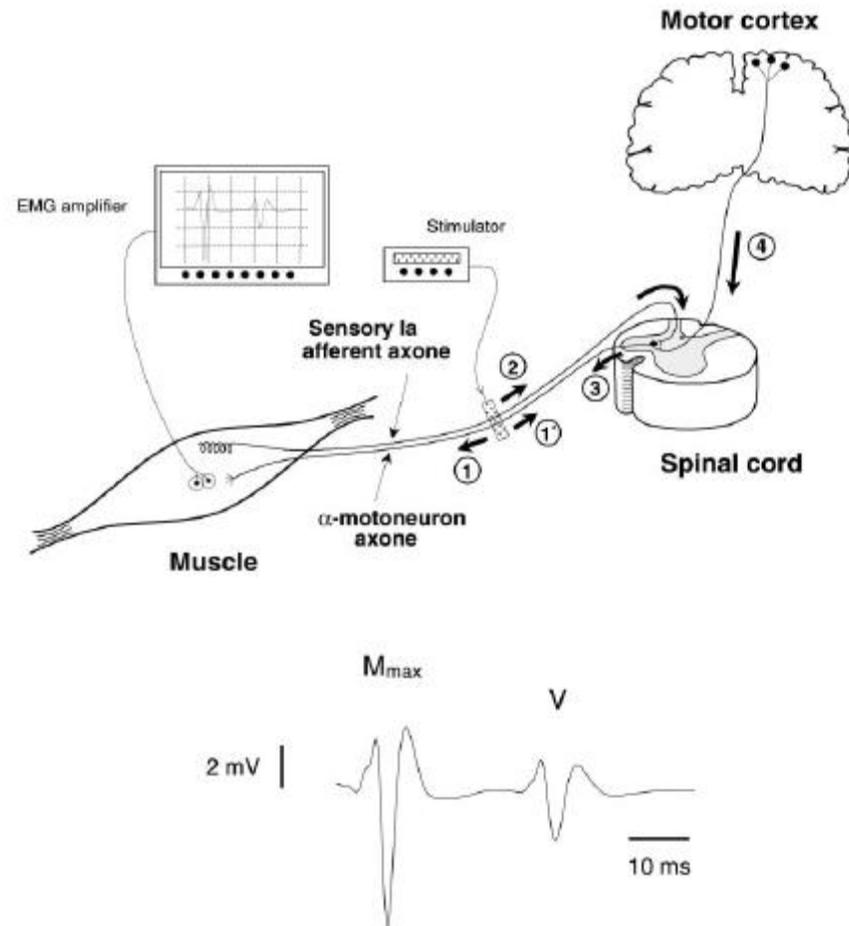


Figure 10. Spinal circuitry pathway followed when eliciting a V-wave. Aagaard, P., Simonsen, E.B., Anderson, J.L., Magnusson, P., & Dyhre-Poulsen, P. (2002). Neural adaptations to resistance training: changes in evoked V-wave and H-reflex responses. *Journal of Applied Physiology*, 92: 2309-2318. Figure 2, page 2311.

## CHAPTER 3: MATERIALS AND METHODS

This chapter will focus on the methodology that was used to answer the research question presented in Chapter 1. A discussion of the sample size estimation performed and the subjects will be followed by a description of the experimental design including the measurement schedule, apparatus and testing position, as well as the testing protocol. This chapter will finish by detailing the techniques that were utilized, followed by a discussion of the statistical analyses that were used.

### 3.1 Sample Size Estimation and Description of Participants

Sample size estimation was accomplished using means, standard deviations, and the intraclass reliability coefficient for maximal isometric wrist flexion strength obtained using a measurement schedule similar to that proposed in this study. Kroll (1963a) studied the effects of repeated assessment of maximal isometric wrist flexion strength. Participants were tested on three successive days, followed by another three successive days of testing three weeks, and three months later. The means, standard deviations, and intraclass reliability coefficient of interest were for the initial three consecutive days and the first retest three weeks later. The calculations, reported in Appendix A, resulted in a sample size of 10 participants per group for a total of 20. However, to protect against the fact that observed error variances and reliability may be lower, the study aimed to recruit 13 participants per group for a total of 26 participants.

Inclusion criteria included the stated absence of neurological or musculoskeletal disorders of the upper limb, right-hand dominance, and had not performed any forearm resistance training in the past year. Participants completed a PAR-Q questionnaire and if

they answered “yes” to any questions, especially those pertaining to hypertension, they were excluded from the study. All participants completed written informed consent forms as approved by Brock University Research Ethics Board (REB#12-281) (Appendix B).

### **3.2 Preliminary Procedures**

Prior to the first testing session, participants were invited into the laboratory to become familiarized with the nature of the experiment and the equipment. Participants then signed an informed consent document (Appendix B), which outlined the requirements of participation, including the inherent risks, possible benefits, and the right to discontinue at any point in time without prejudice. Next, participants filled out a PAR-Q and demographics questionnaire (Appendix B). Anthropometric measurements of the forearm were then taken (Appendix B) and upon completion of these procedures, all four testing sessions were scheduled.

### **3.3 Experimental Design**

#### ***3.3.1 Apparatus and Testing Position***

All procedures took place inside the Faraday cage within the Electromyographic Kinesiology Laboratory at Brock University. Participants were seated at a testing table at a height that allowed the elbow to rest at 160° of extension (Figure 11). A custom-made jig was designed to isolate the hand during isometric contractions of the wrist flexors and extensors. Restraints for the hand were mounted onto a lever arm that was attached to a load cell (JR3 Inc., Woodland, CA). The load cell was secured to base of the testing table. The hand was placed in a half-supinated position within restraints that contacted the volar and dorsal surfaces (Figure 12). Each surface was padded with 1 cm of foam.

The forearm and hand were placed so that the axis of rotation of the wrist was aligned with the axis of rotation of the lever arm on the load cell, just beyond the surface of the table. As well, there were restrains for the forearm to minimize extraneous movements. It was required for the arm not being tested to rest on the testing table. An oscilloscope (VC-6525, Hitachi, Woodbury, NY) was placed at eye level in front of the participant (Figure 13). The oscilloscope was used to display the torque levels achieved during the contractions.



Figure 11. Experimental apparatus and testing position of the participant during each testing session.

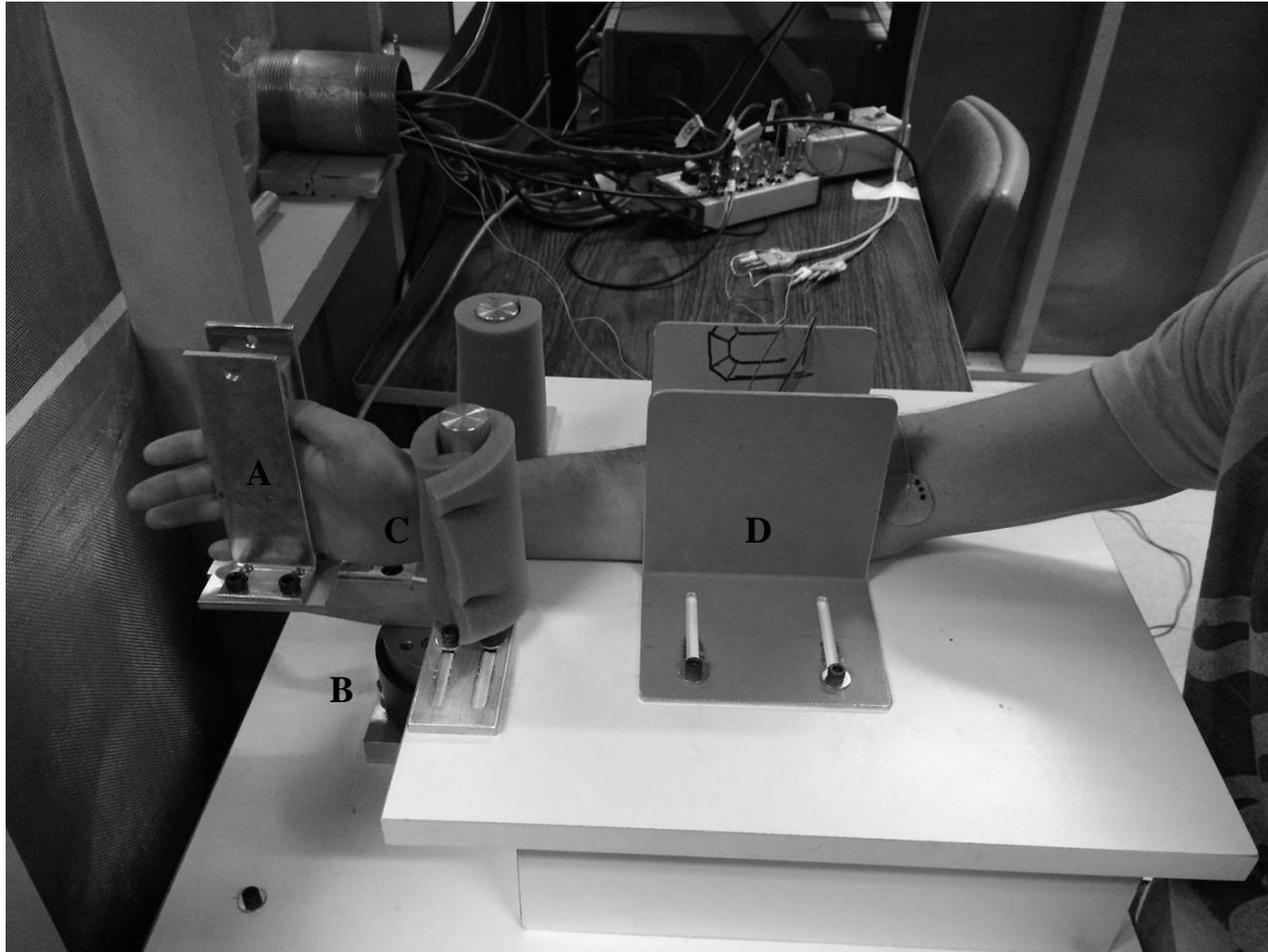


Figure 12. Apparatus and load cell set-up. A) Hand restraints; B) Load cell attached to testing table; C) Wrist axis of rotation aligned with lever axis of rotation; D) Forearm restraints.



Figure 13. A participant's view of the oscilloscope displaying their maximum torque level and target lines.

### 3.3.2 Measurement Schedule

There were four separate testing sessions, each lasting approximately 1.5 hours. The first three sessions occurred over three days with 48 hours between each session and the fourth session occurred two weeks after the third session. Participants were randomly assigned to one of two groups: a control group or an experimental group. The control group performed 5-second maximal isometric contractions of the wrist flexors. The experimental group executed a 5-second maximal isometric contraction of the wrist extensors immediately prior to a 5-second maximal isometric contraction of the wrist flexors. Anthropometric data obtained during the preliminary test session was used to predict each participant's maximal isometric wrist flexion strength. Participants were then ranked and matched and randomly assigned by pairs into either the control or experimental group. Green and colleagues (2012) showed that a multiple regression equation that included body weight, segment length and limb circumference was an excellent predictor ( $R^2=0.56$ ) of maximal isometric elbow flexion strength. A pilot study ( $N=6$ ) was conducted to develop a similar multiple regression equation for maximal isometric wrist flexion strength ( $R^2=0.86$ ). The resulting equation was:

$$\text{Predicted peak torque (Nm)} = -29.85958 + 0.25507(\text{Weight}) + 1.68481(\text{Wrist Circumference})$$

The first three test sessions were identical for both groups, with the exception of the contraction pattern performed. The fourth test session (retention/transfer test) followed a similar protocol, except the participants performed five of their assigned contraction pattern (retention test), followed by five of the opposite contraction pattern (transfer test). The order of the contractions was counter-balanced across the two groups.

Five maximal M-waves and ten H-reflexes were evoked in the flexor carpi radialis (FCR) before and after maximal isometric strength assessment. Starting with M-wave data collection, there was 15-seconds between each evoked potential. Hoffman (H) reflexes were then evoked at 15-second intervals, five-minutes after the last maximum M-wave. Another five-minute rest period preceded maximal isometric strength assessment. Participants then performed ten trials of their assigned contraction pattern. Each maximal isometric contraction was five seconds in duration with three-minutes of rest between each contraction to minimize fatigue (Clarke & Stull, 1969). The V-wave was evoked in the middle of each maximal isometric wrist flexion strength trial. The protocol for all four sessions can be seen in Figure 14.

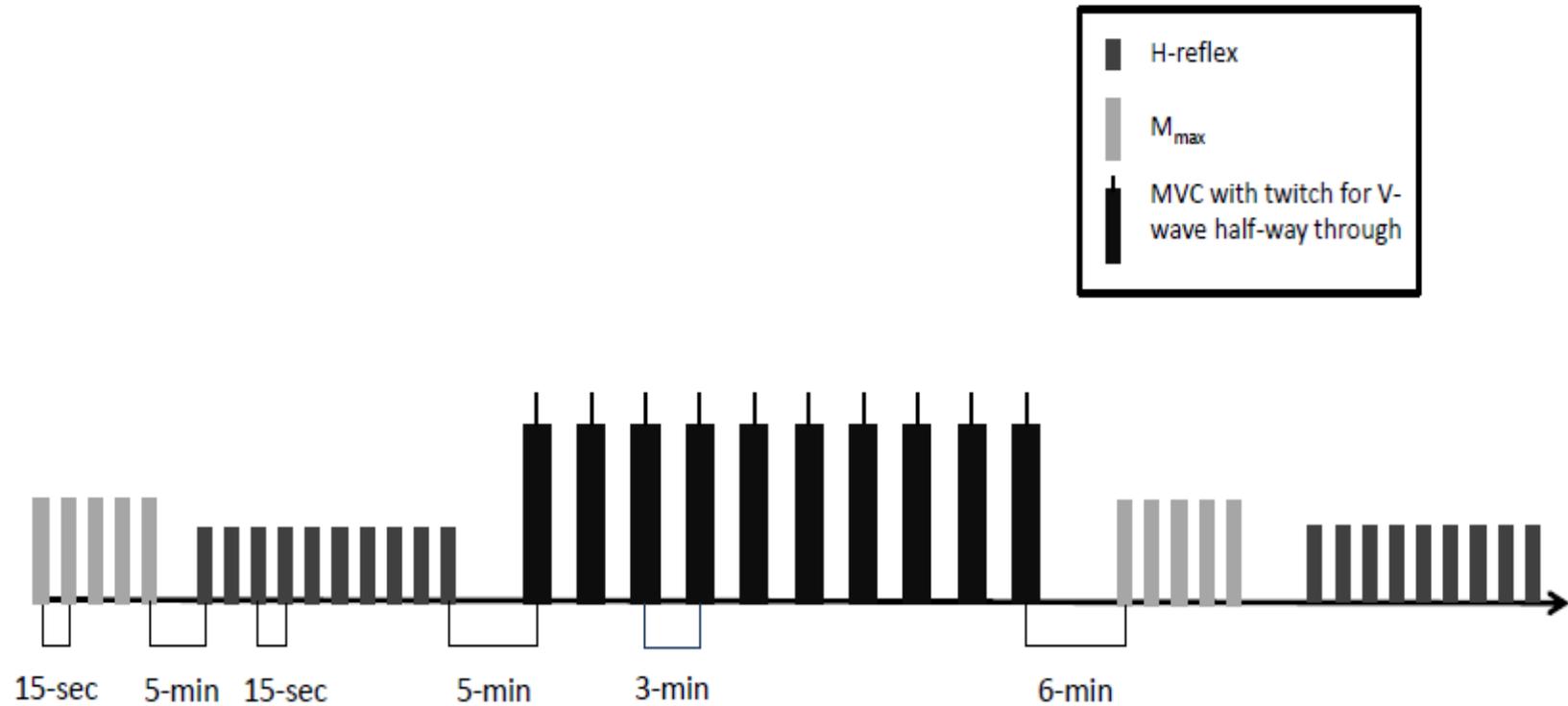


Figure 14. Testing protocol that was carried out during all four sessions for both groups. During the first three sessions, participants performed the assigned contraction pattern. During the fourth session participants performed five of the assigned contraction pattern and five of the non-assigned contraction pattern.

### **3.3.3 Recording Voluntary EMG**

At the beginning of each session, the right forearm was prepped for testing. The electrode locations were shaved, cleansed with isopropyl alcohol, and lightly abraded (NuPrep®, Weaver and Company, Aurora, CO) to maintain skin-electrode impedance below 10 k $\Omega$  (Grass EZM Electrode Impedance Meter, Astro-Med Inc., Warwick, RI). Using an impedance meter (Grass EZM Electrode Impedance Meter, Astro-Med Inc., Warwick, RI) skin impedance was measured before and after the protocol each test session for both the flexor carpi radialis (FCR) and extensor carpi radialis (ECR) muscles. During each test session, a thermometer was also secured on the skin's surface beside the FCR electrodes to monitor skin temperature. The motor points of the FCR and ECR were then located using a low-level repeated electrical stimulation on the skin's surface. The motor point was defined as the point at which a muscle twitch was still visible with the lowest level of stimulation. Once located, these points were marked with indelible ink for electrode placement. Pediatric-sized electrodes (3mm electrode diameter, F-E9M 11mm, GRASS Technologies, Astro-Med, Inc., Warwick, RI) with an inter-electrode distance of 1cm were placed in a bipolar electrode configuration and used to measure the electrical activity of the FCR and ECR muscles during voluntary and evoked contractions. The electrodes were affixed with a two-sided tape and electrolyte gel (Signa Gel®, Parker Laboratories, Fairfield, NJ). A self-adhesive ground electrode was placed on the dorsal side (back) of the hand (Figure 15) for electrical safety and to minimize noise.

To ensure the electrode placement was consistent throughout testing sessions, the electrodes were traced with indelible ink. The participants were asked to maintain these

tracings between sessions and were welcome to come to the laboratory to have the tracings maintained if needed. Although, maintaining the tracings was helpful for the investigator, it was not necessary. If a participant was unable to maintain a tracing, the location of electrode placement was found using the protocol to locate the motor point that is discussed above. These procedures have been shown to result in high intraclass reliability coefficients suitable for documenting surface electromyographic (sEMG) activity obtained over long periods of time (Calder et al., 2005; Christie et al., 2010; Gabriel, 2002; Gabriel, Basford, & An, 2001).

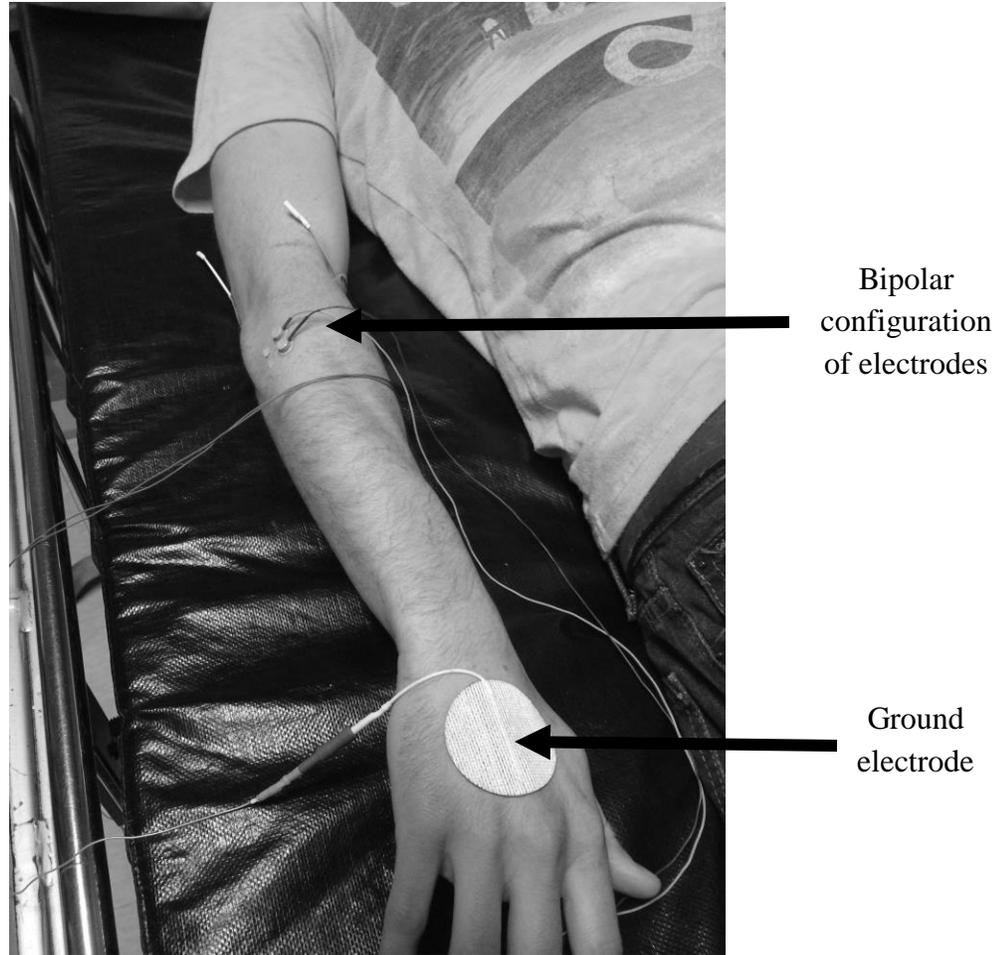


Figure 15. Experimental setup of sEMG electrodes.

