

EVALUATING MUSCULOSKELETAL STIFFNESS IN THE UPPER EXTREMITY

A BIOMECHANICAL EVALUATION OF LIGAMENT AND MUSCULAR
STIFFNESS IN THE DISTAL UPPER EXTREMITY

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ABSTRACT

The purpose of this thesis was to evaluate musculoskeletal contributions to joint stiffness in the distal upper extremity. An *in-vitro* and *in-vivo* approach was used to examine muscle and ligament contributions to mechanical joint stiffness at the elbow and wrist. In Chapters 2 and 3 an *in-vitro* approach was used to evaluate ligament contributions to carpal tunnel mechanics. Chapter 2 documented transverse carpal ligament (TCL) mechanical properties and provided a calculation of TCL length when stretched, which confirmed the ligaments importance in carpal tunnel mechanics and carpal bone stability. Chapter 3 quantified mechanical properties of the TCL at six different locations using a biaxial tensile testing method. It was found that the complex TCL fibre arrangement makes the tissue properties location dependent. The TCL contributes to carpal tunnel mechanics and carpal stability and the ligament contributions are different depending on the tissue location tested. Chapters 4 and 5 focused on the effects of hand loads and arm postures on the muscular response to sudden arm perturbations. The elbow flexors demonstrated stiffness contributions immediately prior to a perturbation and were influenced by posture and hand loading. The forearm muscles provided a small contribution to elbow joint stiffness. Chapter 6 also found muscular contributions that increased wrist joint stiffness immediately prior to a sudden perturbation. Additionally, for a small grip-demanding task, forearm muscle co-contraction resulted in large increases in wrist joint stiffness.

This thesis has provided a detailed analysis of the TCL which improves our understanding of the carpal tunnel and specific mechanisms of injury. It is the first to

document individual muscle contributions to elbow and wrist joint stiffness. The comprehensive analysis of ligament and muscular contributions to joint stiffness has provided insight into joint stability in the distal upper extremity. This can improve our understanding of injury caused by sudden joint loading.

Keywords: Distal Upper Extremity, Joint Rotational Stiffness, Stability, Muscle Stiffness, Ligament Stiffness, Cadaver, Occupational Biomechanics

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Last, but not least I wish to dedicate this work to my grandparents (Nan Jenkins and Pop Holmes) who unfortunately didn't get to see the final document, but I know you have been with me every step of the way.....

THESIS FORMAT AND ORGANIZATION

This thesis contains material from the PhD work of Michael W.R. Holmes and has been prepared in a “sandwich” format as outlined in the McMaster University School of Graduate Studies’ Guide for the Preparation of Thesis. The thesis begins with a general introduction to the research area (Chapter 1), followed by 4 studies that have been prepared as 5 manuscripts and individual thesis chapters (Chapters 2-6). The thesis ends with a concluding chapter (Chapter 7) that provides a discussion of the findings and recommendations for future research in the area.

The thesis has been divided into two parts:

Part I (Chapters 2 and 3) investigates ligament contributions to carpal tunnel mechanics and wrist joint stiffness. Chapter 2 is ‘in press’ in the *Journal of Orthopaedic Research* and Chapter 3 has been submitted for publication in the same journal.

Part II (Chapters 4, 5 and 6) of this thesis investigates the effects of arm posture and hand loading on muscle activity while providing a detailed analysis of individual muscle contributions to joint rotational stiffness at both the elbow (Chapters 4 and 5) and wrist (Chapter 6). Chapters 4, 5 and 6 have been either submitted or prepared in manuscript form and will be submitted upon completion of the dissertation.

CONTRIBUTIONS TO PAPERS WITH MULTIPLE ATUHORS

Part I:

Chapter 2 – In Press

Holmes, M.W.R., Howarth, S.J., Callaghan, J.P., and Keir, P.J. Carpal tunnel and transverse carpal ligament stiffness with changes in wrist posture and indenter size. In Press, *Journal of Orthopaedic Research*, DOI 10.1002/jor.21442.

Chapter 3 - Submitted for Publication

Holmes, M.W.R., Howarth, S.J., Callaghan, J.P., and Keir, P.J. Mechanical properties of the transverse carpal ligament under biaxial strain. Submitted to the *Journal of Orthopaedic Research*.

Contributions

Chapter 2 of this thesis has been accepted for publication in the *Journal of Orthopaedic Research*. Chapter 3 has been prepared for submission to the same journal. Both studies were primarily the work and conception of Michael W.R. Holmes and Dr. Peter J. Keir.

All co-authors have contributed to these chapters. The methodology, collection, analysis, interpretation and manuscript preparation was performed by Michael Holmes. Dr. Keir was a significant contributor to the conception of this work, data interpretation and manuscript preparation. Samuel Howarth was instrumental in data collection for both studies. Samuel Howarth and Dr. Jack Callaghan have provided input for each manuscript. This work would not have been possible without Dr. Callaghan's *in-vitro* laboratory at the University of Waterloo, Waterloo, Canada.

Part II:

Chapter 4 - Submitted for Publication

Holmes, M.W.R., and Keir, P.J. Posture and hand load alter muscular response to sudden elbow perturbations. Submitted to the *Journal of Electromyography and Kinesiology*.

Chapter 5 - Formatted for Publication

Holmes, M.W.R., and Keir, P.J. Muscle contributions to elbow joint rotational stiffness in preparation for sudden external arm perturbations. Prepared for submission to *Human Movement Science*.

Contributions

Both authors have contributed substantially to these chapters. The study conception, methodology, data collection, analysis and manuscript preparation was performed by Michael Holmes with significant input at all stages from Dr. Peter Keir. Dr. Jim Potvin provided consultation during the analysis stage of chapter 5.

Chapter 6 - Formatted for Publication

Holmes, M.W.R., and Keir, P.J. Forearm muscle co-contraction and individual muscle contributions to joint rotational stiffness during flexion and extension wrist perturbations. Prepared for submission to *Human Movement Science*.

Contributions

The study conception, methodology, data collection, analysis and manuscript preparation was performed by Michael Holmes and Dr. Peter Keir. Dr. Jim Potvin provided consultation during the analysis stage of chapter 6.

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LIST OF ABBREVIATIONS

WMSD:	Work related musculoskeletal disorders
CNS:	Central nervous systems
JRS:	Joint rotational stiffness
TCL:	Transverse carpal ligament
FR:	Flexor retinaculum
CTS:	Carpal tunnel syndrome
ANOVA:	Analysis of variance
LSD:	Least Significant Difference
EMG:	Electromyography
MVE:	Maximal voluntary excitation
MVC:	Maximal voluntary contraction
AD:	Anterior deltoid
TB:	Triceps brachii
TRI _{Lo} :	Triceps brachii long head
TRI _{La} :	Triceps brachii lateral head
TRI _M :	Triceps brachii medial head
BB:	Biceps brachii
BI _L :	Brachii long head
BI _S :	Brachii short head
BRA:	Brachialis
BRD:	Brachioradialis

FCR:	Flexor carpi radialis
FDS:	Flexor digitorum superficialis
FCU:	Flexor carpi ulnaris
ECR _L :	Extensor carpi radialis longus
ECR _B :	Extensor carpi radialis brevis
ED:	Extensor digitorum communis
ECU:	Extensor carpi ulnaris
CCI:	Co-contraction index
JRS _M :	Individual Muscle joint rotational stiffness
JRS _T :	Total joint rotational stiffness
MJRS _P :	Maximum joint rotational stiffness potential
CSA:	Cross sectional area
PCSA:	Physiological cross sectional area

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CHAPTER 1: INTRODUCTION

1.1 Introduction

Humans interact continuously with objects in the environment and a fundamental understanding of how the musculoskeletal system functions to safely complete the required demands of a task is vital for an injury free system. Seemingly trivial human-object interactions require the musculoskeletal system to instantaneously find an optimal solution to coordinate a movement pattern that both successfully and safely accommodates the task demands. In the upper extremity, activities of daily living involve the control and coordination of multiple joints with a redundant number of muscles capable of performing a given task. Despite our innate ability to interact with the environment, specific mechanisms can place the musculoskeletal system at an increased risk for injury. From an occupational perspective, musculoskeletal disorders to the distal upper extremity are recognized as a close second to back injury claims reported in the workplace (WSIB, 2009) and present a large financial burden to the economy. These injury claims are concerning given that hand and arm movements are imperative to just about all activities of daily living and often leave a large number of people unable to work or perform everyday activities.

In 2009, 19.7% of all lost time claims in Ontario were related to the upper extremity (WSIB, 2009). Additionally, there is evidence to support that upper extremity health care costs are larger than those pertaining to other regions of the body (Silverstein et al., 1998). Epidemiological studies demonstrate a strong relationship between

workplace factors (including awkward posture, large hand force and highly repetitive movements) and the reporting of work related musculoskeletal disorders (WMSDs) to the hand and wrist (NIOSH, 1997). As a result, the effects of arm postures, hand loads and repetitive motion on muscle activity and joint loading has been substantially documented at the wrist, elbow and shoulder during static and controlled dynamic contractions (An et al., 1981; Au and Keir, 2007; de Groot et al., 2004; Dul et al., 1984; MacDonell and Keir, 2005; Mogk and Keir, 2003; Sporrang et al., 1995, 1996). Workplace factors have combined relationships that promote increases in the likelihood of injury to the distal upper extremity (Moore et al., 1991; NIOSH, 1997; Silverstein et al., 1986), which lead to complex investigations. The realm of disorders to the upper extremity can develop due to sudden and unexpected loading or slow and progressive loading over long periods of time. The largely varied pathomechanics of upper extremity injuries creates difficulty in defining a precise causal path.

Despite considerable research on specific mechanical relationships to injury in the distal upper extremity, the prevalence of work related injuries remains high. Some of these research areas have included, but are certainly not limited to, the aetiology of muscle disorders due to loading in the hand and forearm (Jonsson, 1982; Mogk and Keir, 2003; Snijders et al., 1987; Veiersted et al., 1993), the aetiology of passive tissue loading in the hand and forearm (Armstrong and Chaffin, 1978; Keir et al., 1996), nerve disorders in the hand (Keir et al., 1997, 1998; Moore et al., 1991; Rempel et al., 1998), muscle fatigue (Bystrom and Kilbom, 1990; Hagg and Milerad, 1997), and the motor control of arm movements (De Serres and Milner, 1991; Franklin et al., 2003; Franklin et al., 2007;

Milner et al. 1995; Perreault et al., 2001; Perreault et al., 2004). Interestingly, those investigating spine biomechanics and the aetiology of low back pain/injury have focused on analogous research topics, but more recently, a focus on spinal stability has provided new insight into specific mechanisms of low back pain (Cholewicki and McGill, 1996; Crisco and Panjabi, 1992; Potvin and Brown, 2005; Reeves et al., 2006). To date, research investigating musculoskeletal contributions to upper extremity joint stability has seen limited consideration and is therefore a primary focus of this thesis, with the goal of providing new insight into disorders of the upper extremity.

The ability of the musculoskeletal system to maintain joint stability is critical for successful movement, and a better understanding of how joint stability is controlled in the upper extremity will enhance our understanding of injury. The upper extremity complex consists of many muscles and joints, resulting in a highly redundant system with many degrees of freedom. This complexity will influence how the system coordinates optimal movement. If a system or joint is unstable, small perturbations can lead to unpredictable movements that may result in tissue injury. In many everyday situations, the upper extremity is placed in situations where interactions with external objects apply variable forces to the hand, challenging the system's ability to maintain joint stability. An obvious example for the distal upper extremity involves the use of hand tools (Rancourt and Hogan, 2001), which require the upper extremity musculature to provide a stabilizing means to counter the instability of the tool. Muscular actions to counter an external disturbance are governed by the central nervous system (CNS) and its ability to regulate control of the musculoskeletal system. The ability of the CNS to provide control will

depend on the external conditions and task demands which have a strong influence on safe and successful motion.

The investigation of mechanical joint stability involves an understanding of the complex musculoskeletal system. Panjabi (1992) proposed a conceptual basis for understanding spinal stability and this work can be applicable to the upper extremity. Panjabi (1992) identified three areas that will aide a joint in the maintenance of stability, including: i) the passive system (ligaments), ii) the active system (muscle), and iii) the neural control system (nerves and CNS). When these systems are able to work collectively, a joint is more capable of withstanding an external disturbance, within a margin of safety. If an error ensues in one of these systems, joint injury or failure could occur. Building upon these three areas, Latash and Zatsiorsky (1993) showed that one effective method to increase stability is through joint stiffness and a complete joint stiffness analysis involves the combination of individual stiffness contributions from muscles, tendons, ligaments, cartilage and bones. Considering each of the aforementioned structures more specifically, muscles respond with spring and damper like properties (Hill, 1938; Hill, 1950) and thus the stiffness of a muscle will contribute to overall stability of a structure or joint (Bergmark, 1989). Additionally, some joints may be inherently stable due to the anatomical arrangement of boney segments (Bryce and Armstrong, 2008; Safran and Baillargeon, 2005), while other joints require ligamentous contributions. At the elbow joint for instance, articular surfaces of the distal humerus, proximal ulna, and proximal radius vary in size and geometry, providing congruent surfaces that form constraints to movement (Bryce and Armstrong, 2008). At the wrist

joint however, a large number of carpal bones and ligaments interconnect in such a way that carpal instabilities are quite common (Dias and Garcia-Elias, 2006; Larsen et al., 1995; Linscheid et al., 1972). Finally, ligaments have mechanical properties that respond in a viscoelastic manner with varying mechanical stiffness depending on the ligament being tested. Ligamentous contributions to joint stability are largely reliant on joint mobility. Studies specific to the upper extremity have shown the importance of ligamentous contributions to stability at the shoulder (Blasier et al., 1997; Burkart and Debski, 2002; Veeger and van der Helm, 2007), elbow (Bryce and Armstrong, 2008; Regan et al., 1991; Safran and Baillargeon, 2005) and wrist (Berger, 2001; Fisk 1984; Garcia-Elias et al., 1989a,b; Mayfield et al., 1976; Short et al., 2007; Xu and Tang, 2009).

It is apparent that the mechanical properties of the tissues surrounding a joint collectively contribute to joint stability, and a better understanding of the individual tissue contributions can provide insight into tissue failure and joint injury. In mechanical terms, stability is determined in a binary sense, with a system considered stable or unstable. However, from a biomechanical perspective, the understanding of a system's ability to adapt or respond to a disturbance is beneficial for understanding injury during sudden loading events. Reeves et al. (2006) provided insight into the level or magnitude of stability and proposed the term 'joint robustness' as a means of describing a system's ability to respond to a given disturbance. If a joint is more robust, it is better able to accommodate a disturbance and return to a state of equilibrium, which provides a margin of safety for the joint. Considering the term robustness further, the ability of a system to adapt or resist a perturbation can be influenced by a number of factors that modulate the

mechanical impedance of the joint. These factors include joint parameters such as muscle and joint stiffness, dampening and limb inertia. The mechanical impedance of a joint reflects the system properties determined by inertial, viscous and elastic elements (Latash and Zatsiorsky, 1993) and impedance is typically used to quantify resistance of a mechanical system to movement (Milner, 1995). Therefore, stiffness is a mechanical property of impedance and thus, impedance may be a more appropriate term than robustness when providing insight into the level or magnitude of stability. In the spine, the reference is often made to suggest that, in an unstable spine, the system may ‘buckle’ and result in joint injury and/or failure (Brown and McGill, 2009; Crisco et al., 1992; Potvin and Brown, 2005). In the upper extremity, and under dynamic conditions, the term mechanical impedance may be more relevant given that joint stability is frequently considered in the context of investigating the control of posture and movement. Human interaction with the environment is one of the most fundamental, yet necessary, aspects of human movement and the mechanical impedance of muscle during this interaction has been considered in arm movements for many years (Hogan, 1984; Hogan, 1990; Milner, 1995). Perreault et al. (2004) explained that maintaining a stable arm posture during a task requires muscular force to complete the task, while maintaining joint stability sufficient to reject any external disturbances that may occur. In the realm of motor control, understanding how the mechanical properties of the environment compromise arm stability have been quantified using measurements of endpoint stiffness and many authors have contributed to the literature (Franklin and Milner, 2003; Hogan, 1985; Perreault et al., 2001; 2004). However, a systems approach has traditionally been

considered, with the evaluation of mechanical impedance for the entire arm during basic movements. A better understanding of individual muscle and ligament properties, which largely contributes to overall mechanical impedance of a joint, will help provide knowledge of how the CNS controls posture and movement. The quantification of individual tissue properties will also provide context to how muscles coordinate, and how structures are loaded, during sudden disturbances.

Considering the joint parameter of stiffness further, research on the upper extremity (De Serres and Milner, 1991; Stokes and Gardner-Morse, 2000), as well as the spine (Cholewicki and McGill, 1996; Brown and Potvin, 2005), has suggested that muscle stiffness is a fundamental and essential contributor to mechanical impedance. Hogan (1985) demonstrated that the elastic properties of muscle contribute to the stability of the hand when faced with unpredictable disturbances. However, it is important to note that changes in joint stiffness can be modulated through adaptations to muscle that include changes in individual muscle activity, the level of co-contraction, muscular synergy and limb position (van Loon et al., 2001). This is of particular importance in the upper extremity due to the nature of the system, the ability for considerable joint movement, and the complex muscular demands. These adaptations, which modulate joint stiffness, are closely linked to workplace factors of posture, force and repetition that have been previously discussed. As noted by Hogan (1990), the most important aspect for changing the mechanical impedance of a joint is limb position, since changing limb configuration will influence joint stiffness, viscosity and inertia at the hand. Thus, Hogan

(1990) suggested that the single most important strategy for controlling a hand-object interaction is the choice of posture.

With changes in limb position, the force and moment generating capacity of a muscle will change, thus understanding individual muscular actions throughout a range of motion is needed beyond muscle activity alone. Muscle stiffness increases with muscle activation, and muscle co-contraction (or co-activation) is often considered a primary method used by the CNS to stabilize the position of a limb (Bergmark, 1989; De Serres and Milner, 1991; Hogan, 1990; Milner et al. 1995; Murray, 1988). Muscle co-contraction is typically defined as the simultaneous activation of antagonist (or agonist) muscles that cross a joint. Hughes et al., (2001) also suggested that muscle co-contraction can be interpreted as activity in excess of that needed to produce the desired movement. Thus, if the magnitude of muscle activation is in excess of that needed to produce a movement, it must serve an additional purpose, which has typically been considered a method to stiffen the joint (Bergmark 1989; Cholewicki and McGill, 1996; Milner et al., 1995). In the upper extremity, co-contraction has been extensively studied and shown to increase with load instability, decrease with practice of a movement, improve movement accuracy and increase joint stiffness and damping (Gribble et al., 2003; Milner and Cloutier, 1993; Milner and Cloutier, 1998; Milner et al., 1995; Osu et al., 2004; Selen et al., 2005; Visser et al., 2004). However, increased muscle co-contraction will inherently increase joint loading and can be metabolically inefficient (Hogan, 1984). Hogan (1984) elegantly evaluated muscle co-contraction of the biceps and triceps during a simple postural maintenance task. He was able to demonstrate that

maintaining posture via reflex feedback would be more energy efficient than continuous muscle co-contraction, however, the inherent delays associated with such a pathway would be a large limitation to the approach. Performing a theoretical analysis of muscle co-contraction at the elbow joint, Hogan showed that co-contraction was necessary to stabilize the joint during small perturbations. Because of this metabolically costly approach to joint stiffness, it is likely that an optimization is required from the neuromuscular system since an increase in joint loading may lead to injury, but it can also result in added stability (Stokes and Gardner-Morse, 2003).

Besides changes in limb position, muscular co-contraction has been extensively evaluated during hand gripping tasks and wrist movements, with a focus on forearm muscle activation (Claudon, 1988; Cort et al., 2006; Halpern and Fernandez, 1996; Mogk and Keir, 2003; Snijders et al., 1987; Volz et al., 1980). Milner and Cloutier (1993) suggested that during sudden wrist perturbations, participants would adopt a strategy that increases wrist stiffness by increasing activation of the forearm muscles. This increased activation is typically accomplished via grip force requirements that are necessary for the interaction of objects in the environment. The influence of grip force on forearm muscle activity has been well documented (Cort et al., 2006; Mogk and Keir, 2003). Mogk and Keir (2003) observed large increases in muscle co-contraction of the wrist extensors during gripping which, again, is considered a necessary mechanism to help stabilize the wrist joint (De Serres and Milner, 1991; Snijders et al., 1987). However, the majority of forearm evaluations involve isometric gripping tasks, static postural demands, or slow, controlled arm movements. To date, there has been limited information on the effects of

arm posture and hand loading on forearm muscle co-contraction during sudden externally applied loads that cause involuntary joint rotation, and ultimately, the requirement of the CNS to maintain control during motion.

Traditional stability analyses can be complex even for static evaluations due to the requirement of static equilibrium in most stability calculations, hence the need to balance joint moments. Performing these calculations about a complex joint can be even more difficult to accomplish and quantifying joint stability using dynamic approaches have been virtually nonexistent. Furthermore, most joint stability approaches have evaluated stability for the entire system. Potvin and Brown (2005) acknowledged that the importance of quantifying individual muscle contributions to joint stiffness and, ultimately, joint stability could have profound implications in understanding joint safety. As a result, Potvin and Brown (2005) developed an equation to determine individual muscle contributions to joint rotational stiffness (JRS). The equation allows for the quantification of individual muscle contributions to JRS at any time point throughout a perturbation using three main parameters of the muscle, including: i) muscle force, ii) three-dimensional coordinates of the origin, insertion and node/wrap points for a given muscle crossing the joint, and iii) a constant relating muscle force and length to muscle stiffness. An evaluation of joint stiffness from this perspective negates some of the previous complications with traditional stability analysis and provides context for a muscle's contribution to the system's overall stiffness. The approach can be applied throughout an entire perturbation, providing information about each muscle's ability to respond or adapt to a disturbance at any time during motion.

Most work relating to the upper extremity has, to date, evaluated joint stiffness at the endpoint of a movement (Franklin and Milner, 2003; Franklin et al., 2007; Perreault et al., 2001; Perreault et al., 2004), with limited insight into how initial postural states or muscular demands influence the resultant motion. Furthermore, the investigation of overall joint stability at the shoulder (Anglin et al., 2000; Oosterom et al., 2003; Veeger and van der Helm, 2007), elbow (Giesl et al., 2004; Stokes and Gardner-Morse, 2000), and wrist (Cooney et al., 1989; Garcia-Elias, 1995; Larsen, 1995; Linscheid et al., 1972) has been investigated substantially. However, none of these studies have quantified individual muscle contributions, thus conclusions regarding potential joint and tissue injury are limited. A better understanding of the influence of posture and loading demands on muscle co-contraction in the distal upper extremity, and subsequently how muscle co-contraction modulates muscular contributions to joint stiffness, needs to be quantified. The JRS approach could provide new insight into the mechanical risk factors associated with joint injury in the upper extremity.

1.2 Purpose and Hypotheses

The global purpose of this thesis was to quantify musculoskeletal contributions to stiffness in the distal upper extremity. Joint stiffness is modulated by load and posture, and thus was a common theme across all studies in this thesis. In particular, a variety of hand loading and arm posture demands were tested to investigate the effects on muscular and ligamentous contributions to elbow and wrist joint stiffness. Muscular contributions are imperative to joint stiffness and for maintaining joint stability, and therefore an in-depth investigation of the forearm and elbow musculature was performed to evaluate

individual muscle contributions to elbow and wrist joint stiffness during sudden arm perturbations. At the wrist, ligamentous contributions are important for carpal bone stability, therefore mechanical properties of the carpal tunnel and transverse carpal ligament (TCL) were considered. It was anticipated that a better understanding of upper extremity joint stability could be an essential missing link to lowering the prevalence of musculoskeletal pain and injury to the distal upper extremity.

The specific purpose and hypotheses for each chapter is outlined below:

Chapter 2

Ligaments are essential to maintaining wrist joint and carpal bone stability. In particular, the TCL is thought to contribute to wrist joint stability, carpal bone stability, carpal tunnel mechanics and act as a pulley system for the flexor tendons. It is clearly an important component of the wrist structure; however, the TCL is poorly understood with respect to its exact function.

Purpose: To investigate the mechanical properties of the intact cadaveric TCL by directly loading the structure in different wrist postures.

Hypotheses: The characterization of TCL stiffness will increase with an increase in indenter size. The largest indenter will produce the greatest stiffness as it will provide indentation to the entire carpal tunnel. Finally, cadaver arms placed in a flexed wrist posture will produce greater stiffness than neutral or extended postures, due to an interaction with the flexor tendons.

Chapter 3

This study was a follow-up to Chapter 2. Although providing information on carpal stability and carpal tunnel mechanics, the methodology used in Chapter 2 makes the distinction between carpal tunnel stiffness and TCL stiffness difficult to accomplish.

Purpose: To measure mechanical properties of the cadaveric TCL at six different locations using a biaxial tensile testing method. The TCL was evaluated in isolation from other carpal tunnel structures.

Hypothesis: TCL sections located close to the attachment sites of the ligament will demonstrate increased stiffness, primarily due to thickness in these regions.

Chapter 4

Purpose: To investigate effects of arm posture and hand loading on upper extremity muscle activity during sudden elbow perturbations. Specifically, muscle activations and coordination strategies were evaluated pre-perturbation and post-perturbation to understand voluntary and involuntary muscular responses to sudden loading. The multi-joint muscles of the forearm, which cross the elbow joint, were also considered.

Hypotheses: Despite maintaining similar postural demands, muscle activations will be affected by changes in body orientation. Furthermore, there will be an increased muscular response with the fluid filled tube as a result of increased task complexity.

Chapter 5

As a follow-up to Chapter 4, a musculoskeletal model was developed to investigate the effects of arm posture and hand loading on elbow joint stability using a JRS analysis.

Purpose: To quantify individual forearm and elbow muscle contributions to joint stiffness at the elbow in preparation for a sudden perturbation.

Hypotheses: Changes in body orientation and hand loading tasks will influence elbow co-contraction, and thus, overall joint stiffness. In particular, the standing posture and fluid hand load combination will provide the greatest muscle co-contraction. Finally, due to the anatomical orientation, the forearm muscles will provide a contribution to JRS at the elbow.

Chapter 6

In Chapter 5, the forearm and elbow muscles were evaluated to understand their contributions to elbow JRS. However, a complete evaluation of forearm muscle contributions to JRS also requires the investigation of these multi-articular muscles to wrist JRS.

Purpose: To document muscle co-contraction as well as individual muscle contributions to wrist JRS in preparation for sudden perturbations. Furthermore, a variety of hand loading demands were evaluated to better understand how the influence of muscular co-contraction would affect the forearm muscle contributions to joint stiffness.

Hypotheses: With an increase in grip force requirement there will be increased forearm muscle co-contraction and overall wrist joint stiffness. The predominant

wrist flexor/extensor muscles (ECR and FCR) will provide the largest individual muscle contributions to JRS. The effect of timing knowledge will influence overall wrist joint stiffness, regardless of the grip force requirement.

In summary, this thesis implements both an *in-vitro* and *in-vivo* approach to examine ligament and muscle contributions to joint stiffness at the elbow and wrist joints. This work quantifies ligamentous mechanical properties and thus, contributions to carpal tunnel and wrist joint stability. It also provides a comprehensive evaluation of muscular contributions to joint rotational stiffness at the elbow and wrist. In particular, the *in-vitro* studies and forearm muscle evaluation at the wrist joint provides a comprehensive analysis of tissue contributions to wrist joint stiffness. This work was extended to the elbow joint, to provide a complete analysis of the forearm musculature. Figure 1.1 provides an overview of the dissertation, illustrating each study's contribution to the understanding of ligament and muscular contributions to joint stiffness in the distal upper extremity.

This work will ultimately provide a foundation for the understanding of musculoskeletal contributions to joint stability in the distal upper extremity. The quantification of individual muscular contributions will aid in the understanding of joint injury due to sudden loading, and provide insight into how posture and loading demands influence how muscles become injured during joint disturbances.

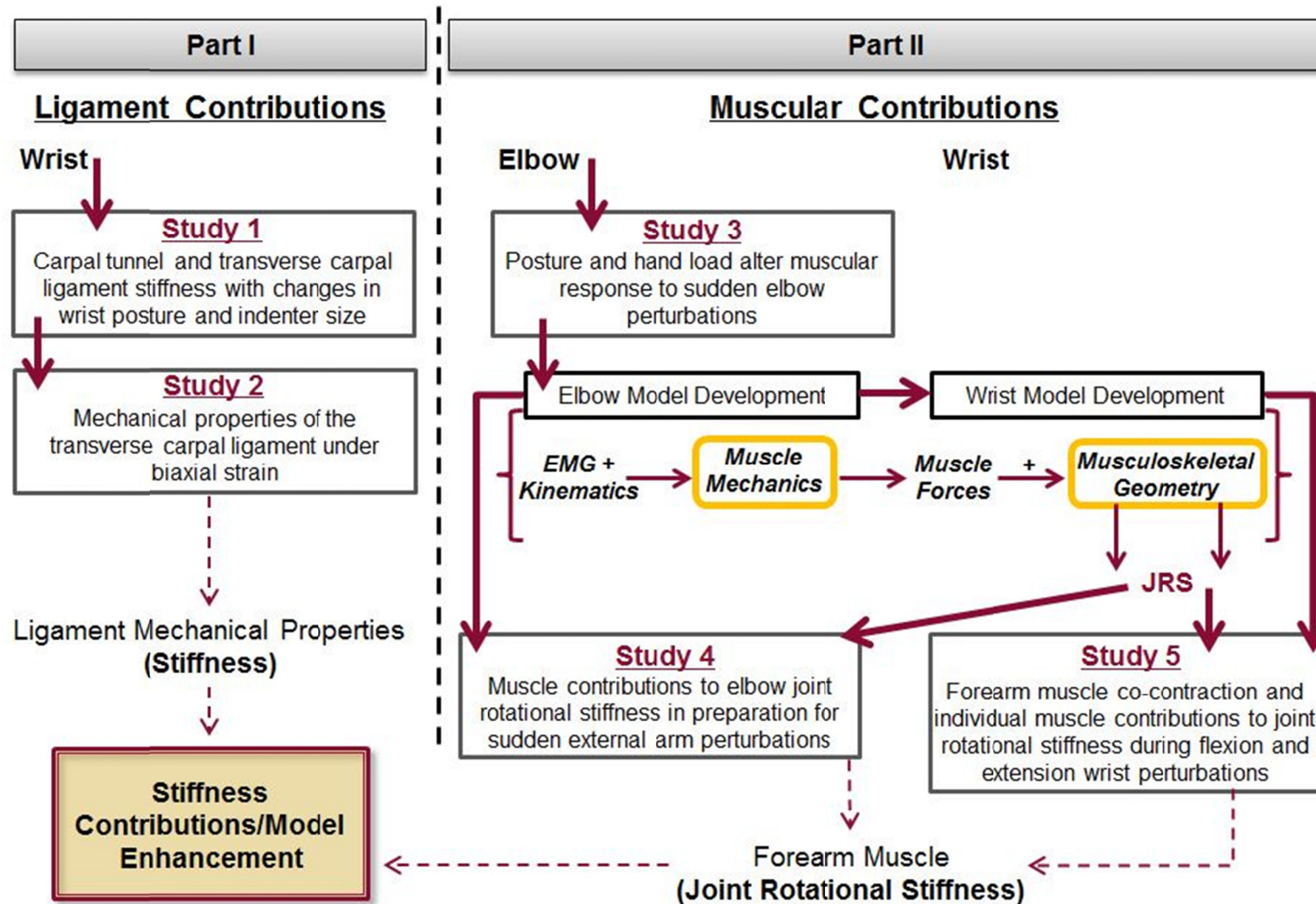


Figure 1.1: Flowchart highlighting the progression of each study towards the quantification of ligament and muscular contributions to joint stiffness in the distal upper extremity.

CHAPTER 2

Study 1: Carpal Tunnel and Transverse Carpal Ligament Stiffness with Changes in Wrist Posture and Indenter Size

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Carpal Tunnel and Transverse Carpal Ligament Stiffness with Changes in Wrist Posture and Indenter Size

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ABSTRACT: This study investigated the effects of loading and posture on mechanical properties of the transverse carpal ligament (TCL). Ten fresh-frozen cadaver arms were dissected to expose the TCL and positioned in the load frame of a servo-hydraulic testing machine, equipped with a load cell and custom made indenters. Four cylindrical indenters (5, 10, 20, and 35 mm) loaded the TCL in three wrist postures (30° extension, neutral and 30° flexion). Three loading cycles with a peak force of 50 N were applied at 5 N/s for each condition. The flexed wrist posture had significantly greater TCL stiffness (40.0 ± 3.3 N/mm) than the neutral (35.9 ± 3.5 N/mm, $p = 0.045$) and extended postures (34.9 ± 2.8 N/mm, $p = 0.025$). TCL stiffness using the 10 and 20 mm indenters was larger than the 5 mm indenter. Stiffness was greatest with the 20 mm indenter, which had the greatest indenter contact area on the TCL. The 35 mm indenter covered the carpal bones, compressed the carpal tunnel and produced the lowest stiffness. The complexity of the TCL makes it an important part of the carpal tunnel and the mechanical properties found are essential to understanding mechanisms of carpal tunnel syndrome. © 2011 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. *J Orthop Res*

Keywords: carpal tunnel; carpal tunnel syndrome; transverse carpal ligament; stiffness; cadaver; wrist

The carpal tunnel is the anatomical space formed by the carpal bones, carpal and transverse carpal ligaments (TCLs). The tunnel is enclosed on the volar side by the TCL and links the anterior forearm to the mid-palmar space allowing passage of the median nerve and nine flexor tendons. The TCL and flexor retinaculum have often been considered synonymous, however, the TCL is distinct as the middle section of the flexor retinaculum and roof of the carpal tunnel.^{1,2} The TCL attaches to the scaphoid and trapezium on the radial side and ulnarly to the pisiform and hook of the hamate. Consisting of thick connective tissue³ and fibers oriented in several directions⁴ the TCL is distinguishable from other tissues making up the flexor retinaculum. The TCL influences tendon, nerve, and bone movement, while also being a component of the flexor tendon pulley system.^{5,6} As a result, the TCL plays an important part in carpal tunnel mechanics, which is evident in investigations that section the TCL.⁷ However, the exact role of the TCL in contributing to carpal stability and carpal tunnel mechanics has been equivocal,^{8,9} emphasizing the need for further evaluation of the ligament's mechanical properties.

Sectioning the TCL has been shown to alter carpal tunnel dimensions and carpal bone movement,⁷ suggesting that the TCL contributes to carpal stability. However, Garcia-Elias et al.⁹ found that the role of the TCL was insignificant in the prevention of abnormal carpal dynamics. They suggested a more likely purpose for the TCL is to withstand large forces, support the metacarpals and protect contents within

the carpal tunnel.⁹ There may be gender differences in TCL properties as Li¹⁰ found female carpal tunnels to be less compliant than males using a manual indentation technique in vivo. Furthermore, the viscoelastic nature of the TCL has prompted an evaluation of non-surgical tunnel expansion via manipulative treatment for carpal tunnel syndrome (CTS) rather than surgical release. Sucher et al.¹¹ demonstrated a 2.6 mm expansion of the carpal tunnel in cadavers when statically loading the TCL for 3 h. Using a different protocol, Li et al.¹² applied palmarly directed forces to the dorsal (interior) surface of cadaveric TCLs and concluded that TCL length was unaffected by loading protocol and arch width narrowing was the result of carpal bone movement.

The mechanics of the carpal tunnel and the TCL are needed to explain aspects of CTS development. For instance, wrist size and posture influence the size and shape of the carpal tunnel^{13,14} and thus influence pressure within the tunnel,^{15,16} potentially leading to compression of the median nerve. Additionally, mechanical compression of the median nerve can result from physical contact with the TCL.¹⁷ Despite links between CTS and carpal tunnel mechanics, little is known about the mechanical properties of the carpal tunnel and TCL. The TCL helps maintain normal function of the wrist and plays a role in defining pressure and compression in the carpal tunnel. Thus, improved knowledge of carpal tunnel and TCL mechanical properties would be beneficial in preventing and rehabilitating CTS.

