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Role of Sensation in Altered Phalanx Grip Force in Persons with Stroke

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ROLE OF SENSATION IN ALTERED PHALANX GRIP FORCE IN PERSONS WITH STROKE

by

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ABSTRACT
ROLE OF SENSATION IN ALTERED PHALANX GRIP FORCE IN PERSONS WITH STROKE

by

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Many individuals experience hand impairment after stroke leading to decreased ability to perform daily living activities. Previous research studies have investigated how stroke survivors' pinch grip control differs from healthy individuals, even though many individuals can only grasp with power grip after stroke. Furthermore, many stroke survivors experience tactile sensory deficit in their paretic limb in addition to motor deficit. It is currently unknown how stroke induced tactile sensory deficit affects power grip force directional control, which is important in terms of preventing object slippage and power grip normal force generation. Additionally it is unknown if power grip could be improved through tactile sensory enhancement. This dissertation investigated how stroke survivors' power grip force control is different from healthy individuals. Also, the effect of stroke induced tactile sensory deficit on power grip force control and the benefits of a sensory enhancement method using remote subsensory vibrotactile noise on power grip phalanx force deviation was assessed. In addition, the effect of noise on the tactile sensation for stroke survivors with tactile sensory deficit and their performance on two dynamic gripping tasks, the Box and Block Test ('BBT', number of blocks moved in

60 seconds) and the Nine Hole Peg Test ('NHPT', time to pick up, place, and remove 9 pegs from 9 holes), were investigated. The theoretical framework of this dissertation is that tactile sensation is critical for grip control and impairment or enhancement of tactile sensation impacts power grip force control post stroke. Results showed that stroke survivors, especially those with tactile sensory deficit, gripped with increased phalanx force deviation compared to healthy individuals, showing reduced directional force control and increasing their chances of dropping objects. Remote subsensory vibrotactile noise improved fingertip and upper palm tactile sensation for stroke survivors with tactile sensory deficit. The noise also improved phalanx force directional control during power grip (reducing phalanx force deviation) for stroke survivors with and without tactile sensory deficit and age-matched healthy controls and improved the BBT score and time to complete the NHPT for stroke survivors with tactile sensory deficit. Overall, stroke survivors, particularly those with tactile sensory deficit, appear to have reduced phalanx force control during power grip, which may biomechanically result from a muscle activation pattern. Remote subsensory vibrotactile noise may have enhanced tactile sensation and hand motor control via stochastic resonance and interneuronal connections and could have potential as a wearable rehabilitation device for stroke survivors. This dissertation contributes to the long term goal of increasing stroke survivors' independence in completing daily living activities.

DEDICATION

This is dissertation is dedicated to my mother, Jo, and my step-father, Doug, for their faithful and continuous support throughout my education.

I would also like to dedicate this dissertation to my grandparents, Kay and Fred, for the constant encouragement they provided.

I could not have succeeded without you guys.

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ABREVIATIONS

COF – Coefficient of friction

Atan - arctangent

FDP- flexor digitorum profundus

DI - dorsal interosseous

FDI - First dorsal interosseous

FDS-flexor digitorum superficialis

EDC- extensor digitorum communis

LUM- lumbricals

PI- palmer interosseous

MCP- metacarpophalangeal joint

FA I- fast adapting I tactile receptors

FA II- fast adapting type II tactile receptors

SA I – slow adapting I tactile receptors

SA II- slow adapting I tactile receptors

CNS - central nervous system

EMG – electromyography

MVC – maximum voluntary contraction

RMS – root mean square

ANOVA - analysis of variance analysis

SE – standard Error

SD – standard Deviation

NHPT – Nine Hole Peg Test

BBT – Box and Block Test

EEG- electroencephalography (EEG)

R –Right hand dominance

L – Left hand dominance

ACKNOWLEDGMENTS

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Chapter 1: Introduction[1]

1.1 Stroke Survivors' Hand Sensorimotor Impairment

There are greater than 7 million stroke survivors that currently reside in the

United States of America (Roger et al. 2012). Even after normal physical therapy

measures, up to 75% of stroke survivors experience persisting motor impairment in the

hands after 6 months of the stroke event (Feys et al. 1998; Olsen 1990; Parker et al.

1986). Loss of hand function can lead to limb disuse and can further worsen the level of

stroke survivors' disability. In addition, loss of hand function reduces independence by

leading to dependency on others to complete both simple and complex daily living

activities.

Hand function is particularly affected by stroke, potentially due to the high degree

of cortical control with the motor cortex and corticospinal pathways responsible for

controlling the hand muscles (Kamper 2012 ; Strick and Preston 1982). Altered activation

of the muscles controlling the hand has been observed for stroke survivors (Cruz et al.

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[1] Information presented in this chapter is adapted from published material as follows and is used with permission from:

Enders LR, Seo NJ, "Phalanx force magnitude and trajectory deviation increased during power grip with an increased coefficient of friction at the hand-object interface", Journal of Biomechanics, Elsevier, 2011, 44(8):1447-53.

Enders L.R. "Effect of hand-object friction on grip force application and hand function" Master's Thesis, University of Wisconsin-Milwaukee, 2010.

2005; Kamper et al. 2003; Kamper and Rymer 2001; Lang and Schieber 2004b), as well as a change composition of the muscles controlling the hand (Dattola et al. 1993; Hafer-Macko et al. 2008; Hu et al. 2007; Landin et al. 1977), muscle spasticity (Bhakta et al. 1996; Nathan et al. 2009), and muscle atrophy (Triandafilou and Kamper 2012) (Figure 1). Similarly, hand somatosensory feedback is often diminished post stroke (Carey 1995; Carey and Matyas 2011; Kim and Choi-Kwon 1996), potentially due to the damages in the somatosensory pathway as well as somatosensory cortex hand area. Since proper hand motor control depends significantly on sensory feedback from the hands (Johansson and Westling 1984), stroke induced sensory loss in the hand could contribute to decreased hand motor control. Therefore, stroke survivors are at a high risk for hand deficit due to neuronal damages and subsequently, reduced neuronal resources in the motor and sensory systems.
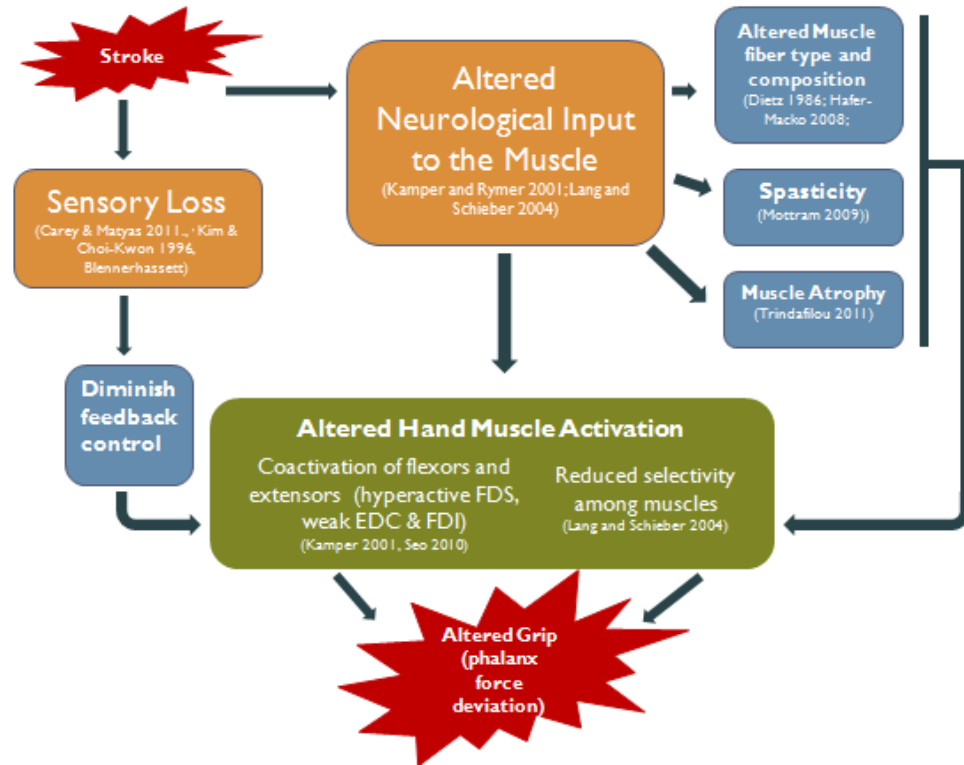
Figure 1: Stroke power grip may be altered (such as increased phalanx force deviation) due to altered neurological input, changes in composition of the muscles controlling the hand, muscle spasticity, muscle atrophy, and stroke induced sensory deficit.