### ***3.3.4 Evoked Potentials***

The median nerve supplying the FCR was stimulated (Grass Telefactor S88, Astro-Med Inc) to obtain the M-wave, H-reflex, and V-wave. Palpating the biceps tendon in the bicipital groove and moving medially located the median nerve; a pulse can be found where the cathode was placed. The cathode (3.2 cm diameter, 879100, Axlegard Manufacturing Co., Ltd., Fallbrook, CA) and anode (5 cm, diameter, CF5000, Axlegard Manufacturing Co., Ltd., Fallbrook, CA) were self-adhesive pad electrodes (Figure 16). The anode was placed on the posterior aspect of the upper arm directly below the cathode. Both electrodes were connected in series with an isolation unit (Grass Telefactor SIU8, Astro-Med Inc., West Warwick, RI) and a stimulator (Grass Telefactor S88, Astro-Med Inc.) that delivered a constant current (150 mA) square-wave pulse, 0.5 ms in duration. The level of stimulation needed to obtain a maximum M-wave was found by slowly increasing the voltage level until the amplitude plateaued (Tucker & Tüker, 2007). H-reflexes were evoked by using a stimulation level that evoked a M-wave that had an amplitude that was  $5\pm 3\%$  of the maximum M-wave (Christie et al., 2005). V-waves were obtained using supramaximal (110%) stimulation during the voluntary isometric wrist flexion contractions (Aagard et al., 2002; 2003; Vila-Chã et al., 2012).

### ***3.3.5 Instructions to Participants***

During the voluntary contractions, participants were instructed by the investigator to maximally contract their forearm muscles. A target line representing the participant's maximum force was presented on the oscilloscope (Hitachi, VC-6525). This target line served two functions. First, participants were instructed to contract as hard and as fast as possible in order to move their trace to or above the target line. Second, participants were

instructed to maintain their force trace parallel to the target line in order to maintain a steady force level. Along with the visual feedback presented on the oscilloscope, participants were shown a picture of what an “optimal” force trace looks like. This was presented as a frame of reference to help participants understand the task. Participants were instructed that they were required to use the visual feedback during all maximal voluntary contractions. The visual feedback was only provided during the first three test sessions. During the retention test, all visual feedback was removed to assess motor learning.

The work-to-rest ratio for the voluntary contractions was controlled by a tape recording. For the control group, the tape recording said: “Ready...Three, two, one, flex”. When the word “flex” was heard, the participants were required to flex at the wrist, as hard and as fast as possible. Participants held the contraction until they heard the word “relax”. The sequence was repeated ten times, with three-minutes between each contraction. Instructions for the experimental group said: “Ready...Three, two, one, extend”. Participants then extended the wrist, as hard and as fast as possible when they heard the word “extend”. The contraction was held until the tape recording instructed them to “flex” wherein an immediate maximal wrist flexion was required. The participants maintained the wrist flexion contraction until they heard the word “relax”. Ten extension-to-flexion contraction patterns were completed at three-minute intervals. No verbal encouragement was provided during the voluntary contractions. After receiving instructions, the participant was asked to repeat the instructions and expectations, to ensure that they understood the task and the visual feedback they were receiving.



Cathode placement  
on the median nerve.  
Anode is placed  
directly in line on the  
opposite side of the  
joint.

Figure 16. Setup of stimulation electrodes.

### **3.4 Signal Processing**

All data was collected inside the Faraday cage located in the Electromyography Laboratory which maintained a signal to noise ratio of approximately 20 dB. The sEMG signals were amplified (Grass P511, Astro-Med, Inc., Warwick, RI) to maximize the resolution of the 16-bit analogue-to-digital convertor (PCI-6251, DATAQ Instruments, Akron, OH) and band-passed filtered (3-1000 Hz). Both force and sEMG signals were digitized at 2048 Hz (DASYLab, DASYTEC National Instruments, Amherst, NH). The force signal was low-passed filtered (20 Hz, 3 dB) using a 4th order Butterworth digital filter offline in MATLAB (The Mathworks Inc., Natick, MA).

### **3.5 Data Reduction and Criterion Measures**

The following criterion measures were obtained from a one-second window in the middle of each five-second-wrist flexion contraction: mean maximal torque, and root-mean-square (RMS) sEMG amplitude for the FCR and ECR. (Figure 17-18). The peak-to-peak amplitude of the M-waves, H-reflexes and V-waves were also determined (Figure 19-20) for reliability purposes.

Additionally, variability measures were calculated on the MVCs to assess learning. Variability in maintaining a constant torque was assessed by calculating the RMS error of the middle 3.5 seconds of the torque trace. This measure represents the variability of the horizontal portion of the torque trace itself, not relative to the horizontal target line. Prior to the RMS error calculation, the torque trace was normalized to its maximum value. To assess the variability in the shape of the entire waveform, variance ratios (VRs) for torque, and the FCR and ECR sEMG waveforms were calculated.

For every block of 5 contractions for each participant, VRs were calculated in the following way for torque and FCR and ECR sEMG. First, a mean waveform was calculated for the block of five trials. For each individual waveform within the block of five trials, the squared deviation (variance) between each point and its corresponding mean point across waveforms was calculated. The mean of the squared deviations was then calculated to represent the mean variance “within” waveforms. A grand mean, which was a single value for all the data points constituting the five trials, was then determined. The squared deviation between each point of the five waveforms and the grand mean was then calculated. The mean of these squared deviations then represented the variance “between” waveforms. Statistically, the VR is interpreted in a way similar to the intraclass correlational analysis of variance model (Christie et al., 2010). The lower the variance “within” each waveform, the smaller the differences that can be detected between waveforms, resulting in a higher variance “between” waveforms. Thus, a lower VR (within/between) indicates a higher reproducibility of waveform shape.

Prior to calculating the VR, the sEMG signals were first linear envelope detected at 20 Hz with a zero phase shift 4th order Butterworth digital filter. Force was similarly filtered at the same low-pass cutoff frequency. The signals were then aligned 500 ms before to 500 ms after the force onset and termination, respectively. Finally, each signal was time-normalized by interpolating each trace to 8,000 data points. The VR was then calculated for each block of five trials. The VR was calculated according to the following formula:

$$VR = \frac{\frac{\sum_{t=1}^T \sum_{n=1}^N (y_{t,n} - \bar{y}_t)^2}{T(N-1)}}{\frac{\sum_{t=1}^T \sum_{n=1}^N (y_{t,n} - \bar{y})^2}{T N - 1}}$$

where  $T$  is the number of data points required (8,000),  $N$  is the number of trials in the VR (five per ratio),  $y$  represents a single trace with  $t$  being each point ( $t_1$  is the first point of a single trial), therefore  $\bar{y}_t$  is the average of the five trials at each point, and  $\bar{y}$  is the single value of an averaged waveform across all points. All data reduction was performed using MATLAB software (The Mathworks, INC., Natick, MA).

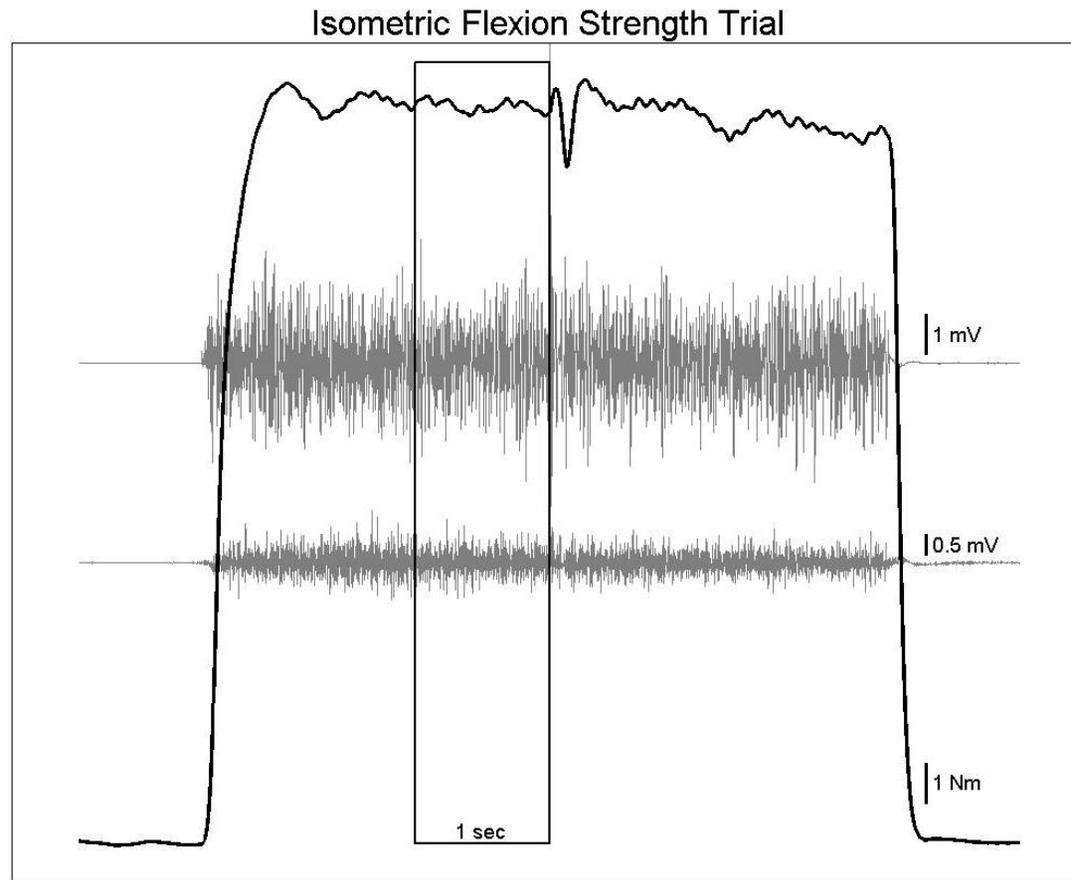


Figure 17. Criterion measures (torque, RMS) extracted from a 1-sec window occurring immediately before the stimulation during the 5-sec isometric flexion strength trial. Grey traces represent sEMG activity of the FCR and ECR. Black trace represents torque.

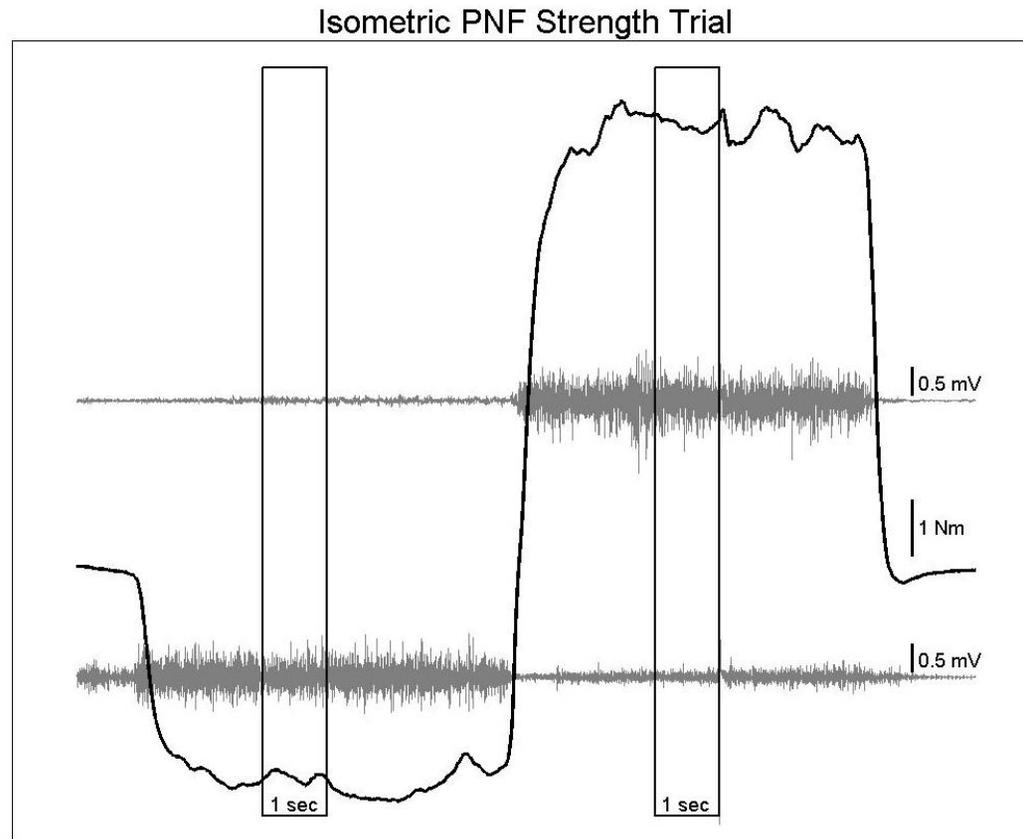


Figure 18. Criterion measures (torque, RMS) extracted from two 1-sec windows, occurring immediately before the stimulation during the flexion portion and in the middle of the extension portion of the trial. Grey traces represent sEMG activity of the FCR and ECR. Black trace represents torque.

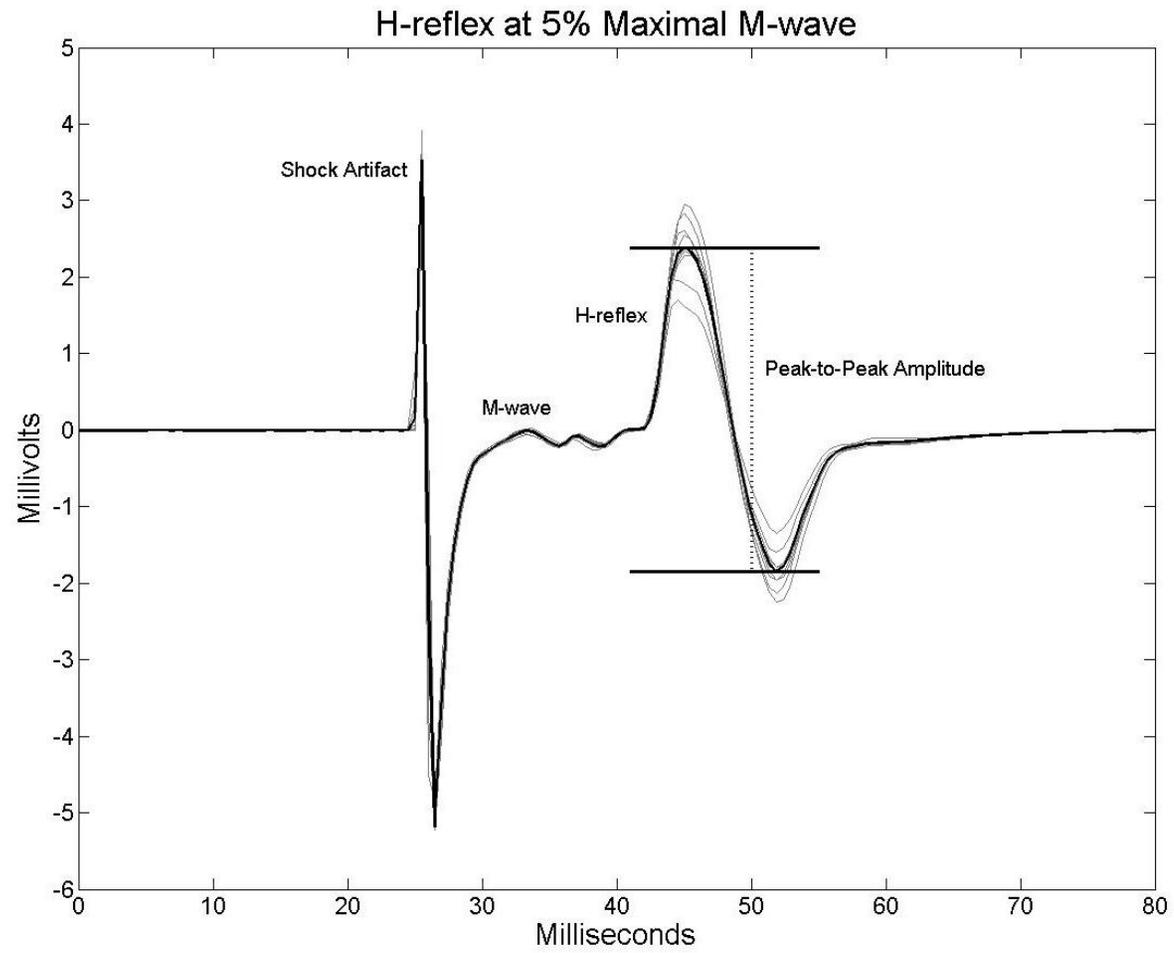


Figure 19. Defining peak-to-peak amplitude of an H-reflex.

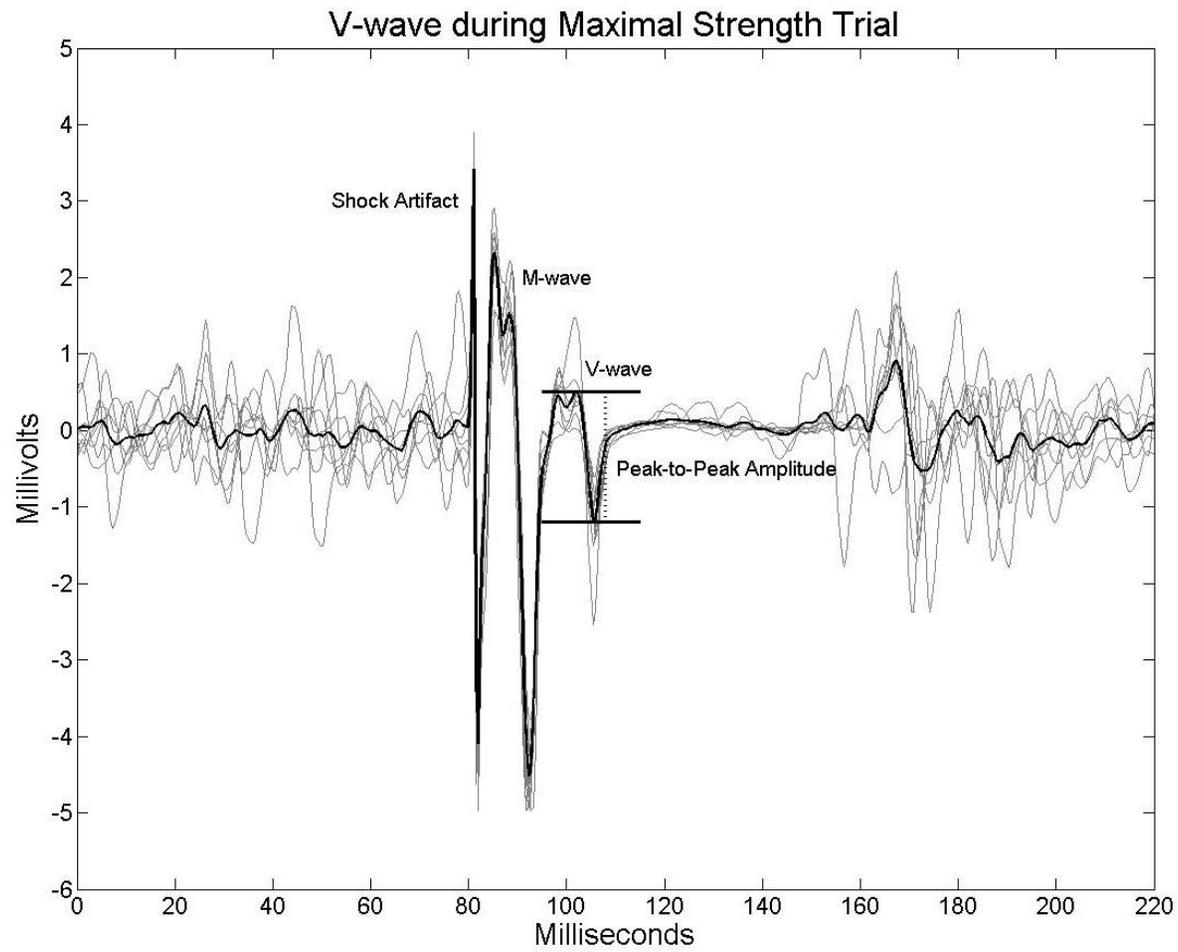


Figure 20. Defining peak-to-peak amplitude of a V-wave.

