

**Examining Neuromuscular Fatigue of the Elbow Flexors During Maximal Arm
Cycling Sprints in a Pronated and Supinated Forearm Position**

By

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Abstract

During high intensity exercise, an individual's ability to generate and maintain force is decreased resulting in a decline in muscle performance. This decrease in force is referred to as neuromuscular fatigue (NMF). There have been several studies examining NMF during maximal leg cycling sprints. Maximal repeated leg cycling sprints have been shown to induce peripheral fatigue early and this fatigue persists throughout the sprints, while central fatigue occurs towards the end of the sprints. To date, only one study has examined the effects of maximal arm cycling sprints on NMF and similar patterns of peripheral and central fatigue have been reported. Many studies have shown that the specific task being performed can alter NMF. For example, the development of NMF has been shown to be different during maximal running and leg cycling exercises at the same workload. This suggests that NMF appears to develop differently depending on the specific action of the muscles involved. Although not examining NMF, two studies have examined the effect of forearm position during constant load arm cycling. These studies found that forearm position can influence muscular activity and brain and spinal cord excitability during constant load arm cycling. Despite the present research, it is currently unknown if the development of NMF is different following arm cycling sprints in different forearm positions. Therefore, the purpose of this study was to compare the development of NMF during repeated arm cycling sprints in pronated and supinated forearm positions. This study add to the current understanding of how NMF influences exercise performance, and may aid in the development of training protocols for rehabilitative and athletic purposes.

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List of Abbreviations

- ATP – Adenosine triphosphate
- CMEP – Cervicomedullary evoked potential
- EMG – Electromyography
- iEMG – Integrated EMG
- ITT – Interpolated twitch technique
- LICI – Long-interval intracortical inhibition
- MEP – Motor evoked potential
- MVC – Maximal voluntary contraction
- NMF – Neuromuscular fatigue
- RMS – Root mean square
- RPE – Rating of perceived exertion
- SICI – Short-interval intracortical inhibition
- TMES – Transmastoid electrical stimulation
- TMS – Transcranial magnetic stimulation

Chapter 1 Introduction

1.0 Overview

Arm cycling is commonly used in rehabilitation clinics, research laboratories, and fitness training centers as a form of exercise. Arm cycling can be performed continuously over a long period of time at a moderate intensity (i.e., endurance exercises; Bressel et al., 2001) or performed intermittently over a short period of time at a high intensity (i.e., sprinting exercises; Pearcey et al., 2016). Intermittent high intensity exercise is a time-efficient strategy to induce similar physiological and performance adaptations to traditional endurance exercises. As a result, intermittent high intensity exercise has been shown to be an effective exercise regime to improve overall health and exercise tolerance (Jung et al., 2016; Gibala et al., 2008). It is commonly believed that high intensity exercise should only be performed by the athletic population; however, untrained individuals (Jung et al., 2016) and individuals undergoing rehabilitation for spinal cord injuries (Astorino & Thum, 2016) have reported higher enjoyment during brief sessions of submaximal or supramaximal interval training, despite greater metabolic strain compared to moderate intensity endurance training.

Fatigue commonly occurs during endurance and sprinting exercises, but occurs more rapidly with sprinting exercises (Bishop, 2012). Exercise-induced fatigue develops as a result of sustained muscular activity and is referred to as neuromuscular fatigue. Neuromuscular fatigue causes a reduction in the ability of the exercising muscle to generate force, and thus results in a decline in exercise performance (Gandevia, 2001). Having a better understanding of how fatigue contributes to a decrease in exercise performance may contribute to the development of better training protocols for rehabilitative, recreational,

and athletic purposes to improve overall physical performance and/or health status. When it comes to training performed with arm cycling one aspect that has received almost no attention is the effect of forearm position on fatigue development. Such knowledge would be valuable in regard to the development of exercise programs that are tailored to various populations.

1.1 Purpose

The purpose of this study was to examine neuromuscular fatigue of the elbow flexors during maximal arm cycling sprints in a pronated and supinated forearm position.

1.2 Research Hypotheses

There are two main hypotheses for this study:

1. Neuromuscular fatigue of the elbow flexors will occur following 10, 10-second arm cycling sprints in both forearm positions (i.e., pronated and supinated).
2. The proposed measurement techniques discussed below will demonstrate a greater degree of neuromuscular fatigue in the elbow flexors during the supinated forearm position.

1.3 References

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Chapter 2 Review of Literature

2.0 Introduction

Upper and lower-body maximal intensity repeated sprint protocols are used to examine the effect of maximal exercise on the neuromuscular system (Girard et al., 2013; Hureau, et al., 2016; Monks et al., 2016; Pearcey et al., 2015; Pearcey et al., 2016). Repeated sprint protocols consist of repeated bouts of brief (≤ 10 seconds) maximal or near maximal work interspersed with recovery periods ranging from 10 to 300 seconds (Balsom et al., 1992; Duffield et al., 2009). The performance of maximal intensity sprints requires a high turnover rate of skeletal muscle adenosine triphosphate (ATP) (Bishop & Clauduis, 2005), and the majority of the energy required to restore this ATP is provided by phosphocreatine breakdown and anaerobic glycolysis (Spriet et al., 1985). Due to the reduction in available energy in the phosphocreatine and anaerobic glycolytic pathways following the onset of exercise, maximal intensity repeated sprint exercises have been associated with the development of fatigue (Bishop & Clauduis, 2005).

Fatigue is a common consequence of repeated or sustained muscular activity (Gandevia, 2001; Amann, 2011; Taylor & Gandevia, 2008; Girard et al., 2008). It is characterized by an acute reduction in the efficiency of performing a task. More specifically, during maximal sprinting exercises, an individual's ability to generate and maintain force is decreased resulting in a decline in muscle performance (Gandevia, 2001). The exercise-induced reduction of the force-generating capacity of working skeletal muscle is referred to as neuromuscular fatigue (Amann, 2011; Gandevia et al., 1996). Several studies have examined neuromuscular fatigue with lower-body maximal leg cycling sprints

(Billaut et al., 2005; Billaut et al., 2006; Duffield et al., 2009; Pearcey et al., 2015; Monks et al., 2016; Racinais et al., 2007) with many variations in the number of sprints performed, the resistive load applied, and the duration of work and recovery. However, to date, only one study has examined neuromuscular fatigue following upper-body maximal arm cycling sprints (Pearcey et al., 2016). Moreover, the development of neuromuscular fatigue has been shown to be different depending on the specific details of the task being performed (Babault et al., 2006; Enoka & Stuart, 1992). During submaximal arm cycling, forearm position has been shown to influence muscle activity as well as the output of the motor system (i.e., corticospinal excitability) differently (Bressel et al., 2001; Forman et al., 2016). It is currently unknown if maximal arm cycling sprints when performed using a pronated or supinated forearm position will have a similar neuromuscular fatigue profile. Pearcey et al. (2016) reported neuromuscular fatigue of the biceps brachii during and following pronated arm cycling sprints. Given that biceps brachii is a less efficient elbow flexor when the forearm is pronated (Drake, Vogl, & Mitchell, 2009; Kleiber, Kunz, & Disselhorst-Klug, 2015), perhaps cycling in a supinated forearm position that allows biceps brachii to become a more efficient elbow flexor would impact the degree to which biceps brachii becomes fatigued. Reviewing existing research on lower and upper body exercises is important for understanding how forearm position may influence the neuromuscular fatigue profile. This review will discuss: 1) neuromuscular fatigue and how it is assessed, 2) current literature on the development of neuromuscular fatigue with maximal intensity lower and upper body exercise, 3) the potential effect of forearm position on the development of neuromuscular fatigue during upper body cycling sprints and 4) clinical and practical applications.

2.1 Neuromuscular Fatigue

For the purpose of this review, the term fatigue will be used in the context of neuromuscular fatigue. Neuromuscular fatigue is an inevitable consequence of performing any sustained physical activity. It is defined as an exercise-induced decrease in muscular performance usually seen as a reduction in the ability of the muscle to maintain or produce force or power (Gandevia, 2001). Neuromuscular fatigue includes processes at all levels of the motor pathway between the brain and muscle and thus, can be divided into peripheral and central elements (Billaut et al., 2006; Billaut & Basset, 2007; Gandevia, 2001).

Peripheral fatigue causes biochemical changes near the neuromuscular junction or terminal branches of the motor axon or within the muscle (Bishop, 2012). During high intensity sprinting exercises, peripheral fatigue occurs early and continues throughout the exercise bout (Gandevia, 2001; Pearcey et al., 2015; Pearcey et al., 2016). Potential mechanisms of peripheral fatigue may include impairments of the excitation contraction coupling process and sarcolemma excitability (Allen et al., 2008; Bishop, 2012). These impairments are influenced by the release and restoration of intracellular calcium from the sarcoplasmic reticulum and the reduced sensitivity between contractile proteins and calcium (Allen et al., 2008; Bishop, 2012; Glaister, 2005). Additionally, increases in blood lactate (Pearcey et al., 2015), hydrogen ions, inorganic phosphate, and muscle deoxygenation (Racinais et al., 2007) may contribute to peripheral fatigue by causing inhibitory effects on force development and/or calcium sensitivity (Allen et al., 2008). Assessing peripheral fatigue during sprinting exercises is important as it has been associated with decreases in performance during repeated sprint exercise (Glaister, 2005).

In contrast, central fatigue occurs proximal to the motor axons and comprises a failure of the central nervous system (i.e., brain and spinal cord) to “drive” the motoneurons resulting in a reduction in voluntary activation of the muscle (Gandevia, 2001; Taylor & Gandevia, 2008). During high intensity exercise, central fatigue mainly occurs later during an exercise bout (Gandevia, 2001). The development of central fatigue is often accompanied by a decline in motor unit firing rate (Gandevia, 2001; Taylor & Gandevia, 2008), although this decline cannot explain the mechanisms behind central fatigue. However, it is likely that the mechanisms mediating the slowing of motor unit firing is a main component of the development of central fatigue. At the level of the motoneurone pool, three mechanisms have been suggested to be underlying motoneurone slowing: 1) a decrease in excitatory input, 2) an increase in inhibitory input, and/or 3) a decrease in the responsiveness of the motoneurons (Taylor & Gandevia, 2008). During fatiguing exercise, inputs from muscle afferents, recurrent inhibition, as well as descending drive innervating the exercising muscles are likely factors that influence the output of the motoneurone pool (Gandevia et al., 1995; Taylor & Gandevia, 2008). Therefore, the decrease in voluntary activation that is observed with central fatigue can be due to supraspinal (i.e., cortical neurones) and/or spinal (i.e., alpha-motoneurons) factors.

Electrical stimulation of the peripheral motor nerves or transcranial magnetic stimulation (TMS) of the motor cortex during a maximal voluntary contraction (MVC) can be used to deduce central fatigue by producing a superimposed twitch (Gandevia et al., 1996). An increased superimposed twitch during peripheral nerve stimulation or TMS implies that a larger proportion of the central nervous system is unable to deliver excitatory input to the motor units to maximally activate the exercising muscle (Gandevia et al., 1996;

Taylor & Gandevia, 2008). Although both electrical stimulation of the peripheral nerve and magnetic stimulation of the motor cortex can measure central fatigue, TMS allows researchers to determine whether or not the central fatigue has a supraspinal component. When the superimposed twitch elicited by TMS increases following a fatiguing task, the motor cortical output is not optimal, which may suggest supraspinal fatigue (Gandevia et al., 1996; Sidhu et al., 2009). On the contrary, an increased superimposed twitch following peripheral nerve stimulation suggests central fatigue has occurred, but this method is unable to distinguish between supraspinal and spinal components responsible for central fatigue. Additionally, TMS can be used to elicit motor evoked potentials (MEPs) in surface electromyography (EMG), which can provide additional insight into the factors that may be contributing to central fatigue (Gandevia et al., 1996). Researchers frequently examine the peak-to-peak amplitude and area of these TMS-evoked MEPs to provide an instantaneous evaluation of corticospinal excitability (Di Lazzaro et al., 1998; Gandevia, 2001; Rothwell, 2007). Essentially, any alterations in MEP amplitudes between trials suggests that the excitability of the corticospinal pathway, comprised of neurones at the supraspinal and spinal level, as well as the muscle fibres, has changed (Gandevia, 2001; Pearcey et al., 2016). To distinguish between alterations in supraspinal and spinal excitability contributing to central fatigue, transmastoid electrical stimulation (TMES) can be used in combination with TMS. In this case both evoked potentials are normalized to the maximal muscle compound action potential to control for changes distal to the motor neurone. TMES produces cervicomedullary evoked potentials (CMEPs), which are short-latency excitatory responses to stimulation of the axons of the corticospinal tract at the cervicomedullary junction (Taylor & Gandevia, 2004). Following a fatiguing task, if there

are decreases in CMEP amplitudes while MEP amplitudes remain unchanged, this suggests that the mechanisms responsible for central fatigue are mediated mainly by spinal mechanisms. In contrast, if there are decreases in MEP amplitudes and CMEP amplitudes remain unchanged, this suggests that supraspinal factors may be largely responsible for changes in central fatigue. Distinguishing between supraspinal and spinal factors can often be difficult as both MEP and CMEP amplitudes can decrease simultaneously with fatigue, highlighting the importance of more detailed investigations to further understand the mechanisms contributing to central fatigue.

Understanding central fatigue is complex, as supraspinal and/or spinal factors can contribute to the observed decrease in voluntary activation. In order to have a more comprehensive understanding of central fatigue, this section will discuss some of the important studies that have contributed to the understanding of central fatigue. A study by Taylor and colleagues (1996) examined EMG responses to TMS and TMES during a sustained two-minute MVC of the elbow flexors (i.e., biceps brachii and brachioradialis) following brief repeated MVCs of the elbow flexors used for control values. These authors found that the MEP area increased in size, whereas the CMEP area remained relatively unchanged during the sustained two-minute MVC. This suggests that the increase in MEP area was due to changes in excitability of the motor cortex during elbow flexor MVCs. Taylor et al. (1996, 2000) also reported an increase in the length of the silent period during sustained or repeated MVCs of the elbow flexors. The silent period is an interruption in the ongoing surface EMG signal after a MEP has been evoked using TMS and can last for more than 200 ms (Gandevia, 2001; Taylor & Gandevia, 2008). The first 100 milliseconds consist of both supraspinal and spinal mechanisms, while the latter part of the silent period

is mainly due to intracortical inhibition (Fuhr et al., 1991). As a result, changes in the length of the silent period may suggest alterations in intracortical inhibition (Priori et al., 1994). Taylor et al. (1996) found that with the addition of spindle input by tendon vibration at the end of the sustained MVC, the silent period did not recover. These findings suggest that the changes in the silent period following the fatiguing task were unlikely due to alterations in spinal excitability or changes in afferent input to the cortex. Thus, these findings were likely due to changes from intrinsic cortical processes and/or altered voluntary drive to the motor cortex (Taylor et al., 1996). McKay et al. (1994) reported similar lengthening of the silent period during fatiguing voluntary contractions of the tibialis anterior muscle, without an increase in the size of the MEP. Using MEP and CMEP amplitudes in combination with changes in silent period may help provide a better understanding of the supraspinal and spinal factors responsible for central fatigue.

