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Rate Modulation of the Human Anconeus Muscle During High-Intensity Dynamic Fatigue of the Elbow Extensor Muscle Group

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Graduate Program in Kinesiology

A thesis submitted in partial fulfillment of the requirements for the degree in Master of Science

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RATE MODULATION OF THE HUMAN ANCONEUS MUSCLE DURING HIGH-INTENSITY DYNAMIC FATIGUE OF THE ELBOW EXTENSOR MUSCLE GROUP
(Thesis Format: Monograph)

by

Brianna Lynn Cowling

Graduate Program in Kinesiology

A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science

The School of Graduate and Postdoctoral Studies
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Abstract

**PURPOSE:** To evaluate anconeus motor unit firing rate (MUFR) as a function of time to task failure (TTF) during maximal velocity elbow extensions at a moderately heavy load.

**METHODS:** Two fine-wire intramuscular electrode pairs were inserted into the anconeus to record MUFR in twelve male participants (25±3y). Individual MUs were tracked throughout a three-stage dynamic elbow extension fatigue protocol. Mean MUFR were calculated for the following time ranges: 0-15%, 45-60%, and 85-100% TTF. **RESULTS:** Following the fatigue task, with a mean TTF of 83s, peak power decreased 64% compared to baseline. Data from 20 anconeus MUs showed changes in MUFR from ~36 Hz (0-15% TTF) to ~28 Hz (45-60% TTF) to ~23 Hz (85-100% TTF). **CONCLUSION:** During high-intensity maximal velocity dynamic contractions, anconeus firing rates decreased substantially. The relative decrease in MUFR after this task is in accordance with that reported for sustained high-intensity isometric tasks in other muscles.

**Keywords**

Motor neuron, Discharge rate, Concentric, Velocity-dependent contraction, Rate coding
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“Beyond a wholesome discipline, be gentle with yourself.”

Max Ehrmann, “Desiderata”
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<td>ANOVA</td>
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<td>EC</td>
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<td>potentiated twitch</td>
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<td>TTF</td>
<td>time to task failure</td>
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<td>Tw&lt;sub&gt;max&lt;/sub&gt;</td>
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<td>maximal velocity at a given load (x)</td>
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Glossary of Terms

**Concentric contraction** - A muscle contraction in which muscle length shortens with a change in joint angle.

**Isometric contraction** - A static muscular contraction whereby there is no change in joint angle or muscle length.

**Motor unit (MU)** – A single motor neuron and all the muscle fibres it innervates.

**Motor unit firing rate (MUFR)** - The number of single motor unit action potentials that are fired per second; measured in Hertz (Hz).

**Neuromuscular fatigue** - An exercise-induced reduction in torque or power production, regardless of whether the task can be successfully performed.

**Neuromuscular junction** - The point of interaction between the motor end plate of the muscle and the terminal axon of the motor neuron.

**Power** - The product of torque and radial velocity, expressed in Watts (W).

**Time to task failure** - The time from when a participant begins a task to the time when they can no longer complete the task adequately, as defined by the experimenter.

**Torque** - Also referred to as “moment”. Torque is the product of the length of the moment arm, the magnitude of the force vector, and the sine of the angle between the force vector and the moment vector. It is expressed in Newton•metres (Nm).

**Voluntary activation** – Ability of the central nervous system to maximally activate muscle.

**Velocity-dependent contractions** - Movement through a range of motion where the imposed load remains constant and velocity is free to vary.
Chapter 1

1 Literature Review

1.1 General Introduction

The intention to move originates in the central nervous system, specifically in premotor centres of the brain. Through various neuronal connections and pathways, an electrical signal (action potentials) travels from the brain to the spinal cord and activates motor neurons who send a signal to the muscle or muscle group that is involved with the intended movement. When the signal crosses the neuromuscular junction and is received by the muscle, it is converted into tension by muscle contraction through a series of electrochemical steps. For angular joint movement to occur (i.e.: a dynamic shortening, or concentric, contraction), the muscle must produce enough force to overcome a load. The rate at which force is produced over a distance (range of motion) is the resultant velocity and is used to determine the power of the dynamic contraction. Mechanical output can be altered by physiological status such as training, disease, or acute neuromuscular fatigue. These states will affect neuromodulation at various points along the motor pathway from the brain to the muscle. This thesis will focus on neuromodulation at the spinal level related to maximal dynamic neuromuscular fatigue.

Previous investigations have given considerable insight into neuromodulatory mechanisms, but mainly in isolated animal preparations or during isometric (no muscle shortening) or slow, lightly loaded dynamic contractions in human models. However, it is valuable to explore these mechanisms during dynamic movements at a whole systems level in vivo, and the human model offers some advantages for these studies.

1.2 Neuromuscular Transmission

Voluntary movement is possible due to the interaction between the nervous and muscular systems (Figure 1). The intent to move is initiated in premotor centres of the brain and occurs approximately 100 milliseconds before muscle activation (1). Signals from the central nervous system are sent via the corticospinal tract to alpha motor neurons (α-mn) in the spinal cord. The α-mn is considered the final common pathway of neuromuscular transmission (2). At this point, inhibitory and excitatory neuromodulatory inputs of varying
strengths from various origins converge and influence the final signal sent to the muscle (3–5). Electrical signals are transmitted via axons of one neuron and are received via dendritic processes of another. Potentials of varying amplitudes arrive at the dendrite and are summated in the cell body. If the sum of the inputs results in depolarization of ~20-30 mV from the resting potential (~ -70 mV) to ~ -50 mV, the threshold for action potential initiation is reached. Action potential generation occurs at the base of the axon (axon hillock) and is propagated in an “all-or-none” response along the axon with invariant amplitude. At this point, the frequency of electrical impulses (firing rate) is what dictates the strength of the signal and ultimately affects muscle force gradation.

Figure 1. Simplified schematic of the motor pathway
Axons from the α-mn extend to the muscle, forming a chemical synapse (Figure 2). A motor unit (MU) includes a motor nerve and the muscle fibres it innervates. MUs vary in size and number depending on the muscle under investigation (6–8). Nerve-muscle connections are concentrated in areas termed motor points, which are located approximately mid-length along the muscle. The number of motor points varies depending on the muscle (9). The neuromuscular junction is the communication point between muscle and nerve. If the signal is robust at this point and is received by the muscle, the intent to move is translated by excitation-contraction (E-C) coupling processes into actin and myosin sliding filament interactions resulting in muscle force generation. The final action is muscle shortening, which, by attachments to bony levers across a joint, causes angular joint rotation and limb movement. In detail, when an electrical impulse in the form of an action potential reaches the terminal axon, acetylcholine is released from vesicles at the presynaptic terminal and diffuses across the ~30 nm space between the nerve and muscle (synaptic cleft). Acetylcholine then binds to postsynaptic receptors on the motor end plate of the muscle, resulting in the opening of ligand-gated ion channels that permit the entry of cations. Cation influx results in depolarization of muscle cell. If the threshold for action potential generation is reached, the motor end plate potential is propagated in both directions along the surface membrane and into the T-tubules of the muscle. This electrical activity begins the process of E-C coupling by triggering the release of calcium from the sarcoplasmic reticulum. The released calcium binds to troponin on the actin filament. This exposes the cross-bridge binding sites on actin and allows it to connect to myosin. The sliding filament action of the actin-myosin complex (cross-bridge cycling) results in sarcomere shortening and production of muscle force (10).
1.3 Motor Unit Control Strategies