### 3.6 Statistical analysis

Prior to the main analyses, all data were screened to determine if there were outliers greater than three standard deviations associated with technical errors. Since the experimental design involved repeated measures, the assumptions for a split-plot factorial (SPF- $p.qr$ ) analysis of variance (ANOVA) were tested. The assumptions included normality (probability plots), and homogeneity of variances and sphericity (Mauchley's test). Participants completed ten trials of maximal isometric strength assessment on each of the four test days, for a total of forty trials, however, only twenty-five trials were analyzed (five contractions from each day 1-3, five retention contractions, and five transfer contractions). Table 1 illustrates the specific statistical model that was used to evaluate the criterion measures. The SPF- $p.qr$  ANOVA had one between-subjects factor ( $p$ =group) and two within-subjects factors ( $q$ =blocks and  $r$ =trials). When appropriate, Bonferroni-corrected orthogonal contrasts were performed for savings analysis to assess retention and transfer. More complex first-order interactions were explored using orthogonal polynomials to evaluate trends in the mean across days (Kirk, 2012). Significance was established at the 0.05 probability level.

Intraclass correlational analysis of variance was used to assess the reliability of the criterion measures as outlined in Christie and colleagues (2010). Reliability analysis of the data required two different ANOVA models: one to examine the consistency of scores within subjects, and the other to test the stability of the means across test days. The first model was a fully nested ANOVA with trials nested within days, and days nested within subjects. The mean squares obtained from the fully nested model were used to calculate the intraclass reliability coefficient ( $R$ ). Details of the calculations are presented

in Appendix C. The stability of means across test days was then evaluated using a complementary two-factor (days  $\times$  subjects) ANOVA.

Table 1. Sample Split-plot Factorial (SPF-p-qr) ANOVA for mean torque across test sessions.

Source	<i>df</i>	SS	MS	F	<i>p</i>
<b>Between Groups</b>					
Groups	1	303.02	303.02	3.00	0.097
Subjects(Groups)	22	2224.67	101.12		
<b>Within Groups</b>					
Blocks	4	208.80	52.20	9.36	<0.0001
Groups x Blocks	4	18.29	4.57	0.82	0.516
Blocks x Subjects(Groups)	88	490.78	5.78		
Total	119	3245.57	27.27		

\*Significance at  $p < 0.05$ .

## CHAPTER 4: RESULTS

### 4.1 Participant Characteristics

In total, 26 male participants volunteered to participate in the study and completed the introduction session. However, only 25 participants completed all four testing sessions. To maintain equal groups, data for 24 participants was used in the final analysis. The data for the matched pair of the participant who did not complete the four testing sessions was omitted from the statistical analysis. This resulted in a final sample size of 24 participants, with 12 participants in each group.

The participants' ( $N=24$ ) physical characteristics, predicted peak torque, baseline isometric mean torque and baseline surface electromyographic (sEMG) root-mean-square (RMS) amplitude for the flexor carpi radialis (FCR) and extensor carpi radialis (ECR) are presented in Table 2. Paired samples  $t$ -tests were performed on each physical characteristic (refer to Table 2), predicted peak torque and baseline torque and sEMG measures to ensure there were no statistical differences between the two groups.

At the beginning and end of each test session, skin impedance and temperature were recorded. Mean values and standard deviations for skin impedance recorded on the FCR and ECR, as well as skin temperature measured on the FCR are presented in Table 3. The average skin impedance decreased by 1.71 k $\Omega$  ( $\Delta 24\%$ ) for the FCR and 0.72 k $\Omega$  ( $\Delta 14\%$ ) for the ECR. Despite a significant  $t$ -test ( $p < 0.05$ ), no practical significance can be placed on small changes in impedance below 10 k $\Omega$ , while these values are still well within the accepted range for sEMG (Hewson et al., 2003). There was a significant ( $p < 0.05$ ) increase in skin temperature (1.26 $^{\circ}\text{C}$ , or  $\Delta 4\%$ ). It has been argued that a comparably small change might alter the sEMG signal (Rutkove, 2000; Winkel &

Jørgensen, 1991). However, only the first five contractions performed were analyzed as part of the experimental design, which should minimize temperature related-effects on the sEMG signal (Masuda et al., 1999).

Table 2. Means (M) and standard deviations (SD) for the physical characteristics of the participants by Group.

Physical Characteristic	Flexion Group M $\pm$ SD (n=12)	PNF Group M $\pm$ SD (n=12)
Age (years)	23.42 $\pm$ 2.31	23.33 $\pm$ 2.31
Height (cm)	179.4 $\pm$ 5.89	178.5 $\pm$ 5.70
Weight (kg)	79.15 $\pm$ 8.65	77.60 $\pm$ 8.44
Wrist Circumference (cm)	16.76 $\pm$ 0.53	16.63 $\pm$ 0.88
Predicted Peak Torque (Nm)	18.59 $\pm$ 2.80	17.94 $\pm$ 3.36
Torque (Nm) – Day 1	14.40 $\pm$ 4.29	12.65 $\pm$ 4.74
FCR sEMG (mV) – Day 1	0.31 $\pm$ 0.19	0.33 $\pm$ 0.22
ECR sEMG (mV) – Day 1	0.14 $\pm$ 0.10	0.14 $\pm$ 0.05

Significant difference between groups, \* =  $p < 0.05$

Table 3. Means (M) and standard deviations (SD) for impedance and temperature at the beginning (pre) and end (post) of all test sessions.

Measurement	Pre M $\pm$ SD	Post M $\pm$ SD
Impedance – FCR (k $\Omega$ )	7.05 $\pm$ 2.21	5.34 $\pm$ 2.30*
Impedance – ECR (k $\Omega$ )	5.12 $\pm$ 2.02	4.40 $\pm$ 1.74*
Temperature ( $^{\circ}$ C)	30.60 $\pm$ 1.13	31.86 $\pm$ 1.01*

Significant difference between pre and post, \* =  $p < 0.05$

## 4.2 Data Screening

Prior to analysis, the data were screened for outliers, which were defined as values that fell three standard deviations away from the variable mean. Any value that fell outside the  $\pm 3$  standard deviation range was examined to determine if it was physiologically reasonable or so extreme that it might be due to technical error. It was determined that all observed values would be included in the analysis

## 4.3 Statistical Assumptions

All data were tested for the assumptions of a split-plot factorial (SPF-*p.qr*) analysis of variance (ANOVA). Probability plots, and skewness and kurtosis measures were examined for each variable. For all variables, skewness values were below 3, while kurtosis values were below 9. It has been stated that ANOVA results are robust to departures from normality for balanced designs with moderate sample sizes (Glass, Peckham, & Sanders, 1972).

The assumption of homogeneity of variances was analyzed using Mauchly's test of sphericity. The tests were significant ( $p$ 's < 0.05) for most variables. In such cases, either Greenhouse-Geisser Epsilon (G-G) or the Huynh-Feldt Epsilon (H-F) corrected degrees of freedom  $F$ -tests may be used. However, significant experimental effects were well beyond the 0.05 probability level, with little difference between the uncorrected and the corrected  $F$ -tests. Similarly, if an experimental effect was non-significant, the probability values were greater than 0.05 for both uncorrected and corrected  $F$ -tests.

## **4.4 Reliability Analysis**

### ***4.4.1 Mean Torque***

The first five trials on the four days of testing were used for reliability analysis of criterion measures obtained during voluntary contractions. Tables 4 and 6 present the means, standard deviations and ANOVA *F*-ratios used to evaluate the stability of the measures for the control and experimental groups, respectively. The expected increase in mean maximal isometric torque associated with repeated strength assessment occurred for both the control (4.5 Nm, 23.4%) and experimental (3.1 Nm, 19.6%) groups. The intraclass correlation coefficients, true score error (between subjects), day-to-day error, and trial-to-trial error are reported in Tables 5 and 7. The lack of stability in means was compensated by highly consistent scores within subjects. The intraclass correlation coefficient was 0.80 for the control group and 0.94 for the experimental.

### ***4.4.2 Surface Electromyographic Activity***

From the first to last test session, the control (2.9%) and experimental (6.9%) groups exhibited significant increases in FCR sEMG RMS amplitude (see Tables 4 and 6). While statistically significant, the practical importance of such changes is debatable as the degrees of freedom for the *F*-test are quite large (McIntosh & Gabriel, 2012). Nevertheless, the consistency of participants within both groups was high (see Tables 5 and 7). Flexor carpi radialis sEMG RMS amplitude had an intraclass correlation coefficient of 0.84 for the control group and 0.92 for the experimental groups.

Both groups exhibited a decrease in ECR sEMG RMS amplitude across the four days. The decrease was significant for the experimental group, which experienced a

50.1% reduction in sEMG activity while the control group only underwent a 3.4% decrease. The slight decrease in means for the control group had a modest impact on the intraclass correlation coefficient. Participants in the control group were still considered very consistent with an intraclass correlation coefficient of 0.82 (Table 5).

It would appear that the lack of stability in ECR sEMG RMS means across days contributed to a reduction in the intraclass correlation coefficient in the experimental group. The observed intraclass correlation coefficient of 0.15 would normally be deemed too low for data analysis (Table 7). However, inspection of Figure 21 suggests that the intraclass correlation coefficient was highly influenced by homogeneous scores for the experimental group. In comparison to the control group, the effect can be observed as a greater clustering of individuals' means (circles) around the group mean (dotted line). The vertical lines are standard deviations of all the scores across days and trials for the individual participant to illustrate consistency. The decrease in means across days would increase the vertical bars to some degree more than the control group (the SEM was 0.24 mV rather than 0.20 mV). The slightly higher SEM does not account for the clustering of individual means (circles) around the group mean (dotted line), which lowers the between subjects mean squares (true score). The intraclass correlation coefficient is therefore artificially lower than what would normally be expected due to an experimental effect. Therefore, the ECR data was used for further analyses.

#### ***4.4.3 Evoked Potentials***

Both groups exhibited a significant decrease in maximal M-wave peak-to-peak (P-P) amplitude across the four test sessions. There was 12.8% decrease for the control group and a 12.5% decrease for the experimental group, see Tables 4 and 6, respectively.

While both groups exhibited a similar decrease in stability of the means across test days, the consistency of participants within groups was lower for the control ( $R=0.63$ ) than for the experimental ( $R=0.82$ ) group. Inspection of Tables 5 and 7 reveals that the control group (29.4%) had a lower true score variance than the experimental group (52.8%), respectively. Figure 22 shows that the lower intraclass correlation coefficient for the control group was due to more homogeneous scores. The effect is illustrated as a tighter clustering of the individual means (circles) around the group mean (dotted line) for the control group than for the experimental group. The analysis once again reveals that an experimental effect can decrease the stability of means across days while the consistency of scores within subjects can remain high, albeit somewhat lower due to an increase in the SEM reducing the true score variance.

The H-reflex could be evoked in only fourteen participants while the V-wave could be observed in only eight participants. Since the reasons for the low response rate are technical which are easily solved for future studies, a reliability analysis of these measures is warranted but the low numbers prevent hypothesis testing in the current study. Both groups exhibited significant alterations in H-reflex P-P amplitude across the four test days. There was a 41.3% decrease for the control group while the experimental group exhibited a 12.5% increase, see Tables 4 and 6, respectively. As discussed above, the decrease in stability of the means across test days had only a modest impact upon the consistency of the scores (see Tables 5 and 7). The intraclass correlation coefficient was 0.82 for the control group and 0.90 for the experimental group.

Similar to the H-reflex, the control and experimental groups exhibited significant alterations in the V-wave P-P amplitude but opposite in the direction of change. There

was an 11.3% decrease for the control group while the experimental group exhibited a 21.6% increase, see Tables 4 and 6, respectively. A decrease in stability of the means across test days has previously been shown to have a modest impact upon the consistency. However, it is important to note for future work, that the portion of the variance due to trial-to-trial error was higher in comparison to the other measures presented thus far (see Tables 5 and 7). As a result, the intraclass correlation coefficient was 0.75 for the control group and 0.79 for the experimental group. The consistency of participants within both groups would be considered sufficient to use the measure for further analysis had the response rates been greater.

Table 4. Analysis of variance for test days (1-3) and retention test (4) for the flexion group for torque, flexor carpi radialis surface electromyographic root-mean-square amplitude (FCR RMS), extensor carpi radialis surface electromyographic root-mean-square amplitude (ECR RMS), maximal M-wave peak-to-peak amplitude (M-wave P-P), H-reflex peak-to-peak amplitude (H-reflex P-P), and V-wave peak-to-peak amplitude (V-wave P-P).

		Torque (Nm)	FCR RMS ( $\mu$ V)	ECR RMS ( $\mu$ V)	M-wave P-P (mV)	H-reflex P-P (mV)	V-wave P-P (mV)
Test Day		M $\pm$ SD	M $\pm$ SD	M $\pm$ SD	M $\pm$ SD	M $\pm$ SD	M $\pm$ SD
1		14.4 $\pm$ 4.2	310.5 $\pm$ 188	136.6 $\pm$ 99	6.57 $\pm$ 2.12	0.63 $\pm$ 0.53	1.51 $\pm$ 1.14
2		17.8 $\pm$ 3.7	412.9 $\pm$ 327	139.4 $\pm$ 97	6.92 $\pm$ 2.64	0.55 $\pm$ 0.44	1.35 $\pm$ 0.77
3		18.7 $\pm$ 4.4	410.7 $\pm$ 282	141.1 $\pm$ 95	7.28 $\pm$ 3.40	0.45 $\pm$ 0.33	1.86 $\pm$ 2.10
4		18.9 $\pm$ 6.3	319.8 $\pm$ 150	132.0 $\pm$ 103	5.73 $\pm$ 3.17	0.37 $\pm$ 0.40	1.34 $\pm$ 0.36
Percent Change		23.8% (4.5)	2.9% (9.3)	-3.4% (-4.6)	-12.8% (-0.84)	-41.3% (-0.26)	-11.3% (-0.17)
ANOVA F-Ratios	<i>df</i>						
Days	3	119.99**	33.27**	0.49	26.36**	14.08** <sup>+</sup>	2.01 <sup>+</sup>
Subjects	11	143.71**	148.17**	62.62**	78.43**	65.23** <sup>+</sup>	23.10** <sup>+</sup>
Days $\times$ Subjects	33	20.62**	22.82**	12.46**	28.99**	11.49** <sup>+</sup>	6.66** <sup>+</sup>
Within Cells	192						

Significant difference between days, \* =  $p < 0.05$ , \*\* =  $p < 0.01$ . The following formula was used to calculate percent change between test session 1 and 4: Percent change =  $(1 - (\text{smaller number}/\text{larger number})) \times 100$  from Day 1 to Day 4. The voluntary surface electromyographic activity is in microvolts for precision in calculating percent changes. <sup>+</sup> denotes a difference in *df* because of a different sample size.

Table 5. Intraclass correlation analysis of variance for test days (1-3) and retention test (4) for the flexion group for torque, flexor carpi radialis surface electromyographic root-mean-square amplitude (FCR RMS), extensor carpi radialis surface electromyographic root-mean-square amplitude (ECR RMS), maximal M-wave peak-to-peak amplitude (M-wave P-P), H-reflex peak-to-peak amplitude (H-reflex P-P), and V-wave peak-to-peak amplitude (V-wave P-P).

Source	<i>df</i>	Torque	FCR RMS	ECR RMS	M-wave P-P	H-reflex P-P	V-wave P-P
<sup>MS</sup> Subjects	11	314.66	0.84	0.12	78.43	2.45 <sup>+</sup>	17.17 <sup>+</sup>
<sup>MS</sup> Days within Subjects	36	63.28	0.13	0.02	28.77	0.44 <sup>+</sup>	4.26 <sup>+</sup>
<sup>MS</sup> Within Cells	192	2.19	0.01	0.0002	0.25	0.04 <sup>+</sup>	0.74 <sup>+</sup>
$(\sigma_{e_1}^2 - Trials)$		2.19 (8.1%)	0.01 (8.5%)	0.002 (17.2%)	0.25 (2.9%)	0.04 (17.1%)	0.74 (35.5%)
$(\sigma_{e_2}^2 - Days)$		12.12 (45.3%)	0.03 (38.9%)	0.004 (37.0%)	5.70 (67.6%)	0.08 (37.1%)	0.70 (30.9%)
$(\sigma_{true}^2 - True)$		12.57 (46.6%)	0.04 (52.9%)	0.005 (45.3%)	2.48 (29.4%)	0.10 (45.8%)	0.65 (30.9%)
Grand Mean		17.45 Nm	0.36 mV	0.14 mV	6.63 mV	0.50 mV	1.51 mV
SEM		3.47 Nm	0.16 mV	0.07 mV	2.43 mV	0.26 mV	0.75 mV
R		0.80	0.84	0.82	0.63	0.82	0.75

<sup>+</sup> denotes a difference in *df* because of a different sample size.

Table 6. Analysis of variance for test days (1-3) and retention test (4) for the PNF group for torque, flexor carpi radialis surface electromyographic root-mean-square amplitude (FCR RMS), extensor carpi radialis surface electromyographic root-mean-square amplitude (ECR RMS), maximal M-wave peak-to-peak amplitude (M-wave P-P), H-reflex peak-to-peak amplitude (H-reflex P-P), and V-wave peak-to-peak amplitude (V-wave P-P).

		Torque (Nm)	FCR RMS ( $\mu$ V)	ECR RMS ( $\mu$ V)	M-wave P-P (mV)	H-reflex P-P (mV)	V-wave P-P (mV)
Test Day		M $\pm$ SD	M $\pm$ SD	M $\pm$ SD	M $\pm$ SD	M $\pm$ SD	M $\pm$ SD
1		12.7 $\pm$ 4.7	322.9 $\pm$ 224	137.7 $\pm$ 51	7.37 $\pm$ 3.53	0.70 $\pm$ 0.46	0.91 $\pm$ 0.37
2		13.8 $\pm$ 5.1	397.8 $\pm$ 378	113.9 $\pm$ 51	7.69 $\pm$ 3.78	1.36 $\pm$ 2.01	1.23 $\pm$ 0.38
3		15.0 $\pm$ 5.1	341.9 $\pm$ 280	88.4 $\pm$ 42	5.18 $\pm$ 2.08	0.96 $\pm$ 1.53	0.96 $\pm$ 0.78
4		15.8 $\pm$ 6.1	346.9 $\pm$ 307	68.7 $\pm$ 25	6.45 $\pm$ 4.27	0.80 $\pm$ 0.84	1.16 $\pm$ 0.12
Percent Change		19.6% (3.1)	6.9% (24)	-50.1% (-62.7)	-12.5% (-0.92)	12.5% (0.10)	21.6% (0.25)
ANOVA F-Ratios	<i>df</i>						
Days	3	59.61**	7.92**	63.86**	76.23**	41.72** <sup>+</sup>	1.44 <sup>+</sup>
Subjects	11	260.53**	188.99**	16.50**	167.95**	453.97** <sup>+</sup>	10.34** <sup>+</sup>
Days $\times$ Subjects	33	9.24**	15.52**	9.46**	26.10**	47.71** <sup>+</sup>	2.55** <sup>+</sup>
Within Cells	192						

Significant difference between days, \* =  $p < 0.05$ , \*\* =  $p < 0.01$ . The following formula was used to calculate percent change between test session 1 and 4: Percent change =  $(1 - (\text{smaller number}/\text{larger number})) \times 100$  from Day 1 to Day 4. The voluntary surface electromyographic activity is in microvolts for precision in calculating percent changes. <sup>+</sup> denotes a difference in *df* because of a different sample size.

Table 7. Intraclass correlation analysis of variance for test days (1-3) and retention test (4) for the PNF group for torque, flexor carpi radialis surface electromyographic root-mean-square amplitude (FCR RMS), extensor carpi radialis surface electromyographic root-mean-square amplitude (ECR RMS), maximal M-wave peak-to-peak amplitude (M-wave P-P), H-reflex peak-to-peak amplitude (H-reflex P-P), and V-wave peak-to-peak amplitude (V-wave P-P).

Source	<i>df</i>	Torque	FCR RMS	ECR RMS	M-wave P-P	H-reflex P-P	V-wave P-P
<sup>MS</sup> Subjects	11	503.97	1.47	0.01	167.95	27.71 <sup>+</sup>	2.61 <sup>+</sup>
<sup>MS</sup> Days within Subjects	36	26.00	0.12	0.01	30.28	2.85 <sup>+</sup>	0.55 <sup>+</sup>
<sup>MS</sup> Within Cells	192	1.93	0.10	0.001	0.12	0.06 <sup>+</sup>	0.25 <sup>+</sup>
$(\sigma_{e_1}^2 - Trials)$		1.93 (6.31%)	0.10 (8.0%)	0.001 (26.9%)	0.12 (0.92%)	0.06 (3.3%)	0.25 (60.8%)
$(\sigma_{e_2}^2 - Days)$		4.81 (15.71%)	0.02 (22.3%)	0.02 (69.8%)	6.03 (46.3%)	0.56 (30.0%)	0.06 (14.4%)
$(\sigma_{true}^2 - True)$		23.90 (77.98%)	0.08 (69.7%)	0.0001 (3.4%)	6.88 (52.8%)	1.24 (66.8%)	0.10 (24.8%)
Grand Mean		14.31 Nm	0.35 mV	0.10 mV	6.67 mV	0.96 mV	1.07 mV
SEM		2.90 Nm	0.17 mV	0.13 mV	6.97 mV	0.52 mV	0.29 mV
R		0.95	0.92	0.15	0.82	0.90	0.79

<sup>+</sup> denotes a difference in *df* because of a different sample size.