To further probe the impact of fatigue on the silent period short-interval intracortical inhibition (SICI) (Benwell et al., 2006), long-interval intracortical inhibition (LICI) (Benwell et al., 2007), and corticospinal excitability (Benwell et al., 2006; Benwell et al., 2007) with fatiguing hand exercises have been studied to determine if the changes in LICI and/or SICI corresponded to the changes in the silent period duration. An increase in MEP amplitude and silent period were reported in both studies. Benwell et al. (2006) found that SICI declined and likewise, Benwell et al. (2007) found that LICI declined with the fatiguing hand exercise protocol. The studies found that the fatiguing hand exercise protocol resulted in a constant increase in corticospinal excitability observed by the increase in MEP size and silent period and was associated with a decline in both SICI and LICI. These observations suggest that during fatigue there is an increase in the central

motor drive through changes in both inhibitory and excitatory networks, implying different neuronal populations may be affected. Benwell et al. (2007) suggested that the increase in the silent period duration may be due to processes of central fatigue in centres upstream of the primary motor cortex. Additionally, the authors suggested that the decrease in LICI along with the increase in MEP amplitude may be a compensatory mechanism of central fatigue to increase output of the primary motor cortex. In contrast to these above findings, Fernandez-del-Olmo et al. (2013) found that there were no changes in MEP/EMG ratio or silent period during brief MVCs following two 30-second Wingate tests (i.e., maximal leg cycling sprints), despite significant decreases in voluntary activation of the knee extensors (e.g. 34% decrease from baseline). These results indicate that alterations in corticospinal excitability may not always be observed with central fatigue, and therefore, potential mechanisms of central fatigue may vary depending on the exercise type, intensity, and duration.

Another potential mechanism of central fatigue is the increased activation of group III/IV muscle afferents that have been shown to inhibit the cortex and decrease central motor drive during isometric contractions (Gandevia et al., 1996; Taylor et al., 1996) and high intensity cycling (Amann et al., 2008; Amann et al., 2011; Sidhu et al., 2017). Amann et al. (2008) were the first to demonstrate that sensory feedback from the exercising limb exerts inhibitory influence on central motor drive. These findings were confirmed by Amann et al. (2011) who found that the blockade of group III/IV muscle afferents enhanced central motor drive during the latter phases of constant load leg cycling. Without the inhibitory influence of group III/IV muscle afferents, participants were able to demonstrate improvements in exercise performance. Thus, these findings confirm that the inhibitory

influence of group III/IV muscle afferents facilitate central fatigue. Recently, Sidhu et al. (2017) investigated the effect of group III/IV muscle afferents and corticospinal excitability during a non-fatiguing and fatiguing leg cycling exercise. The results suggested that in the absence of fatigue, group III/IV muscle afferent feedback facilitates the excitability of the motor cortical cells, while inhibiting the excitability of motoneurons. However, in the presence of fatigue, the results suggested that group III/IV muscle afferent feedback exerts an inhibitory influence on the excitability of the motor cortical cells without affecting the excitability of spinal motoneurons. In summary, the development of central fatigue during fatiguing locomotor exercises is believed to be at least partly due to group III/IV muscle afferent feedback.

2.2 Assessment of Neuromuscular Fatigue

Neuromuscular fatigue can be assessed using a number of different techniques such as, the interpolated twitch technique (ITT), EMG, TMS, and TMES (Gandevia, 2001). Although TMS and TMES contribute to the understanding of mechanisms responsible for central fatigue, this thesis will not use these techniques to help answer the research question. Therefore, this review will focus on how the ITT and EMG can be used to assess neuromuscular fatigue, as well as methodological considerations for each technique.

2.2.1 The Interpolated Twitch Technique and its Methodological Considerations

The interpolated twitch technique (ITT) was developed by Merton in 1954 (Merton, 1954) to examine the ability of the central nervous system to fully activate a muscle (i.e., voluntary activation) (Behm et al., 1996). The ITT can detect the presence of muscles fibers not activated during a voluntary contraction by applying an electrical stimulation to the

motor point during (i.e., interpolated or superimposed twitch) and following (i.e., potentiated twitch) an MVC (Behm et al., 1996; Shield & Zhou, 2004). It is a reliable (Behm et al., 1996) and valid (Behm et al., 1996; Taylor, 2009) measure of the voluntary activation of the muscle, and thus has been used many times in the literature. Using ITT, the central component of neuromuscular fatigue can be quantified by measuring the amplitude of the interpolated twitch force during the MVC. The interpolated twitch is also known as superimposed twitch and will be referred to as the superimposed twitch throughout this paper. An increased superimposed twitch force is due to additional increases in force above the maximal contraction, which suggests that there are additional motor units that have not been fully recruited or firing at maximal output (Herbert & Gandevia, 1999). The peripheral component of neuromuscular fatigue can be quantified by measuring the amplitude of the potentiated twitch force (Behm & St-Pierre, 1997; Belanger & McComas, 1981; Merton, 1954; Taylor & Gandevia, 2008). A percentage of voluntary activation can be calculated as a ratio of the amplitude values between the superimposed and potentiated twitch forces, using the following equation: **Voluntary Activation (%) = [1 – (superimposed twitch/potentiated twitch)] × 100** (Shield & Zhou, 2004). Thus, as the amplitude of the superimposed twitch force increases following a fatiguing maximal exercise bout, a decrease in the percentage of voluntary activation is observed. This decrease suggests the occurrence of central fatigue. In contrast, a decrease in the potentiated twitch amplitude following the maximal exercise bout would suggest the occurrence of peripheral fatigue (Shield & Zhou, 2004; Taylor & Gandevia, 2008). There are many methodological considerations with the ITT that will be discussed below.

2.2.2 Stimulation Intensities

Submaximal stimulation or supramaximal stimulation can be used for the ITT (Shield and Zhou, 2004), and both methods have been shown to be useful in unfatigued muscle (Rutherford et al., 1986). However, using submaximal stimulation for fatigue studies may not be appropriate since the threshold of motor axons increases during fatiguing contractions causing stimulation at any specific intensity to activate fewer motor units (Vagg et al., 1998). Thus, determining an optimal stimulus intensity is required to allow for complete recruitment of motor units in a fatigued muscle (Adam & DeLucam 2005; Neyroud, et al., 2014). Optimal stimulus intensity can be determined when there is a plateau in the evoked force, despite an increase in stimulation intensity (Adam & De Luca, 2005). Neyroud et al. (2014) sought to determine an appropriate stimulation intensity for adequate assessment of neuromuscular fatigue of the knee extensors. Three stimulation intensities (100, 120, and 150% of the optimal stimulus intensity) were used to assess voluntary activation before, during, and after a fatiguing isometric exercise. The authors determined that using 100% of the optimal stimulus intensity may overestimate the degree of peripheral fatigue because the intensity was not enough to ensure maximal recruitment of all motor units in the fatigued state. As well, they found that using 150% of the optimal stimulus intensity stimulations resulted in greater discomfort with doublet stimulations and an increased co-activation of the antagonist muscle (i.e., biceps femoris); whereas the 120% of optimal stimulus intensity stimulation did not result in significant differences in co-activation or discomfort when compared to 100% of optimal stimulus intensity. Therefore, Neyroud et al. (2014) recommended using 120% of optimal stimulus intensity when using the ITT for assessment of neuromuscular fatigue as it produces reliable measurements

while minimizing discomfort levels. The use of 120% of optimal stimulus intensity was also reported by Pearcey et al. (2016) for the ITT to assess neuromuscular fatigue of the elbow flexors following maximal arm cycling sprints.

2.2.3 Signal to Noise Ratio

When applying an electrical stimulation to the motor point of the muscle during an MVC, the superimposed twitch force response will decrease compared to submaximal contractions, while the fluctuations in voluntary force will increase. This results in a decreased signal-to-noise ratio (i.e., changes in the superimposed twitch force may go unnoticed; Dowling et al., 1994; Galganski et al., 1993). One way of improving this signal-to-noise ratio is through manipulation of the number of interpolated stimuli used to elicit the twitch and therefore estimate voluntary activation following a fatiguing task. Using one single stimulus interpolated over voluntary contractions was originally described by Merton (1954) and was used by other authors (e.g. Allen et al., 1995; Rutherford et al., 1986). It is now suggested that using two or more stimuli (i.e., doublet, triple, or quadruple) will result in larger evoked forced and thus, changes in evoked force will be more easily detected (Allen et al., 1998; Behm et al., 1996). Furthermore, the use of single stimuli is unsuited in studies assessing neuromuscular fatigue (Shield and Zhou. 2004) because changes in excitation-contraction coupling will cause a greater decrease in evoked responses using single stimuli when compared to evoked responses using multiple stimuli (Bigland et al., 1986; McKenzie et al., 1986). Thus, it has been suggested to use multiple stimuli to increase the single-to-noise ratio in order to increase the sensitivity of the ITT. More specifically, doublet stimuli separated by 10 ms is the most commonly used

interpolated stimuli to study central fatigue following a fatiguing task (Behm et al., 2002; McKenzie et al. 1992; Bigland et al., 1986; Pearcey et al., 2016, Pearcey et al., 2015, Monks et al., 2016).

2.2.4 Mechanisms influencing the measurement of voluntary activation

There are many complex mechanisms that may influence the relationship between potentiated twitch force and superimposed twitch force (i.e., voluntary activation) (Allen et al., 1998; Gandevia, 2001; Merton, 1951). One important mechanism to consider is an interpolated stimulus induces action potentials that propagate orthodromically (i.e., soma to axon terminal) and antidromically (i.e., axon terminal to soma) in motor and sensory axons (Allen et al., 1998; Shield and Zhou, 2004). The amplitude of the twitch may be influenced by antidromic action potentials colliding with orthodromic potentials in the motor axon which reduces the rate of motoneurone discharge immediate following the stimulus. To study factors that influence the amplitude of the superimposed twitch force, Herbert and Gandevia (1999) used a computer model of the adductor pollicis motoneurone pool. The computer model, based on experimental data, allowed the researchers to examine the effects of antidromic potentials on twitch amplitudes by comparing it to a hypothetical situation in which antidromic potentials do not occur. When antidromic collisions in motoneurons were excluded from the model, the superimposed twitch force amplitude was slightly increased at all contraction intensities. These findings suggest that both antidromic and spinal effects can decrease the amplitude of the superimposed twitch forces in submaximal and maximal contractions. Another mechanism to consider is the influence of unstimulated synergist and antagonist muscles on force production. If any antagonist

muscles are inadvertently stimulated, there may be slight decreases in the size of the superimposed twitch force (Gandevia, 2001). Furthermore, Allen et al. (1998) investigated the contribution of torque (i.e., moment of force) from synergist elbow flexor muscles by examining voluntary activation of the brachioradialis during a series of MVCs of the elbow flexors. When separate stimulations over the biceps brachii and brachioradialis were compared, voluntary activation of brachioradialis (median 91.5 %, range 68.9% to 100%) was lower than for the biceps brachii (median 99.1%, range 78.5%-100%). These results suggest that extra force may be generated by the brachioradialis at high voluntary torques. Since force would be increasing independently of an increase in voluntary drive to the stimulated muscle, misinterpretations of voluntary drive to the elbow flexors may occur. However, the level of voluntary activation remains valid if voluntary activation is calculated by expressing the responses evoked by a stimulus during voluntary efforts over the response evoked by the same stimulus in the relaxed muscle. Measurements of voluntary activation using the ITT with MVCs have been shown to be reproducible from day to day (Allen et al., 1995), and the technique is capable of detecting decrements in voluntary activation of less than 1% (Allen et al., 1998). Therefore, the ITT is a reliable and valid measure of voluntary activation of the muscle following a fatiguing task, despite the complex mechanisms related with the ITT discussed above.

2.2.5 Electromyography and its Methodological Considerations

Electromyography (EMG) is a recording of action potentials that are the result of depolarization and repolarization within the muscle fibre. All of the action potentials within the muscle that are detectable under the electrode site produce a motor unit action potential

(Medved & Cifrek, 2011; Konrad, 2005). Motor unit action potentials can be observed as a bipolar signal consisting of a symmetrical distribution of positive and negative amplitudes (i.e., mean value equals zero) (Konrad, 2005). The amplitude of the EMG signal produced by the recording of the motor unit action potentials is usually filtered and rectified, integrated or root mean square (RMS) values are calculated from the raw data (Rouffet & Hautier, 2008).

There are invasive and non-invasive methods for recording EMG. Invasive EMG recordings use fine wires or needles that are inserted directly into the muscle and thus, the recording is from a small volume of the muscle (Hug & Dorel, 2009). Surface EMG is a non-invasive approach to record muscular activity by using electrodes that are placed on the skin surface above the muscle of interest. The use of electrodes over the skin surface provides information from a large volume of muscle which is a better representation of the total muscle mass (Konrad, 2005). Therefore, surface EMG is the preferred method for recording electrical activity of muscles during dynamic movement (Hug & Dorel, 2009). For proper interpretation of the surface EMG signal, various factors that can influence the EMG signal must be considered. The main physiological factors include: muscle fibre membrane properties (e.g. muscle fibre conduction velocity), motor unit properties (e.g. firing rates), and muscle tissue characteristics (e.g. fibre diameter and subcutaneous tissue). Other non-physiological factors include: electrode location and orientation, cross talk (i.e., contamination of other muscles electrical activity), motion artifact (i.e., movement of electrodes or cables during the motor output), and external noise (Deluca, 1997, Hug & Dorel, 2009; Konrad, 2005; Rau et al., 2004).

DeLuca (1997) stated that there are three main applications of surface EMG when studying human movement. Surface EMG can be used to study the initiation of muscle activation, the relationship of force and muscle activity, and fatigue processes within the muscle. Monitoring surface EMG during fatiguing tasks is of interest as it allows researchers to assess the nervous system's ability to activate the contracting muscle by quantifying changes in the electrical activity of the muscle (Dimitrova & Dimitrov, 2003; Girard et al., 2011). When using surface EMG to measure fatigue, using the median frequency calculated from the frequency spectrum of the EMG signal is preferred (DeLuca, 1997). The EMG median frequency is defined as the frequency at which 50% of the total power within the epoch is reached (Konrad, 2005). During muscular contractions, neuromuscular fatigue causes changes in the amplitude and frequency of the EMG signal. The amplitude will show an increase with increasing motor unit recruitment until the nervous system is maximally activated, whereas the median frequency of the total power spectrum will generally result in a shift from higher frequencies to lower frequencies over the contraction period (Konrad, 2005). This suggests that decreases in the median frequency of the total power spectrum is observed during fatiguing contractions.