Human movement is rarely accomplished through activation of a single MU. Voluntary motor control is typically accomplished through the coordinated activity and modulation of multiple MUs (motor neuron pools) within a muscle, as well as groups of muscles. To successfully complete a task, the neuromuscular system uses recruitment and rate coding to gauge mechanical output. In 1929, Adrian and Bronk (11) described these two primary mechanisms of force gradation. MU recruitment involves increasing the number of active MUs, whereas MU rate coding involves increasing the rate at which the motor neuron transmits impulses to the muscle. Although up-regulation of both mechanisms occurs during increasing levels of muscle force, the recruitment process is often completed before...
maximum output is achieved and rate coding completes the attainment of maximal force output from a muscle. Recruitment order of MUs is determined by the size of the motor neuron according to Henneman’s size principle, which states that smaller motor neurons are activated before larger motor neurons (12). Rate coding works by increasing the frequency of action potential trains to the muscle and the response is based on the inherent contractile quality (speed) of the muscle. If low-frequency stimulation is delivered (sub-tetanic or unfused tetanic evoked contractions), the mechanical response is unfused, because twitch overlap is not optimal. When optimal summation occurs, force increments are smoothed and a fused tetanic contraction is produced. The optimal overlap of the twitch responses allows controlled, fluid movements.

Considerably, understanding of the organization of motor neurons, their behaviour, and anatomical features of a MU has been gained from reduced preparations as well as in human designs (13), and mainly in isometric contractions. Technical limitations related to recording MU activity with changes in muscle length have prevented researchers from recording single MU firing rates (MUFRs) during fast, dynamic contractions (14). Therefore, the majority of investigations of MUFR involve isometric or slow dynamic contractions, which limits the application of the findings.

1.4 Electromyography

Electromyography (EMG) is used to record electrical muscle activity. EMG signals can be affected by anatomical factors such as the thickness of subcutaneous tissue (for surface EMG), tissue inhomogeneities, and size of a MU territory (15). Characteristics of the detection system such as interelectrode distance as well as electrode size and placement can influence EMG signals (15). Large and rapid changes in fascicle length can cause electrode displacement, which constitutes a geometrical factor that negatively influences the quality of both indwelling and surface electrode EMG signals (15). This factor is a concern in the investigation of MUFR changes related to dynamic neuromuscular fatigue because the active muscle tissue may move in relation to the skin. Skin displacement relative to underlying muscle tissue can be minimized somewhat by using indwelling fine wire electrodes, but in past studies, the amount of muscle shortening in relation to overlying fascia and speed of movement needed to be kept to a minimum to achieve suitable discriminate MUFR
recordings. There are significant changes in fascicle length during dynamic contractions that make it difficult to track trains of single MUs.

Surface EMG is a non-invasive technique that assesses global activity over the skin’s surface, whereas single MU activity can be recorded with intramuscular EMG using concentric, monopolar needles or fire wire electrodes. Intramuscular recordings provide a distinct advantage because thickness of the subcutaneous layer does not distort the signal. A concentric needle is widely used clinically because it provides a low signal-to-noise ratio and precise recordings, but is limited to use during low force, static contractions (14). With increasing muscle force, electrode movement results in a change in the recording surface and subsequent variability in recordings. Fine wire EMG collection involves inserting pairs of wire electrodes into the muscle using a hypodermic needle. The amount of exposed ( uninsulated) area dictates the specificity of the recording area that will be acquired. Precise, single MU EMG recordings can be collected using a small (<1mm) exposed area. The tips of the wires are “hooked” into the muscle, allowing higher force isometric, slow dynamic, and repetitive contractions with this technique. Recently, it has been shown that single MU recordings can be obtained during maximal velocity dynamic contractions using a unique muscle model (16,17).

1.5 Elbow Extensor Muscle Group

The elbow extensor muscle group includes the three heads of the triceps brachii (medial, long, and lateral) and the anconeus. This muscle group is innervated by the radial nerve, but each component has distinct characteristics in terms of fibre composition, force profile, and architecture (18–21). The triceps brachii is a fusiform muscle largely involved with elbow extension. The long head originates on the infraglenoid tubercle of the scapula and plays a role in resisting dislocation of the shoulder, particularly during adduction. The lateral head originates on the posterior surface of the humerus, superior to the radial groove and the medial head originates on the posterior surface of the humerus, inferior to the radial groove. All three heads of the triceps brachii insert on the proximal end of the olecranon of the ulna and fascia of the forearm. The anconeus is a small, triangular muscle that originates on the lateral epicondyle of the humerus and inserts on to the lateral surface of the olecranon and superior part of the posterior surface of the ulna. The anconeus assists the triceps in
extending the forearm and also stabilizes the elbow joint (22). It may also play a role in adduction of the ulna during pronation (22). The anconeus is primarily a slow twitch (Type I) muscle (19), whereas the triceps brachii has a relatively greater proportion of fast twitch (Type II) muscle fibres, making it a faster contracting muscle. It has been reported that the triceps brachii muscle group has a higher twitch amplitude and faster contraction time and half-relaxation time compared to the anconeus (19). However, the force exerted by the anconeus to produce an elbow extension is not negligible, as it contributes up to ~15% of the total elbow extension force (23). MUFR increases steadily for the triceps brachii as % maximum voluntary isometric contraction (MVC) is increased. Unlike the triceps, there is evidence that the anconeus is fully recruited at low force levels (<30% MVC) (19).

Results from investigations of fast isometric (ballistic) contractions have demonstrated lower recruitment thresholds and higher MUFRs compared with slow isometric contractions (23–29). Although these results are useful to gain insight into the changes in neuromuscular communication during movement, there are fundamental differences in cortical excitability and MU behaviour in dynamic compared to isometric contractions (30,31). To produce a maximal velocity contraction, higher synaptic input is likely required compared to slow dynamic or isometric contractions. To maximize performance, in terms of speed of movement, it is important to utilize the higher limit of MUFR and lower limit of recruitment threshold. Due to the technical difficulties associated with recording and following single MU action potentials throughout a fast dynamic protocol, the anconeus model has proved useful to explore changes in MUFR with maximal velocity dynamic contractions to the point of task failure.

1.6 The Anconeus Muscle

Single MU recordings can be reliably detected in the anconeus during movement due to its distinct anatomical features, making it an attractive model for the study of neuromuscular properties during dynamic fatigue. Clear intramuscular EMG recordings are possible because the anconeus is a small muscle with relatively few (~25 to ~60) MUs (7). Additionally, the anconeus experiences small absolute, but similar relative changes in fascicle length during a dynamic movement, compared to larger muscles (7). Previous investigations revealed that the anconeus experiences less shortening per unit of displacement compared to the heads of
the triceps brachii (20,21). Additionally, it has been shown that the anconeus is active in elbow extension at all angles, contraction intensities, and elbow extension velocities (18,32,33). This muscle has recently been used to assess MUFR during submaximal and maximal dynamic contractions of the elbow extensors over the course of a submaximal dynamic fatigue protocol of moderate intensity (40% MVC load at 60% of maximal velocity) to velocity failure (17). Following this protocol, MUFR decreased ~20% for maximal dynamic contractions; however, no change in MUFR was observed for submaximal dynamic contractions.