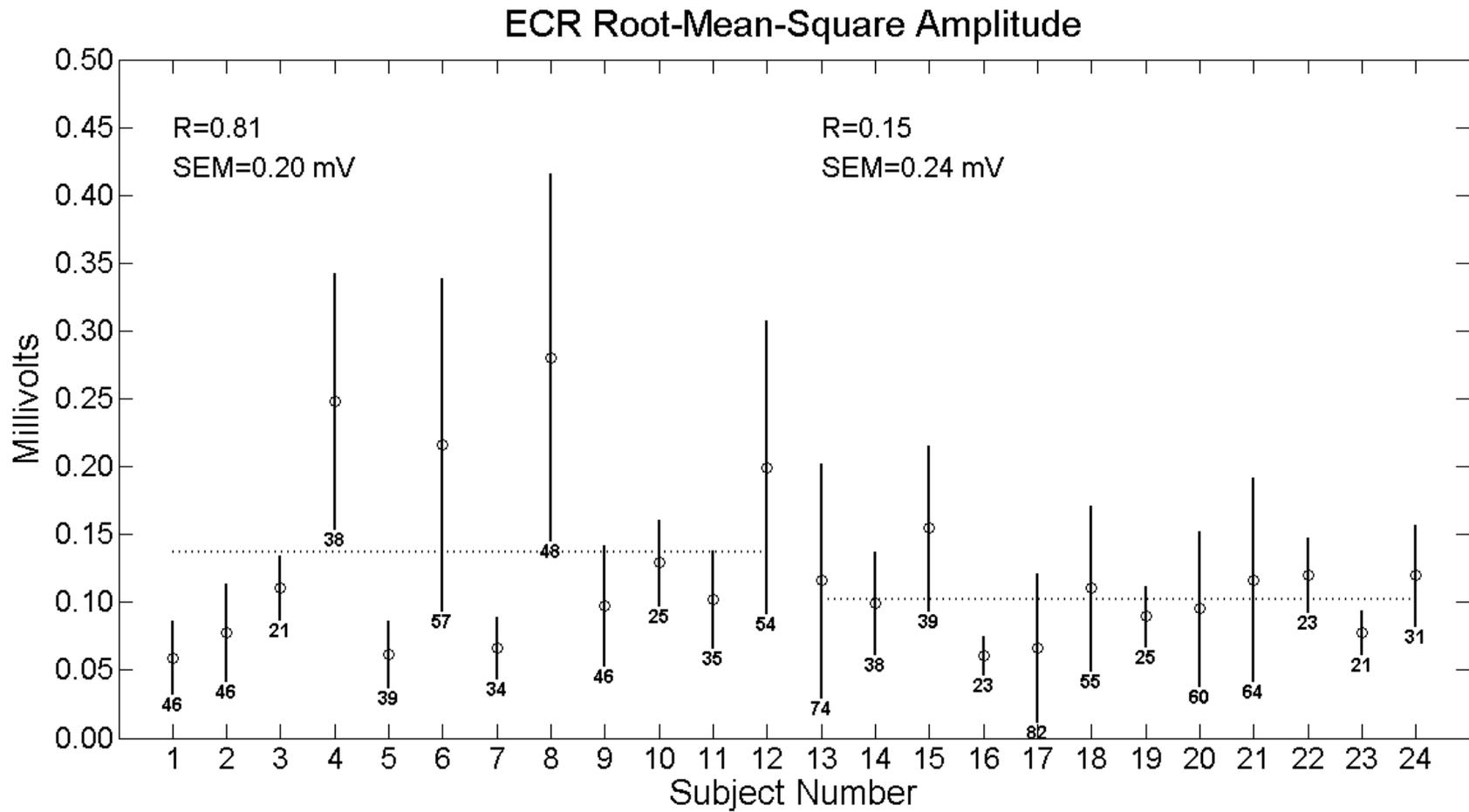


Figure 21. Means and standard errors for ECR sEMG RMS amplitude across both groups. Subjects 1-12 are the control group, while 13-24 are the experimental group.

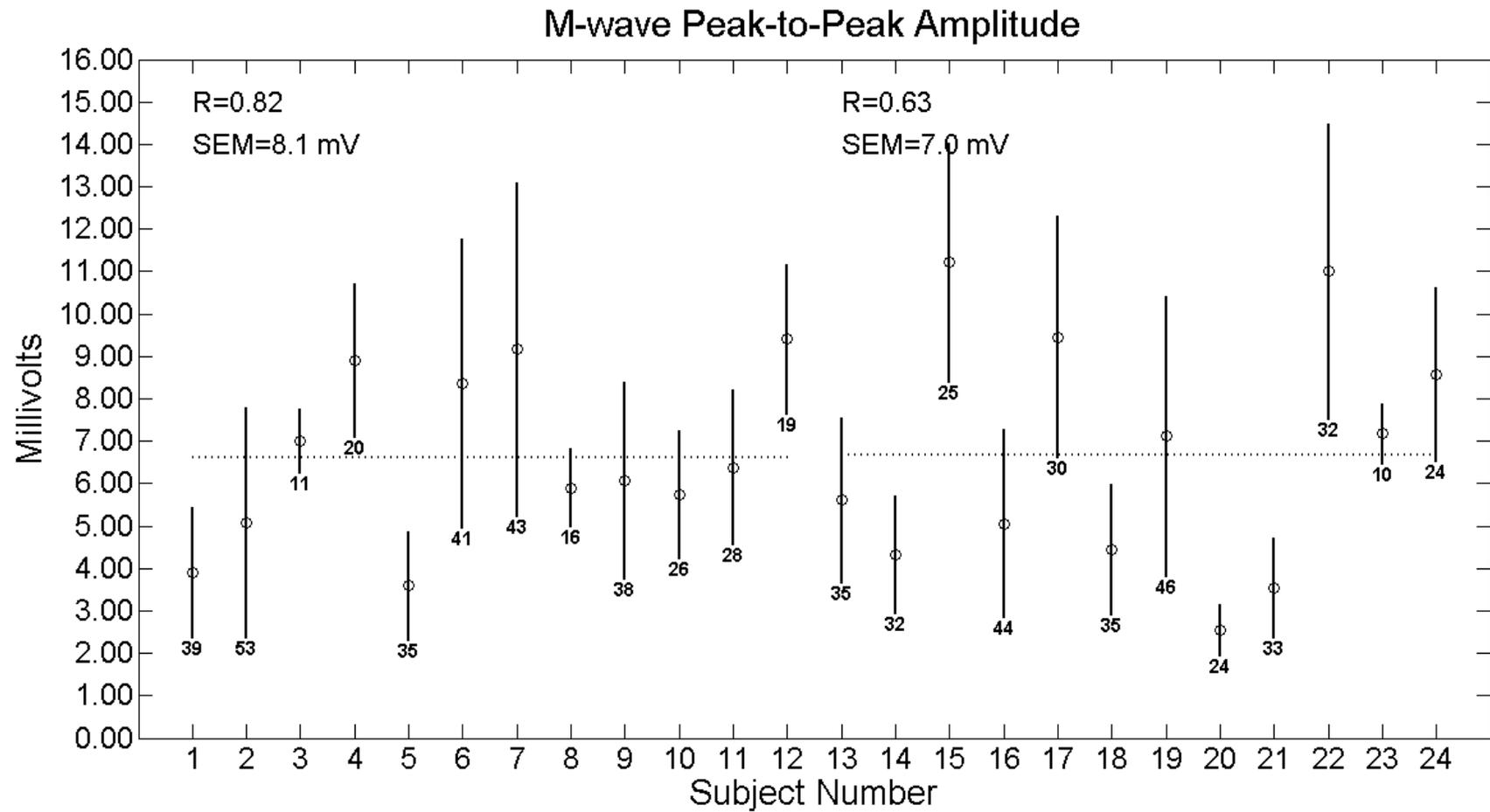


Figure 22. Means and standard errors for M-wave peak-to-peak amplitude across both groups. Subjects 1-12 are the control group, while 13-24 are the experimental group.

## 4.5 Magnitude Variables

On the first three testing days, participants performed ten maximal voluntary isometric contractions of the pattern they were assigned: either a 5-second isometric wrist flexion contraction or a 5-second isometric wrist extension contraction immediately followed by 5-second isometric wrist flexion contraction. During the retention test, performed two-weeks after session three, participants performed five contractions consisting of the pattern they were assigned (retention test), followed by five of the opposite contraction pattern (transfer test). The first five contractions from days 1-3 were assessed, as well as the five retention and five transfer contractions from day 4. For statistical analysis, the data were divided into five blocks. Blocks 1-3 represent the five contractions completed on the first three days of testing, while Block 4 consists of the five retention contractions and Block 5 the five transfer contractions. For each measure, a split-plot factorial ANOVA was performed to assess differences between the two groups. If the omnibus  $F$ -test was significant, orthogonal contrasts were performed to assess changes in performance between Block 3 and 4, retention on Block 4 relative to Block 1, and transfer on Block 5 relative to Block 1. The probability level was Bonferoni-corrected for each successful comparison.

### 4.5.1 Mean Torque

The  $p$ -value was non-significant for both the Groups main effect ( $p > 0.05$ ) and the Group  $\times$  Blocks ( $p > 0.05$ ) interaction term. Before examining the significant main effect for Blocks ( $p < 0.05$ ), it was important to determine if there was a difference in mean strength level between groups. The grand mean difference between groups was 3.12 Nm (18%), which is not trivial for wrist flexion strength and might lead to the

suspicion that the study is underpowered. However, as will be detailed in subsequent paragraphs, the sEMG magnitudes for both groups were nearly identical and the addition of a large number of participants would “not” result in significant differences. The differences in mean strength level between groups were most like due to the matching based on predicted strength (see Figure 23). The prediction equation had a standard error of estimate of 2.43 Nm. Further, the differences between groups on the first test session, while non-significant, was 1.75 Nm (12%). The data were therefore collapsed across groups for further analysis of the Blocks main effect.

The means and standard errors for each group across the five blocks can be seen in Figure 24. Orthogonal contrasts showed a significant ( $p < 0.01$ ) increase from Block 1 to the retention test on Block 4 (3.81 Nm, 20.2%). The transfer test on Block 5 was also significantly ( $p < 0.01$ ) greater (2.41 Nm, 15.1%) than Block 1. There was no significant difference ( $p > 0.05$ ) between Blocks 3 and 4.

#### ***4.5.2 Surface Electromyographic Activity***

Figure 25 illustrates the means and standard errors for both FCR and ECR sEMG RMS amplitude for both groups. Similar to mean torque, the Group main effect ( $p > 0.05$ ) and the Group  $\times$  Blocks ( $p > 0.05$ ) interaction term were non-significant for the FCR sEMG RMS amplitude. There was, however, a slight decrease in FCR sEMG RMS amplitude amounting to 8.2% (0.026 mV, 26  $\mu$ V) for the Blocks main effect that was of interest to explore further ( $p > 0.05$ ). The FCR sEMG RMS amplitude increased across the first three blocks then decreased until Block 5. The overall pattern of means followed a quadratic trend that accounted for 64.2% of the variance ( $p < 0.05$ ). While the quadratic

trend was significant, orthogonal contrasts detected no significant differences between individual means.

The Group main effect ( $p > 0.05$ ) and the Group  $\times$  Blocks ( $p > 0.05$ ) interaction term were non-significant for ECR sEMG RMS. There was a significant main effect for Blocks ( $p < 0.05$ ) that was explored further with orthogonal contrasts. The sEMG RMS amplitude of the ECR exhibited a 19.5% (0.0224 mV or 22.4  $\mu$ V) decrease from Block 1 to the retention test on Block 4 ( $p < 0.05$ ). The decrease in sEMG RMS amplitude continued for a total of 59.7% (0.0513 mV or 51.3  $\mu$ V) between Block 1 and the transfer test on Block 5 ( $p < 0.01$ ). There was no significant difference ( $p > 0.05$ ) between Blocks 3 and 4.

#### ***4.5.3 Muscle Coordination***

To assess potential changes in muscle coordination across the blocks, coactivation ratios were calculated by dividing the ECR sEMG RMS amplitude (antagonist) by the FCR sEMG RMS amplitude (agonist). The means and standard errors are presented in Figure 26. The Group main effect ( $p > 0.05$ ) and the Group  $\times$  Blocks interaction term ( $p > 0.05$ ) were non-significant for coactivation ratio. There was a significant main effect for Blocks ( $p < 0.05$ ) that was explored further with orthogonal contrasts. The coactivation ratio exhibited a 36.1% decrease from Block 1 to the retention test on Block 4 ( $p < 0.01$ ). The decrease in coactivation ratio was maintained so that there was a 35.2% reduction between Block 1 and the transfer test on Block 5 ( $p < 0.05$ ). No significant difference was observed between Blocks 3 and 4 ( $p > 0.05$ ).

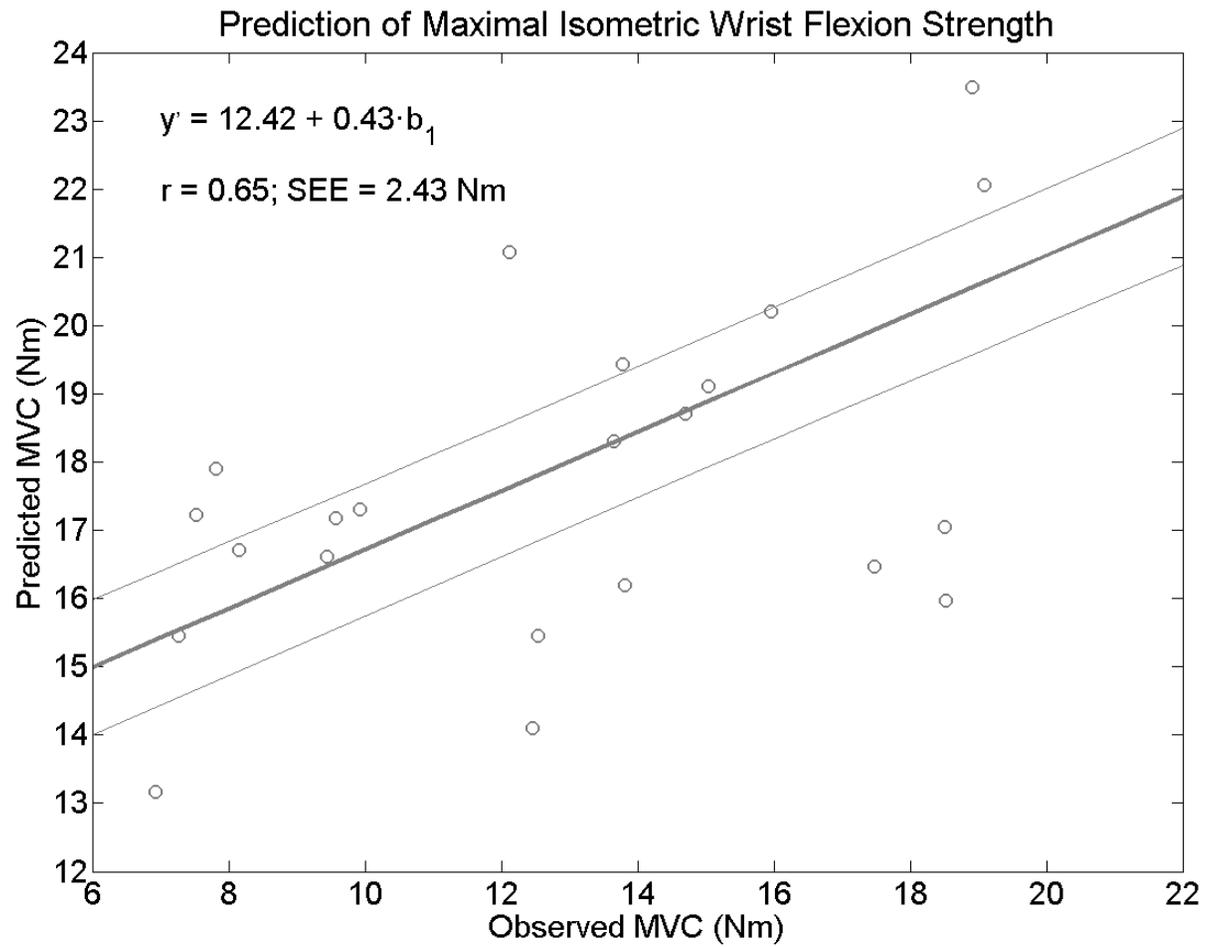


Figure 23. Correlation between predicted peak torque and observed peak torque on day 1.

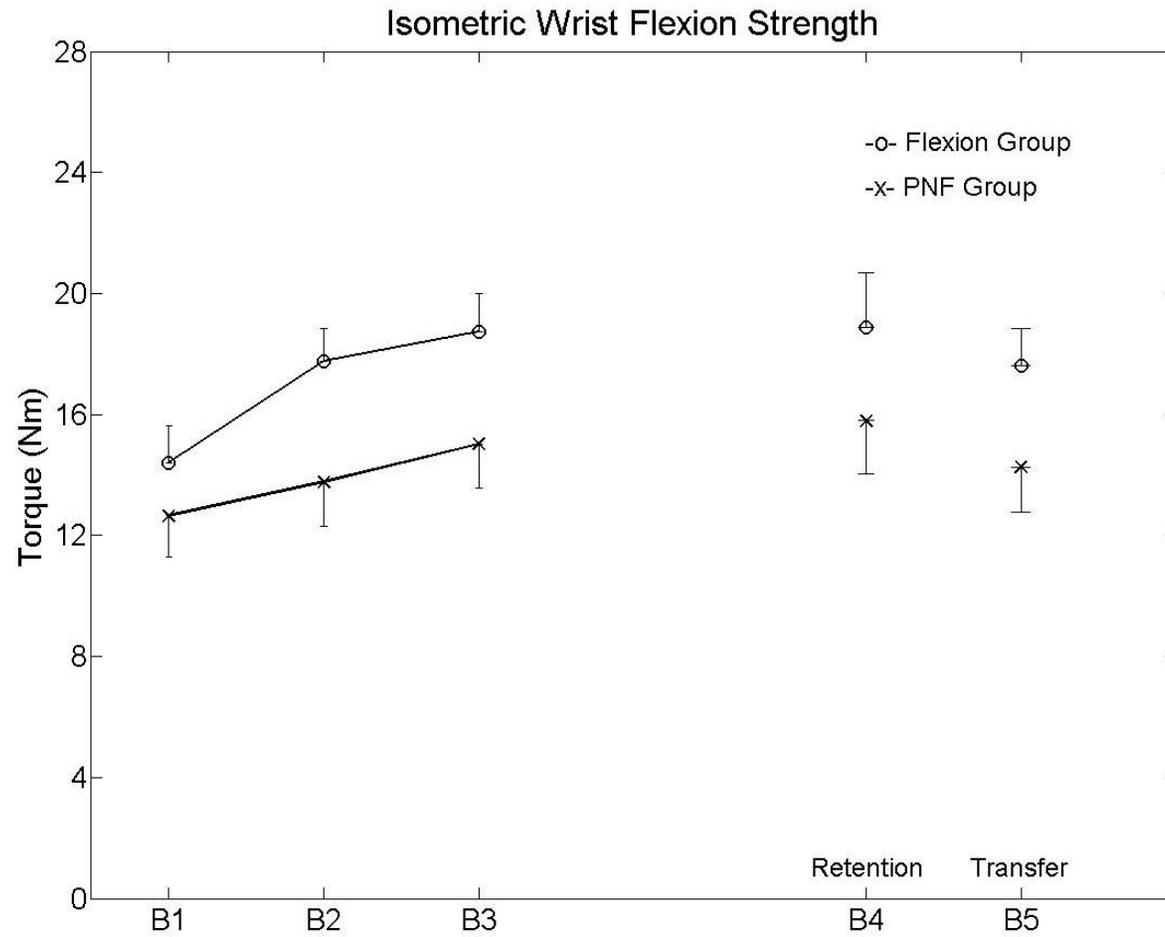


Figure 24. Mean torque for both the flexion and PNF groups across the five blocks. Each block is a mean of five contractions. Standard errors are represented by the vertical lines.