Many authors have studied the effect of neuromuscular fatigue on EMG parameters during isometric contractions (Bigland-Ritchie et al., 1986; Moritani et al., 1981) and during dynamic exercise (Billaut et al., 2005; Billaut et al., 2006; Hautier et al., 2000). Piper (1912) was the first to report a reduction in frequency of the surface EMG (Piper rhythm), while Cobbs and Forbes (1923) reported a consistent increase in the amplitude of the surface EMG recording during a sustained fatiguing contraction. This suggests that approximately 100 years ago, the EMG power spectrum shifts and alterations in EMG

amplitude were observed during fatiguing tasks. The EMG power spectrum shifts during fatigue have been reported by many authors since then and is suggested to be partly due to the slowing of the muscle fiber propagation velocity (DeLuca, 1997; Millis, 1982; Vøllestad, 1997). In contrast, interpreting changes in the amplitude of the EMG signal can be more complicated and contradictory. Many studies have reported decreases in EMG amplitudes (e.g. integrated and/or RMS) following fatiguing tasks (Billaut & Smith, 2010; Mendez-Villanueva et al., 2007; Racinais et al., 2007); however, increased (Billaut & Basset, 2007) and almost unchanged (Billaut et al., 2005; Hautier et al., 2000) EMG amplitudes have also been reported. The changes in EMG amplitude may depend on whether the contractions required maximal or submaximal effort, as well as the degree of fatigue reported (Bishop, 2012; Gibson et al., 2001; Taylor & Gandevia, 2008). For example, with maximal effort repeated sprints where greater magnitudes of fatigue have been reported, decreases in EMG amplitude have been observed (Mendez-Villanueva et al., 2007; Racinais et al., 2007).

Studies by Hautier et al. (2000) and Billaut et al. (2005) demonstrated that analyzing EMG parameters during maximal leg cycling sprints are useful for understanding the influences of neuromuscular fatigue on the activation and intramuscular coordination of specific lower limb muscles throughout the exercise bout. Hautier et al. (2000) examined the changes in EMG that occur during fatigue by having participants perform 15 repeated 5-second maximal sprints on a cycle ergometer interspersed with 25 seconds of rest. The authors found that there was significantly less RMS EMG amplitude for the knee flexors/antagonist muscles (i.e., biceps femoris and gastrocnemius) during the 13th sprint compared to the first sprint. No amplitude changes were observed in gluteus maximus,

vastus lateralis, or rectus femoris, despite decreases in force production. These findings demonstrate a decrease in the efficiency of the EMG signal recorded on power producer muscles (i.e., vastus lateralis and gluteus maximus). As well, the authors suggest that the reported decrease in EMG activation from the first to 13th sprint for the antagonist muscles (i.e., biceps femoris and gastrocnemius) may demonstrate an efficient adaptation of the inter-muscle coordination in efforts to decrease coactivation as agonist force decreased throughout the sprinting protocol.

Similarly, Billaut et al. (2005), used surface EMG to analyze inter-muscle coordination following 10 intermittent 6 second cycling sprints, with 30 seconds of recovery. Their results indicated that fatigue caused a time delay in the muscle activation between vastus lateralis and biceps femoris, while integrated EMG of these muscles remained unchanged. As well, there was a reduction in maximal power output, which occurred in parallel to changes in the muscle coordination pattern after fatigue. These results demonstrate the importance of using EMG analysis when measuring neuromuscular fatigue as it may provide researchers with a better understanding of inter-muscle coordination that may occur as a result of fatigue.

2.3 Sprinting Exercises

The degree of reduction in maximal voluntary force or power and task performance is influenced by the intensity of muscular activity. During submaximal exercise, neuromuscular fatigue can occur without a reduction in task performance since the nervous system can compensate for reductions in force or power by increasing motor unit recruitment (Gibson et al., 2001; Taylor & Gandevia, 2008). In contrast, the nervous system

is unable to compensate during maximal exercise as the nervous system is maximally activated (i.e., all motor units are firing to produce maximal force) throughout the exercise (Gibson et al., 2001; Taylor & Gandevia, 2008). Therefore, high intensity exercise that requires maximal effort, such as sprinting, is useful when studying neuromuscular fatigue.

Sprinting exercises consist of brief periods of maximal work interspersed with relatively short recovery periods and are an essential component of many sports (e.g. soccer, hockey, basketball, and badminton.). Since maximal intensity repeated sprint exercises have been associated with the development of fatigue and decrements in physical performance, the ability to perform repeated sprints in combination with short recovery periods is fundamental to successful athletic performance (Bishop & Girard, 2010). Sprinting exercises are commonly performed during leg cycling, arm cycling, and running. Reductions in maximal power output (e.g. during cycling) or speed (e.g. during running) are common performance decrements observed during subsequent sprints (Bishop, 2012). Some factors responsible for these performance decrements include: limitations in energy supply (e.g. ATP provided by phosphocreatine breakdown and anaerobic glycolysis), metabolic by-product accumulation (e.g. lactate, hydrogen ions, inorganic phosphate ions), impairment in calcium kinetics, reduced excitation of the sarcolemma (e.g. increase in extracellular potassium ions), and failure to fully activate the contracting muscle (Bishop & Girard, 2010). Repeated sprints can be classified into two types of exercise: intermittent-sprint and repeated-sprint exercise (Girard et al., 2011). Intermittent-sprint exercise is described as short duration sprints (≤ 10 seconds) combined with sufficient recovery periods (60 to 300 seconds) that allow for close to full recovery of sprint performance. In

contrast, repeated-sprint exercise is described as short duration sprints (≤ 10 seconds) combined with brief recovery periods (≤ 60 seconds) (Balsom et al., 1992; Duffield et al., 2009). The duration of the recovery period has been shown to influence performance differently (Bishop & Claudius, 2005). During intermittent sprint exercise, there may be little or no performance decrement (Bishop & Claudius, 2005) whereas, with repeated sprint exercise, there is evident performance decrements (Bishop et al., 2004). Bishop et al. (2004) suggested that longer recovery periods may allow for additional return of energy stores (i.e., ATP and phosphocreatine) to prevent evident performance decrements. These findings are supported by Hureau et al. (2016) and Monks et al. (2016). Both studies reported that with shorter recovery times, there were greater decreases in peak power output during leg cycling. Although there was a significant effect of recovery duration on sprint performance, the recovery duration had no effect on the development of neuromuscular fatigue on the knee extensors. More specifically, both studies reported similar decreases in maximal force, voluntary activation, and potentiated twitch force, irrespective of recovery time. In contrast, Billaut and Basset (2007) reported that the shorter recovery pattern with successive leg cycling sprints may lead to greater neuromuscular adjustments and thus, fatigue processes may be differentially affected by the duration of recovery periods between sprints. The authors reported that decreasing recovery durations were associated with higher RMS EMG values. The differences in these findings may be due to the parameters used to assess neuromuscular fatigue. These contradictory findings suggest that more research on sprinting is required to gain a better understanding of the contribution of peripheral and central fatigue to the decline in exercise performance.

2.4 Neuromuscular Fatigue During and Following Maximal Repeated Sprints

Many studies have examined the development of neuromuscular fatigue during and following maximal leg cycling sprints (Billaut et al., 2005; Billaut et al., 2006; Girard et al., 2013; Hureau et al., 2016; Monks et al., 2016; Pearcey et al., 2015). In general, maximal repeated leg cycling sprints induce peripheral fatigue early and this fatigue persists throughout the sprints, while central fatigue occurs towards the end of the sprints. A recent study by Hureau and colleagues (2016) examined the development of neuromuscular fatigue during six separate days of maximal leg cycling sprint exercises with different lengths of passive rest in between sprints. Participants were asked to perform 1, 4, 6, 8, and 10, 10-second sprints with 30 seconds of passive recovery between sprints, as well as 8, 10-second sprints with 10 seconds of passive recovery between sprints. Using pre- and post-exercise measures, the authors reported reductions in power output, twitch force, as well as voluntary activation of the quadriceps from the first to sixth sprint for both 30-second and 10-second recovery periods. They concluded that both peripheral and central fatigue significantly contributed to the decline in power output, and that this decline may be due to the central nervous system limiting motor output to prevent excessive muscle fatigue. Similar findings were reported by Monks et al. (2016) and Pearcey et al. (2015). Using similar set-ups and methods, these studies found that following ten, 10 second sprints with either 30 or 180 seconds of recovery between sprints, there were significant decreases in MVC, voluntary activation, and twitch force of the dominant leg knee extensors. These two studies also examined the time-course of neuromuscular fatigue by taking measurements prior to the sprinting protocol (pre-sprint), and immediately following the completion of the fifth (post-sprint 5), and tenth (post-sprint 10) sprints. Decreases in total

work and knee extensor force were observed post-sprint 5, and the authors attributed this to peripheral fatigue. Following post-sprint 10, there were decreases in knee extensor force, voluntary activation, as well as potentiated twitch, which were attributed to the presence of both peripheral and central fatigue. These findings further support that peripheral fatigue occurs early and continues throughout the exercise bout, while central fatigue occurs later during maximal leg cycling sprints.

Pearcey et al. (2016) was the first study to examine the effects of maximal arm cycling sprints on neuromuscular fatigue. This experiment consisted of ten, 10-second arm cycling sprints interspersed with 150 seconds of rest. The study reported that maximal arm cycling sprints induced decreases in power during the sprints, as well as decreases in MVC force, potentiated twitch force, and voluntary activation of the elbow flexors following sprint 10. Similar to the findings from leg cycling studies, this study supports that both central and peripheral fatigue occurred in the elbow flexors, and that the pattern of fatigue is also similar supporting that peripheral fatigue occurs early, and central fatigue occurs later during the fatiguing exercise protocol. Additionally, Pearcey et al. (2016) also examined the effect of maximal arm cycling sprints on corticospinal excitability. When MEPs and CMEPs were compared from sprints 1 to 5 and 6 to 10, a decrease in supraspinal excitability following sprint 5 and an increase in spinal excitability following sprint 10 of the biceps brachii during a weak contraction was reported. The authors suggested that the decreased supraspinal excitability may have resulted from inhibition and disfacilitation of the motor cortex. These findings suggest that the central fatigue that was observed following sprints 5 to 10 was at least partly due to the increased feedback to the central nervous system from the group III/IV muscle afferents. Furthermore, a significant increase

in rating of perceived exertion (RPE) from sprints 1 to 10 was observed, indicating that the participants probably found each subsequent sprint more fatiguing. Since rating of perceived exertion (RPE) and pain during high-intensity cycling exercises are moderately correlated (Borg et al., 1985), the observed increase in RPE may suggest that the participants also found each subsequent sprint more painful. If the arm cycling sprints did in fact increase participants' pain, then this would further support that the increased activation of the group III/IV muscle afferents were at least partly contributing to the decrease in voluntary activation (i.e., central fatigue). Although Pearcey and colleagues (2016) have contributed substantially to the research on arm cycling sprints and neuromuscular fatigue, more research is required in order to develop a more inclusive understanding of the neuromuscular fatigue response to maximal repeated arm cycling sprints.

2.5 Arm cycling with Different Forearm Positions

Neuromuscular fatigue has been shown to be altered differently depending on the specific fatiguing task. Factors such as the type of contraction (i.e., dynamic versus isometric contraction), the musculature group involved, the intensity, and the duration of the exercise have all been shown to influence the development of neuromuscular fatigue (Girard et al., 2008). Previous research has demonstrated that neuromuscular fatigue appears to develop differently depending on the particular action of the muscle (Babault et al., 2006; Enoka & Stuart, 1992; Girard et al., 2011). One area of research that has received relatively little attention in relation to fatigue is how the fatigue of biceps brachii is altered during repeated arm cycling sprints when forearm position is changed. Comparing the

effect of forearm position during arm cycling sprints may allow researchers to assess this area of research. As previously discussed, Pearcey et al. (2016) was the only study to assess neuromuscular fatigue of the elbow flexors during maximal arm cycling sprints. The sprints were performed with the forearm in a pronated position. When the forearm is in a pronated position, biceps brachii is an agonist for elbow flexion and may not be the prime mover. However, when in a supinated forearm position, biceps brachii becomes the prime mover for elbow flexion (Drake et al., 2009). Therefore, it is possible that the fatigue profile of biceps brachii will be different when the muscle is acting as a prime mover for elbow flexion during supination, as opposed to an agonist for elbow flexion during pronation. Knowing if the fatigue profile of the biceps brachii is altered by forearm position would be beneficial, as this may impact experimental outcomes and alter conclusions drawn by researchers on the development of fatigue of the elbow flexors. Since there is currently no research in neuromuscular fatigue following maximal arm cycling sprints with different forearm positions, the following section will draw on studies examining muscle activity and corticospinal excitability during constant load arm cycling to discuss the potential for a difference in the development of neuromuscular fatigue.

Current research suggests that forearm position influences the activity of the central and peripheral nervous system. For example, Kleiber, Kunz, and Disselhorst-Klug (2015) investigated the relationship of muscular coordination of the brachioradialis and biceps brachii during elbow flexion in a pronated, supinated, and neutral forearm position using surface electromyography (EMG). The authors found that there was a significant difference observed in the muscle activity of the brachioradialis, but not the biceps brachii in the pronated forearm position compared to the supinated and neutral forearm position. A study

by Bressel et al. (2001) was one of the first to investigate the influence of forearm position during arm cycling but did so during constant load arm cycling and investigated the muscle activation via surface EMG. The participants arm cycled on an arm ergometer at a moderate intensity (25W, 60 rpm) for three minutes in three different forearm positions (pronated, supinated, and neutral). Surface EMG was recorded from the biceps brachii, lateral head of triceps brachii, middle deltoid, infraspinatus, and brachioradialis. Brachioradialis had greater muscle activity for the neutral forearm position compared to the other forearm positions, and the infraspinatus muscle showed greater muscle activity for the neutral versus supinated forearm position. The results of this study demonstrate that of the elbow flexors examined, only the muscular activity of brachioradialis and not biceps brachii are influenced by forearm position during a constant workload arm cycling task. This study highlighted the importance of further research in this area to determine if different forearm positions affect muscular activity at different loads since their results are only relevant to the load used in their study. Another study by Forman and colleagues (2016) was the first to demonstrate that corticospinal excitability to the biceps brachii was higher while arm cycling with the hand in a neutral position rather than a pronated position. While these studies did not investigate neuromuscular fatigue during maximal exercise, they do suggest that the central nervous system is modulated differently with different forearm positions during arm cycling. These findings may also suggest that different forearm positions during maximal arm cycling sprints may influence the development of neuromuscular fatigue differently. To date, the effect of forearm position during maximal arm cycling sprints has yet to be investigated. Therefore, a study examining the effect of forearm position on

neuromuscular fatigue following maximal arm cycling sprints would be an effective contribution to the existing literature.