1.7 Neuromuscular Fatigue

Fatigue is a commonly used term that can result in a variety of psychological, physiological, or mechanical manifestations. For the scope of this thesis, neuromuscular fatigue is a more appropriate term to describe the exercise-associated changes that occur as a result of fatigue at any point along the motor pathway from the level of the brain to the muscle. This physiological process begins when exercise commences and develops progressively until volitional termination of the task or at the point of task failure. The neuromuscular system uses various strategies to adapt to the demands placed on it and to delay exhaustion. Neuromuscular fatigue is expressed as a decreased ability to generate maximal torque/power or an increased effort to maintain a submaximal level of mechanical output, regardless of whether the task can be performed successfully (34). The decrease in mechanical output that is characteristic of prolonged or repeated intermittent exercise can occur due to changes at several points along the motor pathway. Neuromuscular fatigue can result from a decrease in the ability of the central nervous system to activate motor neurons (central fatigue) or a failure of the muscle contractile apparatus (peripheral fatigue). The effects and origin of neuromuscular fatigue depend on a variety of factors associated with the parameters of the task (35), or the characteristics of the subjects under study (36). Various techniques have been used to gain insight into the changes that occur when the neuromuscular system is stressed by continuous or repetitive activation.

1.7.1 Techniques Used to Assess Neuromuscular Fatigue

Neuromuscular fatigue has been widely investigated during static contractions (36–41) and, much less comprehensively, during whole body exercise or dynamic movements (42–45).
Therefore much less is known about MU behaviour in response to dynamic fatigue due to technical challenges associated with recording techniques. The vast majority of MUFR studies in animals and humans have provided insight on fatigue processes gained from isometric or static contractions because single MUs can be tracked with relative ease throughout the protocol.

The interpolated twitch technique (ITT) is a method that is extensively used in neuromuscular research to quantify levels of voluntary activation, a term that describes the ability to fully activate skeletal muscle in a voluntary contraction. A supramaximal electrical stimulus is delivered to either the muscle or the peripheral nerve supplying the agonist muscle during the plateau phase of a maximal effort isometric contraction. If there is no further increase in torque when the stimulus is delivered, all available MUs are considered to be optimally activated. If there is an additional increase in force above the maximal force contraction (superimposed twitch), this is an indication that not all MUs have been recruited or activated completely. A stimulus is also delivered following the maximal contraction, evoking a mechanical response (potentiated twitch) at rest. The percentage voluntary activation (%VA) can be determined by expressing the amplitude of the superimposed twitch as a function of the amplitude of the potentiated twitch. A decrease in voluntary activation infers that there is a decrease in the ability of the central nervous system to maximally activate skeletal muscle, which has been reported during prolonged exhaustive exercise (44–48).

The neuromuscular system can adapt to perturbations, such as fatigue, to protect the body from damage and to maintain the desired level of force. With repetitive, high-intensity fatiguing contractions, twitch contraction time and maximum rate of tension development slow and twitch amplitude decreases as a function of biochemical changes that occur along the motor pathway (49). Changes in concentration of certain metabolic by-products (calcium, hydrogen ions, inorganic phosphate, reactive nitrogen and oxygen species) can influence the binding states of actin and myosin contractile proteins, thereby decreasing force-generating capacity of cross-bridges. When twitch contraction time is slowed, a lower frequency of stimulation can be sufficient to maintain maximal force. MUFR slows to match these changes in twitch characteristics and optimize twitch summation. Decreased MUFRs have been widely reported following sustained or repeated maximal fatigue protocols (36,37,39).
Following submaximal isometric fatigue protocols, select MUs showed no change (37,50–53) and others showed decreases in firing rate (54–56). Additionally, the few investigations that employed slow and lightly loaded dynamic fatigue protocols revealed that MUFR during submaximal contractions changed variably (42,57). However, due to technical limitations associated with tracking and discriminating single MUs, there is little known regarding the changes in MUFR following high-intensity, maximal-velocity dynamic fatigue.

1.8 Purpose and Hypotheses

This thesis will contribute to the understanding of MU control strategies by employing a maximal velocity dynamic fatigue protocol. The purpose is to exploit the unique anatomical and physiological features of the anconeus to assess MUFR of the anconeus and mechanical output of the whole elbow extensor group as a function of time to task failure (TTF) during a maximal velocity elbow extension protocol using a moderately heavy load. It has been shown that MUFRs are higher in dynamic contractions compared to isometric contractions (16,28,29,58). High MUFR allows for enhanced MU twitch tension summation that is required for high rates of force develop and to produce maximal dynamic contractions. It was hypothesized that MUFR would decrease over the course of the fatigue protocol and to a greater extent than previous results reported for maximal isometric and submaximal dynamic fatigue protocols.
Chapter 2

2 Introduction

When high-intensity exercise is performed, fatigued-related changes occur along the motor pathway from the level of the brain to the muscle. The physiological process of neuromuscular fatigue begins when exercise commences and develops progressively until volitional termination of the task or at the point of task failure. Neuromuscular fatigue is expressed as a decreased ability to generate maximal torque/power or an increased effort to maintain a submaximal level of mechanical output, regardless of whether the task can be performed successfully (34). Manifestation of neuromuscular fatigue varies depending on a variety of factors including the muscle under investigation (59,60), the task being performed (maximal or submaximal, dynamic or static) (61), age (41,59,62) and sex (63) of the participants, and the method used to assess fatigue (64). It is therefore important to consider these variables when designing a protocol to assess MU behaviour. Compared with isometric contractions, there may be fundamental differences in dynamic contractions that can influence the neuromuscular fatigue process, such as rate of energy expenditure, degree of afferent inhibition, or metabolite accumulation (64). Additionally, the production of a maximal velocity contraction likely requires higher synaptic input compared to slow dynamic or isometric contractions. To maximize performance, in terms of speed of movement, it is important to utilize higher MUFRs with lower recruitment thresholds.

It has been widely reported that following high-intensity isometric fatiguing protocols MUFRs are decreased (36,37,39,56). Protocols that consisted of submaximal and periodic maximal contractions found that MUFR declined by ~30% for maximal contractions, but no change was found for submaximal contractions or there were slight increases (40,65). For dynamic shortening contractions, due to technical limitations associated with tracking and discriminating single MUs, there is little known regarding the changes in MUFR following high-intensity, maximal-velocity dynamic fatigue.

Active muscle shortening and whole muscle architectural changes can affect the muscle-electrode interface during dynamic contractions. Repeated contractions necessary to induce substantial fatigue compound the challenges associated with single MU recordings due to
changes in both force and velocity. These characteristics make single MU recordings difficult during contractions when muscle length is changing. In an attempt to minimize these movement-related challenges, investigators have constrained the velocity by employing isokinetic contractions usually of slower velocities (~50°/s), lighter loads (~20% MVC), or have curtailed joint range of motion to limit muscle length changes (42,57). At these submaximal dynamic levels, MUFR changed variably among recorded units (42,57). A dynamically fatigued neuromuscular system may use different MU control strategies compared to one that is fatigued isometrically, because there are additional variables that impact a voluntary contraction through a range of motion (64). Although some improvement is gained over isometric contractions, most dynamic paradigms used to date are somewhat artificial in relation to natural shortening contractions.

Because of its distinct anatomical features, the anconeus is an attractive muscle for the study of neuromuscular properties during dynamic fatigue. Clear intramuscular EMG recordings are possible in this muscle because the anconeus is a small muscle with few MUs and experiences small relative changes in fascicle length during a dynamic movement (7,66). This model has recently been used to assess MUFRs over the course of a submaximal dynamic fatigue protocol (17). Results showed that submaximal dynamic elbow extensions to fatigue did not result in a change in MUFR for the submaximal dynamic contractions per se, but MUFR decreased ~20% for maximal dynamic contractions during the protocol (17).