Previous research investigations for stroke survivors' hand grip have focused predominantly on pinch grip (Hermsdorfer et al. 2003; McDonnell et al. 2006; Nowak et al. 2003), even though many stroke survivors with severe hand impairment are limited to grasping with power grip due to impaired finger individuation (Gowland et al. 1995; Lang and Schieber 2004b). Power grip, also referred to as cylindrical grip, is a type of hand grip technique where the fingers and thumb work together against the palm to produce force against the object being gripped (Landsmeer 1962) (Figure 2). Power grip is often utilized in tasks such as holding a bottle and carrying a cup of water. Studies that have focused on stroke survivors' power grip, have shown reduced power grip strength

(Radhakrishnan and Nagaravindra 1993), but have not investigated how power grip force

control is altered post stroke (discussed in Aim 1).
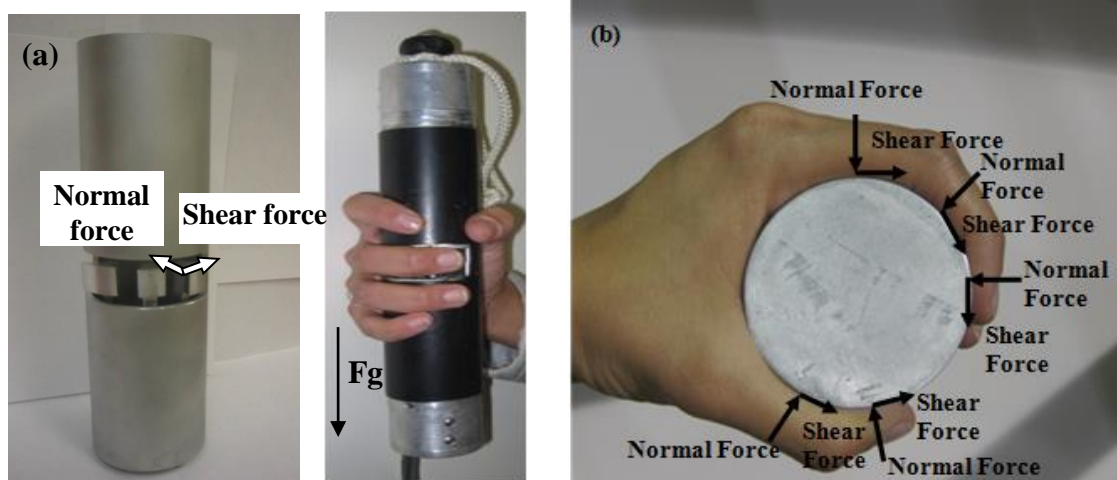
Figure 2. Proximal-distal shear force (tangential to the cylinder surface) and normal forces (perpendicular to the cylinder surface) can be measured on three contact pads that align with each phalanx of a finger during power grip using a new instrumented grip dynamometer (Enders and Seo 2011) (a). Normal and shear forces can be produced by the thumb and fingers against a cylindrical object during power grip (b). The cylinder was held so that the long axis of the cylinder was parallel to the direction of gravity, Fg.

In order to fully comprehend power grip impairment for stroke survivors,

different aspects important for power grip force control are outlined in this Introduction.

To understand how power grip control can be characterized for stroke survivors, the

different force elements that are commonly produced during power grip and how these

force elements contribute to gripping stability is first discussed. Second, important

muscles involved in controlling power grip force and the effect that altered muscle

activation resulting from the stroke could have on this power grip force control will be

discussed, followed by the importance of sensory feedback on phalanx force control and

the effect of stroke induced sensory deficit on phalanx force control. Finally, the effect that potential that sensory enhancement may have on stroke survivors' hand motor impairment is explained.

1.2 Elements of Power Grip

Power grip involves force generation by each phalanx of the five digits (Amis 1987; An et al. 1980; Lee and Rim 1991; Radhakrishnan and Nagaravindra 1993). During power grip, each phalanx of the digits produces force in three dimensions. When gripping a vertical cylinder, the three dimensional force at one phalanx can be decomposed to 1) normal force in the direction perpendicular to the cylinder surface, 2) shear force in the gravity direction, and 3) shear force in either the proximal or distal direction relative to a digit (referred to as proximal-distal shear force hereafter) (Figure 2). The shear force in the gravity direction is determined by the weight of the grasped object (Johansson and Westling 1984; Westling and Johansson 1984a). The proximal-distal shear force does not directly contribute to lifting of the grasped object, but people still do apply this shear force during power grip (Amis 1987; Irwin and Radwin 2009). Many of the research studies have not had the capacity to record both normal and shear forces from the individual phalanges of the finger during a power grip (Boissy et al. 1999; Naik et al. 2011). Even the use of three miniature load cells to align with the phalanges would be too bulky in structure to allow for a typical power grip posture. Recently, a new grip dynamometer has been developed that has the capacity to quantify normal and shear forces from each of three phalanges independently and simultaneously (Figure 2a)

(Enders and Seo 2011). Using this device, it was found that young healthy individuals produced a proximal-distal shear force during power grip that was, on average, 22% of normal force magnitude (Enders and Seo 2011). The ratio of proximal-distal shear force to normal force significantly varied between .09 and .43 depending upon the coefficient of friction between the surface and the finger skin, the grip effort level, finger, and the phalanx (Enders and Seo 2011). Therefore, it appears that young healthy individuals increase and decrease proximal-distal shear to normal force ratio depending on the demands of the power grip task and that proximal-distal shear force is an important force element applied during power grip.

Altering proximal-distal shear force during power grip could affect grip control and normal force generation during power grip. The extent of phalanx force deviation in the proximal-distal direction (calculated as the arctangent of the ratio of the phalanx proximal-distal shear and normal forces) can be used as an indication of phalanx force directional control and object stabilization (Figure 3). Stable grip requires phalanx force direction to not deviate from the direction normal with respect to a gripped object's surface, by more than an angle calculated as the arctangent of the coefficient of friction (COF) between finger skin and the object's surface, termed the 'cone of friction' (MacKenzie and Iberall 1994). Phalanx force deviations outside the cone of friction lead to finger slippage (MacKenzie and Iberall 1994; Seo et al. 2010). In addition to preventing object slippage form the hand, controlling phalanx force deviation has been shown to be necessary in preventing unwanted rotational forces that may occur during gripping (Kinoshita et al. 1997). Furthermore, recent evidence suggests that proximal-

distal shear force can contribute to people's normal force generation capacity. A biomechanical model (Wu et al. 2009) and empirical evidence (Seo et al. 2007) suggest that proximal-distal shear force can affect normal force generation, even when the muscle force is constant. Specifically, proximal-distal shear force on a phalanx can generate a moment about the joint proximal to the phalanx, independent from the joint moment produced by muscles (Seo et al. 2007; Wu et al. 2009). Thus, proximal-distal shear force can contribute to increased phalanx normal force. For example, distally-directed shear force at the distal phalanx (equal to proximally-directed reaction force from a cylinder to the distal phalanx) has been shown to increase distal phalanx normal force (Seo et al. 2007; Valero-Cuevas 2000; Wu et al. 2009). Individuals may utilize this mechanism to intentionally increase proximal-distal shear force to increase normal force during power grip. Proximal-distal shear force, therefore, is an important element of power grip in terms of grip control and power grip normal force generation.