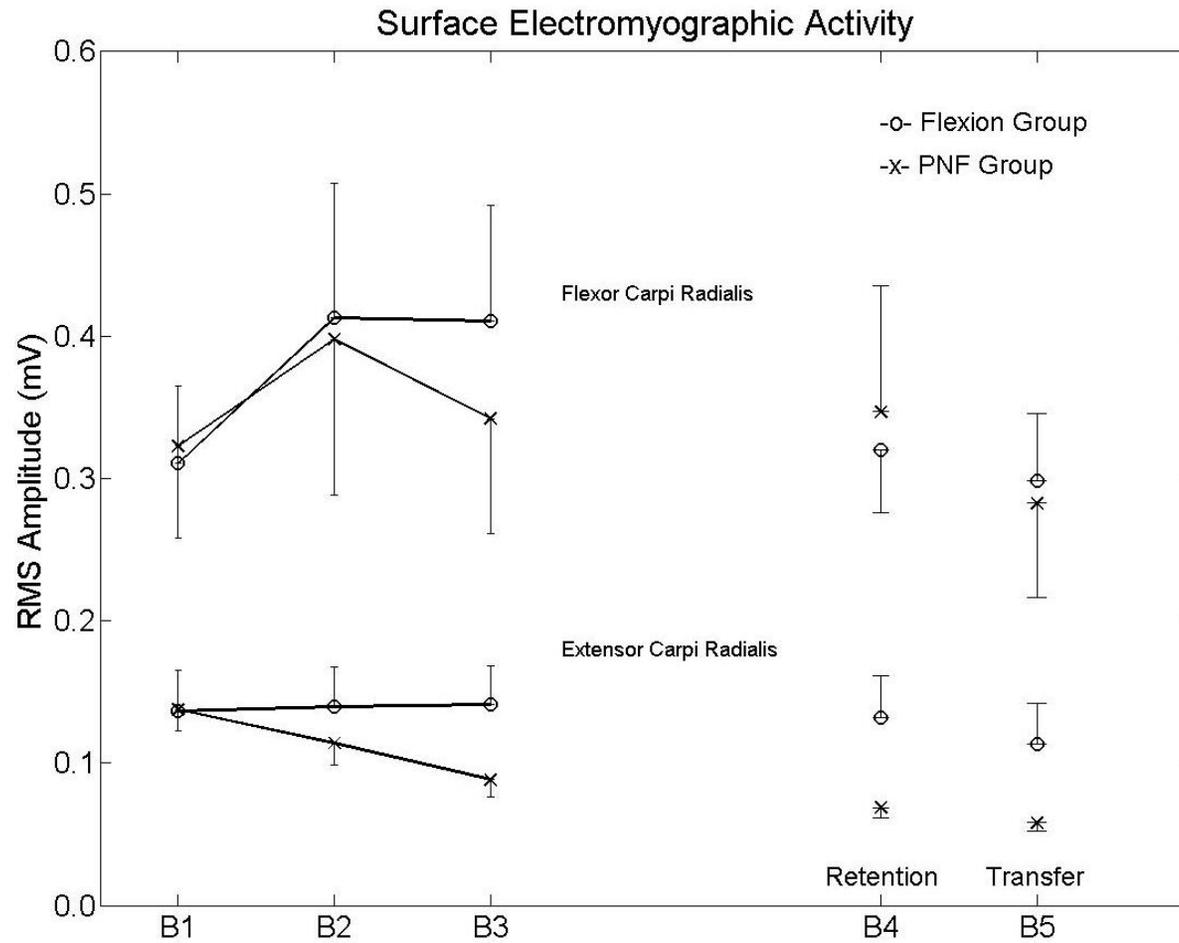


Figure 25. Surface electromyographic RMS amplitude for both the flexion and PNF groups across the five blocks. Each block is the mean of five contractions. Standard errors are represented by the vertical lines.

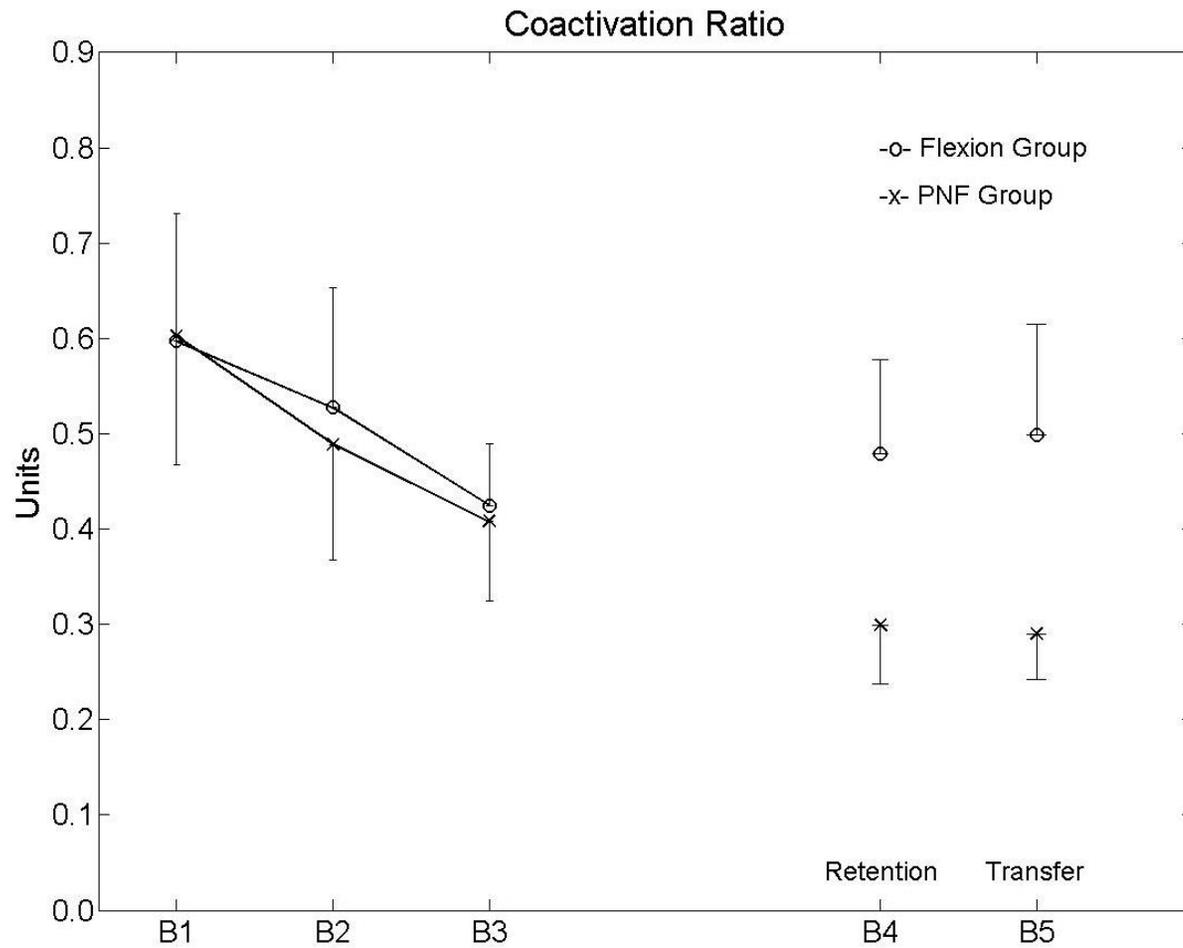


Figure 26. Coactivation ratios for both the flexion and PNF groups across the five blocks. Each block is the mean of five contractions.

Standard errors are represented by the vertical lines.

## 4.6 Variability

Target variability was defined as the variability in maintaining a constant torque and represents the variability of the horizontal portion of the torque trace itself, not relative to the horizontal target line. During each test session, participants were instructed to try as hard as possible to maintain a steady force during each contraction, therefore normalized RMS error of the middle 3.5 seconds of the contraction was calculated to assess the variability between days in maintaining a steady torque. The variance ratio (VR) was calculated for the torque trace, as well as for both the FCR and ECR sEMG waveforms. The VR quantifies the variability in the shape of the entire waveform and can provide insight into the stability of motor performance (Figure 27). Split-plot factorial ANOVAs were used to assess differences between the two groups for the measures of variability

### 4.6.1 Target Variability

The means and standard errors for normalized RMS error for each group are depicted in Figure 28. The Group main effect ( $p > 0.05$ ) and the Group  $\times$  Blocks interaction term ( $p > 0.05$ ) were non-significant for RMS error. There was a significant main effect for Blocks ( $p < 0.01$ ) that was explored further with orthogonal contrasts. The RMS error exhibited a 30.5% decrease from Block 1 to the retention test on Block 4 ( $p < 0.01$ ). The reduction in RMS error continued so that a 26.2% difference was observed between Block 1 and the transfer test on Block 5 ( $p < 0.01$ ). No significant difference was observed between Blocks 3 and 4 ( $p > 0.05$ ).

## 4.6.2 Motor Output Variability

### 4.6.2.1 Torque Variance Ratio

Figure 29 shows the means and standard errors for the torque VR. There was no main effect for Groups ( $p > 0.05$ ) but the Group  $\times$  Blocks interaction term was significant ( $p < 0.05$ ). Orthogonal polynomials were used to analyze differences in trends for the means across blocks between the two groups. The analysis is more efficient because it does not involve numerous comparisons to describe the underlying interaction effects. The control exhibited a 51.6% decrease in torque VR between Block 1 and 4. However, the transfer condition resulted in an increase in torque VR so that there was only a 1.1% difference between Blocks 1 and 5. The result was a significant quadratic trend that accounted for 98% of variance in means across blocks ( $p < 0.01$ ). In contrast, the experimental group exhibited a 69.3% decrease in torque VR from Block 1 to 5. The linear trend component was significant, which accounted for 75.1% of the variance in means across blocks ( $p < 0.01$ ).

### 4.6.2.2 Surface Electromyographic Variance Ratio

The variance ratios for the FCR sEMG waveform followed the same pattern as means for the torque VR (see Figure 30). The Group main effect was not significant ( $p > 0.05$ ) but the Group  $\times$  Block interaction term was significant ( $p < 0.05$ ). The control group exhibited a 15.0% decrease in FCR VR between Block 1 and 4. However, the transfer condition resulted in an increase in FCR VR so that there was only a 0.5% difference between Blocks 1 and 5. The result was a non-significant quadratic trend that accounted for 80% of variance in means across blocks ( $p > 0.05$ ). In contrast, the experimental group exhibited a 17.6% decrease in torque VR from Block 1 to 5. Only the

linear trend component was significant, which accounted for 90% of the variance in means across blocks ( $p < 0.05$ ).

Alterations in the variance ratios for the ECR sEMG waveform are presented in Figure 31. The control group had a lower (12.3%) ECR VR than the experimental group, resulting in a probability value of  $p=0.05$ . The Group  $\times$  Block interaction term had a probability value of  $p < 0.05$  that was explored further with orthogonal polynomials for trend analysis.

The control group exhibited a 9.4% decrease in ECR VR between Block 1 and 4. However, the transfer condition resulted in an increase in ECR VR beyond Block 1; there was only a 1.8% difference between Blocks 1 and 5. The result was a significant quadratic trend that accounted for 67% of variance in means across blocks ( $p > 0.05$ ). In contrast, the experimental group exhibited a 12.1% increase in ECR VR from Block 1 to the retention test on Block 4. There was then a 15.1% decrease in ECR VR for the transfer test on Block 5. The pattern of means across blocks therefore followed a quadratic trend that accounted for 87.1% of the variance ( $p < 0.05$ ).

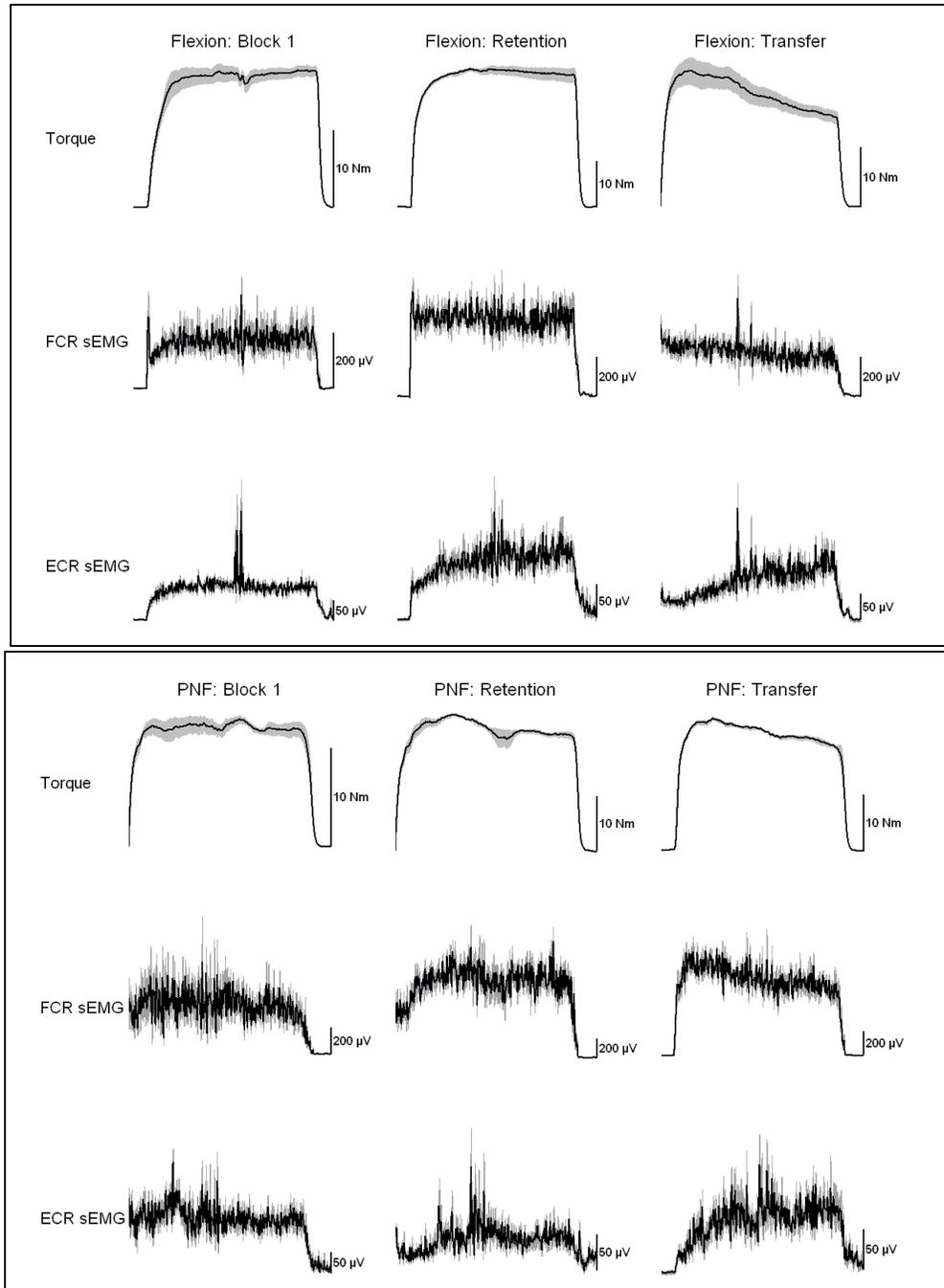


Figure 27. Sample force and sEMG traces collected during Block 1, the retention test, and the transfer test for two participants, one from the flexion group and the other from the PNF group. Gray shading represents plus-minus one standard error. Figures provide a qualitative view of motor output variability.

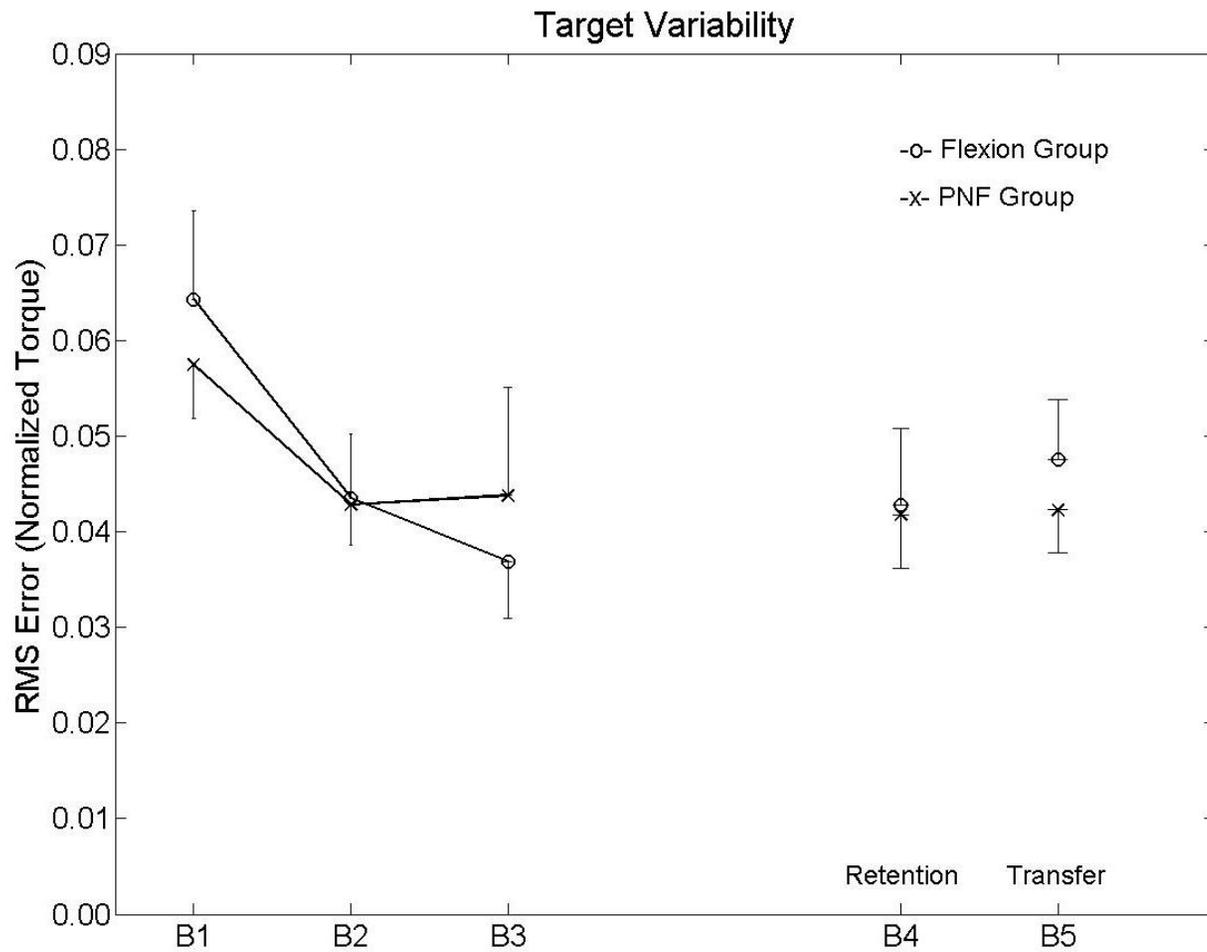


Figure 28. Normalized RMS error for target variability across the five blocks. Each block is the mean of five contractions. Standard error is denoted by the vertical lines.

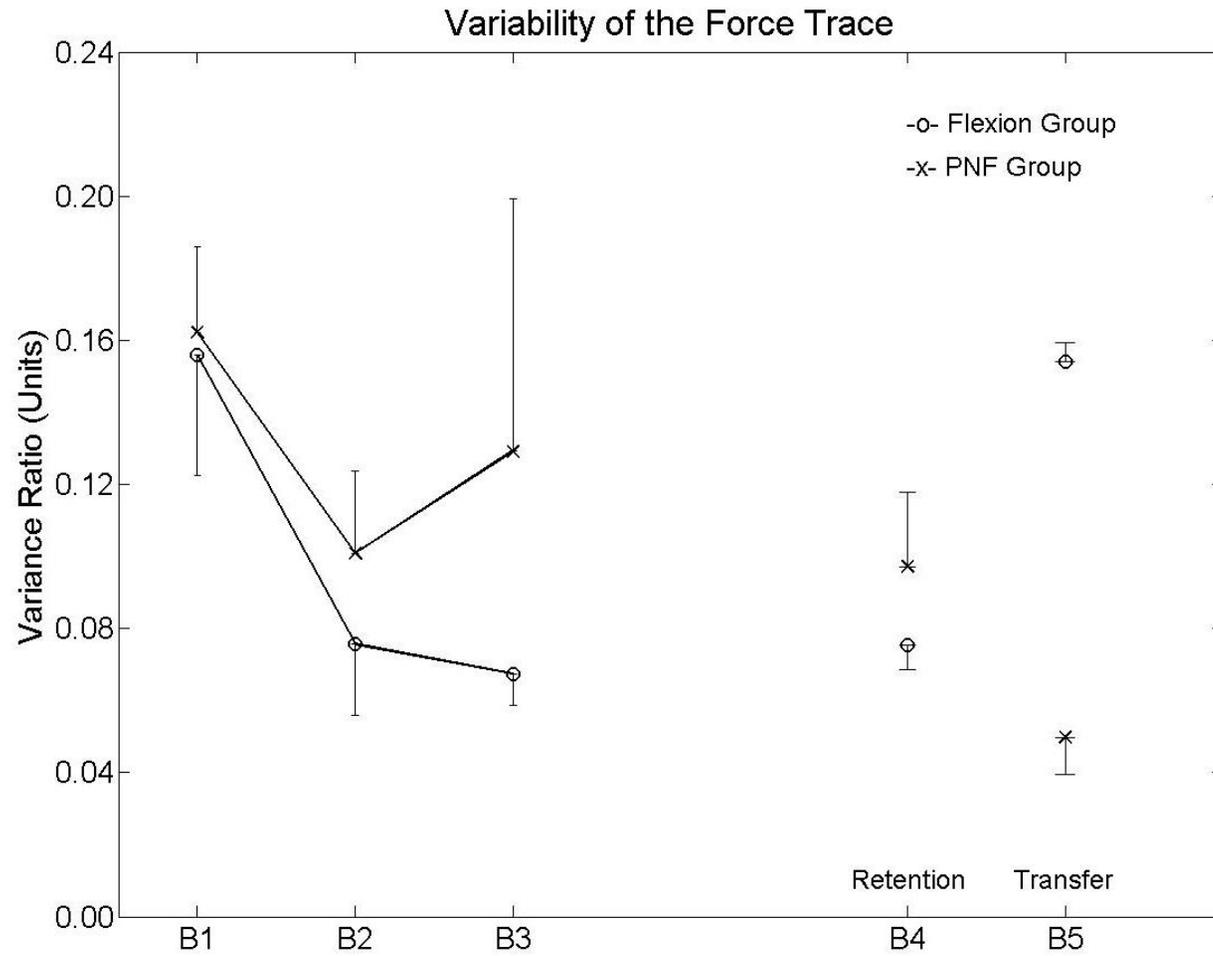


Figure 29. Torque waveform variability as measured by variance ratios across the five blocks. Each block is a mean of five contractions. Standard error is denoted by the vertical lines.