2.6 Clinical and Practical Implications

Arm cycling is commonly used in rehabilitation clinics, research laboratories, and fitness training centers as a form of exercise (Bishop & Claudis, 2005; Bressel et al., 2000; Clausen et al., 1970; DiCarlo, 1988; Pearcey et al. 2015). Some physiological adaptations associated with arm cycling training that have been reported in both healthy and rehabilitative populations include increased maximal oxygen consumption (VO_2max), decreased resting and exercising heart rates, and increased physical work capacity (Clausen et al., 1970; DiCarlo, 1988). By understanding how fatigue contributes to a decrease in exercise performance, the information from arm cycling research may be able to contribute to the development of training protocols for rehabilitative and athletic purposes and may help to enhance overall physical performance (Bressel et al., 2000). Research investigating the neuromuscular response during maximal cycling sprints will provide more information on athletic training development, as the ability to recover and reproduce maximal effort in subsequent sprints may influence sport performance (Bishop & Claudis, 2005). Additionally, understanding the influence of forearm position on neuromuscular fatigue may help guide better rehabilitation strategies, which may be especially useful for individuals with spinal cord injury.

There is a growing interest in arm cycling training for individuals with spinal cord injuries (DiCarlo, 1988; Valent et al., 2008; Astorino & Thum, 2016). Individuals with spinal cord lesions tend to lose motor function below the level of the lesion, and this may

result in a more sedentary lifestyle. This may also lead to a decreased physical work capacity, which is a direct consequence of the paralysis of the body (Haisma et al., 2006). Deconditioning is likely to occur as it becomes more difficult to maintain an active lifestyle (Haisma et al., 2006; Valent et al., 2008). For this reason, it is important to determine effective exercise-based recovery options to improve exercise adherence and overall health status in this population (Astorino & Thum, 2016). Research examining arm cycle ergometry training of low to moderate intensity has shown significant training effects such as, improvements in VO₂ max, peak power output, upper body muscle strength, and pulmonary function (DiCarlo, 1988; Valent et al., 2008). Other studies have examined high intensity arm cycling training in individuals with spinal cord injuries and have reported increases in peak stroke volume and peak oxygen uptake (Brurok et al., 2011; Hasnan et al., 2013). Recently, Astorino and Thum (2016) compared physiological and perceptual responses between high intensity interval training, sprint interval training, and continuous endurance exercise in individuals with chronic spinal cord injury who were habitually active (≥ 4 h/week). The results showed that there was significantly higher enjoyment reported during high intensity interval training and sprint interval training compared to endurance training, despite higher metabolic strain. Since no side effects were reported with higher intensity exercise and training adaptations may be maximized by training at high intensities, this highlights the importance of gaining a better understanding of neuromuscular fatigue during maximal arm cycling sprints for various populations.

2.7 Conclusion

In conclusion, there is an abundance of existing literature on neuromuscular fatigue following maximal sprinting exercise. There are various valid and reliable techniques used for assessing neuromuscular fatigue; however, methodological considerations for each technique must be considered when interpreting the results of studies. The development of neuromuscular fatigue has been extensively researched to arrive at the conclusion that peripheral fatigue occurs early and continues throughout the exercise bout, while central fatigue occurs later. As well, the available research demonstrates that neuromuscular fatigue develops differently with different tasks, but there is still more research required to have a better understanding of how altering a task can affect performance and neuromuscular fatigue profiles. Therefore, the purpose of this thesis will be to examine neuromuscular fatigue of the elbow flexors during maximal arm cycling sprints in a pronated and supinated forearm position.

2.8 References

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Chapter 3

The effects of forearm position on neuromuscular fatigue of the elbow flexors during maximal arm cycling sprints

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Running Head: The effects of forearm position on neuromuscular fatigue during arm cycling sprints

3.0 ABSTRACT

Neuromuscular fatigue has been shown to be altered by duration, intensity, volume, as well as the specific action of the contracting muscles. Since neuromuscular characteristics of fatigue are influenced by the action of the muscle, this study sought to examine the effect of forearm position (i.e. pronated vs supinated) during maximal arm cycling sprints on neuromuscular fatigue (NMF) of the elbow flexors. Over two separate days, NMF was assessed in thirteen healthy male participants (age: 25.7 ± 5.3 years) during 10, 10s arm cycling sprints interspersed by 60s of rest, performed on an arm cycle ergometer in a counter-clockwise direction using both a pronated and supinated forearm position. Prior to and following sprinting, maximal voluntary contractions (MVCs) and potentiated twitch forces were recorded from both the elbow flexors and extensors, and elbow flexors only, respectively. The EMG median frequencies of the biceps brachii and brachioradialis during the elbow flexor MVCs and triceps brachii during the elbow extensor MVCs were recorded for pre-sprint 1 and post-sprint 10. Performance measures (e.g. peak power output, fatigue index), perceived level of pain and rating of perceived exertion (RPE) were recorded following each sprint. During pronated sprinting, participants were able to produce greater peak power output ($p = .045$), but also demonstrated greater fatigue index ($p < .001$) than during supinated sprinting. However, following supinated sprinting, the percentage decrease in potentiated twitch force from pre- to post-sprinting was significantly greater ($p < .001$) than that following pronated sprinting ($45.1 \pm 10.2\%$ vs $24.6 \pm 18.1\%$). Median frequency was affected by forearm position, as there were greater decreases in triceps brachii median frequencies ($p = .007$) and overall higher median frequencies for brachioradialis ($p = .038$) reported in the pronated condition. In addition, irrespective of

forearm position, sprint performance decreased from sprint 1 to sprint 10 and there was a significant effect of time for pain ($p < .001$) and RPE ($p < .001$). Interestingly, despite decreases in elbow flexor MVC force ($p < .001$) following the maximal sprints, there were no changes in elbow extensor MVC force ($p = .357$). Collectively, these findings suggest that there are differences in the NMF profile of the elbow flexors following arm cycling sprints when done in either a pronated or supinated forearm position.

Key words: Neuromuscular fatigue, forearm position, elbow flexors, arm cycling, sprints

3.1 INTRODUCTION

Sprinting exercises consists of brief periods of maximal work interspersed with relatively short recovery periods (Billaut et al., 2006; Bishop & Girard, 2010; Hureau et al., 2016; Monks et al., 2016; Pearcey et al., 2016, Pearcey et al., 2015). Repeated sprints can be used to examine neuromuscular fatigue and are often classified into two types of exercise: intermittent-sprint and repeated-sprint exercise (Girard, Mendez-Villanueva, & Bishop, 2011). Intermittent-sprint exercise is defined as short duration sprints of ≤ 10 seconds with recovery periods of 60 to 300 seconds. Repeated-sprint exercise is defined as short duration sprints of ≤ 10 seconds with brief recovery periods lasting ≤ 60 seconds (Balsom et al., 1992; Duffield et al., 2009). Reductions in maximal power output (e.g. during cycling) or speed (e.g. during running) are common performance decrements observed during both intermittent- and repeated-sprints (Bishop, 2012; Hureau et al., 2016; Monks et al., 2017; Pearcey et al., 2015). This decrease in muscular performance observed during repeated sprints is referred to as neuromuscular fatigue, which is defined as an exercise-induced reduction in the force generating capacity of working skeletal muscle (Amann, 2011; Gandevia et al., 1996).

Neuromuscular fatigue includes processes at all levels of the motor pathway between the brain and the muscle, and thus is comprised of both central and peripheral factors (Bishop, 2012; Billaut et al., 2006; Gandevia, 2001; Pearcey et al., 2015). Peripheral fatigue results from biochemical changes near the neuromuscular junction or terminal branches of the motor axon or within the muscle (Bishop, 2012). These biochemical changes may include metabolic by-product accumulation (e.g. lactate, hydrogen ions,

inorganic phosphate ions), impairment in calcium kinetics, and reduced excitation of the sarcolemma (e.g. increase in extracellular potassium ions). In contrast, central fatigue occurs proximal to the motor axons and is related to a failure of the central nervous system (i.e., brain and spinal cord) to “drive” the motoneurons (i.e., a reduction in voluntary activation of the muscle) (Gandevia, 2001; Taylor & Gandevia, 2008). This reduction in voluntary activation of the muscle is due to supraspinal (i.e., cortical neurones) and/or spinal (i.e., alpha-motoneurons) factors causing alterations in excitatory and inhibitory input to the muscle afferents and descending drive to the exercising muscles (Gandevia et al., 1995; Gandevia et al., 1996; Taylor & Gandevia, 2008).

Several studies have examined the effects of leg cycling sprints on the development of neuromuscular fatigue (Billaut et al., 2005; Billaut et al., 2006; Girard et al., 2013; Hureau et al., 2016; Monks et al., 2016; Pearcey et al., 2015; Racinais et al., 2007). Following leg cycling, decreases in maximal voluntary contraction (MVC) force, potentiated twitch force and voluntary activation of the knee extensors have been reported (Girard et al., 2013; Hureau et al., 2016; Monks et al., 2016; Pearcey et al., 2015; Racinais et al., 2007). To date, only one study (Pearcey et al., 2016) has examined the effects of arm cycling on the development of neuromuscular fatigue. Arm cycling sprints in a pronated forearm position resulted in decreases in MVC force, potentiated twitch force and voluntary activation of the elbow flexors, which are similar to the findings observed during leg cycling sprints. In general, research examining both leg and arm cycling sprints has found that they induce peripheral fatigue early and this fatigue persists throughout the sprints, while central fatigue occurs towards the end of the sprints (Hureau et al., 2016, Pearcey et al., 2016, Pearcey et al., 2015).

Factors such as the type of contraction (i.e., dynamic versus isometric contraction), the musculature group involved, the intensity, and the duration of the exercise have all been shown to influence the development of neuromuscular fatigue (Girard et al., 2008). Previous research has demonstrated that neuromuscular fatigue appears to develop differently depending on the particular action of the muscle (Babault et al., 2006; Enoka & Stuart, 1992; Girard et al., 2011). Current research suggests that forearm position influences the activity of the central and peripheral nervous system. For example, Kleiber, Kunz, and Disselhorst-Klug (2015) investigated the relationship of muscular coordination of the brachioradialis and biceps brachii during elbow flexion in a pronated, supinated, and neutral forearm position using surface electromyography (EMG). The authors found that there was a significant difference observed in the muscle activity of the brachioradialis, but not the biceps brachii in the pronated forearm position compared to the supinated and neutral forearm position. Bressel et al. (2000) was the first to examine the effect of forearm position during moderate intensity arm cycling and reported differences in the muscle activity of the brachioradialis, but not the biceps brachii using surface EMG in a neutral position compared to a pronated and supinated forearm position. Additionally, a study by Forman et al. (2016) examined the influence of neutral and pronated forearm positions on corticospinal excitability to the biceps brachii during a non-fatiguing arm cycling protocol. In support with previous findings, the authors reported no differences in muscle activation of the biceps brachii between forearm positions. Conversely, Forman et al. (2016) found that forearm position altered corticospinal and spinal excitability to the biceps brachii during arm cycling as corticospinal and spinal excitability were higher while arm cycling with a neutral rather than a pronated forearm position. While these studies did not

investigate neuromuscular fatigue during maximal exercise, they do suggest that the central nervous system is modulated differently during dynamic exercises involving elbow flexion in different forearm positions. The effect of forearm position on neuromuscular fatigue during maximal arm cycling sprints has yet to be investigated. The biceps brachii is an agonist for elbow flexion when the forearm is in a pronated position. In contrast, the biceps brachii becomes the prime mover for elbow flexion when the forearm is in a supinated position (Drake, Vogl, & Mitchell, 2009; Kleiber, Kunz, & Disselhorst-Klug, 2015). Therefore, the fatigue profile of the biceps brachii may be different when the muscle is acting as a prime mover for elbow flexion during supination, as opposed to an agonist for elbow flexion during pronation. This highlights the importance of examining how the function of a muscle may alter fatigue during arm cycling.

The purpose of this study was to investigate the effects of pronated and supinated forearm positions on neuromuscular fatigue during maximal intensity arm cycling sprints. First, we sought to determine how forearm position influences sprint performance, pain, and rating of perceived exertion during the sprinting protocol and secondly, how forearm position influences elbow flexion and extension force and elbow flexor twitch force and elbow flexor and extensor EMG median frequencies measured pre- and post-sprint. There are two main hypotheses for this study. The authors believe that neuromuscular fatigue of the elbow flexors will occur following the arm cycling sprints in both forearm positions (i.e., pronated and supinated) and there will be a greater degree of neuromuscular fatigue in the elbow flexors during the supinated than pronated forearm position.

3.2 METHODS

A randomized cross-sectional within-subject study design was used to examine the effect of pronated and supinated forearm positions on neuromuscular fatigue of the elbow flexors following maximal arm cycling sprints.

3.2.1 Participants

Thirteen recreationally active (~10 h of activity/week) male participants (height 179.8 ± 7.6 cm, weight 87.3 ± 4.9 kg, age 25.7 ± 5.3 years) were recruited from the university population to participate in this study. All participants were accustomed to maximal bouts of exercise and had prior experience with performing arm cycling and MVCs with the interpolated twitch technique (ITT) protocol. All participants read and signed a written informed consent form prior to the study. The participants were instructed to complete the Physical Activity Readiness Questionnaire Plus (PAR-Q+) and follow the Canadian Society for Exercise Physiology (CSEP 2003) preliminary instructions (no eating, drinking caffeine, smoking, or drink alcohol for 2, 2, 2, or 6 h, respectively) prior to the start of testing. Additionally, each participant was asked to refrain from heavy exercise 24 h before testing. The Memorial University of Newfoundland Interdisciplinary Committee on Ethics in Human Research approved the study (#20151742-HK), which was administered in accordance with the Tri-Council guidelines in Canada with full disclosure of potential risks to participants.

3.2.2 Arm-cycle ergometer sprint protocol

All arm-cycling sprints were completed using a Velotron ergometer (DynaFit Pro, RacerMate, Seattle, Washington), which was modified for arm cycling (Fig. 1B). The Velotron ergometer can only record data and apply load when cycling is performed in one

direction. This meant that for the present study participants were required to cycle backwards (i.e. in a counter-clockwise direction). Previous research has suggested that backward arm cycling represents a simple reversal from forward arm cycling and similar patterns of EMG and cutaneous reflex amplitudes were reported for both directions (Zehr and Hundza, 2004). Therefore, the decision to cycle in a backward direction was justified for the current study.

For all the cycling trials participants were seated in a chair with their feet strapped to the floor. The Velotron ergometer was positioned so that the center of the crank was in line horizontally with the participant's acromion. Each sprint was preceded by 10 seconds of comfortable cycling at 100 rpm. The sprint phase began immediately following the 10 seconds of slow cycling (Fig. 1C). During all sprints the mechanical brake applied a 5% torque factor. The 5% torque factor resulted in a resistance that was equal to 5% of the participant's bodyweight. This was based on the work of Forbes et al. (2014) who reported that this level of resistance resulted in the highest mean power output over 30 seconds in trained individuals.

All participants were instructed to accelerate following the initiation of the mechanical brake that occurred immediately after the 10-second countdown displayed on Velotron Wingate Software version 1.0 (RacerMate, Seattle, Washington) that was made visible to each participant during the testing protocol. Verbal encouragement was given to each participant to cycle as fast as they could for the 10 sprints. The sprint phase was followed by 60 s of rest where participants were asked to rate their perceived exertion (RPE) using Borg's RPE scale (Borg, 1982). This scale allows participants to rate their

subjective exercise intensity on a scale of 6-20, where 6 is equivalent to complete rest and 20 is equivalent to maximum effort. Perceived ratings of pain were also recorded during each rest period using a scale of 0-10, where 0 is equivalent to no pain and 10 is equivalent to extremely intense pain. This process was repeated 10 times, for a total of 10 sprints.