$\theta = \text{ATAN (COF)} = $ "Cone of Friction"

Phalanx force deviation

$(\alpha) = \text{atan}\left(\dfrac{shear\ force}{normal\ force}\right)$
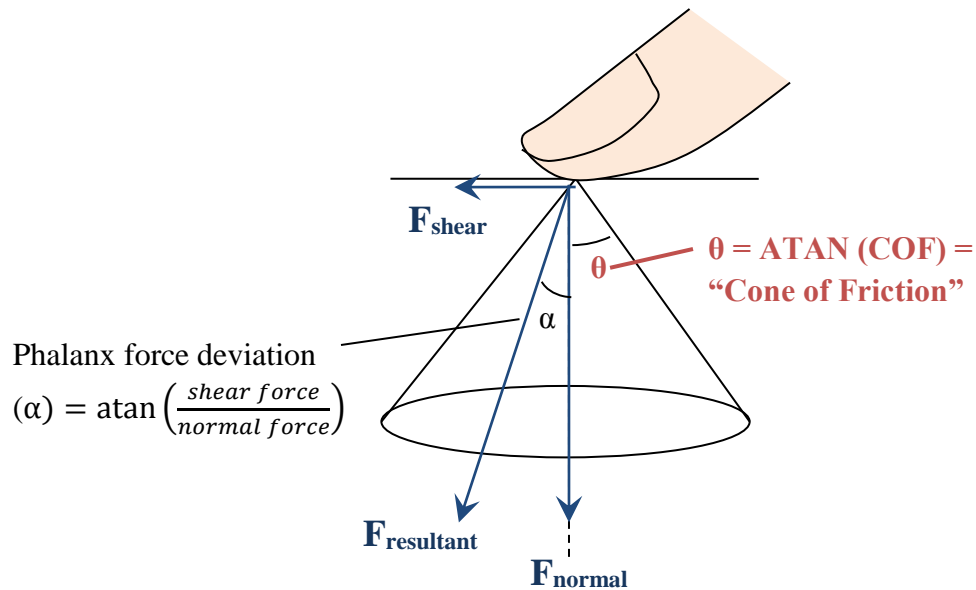
Figure 3: Phalanx resultant force, $F_{resultant}$, should not deviate from the normal direction with respect to the object surface (referred to as phalanx force deviation, α), more than that of certain angle range termed the 'cone of friction' (θ, determined by arctangent (atan) of the COF)in order to avoid slippage between the finger and the grip surface. In other words, the ratio of proximal-distal shear force (F_{shear}) (distal direction shown) to normal force (F_{normal}) should be less than COF (MacKenzie and Iberall 1994). The greater the COF, the greater the angle range that allows stable contact.

Altered grip force control has previously been shown for stroke survivors during pinch grip. Specifically, stroke survivors have displayed increased safety margin (Hermsdorfer et al. 2003) and reduced force control and timing (Hermsdorfer et al. 2003; Nowak et al. 2003). In addition, stroke survivors have displayed digit force deviations that are twice that of the non-paretic hand during pinch grip, leading to frequent finger slippage (55% of the trials)(Seo et al. 2010). However, how phalanx force control is altered for stroke survivors during power grip has not been studied. Therefore, phalanx force control (characterized by the extent of phalanx force deviation) is used to investigate stroke survivors' power grip (discussed in Aim 1).

1.3 Role of Altered Muscle Activation in Stroke Survivors' Reduced Phalanx Force Control

Grip requires coordination among all muscles for the hand to produce grip force, and disruption in the balance among individual muscles' force outputs can directly alter grip force control and magnitude (Johanson et al. 2001; Kutch and Valero-Cuevas 2011; Valero-Cuevas 2000). Weakening of any single muscle can limit the force production in a specific direction (Kutch and Valero-Cuevas 2011). Muscle activation is often altered for stroke survivors, in-part due to impaired neurological control (Kamper and Rymer 2001; Lang and Schieber 2004b), changes in muscle fiber type composition (Dattola et al. 1993; Hafer-Macko et al. 2008; Hu et al. 2007; Landin et al. 1977), muscle spasticity (Bhakta et al. 1996; Nathan et al. 2009), and muscle atrophy (Triandafilou and Kamper 2012). Altered muscle activation for stroke survivors' could reduce power grip force control (discussed in Aim 1).

The muscle groups controlling finger forces, for instance the index finger, can be broken down into two groups: intrinsic (i.e., lumbricals (LUM), first dorsal interosseous (DDI), palmer interosseous (PI)) and extrinsic muscles (i.e. flexor digitorum profundus (FDP), flexor digitorum superficialis (FDS), extensor digitorum communis (EDC)) (Figure 4). Each muscle of the finger is comprised of a different amount of Type I (slow-twitch, slow to fatigable fibers) and Type II (fast-twitch, fast to fatigable fibers), which assist in the delicate synchronization between the muscles to correctly coordinate force

control of the hand (Hwang et al. 2013). Intrinsic muscles are predominantly comprised

of Type II muscle fibers, while extrinsic muscles are composed mostly of Type I muscle

fibers (Hwang et al. 2013).
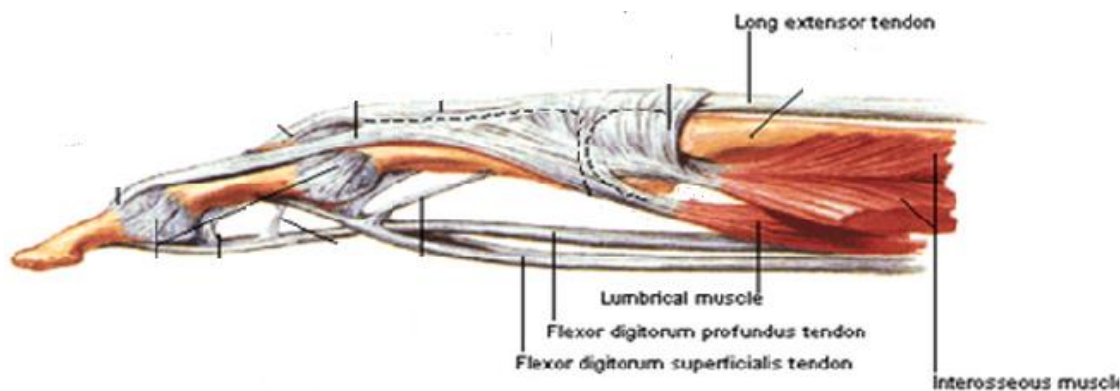
Figure 4: The two main groups of muscles that are important for power grip are the: 1)
intrinsic muscles (shaded in red) and 2) extrinsic muscles (shaded in green). These
muscles groups for the index finger include the intrinsic muscles: the lumbricles (LUM)
and the dorsi interossei (DI) which are important for force directional control and
produce some flexion force, and the extrinsic muscles: the flexor digitorum profundus
(FDP), flexor digitorum superficialis (FDS), and the extensor digitorum communis
(EDC) which are important for force production and finger stabilization (Lauer et al.
1999; Li et al. 2001; Long et al. 1970; Stack 1962).

In general, the extrinsic muscles supply much of the gripping force, while the

intrinsic muscles are important some force generation and fine motor control (Long et al.

1970). The degree, to which either muscle group is involved during a particular grip,

depends on the level of flexion of the phalanges and where the force is being applied

along the finger. For instance, during power grip the index finger's extrinsic muscles are

the producing the greatest amount of force and the index finger's intrinsic interosseous

muscle provides rotation of the phalanges and produces flexion force and stabilization of

the metacarpophalangeal joint (MCP) (Lauer et al. 1999; Li et al. 2001; Long et al. 1970; Stack 1962).

Specific weakening of the extrinsic muscles results in a loss of grip force (Long et al. 1970; Shinohara et al. 2003; Valero-Cuevas et al. 2000) and may affect the mechanical coupling of the fingers, since the extrinsic muscles interact with more than one finger at a time (Schieber 1995). Intrinsic muscle weakness (due to their predominantly Type II muscles fiber composition) have been shown to be particularly prone to atrophy due to age (Larsson et al. 1978; Lexell 1995), diabetes (Bus et al. 2002), and stroke (Dattola et al. 1993; Dietz et al. 1986; Hafer-Macko et al. 2008; Hu et al. 2007; Landin et al. 1977). Specific weakening of the intrinsic hand muscles can cause destabilization of the MCP joint, reduce flexion force, and reduces control of directed fingertip forces (Stack 1962; Valero-Cuevas et al. 2000). However, as previously mentioned, that weakening of any single muscle of the hand can actually affect force production in a specific direction (Kutch and Valero-Cuevas 2011).Therefore, for appropriate control of phalanx forces to achieve the desired force generation during power grip, coordination of both the intrinsic and extrinsic muscles of the hand is important.

1.4 Role of Diminished Sensory Feedback in Stroke Survivors' Reduced Phalanx Force Control

In addition to altered neurological input to the muscles for stroke survivors, diminished sensory feedback could also contribute to stroke survivors altered grip. Sensation loss of the upper extremity affects somewhere between 50% and 85% of all stroke survivors (Carey 1995; Carey and Matyas 2011; Kim and Choi-Kwon 1996) and could altered power grip force control because sensory feedback is critical in terms controlling grip forces (Johansson and Westling 1984). Therefore, the effects of stroke induced sensory deficit on power grip should be investigated (discussed in Aim 2).