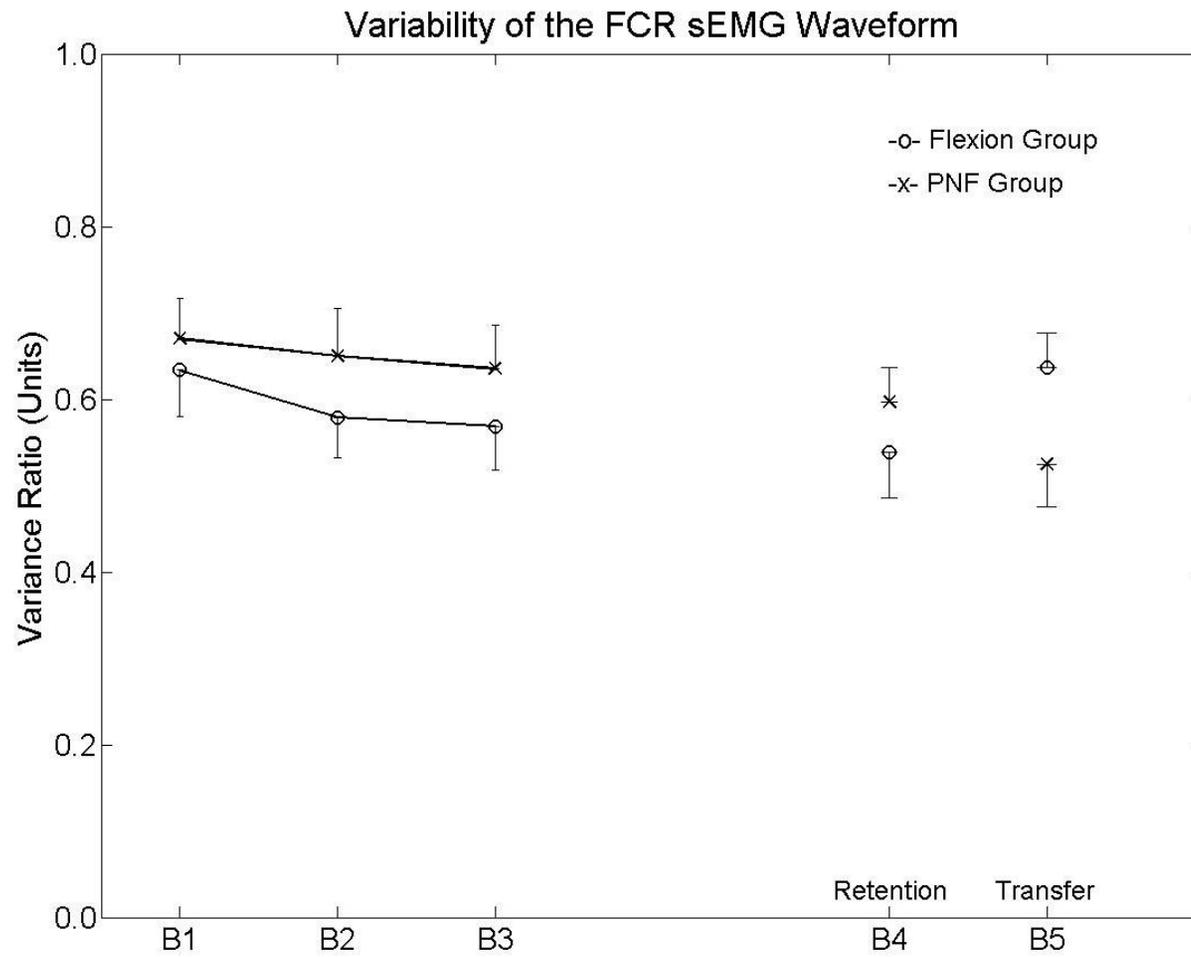


Figure 30. Flexor carpi radialis sEMG variability as measured by variance ratios across the five blocks. Each block is the mean of five contractions. Standard error is denoted by the vertical lines.

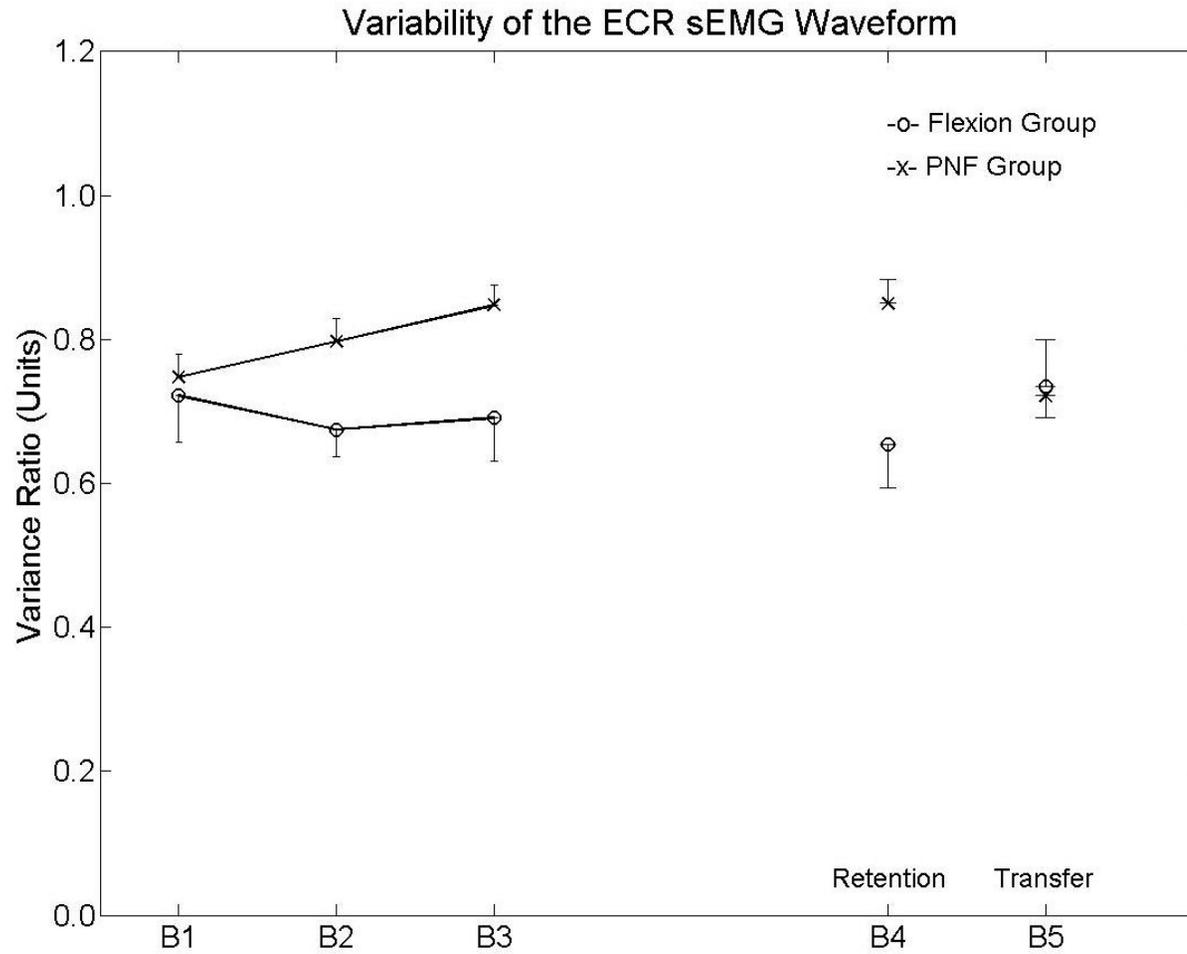


Figure 31. Extensor carpi radialis variability as measured by variance ratios across the five blocks. Each block is a mean of five contractions. Standard error is denoted by the vertical lines.

## CHAPTER 5: DISCUSSION

In the present study, the control group performed maximal voluntary isometric contractions of the wrist flexors, while participants in the experimental group completed a maximal isometric contraction of the wrist extensors immediately prior to an isometric contraction of the flexors. The wrist extension-to-flexion contraction pattern was theorized to result in proprioceptive neuromuscular facilitation (PNF). However, it was equally possible that the “reversal of antagonists” contraction pattern interfered with motor learning-related increases in strength. For the purposes of this study, motor learning was defined as a change in strength, coactivation level, and/or variability of the task performance that occurred during the initial learning phase (first three test sessions) and was retained during the 2-week retention test and/or transfer test.

Participants ( $N=24$ ) were matched in pairs based on predicted strength and randomly assigned to either the control or experimental group. All participants completed four testing sessions; the first three occurring during a one-week period (48 hours between each session) and the fourth two-weeks after the third session. The first three sessions were identical for all participants with the exception of the contraction pattern performed. Participants performed ten maximal voluntary isometric wrist contractions, as assigned, during each session. Retention and transfer tests were administered in succession during the fourth session, two-weeks later. The control and experimental conditions were crossed during the transfer test: the control group performed the “reversal of antagonists” contraction pattern and the experimental group completed agonist only contractions. The transfer test was used to explore if any motor-learning adaptations that occurred during the learning phases and retained during the retention

test, could be transferred to a different type of contraction pattern. Results from the transfer test may provide insight into the functional significance of each contraction pattern and whether or not it would be useful to retain in rehabilitation programs.

The contraction pattern had no effect on the “quick jumps in strength” phenomenon that is due solely to the administration of maximal isometric contractions for strength assessment (Calder & Gabriel, 2007; Kroll, 1963a; McIntosh & Gabriel, 2012). There were no differences in the magnitude of muscle activity, and both groups exhibited the same motor learning-related decreases in coactivation. As well, both groups exhibited similar decreases in task variability (RMS error). The alterations outlined above were retained over a two-week period, as there were no significant differences between the final testing session (Block 3) and the retention test (Block 4). As well, these alterations were present during the transfer task when the conditions were crossed. The groups did however exhibit differences in motor output variability (VRs). When required to perform the reversal of antagonists contraction pattern the control group underwent an increase in variability whereas the experimental group had a pronounced decrease in variability with agonist only contractions. The FCR VR (agonist) followed the same trends as the torque VR, while the most pronounced differences were with respect to the ECR VR (antagonist). For ECR VR, the two groups exhibited opposite adaptations. The control group had a decrease in variability until participants performed the reversal of antagonists contraction during the transfer test, while the experimental group had an increase in variability until participants performed agonist only contractions during the transfer test.

## 5.1 Reliability

Reliability analyses were performed on each group, to assess the stability and consistency of each measure. The following paragraphs will focus on the reliability results for only those measures used for hypothesis testing. The intraclass correlation coefficient (ICC) for mean torque across trials and days was 0.80 for the control and 0.95 for the experimental groups, suggesting good consistency. These values are within the range (0.93-0.95) of what has been previously reported (Kroll 1962; 1963a; 1963b) for the wrist flexors over multiple studies.

Intraclass correlation coefficients for FCR sEMG RMS amplitude were 0.84 and 0.92 for the control and experimental groups, respectively. El Bouse, Gabriel, and Tokuno (2013) reported a comparably high ICC of  $R=0.95$ . In contrast, Barr et al. (2001) reported an ICC of  $R=0.37$ . Differences between values could be attributed to the fact Barr et al. (2001) performed reliability analysis on log-transformed signals that were normalized with respect to the maximal voluntary contraction (MVC). Normalization alters the “spread of scores”, decreasing the between subjects variance, producing the same effect depicted in Figure 21. The result is an artificially lower ICC as described in Section 4.4.2.

Intraclass correlation coefficients for ECR sEMG RMS amplitude were 0.82 and 0.15 for the control and experimental groups, respectively. The explanation for the low ICC value for this measure can be found in Section 4.4.2, as well as justification for using this measure in the final analysis. For the control group, true score variance accounted for 45.3% of the total variance, whereas for the experimental group it only accounted for 3.4% of the total variance. The experimental group was naturally more homogenous with

respect to ECR sEMG RMS amplitude, and it underwent systematic changes across test sessions suggestive of experimental effect “not” error. The two factors combined so that the majority of variance (69.8%) for the experimental group was day-to-day error. The intraclass correlation coefficient for the experimental group is similar to the ICC ( $R=0.22$ ) reported by Barr et al. (2001) who produced the same effect through normalization.

## 5.2 A Comparison of Normal Values

In the current study, isometric wrist flexion torque was  $15.9 \pm 5.4$  Nm. These values are similar to those previously reported for college-age males:  $11.3 \pm 3.0$  Nm (Al-Eisawi et al., 1998),  $14.81 \pm 5.2$  Nm (Vanswearingen, 1983),  $13.7 \pm 3.5$  Nm (Seo et al., 2008). Other investigators have reported higher values:  $25 \pm 6$  Nm (Harbo, Brincks, & Anderson, 2012),  $24.9 \pm 5.9$  Nm (Salonikidis et al., 2009), and  $25.5 \pm 6.1$  Nm (Salonikidis et al., 2011). Differences between studies may be attributed to differences in test position and isolation of the joint within the apparatus. Static forearm position in pronation/supination and flexion/extension can affect muscle length and moment arms resulting in large differences in wrist joint torque (Buchanan et al., 1993; Gonzalez, Buchanan, & Delp, 1997). The mechanics of the task and the recruitment of additional muscle synergies can also be affected by whether or not participants “gripped” a handle or performed isometric contractions with an open hand to minimize involvement of the fingers (Sanes, 1986; Hallbeck, 1994; Leger & Milner, 2000).

There is one study on maximal isometric wrist flexion strength that has reported non-normalized sEMG values. Flexor carpi radialis sEMG RMS amplitude was  $344.5 \pm 265$   $\mu$ V during maximal isometric wrist flexion, which is comparable to  $420 \pm 90$   $\mu$ V

reported by Mizuno, Secher, and Quistorff (1994). To offer a further basis of comparison, Axelson (2005) observed an activation level of 290  $\mu\text{V}$  (129-806  $\mu\text{V}$ ) during maximal effort, ballistic wrist flexion. Axelson (2005) is also the only investigator to provide sEMG activity levels for the ECR. The current study observed an ECR sEMG RMS amplitude of  $113.0 \pm 84 \mu\text{V}$ , which is within the range of 36  $\mu\text{V}$  (1-141  $\mu\text{V}$ ) reported by Axelson (2005). The lack of comparative values for sEMG encouraged an additional examination of the FCR maximal M-wave peak-to-peak (P-P) amplitude data, although it was not used in the analyses. The P-P amplitude of the maximal M-wave was  $6.65 \pm 3.2 \text{ mV}$ , which is within the range of  $4.41 \pm 0.87 \text{ mV}$  (2.18–6.10 mV) reported by Christie et al. (2005).

### **5.3 Hypotheses for the Magnitude Variables**

The previous section showed that the current thesis has added value in providing non-normalized sEMG means, standard deviations, and intraclass reliability coefficients for the FCR and ECR necessary for pre-experimental planning of the appropriate sample size for future experiments using a maximal isometric wrist flexion model. The data were reliable and the values are within expected ranges as reported within the literature. The following paragraphs and sections, will discuss the main hypotheses for the magnitude variables. It is important to remember that that the retention (Block 4) and transfer (Block 5) tests were administered without any feedback.

#### ***5.3.1 Mean Torque***

Both groups exhibited the “quick jumps in strength” phenomenon first demonstrated by Kroll in 1962. That is, a measurement schedule consisting of the administration of maximal isometric contractions for strength assessment, which results

in a significant increase in strength in the absence of any other training. Hellebrandt (1958) proposed that motor learning plays a significant role in strength development as early as 1958, but Kroll (1962) provided evidence that it may actually contaminate baseline measures prior to resistive exercise intervention. As a result, a familiarization period is necessary to subtract-out motor learning effects from intervention studies (Calder & Gabriel, 2007; Green, Parro, & Gabriel, 2014).

There was a 20.2% increase in maximal isometric wrist flexion strength observed in the present study, which is greater than the 8-15% gains observed by Kroll (1963a), who used a similar measurement schedule. However, participants in the current work performed ten contractions on each consecutive test session versus five contractions as required by Kroll (1963a). It was shown that massed practice (ten contractions/day) allowed for better entrainment of an internal model of the resistive exercise task performance (McGuire et al., 2014). The consecutive days then allowed for refinement and consolidation of the internal model through distributed practice (McGuire et al., 2014). The increase in strength was retained over a two-week rest period and transferred when the experimental conditions were crossed, which suggests “relative permanence for the practiced skill” and that motor learning had occurred (Etnier & Landers, 1998; Katak & Winstein, 2012; Kohl & Gauadagnoli, 1996; Lai & Shea, 1999; Wright & Shea, 2001). Further support for motor skill learning is given by the fact that any gains associated with physiological adaptations would have dissipated (Häkkinen & Komi, 1983; Mujika & Padilla, 2001).

Consistent with Gabriel, Basford, and An (1997), the reversal of antagonists contraction pattern did not interfere with motor learning-related increases in strength. In a

previous study by Gabriel and Kroll (1991), participants who performed maximal isometric elbow extension-to-flexion contractions underwent a progressive decrease in baseline strength and mean strength-endurance (fatigue resistance). In contrast, participants who performed maximal isometric contractions of the elbow flexors in isolation, maintained baseline strength and increased mean strength-endurance. Similar to the present study, a tape recording regulated the work-to-rest ratio by providing cues for the timing of contractions throughout all phases of the resistive exercise task. At the same time, participants had to monitor an oscilloscope to obtain feedback about task performance. It may be speculated that the reversal of antagonists resulted in the same type of division of attention that occurs with paired auditory and visual stimuli which increases reaction time (Kroll, 1961; Wulf & Shea, 2002). However, if participants were well-practiced on a simple reaction time task, both simple reaction time and initial paired response reaction time would be unrelated to delays in the second response due to the psychological refractory period (Kroll, 1961). It is reasonable to suggest that the massed trials on each test day allowed our experimental group to become sufficiently well-practiced and demonstrate an increase in strength with a more complex contraction pattern. In support, Gabriel, Basford, and An (1997) used a measurement schedule that included a 30-trial fatigue series, consistent with a massed practice pattern. Participants in the experimental group also performed a 30-trial fatigue series consistent with a massed practice pattern. Moreover, when allowed to perform flexion only contractions in the crossed condition, the experimental group returned to baseline strength levels and they exhibited a significant increase in mean strength-endurance.

While the reversal of antagonists did not interfere with the “quick jumps in the strength” phenomenon, it also did not result in proprioceptive neuromuscular facilitation. These findings support the contention that facilitation effects cannot be manifested unless there is an existing deficit in neural drive as might exist in a patient population (Holt et al., 1969; Bohannon, 1985; Bohannon, 1986; Kroll, 1972a; 1972b; Gabriel et al., 1997). For example, it is possible that maximal isometric contractions of the wrist extensors resulted in autogenic inhibition of the ECR and facilitation of the FCR by Golgi tendon organs (GTOs) just prior to a maximal isometric contraction of the wrist flexors. However, normal sensorimotor integration would suppress the facilitated contraction through the Renshaw cell recurrent inhibition (Alvarez & Fyffe, 2007; Cavallari et al., 1984; Katz & Pierrot-Deseillgny, 1998), or even the GTOs from the FCR (Moore & Kukulka, 1991). It is also possible that central agonist facilitation and antagonist inhibition mechanisms supersede or at least modulate proprioceptive reflex circuits in able-bodied participants under baseline conditions (Geertsen, Lundbye-Jensen, & Nielsen, 2008; Kasai & Komiyama, 1988). In support of this idea, the only evidence of proprioceptive neuromuscular facilitation of strength and sEMG activity in an able-bodied population occurred after serial contractions resulting in a 30% decrement in strength (Hellebrandt et al., 1950; 1951a; 1951b). Execution of facilitatory techniques after the fatigue series resulted in a recovery of strength and sEMG (Hellebrandt et al., 1950; 1951a; 1951b). Gabriel, Basford, and An (2001) demonstrated the same facilitatory effects for when muscle tendon vibration was applied after a 30-trial fatigue series resulting in a 25% decrement in strength.