Previously, a study to document carpal tunnel compliance in vivo used only neutral wrist postures and was not able to address potential confounds between wrist and indenter size.¹⁰ The purpose of the current study was to investigate the mechanical properties of the intact cadaveric carpal tunnel and

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TCL by directly loading the structure in different wrist postures with different size indenters. In addition, carpal bone kinematics were recorded to account for movement. The effects of wrist size and indenter contact area on the mechanical properties of the carpal tunnel were tested using indenters of varying size.

METHODS

Specimen Preparation

Ten fresh-frozen cadaver arms were sectioned at mid-humerus and stored at -20°C . Three male and two female arm pairs with an average age of 75.2 ± 9.3 years were tested. None of the specimens had any known upper extremity musculoskeletal injuries or disorders. Each specimen was thawed overnight at room temperature after which skin and subcutaneous tissues were carefully removed to expose the TCL and its attachment sites. Thenar and hypothenar musculature were removed from the TCL prior to testing. Carpal tunnel contents were left intact.

Data Collection

Following dissection, rigid lightweight flag-like structures with four infrared-emitting diodes were screwed into the pisiform and scaphoid to measure three-dimensional motion of the proximal carpal row (Optotrak Certus, Northern Digital, Inc., Waterloo, Canada). Placements of the rigid structures were confirmed using radiography (Fig. 1). The specimen was then positioned below the compressive actuator of a servo-hydraulic materials testing machine (8872, Instron Canada, Toronto, Canada) equipped with a load cell to which a cylindrical indenter was affixed (Fig. 2A). Four custom made cylindrical indenters were designed with beveled edges and surface diameters of 5, 10, 20, and 35 mm. Each of the four indenters was used to apply loads to the TCL (5, 10, and 20 mm) and carpal tunnel complex (35 mm) in three wrist postures (30° extension, neutral, and 30° flexion). Each indenter was positioned at the mid-point of the ligament



Figure 1. X-Ray image confirming placement of bone pins in the pisiform and scaphoid for carpal bone kinematic measurements.

between the four attachment sites. The approximate area of contact for each indenter is indicated in Figure 2B. PlexiglasTM splints secured the arm in each posture. The hand was attached to the splint using plastic ties across the metacarpals just proximal to the MCP joints. Careful placement of the ties and splint limited tension on the tendons and did not interfere with measuring carpal bone motion.

A loading cycle consisted of the actuator applying a maximum downward force of 50 N at a rate of 5 N/s (adapted from a previous study in vivo,¹⁰ and a maximum load that would not damage the tissue). At the start of testing, 10 preconditioning loading cycles were performed using the 20 mm indenter in the neutral wrist posture. Following preconditioning, three loading cycles were applied in each posture using each indenter in a semi-randomized order (indenter size and posture were randomized but once installed all tests were completed for a given posture). Force, displacement and kinematics of the scaphoid and pisiform were sampled at 100 Hz (Optotrak Data Acquisition, Northern Digital, Inc.). To determine if carpal markers affected indentation, the markers were removed at the end of testing and an additional three loading cycles were performed using a neutral wrist posture and the 20 mm indenter (following 10 additional conditioning cycles). Throughout testing the TCL and surrounding structures were kept moist by spraying with a saline solution (0.91% w/v NaCl).

Data Analysis

Kinematic data were digitally filtered using a low pass Butterworth filter (dual-pass, 2nd order, and 6 Hz cutoff frequency). Prior to data collection, three points on the surface of each bone were digitized around the screw insertion point to provide a fixed spatial relationship between the markers and the bone surface. The centroid of the three digitized points on each bone was determined and the three dimensional vector between the pisiform with the scaphoid was calculated to represent the proximal carpal tunnel width.

Each cycle was divided into loading and unloading curves. Stiffness was measured as the slope of the linear portion of each cycle's load-displacement relationship by means of linear regression (the linear portion was defined by a minimum linear regression R^2 of 0.95). Hysteresis was defined as the area between the loading and unloading curves. All analyses were performed using custom software (MATLAB R2008a, Natick, MA, USA). Actuator displacement and carpal tunnel width was determined at rest (no loading) and at peak loading (50 N). A simplified two-dimensional model at the proximal carpal row was used to represent changes in TCL length assuming linear elastic stretch of the TCL (Fig. 3A). While carpal width was defined as the distance between carpal bone markers, the markers were not inserted at the TCL attachment site, thus a correction was obtained using calipers to measure TCL length from its attachment on the medial border of the scaphoid tuberosity to the pisiform.¹ In this initial analysis, the proximal carpal width was assumed to closely match the midpoint of the carpal tunnel (where the indenter was positioned). The TCL length was used to define the distance from the proximal carpal attachment to the indenter and was assumed to be symmetrical (R_{TCL}). The 35 mm indenter covered the carpal bones and was not included in the calculations. Indenter displacement was measured by the compressive actuator (D_{IND}). All

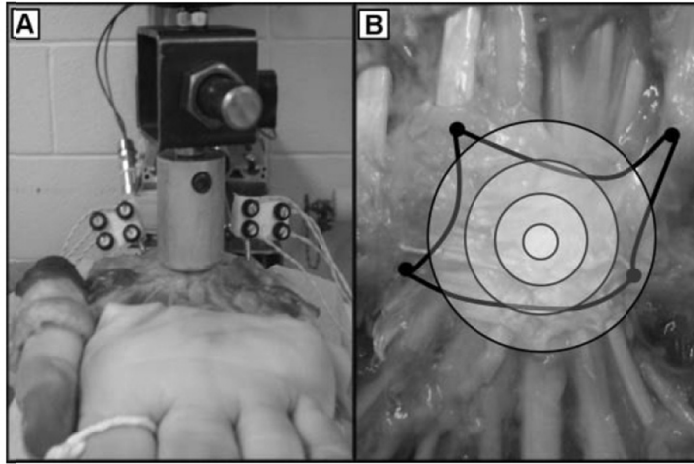


Figure 2. (A) Specimen mounted below compressive actuator of the materials testing machine. Neutral wrist posture and 20 mm indenter shown. (B) Top down view indicating approximate location of each indenter on the TCL (small circle represents 5 mm; large circle represents 35 mm).

displacements were assumed to be due to changes in TCL length from resting length and indenter contact was perpendicular to the ligament. The TCL length at peak indentation (P_{TCL}) was equal to the calculated TCL length on each side of the indenter (L_{TCL}) plus indenter width (W_{IND})

$$P_{TCL} = (2 \times L_{TCL}) + W_{IND} \quad 1$$

Statistical Analysis

Data were averaged across the three trials for each condition. A repeated measures ANOVA was used to evaluate the effect of indenter size, wrist posture, and cycle on indenter displacement and TCL stiffness (SPSS 13.0, Chicago, IL). Significant ANOVA findings were followed up using a least significant difference (LSD) pair-wise comparison. Paired t -tests were used to evaluate stiffness at the start of testing to the conditioning cycles at the end of testing as well as differences due to the placement of kinematic markers. Alpha was set at 0.05.

RESULTS

TCL load-displacement curves exhibited the typical nonlinear toe region followed by a linear elastic region with some hysteresis (Fig. 4). A significant main effect of posture was found for TCL stiffness ($F_{2,20} = 3.87$, $p = 0.038$), with the flexed wrist posture demonstrating greater stiffness (40.0 ± 3.3 N/mm) than neutral (35.9 ± 3.5 N/mm, $p = 0.045$) and extended postures (34.9 ± 2.8 N/mm, $p = 0.025$). A significant main effect of indenter size was also found for TCL stiffness ($F_{3,30} = 5.04$, $p = 0.006$). Stiffness resulting from the 10 and 20 mm indenters were significantly larger than the 5 mm indenter ($p = 0.002$). Stiffness from the 35 mm indenter was significantly less than the 20 mm indenter only ($p = 0.006$). Ligament stiffness using each indenter and posture are found in Figure 5.

A significant main effect of indenter size was found for actuator displacement ($F_{3,30} = 8.16$, $p = 0.0001$),

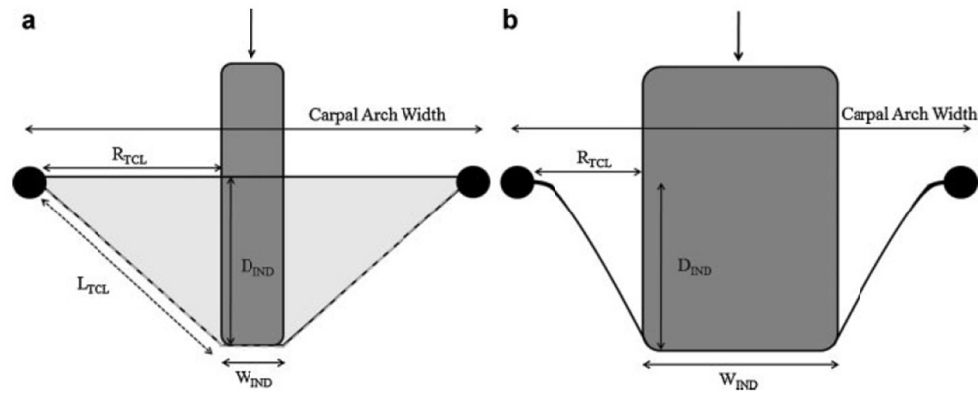


Figure 3. (A) Two-dimensional cross-section of the proximal carpal tunnel used for ligament length calculation. R_{TCL} = length from attachment to edge of indenter; D_{IND} = indenter displacement; W_{IND} = indenter width; L_{TCL} = TCL length on each side of the indenter at peak indentation (50 N load). (B) Theoretical representation of TCL shape during indentation when considering changes in ligament thickness.

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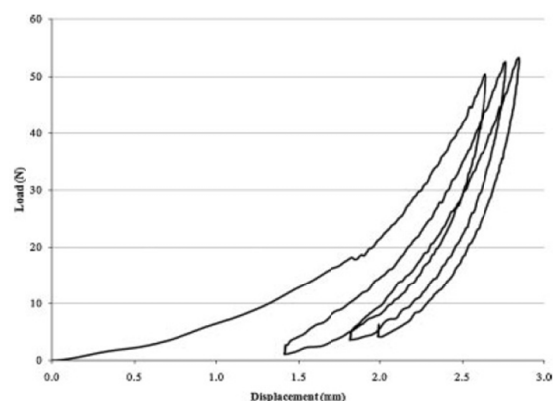


Figure 4. Representative load-displacement curve for three cyclic indentation loads.

due to the 5 mm indenter having greater displacement than the 10 mm ($p = 0.005$), 20 mm ($p = 0.003$), and 35 mm ($p = 0.024$) indenters. Averaged across posture, the 5 mm indenter produced a mean displacement of 2.20 ± 0.19 mm while displacements of 1.83 ± 0.13 , 1.68 ± 0.03 , and 1.82 ± 0.12 mm were found for the 10, 20, and 35 mm indenters, respectively. The flexed posture had less displacement (1.70 ± 0.45 mm) than extension (1.8 ± 0.44 mm) and neutral (2.0 ± 0.50 mm), however this was not significant.

There were no differences between the mean hysteresis for the 10 conditioning cycles before testing (22.8 ± 5.6 N/mm) and the 10 cycles at the end of testing (22.9 ± 5.0 N/mm).

There was no difference in TCL stiffness with or without markers ($p = 0.157$). There was also no difference in carpal width for resting (29.3 ± 0.5 mm) and peak loading (28.7 ± 0.4 mm) ($p = 0.638$). The mean resting TCL length was 20.7 ± 1.8 mm. When averaged across posture, the calculated TCL length (P_{TCL}) at peak loading was 22.1 ± 0.3 , 22.1 ± 0.3 , and

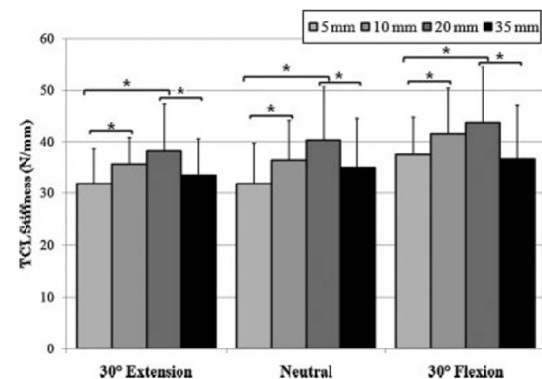


Figure 5. Mean TCL stiffness and SD (N/mm) for indenter size during each wrist posture. The level of significance is noted for individual comparisons, * $p < 0.05$.

23.7 ± 0.8 mm for the 5, 10, and 20 mm indenters, respectively. This represents an average change of 0.58, 0.60, and 2.19 mm for the 5, 10, and 20 mm indenters, respectively. There were no differences in the calculated TCL length when compared across postures. The 35 mm indenter covered the carpal bones, thus TCL length was not calculated.

DISCUSSION

This study investigated the mechanical properties of the TCL by applying cyclic loads with four indenter sizes and three wrist postures. This was the first study to simultaneously test posture and indenter size and we found that our measurements of TCL stiffness were dependent on both. In each posture, ligament stiffness was greatest with the 20 mm indenter which was 21% greater than using the smallest (5 mm) indenter. Interestingly, the largest indenter resulted in lower stiffness than the 20 mm indenter. Given that the large indenter contacted the carpal bones, this suggests that the ligament itself may be stiffer than the bony arch. By measuring kinematics of the carpal bones, this study also showed no significant change in carpal width under load, thus the TCL was stretched in isolation. These findings indicate that, under the conditions tested, indenter size and posture must be considered when evaluating the properties of the carpal tunnel.

In the current study, as indenter size increased, it progressively covered more of the TCL. In a companion study, we found that the radial and ulnar portions of the TCL were thicker than the middle section.¹⁸ This is in agreement with recent work that found the TCL was thicker distally on the ulnar side and proximally on the radial side.² Thus our indenters progressively encroached on the thicker side portions of the ligament, resulting in stiffness increasing from the 5 to 20 mm indenter. Thicker sections of the TCL would be more resistant to stretch and partly explains our small carpal bone movements. The 35 mm indenter spanned the carpal width and compressed the carpal tunnel structure, making a comparison with the other indenters difficult since loading was not applied directly to the TCL (like the smaller indenters). However, the largest indenter (35 mm) produced the lowest stiffness and likely represents stiffness of the carpal tunnel complex rather than the ligament itself as the indenter covered the carpal bones (TCL attachment sites, Fig. 2B) and would have compressed the structure. The 5 mm indenter had the greatest indentation depth (actuator displacement) and lowest stiffness of all other indenters. Due to its small contact area, it acted as more of a point load than the other indenters, generating greater local stress at the indenter-ligament interface. Our finding that indenter contact area affects stiffness measures is important as female carpal tunnels have been reported to be stiffer than males using a manual indentation test with a 10 mm

cylindrical indenter.¹⁰ Given that women tend to have smaller wrists than men,¹³ the relationship of wrist size to indenter size may partially explain some of these findings. Li¹⁰ used a single indenter size, and the greater stiffness for female participants may partly be attributed to the indenter covering a larger portion of the wrist than male participants. In theory, this would have a similar effect to increasing indenter contact area. The increase in stiffness with indenter size (from 5 to 20 mm) found in the current study may partly explain some of the gender differences in Li,¹⁰ since the indenter used in that study would cover more of the carpal arch in smaller (i.e., female) wrists. In the current study, we could not test the effect of sex due to small sample size. However, the largest indenter (35 mm) would have contacted varying degrees of the carpal bone structure depending on wrist size and resulted in lower stiffness. Thus interpretation of the 35 mm indenter results differs from the other indenters as it represents compression of the osseous structure rather than strain in the TCL.

Our TCL stiffness measurements were approximately three times larger than the 10 N/mm found by Li.¹⁰ The larger indentation force in our study appears to account for the difference. Li¹⁰ provided a maximum indentation force of 19.6 N and, as is evident from Figure 4, a larger load was required to reach the linear region in our force–displacement data. Additionally, cadaveric testing of the TCL allowed for the removal of superficial tissues, producing indentation in direct contact with the ligament, and ligament tensile properties have been shown to be unaffected by freezing.²¹

Posture related changes in carpal tunnel dimensions have been shown to influence pressure within the carpal tunnel.¹⁶ Posture had a small but significant effect on TCL stiffness. With the wrist flexed, stiffness was 11% and 14% greater than the neutral and extended postures, respectively (Fig. 4). The greater stiffness could be, in part, attributed to the arrangement of the flexor tendons. In flexion, a slight narrowing of the carpal tunnel may cause a tendon arrangement that provides some lateral support to the carpal arch, potentially influencing our measurements. We did not see a significant change in resting carpal width with posture. It is likely that wrist postures greater than 30° will cause movement of the carpal bones¹⁹ and change the resting TCL length (i.e., length before indentation). Because the scaphoid and pisiform are considered mobile carpal bones²⁰ we expected a change in proximal carpal width during loading. However, the splints used in the present study held the cadaver arm rigidly in place throughout testing, likely limiting carpal bone movement due to posture and isolating the TCL in the three wrist postures.

We determined TCL length changes of up to a 2.1 mm at peak (50 N) load using the 20 mm indenter. This was based on the TCL resting lengths calculated for each specimen and represents a substantial 10.3% increase in length. Our calculation was similar to the

approach of Li et al.¹² who suggested carpal tunnel expansion could be achieved by elongating the TCL and/or by carpal bone movement. Using palmarly directed loads of up to 200 N from within the carpal tunnel, Li et al.¹² found no TCL elongation and suggested that changes in carpal arch width must be due to movement of the carpal bones. At a force of 50 N, as used in the current study, they reported an arch width (between the radial and ulnar edges at the midpoint of the TCL) increase of about 1.5 mm compared to our non-significant 0.6 mm increase (at the proximal carpal row). The differences in force application between our study and Li et al.'s¹² could contribute to these width differences. It is possible that the modest carpal width change may play a role but it appears, based on the current study that TCL lengthening contributes more greatly to changes in carpal tunnel shape. Li et al.¹² simulated the two methods of carpal arch formation (bone movement and TCL elongation) and found that a 1.0 mm increase in TCL length or decrease in arch width both caused a greater than 20% increase in total carpal tunnel area.

There are a few limitations to the current study. Our methods did not isolate the TCL from other carpal tunnel structures. The contents of the carpal tunnel were left intact to replicate in vivo loading and provide an understanding of carpal tunnel stiffness as an intact structure, while a companion study (Keir et al.¹⁸) investigated mechanical properties of the TCL in isolation. While the cadaveric specimens were considered free from wrist injuries, standard concerns about cadaveric tissues apply, including the age of the donors. Our tests indicated no changes in hysteresis or stiffness throughout the study suggesting the TCL stiffness measured throughout out testing protocol was repeatable and unaffected by the frequent cyclic loading. In addition, freezing of specimens has been shown to have no affect on the biomechanical properties (tensile behavior) of ligaments.²¹ The TCL length calculation used in the current study is somewhat limited. First, while indentation with a circular object should cause changes in both length and width of the TCL, we have only considered width changes. Loading was concentrated at the midpoint of the TCL, suggesting equal distribution of the load over the ligament and a linear approximation was used as a minor simplification that reduced variability in TCL length calculations. Second, we assumed a linear change for our calculations of length (similar to Li et al.¹²). A more realistic TCL shape change during indentation which considers the variation in ligament thickness is represented in Figure 3B. Finally, ligament thickness was not considered.

One use of data from the current study is to improve models of the carpal tunnel.¹⁴ The TCL material properties from this study will improve our ability to predict changes in carpal tunnel shape and pressure.¹⁴ Guo et al.⁷ developed an elegant finite element model of the carpal tunnel and modeled the

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TCL as an isotropic material. This is one of the first models to include the TCL and material properties with the purpose of measuring carpal bone movement during the simulation of TCL release. We found changes in TCL mechanical properties with both indenter contact area and wrist posture. These findings show that the mechanical properties of the TCL are different throughout the ligament and modeling the ligament with mechanical properties for different regions may improve the anatomic fidelity of such models. In addition to indenter contact area and wrist posture, the differences in mechanical measures are influenced by changes in TCL thickness at different locations,^{2,18} fiber orientation⁴ and carpal stability. Quantifying TCL thickness at different locations during indentation would be difficult. However, potential errors in stiffness measurement from smaller indenters would be less, and small contact area will better represent loading from internal structures. In addition, the investigation of force transmission and flexor tendon excursions could benefit from these results as the mechanical characteristics of the flexor pulley system (created by the TCL) will be influenced by its material properties.⁶

This study found that our characterization of the TCL changed with changes in indenter contact area and wrist posture. These findings should be considered when evaluating mechanical properties of the carpal tunnel. The measured stiffness changed with indenter size and is important for evaluating differences in stiffness due to carpal tunnel size. Our finding that TCL stiffness differs between wrist postures may be used to improve predictions of posture related changes to the carpal tunnel. Furthermore, the role the TCL plays in carpal tunnel mechanics has been debated and remains uncertain. We found limited movement of the proximal carpal row during our indentation tests, which suggest that changes in carpal tunnel shape were predominantly due to TCL lengthening and not carpal bone movement. This study has documented changes in TCL mechanical properties and the calculated length changes confirm the ligaments importance in carpal tunnel mechanics. The results from the current study will help improve our understanding of the mechanisms of CTS and the flexor tendon pulley system through better knowledge of the material properties of the TCL.

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CHAPTER 3

**STUDY 2: MECHANICAL PROPERTIES OF THE TRANSVERSE
CARPAL LIGAMENT UNDER BIAXIAL STRAIN**

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3.1 Abstract

The transverse carpal ligament (TCL) influences carpal stability and carpal tunnel mechanics, yet little is known about its mechanical properties. The purpose of this study was to investigate the mechanical properties of the TCL in different regions. The TCL was extracted from eight cadaver arms and each ligament was divided into six tissue samples from the following locations; distal radial, distal middle, distal ulnar, proximal radial, proximal middle and proximal ulnar. Five and 15% strains were applied biaxially to each sample at rates of 0.1, 0.25, 0.5 and 1 %s⁻¹. Measures evaluated included thickness, maximum stress and linear elastic modulus. Ligament thickness ranged from 1.22 to 2.90 mm across all samples. Samples from the middle of the TCL were significantly thicker proximally than distally ($p < 0.013$). Tissue location had a significant effect on elastic modulus ($p < 0.001$). Modulus was greatest in the proximal radial samples (mean 2.8 MPa) which were 64% and 44% greater than the distal radial and proximal ulnar samples, respectively. Samples from the middle of the ligament had a modulus that was 20% to 39% greater in proximal versus more distal samples. The TCL exhibits different mechanical properties within different locations. The contribution of the TCL to carpal tunnel mechanics will vary depending on the area of interest within the carpal tunnel. In particular, greater moduli were found near the attachment sites on the carpal bones. These mechanical properties contribute to the understanding of carpal tunnel mechanics which is critical to understanding disorders of the wrist such as carpal tunnel syndrome.

Keywords: Transverse Carpal Ligament, Carpal Tunnel, Cadaver, Mechanical Properties, Elastic Modulus

3.2 Introduction

Carpal tunnel syndrome (CTS) is the most common peripheral compression neuropathy and has a large financial burden to the economy (Atroshi et al., 1999; Foley et al., 2007; Manktelow et al., 2004). The confined area making up the carpal tunnel is enclosed on the volar side by the transverse carpal ligament (TCL). The TCL has been reported to help maintain carpal stability (Fisk, 1984), protect contents of the carpal tunnel (Garcia-Elias et al., 1989a, b) and act as a component of the flexor tendon pulley system (Kline and Moore, 1992; Wehbe, 1993). However, evidence for the exact role of the ligament is lacking, due in part, to its geometry and a fibre arrangement that runs in multiple directions from the four carpal bones to which the ligament attaches (Mashoof et al., 2001; Pacek et al., 2009). A recent study showed that the thickness of the TCL varies throughout the tissue (Pacek et al., 2009), which strongly suggests that investigation of the TCL mechanical properties at different locations is warranted. To date, the mechanical properties of the TCL have only been determined as an intact system, thus distinction between the ligament and the carpal tunnel as a complex has not been achieved.

The mechanical properties of the carpal tunnel have received considerable attention recently, with carpal tunnel stiffness, or compliance, being measured *in vivo* (Li, 2005; Zheng et al., 2006), in cadaveric specimens (Holmes et al., 2010; Li et al., 2009; Tengrootenhuysen et al., 2009) and animal models (Tung et al., 2010). However, these studies focused on the TCL as part of the carpal tunnel complex. In a companion study (Holmes et al., 2010), we found that TCL elongation and carpal tunnel stiffness

measures, from load controlled indentation tests performed on cadaveric wrists, were dependent on both indenter contact area and wrist posture. While the carpal tunnel is often tested as a unit, the contribution of the TCL to the carpal tunnel response is unknown and may behave differently. Pacek et al. (2009) showed that TCL thickness was greatest near its four attachment sites. These findings suggest that the mechanical properties of the TCL also likely vary throughout the tissue, influencing carpal tunnel mechanics, as well as ligament interaction with the flexor tendons and median nerve.

Research on mechanical properties of the TCL at different locations of the carpal tunnel is limited. Xiu and Li (2010) applied medial and lateral (inward, outward) forces to the carpal bones at the distal and proximal levels of the carpal tunnel. They found TCL compliance at the proximal level was 3.2 to 4.3 times greater than at the distal level, and suggested that the carpal tunnel is more flexible at the proximal end. Stuchin (1992) also found that the distal carpal bones were less mobile than those in the proximal row. Additionally, the TCL has been found to be thicker distally (Cobb et al., 1993; Pacek et al., 2009) which likely accounts for variance in carpal tunnel compliance. These studies suggest that specific regions of the TCL vary in stiffness/compliance but, to date, this has not been examined in isolation from other constituent structures of the carpal tunnel.

Manipulative treatment has been proposed as a non-surgical option for rehabilitation of CTS (Moraska et al., 2008; Sucher et al. 2005). The success of therapeutic treatment would benefit from a better understanding of carpal tunnel stiffness and, thus, TCL mechanical properties. Knowing the mechanical properties of the TCL throughout its expanse will improve our understanding of the ligament's role in carpal

tunnel mechanics and its interactions with the flexor tendons and median nerve. These data could lead to major improvements in predictive simulations and models of the carpal tunnel, which would help our understanding of disorders of the wrist, such as CTS (Mogk and Keir, 2007; Mogk and Keir 2009). The purpose of this study was to measure mechanical properties of the cadaveric TCL at six different locations using a biaxial tensile testing method.

3.3 Methods

3.3.1 Ligament Preparation

The TCL was extracted from eight fresh-frozen cadaver arms, representing two male (70 years and 80 years) and two female (62 years and 71 years) pairs. Medical records indicated no musculoskeletal injuries or disorders of the upper extremity for any of the cadavers. Each TCL was stored at -20°C and thawed overnight at room temperature the night before testing, as this process has been shown to have no effect on the tensile properties of ligaments (Woo et al., 1986).

3.3.2 Experimental Protocol

Each excised TCL was cut into proximal and distal segments which were further subdivided into radial, middle and ulnar portions (Figure 3.1). This resulted in 6 tissue samples per excised TCL: distal radial (1), distal middle (2), distal ulnar (3) and proximal radial (4), proximal middle (5), and proximal ulnar (6). A 5 mm x 5 mm sample was taken from each portion (Figure 3.2). After the thickness of each sample was measured at the midpoint using a calibrated laser device (ZX-LD40L Smart Sensor, Omron Canada

Inc., Toronto, ON, resolution = 2 μm), each tissue sample was mounted in a biaxial tensile testing system (BioTester 5000, CellScale, Waterloo, Canada) (Figure 3.3). The tissue was mounted and secured using small rakes. The TCL has fibres running in many directions (Isogai et al., 2002), but each small sample was positioned in the system such that the predominant orientation of the fibres were along the X axis (Figure 3.3). Samples were positioned such that rakes were symmetrically attached to each side of the sample to apply tensile strain through a pair of actuators on each side (i.e. 2 orthogonal pairs). Force applied to the tissue sample was measured by a uniaxial load cell positioned in series with each actuator and had a maximum capacity of 5 N. Strain was applied simultaneously at the same rate, and to the same target strain in both X and Y directions for all biaxial tensile tests.

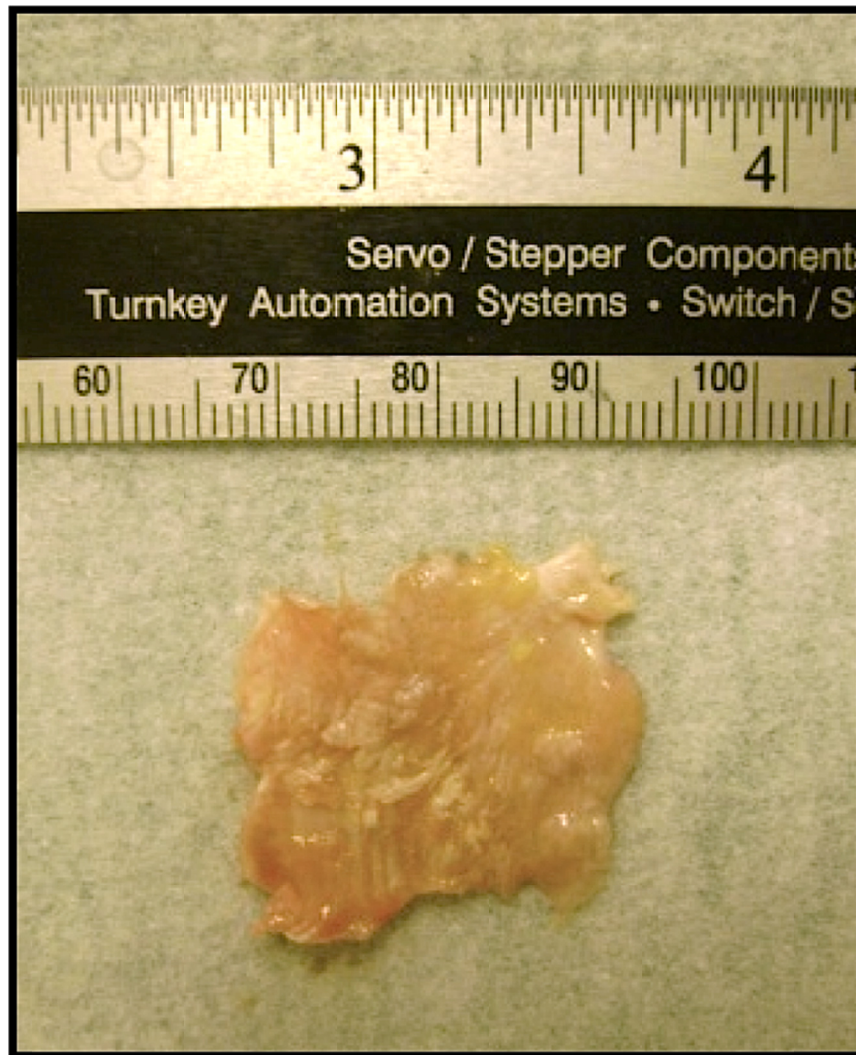


Figure 3.1 TCL extracted from cadaver.

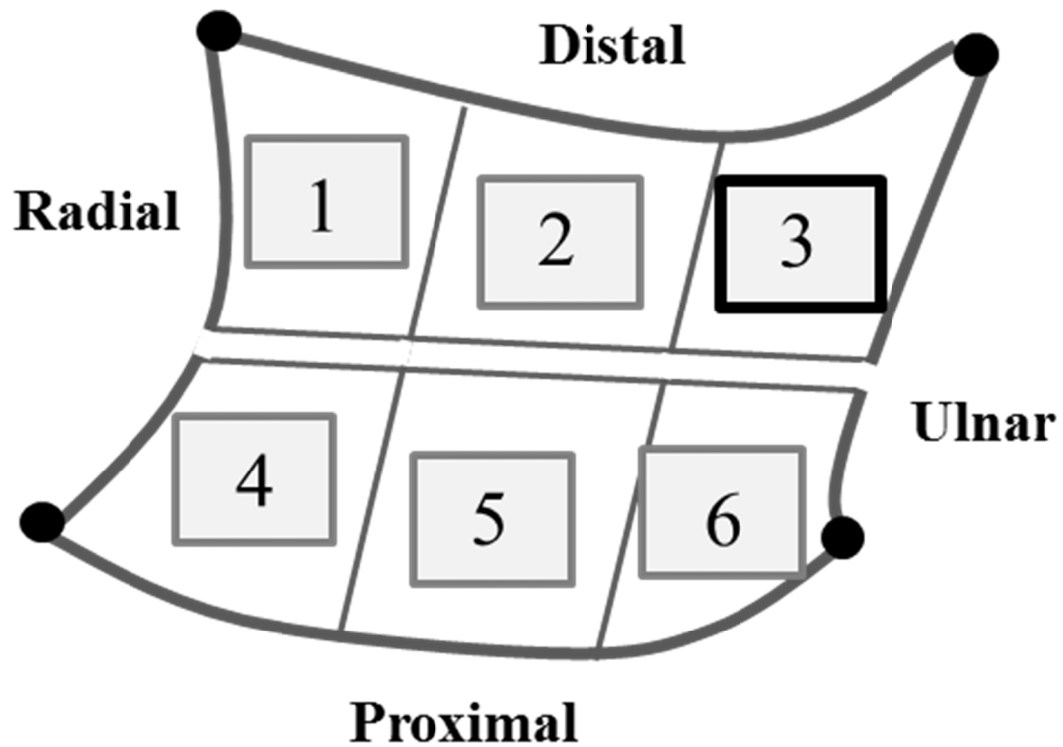


Figure 3.2 Outline of the TCL with attachment sites indicated by black circles. Numbered boxes represent locations of each 5 mm x 5 mm sample. Distal radial (1), Distal middle (2), Distal ulnar (3), Proximal radial (4), Proximal middle (5), Proximal ulnar (6).

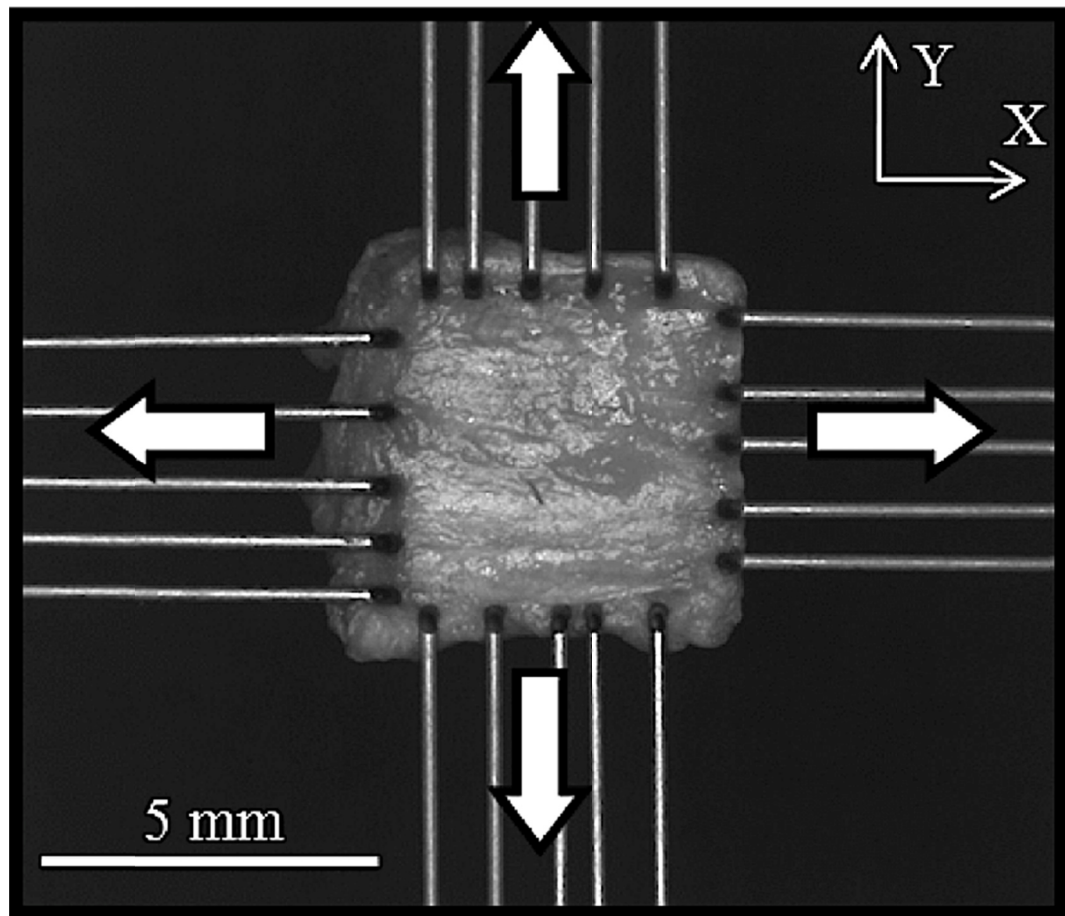


Figure 3.3 Tissue sample (location 3 in figure 3.2) mounted in the biaxial tensile system. Small rakes on each side secure the sample in place. X and Y orientation is noted in upper right corner.