In addition to decreases in MUFR, isometric twitch contractile properties and measures of mechanical function have been shown to change with moderate to high intensity fatigue. Following fatigue evoked by high intensity isometric fatigue, decreased peak twitch (P\text{t}) and increased relaxation time are observed (34). With regard to dynamic fatigue, isometric MVC torque has been shown to decline substantially (~30%) with low (42,43,57) and moderate (17) intensity dynamic tasks. Interestingly, greater relative declines in peak velocity and peak power compared to MVC torque were demonstrated following a moderate dynamic fatigue task of the elbow extensors (17); emphasizing the task-dependent nature of fatigue.

This thesis will further the understanding of MU control strategies by employing a maximal velocity dynamic fatigue protocol. In this study, “dynamic contraction” refers to contractions in which the load remains constant, but the velocity is free to vary throughout the range of
motion (velocity-dependent contraction). Combining a technique to record electrical activity intramuscularly and with a unique muscle model allowed insight into neuromodulation in vivo with mechanical parameters using a task similar to habitual human movement. The purpose was to evaluate anconeus MUFR as a function of TTF during a maximal velocity elbow extension protocol using a moderately heavy load. An additional purpose was to determine whether maximal voluntary contraction, maximal velocity, peak power, and peak twitch torque of the elbow extensors would be altered and to what extent following maximal dynamic fatigue of this muscle group. It was hypothesized that anconeus MUFR would decrease as elbow extensor fatigue progressed, assessed by depressed mechanical function, to the point of task failure. It was expected that these changes would be greater than those previously reported for isometric and submaximal dynamic fatigue protocols.
Chapter 3

3 Methods

3.1 Participants

Twelve healthy young men (25±3 y) who self-reported to be recreationally active, but not systematically trained were recruited for this investigation and completed the protocols outlined below. Participants reported to be free of any known orthopaedic, neuromuscular, or cardiovascular limitations relevant to this protocol. The procedures were approved by the Research Ethics Committee for human subjects at The University of Western Ontario. Participants provided written and verbal informed consent prior to beginning the protocol.

3.2 Experimental Setup

Participants were seated in the Cybex Humac Norm (CSMi Medical Solutions, Stoughton, MA; research toolkit software) apparatus in an upright position with the hip flexed ~90°, left shoulder abducted ~70°, and the forearm secured in a pronated position (Figure 3). The medial surface of the left hand rested against a cushioned metal bar. The hand was secured to the manipulandum with athletic tape. A large Velcro strap was placed horizontally across the chest to secure the participant to the seat. Participants were provided with real-time visual feedback of their position, torque, and velocity on a computer monitor ~1 metre away. Elbow extension (EE) was performed in the transverse plane. During isometric contractions, the position of the lever arm was secured at 90° (neutral) EE. During dynamic contractions, participants were instructed to move a specified load through an 80° range of motion (70° to 150° EE) with a passive return speed of 90°/s from the end position (150° EE) to the start position (70°).
Figure 3. An illustration of the experimental setup.

Elbow extension position, torque, and angular velocity were recorded from the dynamometer and sampled at 100 Hz using a 12-bit analog-to-digital converter (Power 1401; Cambridge Electronic Design, Cambridge, UK) and digitized online using Spike2 (Version 7.0, CED, Cambridge, UK). Two channels of single MU recordings were obtained from the anconeus (each at a sampling rate of 12 500 Hz). Intramuscular EMG of the anconeus was pre-amplified (100-1000x, Neurolog, Welwyn City, England). The signal was band-pass filtered between 10 Hz – 10 kHz. One to three sessions of the protocol on separate visits were necessary (~ 1hr/visit) to obtain an adequate quantity and quality of MUs over the duration of the fatigue protocol. On average, the success rate of the protocol was 30%, meaning that in 30% of sessions, at least one MU was tracked over the course of the protocol.
Doublet twitches (200µs pulse width) were evoked using a stimulator (DS7AH; Digitimer, Ltd., Welwyn Garden City, Hertfordshire, UK) and two custom-made aluminum foil, gel-coated stimulation electrodes. The stimulation electrodes were placed transversely over the muscle bellies of the triceps brachii. The anode (~5 x 6 cm) was placed ~10 cm proximal to the olecranon process of the ulna and the cathode (~5 x 12 cm) was placed ~10 cm distal to the axilla. With the elbow joint angle at 90°, current intensity was increased until twitch torque amplitude showed no further increase. The stimulator intensity was increased an additional 15% to ensure that supramaximal stimulation (115% Tw\text{max}) was delivered.

Two pairs of fine wire (California Fine Wire Company, Grover Beach, CA; 100 µm), hooked-tip needle electrodes were inserted into the belly of the anconeus using a hypodermic needle (Becton Dickinson, Franklin Lanes, NJ; 25G x 5/8). Approximately 1 mm of the insulation was removed from the hooked portion of the needle electrode to create a small recording surface for detection of individual MU action potentials. The common ground was placed over the acromion. In all sessions, after the wires were inserted, the participants were asked to repetitively move a light load through the range of motion to secure the wires into the muscle and to assess signal fidelity.

### 3.3 Baseline Measures

Participants were given ~ 3 minutes of rest before completing 2 to 3 maximum voluntary isometric contractions (MVC) of the elbow extensors at an elbow angle of 90°. For the MVCs, subjects were instructed to produce force quickly and maintain that level of force for ~ 3-5 seconds. ITT was employed to provide a measure of total EE %VA. This technique involved delivering a supramaximal stimulus (115% Tw\text{max}) to the triceps brachii ~ 1 second before (resting), during the maximum torque (superimposed (SIT)) and ~ 1 second after (potentiated (POT)) maximal torque production. Participants were given ~ 3 minutes rest between MVCs. When force did not increase more than 5% in subsequent trials, the highest torque level was taken as the participant’s MVC and was used to determine submaximal loads for the remainder of the protocol. For familiarization, and to determine un-fatigued baseline measures, a series of 3-4 maximum velocity dynamic contractions at 35% isometric MVC load (V\text{max35}) were performed as a baseline measure of dynamic performance. For all
efforts, subjects were instructed to contract as hard and as fast as possible with strong verbal encouragement and visual feedback of their performance was provided.

One MVC with twitch interpolation, and maximum dynamic velocity at 35% MVC load were assessed at 0, 2, 5, and 10 minutes post fatigue. Peak twitch torque was assessed at 0, 5, and 10 minutes post fatigue.

3.4 Fatigue Protocol

A schematic depiction of the fatigue protocol is shown in Figure 4. After a brief (2-3 minute) rest following baseline measures, participants completed a three-stage dynamic elbow extension fatigue protocol, whereby they were asked to move different percentage of MVC loads through an 80-degree range of motion (70°-150°EE). The resistance was adjusted during the fatigue protocol from 45% to 35% to 25% of MVC of the elbow extensors when 50% of the initial velocity of that stage was unattainable. Thus, when the velocity was reduced by 50% with the starting load of 45% MVC the load was reduced to 35% MVC and again to 25% MVC when that velocity declined by 50%. The task was terminated when the velocity at 25% of MVC load decreased by 50% or there was range of motion failure. The purpose was to induce substantial fatigue extending for more than 1 minute (to mimic isometric protocols at high loads) and to minimize range of motion failure while maintaining a moderate velocity. If the initial load of 45% MVC was maintained, task failure would have occurred within ~ 35 seconds. Participants were instructed to perform the contractions at maximal velocity and were given strong verbal encouragement.
Figure 4. A diagrammatic representation of the experimental protocol. Raw torque, velocity and position tracings are shown for each of the tasks. Position is described as degrees from the start position (0 represents 70° elbow extension and 80 represents 150° elbow extension). (A) represents an isometric MVC. (B) represents maximal velocity dynamic contractions at 35% MVC. *The post fatigue measures were repeated at 0, 2, 5, and 10 minutes after the fatigue protocol.