### ***5.3.2 Surface Electromyographic Activity***

Both the control and experimental groups exhibited a slight increase in FCR sEMG RMS amplitude across the three consecutive test sessions, which may be interpreted as an increase in neural drive to the muscle (Sale, 1988; Moritani, 1993; Enoka, 1997). However, a “slight” decrease was evident during the retention test two-weeks later, with a further reduction during the transfer task (the crossed condition) returning close to initial levels. At the same time, there was a reduction in ECR sEMG RMS amplitude that was most evident during the retention test and transfer task, which may be interpreted as a reduction in antagonist coactivation to increase the expression of agonist muscle force (Sale, 1988; Moritani, 1993; Enoka, 1997).

Alterations in agonist-antagonist muscle activity levels have been observed following resistive exercise regimens that involved several hundred contractions (Carolan & Cafarelli, 1992; Laroche et al., 2008; Tillin et al., 2011). The present study corroborates earlier findings for the elbow (McGuire et al., 2014) that alterations in agonist-antagonist muscle activity levels can be observed with a limited number (5–10) of contractions. In contrast, Green, Parro, and Gabriel (2014) report no overt changes in agonist-antagonist sEMG magnitude over 15 maximal isometric contractions of the dorsiflexors, consistent with the observations of Cannon and Cafarelli (1987) for the adductor pollicis muscle. Participants in the Cannon and Cafarelli (1987) study completed 15 contractions, three days a week for 5 weeks. Training-related alterations may have involved changes in motor unit (MU) discharges rates, which do not significantly affect the magnitude of the sEMG signal at full recruitment (Gabriel & Kamen, 2009). The hypothesis is consistent with the observation that the adductor

pollicis has a narrow recruitment range and relies on increases in discharge rates during force gradation (Kukulka & Clamann, 1981). Kamen and Knight (2004) showed that ten maximal isometric contractions of the knee extensors separated by a one-week interval resulted in a 16% increase in strength and a 19% increase in MU discharge rates in the vastus lateralis. Thus, it is possible that adaptations in MU discharge rates with a limited number of contractions might also exist for another larger muscle group like the tibialis anterior (Green, Parro, & Gabriel, 2014).

Across the consecutive test sessions there was a 15.8% increase in FCR sEMG RMS amplitude while ECR sEMG RMS amplitude decreased 16%. In comparison to the first test day, the alterations were retained and continued upon re-test two-weeks later: a 5.0% increase in FRC sEMG RMS amplitude and 26% decrease in ECR sEMG RMS amplitude were observed. The transfer task (crossed condition) followed the same patterns. Insight into the functional significance of alterations in FCR and ECR sEMG RMS amplitude is better provided by the coactivation ratio which showed a progressive decrease across all testing blocks: practice, retention, and transfer, amounting to 35.2%.

The present work extends the earlier findings for adaptations in coactivation during the “quick jumps in strength” phenomenon. When “only” a total of 15 maximal isometric contractions were administered, participants alternated between decreases and increase in coactivation (Calder and Gabriel, 2007). Because the experimental set-up for testing the elbow was similar to the present study, it may be speculated that participants were attempting to find the “optimal” balance between two competing functions ascribed to the antagonist: (1) generating minimally sufficient limb stiffness to decrease force RMS error (Gribble et al. 2003; Osu et al., 2004), (2) while allowing the agonist muscle

to contract unimpeded to maximize the expression of force (Kroll, 1981; Cannon & Cafarelli, 1987).

A re-analysis of Calder and Gabriel (2007) data for measures of variability later revealed that the massed practice of 15 contractions during one session allowed participants to make a better connection between force variability (RMS error) and the variability of motor output (force VR) than did five contractions on three consecutive days, as seen with the distributed practice schedule (McGuire et al., 2014). Thus, the massed practice pattern of ten maximal isometric contractions administered on each of three consecutive days allowed for successful initial entrainment, which was refined and updated resulting in a progressively decreasing coactivation ratio (Milner & Cloutier, 1998; Gribble et al., 2003; Mattar and Ostry, 2007).

Interpretation of sEMG activity is not without controversy. Recent modelling and simulation studies have suggested that it is possible for peripheral-related changes within the muscle to lengthen the intracellular action potential (IAP), which would be detected as an increase in sEMG amplitude. Such changes might occur in calcium-mediated potentiation of skeletal muscle (Arabadzhev, Dimitrov & Dimitrov, 2014). If muscle potentiation played a role in augmenting sEMG RMS amplitude, the first contraction of each consecutive day would serve as a conditioning stimulus and have the lowest scores (Inglis et al., 2011). However, as part of reliability analysis we observed a 15% linear decrease across trials 1 through 5. Furthermore, if peripheral-related training adaptations had occurred within the muscle, they would have been dissipated over the two-week rest period prior to the retention test (Häkkinen & Komi, 1983; Mujika & Padilla, 2001). Cross-talk also looms as a causal factor in the coactivation results (Etnyre & Abraham,

1988; Mogk & Keir, 2003). Extensive testing with electrode configuration and placement during pilot work reduced the observed cross-correlation coefficients between the FCR and ECR sEMG from  $R_{xy}=0.60$  to  $R_{xy}=0.00$ . Further evidence in favor of the absence of cross-talk, is the observation that the FCR and ECR sEMG RMS means followed opposite patterns of change across test sessions.

Since net torque is the summation of all the muscle forces surrounding the joint (Winter, 2009), interpretation of the coactivation ratio based on the kinesiological function of the FCR and ECR during maximal isometric wrist flexion would seem straightforward. However, it is more complicated than it would first appear (Tillin et al., 2011). Based on the works of Gonzalez, Buchanan, and Delp (1997), Leger and Milner (2000), Axelson and Hagbarth (2003), it was assumed that the FCR and ECR would be representative of the flexors and extensors involved in the task. In reality, there is no way to know the exact distribution of forces within any given muscle group, termed in the indeterminate problem (Crowninshield & Brand, 1981). In the simplest case, musculoskeletal anatomy can be used to create an EMG-force relationship to help derive a solution. The physiological cross-sectional areas and moment arms can be used to calculate the joint moment potential of each muscle (Ramsay, Hunter, & Gonzalez, 2009). Electromyographic activity is then used to provide information about the amount of muscle activation and therefore its relative force contribution to net joint torque (Buchanan et al., 1993). There are a number of biomechanical modelling methods used to calibrate the sEMG signal to convert the amplitude into force, yet it remains an active area of research with no generally accepted solution (Erdemir et al., 2007). The ultimate goal for some investigators is to obtain more accurate antagonist muscle force estimates,

so the functional impact of coactivation upon joint mechanics may be determined (Doorenbosch & Harlaar, 2003; Kellis & Katis, 2008; Tillin et al., 2011).

Non-normalized sEMG was analyzed in the present work because calibrating sEMG activity with respect to force is an inappropriate data transformation that violates assumptions required for statistical analysis (Inglis et al., 2013). However, to provide insight into the functional significance of changes in the coactivation ratio, a “post mortem” sEMG-force calibration procedure may be performed. The ECR sEMG RMS amplitude during antagonist muscle action (wrist flexion contractions) was normalized with respect to the RMS amplitude recorded when it contracted as an agonist (wrist extension contractions). The result provides a “rough” estimate of the sEMG unit per isometric force during antagonist muscle function at the wrist (Delp, Grierson, & Buchanan, 1996; Aagaard et al., 2000; Yang & Winter, 1984). It is acknowledged that fascicle lengths, pennation angle, and muscle moment arms can change even during an isometric flexion versus extension contractions (Maganaris, 2000; Simoneau et al., 2012). However, the impact of these variables is the same across test sessions, and it is the pattern of change that is of interest.

The following analysis is for the experimental group because maximal isometric elbow extension strength trials were completed as part of the protocol. Because the two groups followed the same patterns of change in coactivity, it is reasonable to assume that the control group followed the same pattern of change for calibrated ECR sEMG. The experimental group exhibited a linear decrease in ECR coactivity from  $58.0 \pm 24.9\%$  of its maximum on Block 1 down to  $23.6 \pm 9.6\%$  on Block 5. Thus, no matter how antagonist coactivation was assessed, the results showed a decrease associated with

motor-skill learning to allow for more efficient agonist muscle contractions (Cannon & Cafarelli, 1987; Floeter, Danielian, & Kim, 2013; Kroll, 1981).

## **5.4 Variability**

### ***5.4.1 Target Variability***

In the current study, participants were required to contract as hard and as fast as possible, and then maintain the maximum portion of the force trace parallel to a target line on the oscilloscope. The addition of a target line was to help instruct participants how to properly execute a task, as all resistive exercise requires specific technique (Escamilla, 2001; Escamilla et al., 2001; Hay et al., 1983; Madsen & McLaughlin, 1984; Selvanayagam et al., 2011; Steinkamp et al., 1993). To assess task variability, the RMS error of the middle 3.5 seconds of the force trace was calculated. Target variability does not refer to the difference between the participants' force trace and target line; rather it examines the stability of the force trace itself.

Consistent with previous literature, RMS error decreased with practice of the task (McGuire et al., 2014; Newell et al., 2003; Van Dijk et al., 2007). The force trace became more stable as participants in both groups increased in strength. The decrease in RMS error was evident during both the retention test and transfer task (crossed condition). The relative permanence of the RMS error decrease and the reduction in RMS error transferred to a new task is additional evidence that motor-skill learning had occurred (Etnier & Landers, 1998; Katak & Winstein, 2012; Kohl & Gaudagnoli, 1996; Lai & Shea, 1999; Wright & Shea, 2001). Further, the more complex contraction pattern did not interfere with the reduction in RMS error for either the experimental or control groups.

## ***5.4.2 Motor Output Variability***

### *5.4.2.1 Torque Variance Ratio*

Variability of motor output as defined by the variance ratio was recently introduced (Green, Parro, & Gabriel, 2014; McGuire et al., 2014) and as a result, there is limited comparative literature. Both groups exhibited similar reductions in torque VR across the first four blocks of testing: acquisition and retention. A reduction in torque VR means the shape of the torque-time curves became more reproducible, regardless of the complexity of the contraction pattern (Green, Parro, & Gabriel, 2014; McGuire et al., 2014). The relative permanence of decreased motor output variability is further evidence that motor-skill learning was involved in the task (Etnier & Landers, 1998; Kantak & Winstein, 2012; Kohl & Gauadagnoli, 1996; Lai & Shea, 1999).

To examine skill-related changes in force variability, Newell et al. (2003) reported decreases in RMS error, Salonikidis et al. (2009) showed reductions in the coefficient of variation, while Van Dijk et al. (2007) demonstrated decreases in the standard deviation. All of the cited measures are similar to force RMS error used to assess task performance, but do not provide insight into the underlying variability of motor output that generated the task (McGuire et al., 2014). McGuire et al. (2014) previously demonstrated that different practice schedules can produce the same results in terms of task performance (decreased RMS error) but have different effects in terms of the variability of motor output (torque VR). While the reduction in RMS error transferred to the reversal of antagonist contraction pattern for the control group, the more complex task increased torque VR to initial levels.

To explain the difference between the torque RMS error and VR, the similarities of the testing protocol to reaction time experiments involving paired auditory and visual stimuli will be discussed (Kroll, 1961; Wulf & Shea, 2002). Kroll (1969) showed that if participants were well-practiced on a simple reaction time task, both simple reaction time and initial paired response reaction time are unrelated to delays in the second response due to the psychological refractory period. Likewise, the measurement schedule allowed the control group to become well-practiced on agonist only contractions, so that performance (reduced RMS error) was transferred to the more complex contraction pattern. Gaudagnoli et al. (1996) stated:

*“Theorists have suggested that participants’ primary concern early in practice is to understand what it to be done and how performance is evaluated, rather than determining the most efficient way of meeting the task demands.”*

The continued decrease in RMS error simply reflects a transfer of understanding the demands of the task while the higher VR merely reflects the beginning of an iterative process associated with a new contraction pattern (McGuire et al., 2014; Proteau, Marteniuk, & Lévesque, 1992).

#### *5.4.2.2 Surface Electromyographic Variance Ratio*

The FCR VR supports the pattern of change observed for torque VR. That is, the FCR VR for the control group decreased straight through to the retention test, but increased when required to perform the reversal of antagonist during the transfer test. In contrast, the experimental group exhibited a decrease in FCR VR across all five blocks of testing. The FCR VR increased for the control group but decreased for the experimental

during the transfer task (crossed-condition) because of the difference in task complexity (Onushko, Kim, & Christou, 2014). Parallel changes in agonist sEMG VR and force VR while practicing a resistive exercise task is consistent with findings for the dorsiflexors (Green, Parro, & Gabriel, 2014) and the biceps brachii (McGuire et al., 2014). Decreases in the variability of motor unit activity patterns, have been shown to coincide with the decrease in force variability (Christou & Carlton, 2001; Knight & Kamen, 2001; Kornatz et al., 2004).

It would appear that motor learning related differences in outcomes between the two contractions patterns were most evident in the ECR VR. For the control group, the ECR VR followed the same pattern of changes as both the FCR VR and torque VR. There was a decrease that was present upon the retention test and increase when the reversal of antagonists was performed during the transfer task (crossed condition). The experimental group had a generally higher ECR VR than the control group. The ECR VR increased across the three test sessions and was still higher for the retention test. When the experimental group was allowed to perform isolated contractions of the wrist flexors, ECR VR decreased.

It is very tempting to explain the higher variability in ECR sEMG only in terms of task complexity (Wright & Shea, 2001). However, it must be remembered that the whole purpose of the reversal of antagonists technique is to evoke segmental reflexes that would impact the subsequent voluntary contraction. A number of authors have reported that the basic proprioceptive mechanisms are indeed operative (Kasai & Komyama, 1988; Kizuka, Asami, & Tanii, 1997; Gollhofer et al., 1998; Hultborn et al., 1996). Furthermore, there is the potential for training-related descending modulation of these

reflexes before the onset of the contraction (Geertsen, Lundbye-Jensen, & Neilsen, 2008; Lévénez et al., 2005). Participants also attended to auditory cues for the timing of the contraction pattern. The “reaction-time-like” experimental protocol could have easily modulated transcortical reflex loops (Kizuka, Asami, & Tanii 1997). Descending control signals driving the ECR coactivation may therefore be occurring against the backdrop of increased competitive facilitatory and inhibitory inputs to the  $\alpha$ -motoneuron (Ashby, Hilton-Brown, & Stålberg, 1986; Duclay et al., 2011). An increase in the variability of ECR sEMG activity as the muscle switched instantaneously from contraction as an agonist to antagonist, might be expected to occur independent of task complexity (Gabriel, Basford, & An, 2002).

The sEMG signal processing and calculation of the VRs also makes it possible to conceptualize this measure as an assessment of muscle force-pulse variability (Gabriel, Basford, & An, 2002). Overall, sEMG VR findings support the observations that learning-related increases in the performance of maximal effort contractions are associated with decreases in both task variability and variability of the underlying force pulses (Gabriel, 2002; McGuire et al., 2014).

## **5.5 Conclusions**

The present study showed that the proprioceptive neuromuscular facilitation (PNF) technique did not interfere with the “quick jumps in strength” phenomenon. The control and experimental groups exhibited comparable increases in strength, which was both retained and transferred (crossed conditions). A decrease in the ECR-to-FCR sEMG RMS coactivation ratio was the main neuromotor adaptation, which was retained and transferred.

The reversal of antagonists technique did not interfere with motor learning-related decreases in target variability (RMS error) or variability of the torque traces (motor output). Comparable adaptations were attributed to the massed practice pattern because the reduction in torque VR acquired by the control group did not transfer during the crossed condition (the reversal of antagonist contraction pattern). Changes in RMS error and torque VR were associated with alterations in FCR and ECR sEMG VRs. Reductions were retained by both groups. The decrease in sEMG VR transferred to the simple contraction pattern for the experimental group but not to the complex contraction pattern for the control group, cross-validates the effect of a massed practice pattern. The experimental group had a greater ECR sEMG VR during wrist flexion. The increase in variability was attributed to a complex mixture of segmental inputs due to the “conditioning” contraction and/or the “reaction-time like” experimental protocol, not necessarily task complexity.

The results of the present study support the hypotheses that were predicted. The control group displayed increases in strength with concurrent decreases in the variability of the torque and sEMG measures, while the experimental group displayed results supporting the trends seen in the control group.

## **5.6 Future Directions and Implications**

Now that it has been established that the reversal of antagonist technique does not interfere with the “quick jumps in strength” phenomenon if a massed practice pattern is employed, the next step is to determine if PNF is effective when there is a deficit in muscle activation as might exist in an older adult population (Kamen, Sison, Du, & Patten, 1995; Patten & Kamen, 2000; Connelly et al., 1999). Since task complexity of

isometric contractions can reduce motor output in older adults (Barry, Riek, & Carson, 2005), it would be important to determine if a massed contraction pattern would be sufficient to produce comparable strength gains to agonist only contractions. Using isometric contractions, Onushko, Kim, and Christou (2014) also showed that practicing with easier tasks might be advantageous to improve motor learning in older adults. This is particularly relevant because the reversal of antagonists technique resulted in higher antagonist sEMG variability. Chen, Kwon, Fox, and Christou (In Press) recently demonstrated that older adults had impaired motor learning-related alterations in antagonist coactivity during a maximal resistive exercise task that was used to assess skill transfer.

Ultimately, the present thesis topic is important because the reversal of antagonist contraction pattern is part of a suite of proprioceptive neuromuscular techniques that are widely applied for stroke rehabilitation in an older adult population, but there is scant evidence of their therapeutic benefit (Gowland et al., 1992; Westwater-Wood, Adams, & Kerry, 2010; Kollen et al., 2009). At the same time, health-care costs associated with changing population demographics are driving a move towards robotic assisted technology for stroke rehabilitation (Marchal-Crespo & Reinkensmeyer, 2009; Sale et al., 2014; Farmer et al., 2014). Such a device has, for example, already been developed for the wrist (Krebs et al., 2007). While several different training modalities are being explored (Basteris et al., 2014), it is generally agreed that the main benefit is related to dose-response effect (Fasoli et al., 2003). A robotic rehabilitation device can more efficiently deliver a larger number of contractions (termed, sensorimotor training) to the patient than a therapist can while using traditional techniques. The larger number of

contractions is tantamount to massed-practice where improvements are attributed to motor-learning (Fasoli et al., 2003). These issues converge now that PNF contraction patterns are being introduced as part of the therapeutic program delivered by robotic devices. The trade-off between increasing  $\alpha$ -motoneuron recruitment and reducing antagonist coactivity must be assessed (Gowland et al., 1992).

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## APPENDIX A

### Sample Size Estimation

Sample size estimation was based on the primary focus of this thesis: expected changes in maximal isometric strength due solely to measurement schedule. Sample size estimation was based on the procedures outlined by Cohen (1988). Cohen (1988) recommends a ratio of 4:1 to balance the risk of a Type I error against committing a Type II error. The condition is satisfied with the probability of  $\alpha=0.05$  for a Type I error and a probability of  $\beta=0.20$  for a Type II error so that the power ( $1 - \beta$ ) of the test is 0.80. The most difficult aspect of the sample size estimation is to determine a reasonable effect-size (ES) that is deemed non-trivial by the investigator.

The first phase of sample size estimation was to gain an understanding of the variability of the criterion measure and the magnitude of the effect size through a “post-mortem” power analysis of a similar study. Means, standard deviations, and intraclass reliability coefficients were obtained from a similar study on measurement schedule effects upon maximal isometric wrist flexion strength in 20 college age male participants (Kroll, 1963a). The means for the first ( $49.91 \pm 5.71$  lbs) and third ( $51.10 \pm 5.84$  lbs) consecutive days of testing for Test Condition 1 were used to calculate Cohen’s effect size “d” for correlated measures (Case 4). Three consecutive days of testing resulted in a 3.5% (1.69 lbs) increase in maximal isometric wrist flexion strength ( $d_4' = 1.69$  lbs). The slight decrease in the stability between means across days resulted in an intraclass reliability coefficient of  $R=0.91$ , which is still considered quite high but reflects the intrusion of learning effects (Kroll, 1963a).