Once the sample was secured in the biaxial tensile system, each tissue sample was preconditioned with 3 cycles to a maximum of 5% strain at a constant strain rate of $1\text{ }\%s^{-1}$. Following preconditioning, each tissue sample underwent 6 tests, all of which simultaneously produced strain in the X and Y directions. Initially, sets of 3 loading cycles to 5% strain at four specified strain rates were performed in random order. The 4 strain rates were $0.1\text{ }\%s^{-1}$ (loading duration = 50 seconds), $0.25\text{ }\%s^{-1}$ (loading duration = 20 seconds), $0.5\text{ }\%s^{-1}$ (loading duration = 10 seconds) and $1\text{ }\%s^{-1}$ (loading duration = 5 seconds). After these tests to a maximum of 5% strain were completed, two additional tests to a maximum strain of 15% were performed. Each of these tests consisted of a single loading cycle, and was randomly conducted at tensile strain rates of $1\text{ }\%s^{-1}$ or ‘high strain’ (loading duration = 15 seconds), and $0.25\text{ }\%s^{-1}$ or ‘low strain’ (loading duration = 60 seconds). During testing the sample was kept moist by lightly spraying with saline solution (0.91% w/v NaCl).

3.3.3 *Data Analysis*

Biaxial force and displacement in the X and Y directions were synchronized and digitally sampled at a rate of 10 Hz using the manufacturer provided software (LabJoy 5.80, Cellscale, Waterloo, ON, Canada). Stress was calculated at each time point by dividing the measured forces from each load cell by the sample’s measured cross-sectional area. A constant sample thickness was assumed when determining stress for each of the tensile tests. Cross-sectional area was determined using the previously measured sample thickness and the tissue length that was perpendicular to the corresponding direction of applied force. Tissue length (for X and Y directions) were

defined as the distance between rakes on opposing sides of the sample. The original width in each direction was defined as the separation distance between directly opposed rakes when no tension was applied to the mounted sample. Strain was calculated by normalizing the instantaneous specimen width, measured as the distance between directly opposite rakes throughout the tensile tests, to its original (starting) specimen width. For each condition, stress-strain profiles were determined for both X and Y directions. Elastic modulus was determined by taking the slope of the linear portion of the stress-strain curve (beyond the non-linear toe region). The linear portion started at approximately 85% of the maximum strain. The maximum stress was also determined during each loading cycle. Each of these measures was determined using custom software (MATLAB R2008a, Natick, MA, USA).

3.3.4 Statistical Analysis

Trials with a peak strain of 5%, at strain rates of 0.1 %s⁻¹, 0.25 %s⁻¹ and 0.5 %s⁻¹, remained within the non-linear toe region of the stress-strain curve and statistical analyses were not performed. For each of the three remaining strain rate conditions (5% at 1 %s⁻¹, 15% at 1 %s⁻¹ and 15% at 0.25 %s⁻¹) three separate 2 x 6 repeated measures analysis of variance (ANOVA) were used to evaluate the effect of direction (X and Y) and tissue location (distal radial, distal middle, distal ulnar, proximal radial, proximal middle, and proximal ulnar) on elastic modulus and maximum stress. A one-way repeated measures analysis of variance (ANOVA) was used to evaluate the effect of tissue location on the measured tissue thickness (SPSS 13.0, Chicago, IL). Significant

main and interaction effects were compared with a Tukey's HSD post hoc test. A significance level of 0.05 was used for all tests.

3.4 Results

Mean TCL thickness across all samples was 2.14 ± 0.31 mm, ranging from 1.22 mm to 2.90 mm (Table 3.1). Tissue location (i.e. sample) had a significant main effect on TCL thickness ($F_{2,25} = 3.6$, $p = 0.014$). Proximal samples of the TCL were consistently thicker (by a mean of 0.48 mm) than distal samples (Table 3.1). The middle samples were significantly thicker proximally than distally ($p < 0.013$; Table 3.1, location 5 vs. 2). Ulnar portions of the TCL were also significantly thicker proximally ($p < 0.048$) but no significant difference was found on the radial side. There was a trend towards the distal radial and distal ulnar samples (locations 1 and 3) being thicker than the distal middle (location 2) samples by approximately 0.3 mm

Trials performed to maximum specified strains of 5% and 15% at a strain rate of $1\ \%s^{-1}$ and maximum strain of 15% strain at $0.25\ \%s^{-1}$ always reached the linear elastic region of the stress-strain curve. A typical stress-strain profile of one cycle for 15% strain at $1\ \%s^{-1}$ is found in Figure 3.4. The X and Y strain directions produced similar stress-strain profiles in terms of elastic moduli, maximum stress and length of the toe region. At 15% strain only the proximal radial and proximal middle samples had greater moduli in the Y direction than the X direction (average of 0.41 MPa).

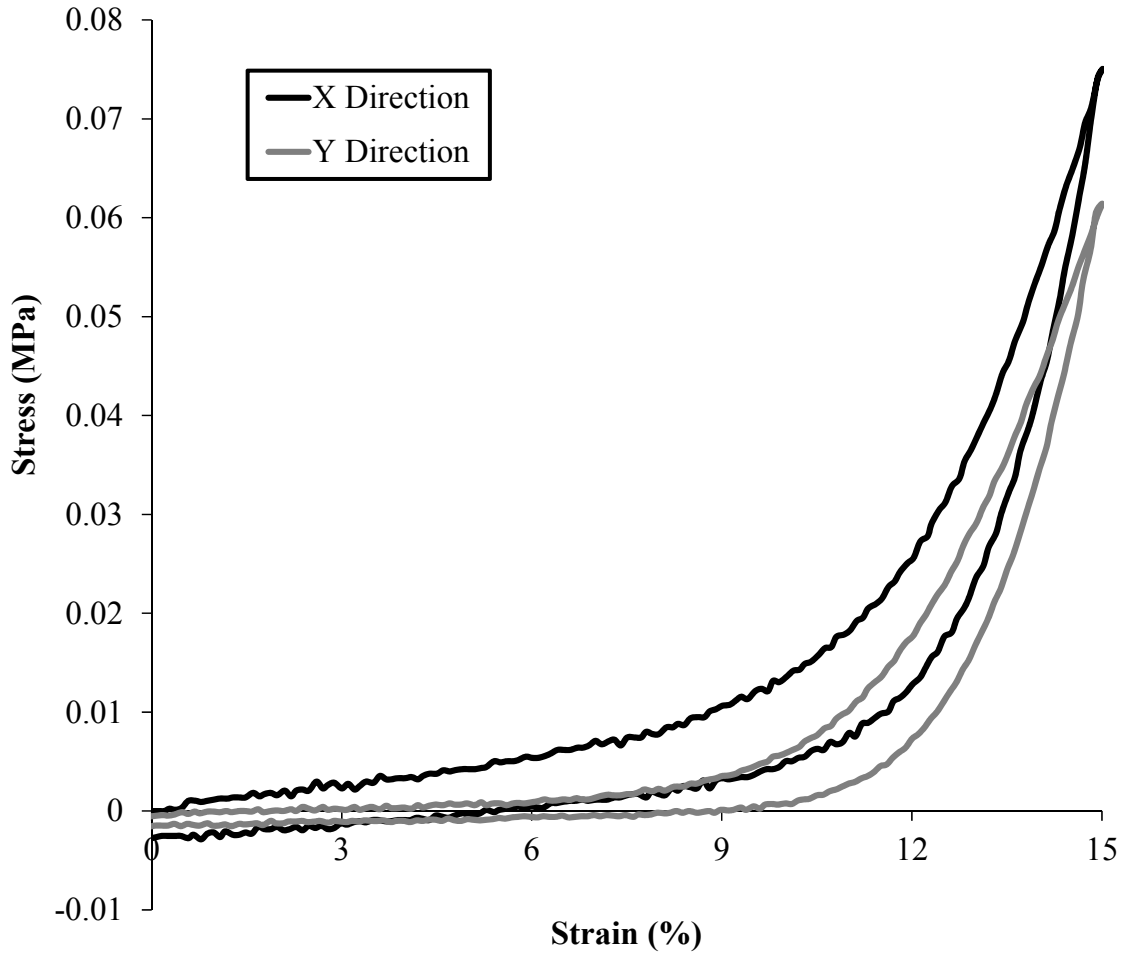


Figure 3.4 Stress-Strain profiles for a tissue sample during 15% strain ($1\ \%s^{-1}$). Black line represents the X direction; Grey line represents the Y direction. Slight negative stress is a consequence of the testing protocol. The rakes were programmed to pull and then return to their exact starting position. This may have caused the tissue to sag in between the rakes, which would pull and cause a slight negative force.

For tests conducted to a maximum of 5% strain ($1\ \%s^{-1}$), there was a significant location x direction interaction ($F_{5,25} = 9.1$, $p = 0.001$). Post-hoc analysis revealed that the proximal radial samples (location 4) had significantly higher elastic moduli than all other locations ($p < 0.05$, both X and Y directions). The proximal radial samples had greater elastic moduli than all other sample locations, with a mean modulus of 0.98 ± 1.16 MPa and 0.78 ± 0.92 MPa in X and Y, respectively. This corresponded to 1.8 (X) and 2.5 (Y) times greater moduli than the next largest (proximal middle) samples. A summary of elastic modulus and maximum stress for all tissue sample locations, along with all statistical comparisons of tissue location for elastic modulus can be found in Table 3.2 for the X strain direction and Table 3.3 for the Y strain direction.

For tests conducted to a maximum of 15% strain, both high and low strain rates ($1\ \%s^{-1}$ and $0.25\ \%s^{-1}$) also demonstrated a significant location x direction interaction ($F_{5,25} = 14.3$, $p = 0.001$ and $F_{5,25} = 16.2$, $p = 0.001$ for $1\ \%s^{-1}$ and $0.25\ \%s^{-1}$, respectively). Modulus was significantly greater in the proximal middle samples than the distal middle samples and this was true for strain rate ($1\ \%s^{-1}$ and $0.25\ \%s^{-1}$) and strain direction (X and Y) (all $p < 0.05$). In the X direction, moduli for the proximal middle samples during the low (mean of 1.22 ± 0.51 MPa) and high (mean of 1.12 ± 0.76 MPa) strain rate conditions were 20% and 39% greater than the distal middle samples, respectively (Table 3.2). In the Y direction, the mean modulus was approximately 65% greater in the proximal middle samples than the distal middle samples (Table 3.3).

For TCL portions close to the attachment sites (i.e. locations 1, 3, 4 and 6), the proximal radial samples had significantly greater moduli than the distal radial samples (p

< 0.01, all strain rates and directions). The proximal radial samples also had significantly greater moduli than the distal ulnar samples (all $p < 0.05$). The greatest modulus was found during the 15% low strain rate condition where the proximal radial samples were 2.76 and 2.77 MPa for the X and Y directions, respectively (Tables 3.2 and 3.3, respectively). This related to a 64% increase in modulus when compared to the distal radial samples and a 44% increase when compared to the proximal ulnar samples.

The maximum stress produced during all strain conditions was less than 0.07 MPa, primarily due to the small force range tested and the thick samples with large cross sectional areas. The statistical results for maximum stress followed closely with elastic modulus for statistical comparisons of tissue location and strain rate.

Table 3.1: Mean tissue thickness \pm standard deviation at each tissue location. Numbers 1-6 represent tissue locations highlighted in Figure 1b (N=8).

Tissue Sample Location			Thickness (mm \pm SD)
Distal	1	Radial	1.97 \pm 0.32
	2	Middle	1.68 \pm 0.32
	3	Ulnar	1.99 \pm 0.24
Proximal	4	Radial	2.30 \pm 0.31
	5	Middle	2.39 \pm 0.21
	6	Ulnar	2.40 \pm 0.40
Mean \pm SD			2.14 \pm 0.31

Table 3.2: Mean (standard deviation) elastic modulus (MPa), maximum stress (MPa) and statistical comparisons of elastic modulus for each tissue sample location in the X strain direction. Note: Numbers found under statistical comparison represents significant differences ($p < 0.05$) between TCL sample locations within each of the three strain conditions. (i.e. for 5% strain, location 2 - distal middle was significantly different than location 4 - proximal radial and 5 - proximal middle).

Tissue Sample Location			Elastic Modulus (MPa)	Maximum Stress (MPa)	Statistical Comparison
<i>5% Strain ($1\ \%s^{-1}$)</i>					
Distal	1	Radial	0.13 (0.06)	0.002 (0.001)	4
	2	Middle	0.18 (0.02)	0.002 (0.003)	4,5
	3	Ulnar	0.20 (0.07)	0.003 (0.001)	4,5
Proximal	4	Radial	0.98 (1.16)	0.013 (0.01)	1,2,3,5,6
	5	Middle	0.54 (0.08)	0.01 (0.003)	2,3,4,6
	6	Ulnar	0.28 (0.10)	0.004 (0.001)	4,5
<i>15% Strain ($1\ \%s^{-1}$)</i>					
Distal	1	Radial	0.72 (0.54)	0.026 (0.012)	3,4,6
	2	Middle	0.68 (0.65)	0.021 (0.002)	3,4,5,6
	3	Ulnar	1.62 (1.19)	0.033 (0.02)	1,2,5
Proximal	4	Radial	1.52 (0.10)	0.056 (0.005)	1,2,5,6
	5	Middle	1.12 (0.76)	0.052 (0.02)	2,3,4,6
	6	Ulnar	1.37 (0.95)	0.04 (0.03)	1,2,4,5
<i>15% Strain ($0.25\ \%s^{-1}$)</i>					
Distal	1	Radial	1.03 (0.82)	0.034 (0.012)	4,6
	2	Middle	0.98 (0.31)	0.033 (0.02)	3,4,5,6
	3	Ulnar	1.55 (0.40)	0.04 (0.014)	2,4,6
Proximal	4	Radial	2.76 (0.34)	0.07 (0.02)	1,2,3,5,6
	5	Middle	1.22 (0.51)	0.05 (0.01)	2,4,6
	6	Ulnar	1.63 (1.20)	0.042 (0.022)	1,2,3,4,5

Table 3.3: Mean (standard deviation) elastic modulus (MPa), maximum stress (MPa) and statistical comparisons of elastic modulus for each tissue sample location in the Y strain direction. Note: Numbers found under statistical comparison represents significant differences ($p < 0.05$) between TCL sample locations within each of the three strain conditions. (i.e. for 15% strain, location 1 - distal radial was significantly different than location 4, 5 and 6).

Tissue Sample Location			Elastic Modulus (MPa)	Maximum Stress (MPa)	Statistical Comparison
<i>5% Strain ($1\ \%s^{-1}$)</i>					
Distal	1	Radial	0.20 (0.12)	0.003 (0.001)	4
	2	Middle	0.19 (0.09)	0.002 (0.001)	4
	3	Ulnar	0.21 (0.04)	0.004 (0.001)	4
Proximal	4	Radial	0.78 (0.92)	0.009 (0.003)	1,2,3,5,6
	5	Middle	0.31 (0.19)	0.005 (0.003)	4
	6	Ulnar	0.29 (0.18)	0.004 (0.001)	4
<i>15% Strain ($1\ \%s^{-1}$)</i>					
Distal	1	Radial	0.67 (0.14)	0.02 (0.001)	4,5,6
	2	Middle	0.52 (0.11)	0.01 (0.01)	4,5,6
	3	Ulnar	0.57 (0.30)	0.02 (0.007)	4,5,6
Proximal	4	Radial	1.77 (0.77)	0.07 (0.013)	1,2,3
	5	Middle	1.45 (0.51)	0.04 (0.01)	1,2,3
	6	Ulnar	1.23 (0.59)	0.04 (0.02)	1,2,3
<i>15% Strain ($0.25\ \%s^{-1}$)</i>					
Distal	1	Radial	0.99 (0.03)	0.03 (0.003)	4,5
	2	Middle	0.78 (0.17)	0.03 (0.001)	4,5,6
	3	Ulnar	0.64 (0.22)	0.023 (0.011)	4,5,6
Proximal	4	Radial	2.77 (1.08)	0.07 (0.02)	1,2,3,6
	5	Middle	2.25 (0.84)	0.05 (0.01)	2,3,6
	6	Ulnar	1.55 (0.77)	0.04 (0.01)	2,3,4,5

3.5 Discussion

This is the first study to use biaxial strain testing of isolated TCL samples at sequential locations throughout the ligament. By isolating portions of the TCL, we found the ligament to be thicker proximally than distally. While not statistically significant, radial and ulnar segments were thicker than the middle segments. The same relationship was found for the tissue's elastic modulus, with proximal segments being consistently stiffer than distal segments. Although thinner, the radial segments of the ligament also had greater moduli than ulnar segments. This study confirmed that mechanical properties of the TCL vary throughout the ligament and that the thicker segments were not always associated with higher moduli. Our results show the dependence of mechanical properties on the ligament's complex fibre arrangement and suggest that the ligament's contribution to carpal tunnel mechanics will vary by location.

Sample location significantly affected the characteristics of the TCL, including thickness, modulus and maximum stress. The middle of the TCL was found to have approximately 20 - 40% greater modulus in the distal samples compared to proximal segments. This difference is substantial considering the proximal portions were consistently thicker than the distal segments. Xiu and Li (2010) applied inward and outward directed forces to the carpal bones (TCL attachments) and measured compliance of the carpal tunnel at both the distal and proximal locations. They reported similar results to the middle segment data in our study, reporting TCL compliance between 3.2 (inward) and 4.3 (outward) times greater at the proximal level, suggesting the distal section of the carpal tunnel was stiffer. We also found that the ulnar and radial segments

produced greater stiffness than the middle of the ligament. In particular, the greatest modulus was found at the proximal radial sample (location 4, Figure 3.2). This section had about three times greater modulus than the proximal middle samples, and was not the thickest location measured. As suggested above, the results of our middle segments support that of previous work (Xiu and Li, 2010). However, when considering tissue samples from locations close to the carpal bone attachment sites, the mechanical properties responded differently. TCL samples from the proximal radial and proximal ulnar locations consistently produced greater moduli than the distal attachment locations.

The overall tissue thickness found in our study was comparable to those reported by others (Cobb et al., 1993; Pacek et al., 2009; Tanzer et al., 1959), but the thickness across samples varied considerably. The noted differences in elastic moduli from the proximal radial and ulnar locations of the TCL can partly be explained by tissue thickness. For instance, we found that the proximal radial samples were approximately 17% thicker than the distal radial samples and this was similar to findings from Pacek et al., (2009) who also isolated specific regions of the TCL. When considering just proximal and distal portions of the ligament, we found that the proximal segments were 26% thicker than the distal segments. It was interesting to note that, while the proximal segments were generally thicker; this did not correspond to our findings of greater ligament elastic modulus (which was found in the distal portion). This may occur due to the TCL exhibiting variability in thickness depending on location, which seems to coincide with the complex arrangement of fibres found previously (Mashoof et al., 2001; Isogai et al., 2002).

With respect to the direction of applied strain, the X direction produced slightly larger, but not statistically significant, modulus than the Y direction, the latter being perpendicular to predominant fibre orientation. The TCL segments all demonstrated a long toe region, typical of collagen fibre un-crimping (Viidik and Ekholm, 1968; Weiss and Gardiner, 2001) as evident in Figure 3.4. Only two of the six ligament locations (proximal radial and proximal middle sections) had greater modulus in the Y direction, likely a reflection of fibre orientation. Care was taken in our protocol to align the predominant fibre orientation with the X axis of our system, but, depending on tissue location, there could have been fibres oriented in other directions. This was unavoidable since the TCL consists of transverse and oblique orientated fibres (Isogai et al., 2002). Given that we found no significant differences in strain direction on modulus or maximum stress, our results indicate that the TCL's intricate design may be ideal for distributing forces throughout the carpal tunnel complex. There are likely deep fibrous layers that contribute to the ligament's overall stiffness, and this anatomical variation could provide a reason why the carpal tunnel is inflexible (Xiu and Li, 2010) and inherently unaccommodating to shape change.

We found no significant differences in moduli or maximum stress between high and low strain rates in the 15% strain trials. This suggests that the TCL may be strain rate independent, however this was not the purpose of our study and the investigation of additional strain rates may provide further insight into this effect. The lack of difference in maximum stress for each strain rate suggests that our results are applicable to low level *in-vivo* loading. These data will contribute to carpal tunnel mechanics and influence

predictions from carpal tunnel models. One recent model (Mogk and Keir, 2008; Mogk and Keir, 2009) documented the effects of wrist posture on carpal tunnel size and shape by predicting carpal bone motion but lacked TCL tissue properties, which can now be included to provide more realistic carpal tunnel mechanics. A second implication of findings from this study is to explore possible manipulative treatments for CTS. Prior to surgical treatment, manual manipulative therapy has been considered an option for the management of CTS (Moraska et al., 2008; Sucher et al. 2005). While the success of such treatment has received mixed reviews from researchers and rehabilitation professionals, Xiu and Li (2010) suggested an alternative therapeutic treatment of inwardly immobilizing the carpal bones to decrease the symptoms of CTS. Based on our findings, we found increased stiffness at the distal level of the carpal tunnel, which may suggest benefits to manipulative treatment that closely isolates the more flexible, proximal carpal tunnel location. This suggestion, however, fails to incorporate effects of ligament creep, which is evident in studies investigating manipulative treatment of the TCL by using a long duration loading protocol (Sucher et al., 2005). Stiffer TCL properties at the attachment sites (as found in the current study) suggest that successful expansion may be more likely in the middle sections of the ligament.

The TCL also acts as a pulley system for the flexor tendons at the wrist (Kline and Moore, 1992; Wehbe, 1993) and contacts the median nerve during wrist movement (Armstrong and Chaffin, 1979; Keir et al., 1997; Keir and Wells, 1999; Ko and Brown, 2007). Interestingly, in a finite element study, Ko and Brown (2007) suggested that the median nerve experienced greater stress due to structural contact rather than fluid

pressure. The TCL provides a mechanical constraint for the superficially located median nerve and places it in contact with the TCL. Based on our findings, structures located close to the radial and ulnar borders of the carpal tunnel would be in contact with a thicker and stiffer region of the TCL. This region would be less accommodating to movement when compared to the middle (and proximal) locations. Further investigations of median nerve location, with knowledge of TCL mechanical properties, could provide insight into CTS development and enhance manipulative treatments for optimal therapeutic success.

There are a few limitations to the current study. First, only biaxial testing was performed. While uniaxial testing was possible, biaxial testing was chosen to replicate *in-vivo* loading conditions imposed upon the TCL. While little work has been done to quantify *in-vivo* TCL loading, the carpal bones are considered mobile (Kaufmann et al., 2006) and capable of rotation and translation during changes in wrist posture. These complex motions, and flexor tendon interaction with the TCL, suggest that *in-vivo* loading more closely relates to biaxial testing. Using the same tensile system, greater modulus has been found using biaxial testing rather than uniaxial testing of the annulus fibrosus (Gregory and Callaghan, 2010), and this would also be expected for our study. Second, technical constraints of the tensile system limited testing to a maximum force range of 5 N. It was evident that the ligament samples tested could tolerate larger mechanical loads and, as a result, ultimate (maximum) stress of our samples could not be determined. Our results are limited to a small force range, however the strain and strain rates selected in this study are likely physiologically relevant during voluntary

movement, and similar to those tested in a study investigating TCL creep (Hinrichs et al., 2001).

This study was the first to measure mechanical properties of the TCL at different locations using biaxial tensile testing. We found that the TCL exhibits different properties at different locations of the ligament. More specifically, locations close to the attachment sites produced the greatest elastic moduli and there were also clear differences between the proximal and distal segments of the TCL. The proximal segments of the TCL had greater moduli than the distal segments and the radial segments had greater moduli than the ulnar segments. This study suggests that the TCL has material and mechanical properties that are location dependent. These properties are not just due to tissue thickness, but can be related to fibre orientation and composition.

3.6 Acknowledgements

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CHAPTER 4

**STUDY 3: POSTURE AND HAND LOAD ALTER MUSCULAR
RESPONSE TO SUDDEN ELBOW PERTURBATIONS**

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4.1 Abstract

Joint stiffness and stability are reliant on coordinated muscle activity, which may differ depending on initial posture and loading during sudden perturbations. This study investigated the effects of arm posture and hand load on muscle activity during perturbations of the arm. Fifteen male participants experienced perturbations to the wrist, causing elbow extension, using a combination of three body postures (standing, supine, sitting) and three hand load conditions (no, solid, and fluid loads), with known and unknown timing. Surface EMG was collected from eight muscles of the right upper extremity. The response to sudden loading was examined using muscle activities pre- (baseline) and post- (reflex) perturbation. During baseline, known timing had greater muscle activity than unknown timing while the opposite was found during the reflex period. During the reflex period with the fluid load, biceps brachii and brachioradialis demonstrated increases in activity of 2.4% and 4.0% of maximum respectively, from supine to standing posture. During the reflex period, the fluid load resulted in forearm co-contraction 23% and 47% greater than the solid and no load conditions. Body orientation and hand loading influenced the muscular response to elbow joint perturbations. Muscle co-contraction at the elbow during known timing perturbations suggests a contribution to elbow joint stability that may reduce injury risk caused by sudden elbow loading.

Keywords: EMG, Elbow, Perturbation, Joint stability, Hand load, Arm posture

4.2 Introduction

A primary function of the upper extremity is to coordinate movement patterns that effectively and efficiently perform tasks with the hand. Humans interact continuously with objects in the environment, which may compromise joint stability. In many cases, passive tissues are not capable of maintaining joint stability and thus an active muscle response is initiated (Panjabi, 1992). For example, the use of a hand tool requires the upper extremity musculature to provide a stabilizing function to counter the action of the tool (Rancourt and Hogan, 2001). Muscular contributions help stabilize a joint by preventing an external disturbance from producing large displacements that have the potential to cause injury. Muscle activity in response to postural demands and hand loading have been well documented at the wrist (Mogk and Keir, 2003), elbow (An et al., 1981; Dul et al., 1984) and shoulder (Au and Keir, 2007; Antony and Keir, 2010) during static and controlled dynamic contractions. However, a better understanding of how the upper extremity musculature functions to maintain joint stability is needed for a variety of conditions.

Understanding musculoskeletal stiffness is an essential component of joint stability and neuromuscular function (Granata et al., 2004). Muscle activation is closely related to stiffness (Darainy et al., 2004; Franklin et al., 2003) with simultaneous activation of muscles crossing a joint increasing joint stiffness and stability (Akazawa et al., 1983; De Serres and Milner, 1991; Stokes and Gardner-Morse, 2000; Osu et al., 2002). However, the perturbation response magnitude can be task and context specific (Hasan, 1992; Lacquaniti 1992). Joint stiffness increases with changes in muscle

activity, co-contraction, muscular synergy and limb position (van Loon et al., 2001). With the forearm vertical, antagonist muscles activate to compensate for gravitational effects on the forearm during elbow contractions, in addition to the influences of force direction and magnitude (Solomonow et al., 1986). During sudden loading, these effects may be augmented due to an increased level of complexity for the neuromuscular system. Milner (2002) evaluated individuals maintaining a stable hand position (endpoint stiffness) using a torque motor that provided mechanical instability and suggested that the nature of the destabilizing condition influenced muscle activation while postural demands influenced how participants compensated for the task. Stokes and Gardner-Morse (2000) had participants hold an arm posture of shoulder and elbow flexion at 90° and subjected it to vertical and horizontal loading. They found that different strategies were used to stabilize the elbow, depending on the load direction, variations in posture and muscle activity.

When the timing of a perturbation is known, participants generally increase muscle activation just prior to the disturbance to help stiffen the joint. However, this voluntary muscular contribution is metabolically inefficient. Alternatively, reflex contributions can act to stabilize the joint. The influence of a reflex response on joint stability varies depending on activation in both antagonist and agonist muscles prior to perturbation (Akazawa et al., 1983; Smeets and Erkelens, 1991; Lewis et al., 2010). Given this dependence on anticipatory muscle action, an understanding of the system's initial state, including postural demands and loading is important. Furthermore, anticipating a perturbation (Koike and Yamada, 2007), as well as the direction and

magnitude of an applied perturbation (Franklin et al., 2003) influences the contribution of the reflex response. The reflex contribution to joint stability in the upper extremity requires further attention given that the neuromuscular delay associated with a reflex can cause instability (Jacks et al., 1988). Muscular co-contraction and reflex actions are necessary muscular support strategies which aid passive tissues in the maintenance of joint stability.

The purpose of this study was to investigate the effects of body orientation and hand loading on upper extremity muscle activity during perturbations causing elbow rotation.

4.3 Methods

4.3.1 Participants

Fifteen right-handed males (179.5 ± 6.0 cm; 81.2 ± 8.5 kg; 26.0 ± 3.0 years) participated in this study. All volunteers confirmed no previous history of upper extremity injury and provided informed consent prior to participation. The study was approved by the McMaster University Human Research Ethics board.

4.3.2 Experimental Protocol

Participants held their right arm in a supinated posture at 90° of elbow flexion while perturbations were applied to the palmar side of the wrist using a pneumatic arm that resulted in elbow extension. A splint was used to maintain a neutral wrist and to provide a consistent rigid target to apply the perturbation force at the centre of the wrist joint. Three body orientations were used to provide challenges to the system (i) lying

supine, (ii) sitting with the shoulder flexed to 90° with the upper arm resting on a table, and (iii) standing (Figure 4.1). In each body posture, participants maintained an elbow flexion angle of 90° under three hand loading conditions (i) holding nothing in the hand (referred to as “no load”), (ii) holding a tube horizontally (“solid tube”), and (iii) balancing a water filled tube (“fluid tube”). Two identical tubes were used for the solid and fluid conditions (34.5 cm long, 2.54 cm diameter and weighing 0.68 kg when filled). Participants assumed the experimental test position with the pneumatic arm of the perturbation device oriented to deliver the push force to the palmar side of the wrist, perpendicular to the forearm (Figure 4.2).

Perturbations were applied with and without the participant’s knowledge of when it would occur. During known timing perturbations, the participant was given a manual trigger and initiated the perturbation when desired. During unknown timing perturbations, the experimenter signaled the start of the trial to the participant and the perturbation was delivered after a random duration of up to 10 seconds. Each body posture, hand load and timing combination was performed in a blocked randomization selection, such that all hand loading and timing combinations were randomly performed in each posture before selection of the next posture. Three trials of each condition were performed with 30 seconds of rest given between trials. Approximately 5 minutes of rest was given between postures to limit the effects of muscular fatigue and to adjust the device.

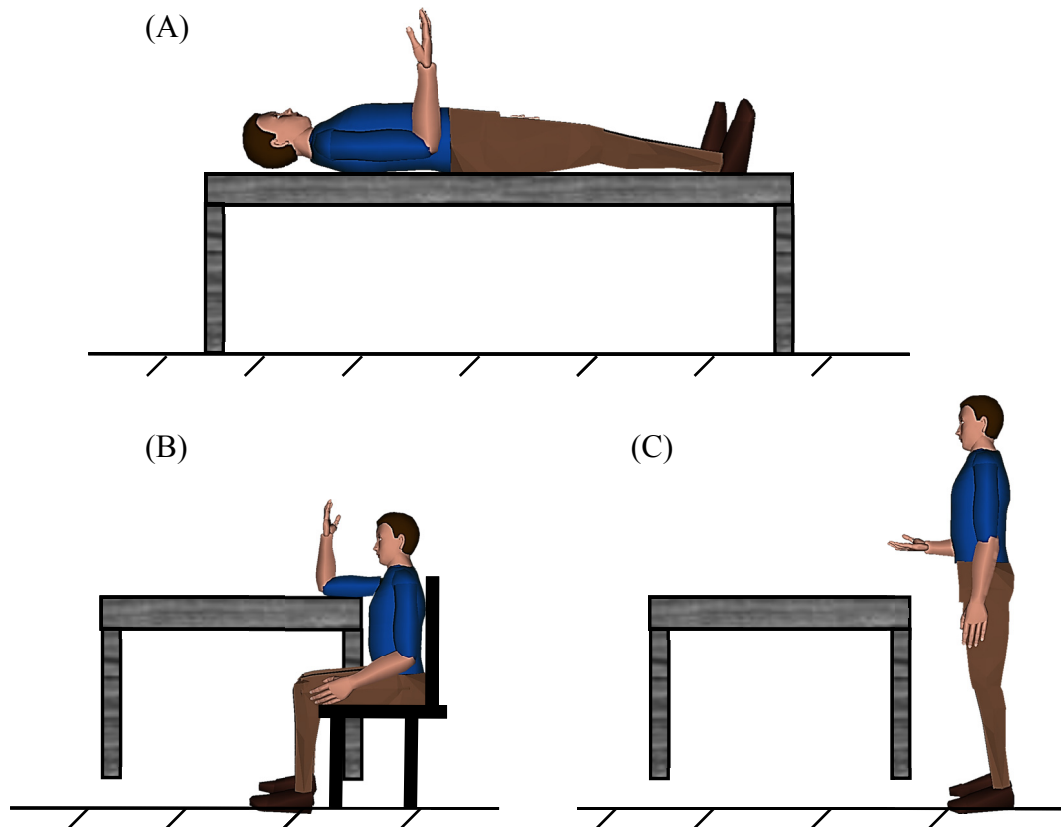


Figure 4.1 The three body orientations: A) supine, B) sitting and C) standing. Note: The perturbation device was orientated to accommodate each body orientation.