3.5 Data Analysis

All data analyses were performed offline using custom software (Spike 2 v 7.0, CED, Cambridge, UK) that facilitates the analysis of peak MVC torque, peak velocity, VA, and twitch contractile properties. Intramuscular EMG signals were high-pass filtered offline at 100 Hz to remove any remaining movement artifact and to facilitate analysis.

Analysis of single MUFRs was performed using a template-matching algorithm (Spike2 v 7.0, CED, Cambridge, UK) that overlays sequential action potentials to identify action potentials of the same MU using temporal and spatial characteristics. Visual inspection was the final deciding factor as to whether an action potential belonged to a MU. Trains of MUs were included in the analysis if they consisted of at least 5 consecutive action potentials (39) and if the same MU fired in at least half of the contractions from each of the time ranges.
when measurements were taken. Additionally, to be accepted as a MU train, the firing rate variability, assessed as the coefficient of variation of interspike intervals, had to be <30% (67). Long interspike intervals (ISI (>150ms) and doublet discharges (ISI <10ms) were excluded from the analysis. Mean MUFRs were calculated for the following three relative time ranges: 0-15% TTF (beginning), 45-60% TTF (middle) and 85-100% TTF (end) (Figure 5).

Peak power was calculated as the maximal product of torque (Nm) and shortening velocity (rad/s) for each contraction. Average peak power was determined for contractions in each of three time bins (0-15%, 45-60%, and 85-100% TTF).

Peak EE power, MVC torque, and velocity at 35% load, and %VA were determined for each subject at baseline, 0, 2, 5, and 10 minutes post-fatigue (pre, post0, post2, post5, post10). %VA was calculated using the following formula: [(1-(SIT/POT))*100]. The average duration of the fatigue protocol was also determined. PT was assessed at baseline and at 0, 5, and 10 minutes post fatigue.
Figure 5. Representative sample data from one participant. From top to bottom, MUFR, Sorted MUAP, and anconeus fine-wire raw EMG are shown from three time points: 0-15%, 45-60%, and 85-100% TTF. A, B, and C are representative of the MUAP shape at each of the three time points.

3.6 Statistical Analysis

Statistical analyses were performed using SPSS 17.0 (IBM, Armonk, NY). Two separate, single-factor repeated measures analysis of variance (ANOVA) tests were performed to assess changes in MUFR and peak power as the fatigue protocol progressed from 0-15% TTF to 45-60% TTF to 85-100% TTF. Repeated contrasts were performed as a post-hoc analysis of a significant omnibus effect. A single-factor, repeated measures ANOVA was performed to assess changes in peak twitch torque at 4 time points (pre, post0, post5, post10). Pairwise comparisons were performed as a post-hoc analysis of a significant omnibus effect. A Dunn-Sidak adjustment was used to correct for multiple comparisons. A repeated measures multivariate analysis of variance (MANOVA) was conducted to assess multivariate effects of peak power, $V_{\text{max35}}$, and MVC at 5 time points (pre, post0, post2, post5, post10). Univariate effects were interpreted (68) and simple contrasts were performed as a post-hoc analysis of a
significant omnibus effect. For all ANOVA and MANOVA tests, Greenhouse-Geisser epsilon adjustments were implemented to remove minor sphericity problems. Partial eta-square calculations were implemented as estimates of effect size (ES). A non-parametric test for repeated measures, the Friedman test, was used to determine whether there were any changes in VA pre and 0, 2, 5, and 10 minutes post-fatigue. A significance level of $p<0.05$ was set for all statistical analyses and all tabular and graphical data are presented at means ± standard deviations (SD).
Chapter 4

4 Results

Mean duration of the fatigue task was 83±17 seconds. From 12 subjects, 20 MUs (1-2 per subject) that met the inclusion criteria and were followed over the course of the fatigue protocol were included in the statistical analysis. Also, from these 12 subjects, peak power, $V_{\text{max35}}$, and MVC torque were assessed at baseline and during recovery. Peak power during the fatigue protocol was assessed for twelve subjects for the 3 time bins from which a MUFR was measured (0-15%, 45-60% and 85-100% TTF). VA and $P_T$ were assessed in only seven subjects because post fatigue twitches were severely depressed and it was not possible to reliably calculate VA or measure $P_T$ in all subjects at this time point. Therefore, these subjects were excluded from statistical analyses of these measures.

Prior to conducting the repeated measures MANOVA, zero-order correlations among all possible combinations of variables were conducted and none was statistically significant, indicating that colinearity was not problematic for the variables included in this test. Results from the repeated measures MANOVA showed a significant multivariate effect [F(12, 132) =8.82, $p<0.05$, ES=0.45]. Significant univariate effects were found for peak power [F(1.97, 21.69)=83.38, $p<0.05$, ES=0.88], $V_{\text{max35}}$ [F(2.45, 26.98)=170.0, $p<0.05$, ES=0.94], and MVC torque [F(2.31, 25.42)=58.73, $p<0.05$, ES=0.84]. Post-hoc simple contrasts showed that post-fatigue measures (post0, post2, post5, post10) for all three variables were significantly different compared to pre-fatigue measures, $p<0.05$ (Table 1). At fatigue (post0) peak power, $V_{\text{max35}}$, and MVC torque were reduced by 64%, 60%, and 37%, respectively (Figure 7). By 10 minutes post fatigue (post10), these measures increased to 88%, 89%, and 85% of baseline, respectively, but were not fully recovered (Figure 7).
Table 1. Results of simple contrast post-hoc analyses for peak power, $V_{\text{max}35}$, and MVC pre and post fatigue.

<table>
<thead>
<tr>
<th>Time</th>
<th>Mean ± SD</th>
<th>Effect Size</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Peak power (W)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>208.5 ± 54.1</td>
<td>- -</td>
</tr>
<tr>
<td>Post0</td>
<td>74.2 ± 30.6*</td>
<td>.92</td>
</tr>
<tr>
<td>Post2</td>
<td>134.7 ± 41.1*</td>
<td>.88</td>
</tr>
<tr>
<td>Post5</td>
<td>166.5 ± 48.7*</td>
<td>.77</td>
</tr>
<tr>
<td>Post10</td>
<td>182.6 ± 57.4*</td>
<td>.50</td>
</tr>
<tr>
<td><strong>$V_{\text{max}35}$ (°/s)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>299.2 ± 37.5</td>
<td>- -</td>
</tr>
<tr>
<td>Post0</td>
<td>120.9 ± 35.8*</td>
<td>.96</td>
</tr>
<tr>
<td>Post2</td>
<td>210.8 ± 36.1*</td>
<td>.93</td>
</tr>
<tr>
<td>Post5</td>
<td>251.3 ± 33.5*</td>
<td>.84</td>
</tr>
<tr>
<td>Post10</td>
<td>271.9 ± 41.0*</td>
<td>.48</td>
</tr>
<tr>
<td><strong>MVC torque (Nm)</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>75.9 ± 27.0</td>
<td>- -</td>
</tr>
<tr>
<td>Post0</td>
<td>47.8 ± 21.6*</td>
<td>.92</td>
</tr>
<tr>
<td>Post2</td>
<td>57.3 ± 24.7*</td>
<td>.91</td>
</tr>
<tr>
<td>Post5</td>
<td>60.8 ± 25.0*</td>
<td>.91</td>
</tr>
<tr>
<td>Post10</td>
<td>64.5 ± 26.3*</td>
<td>.75</td>
</tr>
</tbody>
</table>