In order to obtain, hold, and transfer an object with the hand, it is necessary for a person to apply finger forces that adapt to the physical properties of the object to create a stable and balanced grip. A person may vary the force application strategy (i.e., phalanx force deviation and normal force) in order to apply the correct amount and direction of force on the object's surface, depending on object weight, shape and friction coefficient between finger skin and object surface, using sensory feedback (Gordon et al. 1991; Johansson and Westling 1984; Westling and Johansson 1984b). Initial information regarding the object being gripped, such as the weight, friction, and shape, is sent by the fast adapting I (FA I) tactile receptors in the fingers (Johansson and Westling 1984), which provide information about the tangential shear forces (Macefield et al. 1996). The FA I receptors are also used throughout gripping to detect the need for force adjustment (Johansson and Westling 1984; Macefield et al. 1996). The fast adapting II (FA II) tactile

receptors give indications of rapid changes in the reactive grip forces (Macefield et al. 1996). After contact, during gripping, slow adapting I and II (SA I and SA II) receptors transmit information about the grip reactive forces and tangential shear forces, respectively (Macefield et al. 1996). More sensory information pertaining to reactive grip forces occurring during gripping is sent by the intrinsic and extrinsic hand muscle spindle and tendon-organ receptors and the interphalangeal joint mechanoreceptors (Macefield and Johansson 1996) which, although are less sensitive to tangential shear forces and surface characteristics (i.e., texture) than the tactile sensory receptors, these receptors still assist in controlling phalanx forces and loss of their input can result in increased latencies in reactive grip responses (Häger-Ross and Johansson 1996). Sensory feedback is transmitted to the central nervous system (CNS), which updates any control signals according to the current or any future stages of grip. This feedback control allows a person to use sensory feedback to appropriate the grip forces to prevent the object from slippage (Johansson and Westling 1984).

Sensation loss can occur due to age (Thornbury and Mistretta 1981), peripheral nerve damage (Braune and Schady 1993; Gelberman et al. 1983), or CNS lesions, such as stroke (Carey 1995; Carey and Matyas 2011; Kim and Choi-Kwon 1996). Loss of sensation feedback can lead to imbalance of grip forces, reduce coordination of forces, and yield non-adaptive gripping to varying frictional surfaces both during initiation and hold of grip (Johansson and Westling 1984). Therefore, decreased sensory feedback for stroke survivors may affect phalanx force control during power grip.

1.5 Motor Improvement via Sensory Enhancement

If power grip is found to be altered post stroke and sensory loss contributes to this altered power grip, then a rehabilitation therapy involving sensory enhancement has potential to assist stroke survivors regaining grip function and daily independence. Increasing sensation could give individuals important information regarding grip surface characteristics (Johansson and Westling 1987), magnitude and directional feedback on phalanx force being produced (Augurelle et al. 2003; Blennerhassett et al. 2007; Cole 2006; Hermsdorfer et al. 2003; Monzée et al. 2001; Robertson and Jones 1994) and more information on the finger position and alignment with respect to the object surface (Monzée et al. 2001). Furthermore, increasing somatosensory feedback has been shown to increase cortical excitation and activation in the motor cortices (Kaelin-Lang et al. 2002) and could lead to increased activation of muscles previously diminished in activation due to stroke. In addition, sensory feedback assists in the preservation of the normal cortical representations of both the motor and sensory cortices (Weiss et al. 2004). Reduced tactile sensation can further alter motor function for stroke survivors via impaired cortical sensorimotor representations (Weiss et al. 2004) and increasing tactile sensory feedback has the potential to redirect cortical representation towards normal cortical mapping. Based on this evidence, increasing tactile sensory feedback could be a promising method to improve motor function of the hand of stroke survivors (as discussed in Aim 3).

1.6 Dissertation Objectives

In summary, stroke power grip characteristics such as phalanx force direction may be altered post stroke due to altered neurological activation (Cruz et al. 2005; Kamper et al. 2003; Kamper and Rymer 2001; Lang and Schieber 2004b), a change composition of the muscles controlling the hand (Dattola et al. 1993; Hafer-Macko et al. 2008; Hu et al. 2007; Landin et al. 1977), and stroke induced sensory deficit (Carey 1995; Carey and Matyas 2011; Kim and Choi-Kwon 1996) (Figure 1). The overall objective of this dissertation was to determine the role of tactile sensation in altered power grip post stroke. The theoretical framework is that tactile sensation is critical for grip control and impairment or enhancement of tactile sensation impacts power grip force control post stroke. Specifically, how power grip is altered post stroke, especially with stroke induced finger tactile sensory deficit, was examined. Also investigated was if sensory enhancement via vibrotactile noise could improve stroke survivors' power grip. Stroke survivors' power grip was compared with age-matched neurologically healthy controls. The central hypothesis is that stroke survivors' tactile sensory deficit results in altered power grip with large phalanx force deviation and sensory enhancement improves grip control. These findings will contribute to the current knowledge base regarding altered grip for stroke survivors (Appendix) and can be applied to the development of rehabilitation techniques (such as sensory enhancement via the application of remote

subsensory vibrotactile noise) to improve stroke survivors independence in completing daily living activities. The following aims were proposed to test this hypothesis.

Aim 1: To characterize altered power grip post stroke.

> Study: To determine effects of stroke on power grip force control compared to healthy age-matched controls. It was hypothesized that phalanx force direction during power grip is altered for stroke survivors compared to age matched controls (Chapter 2).

Aim 2: To determine the role of tactile sensory deficit in stroke survivors' power grip control of phalanx forces.

> Study: To determine the difference in phalanx force directional control during power grip between stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and healthy age -matched controls. It is hypothesized that power grip phalanx force control is altered more for stroke survivors with hand tactile sensory deficit compared to stroke survivors without hand tactile sensory deficit and age matched controls (Chapter 3).

Aim 3: To determine the effect of sensory enhancement on power grip phalanx force control.

> Study 1: To determine the effect of remote subsensory vibrotactile noise on fingertip sensation. It is hypothesized that remote subsensory vibrotactile stimulation will increase fingertip sensation for stroke survivors with sensory deficit (Chapter 4).

> Study 2: To determine effects of sensory enhancement (via remote subsensory vibrotactile stimulation) on stroke survivors and age matched controls' power grip. It is hypothesized that remote subsensory vibrotactile stimulation will improve stroke survivors' and age matched controls' power grip phalanx force control (Chapter 5).

> Study 3: To determine effects of sensory enhancement (via remote subsensory vibrotactile stimulation) on dynamic hand grip control of stroke survivors who experience tactile sensory deficit. It is hypothesized that remote subsensory vibrotactile stimulation will improve stroke survivors'

dynamic grip control, as measured by the 9-Hole peg test and Box and Block Test (Chapter 6).

In addition to the aims, the entrepreneurial activity related to making a wearable rehabilitation device using subsensory remote vibrotactile noise and the current stages of prototype development is described (Chapter 7).

Chapter 2: Altered phalanx force deviation during power grip following stroke

Abstract

Many stroke survivors with severe impairment can grasp only with a power grip. Yet, little knowledge is available on altered power grip after stroke, other than reduced power grip strength. This study characterized stroke survivors' static power grip during 100% and 50% maximum grip. Each phalanx force's angular deviation from the normal direction and its contribution to total normal force was compared for 11 stroke survivors and 11 age-matched controls. Muscle activities and skin coefficient of friction (COF) were additionally compared for another 20 stroke and 13 age-matched control subjects. The main finding was that stroke survivors gripped with a 34% greater phalanx force angular deviation of $19\pm2°$ compared to controls of $14\pm1°$ ($p<.05$). Stroke survivors' phalanx force angular deviation was closer to the 23° threshold of slippage between the phalanx and grip surface (=atan(COF) found not to differ after stroke), which may explain increased likelihood of object dropping in stroke survivors. In addition, this altered phalanx force direction decreases normal grip force by tilting the force vector, indicating a partial role of phalanx force angular deviation in reduced grip strength post stroke. Greater phalanx force angular deviation and reduced grip strength may biomechanically result from more severe intrinsic and extensor muscle weakness compared to extrinsic flexor muscles, as empirically observed in stroke survivors. While stroke survivors' maximum power grip strength was approximately half of the controls', the distribution of their remaining strength over the fingers and phalanges did not differ, indicating evenly distributed grip weakness over the entire hand.