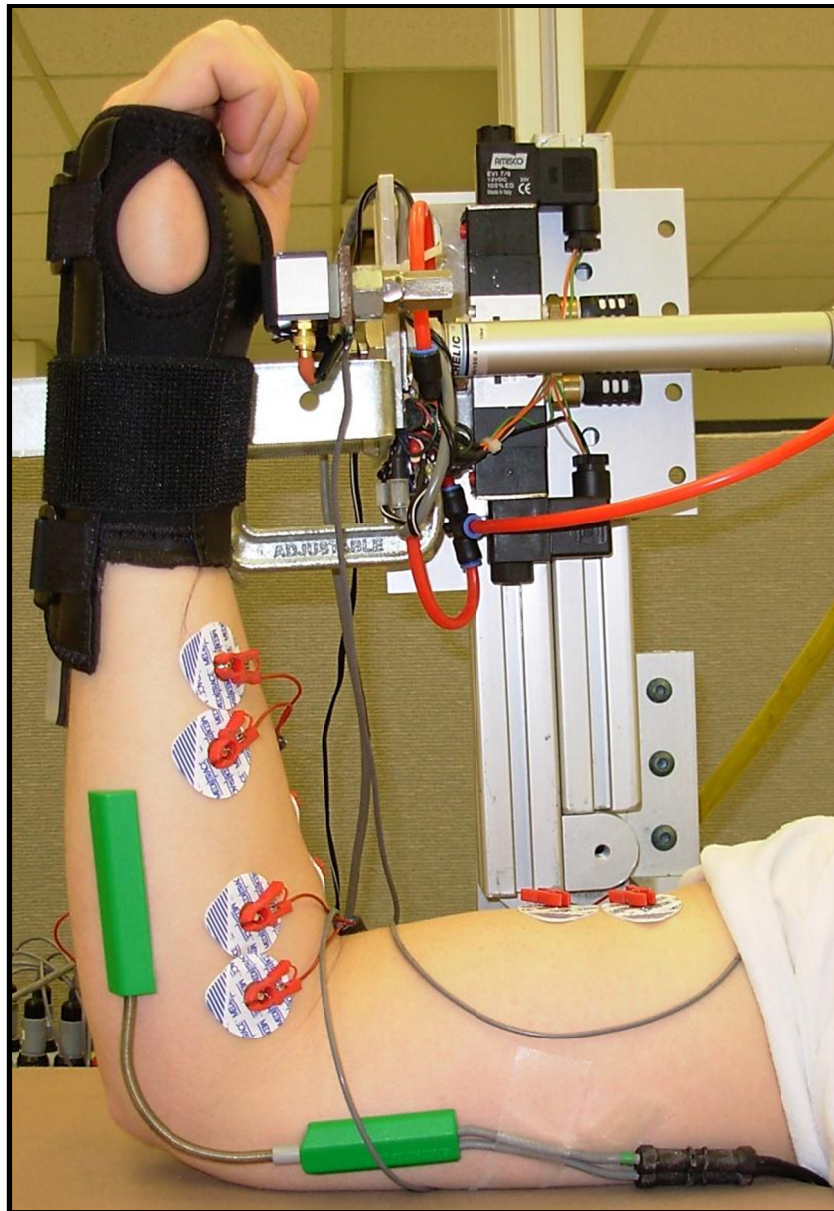


Figure 4.2 Participant preparing for a perturbation during the sitting posture and no load condition. The perturbation device was adjusted to impact each participant in the same location.

4.3.3 Data Collection and Instrumentation

Once familiar with the protocol, each participant was prepared for surface electromyography (EMG) which was collected from eight muscles of the right upper extremity: anterior deltoid (AD), triceps brachii (TB), biceps brachii (BB), brachioradialis (BR), flexor carpi radialis (FCR), flexor digitorum superficialis (FDS), extensor carpi radialis (ECR) and extensor digitorum communis (ED). Electrode sites were prepared by shaving and scrubbing with alcohol prior to placement of disposable Ag-AgCl surface electrodes (MediTrace 130, Kendall, Mansfield, MA, USA) over the muscle belly in line with muscle fibre direction with an inter-electrode distance of 2.5 cm. All electrode placements were confirmed using palpation and manual resistance tests. Muscle specific maximal voluntary isometric contractions were maintained for at least 3 seconds to obtain maximal voluntary excitations (MVE) for each muscle. Maximal contractions were performed twice for each muscle group. EMG signals were differentially amplified and band pass filtered (10 - 1000 Hz; CMRR > 115 dB at 60 Hz; input impedance ~10G Ω ; AMT-8, Bortec Biomedical Ltd., Calgary, AB, Canada).

An electrogoniometer was used to measure elbow angle (SG110, Biometrics, Ltd., Gwent U.K.). The electrogoniometer was centred about the medial epicondyle and secured to the arm with two-sided tape. The perturbation device was equipped with a load cell (MPL-300-CO, Transducer Techniques, Temecula, CA, USA) attached to a metal rod that extended outward in a single plane to deliver the push force. EMG, electrogoniometer and load cell data were sampled at 2048 Hz and A/D converted using a 16-bit A/D system (USB-6229 BNC, National Instruments, TX, USA).

4.3.4 Data Analysis

A quiet trial was collected at the start of testing and used to remove signal bias from each EMG channel. All signals were full-wave rectified and low pass filtered using a 3 Hz cutoff (dual pass, effective 4th order Butterworth) to create a linear envelope of the signal. The maximum activation was found for each muscle specific maximum contraction and used to normalize all EMG signals. The electrogoniometer and load cell data were low pass filtered (dual pass, effective 4th order Butterworth) using 3 Hz and 10 Hz cutoffs, respectively.

Three time periods were examined: (i) baseline, from 150 ms to 100 ms pre-perturbation, (ii) anticipatory, from 15 ms to 0 ms pre-perturbation and (iii) reflex, from 25 ms to 150 ms post-perturbation (Figure 4.3). The start of the perturbation was indicated by a pressure sensor within the perturbation device. The co-contraction index (CCI) was calculated for all 28 muscle pair combinations (Lewek et al., 2004). The CCI provides a measure of muscle co-activation for muscle pairs over a specified time period and uses the ratio between the muscles of lowest and highest normalized activity, multiplied by the sum of the two muscle activities at each sampled point. Average EMG and CCI were investigated at each of the three time periods listed above. As a data reduction measure, the mean co-contraction from all forearm muscle comparisons (FCR-FDS, FCR-ECR, FCR-ED, FDS-ECR, FDS-ED, and ECR-ED) was taken as a “global” forearm flexor-extensor muscle combination.

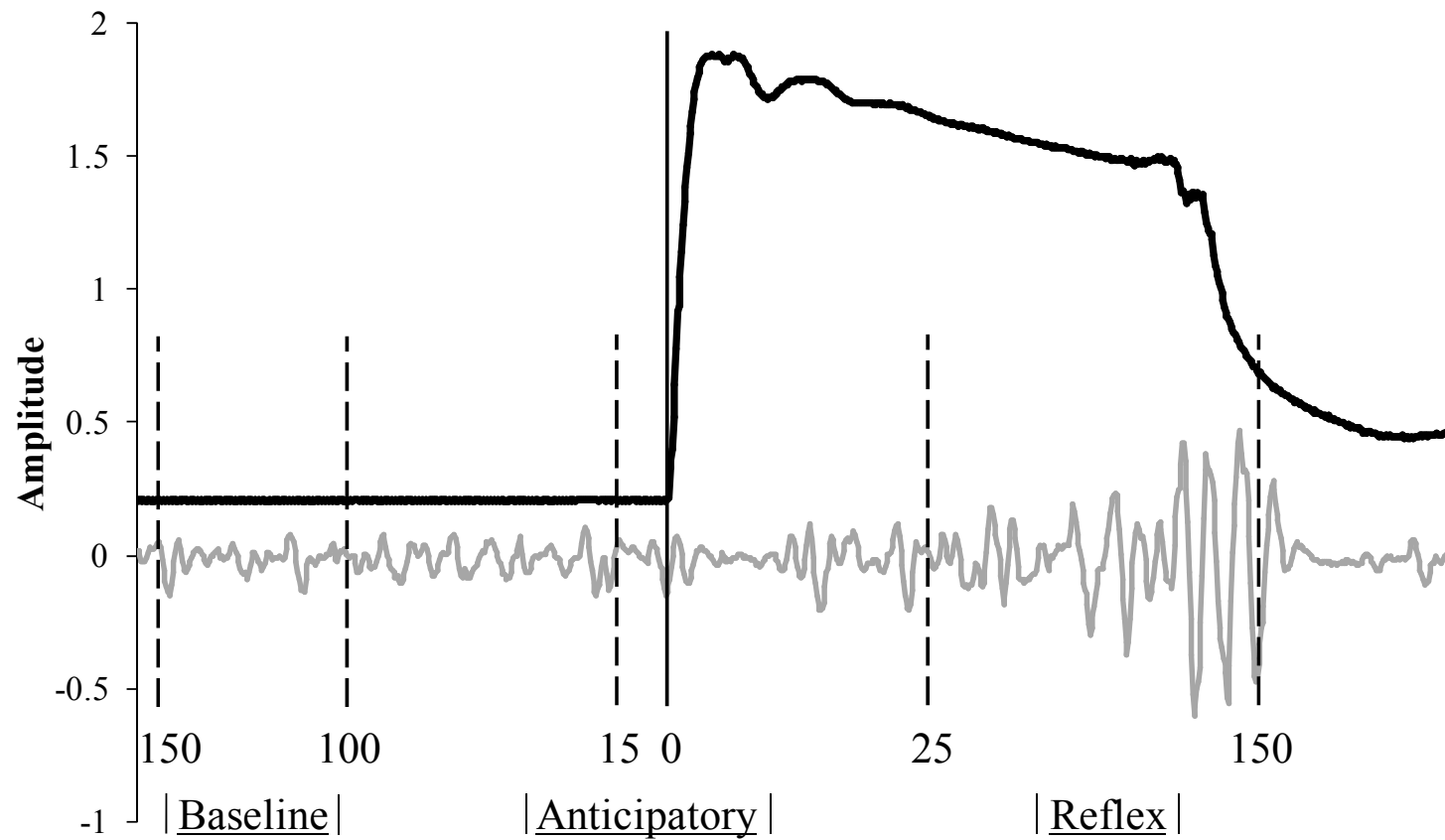


Figure 4.3 Example of one perturbation trial and the time periods analyzed. The black line represents the perturbation device which indicated perturbation onset time. The grey line is a representative EMG signal.

4.3.5 Statistical Analysis

Data were averaged across the three trials for each condition. For each muscle and peak perturbation push force (obtained from the load cell), a 3 (posture) x 3 (load) x 2 (timing knowledge) repeated measures ANOVA was used to evaluate the effects of the independent variables on muscle activity during the baseline, anticipatory and reflex time periods. Significant effects were further evaluated using pair-wise comparisons with Bonferroni correction (SPSS v13.0, IBM Corporation, Somers, NY, USA). An alpha level of 0.05 was used for all statistical analyses.

4.4 Results

The perturbation device delivered a consistent impact to each participant, however, participant resistance to the perturbation varied with posture. The mean peak push force across all trials was 60.0 ± 12.3 N. Standing posture resulted in significantly larger maximum push force (74.4 ± 8.5 N) than the sitting and supine postures ($p < 0.05$), while no differences were found between sitting (58.3 ± 4.4 N) and supine (52.5 ± 8.1 N). There were no significant differences in push force due to hand load or knowledge of perturbation timing.

Muscle activity for the anticipatory period was similar to baseline, thus we will present only the baseline and reflex time periods.

4.4.1 Baseline Time Period (150 - 100 ms pre-perturbation)

There was a posture x load interaction for all muscles during the baseline period (all $F_{4,56} > 3.78$, all $p < 0.01$) except TB ($F_{4,56} = 1.99$, $p = 0.101$). AD, BB and BR

muscles had increased activity during standing when participants held the solid and fluid tubes (Figure 4.4A). During the no hand loading task, AD activity did not change significantly due to posture. However, with the solid tube, AD activity was 1.0% MVE greater during standing than both sitting and supine. With the fluid-filled tube, AD activity increased by 1.2% from sitting to standing and 1.6% MVE from supine to standing (Figure 4.4A). BB activity increased from supine to standing by 1.9% (no load), 1.6% (solid) and 1.0% (fluid) MVE. For both tubes, BR activity increased by 1.0% MVE when standing versus sitting and supine, with no changes in the no load condition. FCR and FDS demonstrated no changes in activity across postures during the no load and solid tube conditions. With the fluid hand load, FCR activity increased during standing by 1.7% and 1.4% MVE from sitting and supine, respectively. With the fluid load, FDS activity increased during standing by 1.7% MVE over the other postures (Figure 4.4B). Opposite to the flexors, the forearm extensors (ECR and ED) had the lowest muscle activity during standing. With the solid hand load, ED activity was 3.5% and 4.8% MVE less during standing than the sitting and supine postures. ECR also demonstrated the largest decrease in activity during the solid condition, decreasing by 1.6% MVE from supine to standing (Figure 4.4B).

Knowledge of perturbation timing had a significant main effect, resulting in higher activity for all muscles (all $F_{1,14} > 4.96$, all $p < 0.05$) except AD ($F_{1,14} = 3.91$, $p = 0.0707$). However, this difference was less than $0.5 \pm 0.3\%$ MVE for all muscles.

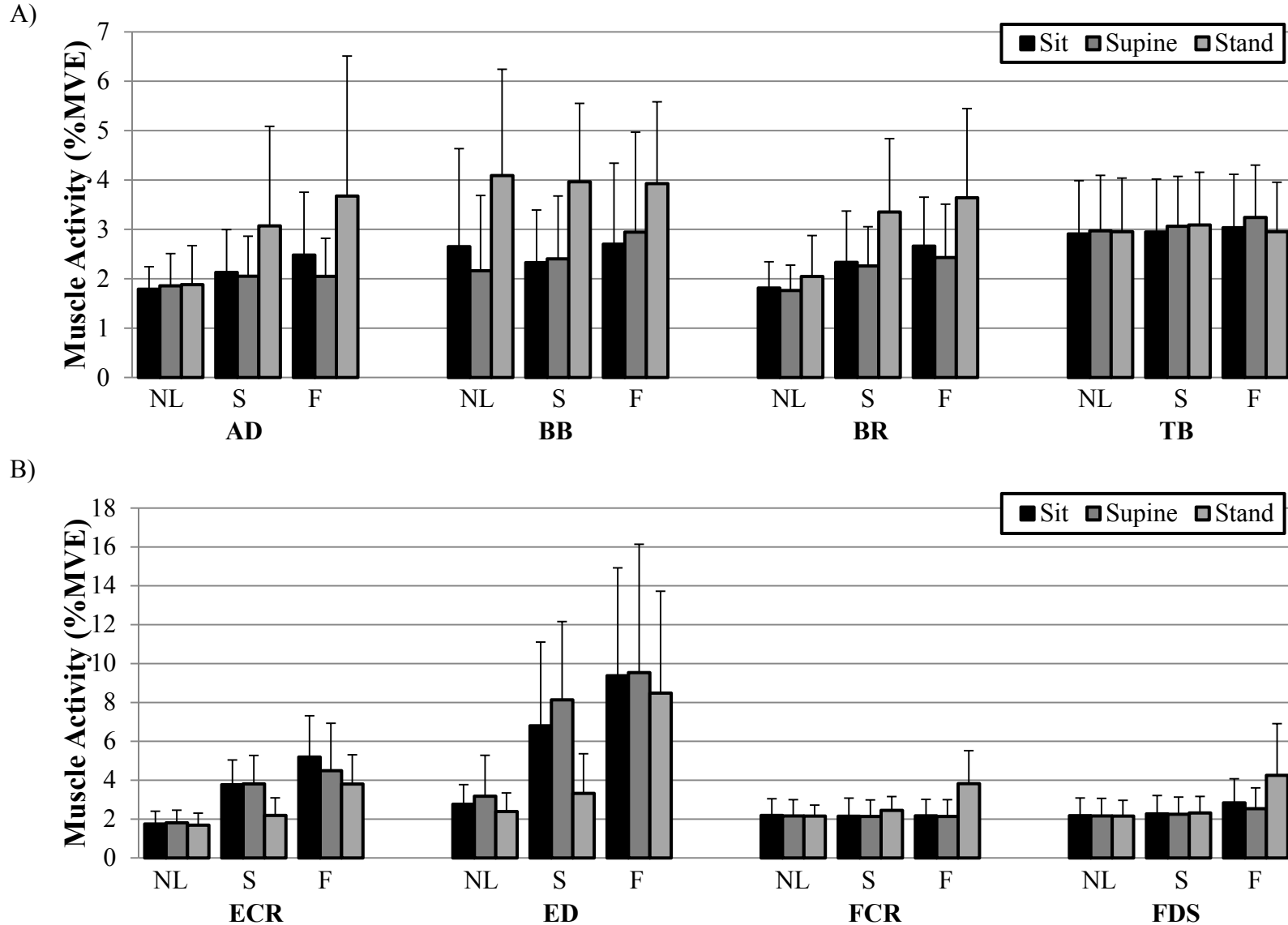


Figure 4.4 Mean muscle activity (%MVE with Standard Deviation) demonstrating the effects of posture and load at baseline for (A) the AD, BB, BR and TB muscles; (B) the forearm muscles (ECR, ED, FCR, and FDS). Note: NL – No load; S – Solid tube; F – Fluid tube. See text for muscle abbreviations.

4.4.2 Reflex Time Period (25 - 150 ms post-perturbation)

There was a significant posture x load interaction for AD, BR, FCR, ECR and ED (all $F_{4,56} > 2.94$, all $p < 0.05$). For AD, muscle activity during the sitting posture with both the solid and fluid tubes was greater than during supine and standing with the same load (Figure 4.5A). Like at baseline, BR and BB muscle activity were greatest during the standing posture and with the fluid tube. When holding the fluid tube, BB and BR increased (by 2.4% and 4.0% MVE respectively) from supine to standing (Figure 4.5A). FDS and FCR also had greater activity when holding the fluid tube during standing. For ECR and ED, there were no changes in activity during the no load and fluid tube conditions. However, when holding the solid tube, ECR and ED activity decreased by 2.4% and 1.5% MVE, respectively from sitting to standing (Figure 4.5B).

A timing x posture interaction was found during the reflex period for AD, TB, BR, FCR, FDS and ECR (all $F_{2,28} > 3.45$, all, $p < 0.05$). Unlike at baseline, unknown timing resulted in increased muscle activity across all postures, with the largest increase from known timing found during standing (Figure 4.6). During standing, unknown timing was 0.7%, 2.1% and 2.8% MVE greater than known timing for BB, TB and BR, respectively. For the forearm muscles during standing, unknown timing increased ECR, ED, FCR and FDS activity by 1.1%, 1.9%, 2.7% and 3.2% MVE, respectively from known timing (Figure 4.6).

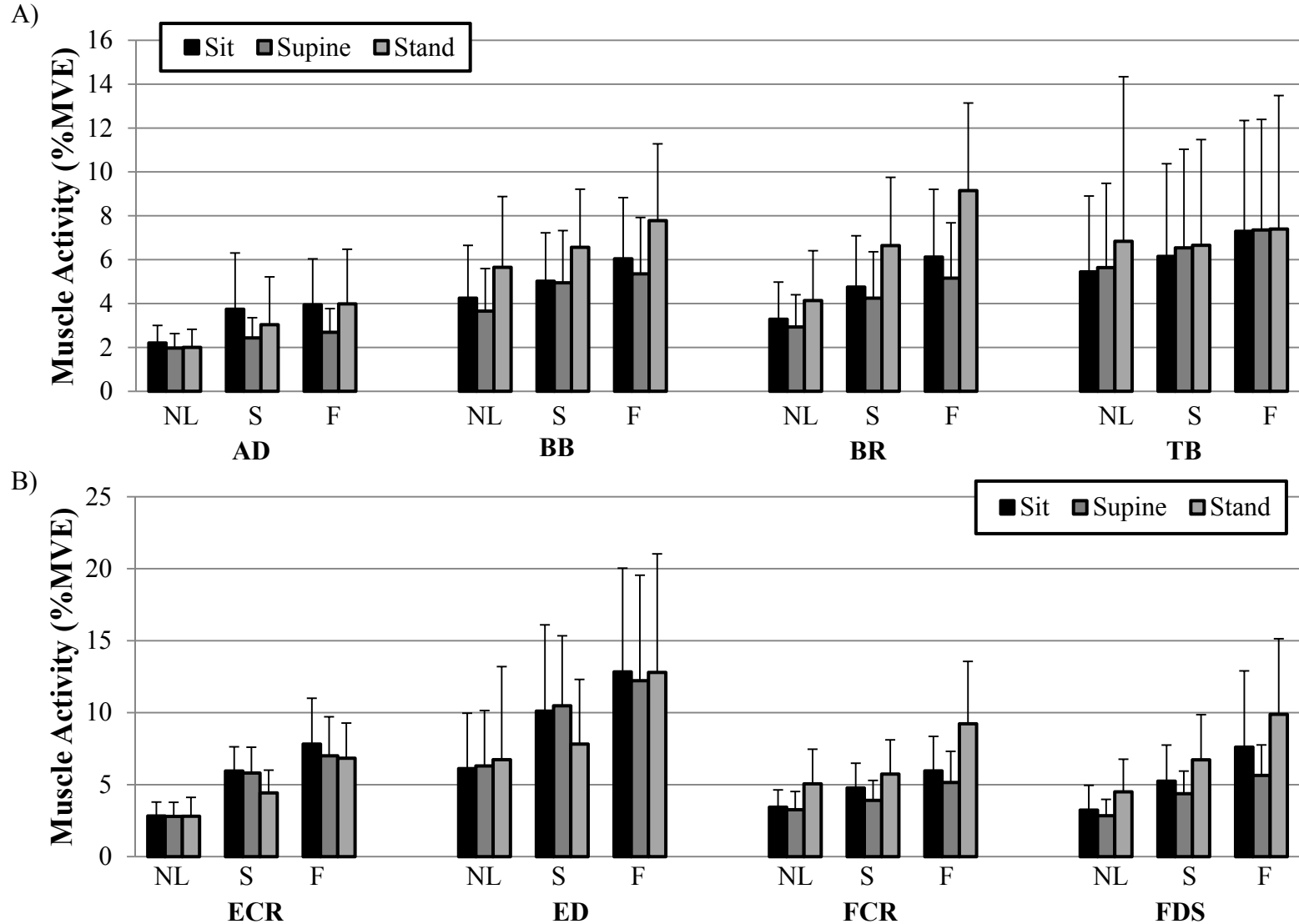


Figure 4.5 Mean muscle activity (%MVE with Standard Deviation) demonstrating the effects of posture and load during the reflex period for (A) the AD, BB, BR and TB muscles; (B) the forearm muscles (ECR, ED, FCR, and FDS). Note: NL – No load; S – Solid tube; F – Fluid tube. See text for muscle abbreviations.

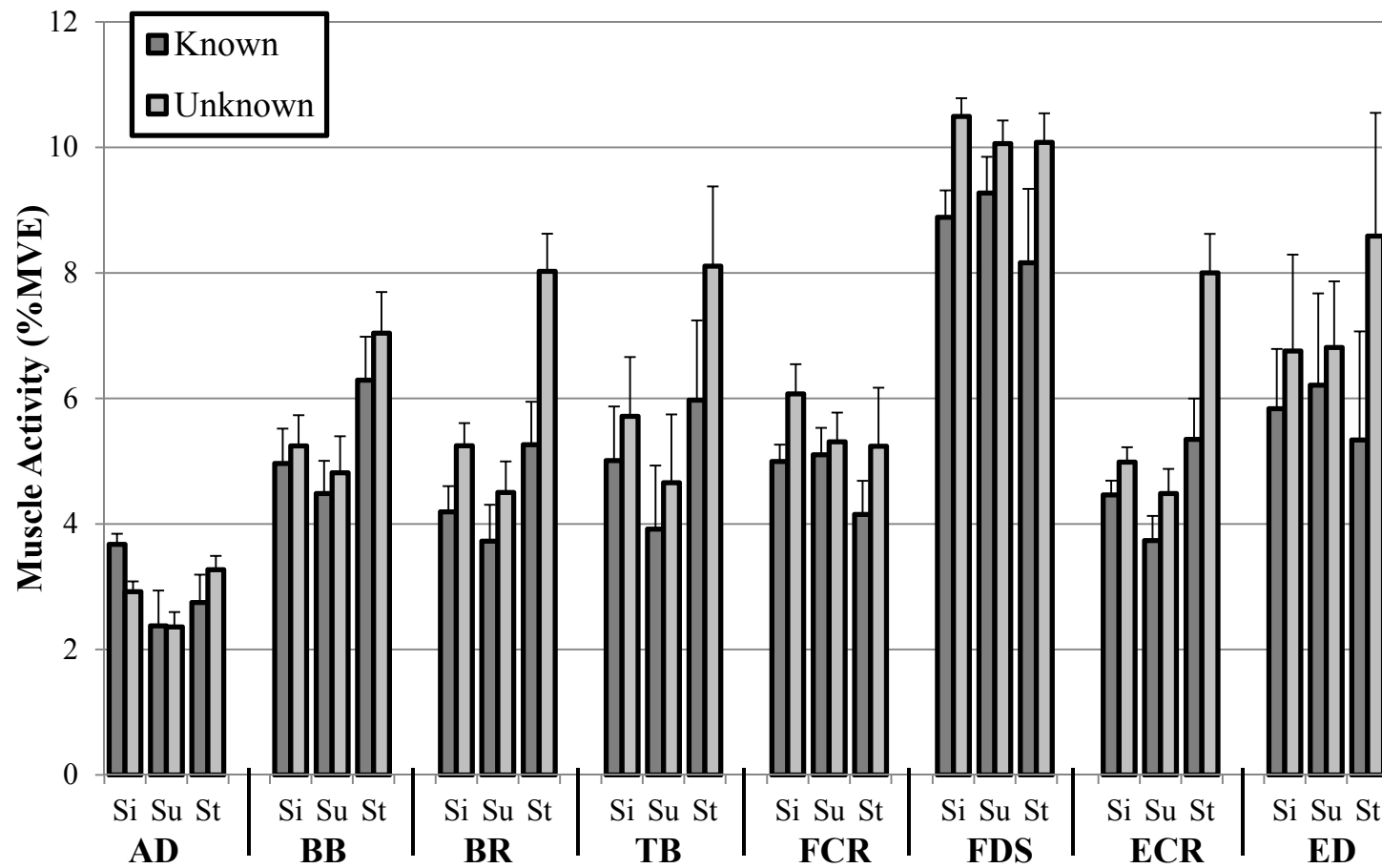


Figure 4.6 Mean muscle activity (%MVE with Standard Deviation) demonstrating perturbation timing knowledge and posture during the reflex period. Note: Si – Sit; Su – Supine; St – Stand. See text for muscle abbreviations.

4.4.3 Muscle Co-contraction

4.4.3.1 Forearm Co-contraction

Co-contraction in the forearm was assessed by obtaining the mean CCI from all forearm flexor-extensor comparisons. There was a posture x load interaction during both baseline ($F_{4,56} = 13.98$, $p = 0.0001$) and reflex ($F_{4,56} = 7.46$, $p = 0.0001$) periods for forearm flexor-extensor CCI. For both time periods, holding the fluid tube during the standing posture resulted in greater co-contraction. The differences were greatest in the reflex period as the mean CCI during the fluid conditions were greater than the no load conditions with a difference in CCI of 3.2, 3.5 and 5.9 for the sitting, supine and standing postures, respectively (Table 4.1).

During baseline, there were no significant differences with timing knowledge ($F_{1,14} = 2.32$, $p = 0.150$). However, the reflex period demonstrated a significant main effect of timing knowledge ($F_{1,14} = 15.39$, $p = 0.002$) with greater co-contraction during unknown timing than known timing perturbations.

4.4.3.2 Elbow Flexor-Extensor Co-contraction

During the reflex period, there was a significant timing x posture interaction ($F_{2,28} = 6.4$, $p = 0.005$) for the biceps-triceps CCI. During standing, co-contraction increased by 25% (increased CCI from 7.0 to 8.8) for unknown timing, but increased by only 3% (from 6.1 to 6.3) and 11% (from 6.1 to 6.7), for the supine and sitting postures, respectively.

Hand load had a significant main effect on CCI at both baseline ($F_{2,28} = 4.39$, $p = 0.022$) and reflex ($F_{2,28} = 17.09$, $p = 0.0001$). The largest differences were seen during

the reflex period where the fluid tube increased co-contraction by 13% (from 6.9 to 7.8) and 38% (from 5.6 to 7.8), from the solid tube and no load conditions, respectively (Table 4.1). There was also a significant main effect of posture at baseline ($F_{2,28} = 7.80$, $p = 0.002$), with the standing posture being larger than sitting ($p = 0.018$) and a trend toward being larger than the supine posture ($p = 0.052$). There were no significant differences at baseline ($p = 0.31$) for knowledge of perturbation timing.

Table 4.1: Mean CCI (standard deviation) for the forearm flexor-extensor and elbow flexor-extensor (biceps-triceps) muscle groups at the baseline and reflex time periods for all conditions.

Perturbation Timing	Body Posture	Hand Load	Forearm Flexor-Extensor		Elbow Flexor-Extensor (Biceps-Triceps)	
			Baseline	Reflex	Baseline	Reflex
Known	Supine	None	3.1 (0.2)	4.0 (0.2)	3.3 (2.1)	5.0 (2.6)
		Solid	3.7 (0.9)	5.7 (1.0)	3.9 (2.1)	6.4 (3.2)
		Fluid	3.9 (1.6)	6.8 (1.5)	4.1 (2.2)	7.0 (4.2)
	Sit	None	3.1 (0.2)	4.3 (0.3)	3.2 (1.8)	4.9 (2.5)
		Solid	3.8 (0.9)	6.7 (0.7)	4.0 (2.2)	6.6 (3.5)
		Fluid	4.4 (1.7)	8.3 (1.1)	4.1 (2.0)	6.8 (3.3)
	Stand	None	3.1 (0.3)	4.0 (0.4)	4.6 (2.0)	5.8 (2.7)
		Solid	3.7 (0.1)	6.3 (0.7)	4.9 (2.1)	7.4 (3.4)
		Fluid	5.7 (0.3)	9.5 (1.1)	4.6 (1.6)	8.0 (3.1)
Unknown	Supine	None	3.0 (0.2)	4.1 (0.4)	3.1 (2.0)	4.8 (2.3)
		Solid	3.6 (0.8)	6.5 (1.2)	3.8 (2.3)	6.2 (2.7)
		Fluid	4.0 (1.2)	8.3 (0.8)	3.9 (1.8)	7.9 (4.3)
	Sit	None	3.6 (0.2)	4.6 (0.4)	3.8 (2.2)	5.7 (2.9)
		Solid	5.3 (1.1)	7.1 (0.6)	3.3 (1.5)	6.3 (2.6)
		Fluid	5.4 (0.8)	7.1 (0.01)	3.5 (1.7)	8.1 (4.6)
	Stand	None	3.1 (0.3)	5.8 (1.4)	4.6 (2.0)	7.6 (3.8)
		Solid	3.7 (0.2)	7.9 (1.1)	5.0 (2.2)	8.5 (3.9)
		Fluid	5.5 (0.4)	12.1 (1.2)	4.9 (2.0)	10.3 (5.0)

4.5 Discussion

This study investigated the influence of body orientation (initial arm posture) and hand load on the muscular response to sudden expected and unexpected perturbations delivered to the wrist. Muscular responses to the perturbation were clearly influenced by posture and hand loading. While small in magnitude, there was also an increase in activity at baseline during the known timing perturbations. The standing posture and fluid load conditions consistently produced the largest muscle activities, except for the forearm extensor muscles. A posture x load interaction was found at both baseline and reflex for most muscles. Elbow co-contraction increased when holding the fluid tube indicating that stabilizing the load challenged the muscular system resulting in greater joint stiffness. The increase in forearm muscle co-contraction likely indicates a forearm muscle contribution to elbow stability, which makes sense intuitively but has not traditionally received much attention. This study demonstrated that changes in arm posture and hand loading will provide altered muscular states prior to a perturbation that will influence the magnitude of muscle co-contraction for both the elbow flexor-extensors, as well as the forearm muscles.

The postures used in this study were anatomically similar, yet body orientation placed different biomechanical demands on the muscular system. The greatest muscle activity for BB and BR was found during the standing posture (approximately 4% and 3.5% MVE, respectively). This was expected, since the mass of the forearm caused an increased external elbow extensor moment, requiring the flexors to maintain equilibrium. In the supine and sitting postures, the forearm acted as an inverted pendulum, which

required little muscle activity to maintain an equilibrium state. During the baseline period (150 ms to 100 ms pre-perturbation), there were no differences in BB and BR muscle activity for the supine or sitting postures, however during the reflex period, sitting produced higher activations than supine due at least partially to a shorter muscle length due to the flexed shoulder.

At baseline, BB and TB muscle activities were unaffected by hand load. However, balancing the fluid tube required greater demands from the muscular system, which was evident by the constant increase in elbow and forearm CCI from no load to solid to the fluid hand loading tasks. Thus it appears that the complexity of the loading increased as intended, with weight and the dynamic nature of the fluid in the tube and as previously demonstrated, the neuromuscular system's ability to selectively adapt joint stiffness by increasing muscular co-contraction was utilized (De Serres and Milner, 1991; Milner et al., 1995).

Furthermore, the constant load x posture interaction evident for the forearm muscles appeared to be influenced by hand load. The forearm flexors (FCR and FDS) demonstrated an increase in muscle activity with the fluid tube, and an interaction with posture resulted in standing producing increased activity from the supine posture. At baseline, FCR and FDS both increased by approximately 1.7% MVE (Figure 4.4B) and during the reflex period by 4.1% (Figure 4.5B). Similar load x posture interactions were observed for the co-contraction measures. The initial arm postures in our study did not affect forearm muscle length, so the changes in activity were partly attributed to another mechanism. Trumbower et al. (2009) investigated the effects of arm posture on endpoint

stiffness using a 3D robotic manipulator and found that arm posture influenced task performance and is a fundamental mechanism to regulate arm stiffness. Therefore, it is likely that our changes in body orientation influenced the participants' ability to compensate for load type. This resulted in a more challenging task during the sitting and standing postures and the forearm flexors were activated to a greater extent to help balance the hand loads.

With respect to AD, we found higher activity for the solid and fluid conditions during sitting, which was somewhat inconsistent with other muscles that predominantly demonstrated increased activation during standing. During static shoulder exertions, our laboratory has demonstrated that shoulder muscle activity (anterior and middle deltoid) is reduced when simultaneously performing a grip task (Au and Keir, 2007; Antony and Keir, 2010). Our results did not demonstrate this, which is consistent with DiDomizio and Keir (2010) who also failed to see this during a simultaneous gripping task with pushing or pulling. The influence of gripping on shoulder muscle activity is likely task specific which is supported by our posture x load interaction effect and is in agreement with Buchanan et al. (1986), who suggested that synergistic muscle actions at the elbow are task specific. During the supine posture, AD activity remained unchanged with hand load, however, significant increases were observed when holding the fluid tube during sitting and standing. The increased AD activity found in the current study during sitting is likely a combination of postural demands for the task, but during standing it may suggest that participants stiffened the shoulder during the elbow perturbations. Discrepancies found in shoulder activation with a concurrent grip task (Au and Keir,

2007; DiDomizio and Keir, 2010) may be dependent on postural demands, since changing body orientation in our study influenced AD muscle activity.

We found a small, but significant, increase in activity when participants self-triggered the perturbation (known timing) during the baseline period. This suggests a pre-emptive muscle response prior to the perturbation and is consistent with previous work involving sudden spine loading (Stokes et al., 2000; Brown et al., 2003). The small activity increases found in the current study suggest that a voluntary neuromuscular response was initiated to help stiffen the elbow joint. When the perturbation was unexpected, we found an increase in muscle activity during the reflex period, representing an involuntary neuromuscular response, similar to that in perturbations of the spine (Cholewicki et al., 1997; Grondin and Potvin, 2009) and upper extremity (Latash, 1994).

During manual tasks of the hands, the forearm muscles must balance moments about the wrist while performing the task and provide adequate wrist joint stiffness to maintain joint stability. These muscles are multi-articular, and are also capable of creating moments at the elbow joint. The increased forearm muscle co-contraction found during the standing posture and fluid tube conditions likely increase wrist stiffness, but should also be considered with evaluating muscle contributions to elbow joint stiffness. We found large increases in forearm extensor muscle activity during many conditions and, while this contributed to increased forearm muscle co-contraction, it is also possible that the forearm extensors contributed to the net elbow extension movement. Unbalanced muscle action can contribute to instability (Jacks et al., 1988), and therefore care needs to

be taken when evaluating forearm muscle contributions to elbow stiffness, since a forearm extensor moment may be counterproductive in stabilizing the elbow. Further investigation using a biomechanical model to evaluate muscle forces and joint rotational stiffness (Potvin and Brown, 2005) would help clarify these issues.

4.6 Conclusions

This study showed that changes in body orientation and hand loading influenced the muscular response to a sudden elbow joint perturbation, and will therefore influence the system's ability to stiffen and stabilize the joint. Knowing the timing of the perturbation produced increased muscle activity prior to the perturbation, and a lack of timing knowledge resulted in higher reflex activity following the perturbation. The standing posture and fluid tube hand loading produced the greatest muscle activity. The complexity of the load increased with the fluid tube and produced a neuromuscular response that increased muscle co-contraction at both the elbow and forearm above that required by the solid tube. The contribution of the forearm muscles to elbow joint stability should be considered when evaluating sudden elbow loading, since an increase in forearm co-contraction will influence elbow joint stiffness. Increased muscle co-contraction due to the standing posture and fluid load will likely influence elbow joint stiffness and thus, contribute to elbow joint stability. Further quantification of the elbow and forearm muscle contributions to elbow joint stability is needed to better understand joint injury risk during sudden elbow loading.