Post-mortem power analysis was accomplished using the non-central  $t$ -distribution, where the error term was for correlated measures. Since the experimental design involves repeated measures, the intraclass reliability coefficient replaces the Pearson's correlation coefficient between paired observations:

$$\sigma_{M_1-M_2} = \sqrt{\sigma_{M_1}^2 + \sigma_{M_2}^2 - 2R\sigma_{M_1}\sigma_{M_2}},$$

where  $\sigma_{M_1}^2$  and  $\sigma_{M_2}^2$  are the standard error of the means for the first and third consecutive test session, respectively. The resulting effects-size is:

$$d = \frac{d4'}{\sigma_{M_1-M_2}}$$

$$d = \frac{1.69 \text{ lbs}}{0.5468 \text{ lbs}} = 3.06$$

Figure 21 shows that the Kroll (1963) means, standard deviations, reliability coefficient, and effect-size resulted in a post-mortem power on 0.80. Post-mortem power analysis is only meaningful if the goal is not to determine how many participants would be required for the observed effect-size to achieve significance (Lenth, 2001). In this case, by coincidence, 20 participants resulted in a power of 0.80 (see Figure 21, left panels).

A similar effect-size was observed as a 3.9% increase in maximal isometric dorsiflexion strength following three days testing, with a 48 hour rest between test sessions (MacIntosh & Gabriel, 2012). The small increase in means across test days had little effect on the reliability with an intraclass correlation coefficient of  $R=0.98$ . Motor learning-related increases in maximal isometric strength of wrist flexors can range from 8-15% with progressively decreasing reliability coefficients down to  $R=0.80$  (Kroll,

1963a). A 5 percent ( $d=4.5$ ) increase in maximal isometric strength of the wrist flexors has been chosen to balance the need to observe an effect-size that is both statistically significant and practically important while securing a reliable criterion measure. Using the same means, standard deviation, and approximated reliability coefficients for the non-central  $t$ -distribution, an estimated sample size of between 10 and 11 participants is required (see Figure 21, right panel). However, to guard against the possibility that the observed variability might be greater than estimated, or that participants may withdraw from the study, 14 matched pairs will be used for a total of 28 participants.

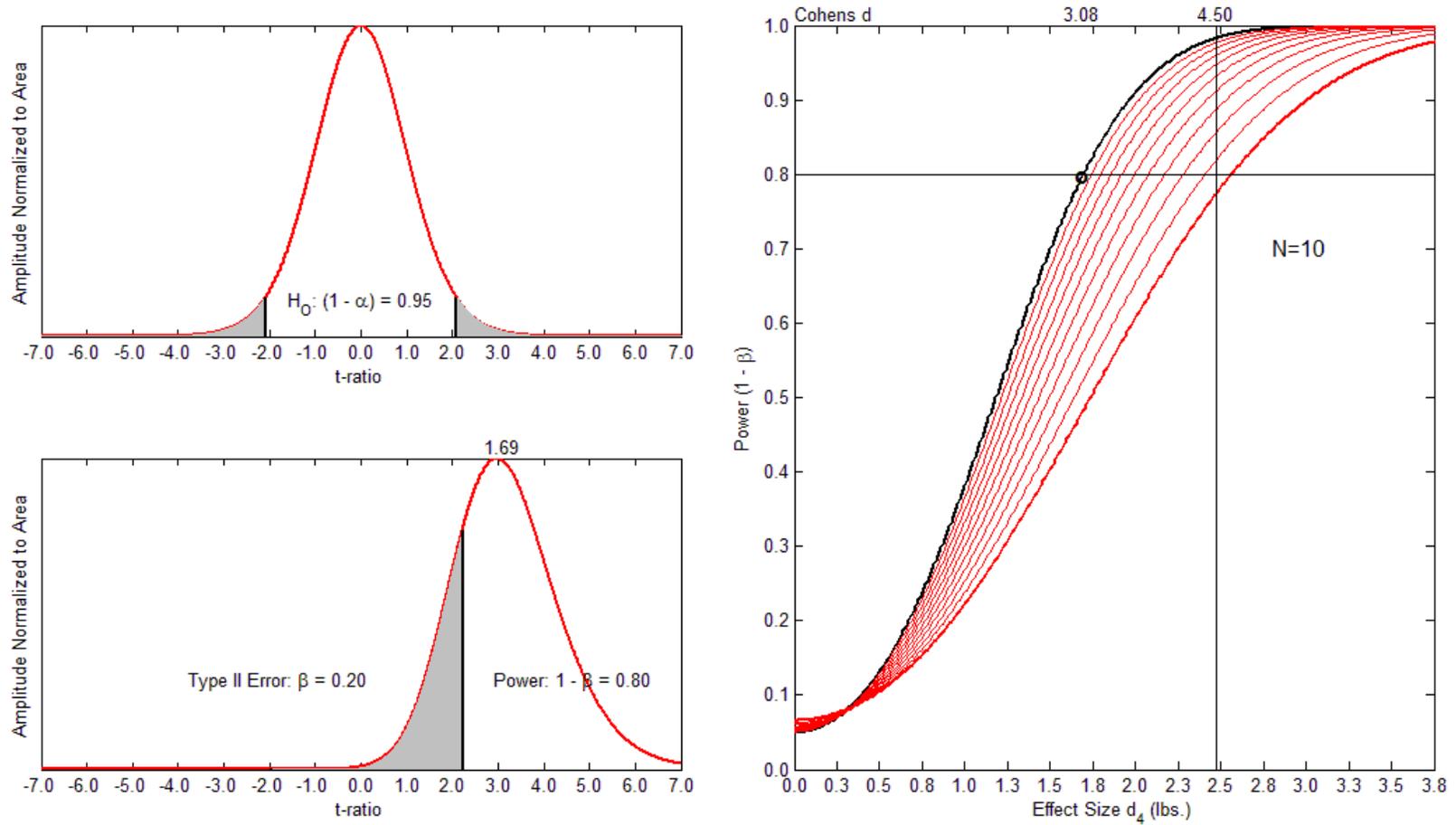


Figure 32. Normal curves and sample size estimation. Each curve in the right figure represents a sample size decrease of 1 participant, starting with the black curve ( $N=20$ ).

## **APPENDIX B**

### **Informed Consent**

Date:

Project Title: Proprioceptive neuromuscular facilitation of the wrist flexors.

Principal Student Investigator: Jessica McGuire, Masters Student  
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### **INVITATION**

You are invited to participate in a study being conducted by Jessica McGuire and supervised by David Gabriel. The purpose of this research project is to investigate the use of a proprioceptive neuromuscular technique (PNF) on muscle electrical activity and force production in the wrist flexors. Participants are required to be college-aged males between 18-30 years, be in overall good health and have no neurological or orthopedic disorders.

### **WHAT'S INVOLVED**

As a participant you will be asked to participate in four separate testing sessions. The first three sessions will occur on consecutive days and the fourth session will occur two-weeks after the third session. Each session will be approximately 1 and one-half hours in length.

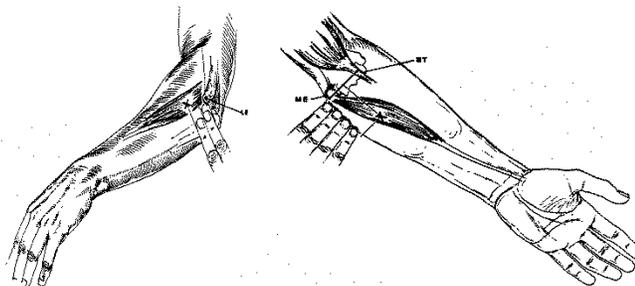
Upon arrival in the lab, you will be asked to complete a PAR-Q questionnaire and a demographic questionnaire (information regarding age, height, weight and weight-training experience), as well as have measurements taken of your forearm (forearm length and circumference). This will only occur during the first test session.

The following paragraphs outline the procedures in sequence that will be followed during all four testing sessions. The dominant forearm will be prepared for testing. Small areas on the forearm will be shaved, lightly abraded, and cleansed with alcohol. These areas correspond to the locations where the electrodes for recording muscle electrical activity will be placed. The procedure is similar to the more familiar electrocardiogram for measuring the electrical activity of the heart muscle. The recording electrodes for forearm

muscle electrical activity are small metallic discs about the size of a shirt button; they will be placed on the skin surface, over the muscles of interest. We are interested in recording electrical activity from muscles on the front (flexor carpi radialis, or FCR) and back (extensor carpi radialis, or ECR) of the forearm, which are depicted in the figure below.

*EXTENSOR CARPI RADIALIS,  
LONGUS AND BREVIS*

*FLEXOR CARPI RADIALIS*



Next, landmarks for the locations of the electrodes will be located using a low-level repeated electrical stimulation on the skin's surface. The level of electrical stimulation will be small and barely perceptible. A metallic probe electrode will explore the skin surface to locate the recording point ("x") for each muscle identified in the figure above. This is the point where the investigator observes a minimally visible muscle twitch, and it will be marked with indelible ink. There will be one pair of electrodes for each muscle, each pair placed on the belly of the muscle just below the point marked "x". A ground electrode for electrical safety will be placed on the back of the hand.

The median nerve which runs next to the tendon of the biceps brachii will then be stimulated by placing a pad electrode about the size of a quarter on the elbow crease. While relaxing the forearm, there will be an electrical stimulation applied to the median nerve that is strong enough to "briefly" flex the wrist involuntarily. At this point, the level of electrical stimulation "may" be perceived as uncomfortable but has been reported to be quite tolerable. You are free to discontinue the procedure if you find it unacceptable.

The involuntary wrist flexion is the result of a large amount of electrical activity generated in the muscle called an "M-wave". A total of 5 stimulations will be required to obtain the 5 M-wave responses. There will be 15 seconds between each stimulation. Next, a barely perceptible level of electrical stimulation will be applied to the median nerve. No movement will occur but we will still be able to record a small amount of electrical activity from the muscle called an "H-reflex". A total of ten responses are necessary, each response occurring at 15 second intervals.

During the next phase of the study you will be asked to perform 10 maximal effort wrist flexion contractions while your hand is secured within a testing device so that there is no movement at the elbow, wrist or hand. Thus, while the contractions are forceful, there will be no movement (isometric). Each contraction will last 5-seconds and there will be 3-minutes of rest between each contraction.

This study is a two-group design and you will be randomly assigned to one of the two groups. One group will perform wrist flexion only (Group 1) and the other group will perform wrist extension immediately before wrist flexion (Group 2) for all four sessions. Performing wrist extension-to-flexion is analogous to “crouching” down just before jumping up.

Halfway through each flexion contraction for both groups, the median nerve will be stimulated at the same level required to obtain an M-wave. However, because the electrical stimulation occurs during a voluntary contraction, it results in a different pattern of electrical activity from the muscle, termed the “V-wave”. After the five contractions, while you are resting, 5 M-waves and 10 H-reflexes will be evoked in the same manner as at the start of the testing session.

Before leaving the lab, the positions of the recording electrodes will be marked with a non-toxic indelible ink and you will be asked to maintain these locations over the testing sessions. You are welcome to come to the lab for help to retrace the electrode at any time, before the location is lost. Maintaining the recording points is only to save time and is not a critical issue. If the tracings are not visible upon returning to the lab, the recording points (“x”) will be identified once again using the same method as described above.

## **POTENTIAL BENEFITS AND RISKS**

Although there are no direct benefits from participating in this study, it should be known that your willingness to participate in this experiment will help the researcher and other scientists optimize therapeutic resistive exercise for patients suffering from muscle weakness. Participating in the current research will also provide you with an opportunity to gain exposure to research and develop knowledge about the neuromuscular system and muscle contractions.

It is not possible to predict all risks and discomforts associated with any research, but according to previous research and experience, the researcher anticipates no major risks associated with this protocol.

1. Participants may sometimes feel a mild discomfort during the preparation of the skin for electrode placement. On occasion, some participants may experience skin irritation associated with the placement of electrodes, but this is usually very mild and will subside in a few hours, or a day.
2. It may be necessary to remove hair over the muscle to record its electrical activity. As a result, it is possible that the participant may be cut by the razor while shaving the skin. To minimize the possibility of wound infection, a new disposable safety razor will be used for each participant. The area will also be cleansed with alcohol and a Band-Aid will be applied if necessary.
3. There are two possible risks associated with electrical stimulation in a healthy-bodied population:
  - a. The first concern is electrical safety which is maintained by grounding both the participant and laboratory equipment. Electrical safety is further

enhanced by the use of an isolation unit that separates the participant from the stimulator.

- b. The second risk is that the participant perceives the electrical stimulus to the nerve as noxious, resulting in vasovagal syncope (i.e., fainting). If the electrical stimulation pads are placed correctly over the nerve, the actual physical discomfort is minimal. However, there is no way to predict how someone will respond subconsciously to the electrical stimulation. The student-investigator will constantly monitor the participant for how well the procedures are being tolerated and will discontinue the protocol if the participant expresses a desire to stop or if the initial signs of fainting are present. A participant has never fainted in the laboratory while following these guidelines. If fainting does indeed occur, the student investigator has been certified in CPR and first aid. Because this reaction is not under the control of the participant, they will be discontinued from further study.
4. There is a very slight possibility of muscle soreness from isometric contractions of the forearm muscles, but this is typically very mild. It will not interfere with normal daily activities and should dissipate within 72 hours.
5. Maximal 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. If any box on the PAR-Q form is checked “yes”, especially ones that may identify or point towards hypertension, participants must be automatically excluded from the study.

## **CONFIDENTIALITY**

Confidentiality of information concerning all participants will be maintained throughout the research and during the publication of the study. The data will be coded without personal reference to you and all information that can be related back to you will be kept in a locked office, to which only the investigating team has access to. Names or material identifying participants will not be released without written permission except as such release is required by law.

## **VOLUNTARY PARTICIPATION**

Participation in this study is voluntary. Refusal to participate if any component of the study or the study as a whole will NOT result in loss of access to any services or programs at Brock University you are entitled to. If you wish to withdrawal from this study at any time during the course of the research, please inform the investigator, Jessica McGuire.

## **PUBLICATION OF RESULTS**

Results of this study may be published in professional journals and presented at conferences. If you would like feedback about the results, you may request for the researcher to contact to you once they are published.

### **CONTACT INFORMATION AND ETHICS CLEARANCE**

If you have any questions about this study or require further information, please contact the principle investigator, Jessica McGuire, or the faculty supervisors, David Gabriel using the contact information provided above. This study has been reviewed and received ethics clearance through the Research Ethics Board at Brock University [12-281]. If you have any comments or concerns about your rights as a research participant, please contact the Research Ethics Office at (905) 688-5550 Ext. 3035, [reb@brocku.ca](mailto:reb@brocku.ca).

Thank you for your assistance in this project. Please keep a copy of this form for your records.

### **CONSENT FORM**

I agree to participate in this study described above. I have made this decision based on the information I have read in the Information-Consent Letter. I have had the opportunity to receive any additional details I wanted about the study and understand that I may ask questions in the future. I understand that I may withdraw this consent at any time.

Name: \_\_\_\_\_

Signature: \_\_\_\_\_ Date: \_\_\_\_\_

\_\_\_\_\_



## PAR-Q Form

Physical Activity Readiness  
Questionnaire - PAR-Q  
(revised 2002)

# PAR-Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69, the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age, and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly: check YES or NO.

YES	NO	
<input type="checkbox"/>	<input type="checkbox"/>	1. Has your doctor ever said that you have a heart condition <u>and</u> that you should only do physical activity recommended by a doctor?
<input type="checkbox"/>	<input type="checkbox"/>	2. Do you feel pain in your chest when you do physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	3. In the past month, have you had chest pain when you were not doing physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	4. Do you lose your balance because of dizziness or do you ever lose consciousness?
<input type="checkbox"/>	<input type="checkbox"/>	5. Do you have a bone or joint problem (for example, back, knee or hip) that could be made worse by a change in your physical activity?
<input type="checkbox"/>	<input type="checkbox"/>	6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?
<input type="checkbox"/>	<input type="checkbox"/>	7. Do you know of <u>any other reason</u> why you should not do physical activity?

If  
you  
answered

## YES to one or more questions

Talk with your doctor by phone or in person BEFORE you start becoming much more physically active or BEFORE you have a fitness appraisal. Tell your doctor about the PAR-Q and which questions you answered YES.

- You may be able to do any activity you want — as long as you start slowly and build up gradually. Or, you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his/her advice.
- Find out which community programs are safe and helpful for you.

## NO to all questions

If you answered NO honestly to all PAR-Q questions, you can be reasonably sure that you can:

- start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.
- take part in a fitness appraisal — this is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also highly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

## DELAY BECOMING MUCH MORE ACTIVE:

- if you are not feeling well because of a temporary illness such as a cold or a fever — wait until you feel better; or
- if you are or may be pregnant — talk to your doctor before you start becoming more active.

**PLEASE NOTE:** If your health changes so that you then answer YES to any of the above questions, tell your fitness or health professional. Ask whether you should change your physical activity plan.

**Informed Use of the PAR-Q:** The Canadian Society for Exercise Physiology, Health Canada, and their agents assume no liability for persons who undertake physical activity, and if in doubt after completing this questionnaire, consult your doctor prior to physical activity.

**No changes permitted. You are encouraged to photocopy the PAR-Q but only if you use the entire form.**

NOTE: If the PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal, this section may be used for legal or administrative purposes.

"I have read, understood and completed this questionnaire. Any questions I had were answered to my full satisfaction."

NAME \_\_\_\_\_

SIGNATURE \_\_\_\_\_

DATE \_\_\_\_\_

SIGNATURE OF PARENT  
or GUARDIAN (for participants under the age of majority) \_\_\_\_\_

WITNESS \_\_\_\_\_

**Note: This physical activity clearance is valid for a maximum of 12 months from the date it is completed and becomes invalid if your condition changes so that you would answer YES to any of the seven questions.**



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## Demographic Information and Physical Activity Questionnaire

Participant Number \_\_\_\_\_

Age: \_\_\_\_\_

Weight: \_\_\_\_\_

Height: \_\_\_\_\_

Sex: \_\_\_\_\_

How many times a week do you participate in physical activity (moderate to vigorous)? -  
\_\_\_\_\_

On average, how many hours per a week (total) do you participate in physical activity?  
\_\_\_\_\_

Do you participate in any racquet activities or physical activities that mainly use the upper limbs? If so, please list activities and frequency:

---



---

What other physical activities do you participate in?

---



---

Do you weight train? If so, how many times a week do you weight train? \_\_\_\_\_

On average, 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

**Anthropometric Measurements****Participant Number:** \_\_\_\_\_*Anthropometric Measurements*

Forearm Length (olecranon process to styloid process of ulna) \_\_\_\_\_

Hand Length (styloid process of ulna to tip of third finger) \_\_\_\_\_

Elbow Circumference (circumference at olecranon process) \_\_\_\_\_

Wrist Circumference (circumference at distal space to styloid process of ulna)  
\_\_\_\_\_Wrist breadth (distance between radial prominence and ulnar styloid process)  
\_\_\_\_\_

Forearm Circumference Proximal \_\_\_\_\_ Distal \_\_\_\_\_

Hand Thickness (thickness of base of hand, cross-section height thenar eminence and  
hypothenar eminence) \_\_\_\_\_

Hand breadth (measured across the distal ends of the metacarpal bones) \_\_\_\_\_

Lever Length (styloid process of ulna to base of third finger) \_\_\_\_\_

## APPENDIX C

### Reliability

Reliability was assessed using two different analysis of variance (ANOVA) models. The first ANOVA was used to establish “consistency” of the measures using a fully nested model wherein trials were nested within days, which were in turn nested within subjects. The mean squares from the fully nested model outlined below were used to calculate the intraclass correlation coefficient, which was used to assess consistency. The second ANOVA model was used to examine the “stability” of the means across test sessions. This model had two factors (days  $\times$  subjects) and the repeated measurements (trials) on each subject in each day constituted a “within-cells” replication of measures. Therefore, for a measure to be considered reliable, it must have exhibited both consistency and stability. The intraclass correlation coefficients (R) were calculated as follows:

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

$$\sigma_{true}^2 = MS_{trials}$$

$$\sigma_{e_1}^2 = \frac{MS_{days} - MS_{trials}}{n'}$$

$$\sigma_{e_1}^2 = \frac{MS_{subjects} - MS_{days}}{a' \cdot n'}$$

The mean squares for subjects ( $MS_{subjects}$ ), days ( $MS_{days}$ ), and trials ( $MS_{trials}$ ) were extracted from the fully nested ANOVA to calculate the reliability coefficient. In the above equations  $a'$  was the number of days and  $n'$  was the number of trials. The total

variance was the sum of all variances. The portion of variances attributable to day-to-day ( $\sigma_{e_2}^2/100$ ), trial-to-trial ( $\sigma_{e_1}^2/100$ ), and between-subjects ( $\sigma_{true}^2/100$ ) error was computed to identify the amount of variability at each level of measurement (Calder et al., 2005; Christie et al., 2010).