2.1 Introduction

Currently more than 7 million stroke survivors reside in the United States of America (Roger et al. 2012). Many of these stroke survivors suffer from impaired motor function in their hands and arms (Gray et al. 1990; Nakayama et al. 1994; Parker et al. 1986). Loss of hand function leads to dependency on others to complete both simple and complex daily living activities. As such, many studies examined how pinch grip control is altered after stroke (Hermsdorfer et al. 2003; McDonnell et al. 2006; Nowak et al. 2003). However, many stroke survivors suffering from severe impairment can grasp only with a power grip, and cannot perform a pinch grip due to impaired finger individuation (Gowland et al. 1995; Lang and Schieber 2004b). Yet, currently little knowledge is available on altered power grip after stroke, other than a reduced power grip strength (Boissy et al. 1999).

Power grip characteristics, such as phalanx force direction and force distribution over the hand, may differ post stroke. Biomechanics studies have shown that not only the action of the long finger flexor muscles but also the action of the extensor muscles and intrinsic hand muscles are important for controlling the force direction and distribution (Li et al. 2000; Valero-Cuevas et al. 2000). Altered muscle activation patterns, especially with under-activated intrinsic and extensor muscles, have been observed post stroke (Cruz et al. 2005; Kamper et al. 2003; Kamper and Rymer 2001; Lang and Schieber 2004b) and may disrupt the delicate balance among multiple hand muscles necessary for

force directional control or natural force distribution during power grip (Kutch and

Valero-Cuevas 2011; Li et al. 2001). Alternatively, changes in skin frictional properties,

if there are any after stroke, could affect the slipperiness of the finger skin against the

grip surface and modify grip force control, as it does for aging adults (Cole 1991).

These stroke related changes could affect power grip characteristics such as

phalanx force direction and force distribution over the hand, which can lead to the

decreased object stability and object dropping that is frequently observed in persons with

impaired hand function (Pazzaglia et al. 2010). Stable grip requires that phalanx force not

deviate from the direction normal to a gripped object's surface by more than an angle

defined as the 'cone of friction' (Figure 5), which is calculated as the arctangent of the

coefficient of friction (COF) between finger skin and the object's surface (MacKenzie

and Iberall 1994). Phalanx force direction outside the cone of friction leads to finger

slippage, which has been observed in stroke survivors during pinch grip (Seo et al. 2010).

In addition, deviation from the typical grip force distribution of the highest force

concentration on the distal phalanx directed toward the palm (Amis 1987; Kong and

Lowe 2005; Lee et al. 2009) could result in reduced grip force (Seo et al. 2007), object

rotation out of the hand (Kinoshita et al. 1997; Latash et al. 2002; MacKenzie and Iberall

1994), and discomfort (Gurram et al. 1993).
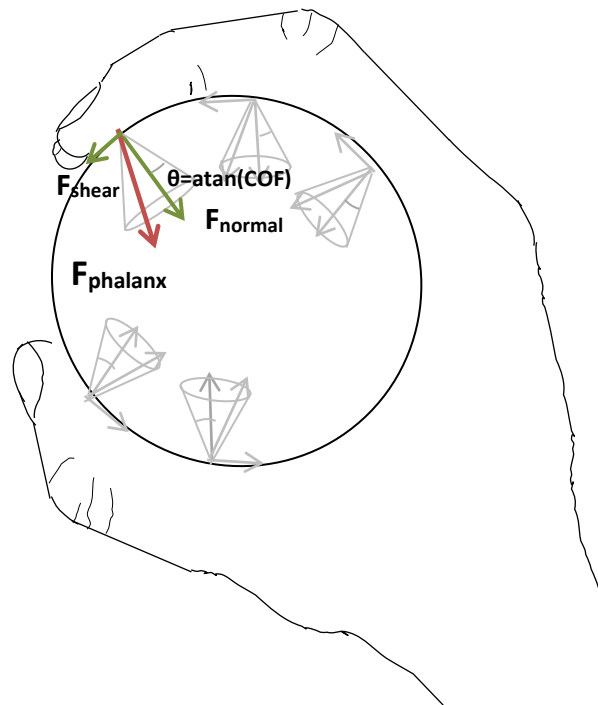
Figure 5: Stable grip without slippage requires that each phalanx's force not deviate from the direction normal to the object surface more than the cone of friction angle (θ), determined as arctangent of the coefficient of friction (COF) between the hand and grasped object (MacKenzie and Iberall 1994). Phalanx force deviations outside the cone of friction can lead to hand-object slippage

Despite these important functional implications for grip stability and strength, knowledge is sparse on the extent of altered phalanx force direction and altered force distribution across the fingers and phalanges during power grip post stroke. This knowledge gap is perhaps due to a lack of proper equipment. A recent development of an instrumented cylinder that has the capacity to measure not only normal force but also shear force from each phalanx and finger independently (Enders and Seo 2011) enables quantitative characterization of phalanx force direction and distribution during power grip post stroke. This new information on post-stroke power grip characteristics can provide

greater insight into stroke survivors' grip abnormality, especially the extent that altered phalanx force direction and distribution account for reduced whole-hand grip stability and strength.

The goal of this study was to characterize the altered power grip for people with stroke as compared with age-matched neurologically-intact (control) persons. The first experiment investigated the extent to which stroke survivors' phalanx forces deviated from the normal direction and the distribution of normal forces across the phalanges and fingers compared to controls during static power grip at 100% and 50% maximum perceived effort. In addition, the ability to approximate 50% of the maximum power grip force was examined to gauge the potential role of somatosensation in altered power grip post stroke. Upon observing greater phalanx force angular deviation post stroke, the second experiment was performed to examine potential mechanisms for altered phalanx force direction by comparing hand muscle activity between stroke survivors and healthy controls. In addition, the COF between the finger skin and grip surface was measured to determine any decrease in skin slipperiness that might allow greater phalanx force angular deviation after stroke.

2.2 Methods

2.2.1 Subjects

Eleven chronic stroke survivors (mean age ± standard deviation (SD) = 64 ± 11 years) and 11 age matched neurologically intact control subjects (65 ± 10 years) participated in Experiment 1 (Table I). Twenty chronic stroke survivors (mean age ± SD = 59 ± 11 years) and 13 age- matched control subjects (57 ± 8 years) participated in Experiment 2 (Table II). Roughly half of the participants were females for each Experiment. Six of the subjects returned from Experiment 1 to participate in Experiment 2. All stroke survivors had time since stroke greater than 6 months. The mean motor impairment for the stroke survivors in Experiment 1 was Stage 5 ± 2 out of the maximum score of 7 on the Chedoke-McMaster Stroke Assessment Hand Section (Gowland et al. 1995) and Stage 5 ± 2 in Experiment 2. For the hand and wrist subdivision of the Fugl-Meyer Assessment (Fugl-Meyer et al. 1975), the mean motor impairment was 19 ± 5 out of the maximum score of 24 for the stroke survivors in Experiment 1, and 19 ± 7 in Experiment 2. All subjects signed a consent form and followed a protocol approved by the Institutional Review Board.