4.7 Acknowledgements

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CHAPTER 5

**STUDY 3: MUSCLE CONTRIBUTIONS TO ELBOW JOINT
ROTATIONAL STIFFNESS IN PREPARATION FOR SUDDEN
EXTERNAL ARM PERTURBATIONS**

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5.1 Abstract

Understanding joint stability is beneficial for assessing injury risk and joint failure. However, there has been limited work quantifying individual muscle contributions to elbow joint stability in the upper extremity during sudden external perturbations. The purpose of this study was to examine the joint rotational stiffness provided by individual upper extremity muscles that contribute to elbow joint stability. Fifteen male participants held a combination of three body postures (standing, supine, sitting) and three hand loading tasks (no load, solid load, fluid load) while a sudden perturbation caused elbow extension. Elbow joint angles and activity from eight upper extremity muscles were collected and used as input to a biomechanical model to determine each muscle's contribution to elbow joint rotational stiffness (JRS_M). Averaged across all experimental trials, JRS_M was greatest for brachialis ($30.4 \pm 1.9\%$), followed by brachioradialis ($21.7 \pm 2.2\%$), biceps short ($19.7 \pm 0.8\%$) and long head ($15.5 \pm 1.2\%$). The combined JRS_M for the forearm muscles and triceps was $5.5 \pm 0.6\%$ and $9.2 \pm 1.9\%$, respectively. The contribution of the primary elbow flexors and forearm muscles were greater immediately prior to the sudden perturbation than during the baseline period (100-150 ms before perturbation). JRS_M for the primary elbow flexors were influenced by posture, and displayed the highest values when standing. The primary elbow flexors contributed most to elbow joint stability and provided an increased neuromuscular response just prior to the perturbation, which is beneficial for stiffening the joint and minimizing the chance of joint injury due to sudden elbow loading. This

study documents the primary muscles that contribute to the maintenance of joint stability, which can lower the risk of joint injury during sudden perturbations.

Keywords: Upper Extremity, Elbow, Joint Rotational Stiffness, Joint Stability, Biomechanical Modeling

5.2 Introduction

The capacity of the musculoskeletal system to maintain joint stability is critical for successful human movement. Moreover, an understanding of joint stability can be beneficial for assessing injury risk since inadequate joint stiffness can result in joint failure. Mechanically, stability is considered to be binary, without level or magnitude, meaning that a system is either stable or unstable. However, in biomechanical terms, it is beneficial to understand a system's ability to adapt or respond to a disturbance. Thus, parameters such as joint stiffness help provide a margin of safety, which makes a joint more robust to a perturbation, and thus able to maintain a stable behaviour (Reeves et al., 2006). The level of joint stiffness can be modulated through contributions from individual muscles, tendons, ligaments, cartilage and bone (Latash and Zatsiorsky, 1993). Panjabi (1992) suggested that passive tissues alone are generally not capable of maintaining joint stability and thus muscular contributions must play a critical role. A joint is considered stable when it can maintain, or return to, a state of equilibrium following an external perturbation and muscular contributions help stabilize the joint by preventing the disturbance from producing unpredictable movements that may result in tissue injury. Typically, joint stiffness is largely influenced by muscular action as demonstrated in the spine (Cholewicki and McGill, 1996; Crisco and Panjabi, 1991; Potvin and Brown, 2005); however, there has been no work to quantify individual muscle contributions to joint stability in the upper extremity, particularly during sudden external perturbations. Sudden loading to joints in the upper extremity is typical during unexpected disturbances or when “catching” oneself, therefore evaluating the effects of

arm posture and hand loading on upper extremity muscle activity needs to be investigated with reference to muscular contributions to joint stability.

Muscle stiffness increases with muscle activity, leading to overall stiffening of a joint (Cholewicki and McGill, 1996; Darainy et al., 2004; Franklin et al., 2003; Perreault et al., 2001). Furthermore, occupational risk factors for injury, including awkward postures and large forces, have been well documented in the upper extremity (Moore et al., 1991; NIOSH, 1997; Silverstein et al., 1986). From a motor control perspective, numerous studies have focused on the influence of posture on endpoint stiffness during limb movements (Franklin et al., 2003; Franklin et al., 2007; Perreault et al., 2001; Perreault et al., 2004). The focus of these studies was to understand endpoint stiffness and movement accuracy during goal-directed tasks. From a biomechanical perspective, the complexity of the neuromuscular system has made traditional stability analyses challenging. To date, dynamic stability analyses evaluating muscle contributions during sudden loading events have been limited. However, knowledge of the system's initial state, prior to a disturbance, is needed to understand muscular contributions to mechanical joint stability and provide insight into joint safety.

Previously, Holmes and Keir (chapter 4) found small increases in muscle activity just prior to an elbow joint perturbation when knowledge of the timing event was known, which is consistent with previous spine perturbation and quick release studies (Chiang and Potvin, 2001; Grondin and Potvin, 2009; Stokes et al., 2000). This voluntary muscular response represents muscle action, which helps stiffen the joint prior to a disturbance. Holmes and Keir (chapter 4) also found that muscular responses to the

perturbation were influenced by posture and hand loading. These findings agree with van Loon et al. (2001) who suggested that increases in joint stiffness occur with changes in muscle activity, co-contraction, muscular synergy and limb position. Stokes and Gardner-Morse (2000) evaluated elbow joint stability under vertical and horizontal loading conditions and found that participants were able to maintain stable equilibrium by utilizing different strategies including variation in posture and muscle activity. Stokes and Gardner-Morse (2000) evaluated overall elbow joint stability during elbow flexion and extension tasks, yet individual muscle contributions to elbow joint stability have yet to be investigated.

Traditionally, the elbow joint has been considered inherently stable due to a strong bony configuration combined with a large number of ligaments, a deep joint capsule and many muscles crossing the joint (Hamilton et al., 1996; Safran and Baillargeon, 2005). The purpose of this study was to examine individual muscle contributions to joint rotational stiffness at two time periods immediately prior to a sudden external perturbation. In particular, this study investigated the potential for the forearm muscles to contribute to elbow joint stability as a consequence of increased hand loading.

5.3 Methods

5.3.1 Participants

Fifteen right hand dominant males with a mean height of 1.80 ± 0.06 m, mass of 81.2 ± 8.5 kg and age of 26.0 ± 3.0 years participated in this study. All participants self-

reported that they had no prior history of musculoskeletal injury to the upper extremity. Each participant provided informed consent and the study was approved by the McMaster University Human Research Ethics board.

5.3.2 Instrumentation and Data Acquisition

Surface electromyography (EMG) was collected from the following eight muscles of the right upper extremity: anterior deltoid, triceps brachii, biceps brachii, brachioradialis, flexor carpi radialis, flexor digitorum superficialis, extensor carpi radialis longus and extensor digitorum communis. Following electrode site preparation that included shaving and scrubbing with alcohol, disposable bipolar Ag-AgCl surface electrodes (MediTrace 130, Kendall, Mansfield, MA, USA) were placed over each muscle belly and in line with muscle fibre orientation with an inter-electrode distance of 2.5 cm. EMG signals were band-pass filtered (10-1000 Hz) and differentially amplified (CMRR > 115 dB at 60 Hz; input impedance ~10G Ω ; Model AMT-8, Bortec Biomedical Ltd., Calgary, AB, Canada).

Each participant was fitted with an electrogoniometer to measure elbow angle (SG110, Biometrics, Ltd., Gwent, U.K.). The electrogoniometer was attached to the forearm (mid-forearm ulnar side) and upper arm (mid-humerus) with two-sided tape, centered at the medial epicondyle. A custom made pneumatic based perturbation device was equipped with a metal rod (1.0 cm diameter and 20 cm length) that extended outward in a single plane to deliver a push force to each participant's arm. A load cell (MPL-300-CO, Transducer Techniques, Temecula, CA, USA) was attached in series with the metal rod to measure the perturbation push force. EMG, electrogoniometer and load cell data

were sampled at 2048 Hz using a 16-bit analog-to-digital converter (USB-6229 BNC, National Instruments, TX, USA).

5.3.3 Experimental Procedures

Participants performed trials in three body postures and three hand loading conditions while the pneumatic device delivered a sudden perturbation to the wrist, causing the elbow to rotate into extension. Three starting postures provided different challenges to the system and all required the elbow to be flexed to 90° and included: i) lying supine on table, ii) sitting with the shoulder flexed to 90° and upper arm resting on table, and iii) standing (Figure 5.1). Note that the upper arm was aligned with the thorax in both standing and supine, but the forearm acted as an inverted pendulum in the seated and supine postures. Prior to the perturbation, participants maintained each posture under three different hand loading conditions: i) no load in the hand, referred to as “no load”, ii) holding a tube horizontally, or “solid load”, and iii) holding and balancing a water-filled tube horizontally, or “fluid load”. The solid and fluid filled tubes were identical in length (34.5 cm), diameter (2.54 cm) and mass (0.68 kg). Participants wore a wrist brace to ensure a constant neutral wrist posture and to provide a consistent rigid surface to which the perturbation force could be applied. For each condition, the perturbation device was aligned to deliver the push force at the proximal wrist crease. The brace limited wrist rotation, resulting in a perturbation that caused sudden elbow extension (Figure 5.2).

Perturbations were applied with timing both known and unknown to the participant. During known timing perturbations, the participant was given a trigger with which they could manually initiate the perturbation. During unknown timing

perturbations, the experimenter signaled the start of the trial to the participant and the perturbation occurred randomly within 10 seconds. Each body posture, hand load and timing combination was performed in a semi-random order, such that all hand loading and timing combinations were randomly performed in one posture before selection of the next posture. Three trials were performed for each testing combination and 30 seconds of rest was given between trials. Approximately 5 minutes of rest was given between postures to limit the effects of muscular fatigue and to adjust the device. In total, each participant was exposed to 54 perturbations.

Prior to the perturbation trials, maximal voluntary excitations (MVE) were determined for each muscle using muscle specific maximal voluntary isometric contractions. For each muscle, the participant held a muscle specific maximal contraction for 3 seconds. Maximal contractions were performed as follows: (i) anterior deltoid, manually resisted shoulder flexion with the arm flexed to 90°; (ii) biceps brachii and brachioradialis, manually resisted elbow flexion with the elbow flexed to 90° and forearm supinated; (iii) triceps brachii, manually resisted elbow extension in same posture as biceps brachii test; (iv) forearm flexor and extensors, combinations of wrist flexion, wrist extension, radial and ulnar deviation in combination with a maximal hand gripping task. Maximal contractions were performed twice for each muscle group and a minimum of 30 seconds rest was given between maximal exertions.

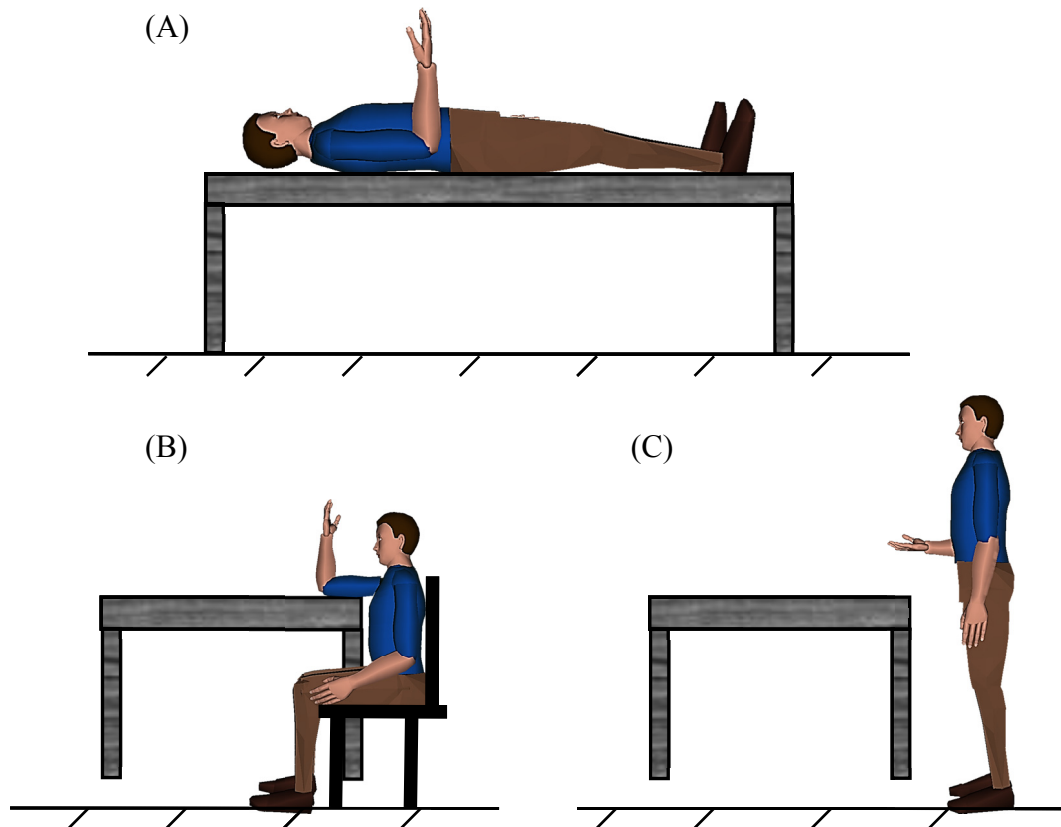


Figure 5.1 The three body orientations: A) supine, B) sitting and C) standing. Note: The perturbation device was orientated to accommodate each body orientation.

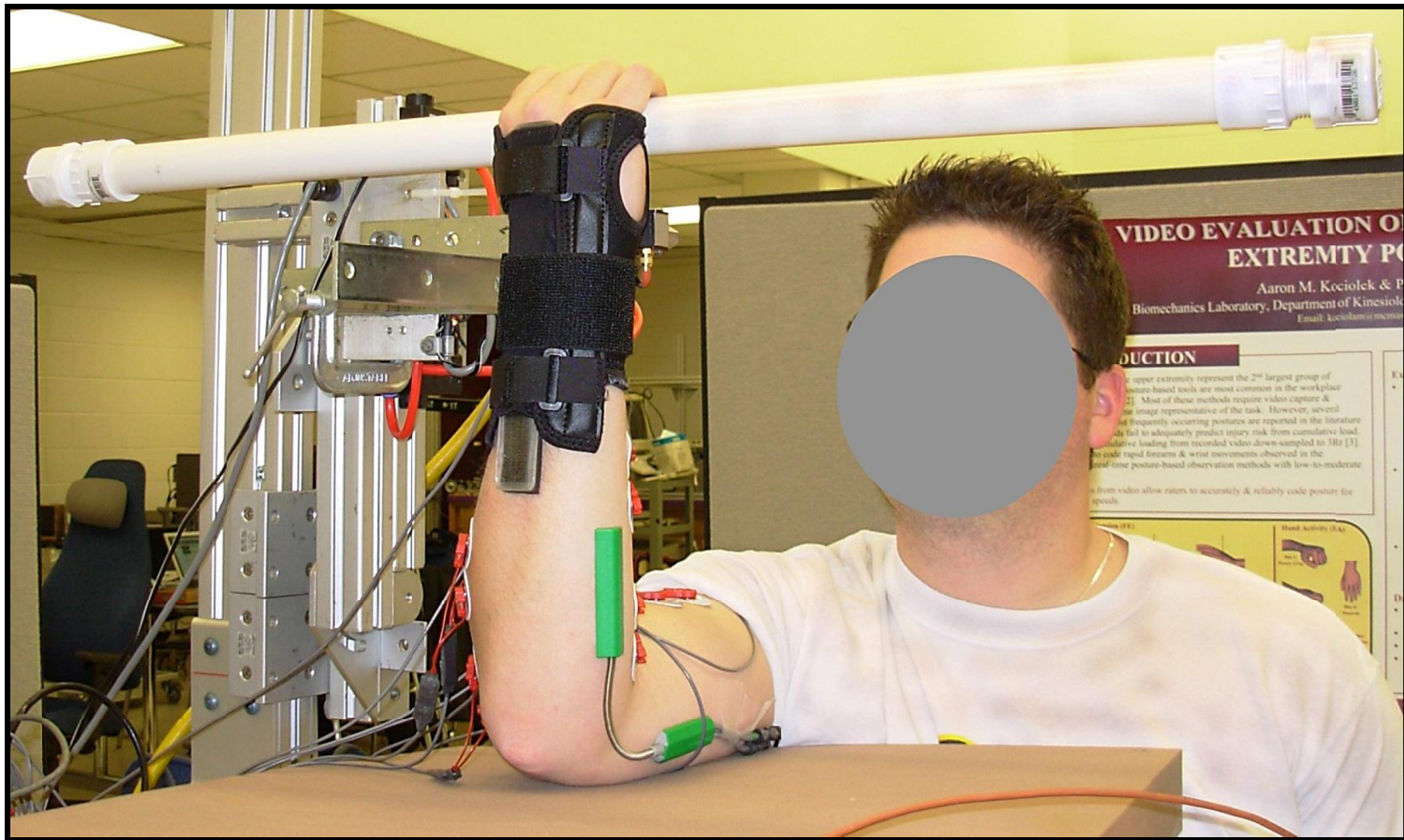


Figure 5.2 Participant preparing for a perturbation during the sitting posture and fluid hand load condition. Note: the perturbation device impacted each participant in the same location on the palmar side of the hand.

5.3.4 Data Analysis

A quiet trial was collected and used to remove signal bias from each EMG channel. All EMG signals were full-wave rectified and digitally low-pass filtered using a 2nd order Butterworth filter with a 3 Hz cutoff. Maximum activation was found for each muscle specific maximum contraction and used to normalize each EMG signal. The electrogoniometer and load cell data were digitally low-pass filtered (2nd order, dual pass Butterworth) with cutoffs of 3 Hz and 10 Hz, respectively. The perturbation device was equipped with a pressure sensor that was used to indicate the start of the perturbation. All data were investigated over a 150 ms time period prior to the perturbation. The baseline time period was defined as 150-100 ms pre-perturbation and the anticipatory time period was defined as 15-0 ms pre-perturbation. These periods were used to investigate the effects of voluntary muscular contributions. As a data reduction measure, all signals were down sampled to 128 Hz. For additional information regarding this protocol and additional EMG analysis, the reader is referred to Holmes and Keir (chapter 4).

An existing upper extremity model (Holzbaur et al., 2005) was used to apply the perturbation data in a musculoskeletal modeling software platform (OpenSIM, Delp et al., 2007). The original model had fifty muscle-tendon actuators and kinematics of the shoulder, elbow, forearm, wrist, thumb and forefinger (Holzbaur et al., 2005). The model was reduced to include only muscles crossing the elbow joint ($n = 12$, see Table 5.1). The 12 modeled muscles were: triceps brachii long head (TRI_{Lo}), triceps brachii lateral head (TRI_{La}), triceps brachii medial head (TRI_M), biceps brachii long head (BI_L), biceps

brachii short head (BI_S), brachialis (BRA), brachioradialis (BRD), extensor carpi radialis longus (ECR_L), extensor carpi radialis brevis (ECR_B), extensor carpi ulnaris (ECU), flexor carpi radialis (FCR) and flexor carpi ulnaris (FCU). The elbow joint was modeled as a 2 degree-of-freedom joint that rotates about a fixed axis passing between the center of the trochlear sulcus and the capitulum (Holzbaur et al., 2005), resulting in flexion/extension and forearm pronation/supination rotation. Elbow joint angles, from the electrogoniometer, were used as input into the upper extremity elbow model to determine each muscle's instantaneous length, velocity and moment arm for each perturbation trial. The instantaneous muscle parameters were used in combination with the EMG to evaluate muscle force generating characteristics using a Hill-type muscle model using optimal fibre length, peak force, tendon slack length and pennation angle for each muscle (Delp and Loan, 1995; Zajac, 1989). Individual muscle parameters for the upper extremity, including muscle architecture were taken from Holzbaur et al. (2005). EMG from triceps brachii was used to activate all three heads of the triceps in the model (TRI_{Lo} , TRI_{La} and TRI_M). Similarly, the collected muscle activity from biceps brachii was used to drive the modeled BI_L , BI_S and BRA muscles. Finally, ECR_L EMG was used to activate ECR_B in the model.

OpenSIM was used to obtain muscle specific three-dimensional coordinates (representing muscle origin, insertion and node/wrap points) at each time point for each trial. The calculated muscle forces from the model and the anatomical muscle coordinates (geometric orientation) were used to determine individual muscle contributions to joint rotational stiffness (Equation 1, Potvin and Brown, 2005).

$$\text{JRS}(\text{m})_Z = F \left[\frac{A_x B_x + A_y B_y - r^2_z}{l} + \frac{q r^2_z}{L} \right] \quad (1)$$

Where, JRS is the joint rotational stiffness contribution of muscle (m) about the z axis (flexion-extension) of the elbow joint. F is the calculated muscle force; l is the three-dimensional length of the muscle vector crossing the elbow joint; L represents the full three-dimensional length of the muscle; r represents the three-dimensional muscle moment arm; A_x , B_x , A_y and B_y represent three-dimensional origin (A) and insertion/node (B) coordinates with respect to the elbow joint; q represents a constant relating muscle force to muscle stiffness and was set to 10 (Potvin and Brown, 2005).

Individual muscle contributions to joint rotational stiffness, about the flexion-extension axis, were calculated at each time point and the sum of all individual muscle contributions to JRS were determined and referred to as the total joint rotational stiffness (JRS_T) for that time point. Each individual muscle contribution was then normalized to the total contribution from all muscles (JRS_T) at that time point to represent the relative contribution of each individual muscle to joint rotational stiffness (JRS_M). The average JRS_M was determined at the baseline and anticipatory time periods outlined earlier. All muscle forces and anatomical coordinate data were extracted from OpenSIM and used to calculate JRS_M using Matlab (R2008a, The Mathworks, Inc., Natick, MA, USA).

Table 5.1: List of muscles crossing the elbow joint in the model. The corresponding muscle lengths (cm) and moment arms (cm) are given for the arm at 90° (pre perturbation). Note: These data represent the standing and supine postures, representing a 50th percentile male (170 cm tall).

Muscle (abbreviation)	Muscle Length (cm)	Flexion Moment Arm (cm)
Triceps brachii long head (TRI _{Lo})	31.03	-1.62
Triceps brachii lateral head (TRI _{La})	19.74	-1.62
Triceps brachii medial head (TRI _M)	18.82	-1.62
Biceps brachii long head (BI _L)	35.89	3.72
Biceps brachii short head (BI _S)	28.81	3.72
Brachialis (BRA)	12.65	2.27
Brachioradialis (BRD)	27.67	6.11
Extensor carpi radialis longus (ECR _L)	26.13	2.27
Extensor carpi radialis brevis (ECR _B)	22.42	0.13
Extensor carpi ulnaris (ECU)	23.64	-0.12
Flexor carpi radialis (FCR)	21.84	1.24
Flexor carpi ulnaris (FCU)	22.83	1.25

5.3.5 Statistical Analysis

Data were averaged across the three trials for each condition. For each muscle, a 2 x 3 x 3 x 2 repeated measures ANOVA was used to evaluate the effects of perturbation timing knowledge (known and unknown timing), posture (sitting, supine and standing), hand load (no load, solid and fluid) and time period (baseline and anticipatory) on the calculated JRS_M. Significant effects were further evaluated using a pair-wise comparison with Bonferroni correction (SPSS v13.0, IBM Corporation, Somers, NY, USA). An alpha level of 0.05 was used for all statistical analyses.

5.4 Results

5.4.1 Perturbation Force and Elbow Flexion Angle

The change in posture had a slight influence on the perturbation magnitude. The mean maximum push force (\pm standard deviation) across all trials was 60.0 ± 12.3 N. Standing had a significantly greater maximum push force (74.4 ± 8.5 N) than the sitting (58.3 ± 4.4 N) and supine (52.5 ± 8.1 N) postures ($p < 0.05$). There were no differences in push force for the hand load and timing knowledge conditions.

Participants successfully maintained the initial posture prior to each perturbation regardless of posture and hand loading conditions. The average arm posture during the baseline and anticipatory time periods was $90.5 \pm 0.56^\circ$ and $89.1 \pm 0.40^\circ$ of elbow flexion, respectively.

5.4.2 Individual Muscle Contributions to Joint Rotational Stiffness (JRS_M)

JRS_M about the flexion/extension axis were averaged for all conditions during the baseline time period and represented in Figure 5.3 to give a general representation of individual muscle contributions to elbow JRS prior to a perturbation. The largest JRS_M about the flexion/extension axis was BRA with a contribution of $30.4 \pm 1.9\%$ JRS_T . The next largest contribution was BRD ($21.7 \pm 2.2\%$ JRS_T), followed by BI_S and BI_L at, $19.7 \pm 0.8\%$ and $15.5 \pm 1.2\%$ JRS_T , respectively (Figure 5.3). In total, the contribution of all three heads of the triceps brachii (TRI_{Lo} , TRI_{La} and TRI_M) at baseline was $9.2 \pm 1.9\%$ JRS_T . The contribution of all forearm muscles (ECR_L , ECR_B , ECU , FCR and FCU) was $5.5 \pm 0.6\%$ JRS_T , with the majority from the extensors.

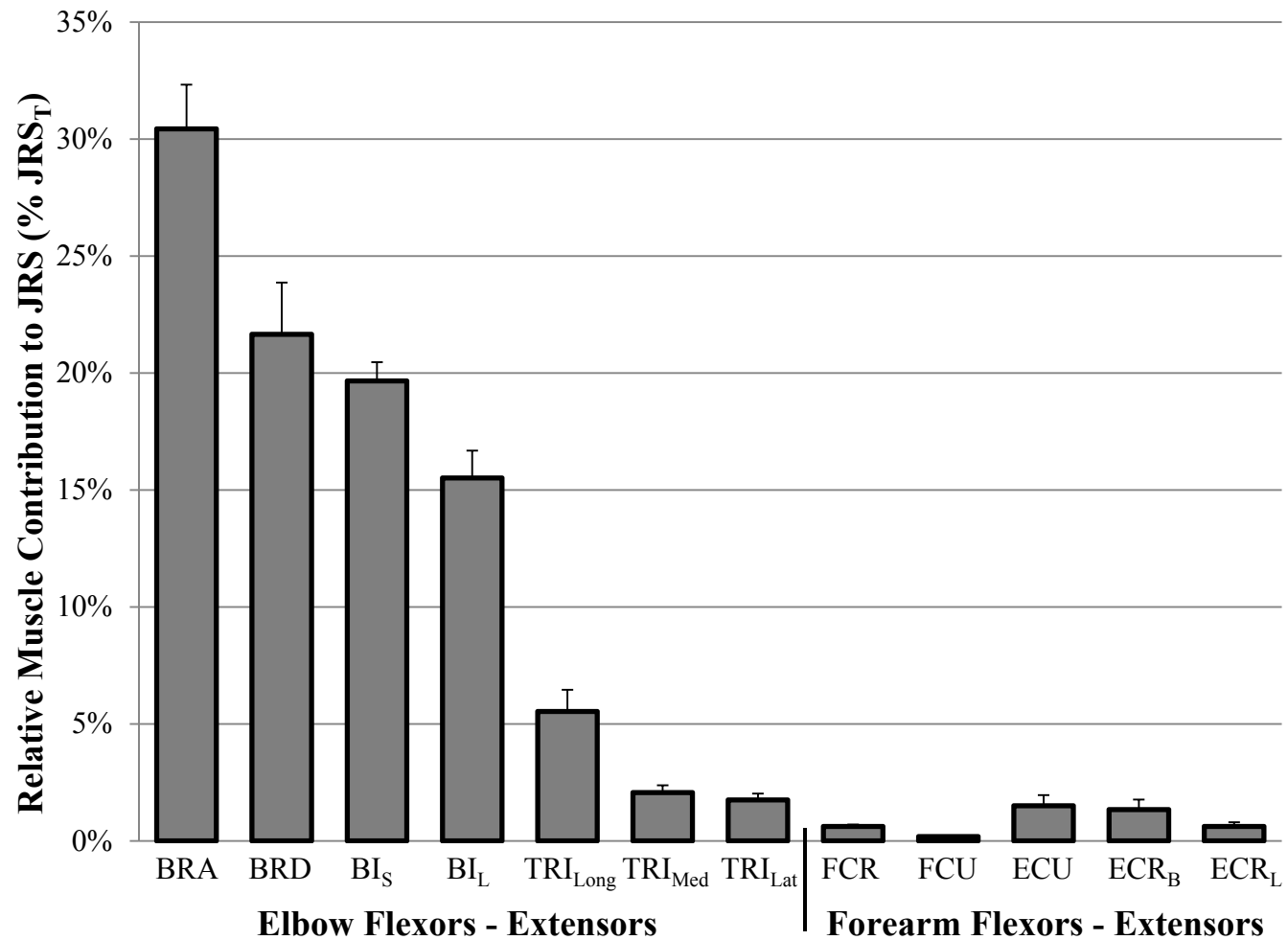


Figure 5.3 Mean JRS_M (%JRS_T with standard deviation) for all muscles during the baseline time period averaged across all posture, hand loading and perturbation timing knowledge conditions. See Table 5.1 for muscle abbreviations.

The changes in JRS_M across experimental conditions were functionally small, however statistically significant differences were found. Eight of the modeled muscles (BRA, BRD, BI_L , BI_S , FCU, FCR, ECU and ECR_B) demonstrated a significant posture x load interaction (all $p < 0.002$) (Figure 5.4). BI_S had a greater contribution during standing than the supine posture, regardless of hand loading condition. During sitting and supine postures, the BRA contribution was larger with no load than solid and fluid conditions. During standing, JRS_M for BRA (no load condition) increased by 4.2% JRS_T from sitting and by 5.0% JRS_T from supine (Figure 5.4A). The contribution from BRD during standing with no hand load was 18.5% JRS_T and increased to 23.8% JRS_T , with the solid load and to 26.3% JRS_T with the fluid load. For ECU and ECR_B , the standing posture resulted in the lowest JRS_M , regardless of hand load (Figure 5.4B). For ECU during the solid load conditions, JRS_M was 1.6% and 1.7% JRS_T for the sitting and supine postures, and decreased to 0.8% JRS_T for standing. For ECR_B the greatest differences were found with the fluid hand load. JRS_M was 1.7% and 1.5% JRS_T for the sitting and supine postures, and decreased to 1.0% JRS_T for standing.

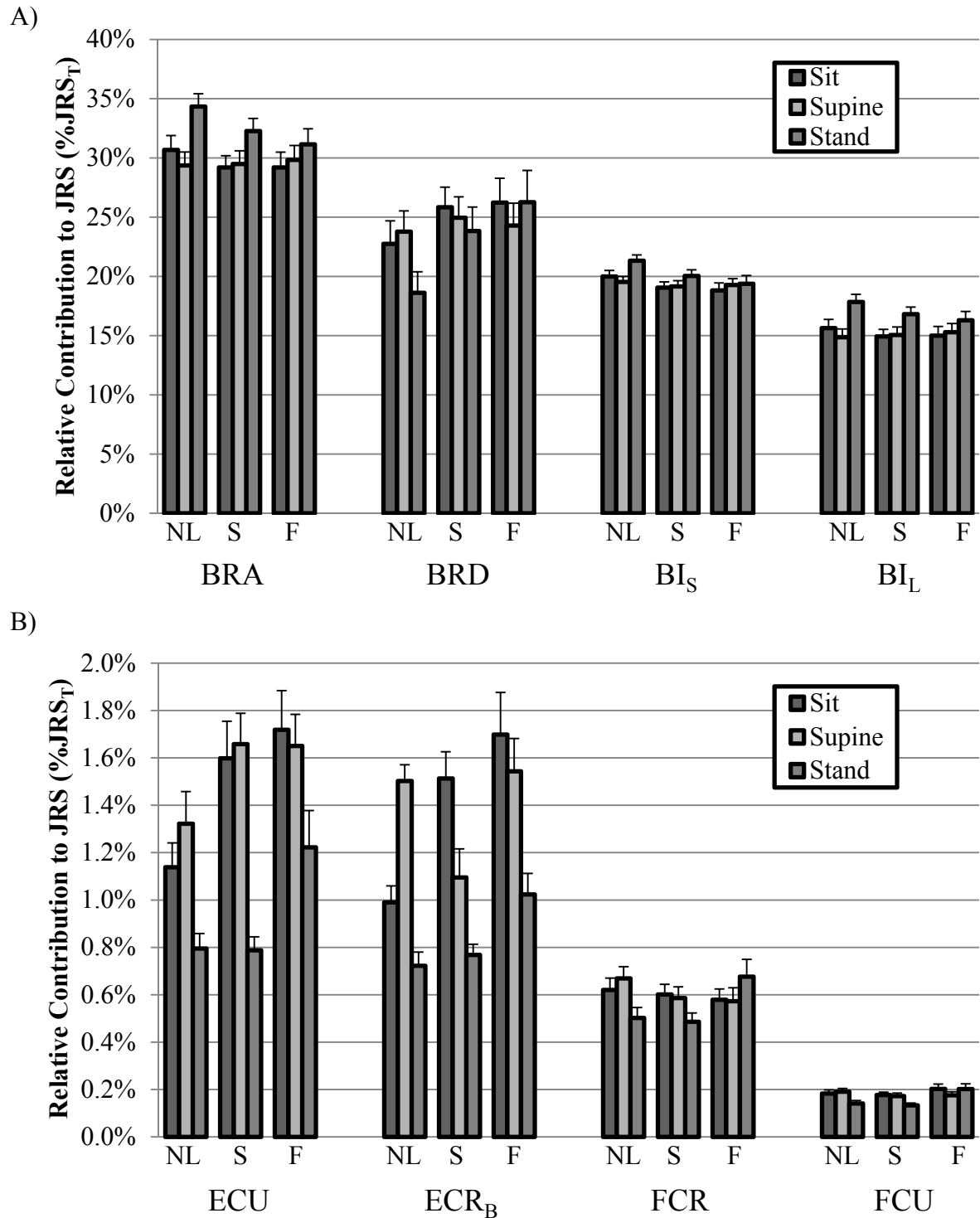


Figure 5.4 Mean JRS_M (%JRS_T with standard deviation) for each hand loading task during each posture for A) The primary elbow flexor muscles, B) The forearm flexors and extensors. See Table 5.1 for muscle abbreviations.

The primary elbow flexors (BRA, BRD, BI_L and BI_S) demonstrated a significant posture x time period interaction (all, $p < 0.023$). JRS_M for BRA, BRD, BI_L and BI_S was consistently greater during the anticipatory time period than at baseline (Figure 5.5). The largest contributions were found during the standing posture, where JRS_M for BI_S increased by 7.4% and BRD increased by 2.4% during the anticipatory period (Figure 5.5). Figure 5.5 highlights the time period x posture interaction for the elbow flexor muscles.

Three of the forearm muscles (ECU, ECR_L and FCR) demonstrated a significant hand load x time period interaction (all $p < 0.002$). JRS_M for ECU, ECR_L and FCR all demonstrated a greater contribution during the anticipatory time period than at baseline (Figure 5.6). ECR_L had the largest increase in JRS_M from the baseline to anticipatory time period, but was only 0.5%. With the fluid hand loading task, ECU and ECR_B demonstrated the largest increase in JRS_M from the baseline to anticipatory time period, yet the increase was only 0.3% (Figure 5.6). Figure 5.6 highlights the forearm muscles during time period and hand loading conditions.

The elbow extensors (TRI_{Lo}, TRI_{La} and TRI_M) were the only muscles to demonstrate a significant main effect of perturbation timing knowledge (all $p < 0.006$) with JRS_M being greater when perturbation timing was known. Posture also had a significant main effect on JRS_M for all heads of the triceps (all $p < 0.001$). Further comparisons revealed that an opposite trend to the elbow flexors was found for posture. Both the sitting and supine postures resulted in a significantly greater contribution than the standing posture ($p < 0.005$ and $p < 0.001$, respectively).