3.2.3 Elbow flexors and extensors force

To determine the elbow flexor and extensor force of the dominant arm, participants sat in a chair in an upright position with the hips, knees, and elbows flexed at 90° while the upper torso was rested against the backrest. With their forearm in a supinated position, participants inserted their dominant wrist into a non-compliant padded strap which contained a load cell (Omegadyne, Inc., Sunbury, Ohio, USA). The load cell detected forces, which were amplified (x2000) (MP-150, BIOPAC Systems Inc., Santa Barbara, CA, USA) and displayed on a computer screen. The data was sampled at 2000 Hz. Participants were instructed to maintain positions of their head, neck, shoulder, and arm while performing the MVCs. Participants were required to perform a maximal elbow flexor MVC by maximally flexing the elbow against resistance, followed by a maximal elbow extensor MVC by maximally extending the elbow against resistance prior to and following the sprinting protocol using the same set-up (Fig. 1 A). Visual feedback and verbal encouragement were given to all participants during contractions.

3.2.4 Electromyography

Electromyography (EMG) recordings were collected from the biceps brachii, the triceps brachii, and the brachioradialis muscles of the participant's dominant arm during the pre and post-sprint protocol (i.e., during MVCs and potentiated twitch force) using

BIOPAC data acquisition system (MP-150, Santa Barbara, CA, USA) and its associated software (AcqKnowledge 4.1). A bipolar configuration of disposable 10 mm Ag-AgCl surface EMG electrodes (MediTrace TM 130 Foam Electrodes with conductive adhesive hydrogel, Covidien IIC, Massachusetts, USA) were positioned 2 cm apart (center-to-center) over the mid-point of the muscle belly of the biceps brachii, lateral head of triceps brachii, and the brachioradialis. A ground electrode was placed over the lateral epicondyle. Indelible ink was used to help identify the same position of electrodes for the latter session. To improve signal quality, thorough skin preparation for all electrodes was required, which included shaving hair off of the desired area, abrading, and cleansing the skin with an isopropyl alcohol swab. EMG signals were differentially amplified (input impedance = 2 M Ω , common mode rejection ratio >110 dB min (50/60 Hz), gain \times 1000, noise <5 μ V) and filtered using a band-pass filter with cut off frequencies of 10 to 500 Hz. Analog to digital conversion was processed using a 16-bit convertor and EMG signals were sampled at 2000 Hz.

3.2.5 Motor Point Stimulation

Electrical stimulation was delivered via adhesive 10 mm Ag-AgCl electrodes secured to the skin on the biceps brachii distal tendon (cathode) and over the motor point (anode; just proximal and medial to the midpoint of the biceps brachii muscle belly. Current pulses were delivered as a doublet (10ms apart, 200 μ s duration, 110-250 mA amplitude) via a constant current stimulator (DS7AH, Digitimer Ltd., Welwyn Garden City, UK). A maximal twitch force was determined by gradually increasing the stimulation intensity until the twitch force of the elbow flexors no longer increased. A supramaximal stimulation

current (i.e., 20 % higher than that required to elicit maximal twitch force) was used for the protocol.

3.2.6 Experimental Protocol

A total of three sessions were required for this experiment: a familiarization session (~20 minutes) and two experimental sessions consisting of either pronated or supinated arm cycling sprints (~30 minutes each). The familiarization session was required for all participants to become accustomed to maximal arm cycling sprints in a pronated and supinated forearm position in a backward direction. The forearm position for the two sprinting sessions were randomly assigned for each participant. With randomization, 8 participants started

Once forearm position was determined during the first session, participants began each experimental session with a 4-min warm-up on a Monark ergometer (Monark 874E, Monark Exercise AB, Sweden) at a self-selected pace. Following the warm up, maximal twitch force of the elbow flexors in the dominant arm was determined prior to and potentiated twitch force was determined following an MVC of the elbow flexors. Elbow extensor MVC was also performed by each participant to determine maximal elbow extensor force. After the MVCs, participants completed ten, 10 second sprints on the Velotron cycle ergometer. Immediately following sprint 10 (within 20 seconds), participants' elbow flexor and extensor MVC force and PT force were measured. Minimizing the transition time from the Velotron cycle ergometer to the start of performing the MVCs post-sprint was imperative, since significant recovery of muscle function occurs 1-2 min following exercise (Froyd, Millet, & Noakes, 2013). Ratings of perceived exertion and pain were also recorded following each of the ten sprints. Participants then returned for

another sprinting session with the same protocol as above, but in the forearm position that was not performed during their first sprinting session. At least 48 hours of rest was given between the two sprinting sessions to alleviate possible delayed onset muscle soreness that may have been caused by the arm cycling sprints.

3.2.7 Data Analyses

All power output data was recorded and calculated using the Velotron Wingate Software version 1.0 and stored on a computer. Similar to Pearcey et al. (2015), six of the following variables were measured during each sprint: (1) peak, mean, and minimum power, (2) peak and minimum RPM, (3) anaerobic capacity (mean power divided by body weight [Watts/kg]), (4) anaerobic power (peak power divided by body weight (Watts/kg)), (5) fatigue index (peak power minus minimum power divided by test duration [Watts/s]) and (6) total work (average Watts multiplied by the duration (J)).

A frequency analysis was done using BIOPAC software (Acqknowledge 4.1). Using this software, a power spectral density analysis was performed, and the median frequency was calculated using methods described in the BIOPAC users' manual (BIOPAC Systems Inc., 2009). The median frequency of the biceps brachii, triceps brachii, and brachioradialis was calculated for each MVC prior to and following the 10 sprints. This was calculated over a 1 second epoch during the MVC to determine EMG median frequency. The EMG median frequency is defined as the frequency at which 50% of the total power within the epoch is reached (Konrad, 2005). The MVC and twitch force were determined by measuring the peak-to-peak amplitude of the trace. Average root mean square (RMS) EMG was calculated for a 1-second epoch during peak MVC force for the

biceps brachii and brachioradialis during the elbow flexor MVC and trieps brachii during the elbow extensor MVC.

3.2.8 Statistical Analyses

All statistics were performed using IBM's SPSS software (IBM SPSS, version 20.0; IBM Corp., Armonk, N.Y., USA). Assumptions of sphericity were tested using Mauchly's test and if violated, degrees of freedom were corrected using Greenhouse-Geisser estimates of sphericity. Two separate two-way repeated measures ANOVAs were performed to compare position (pronated and supinated) and time (pre- and post-sprinting) and to compare position and sprint number (sprint 1 to 10). Specifically, the ANOVAs were used to compare median frequency, peak twitch force, and MVC force at pre- and post-fatigue and RPE, pain, peak, average, and minimum power, peak and minimum RPM, anaerobic capacity, anaerobic power, and fatigue index across all sprint trials (1 to 10) in both the supinated and pronated position. Repeated pairwise comparisons using Bonferroni post-hoc tests were used to determine where significant differences existed when significant main effects and/or interaction effects were found. *F*-ratios were considered statistically significant at the $p < .05$ levels. Descriptive statistics in text and Tables 1, 2, and 3 include mean \pm SD and the figures include mean \pm SE.

3.3 RESULTS

3.3.0 Effect of forearm position on power outputs

The mean \pm SD and percent changes for the measures of power output in pronated and supinated forearm positions are reported in Tables 1 and 2, respectively. The mean \pm SD for sprints 1 to 10 and *p*-values for the measures of power output to compare forearm

position are displayed in Table 3. For all main effects of time complete post-hoc analyses were completed. For the present study, the only results of interest were the comparison between sprint 1 and 10, as it provided an indication of the effectiveness of the fatiguing protocol. As such that is the only post-hoc result that will be reported for main effects of time. For peak power, post-hoc results for sprints 1 to 5 and 6 to 10 will also be reported to determine if there were similar time courses of neuromuscular fatigue, as reported by previous studies (Pearcey et al., 2015; Pearcey et al., 2016; Monks et al., 2016).

There was a significant main effect for sprint number ($F_{(2.603, 31.231)} = 50.382, p < 0.001$) and position ($F_{(1, 12)} = 5.022, p = .045$), but not a significant interaction between position and sprint number ($F_{(9, 108)} = 1.930, p = .055$; Fig. 2) on peak power output. Post-hoc tests revealed that peak power output during pronated sprinting was $3.0 \pm 15.0\%$ higher than supinated sprinting. For the main effect of time, peak power output significantly ($p < .001$) decreased by $22.5 \pm 9\%$ from sprint 1 to sprint 10. As well, peak power decreased by 19.1% from sprints 1 to 5 ($p < .001$) and only 3.9% from sprints 6 to 10 ($p = .999$).

There was a significant main effect for sprint number ($F_{(2.406, 28.876)} = 74.095, p < .001$; $F_{(1.945, 23.335)} = 42.598, p < .001$; $F_{(2.829, 33.953)} = 81.845, p < .001$), but not position ($F_{(1, 12)} = 1.538, p = .239$; $F_{(1, 12)} = 0.906, p = .360$; $F_{(1, 12)} = .519, p = .486$) on average power, minimum power, and peak RPM, respectively. Irrespective of position, average power, minimum power, and peak RPM decreased by $24.0 \pm 7.0\%$, $21.5 \pm 11.5\%$, and $20.5 \pm 7.0\%$, respectively.

There was a significant main effect for sprint number ($F_{(2.840, 34.086)} = 86.369, p < .001$; $F_{(2.809, 33.703)} = 51.712, p < .001$) but not position ($F_{(1, 12)} = .529, p = .481$; $F_{(1, 12)} =$

3.140, $p = .102$) on anaerobic capacity and anaerobic power, respectively. Irrespective of position, anaerobic capacity and anaerobic power decreased by 23.5 ± 7.5 and 22.5 ± 9.0 %, respectively.

There was a main effect for sprint number ($F_{(2,402, 9)} = 18.294, p = .001$) and a main effect for position ($F_{(1,12)} = 6.396, p = .003$) with the pronated position having higher values of fatigue index than the supinated position. A significant interaction effect between position and time ($F_{(4,135, 9)} = 2.819, p = .033$) was also found. The post-hoc test revealed that fatigue index decreased ($p < .001$ and $p = .035$) by $27.7 \pm 18.8\%$ and 18.4 ± 27.8 %, from pre- to post-pronated and supinated sprinting, respectively.

3.3.1 RPE and Pain

There was a significant main effect for sprint number ($F_{(1,851, 22,212)} = 8.109, p < .001$), but not position ($F_{(1, 11)} = 3.850, p = .076$) on RPE (Fig. 3A). As well, there was a significant main effect for sprint number ($F_{(1,382, 15,203)} = 29.479, p < .001$), but not position ($F_{(1, 11)} = 3.850, p = .076$) on perceived rating of pain (Fig. 3B). Irrespective of position, pain and RPE increased by 28.4 ± 21.8 and 26.8 ± 29.0 %, respectively.

3.3.2 MVC force, EMG median frequency and RMS amplitude

There was a significant main effect for time ($F_{(1,12)} = 32.075, p < .001$), but not position ($F_{(1,12)} = 1.040, p = .328$) on elbow flexor MVC forces (Fig. 4A). Irrespective of position, MVC force decreased ($p < .001$), by $14.3 \pm 13.1\%$. There was no significant main effect for time ($F_{(1,12)} = .918, p = .357$) or position ($F_{(1,12)} = 2.725, p = .125$) on elbow extensor MVC (Fig. 4B).

There was a main effect of time ($F_{(1,12)} = 27.688, p < .001$; $F_{(1,11)} = 8.411, p = .014$) on biceps brachii and brachioradialis median frequencies (Fig. 5), respectively during the elbow flexors MVCs. As well, there was a main effect of position on the median frequency for the brachioradialis ($F_{(1,11)} = 5.550, p = .038$) (Fig. 5B), but not biceps brachii ($F_{(1,12)} = .495, p = .495$). There was no interaction effect for either muscle. Irrespective of position, median frequency of the biceps brachii and brachioradialis decreased by $13.8 \pm 10.6\%$ and $11.4 \pm 17.6\%$, respectively. Overall, median frequency of the brachioradialis was $4.3 \pm 11.5\%$ higher in pronation than supination.

There was a significant main effect for time ($F_{(1,12)} = 7.625, p = .017$;) and position ($F_{(1,12)} = 5.092, p = .043$), and an interaction effect between time and position ($F_{(1,12)} = 11.981, p = .005$) on the triceps brachii median frequency during the elbow extensor MVCs (Fig. 5C). Pairwise comparisons revealed that the median frequency decreased ($p = .007$) by $14.9 \pm 15.5\%$ from pre-sprint 1 to post-sprint 10 in a pronated forearm position. There was no difference in the median frequency between pre-sprint 1 and post-sprint 10 in a supinated forearm position ($p = .373$) or between pre-sprint 1 values for the two separate test sessions (i.e., pre-sprint 1 pronation and pre-sprint 1 supination; $p = .440$). The median frequency was $13.3 \pm 14.0\%$ higher ($p = .010$) for post-sprint supination compared to post-sprint pronation.

There was no significant effect for time ($F_{(1,12)} = .007, p = .936$; $F_{(1,12)} = .055, p = .819$) or position ($F_{(1,12)} = 1.503, p = .244$; $F_{(1,12)} = 1.458, p = .250$) on biceps brachii and brachioradialis RMS amplitude during the elbow flexor MVC, respectively. There was a

significant main effect for time ($F_{(1,12)} = 7.483, p = .018$) but not for position ($F_{(1,12)} = .110, p = .746$) on triceps brachii RMS amplitude during the elbow extensor MVC. Irrespective of position triceps brachii RMS amplitude was $8.1 \pm 9.1\%$ higher ($p = .018$) post-sprint.

3.3.4 Potentiated Twitch Force

There was a significant main effect for sprint number ($F_{(1,12)} = 78.960, p < .001$; Fig. 6A) but not position ($F_{(1,12)} = .045, p = .835$), and a significant interaction ($F_{(1,12)} = 13.437, p = .003$) between sprint number and position on potentiated twitch force. Overall, potentiated twitch force decreased by $35.7 \pm 10.2\%$ from pre-sprint 1 to post-sprint 10. There was no difference for potentiated twitch force between supination and pronation at pre-sprint or post-sprint. However, the percentage decrease in potentiated twitch force from pre- to post-sprint was significantly ($p < .001$) greater following supination ($45.1 \pm 10.2\%$) than pronation ($24.6 \pm 18.1\%$; Fig. 6B).