Table I: Stroke Subject Demographics for Experiment 1

Subject	Approximate time since most recent stroke (months)	Type of Stroke	Hand Dominance		Paretic Hand Side	Sex	Age (years)	Fugl-Meyer Score (out of 24)	Chedoke McMaster Score (out of 7)
			Pre stroke	Post Stroke					
1	8	Hemorrhagic	R	R	L	Female	49	24	7
2	44	Hemorrhagic	R	R	R	Female	59	24	7
3	19	Ischemic	R	L	R	Male	79	-	2
4	86	Ischemic	R	R	L	Male	79	-	3
5	16	Ischemic	R	R	R	Female	73	24	6
6	22	Ischemic	R	R	R	Male	50	21	5
7	102	Hemorrhagic	L	R	L	Female	66	9	2
8	45	Ischemic	R	R	L	Female	58	16	6
9	46	Ischemic	R	R	R	Male	69	13	2
10	82	Ischemic	R	R	L	Male	59	16	5
11	80	Hemorrhagic	R	R	R	Male	63	24	7

*R= "Right hand dominance", L= "Right hand dominance"

Table II: Stroke Subject Demographics for Experiment 2

Subjects	Approximate time since most recent stroke (months)	Type of Stroke	Hand Dominance*		Paretic Side	Gender	Age (years)	Fugl-Meyer Score (out of 24)	Chedoke McMaster Score (out of 7)
			Pre Stroke	Post Stroke					
1	36	Unknown	R	L	R	Female	67	5	2
2	47	Hemorrhagic	R	R	L	Female	51	24	7
3	245	Ischemic	L	L	R	Female	55	24	7
4	48	Hemorrhagic	R	R	R	Male	52	21	5
5	53	Ischemic	R	R	L	Male	54	10	2
6	44	Ischemic	R	R	R	Female	75	24	6
7	40	Ischemic	L	L	L	Female	61	16	5
8	51	Ischemic	R	R	R	Male	60	23	7
9	126	Hemorrhagic	L	R	L	Female	67	9	2
10	67	Ischemic	R	R	L	Male	59	16	6
11	7	Ischemic	R	R	R	Male	66	24	7
12	55	Hemorrhagic	L	R	L	Male	32	8	2
13	162	Hemorrhagic	R	R	L	Male	60	22	7
14	52	Ischemic	R	L	R	Male	81	9	2
15	62	Unknown	R	R	R	Female	60	22	7
16	142	Ischemic	L	L	R	Female	53	24	7
17	64	Ischemic	R	R	L	Male	62	24	7
18	8	Unknown	R	R	L	Male	65	24	7
19	44	Ischemic	R	R	L	Female	43	18	3
20	172	Hemorrhagic	R	R	R	Female	50	24	7

*R= "Right hand dominance", L= "Right hand dominance"

2.2.2 Procedure and Analysis

Experiment 1 quantified phalanx force direction and distribution during 100% and 50% maximum power grip for stroke survivors as compared to controls. Experiment 1 also examined the ability to approximate 50% of the maximum grip force for stroke and control subjects. Subjects sat in a chair with the elbow flexed at approximately 90° and the forearm horizontally rested on an arm rest. Subjects performed power grip at 100% and 50% of their maximum perceived effort on a custom-made grip dynamometer

(Enders and Seo 2011) for at least five seconds, while individual phalanges' normal force and shear force in the proximal-distal direction were recorded at 1000 Hz for one finger at a time. For each phalanx, normal and shear forces were measured using two sets of four strain gauges in a Wheatstone bridge configuration instrumented in the custom-made grip dynamometer (Enders and Seo 2011). Two grip efforts were examined to facilitate comparison with previous literature using maximum grip (Radhakrishnan and Nagaravindra 1993) and to include submaximal grip for its relevance to daily activities. No visual feedback was provided to the subjects during gripping. Subjects were instructed to grip in a consistent manner regardless of the finger being measured. Subjects' distal, middle, and proximal phalanges of the finger were aligned with the three measuring pads on the grip dynamometer during power grip. If subjects were unable to correctly align the fingers themselves, the experimenter assisted the subject by aligning their finger with the three pads. Stroke subjects' paretic hand and control subjects' non-dominant hand were used because this hand often acts as the "stabilizing hand" to hold objects while opening containers or performing finer manipulation with the non-paretic or dominant hand (Sainburg 2005; Wang and Sainburg 2007). The entire surface of the grip dynamometer was covered with a paper surface. Measurements of phalanx normal and shear forces for all five fingers at the two effort levels were repeated three times each to obtain averages.

For Experiment 1 data analysis, phalanx force direction and normal grip force distribution were determined from the phalanx shear force and normal force during the two-second static grip period with the highest grip force within each trial. The phalanx

force angular deviation for each phalanx of each finger was quantified as the absolute arctangent of the ratio of mean shear force to mean normal force of that phalanx during the static grip period. A deviation of 0º indicates that phalanx force was in the normal direction, perpendicular to the grip surface with no shear force. Phalanx force deviation of either distal or proximal direction was noted separately. For the normal grip force distribution across the phalanges and fingers, the percentage contribution for each of the 14 individual phalanges of the hand to the total normal force was calculated during the same static grip period. The accuracy of approximating 50% of the maximum grip was examined using the ratio of the sum of each phalanx's resultant forces during the 50% maximum grip to that during the 100% maximum grip. Resultant force was used in this analysis since subjects may use feedback from both the normal and shear forces to approximate 50% of their maximum grip force.

Experiment 2 was performed in the same setting during the same gripping tasks, except that electromyography (EMG) from hand muscles was additionally recorded. EMG data from two extrinsic hand muscles, the extensor digitorum communis (EDC) and flexor digitorum superficialis (FDS), and one intrinsic muscle, the first dorsal interosseous (FDI), were recorded at 1000 Hz (Bortec Biomedical Ltd., Calgary, AZ). The EDC and FDS muscles were investigated to sample the extrinsic muscles, and the FDI was investigated to sample the intrinsic muscles, similar to previous studies (Kamper et al. 2003), to determine if altered phalanx direction was due to either intrinsic or extrinsic muscle-specific weakness post stroke.

Skin was cleaned with alcohol swabs to reduce impedance before the bipolar surface electrodes were placed on the muscle bellies according to literature (Basmajian 1989). The maximum voluntary contraction (MVC) EMG level was also recorded twice for each muscle by performing maximum voluntary contractions against resistance. For the EMG analysis, the root mean square (RMS) EMG with a 20-ms moving window was calculated for the two second static grip period with the highest grip force. These mean RMS values were further normalized by the RMS MVC for each muscle (%MVC). To examine altered muscle activation patterns post stroke, the relative FDI and EDC muscle activities in relation to the FDS muscle activity (calculated as the ratio of FDI to FDS EMG and that of EDC to FDS EMG in %MVC) were compared between stroke survivors and controls.

To compare the skin slipperiness between stroke survivors and healthy controls, the COF between the subjects' finger skin and the paper surface was measured during a series of finger drag tests. The subject's index finger tip was placed on a force transducer covered in paper and the experimenter applied 2N of normal force down on the finger tip, guided by a visual feedback display. Then force was increased in the shear direction until the finger slipped. The shear to normal force ratio at the point of the slip was then defined as the COF for that subject's finger skin and the paper surface. COF was measured twice for each subject.

2.2.3 Statistical Analysis

For Experiment 1 results, two separate mixed-design Analysis of Variance tests (ANOVAs) were used to examine if the phalanx force angular deviation and normal force distribution varied significantly for the subject group, effort level, phalanx, finger, and interactions between the subject group and the effort level, the subject group and the phalanx, and the subject group and the finger. Once a significant subject group effect was found for the phalanx force angular deviation, Pearson correlation was performed to examine the relationship between the motor impairment levels (both in the Chedoke-McMaster Stroke Assessment Hand Section and the hand and wrist subdivision of the Fugl-Meyer Assessment) and the mean phalanx force deviation of stroke survivors. For the ability to estimate 50% of maximum grip force, one-sample t-tests determined if the ratio of grip force during 50% perceived grip to that during maximum grip was significantly different from 50% for each subject group.

For Experiment 2, a mixed-design ANOVA was used to determine if muscle activation, EMG (%MVC), significantly varied for subject group, effort level, muscle, and the interaction between group and effort and the interaction between group and muscle. To further investigate how muscle activation pattern is altered post stroke, another mixed-design ANOVA was used to examine if the relative FDI and EDC EMG (normalized to FDS EMG) varied significantly for the subject group, effort level, muscle, and interactions between the subject group and the effort level and between the subject group and the muscle. For the COF data, a two-sample t-test determined if the stroke

survivors' COF differed from healthy controls. To ensure normality, a square root transformation was applied to the phalanx force deviation, normal force distribution, and COF data and a log transformation was applied to the muscle activation pattern data to result in non-significant skewness (Tabachnick and Fidell 2007), and these transformed data were used for the ANOVAs and t-tests.