*Significantly different than pre, $p<0.05$. 
4.1 Voluntary Activation and Peak Twitch

A Friedman test to assess changes in VA across 5 time points revealed no significant omnibus effect \( \chi^2 (4)=8.71, p>0.05 \). Results from a one-way repeated measures ANOVA revealed a significant omnibus effect for \( P_T \) \( [F(1.93,11.56)=17.59, p<0.05, ES=0.75] \). Post-hoc pairwise comparisons revealed that \( P_T \) decreased immediately post-fatigue (post0) by 73\% (2.5±0.5 Nm) compared to baseline (pre) (9.3±1.2 Nm, \( p<0.05 \)). \( P_T \) was recovered to baseline values by post5 (7.6±0.8 Nm, \( p>0.05 \)) and post10 (6.6±7.7 Nm, \( p>0.05 \)).
4.2 Motor Unit Firing Rate and Peak Power During the Fatigue Protocol

A one-way ANOVA on MUFR across three time points (0-15%, 45-60%, and 85-100% TTF) results demonstrated a significant omnibus effect \( F(1.38, 26.24) = 87.23, p < 0.05, ES = 0.82 \). Post-hoc repeated contrasts revealed that MUFR decreased significantly by 21% from 0-15% TTF (35.5±6.4) to 45-60% TTF [28.2±3.8, \( F(1,19) = 41.49, p < 0.05, ES = 0.69 \]) and decreased an additional 15% during 45-60% TTF to 85-100% TTF [23.0±3.5, \( F(1,19) = 88.30, p < 0.05, ES = 0.82 \)] (Figure 6).

A separate one-way ANOVA on peak power across three time points (0-15%, 45-60%, and 85-100% TTF) results demonstrated a significant omnibus effect \( F(1.24, 13.65) = 115.57, p < 0.05, ES = 0.91 \). Post-hoc repeated contrasts revealed that peak power decreased significantly from 0-15% TTF (159.8±44.3) to 45-60% TTF [96.8±29.6, \( F(1,11) = 108.68, p < 0.05, ES = 0.91 \)] and from 45-60% TTF to 85-100% TTF [43.2±16.2, \( F(1,11) = 81.50, p < 0.05, ES = 0.88 \)].
Figure 7. MUFR changes with fatigue progression from 0-15% TTF to 45-60% TTF to 85-100% TTF. Grey lines represent individual MUFRs and the black dashed line represents mean MUFR. The triangle represents the maximal isometric MUFR of anconeus MUs (as reported in (16)) * Mean MUFR is significantly different from previous time point ($p<0.05$)
Chapter 5

5 Discussion

The present study demonstrated that MUFRs of the anconeus were substantially depressed (>35%) in response to a dynamic fatigue protocol that lasted ~83 seconds and involved repeated maximal velocity elbow extensions at a moderately high load. These changes occurred concurrently with considerable impairments in overall elbow extensor mechanical function, indicating that substantial muscle fatigue was induced. Peak power, maximal isometric torque, peak twitch torque, and maximal velocity were significantly reduced following the fatigue protocol. These findings support the hypothesis that MUFR would decline substantially following the fatigue task, and to a greater extent than what has been previously reported for submaximal dynamic fatigue protocols (42,57). However, the hypothesis that the fatigue induced in this investigation would result in greater depression of MUFR compared to maximal isometric fatigue protocols was not supported (36,37,39).

Dynamic shortening contractions that result in muscle fatigue are accompanied by reductions in MUFR that may contribute to the reduction in maximal velocity and, therefore, loss of power. The anconeus muscle, as a part of the elbow extensor group, may be a valuable proxy indicative of fatigue-induced changes in all portions of the triceps brachii, which are innervated by the radial nerve.

5.1 Anconeus as a model to study neuromuscular fatigue

A novel aspect of this investigation was the ability to successfully record and track individual anconeus MUs during maximal effort fast, dynamic shortening contractions at high loads. Previously, evaluation of MU activity was limited to isometric, or slow, lightly-loaded dynamic contractions (13). Recording single MUs during velocity-dependent contractions has been limited due to the technical problems associated with electrode displacement and movement, which occurs due to joint angle changes that affect muscle length. The ability to track a MU over the course of a fatigue protocol is further complicated by repeated contractions over a large range of motion required for this task. Although the anconeus muscle responds with architectural changes on a similar relative scale as those of other limb muscles (66), it has unique mechanical and anatomical features that minimize electrode
displacement. It has relatively few MUs for its size (7), which likely improves unit selectivity with these indwelling wires. As a neuromuscular model, the anconeus is active at all angles of elbow extension and contraction intensities and its firing rates increase as velocity is increased to maximal levels (18,32,33). Recruitment of MUs is complete by ~25-35% MVC and additional force gradation activity is achieved by increasing firing rates (33,67,69) and thus rates span a large range from ~8 Hz to ~60 Hz (16). It has previously been shown that anconeus MUFR can be reliably tracked during contractions of maximal velocity and throughout substantial changes in joint angle (16,17). This feature has been confirmed in the present study.

5.2 Assessment of neuromuscular fatigue

It is known that fatigue is a task-dependent phenomenon; therefore, it is important to consider various task parameters when designing and comparing fatigue protocols. Velocity-dependent contractions were chosen here because most daily activities involve movements that have an unconstrained velocity with a rather fixed or constant external resistance. A graded fatigue protocol was implemented to extend task duration and enhance the potency of the induced fatigue processes, and concurrently to allow maintained higher velocities and joint range of motion as much as possible throughout the protocol. It was observed here and previously that at high loads, velocity quickly falls and range of motion becomes compromised (70,71).

Despite a voluntarily well-activated elbow extensor isometric MVC (>98%) that did not change during this challenging task, and agrees with previous studies (17,42), results demonstrated an overall reduction in elbow extensor function including significant impairments of both isometric and dynamic measures of fatigue. Following the protocol, isometric MVC, $V_{max35}$, and peak power all declined by 37%, 60%, and 64%, respectively. The fatigue protocol induced greater decreases in the dynamic measures ($V_{max35}$ and peak power) compared to the isometric MVC torque of the elbow extensors. This emphasizes the task dependent nature of fatigue. Because the system was fatigued through repetitive dynamic elbow extensions, rather than isometrically, it would be reasonable to suggest that dynamic measures of mechanical function would be depressed to a greater degree compared to static measures of mechanical function. Although peak power, MVC, and $V_{max35}$ did not
return to baseline levels by 10 minutes post-fatigue, substantial decreases in effect size estimates at that time point indicate that these three measures were approaching recovery.

Two prior investigations fatigued the elbow extensors using lightly loaded (~20%MVC) and slow (50%) dynamic contractions and results showed modest decreases in MVC torque of ~27% (42) and ~25% (57) compared to maximal dynamic fatigue found in the present study. In another study related specifically to the anconeus, Harwood et al. employed a fatigue protocol that consisted of repeated sets of 10 shortening elbow extensions with moderate load and velocity (40% MVC and 60% V_max at 40% MVC) contractions followed by 2 maximal dynamic contractions (40% MVC/V_max40) (17). The mean duration of the fatigue task was ~160 seconds. Following that submaximal dynamic fatigue protocol, results demonstrated decreases in torque, velocity, and power of the maximal dynamic contractions of ~35%, ~45% and ~55%, respectively. Despite finding similar declines in torque (~37%), greater declines in velocity (~60%) and power (~64%) were found in the present study following maximal dynamic fatigue. The production of repeated maximal velocity contractions places an increased demand on the neuromuscular system. Maximal velocity contractions were performed to fatigue; therefore, it is not surprising that maximal velocity and peak power (which has a velocity component), are affected to a greater extent in this investigation compared to the prior submaximal dynamic fatigue study (17). These results emphasize the task-dependent nature of fatigue.