3.4 DISCUSSION

The most significant finding of the current study was that sprint performance was affected by forearm position during repeated arm cycling sprints in a counter-clockwise direction. This study demonstrated that there was more power produced but also a greater decrease in the ability to produce power over time (i.e., a greater decrease in fatigue index) during the pronated sprints than the supinated sprints. As well, pronated and supinated arm cycling sprinting led to differences observed in median frequency of the triceps brachii and brachioradialis during the MVCs that followed the arm cycling sprinting. More specifically, during the elbow extensor MVC there was a significant decrease in triceps brachii median frequency from pre-sprint 1 to post-sprint 10 following pronated sprinting, but not

supinated sprinting. The overall median frequency for brachioradialis was higher during the elbow flexor MVC following pronated compared to supinated sprinting. This suggests that there may have been a slightly different function of the brachioradialis during the pronated arm cycling. The elbow flexor force and EMG median frequency of the biceps brachii decreased in a similar manner from pre- to post-sprint in both hand positions. This was the first study to demonstrate that elbow extensor force remained unchanged following maximal arm cycling sprints, despite decreases in median frequency of the triceps brachii. Lastly, there was a greater reduction in percentage change for potentiated twitch force (i.e., a larger decrease from pre- to post-sprinting) following supinated sprinting. Irrespective of forearm position, counter-clockwise repeated sprint exercise performance decreased from the start to the end of the repeated sprints.

3.4.1 Effect of position on neuromuscular fatigue

The present study demonstrated that there are differences in the neuromuscular fatigue profile during repeated maximal arm cycling sprints when performed in a pronated compared to a supinated forearm position. Greater values of peak power and fatigue index were observed in the pronated forearm position compared to the supinated forearm position. Fatigue index is a measure of one's ability to produce watts per second and can be measured by subtracting the minimum power from the peak power divided by the test duration (Velotron, 2010). Therefore, a decrease in fatigue index from sprints 1 to 10 would indicate that the ability to produce power over time has decreased (e.g. a decrease in peak power and/or minimum power), suggesting fatigue has occurred. Since there were higher values of peak power measured in pronation, it is not surprising that the overall average for fatigue

index was higher in pronation than supination (Table 3). However, comparing the decrease in fatigue index from sprints 1 to 10 demonstrates that over time, participants ability to produce power per second decreased in both positions, but there was a greater decrease in the pronated position compared to the supinated position (i.e., 27.1% vs. 18.6% decrease; Tables 1 and 2). Since peripheral fatigue has been shown to play the most important role in reduced performance with repeated sprinting exercises (Goodall et al., 2015; Perrey et al., 2010), this may suggest that there was a greater degree of peripheral fatigue in the pronated forearm position.

Contradicting the hypothesis that greater peripheral fatigue occurred in the pronated position, is the fact that there was a greater percentage decrease in elbow flexor potentiated twitch force from pre- to post-sprint following supinated ($45.1 \pm 10.2\%$) vs. pronated ($24.6 \pm 18.1\%$) trials (Fig. 6B). This finding indicates that there may have been greater peripheral fatigue during the supinated trials, which is in contrast to the results discussed above. The discrepancy in these results could suggest that the mechanisms contributing to peripheral fatigue may be different depending on forearm position. In the current study, potentiated twitch force (i.e., measure of peripheral fatigue) was only measured from the elbow flexors and thus, did not consider fatigue that may have occurred in the elbow extensors or other active muscles during the arm cycling sprints. While the greater observed decrease in potentiated twitch force in the supinated forearm position may be explained by greater elbow flexor fatigue, the greater decrease in exercise performance in the pronated position might be due to fatigue of other muscles, namely the elbow extensors. During supinated arm cycling, the biceps brachii is in a position to be able to function more effectively as an

elbow flexor (Drake et al., 2009) compared to the pronated forearm position, and hence may explain the greater peripheral fatigue of the elbow flexors (as measured via potentiated twitch force) observed following supinated sprinting. During pronated arm cycling, however, it is possible that the elbow extensors play a larger role to the production of force, as the biceps brachii is not able to function optimally in this position (discussed further below). This idea is supported by the finding that triceps brachii median frequency (a measure of muscle fatigue; Billaut et al., 2006; Gerdle & Fugl-Meyer, 1992) showed a greater decline following pronated sprinting, suggesting that elbow extensor fatigue is greater following pronated sprinting. However, in the current study we were unable to quantify the contribution of muscles to force development during arm cycling, so we can only hypothesize on the role of the triceps brachii in the pronated forearm position. Interestingly, the hypothesis that the biceps brachii fatigued more when in a supinated forearm position, and that the triceps brachii fatigued more in a pronated forearm position, does not agree with the findings related to EMG amplitude and MVC force post-sprint 10, as neither of these measures indicated that fatigue was influenced by forearm position. These inconsistent findings may also be partly influenced by the study design as MVC and twitch force were measured with the hand in the supinated hand position only. This limitation will be discussed further in methodological considerations. Clearly, further research is needed in this area to determine how forearm position impacts elbow flexor and extensor fatigue during arm cycling.

Discussing the biomechanics of the elbow joint is pertinent for understanding the influence of forearm position on neuromuscular fatigue. The biceps brachii and the

brachioradialis are two superficial muscles involved in elbow flexion, while the triceps brachii is the main elbow extensor. As well, the biceps brachii is involved in supination of the forearm, and the brachioradialis is capable of acting as a synergistic muscle for supination and pronation, depending on the position of the forearm (Kleiber et al., 2015). Studies have shown that the brachioradialis changes its contribution to elbow flexion with forearm position (Howard, Hoit, Enoka, & Hasan, 1986; Kleiber et al., 2015; Praagman, Van der Helm, & Veeger, 2010). Kleiber and colleagues (2015) examined muscular coordination of biceps brachii and brachioradialis during elbow flexion in pronation, supination, and neutral forearm positions using surface EMG. The authors found that during elbow flexion, there was a greater contribution of brachioradialis in a pronated forearm position compared to a supinated and neutral position. Several studies have also reported similar findings (De Sousa, & De Moraes Vieira, 1961; Howard et al., 1986; Nakazawa, Kawakami, Fukunaga, Yano, & Miyashita, 1993; Praagman et al., 2010). Additionally, Kleiber et al. (2015) found that the activation level of the biceps brachii remained constant in all three forearm positions. In the current study, forearm position during arm cycling had no effect on the biceps brachii median frequency or elbow flexor MVC force measured post-sprinting. In support of these findings, Bressel et al. (2001) examined the effect of forearm position on muscular activity during three minutes of arm cycling at 60 rpm and found that the biceps brachii activation was not influenced by forearm position, whereas the muscle activity of the brachioradialis was. The brachioradialis' recruitment strategy may be different due to the biomechanical disadvantage of the biceps brachii in pronation (Kleiber et al., 2015). Specifically, the biceps brachii has a biomechanical disadvantage as an elbow flexor in pronation because the biceps tendon is

wrapped around the radial tuberosity (Howard et al., 1986), and therefore, the higher activity of the brachioradialis may be compensating for the lower torque produced by biceps brachii in pronation. The biomechanics of the biceps brachii and brachioradialis in pronation may contribute to the understanding of the present findings. There was an overall higher median frequency of brachioradialis observed following the pronated trials, compared to the supinated trials (Fig. 5B). This may be indicative of a slightly different function of brachioradialis in the pronated forearm position during cycling, which is in support with previous findings (Forman et al., 2016; Kleiber et al., 2015). An increase in median frequency implies a modification in the muscle fiber recruitment and may suggest an increase in conduction velocity of the muscle fibers (Billaut et al., 2003; Gerdle & Fugl-Meyer, 1992). If an increase in the conduction velocity of the muscle fibers did in fact occur, this may suggest that there was a higher activity of the muscle fibers in the pronated forearm position. A higher activity of the brachioradialis in pronation may at least partly explain the greater peak power observed during pronated arm cycling as a higher muscle activity would allow for a greater force contribution. Together, these findings suggest that forearm position influences the fatigue profile and the function of muscles during and following arm cycling. To gain a better understanding of how forearm position influences neuromuscular fatigue and the activation of various muscles, future studies should measure EMG during each sprint and incorporate measurements of central fatigue (e.g. voluntary activation and corticospinal excitability).

3.4.2 Effect of time on neuromuscular fatigue

Irrespective of forearm position, the maximal arm cycling sprinting protocol used

in this study demonstrated evidence of neuromuscular fatigue during the sprint itself and the MVC and potentiated twitch force that followed the sprints. Repeated sprinting led to decreases in peak, average, and minimum power, peak RPM, anaerobic capacity, anaerobic power, and fatigue index. There was also an increase in RPE and pain during the sprints. Following the sprints, potentiated twitch force and elbow flexor but not elbow extensor MVC decreased. As well, median frequency for the biceps brachii and brachioradialis also decreased without any changes reported for EMG amplitudes during the MVCs. These findings indicate that the 10-second repeated sprint protocol resulted in neuromuscular fatigue in both forearm positions.

In the present study, peak power decreased as the number of arm cycling sprints performed increased. Pearcey et al. (2016) reported similar declines in peak power following 10, 10-second arm cycling sprints interspersed with 150 seconds of rest. Other studies examining repeated and intermittent leg cycling sprints have also reported similar findings (Girard et al., 2013a; Hureau et al., 2016; Mendez- Villanueva et al., 2012; Monks et al., 2017; Pearcey et al., 2015; Racinais et al., 2007). Many studies examining leg cycling sprints (Girard et al., 2013b; Mendez- Villanueva et al., 2012; Monks et al., 2017; Pearcey et al., 2015) and one study examining arm cycling sprints (Pearcey et al., 2016) have shown that the rate of decline in power is higher during the first half of the sprinting protocol (e.g., sprints 1 to 5) compared with the last half of the sprinting protocol (e.g., sprints 6 to 10). This study found a similar pattern as there was a 19.1% decline in peak power for sprints 1 to 5 ($p < .001$) and only a further 4.0% decline in peak power for sprints 6 to 10 ($p = .999$). The greater decline in sprint performance during the first half of the sprinting protocol

suggests that the development of neuromuscular fatigue may not be uniform (Collins, Pearcey, Buckle, Power & Button, 2018). More specifically, several studies during leg cycling (Girard et al., 2013b; Hureau et al., 2016; Mendez- Villanueva et al., 2012; Pearcey et al., 2015) and one study during arm cycling (Pearcey et al., 2016) have shown that maximal repeated sprints induce peripheral fatigue early, and this fatigue persists throughout the sprints, while central fatigue occurs towards the end of the sprinting protocol. Therefore, this suggests that peripheral fatigue may have been the main contributor to the decline in sprint performance in the present study, as most of the decline in peak power occurred during the first half of the sprinting protocol. However, the overall decline in sprint performance is most likely attributed to both peripheral and central fatigue as supported by previous research (Hureau et al., 2016; Monks et al., 2016; Pearcey et al., 2015; Pearcey et al., 2016). Central fatigue has been suggested to prevent further increases in peripheral fatigue as a way to protect damage and injury to the exercising muscles (Gandevia, 2001).

Overall, there were decreases in MVC and potentiated twitch force of the elbow flexors following sprinting in both forearm positions. Peripheral fatigue has been suggested to contribute to the decline in MVC and potentiated twitch force following maximal sprinting protocols (Billaut et al., 2006; Pearcey et al., 2016; Racinais et al., 2007). Similar to the present study, decreases in MVC and potentiated twitch force have been reported following several types of sprinting protocols including running (Goodall et al., 2015), leg cycling (Girard et al., 2013a; Hureau et al., 2016; Pearcey et al., 2015, Monks et al., 2017) and arm cycling (Pearcey et al., 2016). In addition to decreases in potentiated twitch force,

Pearcey et al. (2016) also found that there were no changes in the amplitude of the maximal M-wave (M_{\max}) following the arm cycling sprints, which suggests that peripheral fatigue occurred beyond the sarcolemma. While it is impossible to draw similar conclusions based on the data collected for the present study, given the similarities between protocols and participants examined in this and the study by Pearcey et al. (2016), it would seem logical to suggest similar mechanisms in the current study. Changes beyond the neuromuscular junction that may be contributing to the peripheral fatigue include impairments of the excitation contraction coupling process and sarcolemma excitability (Allen et al., 2008; Bishop, 2012). These impairments are influenced by the release and restoration of intracellular calcium from the sarcoplasmic reticulum and the reduced sensitivity between contractile proteins and calcium (Allen et al., 2008; Bishop, 2012; Glaister, 2005). Potential mechanisms for impairments of the excitation contraction coupling process and sarcolemma excitability may include increased blood lactate (Pearcey et al., 2015), increased hydrogen ions (Bishop et al., 2004), muscle deoxygenation (Racinais et al., 2007), and reduced blood lactate removal (Fernandez-del-Olmo et al., 2013). These mechanisms may result in reduced free Ca^{2+} concentration causing reduced cross bridge formation (Goodall et al., 2015), which may partly explain the observed decreases in force development in the current study.

Central fatigue has also been suggested to contribute to the decline in MVC force (Gandevia et al., 1996; Gandevia, 2001). Decreases in MVC force with concurrent decreases in voluntary activation indicate that there is a failure of the central nervous system (i.e., brain and spinal cord) to “drive” the motoneurons, and this can be attributed to

reduced supraspinal (i.e., cortical neurones) and/or spinal (i.e., alpha-motorneurones) “drive” (Gandevia, 2001; Taylor & Gandevia, 2008). Specific measures of voluntary activation, such as interpolated twitch technique and transcranial magnet stimulation, were not examined in this study, which is a limitation and will be discussed in methodological considerations. Pearcey et al. (2016) was the only study to examine voluntary activation of the elbow flexors following maximal arm cycling sprints and the authors found that voluntary activation significantly decreased. They hypothesized that the observed decrease in voluntary activation may have been due to increased feedback to the central nervous system from group III/IV muscle afferents (Pearcey et al., 2016). Studies examining the effect group III/IV muscle afferents have on central motor drive during high intensity leg cycling protocols have found that the activation of group III/IV muscle afferents act to inhibit the motor cortex which promotes central fatigue (Amann et al., 2008; Amann et al., 2011; Sidhu et al., 2017). The present study found that as sprint number increased, the amount of perceived pain also increased. Increased pain can be associated with increased activation of group III/IV muscle afferents which may suggest that the present study also resulted in inhibition of the motor cortex. Future studies examining pronated and supinated arm cycling should include measures of voluntary activation and corticospinal excitability to definitively determine which mechanisms related to central fatigue may have influenced the findings in the present study.