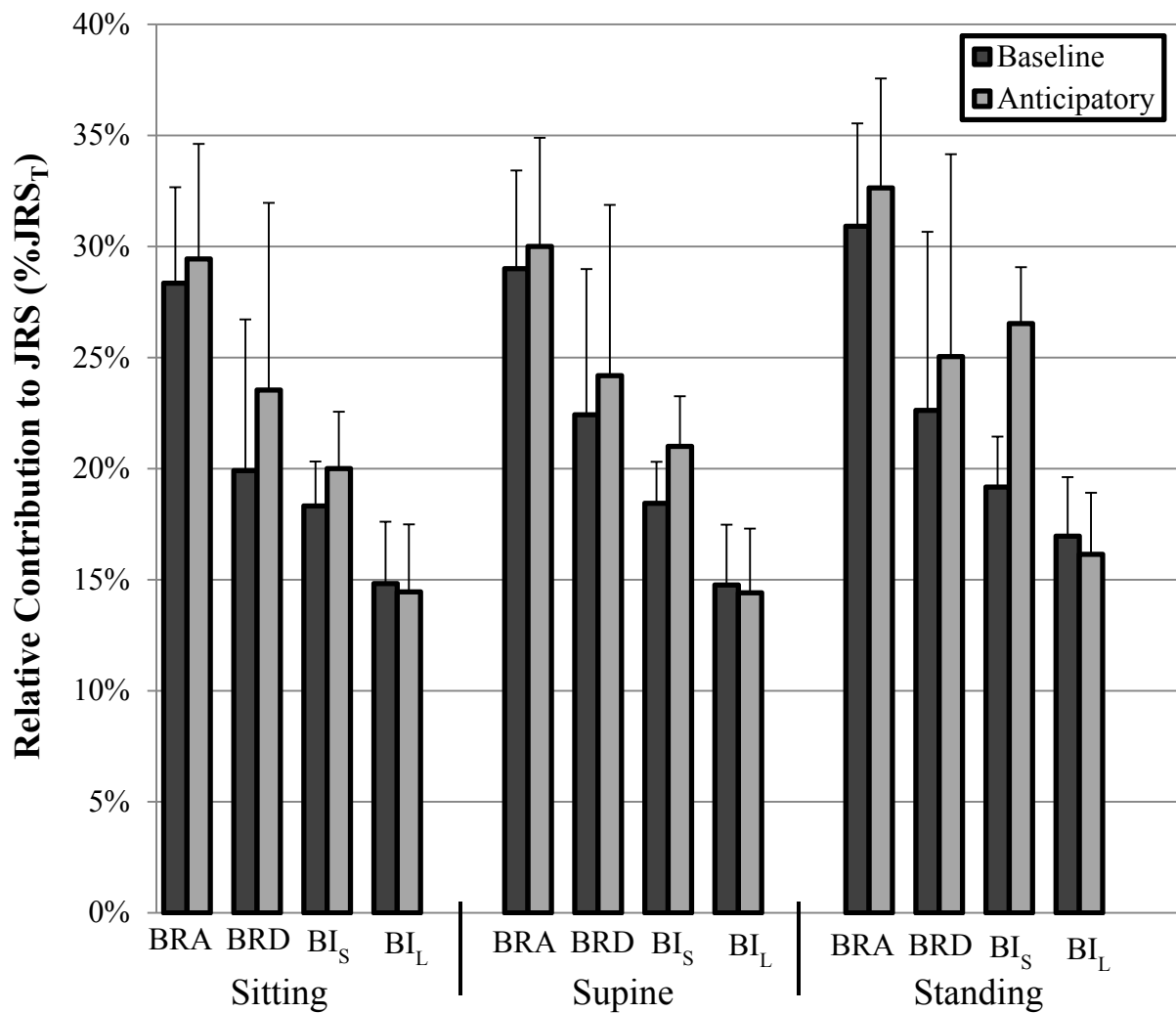


Figure 5.5 Mean JRS_M (% JRS_T with standard deviation) for the primary elbow flexor muscles during the two time periods for each body posture. See Table 5.1 for muscle abbreviations.

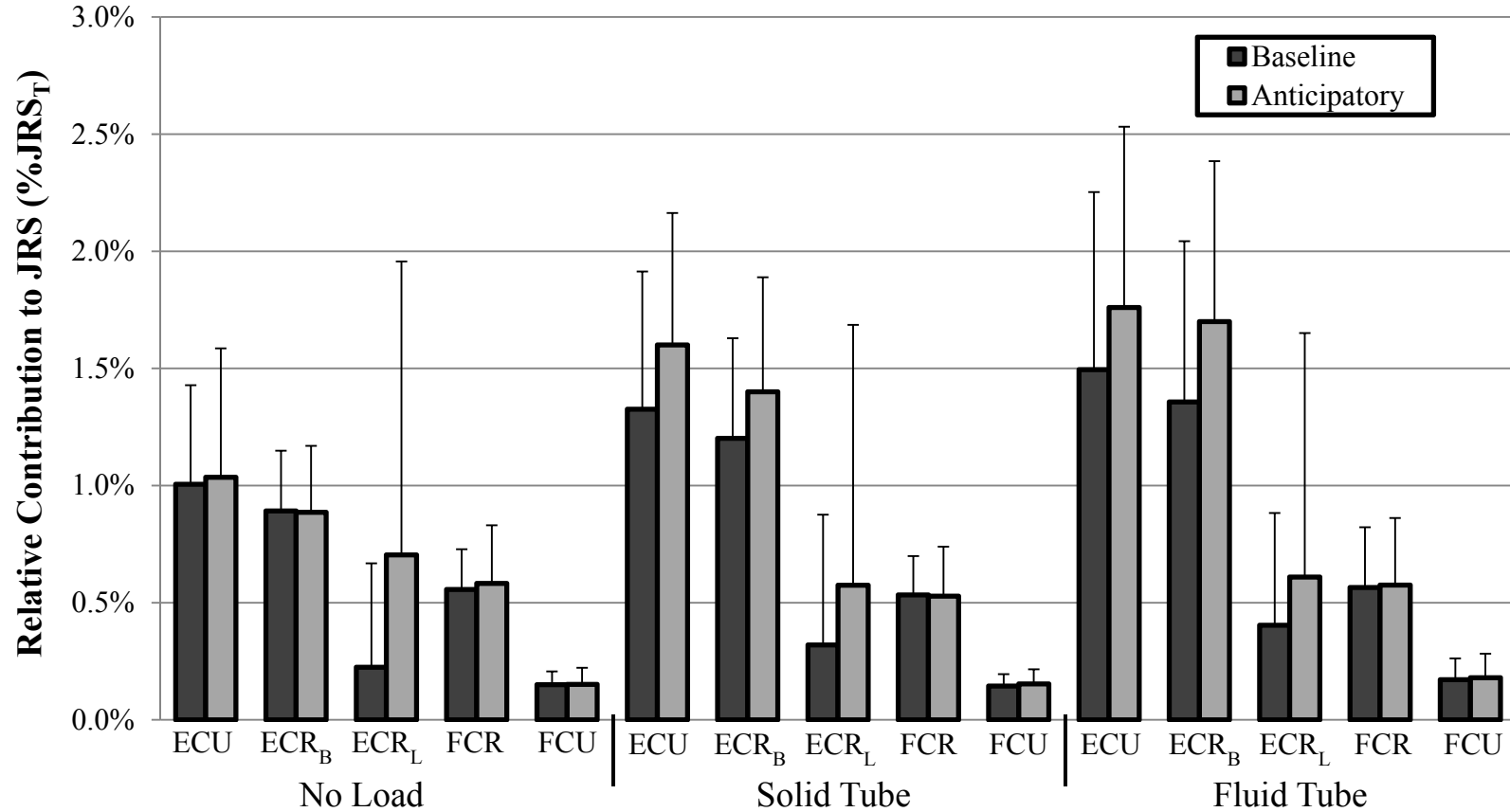


Figure 5.6 Mean JRS_M (%JRS_T with standard deviation) for the forearm flexor-extensor muscles during the two time periods for each hand load. See Table 5.1 for muscle abbreviations.

5.5 Discussion

This study applied sudden expected and unexpected perturbations to the arm of participants in three body postures, which resulted in sudden elbow extension. The current investigation quantified individual upper extremity muscle contributions to joint rotational stiffness during two time periods immediately prior to a sudden perturbation. It was found that regardless of the experimental condition, the primary elbow flexor muscles (BRA, BRD, BI_L and BI_S) consistently had the largest JRS_M and thus provided the greatest influence on mechanical joint stability at the elbow during sudden perturbations of elbow extension. Also, the triceps brachii and forearm muscles contributed marginally to JRS. The primary elbow flexors, and to a lesser extent, the forearm muscles, all demonstrated contributions that were greater immediately prior to the sudden perturbation, which helped stiffen the joint. This is a commonly suggested neuromuscular strategy demonstrated in spine research (Stokes et al., 2000; Brown et al., 2003), but not typically reported in the upper extremity. This study provides new detailed information about individual muscle responses to sudden elbow loading. The forearm muscles were found to be capable of contributing stiffness at the elbow joint, which has not traditionally been considered.

Across all experimental trials, BRA somewhat unexpectedly demonstrated the greatest JRS_M (Figure 5.3). The stiffening potential for a muscle is largely dependent on its geometrical orientation to the joint as is evident from the moment arm being squared in Equation 1 (Potvin and Brown, 2007). While this “geometric stability” has the greatest influence on a muscle’s stiffening potential, the force generating capacity and thus, cross

sectional area (CSA) of a muscle will also help provide stiffness. It was surprising to find that BRA had the largest JRS_M , because in our model the BRA flexion moment arm was approximately 1.6 and 2.7 times smaller than the biceps brachii (BI_L and BI_S) and BRD muscles, respectively. BRA had the smallest moment arm of all major elbow flexors (Table 5.1). However, peak isometric muscle force for BRA was 2.3 and 3.7 times greater than the biceps brachii and BRD muscles, respectively. Another important part of the geometric component for this equation relates to muscle length, and at baseline (starting posture with an elbow flexion angle of 90°), BRA had a relatively short muscle length (Table 5.1). Its short length, in combination with a large CSA and peak isometric force generating potential, suggests that BRA is an important stabilizer for the elbow. Some classic work (Basmajian, 1978; MacConaill, 1946) suggested that muscles which act across the long axis of the forearm (i.e. BRA) did not have a joint stabilizing component when compared to muscles that act more parallel to the forearm (i.e. BRD). Our study did not find this, which is in agreement with Buchanan et al. (1986) who also failed to see this relationship while investigating synergistic relationships of the elbow muscles during isometric contractions. Additionally, Basmajian and De Luca (1985) suggested that BRA was the “workhorse” for elbow flexion activities and according to our findings it appears that BRA is also the primary stabilizer for elbow flexion/extension tasks.

In our model, BRD had the largest elbow flexion moment arm and thus it was not surprising that this muscle contributed significantly to elbow JRS. However, it was generally the second largest contributor to elbow JRS, which is interesting given the

supinated hand posture required by all participants in this study. BRD provides elbow flexion, but is a large contributor to forearm pronation/supination and produces optimal force in a neutral forearm posture. A supinated forearm posture was required by all participants and it is likely that BRD would have had an even greater contribution if we were to test a neutral forearm posture. This emphasizes how knowledge of initial posture and the geometrical orientation of a muscle are of particular importance when evaluating individual muscle contributions to joint rotational stiffness.

Chadwick and Nicol (2000) demonstrated that ECR_L has a comparable elbow flexion moment arm to BRA (also evident in our study, Table 5.1) and suggested that ECR_L should contribute to elbow flexion. Besides ECR_L , the other forearm muscles in our model had relatively small elbow flexion moment arms compared to the other elbow flexors and our study found that the total forearm flexor and extensor contribution to elbow JRS was only $5.5\% \pm 0.6\%$ (Figure 5.3). However, our hand loading task was meant to provide a challenge to the system and not substantially load the forearm muscles. As evident from our EMG analysis (Holmes and Keir, Chapter 4), we found increases in forearm muscle co-contraction due to hand loading, but generally the activations were below 10% MVE. It was surprising to find that ECR_L had a small contribution, even less than ECR_B , given its larger moment arm. Our muscle force estimates are a reflection of our surface EMG recordings, perhaps suggesting that our modeled ECR_B was activated to a greater extent than it may have been during our hand loading tasks (activation taken from ECR_L). Our results suggest that the forearm muscles are capable of providing JRS_M at the elbow; however the magnitude of these

contributions were small. With greater demands at the hand and wrist, greater effects would be expected.

Although the magnitude of our changes in JRS_M , due to arm posture and hand loading, were relatively small, the findings demonstrate that task demands will influence the magnitude of JRS_M . This may suggest that larger muscular demands to the system would have increased the magnitude of these changes, making the findings more functionally meaningful. However, due to the demands of our study, the elbow flexors were influenced the most by postural change and generally demonstrated the greatest increase in JRS_M during standing. The forearm flexors demonstrated the largest differences due to hand loading, with the fluid and solid loads providing slightly greater JRS_M than the no hand loading task. This demonstrates that an increased muscular demand was necessary to stabilize the more challenging fluid filled load. This has also been confirmed at the wrist joint with increasing load instability (De Serres and Milner, 1991).

Time period influenced most muscles in our model. When knowledge of the timing was given, the majority of our modeled muscles consistently demonstrated greater JRS_M during the anticipatory time period than at baseline. This shows that during elbow extension perturbations our participants provided a voluntary neuromuscular response immediately prior to the perturbation that helps stiffen the elbow and stabilize the joint. This is in agreement with other elbow work (Zhang and Rymer, 1997) and also spine perturbations (Brown et al., 2003; Granata et al, 2001). However, to our knowledge this is the first study to document the individual muscle contributions during two preparatory

phases of a perturbation protocol for the upper extremity. As might be expected, we found that BI_S and BRD provided the greatest individual increase immediately prior to the perturbation, which likely represented a slight resistance to the extension perturbation and contributes significantly to limiting the resultant joint motion.

Stokes and Gardner-Morse (2000) investigated elbow joint stability with vertical and horizontal loading conditions and concluded that variations in strategies used to stabilize the elbow joint would have a trade-off between stability and physiological cost. Perturbation timing knowledge will influence joint stiffness prior to a perturbation (Aruin and Latash, 1995, Chiang and Potvin, 2001), but surprisingly, only the triceps brachii muscles were influenced by timing knowledge in our study. This may be the result of a potential learning effect (Osu et al. 2002; Thoroughman and Shadmehr, 1999) due to the large number of perturbations in our protocol and that participants knew the magnitude and direction of our perturbation. The participant likely became familiar with the perturbation direction and adopted a neuromuscular strategy that increased muscular contributions during the reflex time period to help limit the magnitude of joint rotation during sudden unknown perturbations. Future work with unexpected perturbation directions could provide additional information about muscular strategies to help provide joint stability.

There are a few limitations to this work that should be discussed. First, the JRS calculation is dependent on the geometrical orientation of muscle coordinates (Brown and Potvin, 2007; Potvin and Brown 2005). Our results are dependent on the anatomical coordinates of the model (Holzbaur et al., 2005), which is an approximation of an

average adult male and was not scaled to participant anthropometrics. Second, the elbow is a highly redundant system, with many muscles crossing the joint, causing rotation about the flexion/extension axis. Numerous authors have developed models of the elbow (Fisk and Wayne, 2009; Gonzales et al., 1996; van der Helm, 1997; Veeger et al., 1997) while others have recommended optimization strategies and cost functions for the load sharing distribution problem (Buchanan et al., 1989; Dul et al., 1984; Kaufman et al., 1991; Praagman et al., 2010). Our analysis used an EMG driven muscle force analysis. Variability in our JRS calculations will be apparent when using different EMG-force modeling approaches, but we have only expressed relative contributions for each muscle, and the JRS results are more dependent on geometrical orientation rather than force estimates.

5.6 Conclusions

The primary elbow flexor muscles dominated joint rotational stiffness at the elbow while the forearm and triceps brachii muscles provided a small contribution. The forearm and elbow flexor muscles demonstrated increases in JRS_M due to time period, which suggests a neuromuscular preparation strategy for ensuring elbow joint stability. Sudden arm perturbations are common in occupational settings and if the muscular response to a sudden disturbance is poorly coordinated, the consequence may be joint or tissue injury. This study has documented which muscles are best suited to provide a stabilizing contribution to a sudden arm disturbance, while also finding that for the conditions tested, the forearm musculature provides limited support. This is the first study to document forearm contributions to joint stiffness at the elbow and the

investigation of larger forearm loads (experienced during gripping tasks) may reveal larger forearm contribution at the elbow. It is likely that the forearm muscles are activated to what may be required for a task, simply due to a stabilizing role. This may improve our understanding of cumulative loading and overuse injuries of the forearm.

5.7 Acknowledgements

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CHAPTER 6

**STUDY 4: FOREARM MUSCLE CO-CONTRACTION AND
INDIVIDUAL MUSCLE CONTRIBUTIONS TO JOINT
ROTATIONAL STIFFNESS DURING FLEXION AND EXTENSION
WRIST PERTURBATIONS**

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6.1 Abstract

A significant component of joint stiffness is related to muscle co-contraction. In the distal upper extremity, muscular contributions to joint stiffness are essential for the control and stabilization of objects interacting with the hand. The purpose of this study was to investigate the influence of a gripping task on forearm muscle co-contraction as well as individual muscle contributions to joint rotational stiffness prior to wrist flexion and extension perturbations. Ten male participants performed a sub-maximal gripping task (no grip, 5% and 10% of maximum) while a perturbation caused wrist flexion or extension. Wrist joint angles and activity from eleven upper extremity muscles were collected and used as input into a biomechanical model to determine the contribution of each muscle to wrist joint rotational stiffness. The response to sudden loading was examined at two time periods prior to perturbation (baseline and anticipatory). Increased co-contraction was found as grip force requirement increased, corresponding to a 36% increase in overall wrist joint stiffness from no grip to 10% grip. There was an increase in wrist joint stiffness during the anticipatory period (15 ms before perturbation), demonstrating a neuromuscular response to stiffen the joint. The largest individual joint rotational stiffness contribution was from extensor carpi radialis longus and brevis, with contributions of $34.5 \pm 1.3\%$ and $20.5 \pm 2.3\%$, respectively. The greatest contributors to joint rotational stiffness were consistent across conditions, suggesting that all muscle contributions were enhanced, rather than a redistribution of muscle requirements. This study provides insight into how individual forearm muscles modulate wrist joint stiffness. Consideration of these findings can lead to an understanding of how muscles maintain

joint stability, and why specific muscles may become injured during sudden loading events, due to their requirement to help stiffen the joint.

Keywords: Forearm, Wrist, Joint Rotational Stiffness, Joint Stability, Biomechanical Modeling

6.2 Introduction

The forearm and hand represents a redundant musculoskeletal system with a complex arrangement of muscles that must be elegantly coordinated to perform many activities of daily living. To interact with our environment, the forearm musculature transfers loads across the wrist joint and must balance moments created by the flexor and extensor muscle groups. If muscle actions are uncoordinated, imbalances about the wrist may lead to joint instability and injury. Approximately twenty-six muscles cross the wrist joint, providing an additional level of difficulty for the neuromuscular system and for the maintenance of joint stability as many of the muscles provide similar or redundant actions. Work-related musculoskeletal disorders can occur from either sudden (acute) trauma, or low level, continuous loading over long periods of time (Kumar, 1990). Quantifying the potential stabilizing contributions from individual muscles could enhance our current knowledge of wrist joint loading and improve the understanding of hand and forearm injury risk.

Latash and Zatsiorsky (1993) suggested that joint stiffness is modulated by individual contributions from muscles, tendons, ligaments and bones. Thus, a detailed evaluation of joint stability requires knowledge of individual musculoskeletal components that regulate joint stiffness. At the wrist, medical conditions (such as malalignment of the carpal bones) can result in an unstable wrist (Garcia-Elias, 1997), causing carpal bone instability and financial burden to the health care system (Dias and Garcia-Elias, 2006). The carpal ligaments are important to carpal stability (Guo et al., 2009; Holmes et al., 2011; Mayfield et al., 1976; Short et al., 2007), however, a better

understanding of muscular contributions to joint stiffness is also needed. Panjabi (1992) offered that passive tissues of the spine are not able to maintain joint stability during disturbances to a system, thus, muscular contributions are required. Crisco et al. (1992) demonstrated that the spinal system would buckle under relatively small compressive load if only passive tissues contributed to spinal stability. While these findings are not explicitly transferable to the upper extremity, muscular contributions can reduce stress on the carpal ligaments, enhance wrist joint stiffness and carpal stability (Gofton et al., 2004; Tsai, 2009), which is favourable for joint safety and for reducing injury during movements and tasks that load the carpal structures. Further, muscular contributions are essential for the control and stabilization of objects interacting with the hand.

Muscle co-contraction is related to an increase in joint stiffness, predominantly due to the relationship between muscle activity and stiffness (Cholewicki and McGill, 1996; Darainy et al., 2004; De Serres and Milner, 1991). Large increases in co-contraction of the wrist extensors have been observed during gripping (Mogk and Keir, 2003) to help stabilize the wrist joint (De Serres and Milner, 1991; Snijders et al., 1987). Furthermore, hand posture and grip force have large effects on forearm muscle activity (Cort et al., 2006; Mogk and Keir, 2003). However, the majority of these forearm evaluations involve isometric gripping tasks and static postural demands. To date, there has been limited information on forearm muscle activity in preparation for sudden externally applied loads that causes involuntary wrist rotation.

Potvin and Brown (2005) demonstrated that the magnitude of individual muscle contributions to joint rotational stiffness can be determined with knowledge of the origin

and insertion coordinates of a muscle relative to the joint, muscle force, and muscle stiffness. Thus, it is apparent that the evaluation of muscle co-contraction via EMG may not provide all the required information to draw conclusions about joint stiffness. As a result, the Potvin and Brown (2005) equation provides a relatively simplified approach to evaluate individual muscle contributions to joint rotational stiffness. Typically, wrist joint stability has been evaluated at the endpoint of a sudden perturbation or movement (De Serres and Milner 1991; Franklin et al., 2003; Milner et al. 1995) with limited knowledge of how the individual muscles contribute to regulate overall joint stiffness. Due to the redundancy of the forearm musculature and potential for uncoordinated movements, understanding individual muscle contributions to wrist joint stiffness can provide information on how the muscular system modulates joint stiffness during sudden loading.

The purpose of this study was to quantify forearm muscle co-contraction and joint rotational stiffness during sudden perturbations of wrist flexion and extension. Three gripping demands were evaluated to better understand how forearm loading affects the muscular contributions to joint stiffness.

6.3 Methods

6.3.1 Participants

Ten right-hand dominant male volunteers with no history of musculoskeletal injury to the upper extremity participated in this study. Participant age, height, mass, arm length and maximum grip force can be found in Table 6.1. This study was approved by the McMaster University Human Research Ethics board. Each volunteer provided informed written consent prior to participation.

Table 6.1: Mean participant anthropometrics and maximum grip strength (standard deviation).

Anthropometrics	Mean \pm SD
Age (years)	22.7 \pm 2.7
Height (m)	1.78 \pm 0.06
Mass (kg)	77.0 \pm 11.3
Forearm Length (cm)	28.1 \pm 1.4
Hand Length (cm)	23.6 \pm 8.4
Max Grip (N)	502.2 \pm 88.2

6.3.2 Experimental Procedures

Participants performed a sub-maximal gripping task while a pneumatic perturbation device provided a push force, causing wrist rotation. Each participant stood upright next to a table (approximately waist height) with their feet placed at shoulder

width. The right arm was positioned above the surface of the table with 90° elbow flexion, 0° shoulder flexion and abduction, forearm mid-prone and wrist in a neutral posture. Participants held a grip dynamometer (MIE Medical Research Ltd., Leeds, UK; mass = 450 g) that had a light weight PlexiglasTM apparatus (mass = 210 g) attached to provide a consistent rigid target to apply the perturbation, resulting in no contact with the hand (Figure 6.1).

The rod of the perturbation device rested against the grip apparatus and was positioned in two locations to deliver a push force that caused wrist flexion and wrist extension. Restraints allowed the forearm to rest comfortably on a padded surface while restricting wrist and forearm movement for undesired off-axis rotations. The restraints and pneumatic device could be adjusted to accommodate individual anthropometrics such that the perturbation was applied in the same location for each participant.

Prior to a perturbation, participants performed different gripping tasks, which included: i) holding the dynamometer with no grip requirement, ii) maintaining a 5% of maximum grip force, and iii) maintaining a 10% of maximum grip force. The dynamometer had a fixed grip span of 5 cm and visual feedback corresponding to the grip threshold was provided via an onscreen target located on a monitor in front of the participant. Grip precision was set to $\pm 1.5\%$ of maximum for each target level. No penalty was given for poor grip performance; however participants could easily maintain the $\pm 1.5\%$ of maximum criterion. Visual feedback was provided with custom software (LabView 8.5, National Instruments, TX, USA).

Perturbations were applied with timing both known and unknown to the participant. During known timing perturbations, the participant was given a manual trigger and could initiate the perturbation when desired using their left hand. During unknown timing perturbations, the experimenter signalled the start of the trial to the participant and the perturbation occurred randomly within 10 seconds. Perturbation direction was performed in a semi-random order, such that all gripping and timing conditions were completed in one direction before completing the second direction. Three trials were performed for each combination of perturbation direction, grip trial and timing condition with 30 seconds of rest given between trials. Approximately 5 minutes rest were given between the flexion and extension perturbation directions to limit the effects of muscular fatigue and to adjust the device.

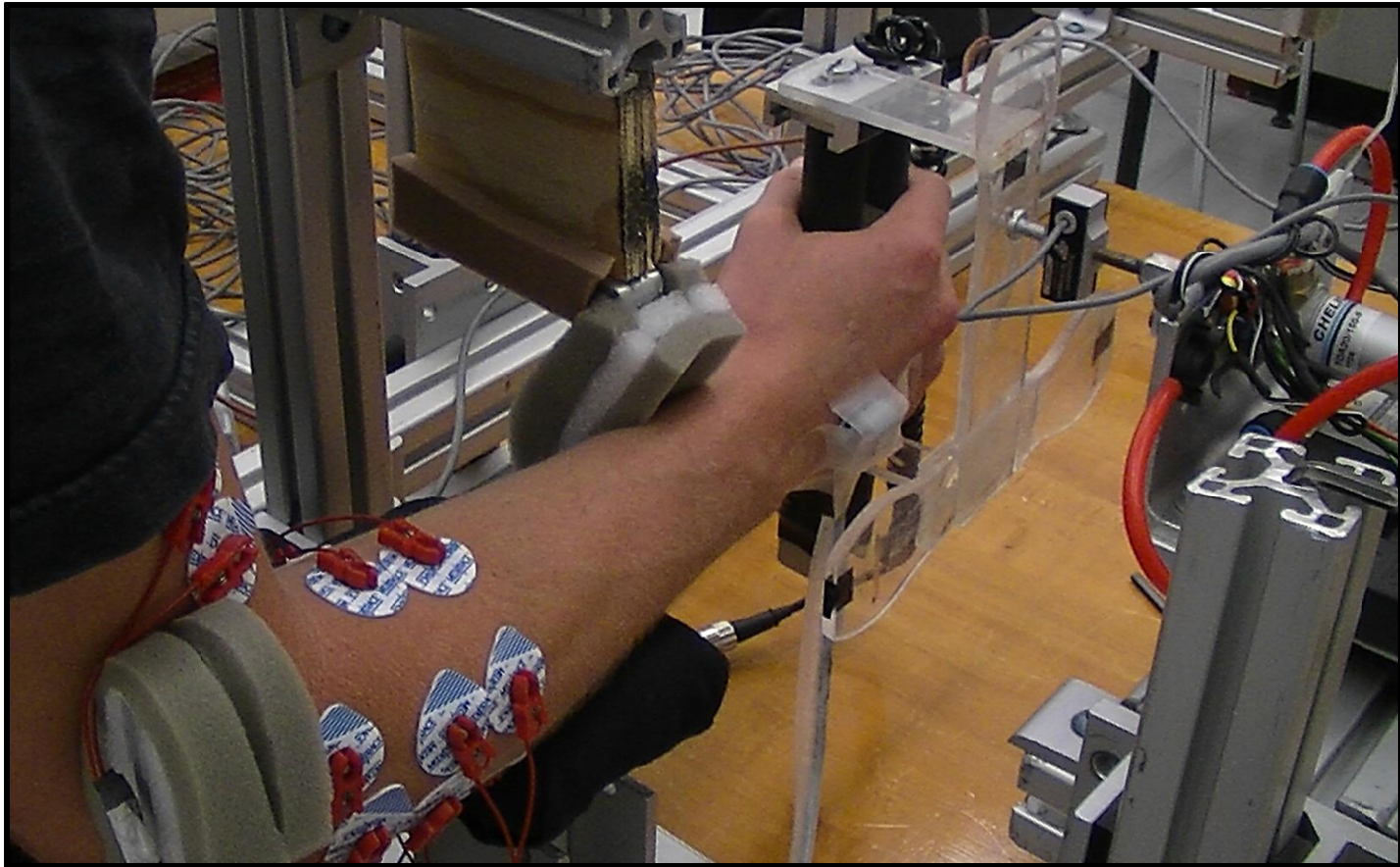


Figure 6.1 Participant preparing for a perturbation. The perturbation device (and load cell) could be adjusted to impact the same location for each participant.

6.3.3 Data Collection and Instrumentation

Surface electromyography (EMG) was collected from eleven muscles of the right upper extremity: triceps brachii lateral head (TB), biceps brachii long head (BB), brachialis (BRA), brachioradialis (BRD), flexor carpi radialis (FCR), flexor carpi ulnaris (FCU), flexor digitorum superficialis (FDS), extensor carpi radialis longus (ECRL), extensor carpi radialis brevis (ECRB), extensor carpi ulnaris (ECU) and extensor digitorum communis (ED). Following electrode site preparation that included shaving and scrubbing with alcohol, disposable bipolar Ag-AgCl surface electrodes (MediTrace 130, Kendall, Mansfield, MA, USA) were placed over each muscle belly and in line with muscle fibre orientation with an inter-electrode distance of 2.5 cm. EMG signals were band-pass filtered (10-1000 Hz) and differentially amplified (CMRR > 115 dB at 60 Hz; input impedance ~10G Ω ; Model AMT-8, Bortec Biomedical Ltd., Calgary, AB, Canada).

Following electrode preparation, a quiet EMG trial was collected and maximal voluntary excitations (MVE) were determined for each muscle using muscle specific maximal voluntary isometric contractions (MVC). For each muscle, the participant held the muscle specific maximal contraction for 3 seconds. Maximal contractions were performed twice for each muscle group and a minimum of 30 seconds rest was given between maximal exertions.

The perturbation device was equipped with a metal rod (1.0 cm diameter and 20 cm length) that extended outward in a single plane to deliver a push force to the gripping apparatus. A load cell (MPL-50-CO, Transducer Techniques, Temecula, CA, USA) was attached in series with the metal rod to measure the perturbation push force. All EMG,

grip force and load cell data were sampled at 2048 Hz using a 16-bit analog-to-digital system (USB-6229 BNC, National Instruments, TX, USA). Hand posture was collected using an electromagnetic motion tracking system (FASTRAK[®], Polhemus Ltd., Colchester, VT, USA). A sensor was attached to the mid-point of the dorsal aspect of the hand (approximately third metacarpal, based on Wigderowitz et al., 2007) using double sided tape and calibrated in the neutral starting posture. The sensor position was selected from pilot testing that determined a location with limited skin movement and would not interfere with the grip apparatus. Hand position and orientation were sampled at 100 Hz and synchronized with the EMG, perturbation device and load cell.

6.3.4 Data Analysis

A quiet trial was collected and used to remove bias from each EMG channel prior to analysis. EMG signals were full-wave rectified and digitally low-pass filtered at 3Hz (2nd order, single pass Butterworth filter). The maximum activation was found from each muscle specific maximum contraction and used to normalize each EMG signal. The load cell and three-dimensional wrist motion data were low-pass filtered at 10 Hz (2nd order, dual pass Butterworth filter). The perturbation device was equipped with a pressure sensor that was used to indicate the start of a perturbation. All data were investigated at three time periods: (i) baseline time period (150 ms to 100 ms pre-perturbation), (ii) anticipatory time period (15 ms to 0 ms pre-perturbation), and (iii) reflex time period (25 ms to 150 ms post-perturbation). The muscle co-contraction index (CCI) was calculated for all 55 muscle pairs (Lewek et al., 2004). The CCI provides a measure of muscle co-activation for muscle pairs over a specified time period and uses the ratio between the

muscles of lowest and highest normalized activity, multiplied by the sum of the two muscle activities at each sampled point.

An existing upper extremity model (Holzbaur et al., 2005) was used to apply the perturbation data (OpenSIM, Delp et al., 2007). The model was reduced to include only the 26 muscles crossing the wrist joint. The model included 13 muscles that could not be collected using surface EMG, thus, muscle force estimates could not be determined. Two of the forearm muscles in the model (FDS and ED) compartmentalize and cross the wrist joint as four tendons that attach to the digits of the hand. In our study, FDS and ED muscle activity was collected from the bulk of each muscle and this was used to drive each of the four compartments in the model. In total, muscle forces were found for 13 muscles and can be found in Table 6.2. The wrist model was capable of forearm pronation/supination and two wrist joint degrees of freedom including wrist flexion and deviation. Wrist joint kinematics were distributed between the proximal and distal carpal rows (Ruby et al., 1988). The collected wrist and forearm angles were used as input into the wrist model to determine the instantaneous length, velocity and moment arm of each muscle. The instantaneous muscle parameters were used in combination with the EMG to evaluate muscle force generating characteristics using a Hill-type muscle model (Delp and Loan, 1995; Zajac, 1989).

OpenSIM was used to obtain muscle specific three-dimensional coordinates (representing muscle origin, insertion and node/wrap points). The calculated muscle forces from the model and the anatomical muscle coordinates were used to determine individual muscle contributions to joint rotational stiffness about all three rotational axes

(Potvin and Brown, 2005). A constant value that relates muscle force and length to muscle stiffness was set to 10 as recommended by Potvin and Brown (2005).

Muscle forces and anatomical coordinate data were extracted from OpenSIM and used to calculate joint rotational stiffness (JRS) (Matlab, R2008a, The Mathworks, Inc., Natick, MA, USA). At each time point, all individual muscle contributions were summed and referred to as total joint rotational stiffness (JRS_T). Each individual muscle contribution was normalized to JRS_T at that time point to represent the relative contribution of each individual muscle to joint rotational stiffness (JRS_M). A second normalization method was performed to express the JRS_T for each experimental condition as a percentage of the theoretical maximum wrist JRS in our model ($MJRS_P$). $MJRS_P$ was determined by performing a theoretical analysis that included setting the forearm extensors to maximum activation to determine the resultant extensor moment. The forearm flexor activation required to maintain static equilibrium of the wrist joint (equal and opposite the extensor moment) in our neutral posture was then determined. Using the theoretical activations, $MJRS_P$ could be determined and each JRS_T during our trials could be normalized as a percent of $MJRS_P$. Mean JRS_M and JRS_T were calculated for the two time periods immediately prior to the perturbation (baseline and anticipatory) for rotations about the flexion/extension axis.

6.3.5 Statistical Analysis

Data were averaged across the three trials for each condition. A 2x2x3x3 repeated measures ANOVA was performed to evaluate the effects of perturbation timing knowledge (known timing and unknown timing), perturbation direction (flexion and

extension), grip level (no grip, 5% MVC and 10% MVC) and time period (baseline, anticipatory and reflex). The dependent variables included, grip force, push force, wrist angle, and CCI for each muscle combination. An ANOVA was also performed for JRS_M and JRS_T to evaluate the effects of timing knowledge, direction and grip level, but only the two pre perturbation time periods (baseline and anticipatory) were included, thus resulting in a 2x2x3x2 repeated measures ANOVA. Significant effects were compared with Tukey's HSD test. The alpha level was set to 0.05 for all statistical analyses (SPSS v13.0, IBM Corporation, Somers, NY, USA).