5.3 Motor unit firing rates

Maximal anconeus dynamic unfatigued MUFRs were comparable to those in previous investigations (16,17). Previous investigations found that firing rates at rest at maximal velocity with 25% and 40% MVC loads were ~39 Hz(16,17), and in this investigation, MUFR at maximal velocity with a 35% MVC load were ~36Hz. In contrast, anconeus MUFRs during an isometric unfatigued MVC have been reported to be ~24 Hz, ranging from 15 to 36 Hz (16). Thus it is clear that dynamic contractions elicit or require higher firing rates than those required for isometric MVCs presumably required to achieve fast dynamic contractions. The importance of high MUFRs to produce fast, dynamic contractions was emphasized in this investigation. Even at task failure in this study, when MUFRs were decreased to approximately 23 Hz, the rate was comparable to maximal isometric unfatigued
firing rates in this muscle (16). Interestingly, despite its fibre type (60-67% slow twitch (ST))(19), mean maximal isometric anconeus MUFRs are higher than that of the soleus (~89% ST; mean: ~11-16 Hz; range: 5-20 Hz), and only slightly lower than that of adductor pollicis (~80% ST) and biceps brachii (~50% ST) (mean: ~30 Hz, range: 12-60 Hz) (72–74).

The decrease in MUFR observed following maximal velocity elbow extensions to task failure in the present study was approximately 35%, from ~36 to ~23 Hz. This substantial decrease provides support for the effect of fatigue, rather than simply due to the effect of decreasing load in the fatigue protocol. For example, if the initial (45% MVC) load was maintained, task failure would have occurred within ~35 seconds, whereas with a graded protocol, task failure required almost 3 times longer to occur.

In other muscle groups, sustained isometric MVC fatigue protocols for 40 to 120 seconds showed ~45% reductions in MUFR in the adductor pollicis (39) and ~36% in the first dorsal interosseous (38). Intermittent maximal fatigue protocols showed reductions in MUFR of 35% in the tibialis anterior (36). Following a sustained 75% MVC fatiguing task in the triceps surae, MUFR was reduced by 35% (41). When 6 second, 50% MVC elbow extension contractions were repeated every ten seconds to fatigue, a reduction of ~30% in MUFR was observed in the triceps brachii (65). It was hypothesized that, because of the higher range of MUFRs possible during dynamic contractions, a greater percentage decline in MUFR would be observed compared to an isometric task; however, this hypothesis was not observed. This may have been because the initial, unfatigued MUFRs are higher for dynamic compared to isometric contractions (39 Hz for dynamic and 24Hz for isometric in the anconeus) (16).

With a higher initial firing rate in dynamic contractions, a greater absolute decrease in firing rate is required to find comparable relative decreases to isometric protocols at task failure. However, isometric fatigue in the anconeus has not been explored; therefore, a direct comparison between fatigue modalities for this muscle model is not yet possible. Likely due to the dynamic nature, rates cannot decline too far if relatively fast velocity and range of motion are to be maintained.

From prior studies that used slow and lightly loaded dynamic elbow extensor fatigue protocols, extremely variable changes in MUFR have been reported (42,57). In one investigation, in 17 of 25 tracked MUs, MUFR declined by at least 2Hz, 4 of which showed
decreases and subsequent increases in firing rates (42). Seven MUs showed no change in firing rate, and one consistently increased. However, taken together, at task failure, overall firing rates decreased by ~24%. In the present investigation, all 20 MUs showed a decline by the end of the fatigue protocol (~83 seconds) and to a greater extent (>10%) than that observed in the previously mentioned study (42). Another study used faster (60% of maximum velocity) and moderately loaded (40% of MVC) dynamic elbow extensions fatigue and assessed anconeus MUFR changes for the submaximal task and between pre and post fatigue maximum velocity contractions (17). Anconeus MUFR declined ~20 % for maximal dynamic contractions; however, there was no difference in MUFR for submaximal dynamic contractions performed pre and post fatigue. Additionally, no change was found in MUFR during maximal velocity contraction beyond 50% TTF indicating that MUFR achieved a nadir by the half-way point of the protocol, which was ~31Hz. In the present study compared to this previous one, greater declines in MUFRs were observed (Figure 6). The progressive decline in MU firing rates and the larger change in firing rates compared to previous investigations in this model may be explained by the potency of the induced fatigue. The design of this protocol to induce substantial fatigue was confirmed by the large reduction in post fatigue MVC (37%), maximal velocity (60%), and peak power (64%) and lack of recovery in these measures by 10 minutes after the fatigue protocol ended. Reductions in calcium and changes in crossbridge kinetics related to muscle fatigue temper muscle fibre torque, power, and velocity (for review see 49,75). In addition, a usual finding of fatigued muscle is depressed and slowed contractile properties (38,41,76,77) and in the present study twitch amplitudes after fatigue were 73% lower, with some twitches too small to accurately measure, indicative of low-frequency fatigue.

5.4 Summary

This investigation demonstrated that anconeus MUs can be recorded during maximal velocity contractions under a moderately high load through a large range of motion (80°), and can also be followed throughout repeated contractions of this intensity to task failure. Additionally, the task-dependent nature of fatigue was emphasized in the results of this study. Dynamic markers of fatigue (reduced peak power and velocity) were substantially affected, and to a greater degree than static markers of fatigue (MVC torque), at task failure. Additionally, the importance of high MUFR to produce fast, dynamic contractions is
reinforced in the results of this investigation. At the point of task failure, MUFR were similar to maximal rates found for isometric contractions in a non-fatigued state (16,17). It is important to recognize that the protocol used to fatigue the neuromuscular system influences the type and degree of mechanical impairments that are expressed.

Similar decreases in MUFR to those found following maximal isometric fatigue were found in this investigation, indicating that a similar mechanism may be used by the neuromuscular system to modulate MUFR in a fatigued state. Interestingly, MUFR declined substantially from the start (0-15% TTF) to the middle (45-60% TTF) of the protocol and further declines were found from the middle to the end (85-100% TTF). This indicates that the reductions in MUFR progressed from the start of the protocol to the point of task failure. This finding is unique to what was found when submaximal dynamic contractions were used to fatigue the elbow extensors. The previously mentioned study found that MUFR during maximal contractions showed substantial declines in the first half of the protocol for maximal velocity contractions, but no further declines in MUFR were observed in the second half of the task (17).

By establishing dynamic MUFR changes related to dynamic fatigue in a healthy, young population, we can then correlate these values with other measures of neuromuscular fatigue. Additionally, we can explore the relationship of varying dynamic fatigue intensities and MUFR in disease or trained populations using the anconeus model.

5.5 Limitations

Although the anconeus model provides a unique opportunity to record MU activity during high-intensity dynamic contractions, there are some limitations associated with this investigation. These limitations can be classified into mechanical and architectural, technical, and electrophysiological.