Neuromuscular fatigue can cause changes in the amplitude and frequency of the EMG signal. The amplitude will show an increase with increasing motor unit recruitment until the muscles are maximally activated, whereas the median frequency will generally

decrease over the contraction period (Konrad, 2005). The present study found decreases in EMG median frequency during MVCs for the biceps brachii and brachioradialis in both forearm positions and for the lateral head of triceps brachii from pre-sprint 1 to post-sprint 10 in pronated forearm positions (Fig. 5). Studies examining fatigue during leg cycling have also reported decreases in median frequencies of the contracting muscles (Billaut et al., 2006; Mendez-Villanueva et al., 2007; Pearcey et al., 2015; Taylor et al., 1997). Therefore, decreases in median frequency reported for biceps brachii and brachioradialis in both forearm positions further demonstrates that neuromuscular fatigue occurred in the elbow flexors following 10, 10-second arm cycling sprints. Despite decreases in median frequency, there were no changes in EMG amplitude for biceps brachii or brachioradialis. In contrast to these findings, many studies have reported decreases in EMG amplitudes following fatiguing tasks (Billaut & Smith, 2010; Mendez-Villanueva et al., 2007; Racinais et al., 2007). However, increased (Billaut & Basset, 2007) and almost unchanged (Billaut et al., 2005; Hautier et al., 2000) EMG amplitudes have also been reported. These contradictory findings demonstrate that further research is needed to draw conclusions on the effect of forearm position on fatigue using EMG amplitudes and median frequencies. Moreover, the next step in this research would be to record EMG amplitudes *during* the cycling sprints to supplement existing information regarding muscle activity and neuromuscular fatigue.

To date, no study has reported differences in the effect of maximal arm cycling sprints between the biceps and triceps brachii. As previously mentioned, the current study found that elbow flexor MVC force decreased, while elbow extensor MVC force remained

unchanged from pre- to post-sprinting in both forearm positions. This may suggest that greater fatigue occurred in the elbow flexors than elbow extensors during maximal repeated arm cycling sprints in the counter-clockwise direction. Little is known about the triceps brachii during fatiguing arm cycling tasks, and therefore previous research examining elbow flexors and extensors during tonic and dynamic contractions will be used to draw conclusions on the findings of the present study. Using a tonic contraction, Martin et al. (2006) sought to determine whether afferents activated by fatigue of the flexor and extensor muscles during elbow flexor and extensor MVCs were different. The authors found that motoneurons of flexor and extensor muscles are affected differently by inputs from group III/IV afferents. Specifically, afferent input from homonymous and antagonist muscles inhibit extensor motoneurons, while facilitating motoneurons innervating the flexors. Additionally, using an arm cycling protocol at 5 and 15% of peak power, Spence et al. (2016) was the first study to report that corticospinal excitability to the biceps and triceps brachii appeared to be modulated differently during arm cycling. More specifically, corticospinal and spinal excitability to the biceps brachii demonstrated a phase dependency, as both measurements were higher during elbow flexion than elbow extension. However, the triceps brachii did not demonstrate phase dependent changes as spinal excitability to the triceps brachii was higher during elbow flexion. These findings suggest that during non-fatiguing arm cycling, the biceps and triceps brachii may be controlled differently by supraspinal and spinal centers. Although corticospinal excitability was not measured in the current study, drawing on these findings may partly explain the observed differences in elbow flexor and extensor force following arm cycling sprints.

3.4.3 Methodological considerations

In the current study, there are several factors to consider in the interpretation of the results. This study required participants to arm cycle in a reverse direction (i.e., counter-clockwise direction). It is possible that the results observed may be unique to backward cycling and not transferrable to the more typically performed forward cycling. However, Zehr and Hundza (2004) compared forward and backward arm cycling and found there were similar EMG patterns and cutaneous reflex amplitudes for rhythmic arm cycling in both forward and backward directions. This suggests that the direction of the cycling performed in the current study should not have influenced the results, but further research is required to be certain.

Most measurements used to quantify fatigue following maximal arm cycling sprints were measured during isometric contractions (i.e., MVC, median frequency, and potentiated twitch force). Performing dynamic movements like arm cycling requires rhythmic and alternating patterns of muscle activation, which is different from isometric contractions. The activation levels of specific muscles may be different in isometric compared to dynamic contractions, which was suggested by Forman et al. (2015). Therefore, using mainly isometric contractions to measure fatigue may not have been sufficient to compare the neuromuscular fatigue profiles in both forearm positions. Additionally, another important consideration in the current study is that all MVCs were performed with the forearm in a supinated position following both the pronated and supinated cycling sprints. Perhaps the isometric contractions should have been performed in the same forearm position as the arm cycling session. We used the same position for both

arm cycling sessions to allow for a better comparison between sessions. An area of interest for future studies is whether the influence of forearm position affects isometric fatigue measurements (e.g. MVC force, EMG median frequency and amplitudes, potentiated twitch force and voluntary activation).

This study did not use specific measures of voluntary activation, which makes it difficult to draw conclusions on the occurrence of central fatigue. Future studies should use the interpolated twitch protocol to compare voluntary activation in both pronated and supinated forearm positions. Additionally, future studies in our lab will measure EMG amplitudes during arm cycling sprints to compare muscle activity in each forearm position. Having a measurement of muscle activity during sprinting protocols will provide insight on the effect of fatigue on the activation and inter-muscle coordination of the active muscles in each forearm position.

Furthermore, the EMG median frequency and amplitude of the triceps brachii must be interpreted with caution as the lateral head, but not the medial and long head, were used to quantify the frequency. A study by Davidson and Rice (2010) suggested that measuring from one muscle portion of the elbow extensors are not representative of the entire muscle group, as shoulder angle and muscle length can affect the EMG activity and fatigability of the elbow extensors during tonic contractions. These findings will be taken into consideration in future studies when using EMG to examine muscle activity during arm cycling.

3.5 CONCLUSION

The present study demonstrates that there were differences in the development of neuromuscular fatigue during maximal arm cycling sprints in pronated and supinated forearm positions. Forearm position did influence sprint performance, EMG median frequency during MVCs, as well as potentiated twitch force of the elbow flexors. This is the first study to demonstrate that there were no changes in MVC force of the elbow extensors, despite decreases in MVC force of the elbow flexors following maximal arm cycling sprints. Future studies examining the differences of forearm position on neuromuscular fatigue should measure voluntary activation pre and post-sprinting and measure EMG during cycling to allow for a more comprehensive comparison between forearm positions during maximal arm cycling sprints.

3.6 ACKNOWLEDGEMENTS

The authors would like to thank Dr. Thamer Alkanani at Memorial University for technical assistance.

3.7 LIST OF TABLES

Table 1. Raw data for peak power, average power, minimum power, peak RPM, minimum RPM, anaerobic capacity, anaerobic power, and fatigue index recorded during each sprint in the pronated forearm position. All data shown as mean \pm SD. Percent changes and p-values represents changes and significance between sprints 1 and 10.

Measurements for each 10-second sprint in the pronated forearm position								
Sprint	Peak Power (W)	Avg. Power (W)	Min. Power (W)	Peak RPM	Min RPM	An. Capacity (mean W/kg)	An. Power (peak W/kg)	Fatigue Index (W/s)
1	607.1 \pm 101.1	501.9 \pm 85.7	405.2 \pm 78.2	165.7 \pm 11.1	104.1 \pm 4.0	5.7 \pm 0.5	7.0 \pm 0.7	20.2 \pm 3.9
2	555.7 \pm 94.6	470.3 \pm 77.5	394.9 \pm 74.7	156.2 \pm 12.7	102.9 \pm 4.1	5.4 \pm 0.5	6.4 \pm 0.6	16.1 \pm 3.4
3	529.2 \pm 85.4	451.9 \pm 71.8	375.5 \pm 56.8	150.4 \pm 12.8	103.5 \pm 4.8	5.2 \pm 0.5	6.1 \pm 0.6	15.4 \pm 4.3
4	510.0 \pm 76.2	425.2 \pm 56.2	355.5 \pm 51.7	142.7 \pm 11.3	103.6 \pm 2.6	4.9 \pm 0.5	5.9 \pm 0.6	15.4 \pm 4.2
5	491.2 \pm 69.5	408.1 \pm 59.2	340.7 \pm 53.1	138.5 \pm 10.8	104.2 \pm 2.8	4.7 \pm 0.5	5.7 \pm 0.6	15.1 \pm 3.6
6	474.6 \pm 77.5	395.8 \pm 64.7	331.9 \pm 53.5	134.9 \pm 10.6	104.1 \pm 3.1	4.5 \pm 0.5	5.5 \pm 0.5	14.3 \pm 2.1
7	457.2 \pm 86.4	385.1 \pm 70.8	326.5 \pm 60.7	131.2 \pm 11.1	104.9 \pm 3.7	4.4 \pm 0.5	5.2 \pm 0.5	13.1 \pm 3.4
8	449.6 \pm 75.3	373.2 \pm 64.3	315.2 \pm 56.7	128.9 \pm 11.0	104.5 \pm 3.6	4.3 \pm 0.5	5.2 \pm 0.5	13.4 \pm 3.1
9	454.8 \pm 83.0	375.1 \pm 71.3	313.5 \pm 58.4	129.2 \pm 11.2	104.9 \pm 4.3	4.3 \pm 0.5	5.2 \pm 0.5	14.1 \pm 3.3
10	462.2 \pm 84.0	378.7 \pm 78.8	316.1 \pm 65.3	130.6 \pm 12.8	105.6 \pm 3.9	4.3 \pm 0.5	5.3 \pm 0.6	14.6 \pm 2.7
Sprints 1 & 10: % change	\downarrow 23.9	\downarrow 24.5	\downarrow 22.0	\downarrow 21.2	\uparrow 1.0	\downarrow 24.6	\downarrow 24.3	\downarrow 27.7

Table 2. Raw data for peak power, average power, minimum power, peak RPM, minimum RPM, anaerobic capacity, anaerobic power, and fatigue index recorded during each sprint in the supinated forearm position. All data shown as mean \pm SD. Percent changes and p-values represents changes and significance between sprints 1 and 10.

Measurements for each 10-second sprint in the supinated forearm position								
Sprint	Peak Power (W)	Avg. Power (W)	Min. Power (W)	Peak RPM	Min RPM	An. Capacity (mean W/kg)	An. Power (peak W/kg)	Fatigue Index (W/s)
1	568.4 \pm 84.7	477.8 \pm 76.0	410.1 \pm 81.7	161.2 \pm 13.8	104.3 \pm 7.7	5.5 \pm 0.6	6.6 \pm 0.8	15.8 \pm 4.4
2	534.5 \pm 78.8	457.4 \pm 74.9	398.5 \pm 72.5	154.5 \pm 12.7	104.8 \pm 6.1	5.3 \pm 0.5	6.2 \pm 0.8	13.6 \pm 3.5
3	508.2 \pm 70.1	434.3 \pm 58.2	380.0 \pm 55.9	147.8 \pm 12.8	104.7 \pm 4.5	5.0 \pm 0.5	5.9 \pm 0.6	12.8 \pm 2.8
4	507.6 \pm 75.4	419.9 \pm 57.2	365.9 \pm 56.1	143.7 \pm 10.8	107.0 \pm 2.4	4.9 \pm 0.5	5.9 \pm 0.4	14.2 \pm 3.7
5	466.7 \pm 52.6	395.4 \pm 49.7	350.5 \pm 50.5	136.2 \pm 12.1	104.3 \pm 2.5	4.6 \pm 0.5	5.4 \pm 0.5	11.6 \pm 2.3
6	467.9 \pm 66.6	388.5 \pm 51.7	339.4 \pm 48.3	134.2 \pm 9.9	104.8 \pm 3.1	4.5 \pm 0.5	5.4 \pm 0.5	12.9 \pm 3.3
7	452.5 \pm 67.2	381.0 \pm 50.2	330.7 \pm 42.0	132.2 \pm 10.8	104.1 \pm 3.8	4.4 \pm 0.5	5.2 \pm 0.5	12.2 \pm 3.6
8	451.9 \pm 71.8	368.9 \pm 46.7	311.9 \pm 37.4	129.2 \pm 10.3	104.2 \pm 4.6	4.3 \pm 0.5	5.2 \pm 0.5	14.0 \pm 6.0
9	448.0 \pm 61.8	369.2 \pm 53.3	315.5 \pm 49.1	128.8 \pm 9.0	105.9 \pm 3.2	4.3 \pm 0.4	5.2 \pm 0.5	13.3 \pm 2.9
10	446.5 \pm 88.8	367.2 \pm 50.8	318.1 \pm 44.6	128.7 \pm 11.3	103.5 \pm 4.0	4.3 \pm 0.5	5.2 \pm 0.7	12.9 \pm 3.6
Sprints 1 & 10: % change	\downarrow 21.4	\downarrow 23.1	\downarrow 22.4	\downarrow 20.2	\uparrow 1.0	\downarrow 21.8	\downarrow 21.2	\downarrow 18.4

Table 3. Power output measurements (averaged over sprint 1 to 10) to compare pronated and supinated forearm positions. All data shown as mean \pm SD and p-values represent significant main effect for forearm position.

Averages of power output measurements for sprints 1 and 10 in each forearm position								
	Peak Power (W)	Avg. Power (W)	Min. Power (W)	Peak RPM	Min RPM	An. Capacity (mean W/kg)	An. Power (peak W/kg)	Fatigue Index (W/s)
Pronation	499.2 \pm 83.3	416.5 \pm 70.5	347.5 \pm 60.9	140.8 \pm 11.6	104.2 \pm 3.7	4.8 \pm 0.5	5.8 \pm 0.6	15.2 \pm 3.6
Supination	485.2 \pm 69.6	410.3 \pm 56.9	352.1 \pm 48.9	139.6 \pm 11.3	105.0 \pm 4.2	4.7 \pm 0.5	5.6 \pm 0.6	13.3 \pm 3.6
<i>p</i> -values	<i>p</i> = .045	<i>p</i> = .239	<i>p</i> = .360	<i>p</i> = .486	<i>p</i> = .403	<i>p</i> = .481	<i>p</i> = .102	<i>p</i> = .001

3.8 LIST OF FIGURES

Fig 1. Experimental Setup. **(A)** Experimental set up for measuring biceps and triceps brachii maximum voluntary contraction (MVC), potentiated twitch (PT) force, and electromyography (EMG). **(B)** Experimental set up for the arm cycling sprints. **(C)** Timeline for experimental protocol. The light grey bars represent submaximal intensity cycling that each participant performed prior to maximal intensity arm cycling. The maximal intensity arm cycling sprints are represented by the dark grey bars and the passive rest periods are represented by the white bars. The arrows pointing downward indicate when MVC and PT were measured.