2.3 Results

The overall static power grip force profiles for stroke survivors and controls obtained from Experiment 1 are shown in (Figure 6). The maximum total normal force for stroke survivors was 43% reduced compared to the control (154 N vs. 270 N). This extent of grip weakness is comparable to a previous study (Boissy et al. 1999). The new finding of this study is that phalanx force angular deviation is substantially greater for the stroke survivors. Altered phalanx force direction post stroke was associated with an altered muscle activation pattern with more reduced FDI and EDC muscle activities compared to the FDS activity and larger force estimation error after stroke, but without any skin friction change. This increased phalanx force deviation was significantly correlated with lower hand motor function in stroke survivors. The distribution of the remaining force across the phalanges and fingers was unaltered in stroke survivors. Detailed results are described below.
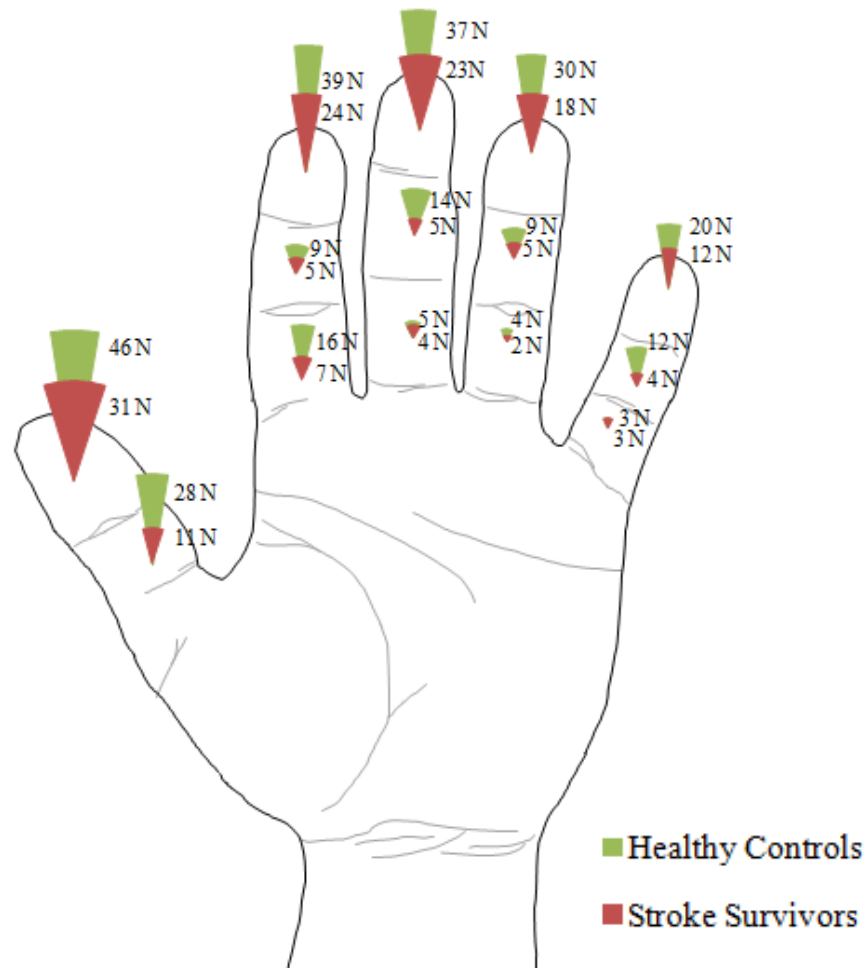
Figure 6: Mean phalanx force angular deviation, shown as the spread of the fan, was significantly greater for stroke survivors compared with healthy controls. Mean grip forces were substantially reduced for stroke survivors compared with healthy controls, as seen by the shorter fan height for stroke survivors.

2.3.1 Increased phalanx force angular deviation for stroke survivors

Stroke survivors gripped with 34% greater phalanx force deviation compared with controls on average (Figure 7a and Appendix B, ANOVA subject group main effect with $p=.03$). Phalanx force deviation was significantly dependent upon the subject group, phalanx, finger, and interaction between the subject group and the finger ($p<.05$). Stroke

survivors' phalanx force deviation was significantly greater than controls' for both grip

efforts and all phalanges (Figure 7b-c and Appendix B, ANOVA subject group main

effect with $p<.01$ and non-significant interactions between subject group and effort and

between subject group and phalanges with p>.05). The stroke survivors' phalanx force

deviation was significantly higher for the thumb, index, and little fingers, compared with

the controls (Figure 7b, ANOVA subject group and finger interaction with $p<.01$, and

post-hoc significance found for the three fingers with p<.05). The frequency of phalanx

force being distally directed was 56% for the stroke subjects, which is comparable to

47% for the controls.
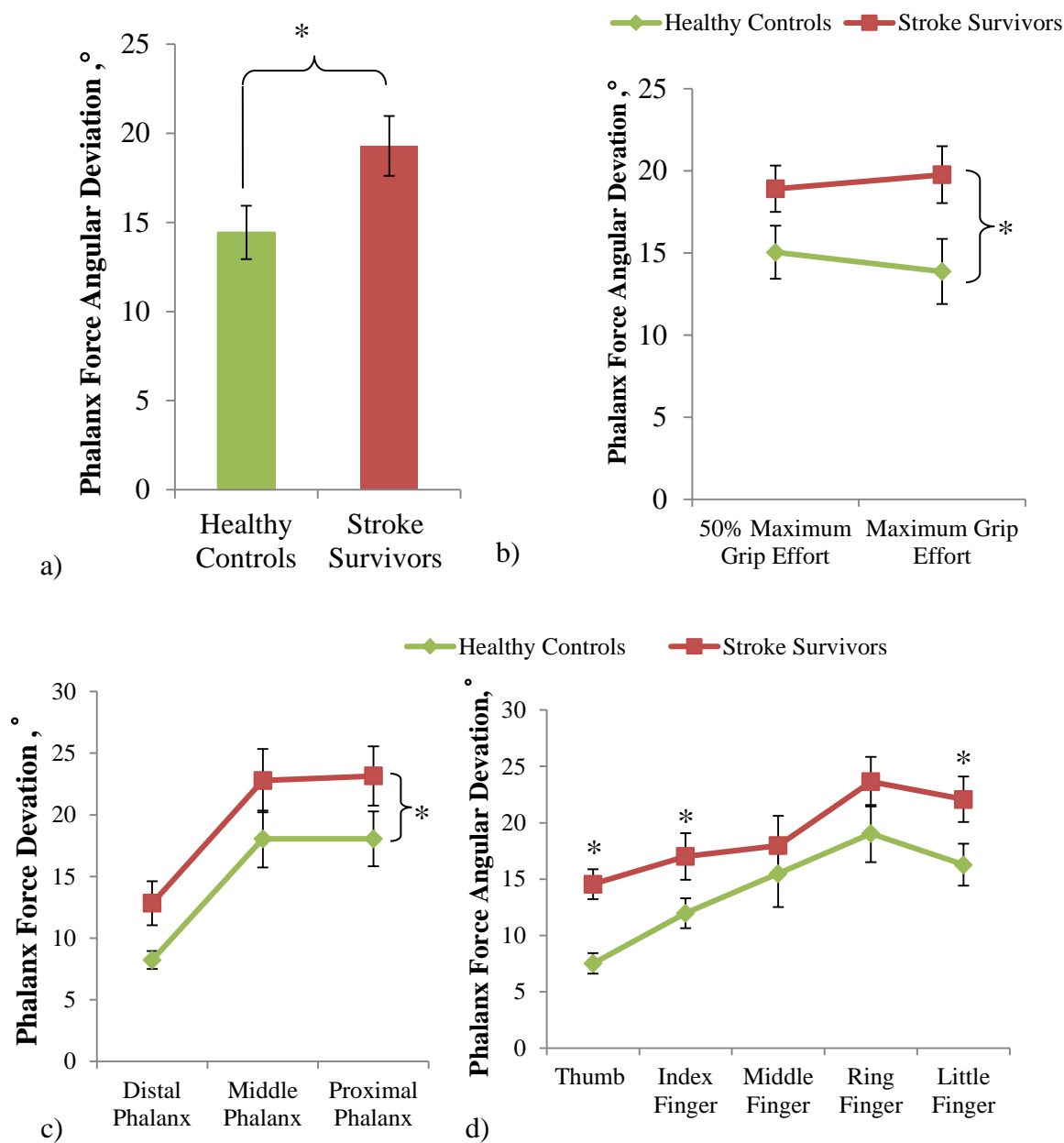
Figure 7: Phalanx force angular deviation was significantly greater for stroke survivors compared with controls (ANOVA subject group main effect with $p<.05$) (effort levels, fingers, phalanges, and subjects pooled) (a), for both 50% and maximum grip effort (b), for all three phalanges (c), and especially for the thumb, index, and little fingers (ANOVA, subject group and finger interaction with $p<.05$, posthoc significance marked with stars) (d). Non-transformed mean ± SE data is shown in the figure.