In terms of mechanics, the anconeus is not a prime extensor of the elbow. It contributes ~15% of the torque production in elbow extension, compared to the cumulative ~85% contribution of the triceps brachii muscle group (23). Architecturally, anconeus volume and cross-sectional area (21,78), pennation angle and fascicle length (79) and elbow extension moments (79) differ from that of those of the triceps brachii. Although, recently it has been
shown that anconeus undergoes similar relative, albeit smaller absolute changes in fascicle length compared to larger muscles (66). Previous investigations have shown the twitch tension of the anconeus to be ~25% of that of the lateral head of the triceps brachii (19). Therefore, the length-tension and force-frequency relationships of the anconeus are different than those of the long head of the triceps brachii (19). Additionally, the contribution of each of the elbow extensors to torque production differs depending on joint angle in isometric contractions and range of motion in dynamic contractions (24). The mechanical output measurements in this investigation are based on the cumulative output of all four elbow extensor muscles (anconeus and the three heads of the triceps brachii), and potentially minor contributions from some forearm muscles. Without direct measurement of torque from the anconeus relative to the other elbow extensors, it is difficult to know how much this muscle was actually contributing to elbow extension in this investigation. It is known; however, that anconeus is active in all forms of elbow extension and at all torque levels. Despite these architectural differences, the anconeus is a unique model to study motor unit activity during dynamic contractions, which, to date and with the techniques currently available for the study of MU activity, has not been feasible in other muscles in vivo.

By using fine wire intramuscular EMG rather than surface EMG, the relationship between MUFR and mechanical output can be investigated with greater precision. Much of the interference associated with recording from the skin surface is eliminated by recording intramuscularly (80), provided there is minimal movement of the electrode when the wires are hooked in to the muscle. We assumed that the characteristics of motor unit action potentials are consistent and distinct from neighbouring MUs. According to previous investigations, different distributions of individual MU twitch tensions within the motor pool depend on the firing rate and recruitment threshold (81–83). It was assumed that motor units recorded in this investigation follow the same basic principles as other muscles, but the distribution of twitch tensions in the anconeus motor unit pool are unknown and, therefore, we can only infer the mechanical output of the anconeus attributed to motor unit activity. This limitation is further compounded by induced fatigue because the input-output relationship of motor neurons is disrupted by decreases in the muscle fibres’ ability to produce torque, velocity, and power (84,85).
Electrophysiological limitations are inherent to any investigation employing EMG recordings to investigate single MU activity. Single MU recordings are made from the sarcolemma and represent the cumulative influence of multiple inputs on the α-mn. The recordings in this investigation represent likely the sum input from spinal and supraspinal factors, and can only be regarded as a final common pathway, rather than allow specific insight into where fatigue-related changes to the neuromuscular activity occur (2). Although the wires were hooked in to the muscle, and presumably the recording surface did not change, there are changes with fatigue to the intracellular action potentials that are recorded in the extracellular space (86,87). A conservative approach was used to classify motor units, but the possibility still exists that erroneous classification occurred by the sorting algorithm or investigator.

Although the anconeus allows for the recording of single MU activity under conditions (moderate to high torque levels and high velocity dynamic contractions) that were not previously accessible, technical limitations made it difficult to obtain a high MU sample size. Participants returned to the lab for 1 to 3 sessions to obtain an adequate (20 MU) sample of MUs. Although repeated trials were performed, the sample size in this investigation was less than in previous studies of MU activity in the elbow extensors (~40 MUs) (57,88). However, in the aforementioned investigations not all MUs were tracked from beginning to end of the fatigue task. We tracked the same MUs throughout the entire fatigue protocol to allow us compare the firing rate of the same MU across three time points. The anconeus likely has few motor units (~25-60) (7); therefore, the population that was collected in this investigation probably represents a significant portion of the motor unit pool, and the motor units have a significantly broad range of firing rate (~23-48 Hz). It is unlikely that a biased existed and that we collected from a pool of similarly-behaving motor units. Also, it might have been useful to have made some global-type EMG recordings of the triceps brachii, but the setup and anatomical space available made this very difficult. A separate EMG study focussing on the triceps brachii during this task could be helpful.

5.6 Future Directions

This investigation has contributed substantially to the current body of literature on MUFR changes with fatigue by extending knowledge of this topic to maximal velocity dynamic fatigue. Only recently, through the exploitation of the anconeus model, has it been possible to
record MU activity during fast, dynamic contractions. More research is required to add to
dynamic fatigue literature and extend the model to learn how MUFR changes with fatigue in
special populations, such as disease states or training. Additionally, fatigue is task, age, and
sex specific. Therefore, it will be important to investigate dynamic fatigue using different
fatigue protocol parameters, and in aged and female populations. It will be important to
investigate whether dynamic neuromuscular properties and behaviour of anconeus motor
units change, and to what degree relative to other muscles under various physiological states.

Although the contractile properties were evaluated during recovery here, future studies
should record recovery of MUFR following this fatigue protocol. Because low-frequency
fatigue, can persist for hours, or, in extreme cases, days (89–91), it would be useful to
evaluate MUFR in relation to factors that contribute to low-frequency fatigue related to
disruption in the E-C coupling process or in muscle fibre structure and integrity (89). Due to
the significant depression of the post-fatigue twitches, it was difficult to assess changes in
twitch properties following fatigue in the current study. To address this issue, an alternative
approach may be to evaluate tetanic measures of twitch properties such HRT from brief 50
Hz stimulation.

Additionally, it will be important to investigate where, along the motor pathway, changes in
voluntary drive are altered. The source of the reduction in voluntary drive may be isolated by
employing various neuromuscular techniques such as afferent stimulation, transcranial
magnetic stimulation, or cervicomedullary stimulation.

Lastly, this model may be used to help resolve whether afferent feedback is different between
fatigue induced by different contraction types (dynamic versus isometric) by ischemic
clamping to trap metabolites and correlate these differences with changes in MUFR between
the two fatigue modalities.
References


Appendix

Appendix A. Ethics approval documentation

Use of Human Participants - Ethics Approval Notice

Principal Investigator: Dr. Charles Rice
Review Number: 18097
Review Level: Full Board
Approved Local Adult Participants: 100
Approved Local Minor Participants: 0
Protocol Title: Neuromuscular control of human movement
Department & Institution: Anatomy & Cell Biology, University of Western Ontario
Sponsor: Natural Sciences and Engineering Research Council

Ethics Approval Date: July 22, 2011 Expiry Date: August 31, 2015

Documents Reviewed & Approved & Documents Received for Information:

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<tr>
<th>Document Name</th>
<th>Comments</th>
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<td>UWO Protocol</td>
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This is to notify you that the University of Western Ontario Health Sciences Research Ethics Board (HSREB) which is organized and operates according to the Tri-Council Policy Statement: Ethical Conduct of Research Involving Humans and the Health Canada/ICH Good Clinical Practice Practices: Consolidated Guidelines; and the applicable laws and regulations of Ontario has reviewed and granted approval to the above referenced study on the approval date noted above. The membership of this HSREB also complies with the membership requirements for REB's as defined in Division 5 of the Food and Drug Regulations.

The ethics approval for this study shall remain valid until the expiry date noted above assuming timely and acceptable responses to the HSREB's periodic requests for surveillance and monitoring information. If you require an updated approval notice prior to that time you must request it using the UWO Updated Approval Request form.

Member of the HSREB that are named as investigators in research studies, or declare a conflict of interest, do not participate in discussions related to, nor vote on, such studies when they are presented to the HSREB.

The Chair of the HSREB is Dr. Joseph Gilbert. The UWO HSREB is registered with the U.S. Department of Health & Human Services under the IRB registration number IRB 00000940.

Signature

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January 2013

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