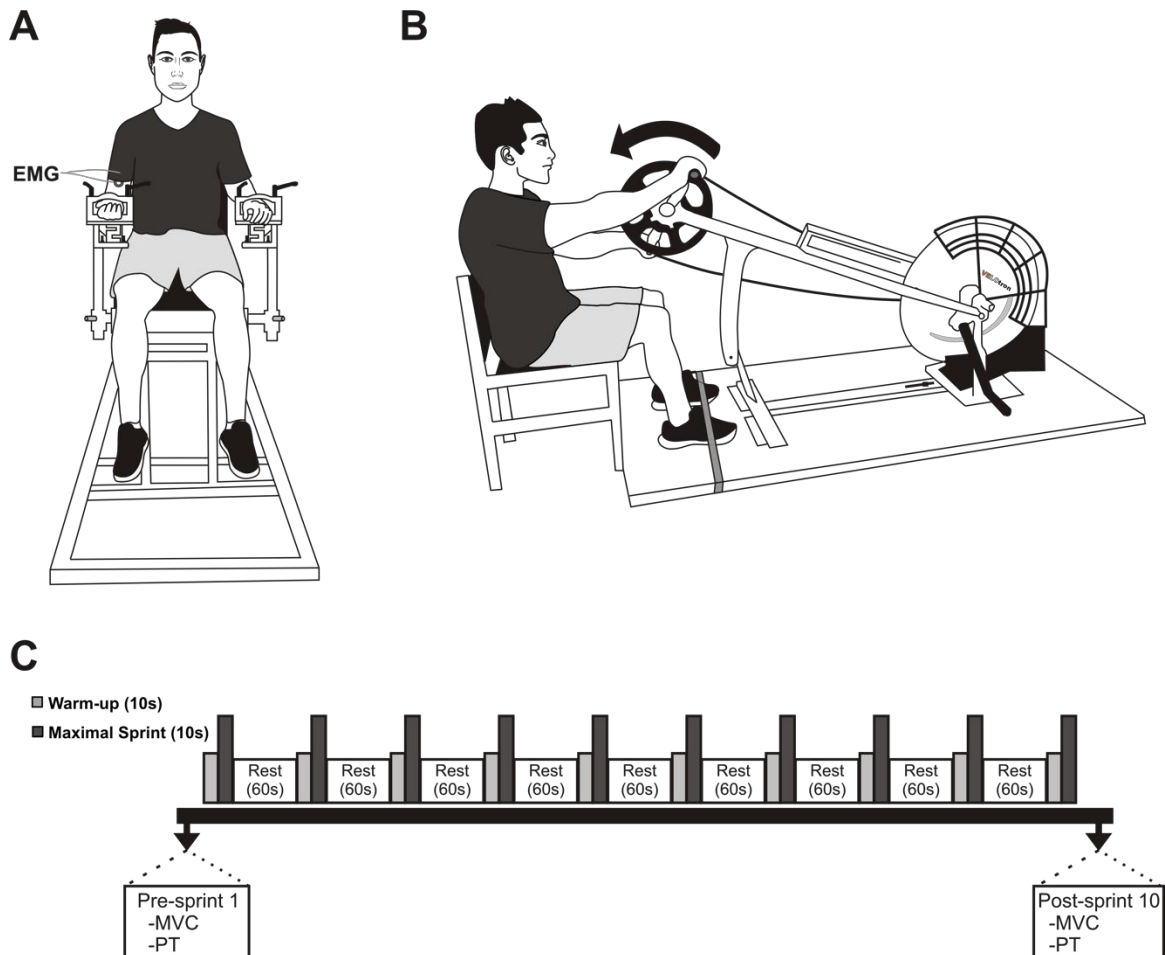


Fig 2. Comparison of peak power (mean \pm SE) for sprints 1 to 10 in pronated and supinated forearm positions. Inset represents position differences for overall peak power averages of sprints 1 to 10 for the pronated and supinated forearm positions. The asterisk represents significant ($p < .05$) time effect for sprints 1 and 10. The dagger represents significant ($p < 0.05$) differences between positions. Each point represents mean.

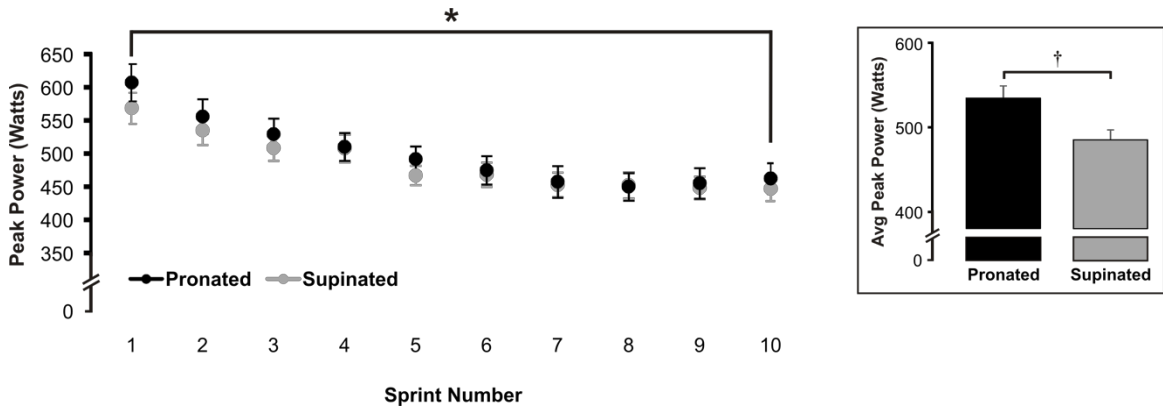


Fig 3. Comparison of (A) RPE and (B) perceived pain data for sprints 1 to 10 in pronated and supinated forearm positions. The asterisk represents significant ($p < .05$) time effect for sprints 1 to 10. Each point represents mean \pm SE.

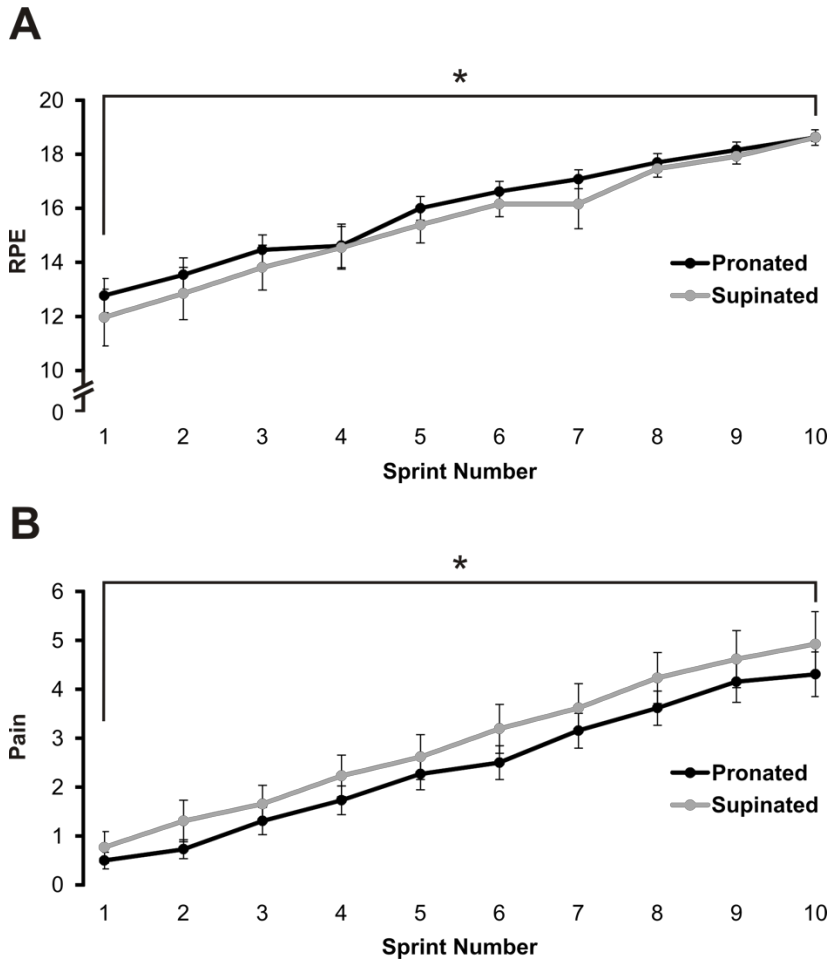


Fig 4. Comparison of (A) elbow flexor (B) and elbow extensor MVC force at pre-sprint 1 and post-sprint. The asterisk represents significant ($p < .05$) difference between pre- and post-sprint. All data shown as mean \pm SE.

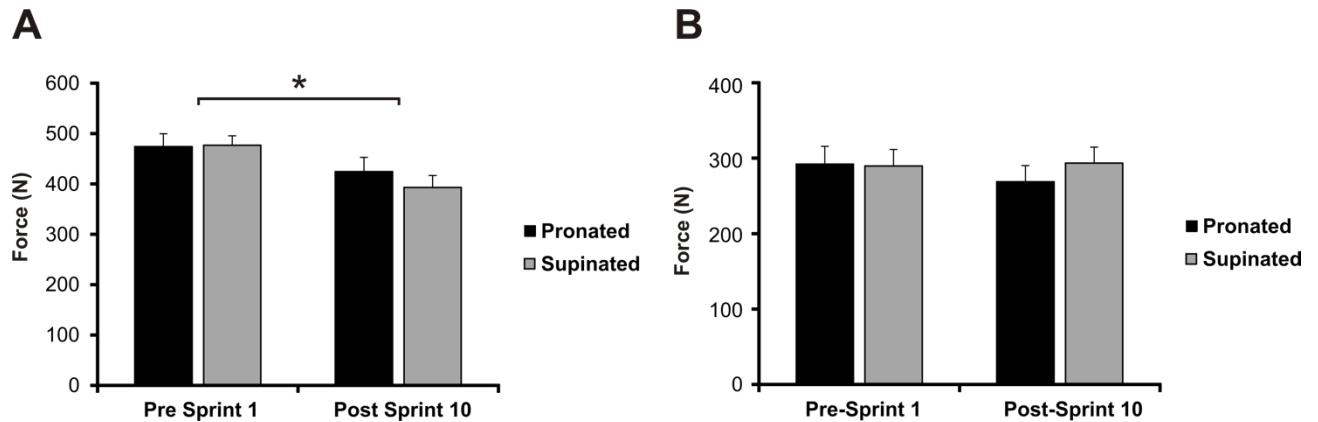
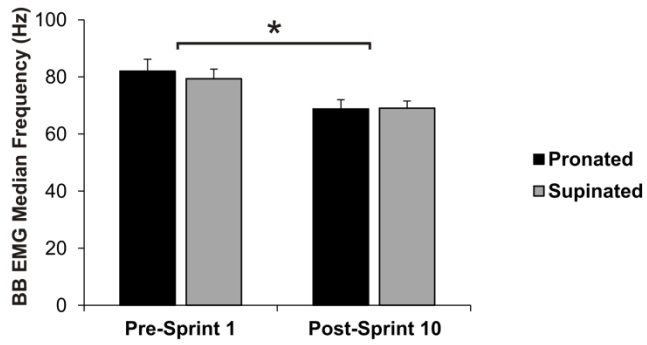
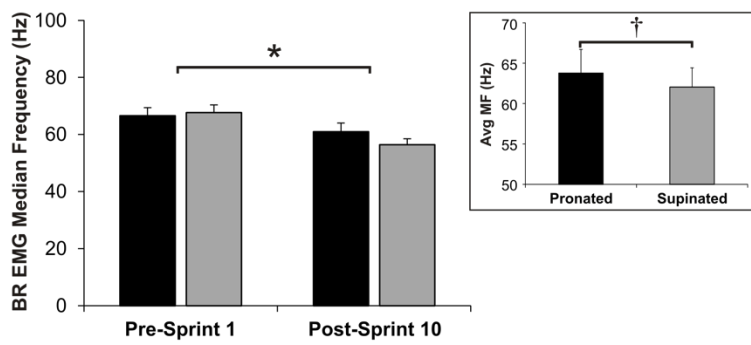


Fig 5. Comparisons of median frequencies at pre-sprint 1 and post-sprint 10 in pronated and supinated forearm positions for (A) biceps brachii (BB), (B) brachioradialis (BR), and (C) triceps brachii (TB). The asterisk represents significant ($p < .05$) time effect for pre and post-sprint. Insets for fig. 5B and C represent average median frequency (MF) for the pronated and supinated positions to demonstrate effect of position. The dagger represents significant ($p < .05$) differences between positions. All data shown as mean \pm SE.

A



B



C

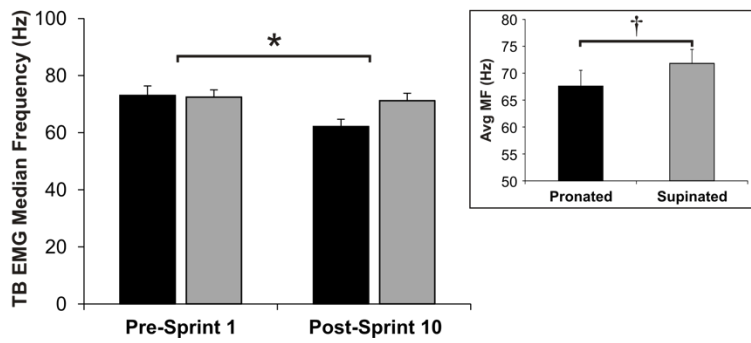
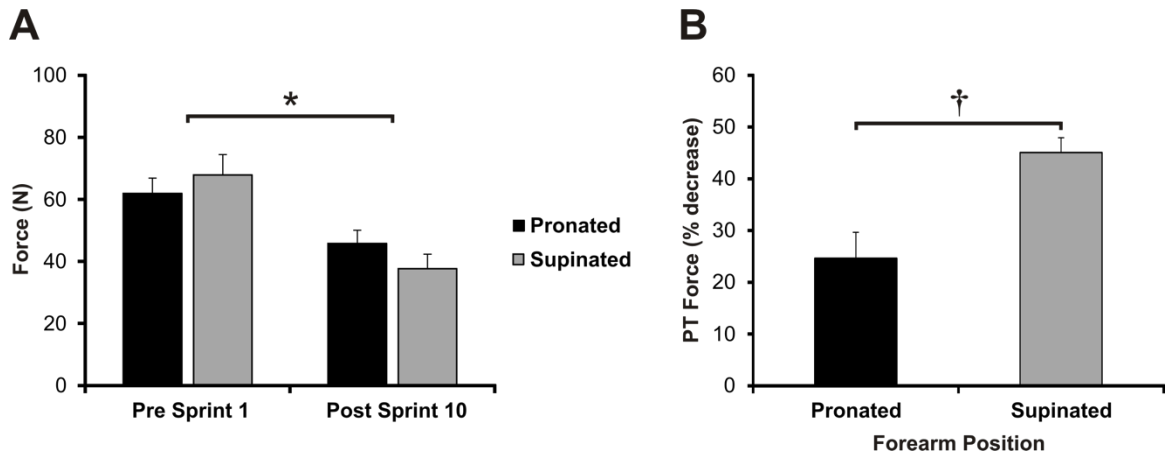


Fig 6. Comparison of potentiated twitch (PT) force at pre- and post-sprinting for pronated and supinated cycling. **(A)** Comparing pre-sprint 1 and post-sprint 10 for pronated and supinated arm cycling. The asterisk represents significant ($p < .05$) difference between pre- and post-sprint. **(B)** Comparing the percentage decrease in each forearm position where the boxes represent the percentage decrease from pre-sprint 1 to post-sprint 10 values for each forearm position. The dagger represents significant ($p < .05$) differences between positions for percentage decrease. All data shown as mean \pm SE. There was a greater percentage decrease ($p < .05$) in PT force following supinated sprinting compared to pronated sprinting as demonstrated by a greater reduction from pre-sprint 1 values.



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