Table 6.2: List of muscles included in the model that cross the wrist joint. Note: “*” indicates one activation used to drive each of the four compartments.

Muscle	Abbreviation
Extensor carpi radialis longus	ECRL _M
Extensor carpi radialis brevis	ECRB _M
Extensor carpi ulnaris	ECU _M
Extensor digitorum communis*	
Digit 2	ED2 _M
Digit 3	ED3 _M
Digit 4	ED4 _M
Digit 5	ED5 _M
Flexor carpi radialis	FCR _M
Flexor carpi ulnaris	FCU _M
Flexor digitorum superficialis*	
Digit 2	FDS2 _M
Digit 3	FDS3 _M
Digit 4	FDS4 _M
Digit 5	FDS5 _M

6.4 Results

6.4.1 Perturbation Push Force, Grip Force and Wrist Rotation

There were no significant differences in perturbation push force for any experimental condition. The mean push force across all trials was 15.8 ± 2.6 N. Participants were able to maintain the target grip force at the high end of the target range. There was a significant effect of grip level ($p < 0.001$) with all three tasks differing significantly. The mean grip force recorded during the baseline time period without grip, 5% MVC and 10% MVC trials were $4.2 \pm 0.3\%$, $7.3 \pm 0.2\%$ and $11.5 \pm 0.4\%$ MVC, respectively. There were no significant differences in grip force due to perturbation direction or time period. Averaged across all conditions, the mean grip force at baseline was $7.8 \pm 3.2\%$ MVC and $7.9 \pm 3.2\%$ MVC during the anticipatory time period.

There were no significant effects of perturbation direction and grip force level for wrist angle. There was a significant timing knowledge x time period interaction for wrist angle ($p = 0.001$). During the reflex period, known timing resulted in $9.4 \pm 1.5^\circ$ more flexion/extension than the unknown timing conditions ($p = 0.001$). The restraints were successful at limiting off-axis rotations. During all flexion and extension trials, there was only slight forearm supination ($2.3 \pm 0.9^\circ$) and ulnar deviation ($4.3 \pm 1.3^\circ$).

6.4.2 Muscle Co-Contraction

There were 55 possible muscle co-contraction pairings. The CCI for five muscle pairings that cross the wrist joint are highlighted below to represent forearm muscle co-contraction (ECRL – FCR, ED – FDS, ECU – FCU, ECRL – ED, FCR – FCU).

Three of the five muscle pairings (ECRL-ED, ED-FDS and ECRL-FCR) demonstrated a significant perturbation direction x grip interaction (all $p < 0.048$). During the no grip trials, wrist flexion perturbations produced a CCI for ED-FDS that was 1.5 times larger than during wrist extension and there were no differences found between directions for the other two gripping levels. During extension, the ECU-FCU muscle pairing for the no grip, 5% and 10% MVC trials was 2.6, 2.0 and 1.8 times larger, respectively, than flexion. An opposite effect was found for ECRL-ED, as CCI during no grip, 5% and 10% MVC trials was approximately 1.5 times larger during flexion than extension (Figure 6.2).

There was a significant effect of time period on CCI for all five muscle pairings (all $p < 0.006$), with an increase in CCI from the baseline to the anticipatory period (Figure 6.3). CCI during the reflex period was greater than baseline for all muscle pairings and greater than the anticipatory period for ECRL-ED, ECU-FUC and ECRL-FCR (Figure 6.3). The average CCI for all five muscle pairings during the reflex and anticipatory periods were 2.8 and 2.3 times larger than at baseline, respectively.

Grip had a main effect on CCI for all muscle pairings (all $p < 0.003$). ECRL-FCR was the only pairing to demonstrate an increase in CCI from the no grip to 5% MVC trial. For all other muscle combinations (ECRL-ED, FCR-FCU, ED-FDS and ECU-FCU) the 10% MVC grip produced greater CCI than the 5% MVC and no grip trials.

ECU-FCU demonstrated a significant main effect of timing knowledge ($p = 0.002$) with unknown timing having greater CCI (4.2 versus 5.8).

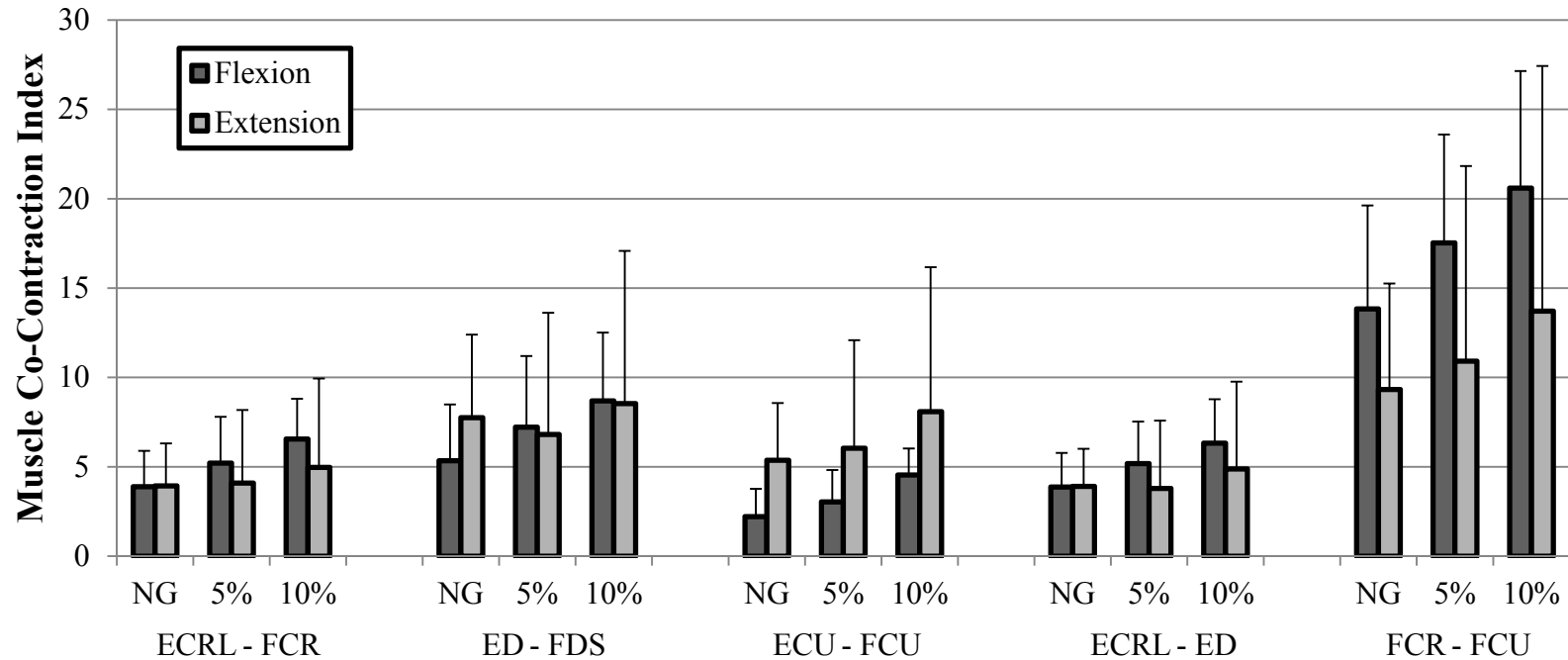


Figure 6.2 Mean muscle co-contraction (with standard deviation) demonstrating the effects of grip and perturbation direction. NG – No grip; 5% – 5% MVC grip; 10% – 10% MVC grip. See text for muscle abbreviations.

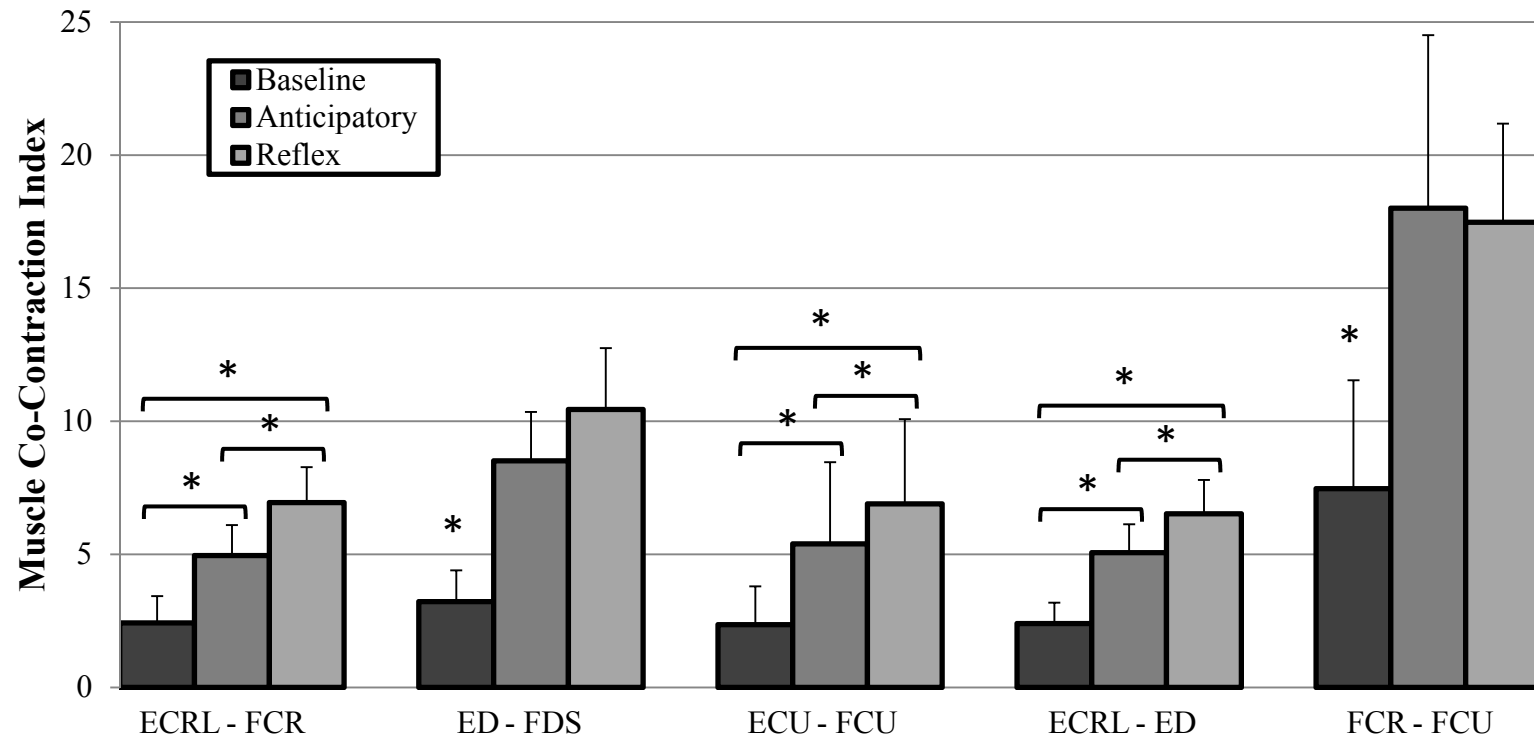


Figure 6.3 Mean muscle co-contraction (with standard deviation) during the three time periods. Significance is indicated for individual time period comparisons, “*” $p < 0.05$. See text for muscle abbreviations.

6.4.3 Maximum Joint Rotational Stiffness

The maximum JRS potential (as a percentage of MJRS_P) was greatest for the flexion/extension axis, followed by pronation/supination and radial/ulnar deviation (18.5, 11.8 and 10.9 Nm/rad, respectively). However, only the flexion/extension axis will be discussed in this communication.

A significant perturbation direction x grip interaction ($p = 0.007$) was found for normalized JRS_T. During perturbations of wrist flexion, the no grip condition was $10.5 \pm 1.9\%$ MJRS_P and the 10% MVC grip was $14.4 \pm 2.6\%$ MJRS_P, which was a 36.4% increase due to the 10% grip (Figure 6.4). There was also a significant main effect of time period on normalized JRS_T ($p = 0.0001$) with the anticipatory period being 35% greater than the baseline period ($13.2 \pm 2.2\%$ versus $9.7 \pm 1.6\%$ MJRS_P, respectively).

6.4.4 Individual Muscle Contributions to JRS (JRS_M)

The relative contribution of each muscle (%JRS_T) did not change due to the experimental conditions. Mean JRS_M for all experimental conditions is represented in Figure 6.5 for the flexion/extension axis. ECRL_M and ECRB_M had the largest contributions at $34.5 \pm 1.3\%$ and $20.5 \pm 2.3\%$ JRS_T, respectively. The four compartments of ED (ED2, 3, 4, 5) and FDS (FDS2, 3, 4, 5), when grouped together represent a total FDS contribution of $16.7 \pm 3.9\%$ JRS_T and $13.0 \pm 0.5\%$ JRS_T for ED. FCR had a very small contribution of 0.5% JRS_T.

Examining the individual compartments of FDS and ED, it was found that FDS2 and FDS3 contributed the most to the overall FDS contribution at $8.2 \pm 1.0\%$ JRS_T and

$7.2 \pm 0.9\%$ JRS_T, respectively. ED4 and ED5 contributed the most to the overall ED contribution at $4.7 \pm 0.5\%$ JRS_T and $3.1 \pm 1.1\%$ JRS_T, respectively (Figure 6.6).

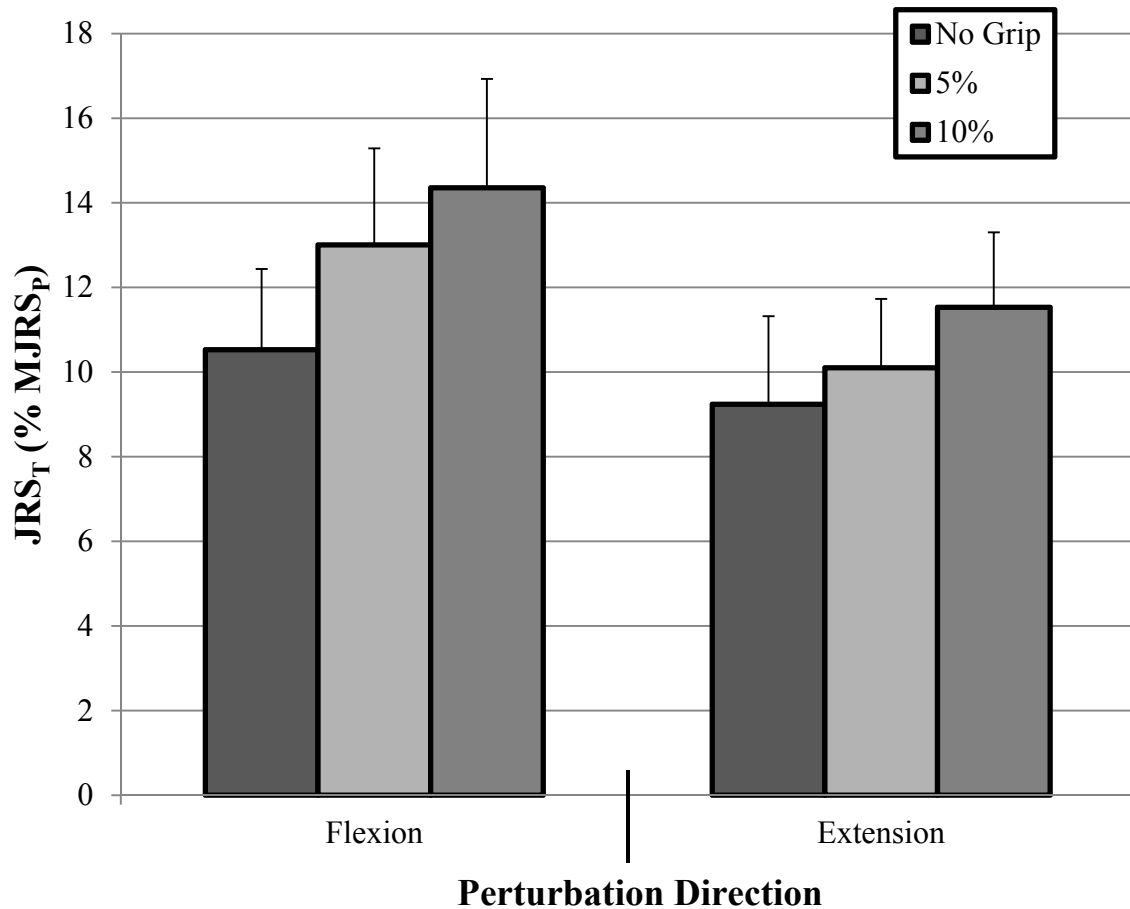


Figure 6.4 Mean JRS_T (with standard deviation) normalized to the maximum potential for our wrist model during the flexion/extension axis. The effects of grip level and perturbation direction are highlighted.

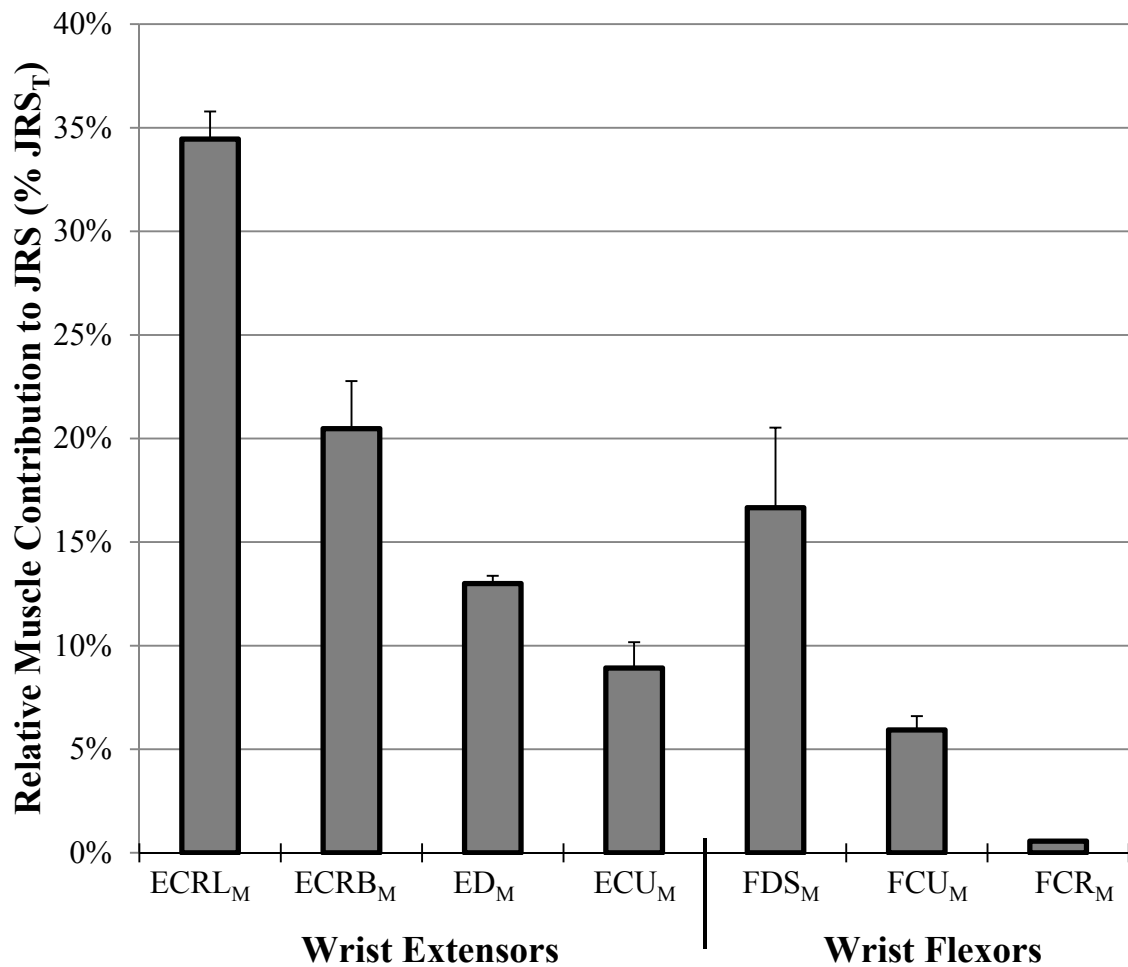


Figure 6.5 Mean JRS_M (with standard deviation) for all muscles during the baseline time period, averaged across all experimental conditions. See Table 6.2 for muscle abbreviations.

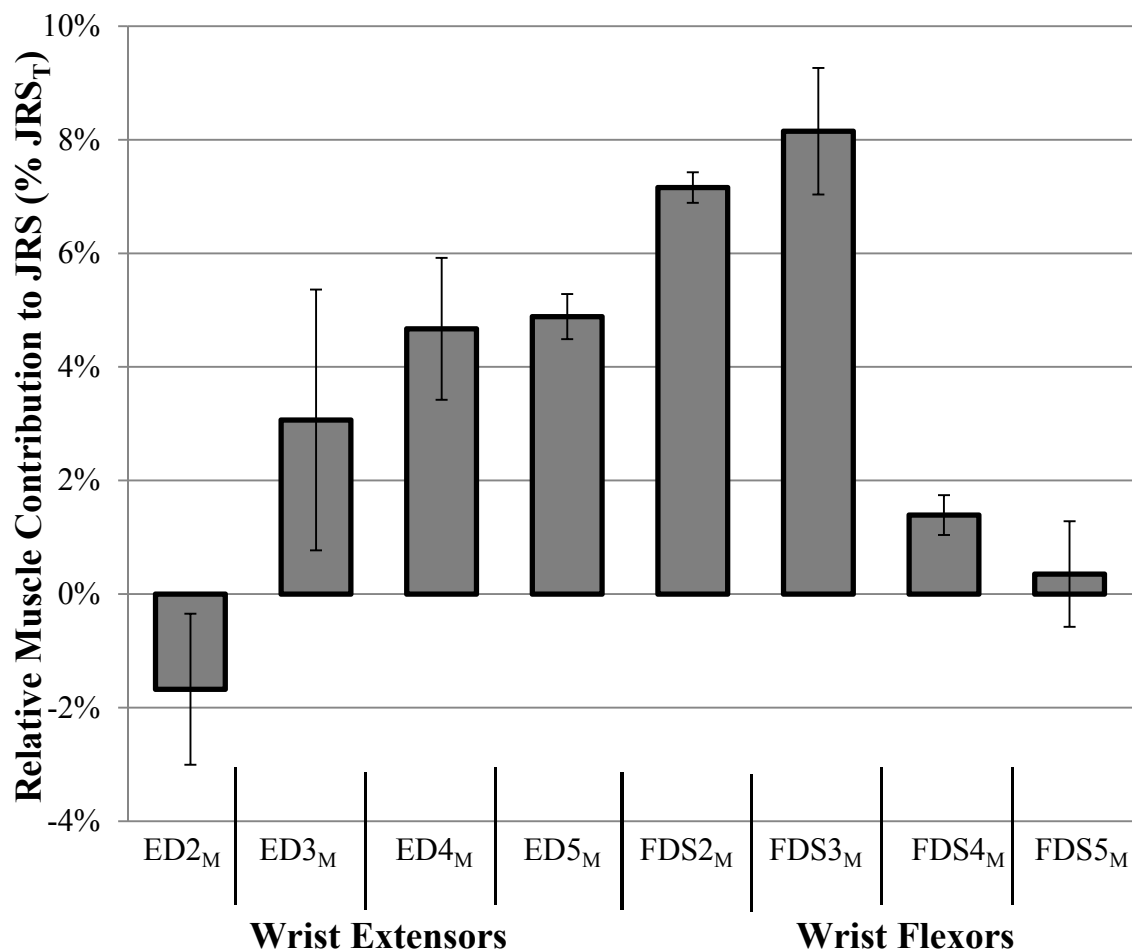


Figure 6.6 Mean JRS_M (with standard deviation) for each muscle compartment that was summated in figure 6.5 to represent ED and FDS, averaged across all experimental conditions for the baseline time period. See Table 6.2 for muscle abbreviations.

6.5 Discussion

This study investigated the effects of a gripping task on sudden expected and unexpected perturbations applied to the wrist joint causing wrist flexion or extension. It has been suggested that increases in co-contraction act to increase wrist joint stiffness; however, there has been little information on the manner in which individual forearm muscles act to increase stiffness. In this study, gripping was performed to modulate the level of forearm muscle co-contraction prior to wrist perturbation and a biomechanical model was used to quantify individual muscle contributions to wrist joint stiffness. Across all forearm muscle pairs, co-contraction increased as the grip demand increased. The 10% MVC grip resulted in a 34% increase in $MJRS_p$, over trials without grip, confirming that a relatively small grip was able to significantly increase wrist joint stiffness. One of the most interesting findings in this study was that, while grip force did not change between the baseline and anticipatory time periods, $MJRS_p$ increased, indicating both the importance of the anticipatory neuromuscular response and the need to assess joint stiffness. This was also the first study to quantify the contributions of individual muscles to wrist joint stiffness and we found that the greatest contributors to JRS (i.e. muscles with the highest JRS_M) were consistent across all conditions. Thus, for our gripping setup, muscle stiffness was up regulated across grip force level rather than redistributing muscle requirements. This helps understand the neuromuscular response to sudden loading as well as how the system increases joint stability to provide a margin of safety for the joint.

Between the baseline and anticipatory time periods, wrist joint stiffness increased by 35% despite no concurrent grip force increase. During the two time periods, grip force remained almost exactly the same, resulting in a non-significant 0.1% MVC difference. This is indicative of a neuromuscular response to help stiffen the wrist joint that was present in both known and unknown timing perturbations and it can be confirmed that gripping did not cause this response. This has been shown previously for known timing as participants stiffen the spine in anticipation for an upcoming perturbation (Brown et al., 2003; Granata et al., 2001). However, we found enhanced wrist stiffening regardless of knowing when the impulse would arrive. This may reflect our protocol since participants were aware that the unknown perturbation would occur within 10 seconds and they appeared to co-contract until the perturbation to ensure a stiffer joint, however, this could be a metabolically inefficient approach (Hogan, 1984). Our protocol also modulated the level of forearm co-contraction prior to each perturbation, which may have also contributed to this finding. Differences found between known and unknown timing protocols have typically occurred with no enhanced pre-activation of the surrounding musculature prior to the perturbation. The nature of our gripping task even resulted in a grip force requirement to simply hold the apparatus and may have contributed to these findings. Furthermore, the purpose of this paper was to investigate muscular responses in preparation for a sudden disturbance. It is anticipated that the investigation of muscular contributions to JRS immediately following a perturbation (reflex time period) will help clarify these findings.

This study utilized a relatively low grip force requirement (10% MVC), yet stiffness increased from 10.5% MJRS_p during the no grip condition to 14.4% MJRS_p during the 10% MVC grip (Figure 6.4). It has been documented that co-contraction is related to joint stiffness (De Serres and Milner, 1991; Franklin et al., 2003); however in these evaluations endpoint stiffness was quantified. During a hand gripping task, large increases in forearm muscle co-contraction has been found and it has been suggested as a primary mechanism to stiffen the wrist joint (Mogk and Keir, 2003; Snijders et al., 1987). Our study measured co-contraction and quantified wrist joint stiffness, thus confirming this hypothesis, since our gripping tasks demonstrated greater forearm muscle co-contraction and an overall increase in wrist joint stiffness.

Averaged across all experimental trials, ECRL_M and ECRB_M had the largest contributions to JRS (Figure 6.5). Due to the nature of the JRS equation (Potvin & Brown, 2005), contributions will be influenced by a number of factors including each muscle's force generating capacity and geometric configuration at the wrist joint. ECRL_M and ECRB_M have the largest physiological cross sectional areas (PCSA), except for FCU_M, thus have large maximum force capacities. Consistent with our data, Mogk and Keir (2003) suggested that during gripping tasks, the wrist extensors are activated to balance the flexor muscles. It appears that the increased demand of the forearm extensors during gripping, particularly ECR, played a role in these muscles providing the largest contributions. ECU and FCU also had large contributions about the flexion/extension axis and were recruited as a result of the gripping demands. It should also be noted that the orientation of each participant's hand in our study may have increased ECR activation

to hold the wrist in a neutral posture. However, it is expected that activation required to balance a potential ulnar deviation moment was minimal.

FCR had only a small contribution to JRS. However, FCR has a small force generating capacity (Gonzales et al., 1997) and the 10% MVC grip likely required little contribution from FCR. Claudon (1998) found that the forearm flexors are preferentially activated at high force levels, while Mogk and Keir (2003) demonstrated much greater extensor than flexor activity during a 5% grip. Furthermore, De Serres and Milner (1991) found that FCR activity remained unchanged by co-contraction of the wrist extensors, whereas FCU activity increased substantially. Our co-contraction measure for the FCR-FCU muscle pairing produced the largest increase of all muscle pairings from the baseline to anticipatory time period (Figure 6.2). Despite this increase (from a CCI of 7.5 to 18.0), FCR still had a small contribution to JRS. This suggests that FCR has a poor geometric contribution to stability (that which is independent of muscle force) and is not an important stabilizer of the wrist. ECU and FCU co-contraction were also found to increase with increasing load instability thereby increasing wrist stiffness (De Serres and Milner, 1991; Milner, 2002). Our CCI results found that the ECU-FCU muscle pairing increased with grip, but only at magnitudes comparable to the other pairings. The ECU-FCU CCI was less than FCR-FCU, thus further demonstrating the importance of each muscles geometric contribution to stability.

We found FDS to be the third largest contributor to JRS ($16.7 \pm 3.9\%$ JRS_T), which supports the findings of Gonzales et al. (1997) who demonstrated that the finger flexors played a major role in wrist flexion. Both FDS and ED have four individual

compartments that contribute to JRS. While FDS and ED have similar sized moment arms, the total PCSA of FDS was much larger than ED, which is reflected in the overall FDS contribution being larger. However, when considering the anatomical nature of FDS, the individual PCSA of each compartment is likely less important to our individual JRS differences than variations in muscle orientation. For instance, FDS2_M and FDS3_M attach on the anterior border of the radius, resulting in a smaller muscle length than FDS4_M and FDS5_M. The length of muscle segments crossing the wrist joint were also smaller (small “l” in the Potvin and Brown, 2005 equation), which contributes favourably to the JRS equation. As evident in Figure 6.6, an apparent negative stiffness was found for the ED2_M compartment. While negative stiffness in the true physical sense is not possible, in terms of the JRS calculation, it would suggest that ED2_M produced a moment that would likely contribute to the overall perturbation direction, and thus had a destabilizing effect in our posture. Brown and Potvin (2007) provided a detailed explanation of this interpretation.

We found that holding the grip dynamometer with no grip requirement resulted in an average grip force of $4.1 \pm 0.3\%$ MVC. This was of similar magnitude to other studies involving a precision grip task (Au and Keir, 2006; Mogk and Keir, 2003). Similar to Smets et al. (2009), we also found that participants were continually at the high end of the $\pm 1.5\%$ MVC criterion that was used for our grip force conditions. Interestingly, Au and Keir (2006) suggested that when presented with a target force, participants maintained a level at the lower boundary. The opposite was found in our study and this could be a reflection of the perturbation protocol. Participants were potentially at the high end of

our grip force criterion because they knew a sudden disturbance was about to occur and this approach would help stiffen the joint prior to perturbation.

There are a few limitations to this study. First, when investigating the forearm muscles with surface EMG, cross-talk can be a concern due to the anatomical arrangement of muscles in the forearm being in close proximity to each electrode site. Particular care was taken to ensure accurate electrode placement and previous work has suggested that cross-talk in the forearm can be minimal with proper configuration (Mogk and Keir, 2003). Finally, due to constraints of surface EMG, deep forearm muscles were not monitored and were not included in our calculations. Buchanan et al. (1993) found that many of the muscles omitted from our model generates minimal wrist moments and are likely not large contributors to wrist joint stiffness.

6.6 Conclusion

This study found that muscular contributions increased wrist joint stiffness immediately prior to a sudden perturbation, while no changes in the magnitude of grip force requirements were found. This study also confirmed that for a relatively small grip demanding task, forearm muscle co-contraction resulted in a 34% increase in wrist joint stiffness. This is the first study to document individual forearm muscle contributions to wrist JRS and it was found that the extensor carpi radialis had the largest contributions while the superficial finger flexors had the largest flexor contributions. This study provides insight into how individual forearm muscles modulate wrist joint stiffness. Consideration of these findings can lead to an understanding of how muscles maintain

joint stability, and why specific muscles may become injured during sudden loading events, due to their requirement to help stiffen the joint.

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CHAPTER 7: THESIS SUMMARY AND DISCUSSION

7.1 Thesis Summary

Stability is affected by the bony configuration at a joint in addition to the active and passive (soft-tissue stabilizers) contributions from surrounding tissues. Knowledge of the biomechanical properties of tissues surrounding a joint is critical for the understanding, diagnoses and treatment of clinical instabilities (Safran and Baillargeon, 2005). This thesis has provided a biomechanical evaluation of both ligamentous and muscular contributions to joint stiffness in the distal upper extremity. By quantifying these contributions, this work has provided insight into how the neuromuscular system adapts and responds to a sudden disturbance, thus providing some insight into mechanical joint stability. The four studies included in this thesis were designed with a common theme that investigated the effects of arm postures and hand loads on individual muscle and ligament contributions to joint stiffness. The thesis was considered in two parts: (i) ligamentous and (ii) muscular.

The first part of this thesis utilized a cadaveric approach to investigate mechanical properties of the carpal tunnel and TCL. The TCL is an important part of the carpal complex with contributions to carpal tunnel mechanics, carpal stability and as a pulley system for the flexor tendons. Despite this, the TCL remained an elusive structure that seemingly had more than one purpose. The anatomical location of the TCL places it in contact with the flexor tendons of the wrist and, given this interaction, was considered important for providing a detailed analysis of wrist stability. To date, mechanical

properties of the TCL have not been documented and it was expected that a detailed analysis of the TCL would address some of the questions that remained about its exact function. Two studies (Chapters 2 and 3) were designed to test TCL mechanical properties and this thesis has helped clarify the ligament's function as an important component of carpal tunnel mechanics. Chapter 2 focused on the effects of loading and posture on mechanical properties of the TCL. It was found that the TCL mechanical properties are not consistent throughout the structure, which suggests that its mechanical contributions to carpal stability vary depending on the location of the carpal tunnel under stress. It was also interesting to note that the TCL appeared to be stiffer than the entire carpal tunnel complex, at least during the vertical loading protocol implemented in Chapter 2. In a continuation of this study, Chapter 3 further evaluated the finding that mechanical properties vary depending on location of the TCL. Using a biaxial tensile testing method, it was found that locations closer to the TCL attachment sites were stiffer than sections at the middle of the ligament. This study indicated that the anatomical orientation and complex TCL fibre arrangement makes the mechanical properties location-dependent.

The primary contributions to joint stiffness are accomplished through the muscular system, and therefore a large component of this thesis focused on the upper extremity musculature. To provide a comprehensive analysis of wrist joint stiffness, and to expand upon the *in-vitro* investigation, it was imperative to document forearm muscle contributions to wrist joint stiffness. However, due to the complex and multi-articular nature of the forearm muscles, an investigation of forearm muscle contributions to joint

stiffness at the elbow was also performed. As a result, this thesis used a JRS approach to quantify individual muscle contributions to elbow and wrist joint stiffness. JRS provided an understanding of the manner in which the musculoskeletal system modulates individual muscle contributions to joint stiffness due to changes in arm posture and hand loads. Ultimately, this work provides insight into how the muscular system prepares for, and responds to, a sudden disturbance such that joint integrity is maintained. Chapter 4 focused on the effects of hand loads and arm postures on the muscular response to sudden arm perturbations. It was found that muscular responses to the perturbations were influenced by posture, hand loading and timing knowledge. It was suggested that increases in muscle activity (and co-contraction) help stiffen the elbow joint and provide stability. As a result, in chapter 5 a musculoskeletal model was developed to evaluate individual muscle contributions to elbow JRS due to posture and hand loading tasks. While the forearm muscles are not considered primary elbow flexors/extensors, they did contribute (albeit slightly) to elbow JRS and this was an important finding from this work. The forearm muscles have not traditionally been considered when evaluating muscular contributions to elbow stability and a better understanding of forearm contributions may provide insight into potential injury risk. It is likely that the forearm muscles may be loaded to a greater extent than that needed to mechanically complete a task, simply due to the stabilizing requirement.

Finally, a musculoskeletal model of the forearm and hand was developed to evaluate individual muscle contributions to wrist JRS. In particular, the effect of forearm muscle co-contraction on joint stiffness was evaluated during sudden wrist perturbations.

As forearm co-contraction increased using a low grip demanding task, a substantial increase in wrist joint stiffness was found. This study also found that immediately prior to a sudden perturbation, a neuromuscular response stiffened the wrist joint, which is likely a safety mechanism to limit joint rotation due to the sudden perturbation and has been previously shown in spinal stability work (Brown et al., 2003; Stokes et al., 2000), but not the upper extremity. It was evident that an analysis of forearm muscle co-contraction may not provide the required information to specifically conclude how the musculature provides a stabilizing contribution to the wrist joint. This is reflected in the JRS equation and this work has shown that muscle orientation (origin, insertion, nodal points and moment arm length) greatly influences forearm muscle contributions to wrist joint stiffness.

In summary, a comprehensive analysis of forearm muscle contributions to wrist and elbow joint rotational stiffness has been performed while also quantifying TCL mechanical properties that demonstrated its importance for maintaining carpal bone stability and proper carpal tunnel mechanics. This thesis provided an approach that had not previously been considered in the upper extremity for the evaluation of mechanical relationships to injury. This thesis is the first to document individual muscle contributions to JRS in the distal upper extremity, while also providing insight into carpal stability using a cadaveric approach. New and valuable information was found concerning how the musculoskeletal system functions to maintain joint integrity.

7.2 Main Research Contributions

7.2.1 TCL Contributions to Carpal Tunnel Mechanics

Chapters 2 and 3 provided a comprehensive evaluation of TCL mechanical properties, which was a vital and necessary component to better understand the ligament's role in carpal tunnel mechanics and carpal stability. Chapter 2 investigated how TCL characteristics were altered due to changes in wrist posture and size of indentation contact area. It was found that a flexed wrist posture had resulted in significantly greater TCL stiffness than the neutral and extended postures. Furthermore, the influence of indenter contact area suggested that as more of the TCL was covered during indentation, stiffness increased. A recent study found that thickness of the TCL varies throughout the tissue (Pacek et al., 2009), which suggested that as our indenters covered more of the ligament; we likely contacted thicker (and stiffer) parts of the tissue. Given that the median nerve and flexor tendons interact with the TCL (Armstrong and Chaffin, 1978; Kline, 1992) and the ligament provides a boundary for the carpal tunnel, it was clear that this work helped confirm the importance of the TCL in carpal tunnel mechanics. However, this work suggested that further insight was needed into the specific characteristics of the ligament.

Building upon this research, a follow-up study (Chapter 3) provided a more direct investigation of the TCL and evaluated individual components of the ligament at different regions of the carpal tunnel. This was the first study to document site dependent differences in the mechanical properties of the TCL, which is vital to further advancing our understanding of the TCL as a critical component to carpal stability. Thickness

varied considerably throughout the ligament, with distinct differences being found closer to the attachment sites of the tissue. This work confirmed that the TCL exhibited different mechanical properties within different locations of the tissue. It is also interesting to note that the complex arrangement of fibres making up the TCL (Mashoof et al., 2001; Isogai et al., 2002) adds further complexity to interpreting its mechanical properties. Interestingly, thickness did not always relate to increased stiffness and this finding can significantly contribute to our understanding of carpal tunnel mechanics. The TCL provides a mechanical constraint for the superficially located median nerve and places it in contact with the TCL. Based on our findings, structures located close to the radial and ulnar borders of the carpal tunnel would be in contact with a thicker and stiffer region of the TCL. This region would be less accommodating to movement when compared to the middle (and proximal) locations. Further investigations of median nerve location with knowledge of TCL tissue properties could provide insight into CTS development.

7.2.2 Interpretation of JRS

A primary focus of this thesis was to investigate the forearm musculature and how arm postures and hand loading tasks influence individual muscle contributions to JRS. To fully evaluate forearm muscle contributions to joint stiffness in the distal upper extremity, an evaluation of contributions at both the elbow and wrist joint was required.

Chapter 5 investigated the individual muscular contributions to elbow JRS. It was found that during perturbations with known timing, participants increased their neuromuscular response just prior to the perturbation, which is beneficial for stiffening

the joint and minimizing the chance of joint injury due to sudden loading. The magnitude of our changes for individual muscle contributions to JRS due to arm postures and hand loading were relatively small; however it was found that the task demands enhanced the magnitude of individual contributions. This would suggest an enhanced overall elbow joint stiffness. Due to the task demands in our study, the elbow flexors were influenced the most by postural change and generally demonstrated the greatest contributions to JRS during standing. The forearm muscles demonstrated the largest difference due to hand loading, with the fluid and solid loads producing enhanced stiffness when compared to the no hand loading task.

To complete the analysis of forearm muscle contributions to JRS, a wrist model evaluated the effects of forearm muscle loading (via a gripping task) on wrist JRS. This study demonstrated that there was a substantial increase in stiffness immediately prior to a sudden perturbation, while no changes in grip force demands were apparent. However, it was surprising to find that the increased anticipatory response was also evident during unknown timing perturbations, a finding that was not expected and did not occur at the elbow. This was an interesting finding that needs further investigation. It may have been a reflection of our perturbation protocol, since multiple trials were performed and resulted in a large number of perturbations. Participants may have anticipated the perturbation (Koike and Yamada, 2007) or became accustomed to the direction and magnitude (Franklin et al., 2003), which would influence the response from the muscular system. However, further analyses of the reflex time period (post perturbation) should also be investigated. It has been shown that the reflex response can be altered depending

on the environment (Akazawa et al., 1983; Perreault et al., 2008) and by the demands of the perturbation (Lewis et al., 2006; Pruszynski et al., 2008). It is likely that differences in the neuromuscular response during known and unknown timing will be more apparent in the reflex period.

It was found that forearm muscle co-contraction increased with grip demands, and this corresponded to an increase in overall wrist joint stiffness. For a relatively small grip demanding task (10% MVC), forearm muscle co-contraction significantly increased wrist joint stiffness. During a gripping task, large increases in forearm muscle co-contraction are observed and it has long been proposed as a mechanism to stiffen the wrist joint (Mogk and Keir, 2003; Snijders et al., 1987). This study measured forearm muscle co-contraction and quantified wrist JRS, thus confirming that wrist stiffness increased with increasing grip demands.

7.2.3 Individual Muscle Contributions to JRS

A primary focus of this thesis was to quantify how arm postures and hand loads influenced individual muscle contributions to JRS at two time periods prior to a sudden perturbation. This work has provided knowledge of which muscles have the greatest potential to help stabilize the elbow and wrist joints. At the elbow joint, it was found that the primary elbow flexor muscles (brachialis, biceps brachii long and short head, and brachioradialis) provided the greatest individual contributions to elbow stiffness. Brachialis had the greatest contribution, which was an important finding since the role of brachialis as a stabilizer of the elbow has received conflicting views (Basmajian, 1978; Buchanan et al., 1986; MacConaill, 1946). It was interesting to note that for the postural

tasks evaluated in this thesis, triceps brachii had a relatively small contribution, suggesting that in the conditions tested, it may not be an important stabilizer of the elbow. However, as suggested by Hogan (1984), postural demands are important when interpreting joint impedance, and thus additional postural demands should be investigated before concrete conclusions can be made about the role of triceps brachii for elbow stability. Two important aspects of this thesis may have contributed to these findings, including the extended triceps length in the postures tested and the nature of our perturbation direction causing arm extension.

It was also found that the forearm muscles which cross the elbow joint provided a small, approximately 5.5%, but potentially important contribution to total elbow JRS. The forearm muscles have not traditionally been considered to provide a stabilizing role at the elbow, however our results demonstrate that, geometrically, they will have a contribution. The forearm extensors, ECR and ECU, provided the largest contributions to JRS within the forearm muscles tested. It is likely that during grip demanding tasks, these forearm muscles may have substantial contributions to elbow joint stiffness due to increased force requirements.

At the wrist joint, ECR longus and brevis provided the greatest contributions to JRS. Mogk and Keir (2003) suggested that during gripping tasks, the wrist extensors are activated to balance the flexor muscles. It appears that the increased demand of the forearm extensors during gripping, particularly ECR, played a role in these muscles providing the largest contributions. Additionally, ECU and FCU also contributed substantially to wrist JRS and this highlights the multi-functional role for many of the

forearm muscles. My perturbation caused wrist flexion/extension, yet large contributions were found from ECU and FCU, which are also wrist deviators. This was not surprising, since these muscles have been shown to have large flexion/extension moment arms (Gonzales et al., 1997). However, this further highlights the complexity of the forearm musculature, which has a redundant number of muscles that have similar actions and muscle orientations that provide contributions to JRS about all rotational axes. While only flexion/extension perturbations were quantified in this thesis, work has already been performed to investigate muscular contributions to pronation/supination perturbations. It appears that in the forearm, many of the muscles will have contributions to JRS for rotations that may not traditionally be considered its primary muscle action. Given that the forearm extensors dominated the overall contribution to wrist JRS, and were the primary forearm contributors to elbow JRS, it is apparent they play a large stabilizing role in the distal upper extremity and thus are vital for maintaining joint integrity.

7.3 Implication of Findings to Injury

In 2009, 19.7% of all lost time claims in Ontario were related to the upper extremity (WSIB, 2009) and there is evidence to support that upper extremity health care costs are larger than those pertaining to other regions of the body (Silverstein et al., 1998). Clearly more needs to be done within the research community to address these concerning injury statistics. Specific mechanical relationships for workplace injuries have been suggested; however the incidence of injury remains high. This thesis has attempted to shift the focus from traditional evaluations of muscle and joint loading,

towards a better understanding of how the neuromuscular system modulates joint stiffness in the distal upper extremity.

The nature of our posture and loading conditions altered muscle co-contraction immediately prior to both the elbow and wrist perturbation studies. As discussed previously, it was interesting to find that there were no differences in muscle activity, muscle co-contraction or individual muscle contributions to JRS between known and unknown timing perturbations at the wrist joint in our work. This would suggest that participants increased activation throughout the unknown timing events, regardless of when the perturbation occurred. This is a metabolically inefficient approach (Hogan, 1984) and results in increased joint loading (Cholewicki and McGill, 1996), which over time, can lead to large cumulative loads (Kumar, 1990) and ultimately contribute to joint injury. The forearm extensor muscles (ECR and ECU) dominated the contribution to overall wrist JRS, thus it would appear that these muscles may experience loading that is greater than that required, due to stability requirements. However, it was assumed that in our protocol co-contraction was a viable and necessary requirement to enhance the level of joint stiffness, and hence, contribute to maintaining joint stability.

During hand intensive tasks (such as gripping), increased activation of the forearm extensors is a necessary mechanism to balance wrist joint moments (Mogk and Keir, 2003; Snijders, 1987). This thesis has demonstrated that many of the forearm extensor muscles (in particular ECR and ECU) are primary contributors to wrist joint stiffness. It was also found that ECR provided a small contribution to elbow JRS, which demonstrates that the extensor muscles are extremely active stabilizers. Many injuries to

the elbow (such as lateral epicondylitis) occur from muscular imbalances in the forearm (Pienimake et al, 2002). Our results confirmed that the forearm extensors play a primary role in stabilizing the wrist and elbow. These results may improve our understanding of why overuse injuries in the forearm develop.

At the wrist, a primary concern for workplace injury revolves around carpal tunnel syndrome (CTS). CTS is the most common peripheral compression neuropathy and has a large financial burden to the economy (Atroshi et al., 1999; Foley et al., 2007; Manktelow et al., 2004). Previous work has documented the effects of wrist posture on carpal tunnel size, shape and pressure (Mogk and Keir, 2007; Mogk and Keir, 2009), but to date, there has been a lack of information on how the mechanical properties of the TCL will alter these predictions. The cadaver work in this thesis will improve our understanding of CTS, since the improved mechanical property information can be added to current models of the carpal tunnel to provide more realistic measures of carpal tunnel mechanics.

7.4 Future Directions

This thesis is the first to document individual ligament and muscle contributions to joint stiffness in the distal upper extremity, and has ultimately provided a starting point for future work that can improve our understanding of joint stability and injury risk.

One of the most important reasons for investigating the TCL and carpal tunnel mechanics was to better understand CTS. The evaluation of clinically diagnosed CTS cadaver arms would help provide a better understanding of how the mechanical

properties of the TCL change with injury and influence carpal tunnel mechanics. Furthermore, manipulation techniques have been used in the past, by rehabilitation professionals, as a means of treatment for CTS. To date, there has been limited support in the research community for these techniques and conducting a similar protocol (Chapter 3) on an injured population would provide insight into potential recommendations for non-surgical rehabilitation of CTS. Finally, previously developed three-dimensional model of the carpal tunnel (Mogk and Keir, 2007) evaluates carpal tunnel mechanics based solely on carpal bone movement. The inclusion of TCL mechanical properties from this thesis could more accurately predict carpal tunnel size and shape due to changes in posture. Taking this work a step further, the flexor tendons and median nerve interact with the TCL during wrist movement and a model with TCL mechanical properties could be used to evaluate flexor tendon excursions and how forces exerted by the flexor tendons impact the TCL as a pulley system for the wrist. The TCL pulley system will ultimately impact the flexor muscle's force generating capacity and should potentially be included in models used to estimate forearm muscle forces.

This thesis was the first to document individual muscle contributions to JRS in the upper extremity and this has led to many additional questions. First, many studies suggest that the forearm extensor muscles fatigue first during gripping tasks (Mogk and Keir, 2003; Snijders, 1987). Performing a protocol that fatigues the forearm extensor muscles prior to sudden perturbation could be used to investigate if the redundant forearm musculature provides an altered strategy to maintain joint stability. It is likely that in a fatigued state, there will be a redistribution of the primary muscle contributions.

Sudden joint angle perturbations are often used to evaluate the neuromuscular response to a disturbance. In this thesis, this type of protocol was used to better understand the neuromuscular systems ability to provide joint safety. In our protocol, a known perturbation direction was always implemented with a relatively low perturbation magnitude. Further research investigating larger perturbation magnitudes and an unknown timing perturbation protocol that would not allow participants to anticipate the disturbance is undoubtedly needed. Finally, a perturbation, where participants are unaware of the direction, may result in different muscular activation patterns and could ultimately be more representative of real life joint angular disturbances during occupational tasks.

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APPENDICES

APPENDIX A: Ethics approval for Study 1 and 2 (Chapters 2 and 3): Cadaveric Research Summary and Authorization



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Cadaveric Research Summary & Authorization

Introduction

As of June 4, 2007 the Office of the Chief Coroner for Ontario & General Inspector of Anatomy (CCO) requires knowledge of all scientific and educational research being performed on cadaveric material obtained within the Province of Ontario under the Ontario Anatomy Act (1990, amended 2006). Subsequently, the Office of the CCO will assess the intended research and will reserve the right to authorize its commencement or continuation within the Province of Ontario.

The purpose for assessment and authorization is to ensure that cadaveric material is used in the most effective, culturally sensitive, and beneficial manner possible.

Assessment by the Office of the CCO can be done prior to concurrently with, or after the Ethics Approval Processes of the Institutions involved.

The CCO merely wants to have knowledge of how cadavers are being used.

Instructions

Please fill out the following sections using clear, brief explanations. To streamline this form, some sections have recommended practises. If you plan on using these practises please reiterate them in your description, adapting them to your location, lab or study. If your study requires a significant diversion from these practises, please explain why and expand on how you plan to accomplish the tasks requested

This page will be signed by you the investigator; the Director for the Education Program in Anatomy; and, upon approval, a representative of the Office of the Chief Coroner for Ontario. Submissions should be returned to the Education Program in Anatomy and will be forwarded to the Chief Coroner for Ontario for review. Submissions can be made via PDF, fax, mail or by hand.

If you have any questions or concerns, please contact the Education Program in Anatomy.

Signatures (page 1, below)

Section 1: Names and Contacts (page 2)

Section 2: Your study (page 3)

Section 3: Transport (page 4)

Section 4: Storage and Security (page 5)

Section 5: Disposal (page 6)

Investigator (print): _____ (sign): _____ Date: _____

Director, Education Program in Anatomy:  _____ Date: _____

Office of the Chief Coroner for Ontario: _____ Date: _____

Approval: _____



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Section 1: Names and Contacts

Please list the names and contact information of the principle investigators in your study, specifying who will be handling the cadaveric materials:

Dr. Peter J. Keir, PhD
Department of Kinesiology, McMaster University
1280 Main Street West, Hamilton, ON L8S 4K1
Phone: (905) 525-9140 x23543 Fax: (905) 523-6011
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- Peter Keir will be available to help prepare specimens for testing and will supervise to ensure proper use and disposal.

Michael W.R. Holmes, Msc.
Department of Kinesiology, McMaster University
1280 Main Street West, Hamilton, ON L8S 4K1
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E-mail: holmesmw@mcmaster.ca

- Michael Holmes will be involved in all dissection and testing procedures of the specimens.
- Michael Holmes will be doing the cadaver handling (transportation, testing and disposal) - with assistance from Peter Keir and Jack Callaghan.

Dr. Jack P. Callaghan, PhD, CCPE
Canada Research Chair in Spine Biomechanics and Injury Prevention
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Phone: (519) 888-4567 x37080 Fax: (519) 746-6776
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- Jack Callaghan will be available at the testing location to provide supervision of proper specimen use, storage and disposal.



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Section 2: Your study

Please provide a brief description of your study (2-3 paragraphs if necessary). If you plan on publishing, include how and where you intend to publish your study:

The purpose of this study is to use an instron testing machine to investigate stiffness properties of the human transverse carpal ligamen (TCL). An analysis of stiffness properties of the TCL is essential to understanding loading characteristics of the carpal bones and surrounding structures. Additionally, a better understanding of TCL function will advance our knowledge of carpal bone kinematics and the TCL's role in wrist joint stability.

To develop sufficient confidence in the proposed technique, it is anticipated that this study will require 10 fresh-frozen cadavers. Following proper dissection to expose the TCL, each specimen will be affixed to an Instron testing machine. The TCL will be loaded using a custom made cylinder apparatus. Four different size apparatus will be used (5, 10, 20 and 35 mm diameters). A loading magnitude from 5 to 50 N will be applied at a 100hz loading rate.

This information will provide insight into compression of the median nerve which will help improve an existing 3 dimensional model of the carpal tunnel and wrist. The advancement of the model will benefit people who have suffered carpal tunnel injury or another related disorders to the hand and wrist.

It is anticipated that the results of this investigation will be published in the Journal of Biomechanics.

This study will take place from September 2008 to August 2009.



Education Program in Anatomy

McMaster University Medical Center
Room 1R1
McMaster University
1200 Main Street West
Hamilton, Ontario
Canada L8N 3Z5

Phone: 905 525-9140 x22273
Fax: 905 525-7400

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Section 3: Transport

Transportation is usually done by an Education Program in Anatomy staff member, travelling with two signed warrants. The first warrant describes the anatomical material being transported and is signed by the Director of the Education Program in Anatomy. The second warrant is a blanket permission to transport by the Chief Coroner of Ontario. The cadaveric material is contained in an appropriate, nondescript case and cannot be left unattended until it has reached its destination.

Please provide a brief description of how you plan to transport the cadaveric materials:

Following the appropriate signed warrants, cadaveric materials will be transported to the University of Waterloo, Waterloo, ON, Canada for testing.

Dr. Peter Keir and/or Michael Holmes will provide private transportation of the cadaveric material in an appropriately sealed and locked cooler container. At no time during transport will the materials be left unattended.

Section 4: Storage and Security

Storage for cadaveric materials must be secure, and the materials themselves treated as biohazardous. Freezers or coolers must have locks with carefully controlled key access. Storage should be in a low traffic area to minimize the potential for unwilling exposure to those who may be sensitive to the nature of human remains.

Please provide a brief description of where and how you plan to store and secure your materials, including: who will have access and how. If you are storing the materials in a cooler or freezer, please list any other biological materials that will also be stored in the cooler/freezer:

The Department of Kinesiology at the University of Waterloo has a locked and secure laboratory section to the building. The Laboratory area for material testing also has additional locked access. Only individuals requiring access to this laboratory for testing will have access.

Materials will be stored in a pad locked, secure freezer until needed for testing. During the time of testing it is anticipated that there will also be porcine spinal units kept in the same storage freezer.

Section 5: Disposal

Cadaveric materials must be returned to either the Education Program in Anatomy at McMaster University, or to the institution they originated from for cremation. Under absolutely no circumstances will cadaveric materials be allowed to enter the conventional waste handling system.

Please provide a brief description on how you will dispose of cadaveric materials, specifically how you will store, label and transport waste tissue:

Following successful testing of cadaveric material, each specimen will be labeled, carefully handled and placed in the appropriate locked storage cooler container for private transport by Dr. Peter Keir and Michael Holmes to the McMaster University School of Anatomy.

APPENDIX B: John Wiley and Sons: Chapter 2 Copyright Agreement

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May 09, 2011

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APPENDIX C: Ethics approval for Study 3 (Chapters 4 and 5): Amendment to current research program

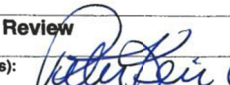
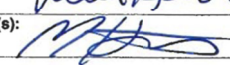
McMaster University Research Ethics Board (MREB)
Change Request Form

Please complete and submit this form, if you wish to make a change or an addition to an approved "Application to Involve Human Participants in Research"
 Submit **two (2)** sets of all materials to:
 c/o Office of Research Services, MREB Secretariat, GH 305/H, x 23142, e-mail: ethicsoffice@mcmaster.ca

TITLE OF RESEARCH PROJECT: Evaluating upper extremity muscle contributions to elbow and wrist joint stiffness				
REB File #: 2007 148 2008 8, 2008		Today's Date: May 12, 2008		Date of Original Ethics Approval: Jan. 8, 2008
	NAME	DEPT./ADDRESS	PHONE	E - MAIL
Faculty Investigator(s)/ Supervisor(s)	Peter Keir	Kinesiology/IWC 216	23543	pjkeir@mcmaster.ca
Student Investigator(s)	Michael Holmes	Kinesiology/IWC A108	20175	holmesmw@mcmaster.ca

The headings below correspond to sections of the McMaster University "Application to Involve Human Participants in Research" form.

1. Please check all boxes that apply to the sections of the original protocol which you wish to change.
2. If you wish to change a test instrument (questionnaire, etc.) or a consent form or letter of information, please submit the entire revised document and highlight the sections which are being changed or added.
3. Please submit this form, the changed documents referred to in 2. (above) if applicable, and the original "Application to Involve Human Participations in Research" form, together with:
 - (a) either a narrative description of the changes which are identified below or:
 - (b) if the changes are substantial, a second "Application to Involve Human Participations in Research" form, highlighting the sections which are different from those contained in the original application.

A. GENERAL INFORMATION <input checked="" type="checkbox"/> Title of Project <input type="checkbox"/> Faculty Investigator(s) <input type="checkbox"/> Faculty Supervisor(s) <input checked="" type="checkbox"/> Student Investigator(s) <input checked="" type="checkbox"/> A.1 Level of Project <input type="checkbox"/> A.2 Funding Status <input type="checkbox"/> A.3 Details of Funding B. PROJECT SUMMARY <input type="checkbox"/> B.1 Purpose/Rational for Research Project <input checked="" type="checkbox"/> B.2 Methodology/Procedures <input type="checkbox"/> B.3 Previous Experience <input type="checkbox"/> B.4 Participants Involved in Study <input checked="" type="checkbox"/> B.5 Recruitment Process <input checked="" type="checkbox"/> B.6 Compensation for Participants <input type="checkbox"/> B.7 Feedback for Participants	C. BENEFITS <input type="checkbox"/> Potential Benefits of the Proposed Study D. RISKS <input type="checkbox"/> D.1 Known/Anticipated Risks of the Study <input type="checkbox"/> D.2 Procedures or Safeguards in Place E. INFORMATION/CONSENT PROCESS <input type="checkbox"/> E.1 Informed Consent/Absence of Consent <input type="checkbox"/> E.2 Process for Parental/Guardian Consent <input type="checkbox"/> E.3 Process for Withdrawal of Consent F. CONFIDENTIALITY <input type="checkbox"/> Procedures to Ensure Confidentiality G. DECEPTION <input type="checkbox"/> Justify Deception & Explain Debriefing H. REB REVIEW <input type="checkbox"/> Frequency of Review Faculty Signature(s):  (PETER KEIR) Student Signature(s):  (MIKE HOLMES)
--	---

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APPENDIX D: Consent form – Chapter 4 and 5August 2nd, 2009**Letter of Information and Consent*****A study evaluating upper extremity muscle contributions to elbow and wrist joint stiffness*****Investigators:** Michael Holmes and Dr. Peter Keir

Principal Investigator: Dr. Peter Keir
 Department of Kinesiology
 McMaster University
 Hamilton, Ontario, Canada
 (905) 525-9140 ext. 23543;

Student / Co-Investigator Michael Holmes
 Department of Kinesiology
 McMaster University
 Hamilton, Ontario, Canada
 (905) 525-9140 ext. 21334;

Purpose of the Study

Despite significant contributions to upper extremity research, the number of work related injuries remain high. It is thought that muscles play a large role in stabilizing a joint and further investigation into how they do this may help provide insight into how people become injured. The purpose of this study is to evaluate how muscles individually contribute to upper extremity activities involving the elbow and wrist.

Procedures involved in the Research

Anthropometric measures (height, weight, arm length and hand length) will be recorded and you will be introduced to the protocol. Immediately following this you will have recording electrodes placed over 11 muscles of the forearm, upper arm and shoulder. These electrodes allow us to record the activity in the muscles under them. To know how active your muscles are, we first need to determine the maximum activity for each muscle through a series of tests for each muscle. A device will also be placed at the elbow joint to measure elbow angle. You will then be required to statically hold 2 postures during testing. One posture, lying on a table with your arm by your side and elbow flexed to 90 degrees and the second posture standing with your upper arm at the side of your body and elbow flexed to 90 degrees. With your arm held in each posture, the device will be positioned in a location that will cause your arm to extend. The device will push a padded rod outward, pushing your arm with it. The extension of your arm will be minimal, causing only a small change in angle at the elbow. The padded area will result in no discomfort to you. Your participation will require about 1.5 hours in the lab.

Potential Harms, Risks or Discomforts:

There is minimal risk associated with participation in this study. You may experience some muscle soreness as a result of the maximal exertions. Although very rare, you may experience a temporary reaction to the adhesive from the surface electrodes. Should you experience any serious discomfort following the study, please contact the principal investigator, Dr. Peter Keir. Due to the nature of the protocol, you will not be allowed to participate if you have been diagnosed with high blood pressure or have previous shoulder and wrist injuries.

Potential Benefits

We hope to understand the loads experienced within the body and relate them to injuries and disorders that develop in the workplace. Ultimately we hope to prevent workplace disorders. The research will not benefit you directly.

Payment or Reimbursement:

You will be financially compensated \$20.00 for your time and participation in this study.

Confidentiality:

Your identity will be kept anonymous and the data collected will be used for teaching and research purposes only. You may be asked if you would be willing to have photos or video of you taken for use in publications and presentations. Photo and video data will only be used with your consent. The information directly pertaining to you will be locked in a cabinet for a maximum of 15 years.

Participation:

Your participation in this study is voluntary. If you decide to participate, you can decide to stop at any time, even after signing the consent form or part-way through the study. If you drop out of the study, your data will only be used with your explicit consent. If you decide to stop participating, there will be no consequences to you and the compensation will be prorated. If you do not want to answer some of the questions you do not have to, but you can still be in the study.

Information About the Study Results:

You may obtain information about the results of the study by contacting Dr. Keir or Michael Holmes. An update will be emailed after completion of the study; if you would like an update your email will be required.

Information about Participating as a Study Subject:

If you have questions or require more information about the study itself, please contact Dr. Keir or Michael Holmes.

This study has been reviewed and approved by the McMaster Research Ethics Board. If you have concerns or questions about your rights as a participant or about the way the study is conducted, you may contact:

McMaster Research Ethics Board Secretariat
Telephone: (905) 525-9140 ext. 23142
c/o Office of Research Services
E-mail: ethicsoffice@mcmaster.ca

CONSENT

I have read the information presented in the information letter about a study being conducted by Michael Holmes and Dr. Keir, of McMaster University. I have had the opportunity to ask questions about my involvement in this study, and to receive any additional details I wanted to know about the study. I understand that I may withdraw from the study at any time, if I choose to do so, and I agree to participate in this study. I have been given a copy of this form.

Name of Participant


In my opinion, the person who has signed above is agreeing to participate in this study voluntarily, and understands the nature of the study and the consequences of participation in it.

Signature of Researcher or Witness

APPENDIX E: Ethics approval for Study 4 (Chapter 6): Amendment to current research program

MREB Approval Certificate

Page 1 of 1

		McMaster University Research Ethics Board (MREB) c/o Office of Research Services, MREB Secretariat, GH-305/H, e-mail: ethicsoffice@mcmaster.ca	
CERTIFICATE OF ETHICS CLEARANCE TO INVOLVE HUMAN PARTICIPANTS IN RESEARCH			
Application Status: New <input type="checkbox"/> Addendum <input checked="" type="checkbox"/> Project Number: 2007 148			
TITLE OF RESEARCH PROJECT: Work-related Upper Extremity Loading			
Faculty Investigator (s)/ Supervisor(s)	Dept./Address	Phone	E-Mail
P. Keir	Kinesiology	23543	pjkeir@mcmaster.ca
Student Investigator(s)	Dept./Address	Phone	E-Mail
M. Holmes	Kinesiology		holmesmw@mcmaster.ca
The application in support of the above research project has been reviewed by the MREB to ensure compliance with the Tri-Council Policy Statement and the McMaster University Policies and Guidelines for Research Involving Human Participants. The following ethics certification is provided by the MREB:			
<input checked="" type="checkbox"/> The application protocol is approved as presented without questions or requests for modification. <input type="checkbox"/> The application protocol is approved as revised without questions or requests for modification. <input type="checkbox"/> The application protocol is approved subject to clarification and/or modification as appended or identified below:			
COMMENTS AND CONDITIONS: Ongoing approval is contingent on completing the annual completed/status report. A "Change Request" or amendment must be made and approved before any alterations are made to the research.			
Reporting Frequency:		Annual: Jan-07-2011	Other:
Date: Jan-07-2008		Chair, Dr. R. Storey/ Vice-Chairs, Dr. Tina Moffat & Dr. Bruce Milliken:	

July 7, 2010
amendment

http://iserv.mcmaster.ca/ethics/mreb/print_approval.cfm?ID=1856

6/22/2010

APPENDIX F: Consent form – Chapter 6

July, 2010

Letter of Information and Consent***A study evaluating upper extremity muscle contributions to elbow and wrist joint stiffness*****Investigators: Michael Holmes and Dr. Peter Keir**

Principal Investigator: Dr. Peter Keir
 Department of Kinesiology
 McMaster University
 Hamilton, Ontario, Canada
 (905) 525-9140 ext. 23543;

Student / Co-Investigator Michael Holmes
 Department of Kinesiology
 McMaster University
 Hamilton, Ontario, Canada
 (905) 525-9140 ext. 21334;

Purpose of the Study

Despite significant contributions to upper extremity research, the number of work related injuries remain high. It is thought that muscles play a large role in stabilizing a joint and further investigation into how they do this may help provide insight into how people become injured. The purpose of this study is to evaluate how muscles individually contribute to upper extremity tasks that involve the hand and wrist.

Procedures involved in the Research

Anthropometric measures (height, weight, arm length and hand length) will be recorded and you will be introduced to the protocol. Immediately following this you will have recording electrodes placed over 10 muscles on the forearm and upper arm. These electrodes allow us to record the activity in the muscles under them. To know how active your muscles are, we first need to determine the maximum activity for each muscle through a series of tests for each muscle. A device will also be placed at the wrist joint to measure wrist angle. Holding your wrist in a neutral posture a perturbation device will be positioned in a location that will cause wrist movement. The location of the perturbation (location of contact with the wrist) will be altered, producing situations that cause wrist flexion, wrist extension, wrist pronation or wrist supination. During each perturbation you will be required to perform three hand loading tasks. The hand loading conditions will require you to hold the bottom part of a tube shaped like a 'T'. One condition will be referred to as "solid" and involves holding the light weight tube. The second condition will be referred to as "fluid" and involves a tube that has the same weight as the tube in the 'solid' condition, but in this case the top part of the tube will be filled with water. Finally, there will be a no hand loading condition. All wrist perturbation directions will be performed using all hand loading conditions.

The device will push a padded rod outward, causing rotation of the wrist. The perturbation will be minimal, causing only a small change in angle at the wrist. A padded area on the device will ensure that the perturbation results in minimal discomfort to you. Your participation will require about 1.5 hours in the lab.

Potential Harms, Risks or Discomforts:

There is minimal risk associated with participation in this study. You may experience some muscle soreness as a result of the maximal exertions. Although very rare, you may experience a temporary reaction to the adhesive from the surface electrodes. Should you experience any serious discomfort following the study, please contact the principal investigator, Dr. Peter Keir. Due to the nature of the protocol, you will not be allowed to participate if you have been diagnosed with high blood pressure or have previous shoulder and wrist injuries.

Potential Benefits

We hope to understand the loads experienced within the body and relate them to injuries and disorders that develop in the workplace. Ultimately we hope to prevent workplace disorders. The research will not benefit you directly.

Payment or Reimbursement:

You will be financially compensated \$15.00 for your time and participation in this study.

Confidentiality:

Your identity will be kept confidential and the data collected will be used for teaching and research purposes only. The information directly pertaining to you will be locked in a cabinet for a maximum of 15 years.

You may be asked if you would be willing to have photos or video of you taken for use in publications and presentations. Photo and video data will only be used with your consent. Your name will not be associated with the images but someone viewing them might recognize your identity.

Participation:

Your participation in this study is voluntary. If you decide to participate, you can decide to stop at any time, even after signing the consent form or part-way through the study. If you drop out of the study, your data will only be used with your explicit consent. If you decide to stop participating, there will be no consequences to you and the compensation will be prorated. If you do not want to answer some of the questions you do not have to, but you can still be in the study.

Information About the Study Results:

You may obtain information about the results of the study by contacting Dr. Keir or Michael Holmes. An update will be emailed after completion of the study; if you would like an update your email will be required.

Information about Participating as a Study Subject:

If you have questions or require more information about the study itself, please contact Dr. Keir or Michael Holmes.

This study has been reviewed and approved by the McMaster Research Ethics Board. If you have concerns or questions about your rights as a participant or about the way the study is conducted, you may contact:

McMaster Research Ethics Board Secretariat
Telephone: (905) 525-9140 ext. 23142
c/o Office of Research Services
E-mail: ethicsoffice@mcmaster.ca

Note: Consent and signature sections are located on the following page.

CONSENT

I have read the information presented in the information letter about a study being conducted by Michael Holmes and Dr. Keir, of McMaster University. I have had the opportunity to ask questions about my involvement in this study, and to receive any additional details I wanted to know about the study. I understand that I may withdraw from the study at any time, if I choose to do so, and I agree to participate in this study. I have been given a copy of this form.

Name of Participant

Signature of Participant

I agree to allow the optional photos and videos of me to be taken during the task.

Photo

Yes _____

No _____

Videos

Yes_____

No _____

Signature of Participant

In my opinion, the person who has signed above is agreeing to participate in this study voluntarily, and understands the nature of the study and the consequences of participation in it.

Signature of Researcher or Witness