Increased phalanx force deviation was significantly and negatively correlated with motor impairment scores of the Chedoke-McMaster Assessment Hand Section (Figure 8a, Pearson Correlation, r = -.84 with p<.05). Stroke survivors' increased phalanx force deviation was also significantly and negatively correlated with a lower motor function score on the hand and wrist subdivision of the Fugl-Meyer Assessment (Figure 8b, Pearson Correlation, r = -.79 with p<.05).
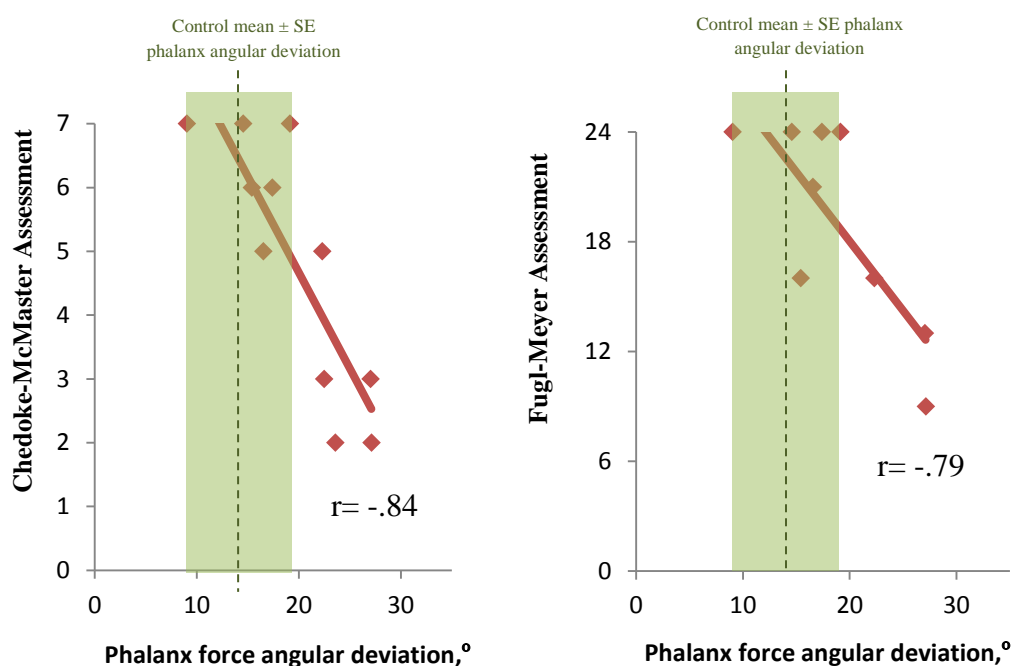
Figure 8: Stroke survivors' increased phalanx force deviation was significantly correlated with lower motor function scores of the Chedoke-McMaster Assessment Hand Section (Pearson Correlation, r = -.84 with p<.05) (a) and the hand and wrist subdivision of the Fugl-Meyer Assessment (Pearson Correlation, r = -.79 with p<.05) (b).

2.3.2 Similar grip force distribution

The distribution of normal force across the fingers and phalanges was similar between stroke and controls: They both gripped with the largest normal force produced by the distal phalanges (Figure 9a,c and Appendix B) and the thumb (Figure 9b,d and Appendix B), consistent with the previous study (Radhakrishnan and Nagaravindra 1993). The percent contribution of the phalanx normal force to the total normal force was significantly dependent on the phalanx and the finger (ANOVA with $p<.05$), but not significantly dependent on any other factor or interaction with the subject group (ANOVA with $p>.05$). Similar observations were made when resultant force magnitudes (instead of normal force) were examined for distribution across the fingers and phalanges, with no significant difference between the two subject groups.
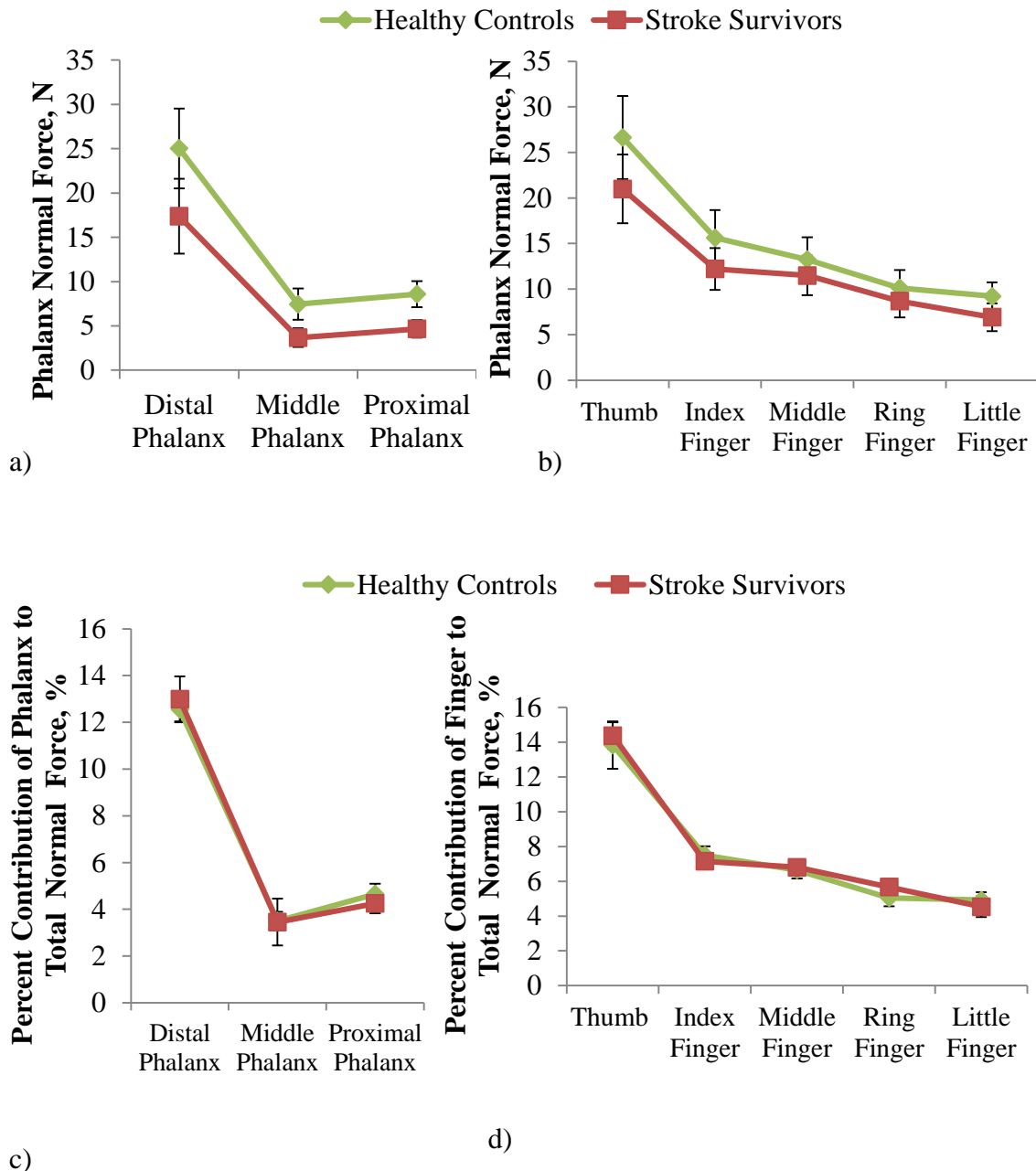
Figure 9: The distribution of phalanx normal force across the phalanges (a and c) and fingers (b and d) for stroke and control subjects. Percent contribution (c and d) of the individual phalanges to total normal force was not significantly dependent upon the interaction of subject group and phalanx or the interaction of subject group and finger (ANOVA with $p>.05$) (d). Non-transformed mean ± SE data is shown in the figure.

2.3.3 Overestimation of 50% grip for stroke survivors

During the 50% effort grip, the control subjects gripped with 46% ± 3% (mean ± SE) of their maximum grip force, which was not significantly different from 50% (Figure 10 and Appendix B, t-test with *p*>.05). On the other hand, the stroke survivors gripped with, on average, 68% ± 11% of their maximum grip force, which was significantly different from 50% (Figure 10 and Appendix B, t-test with *p*<.05).
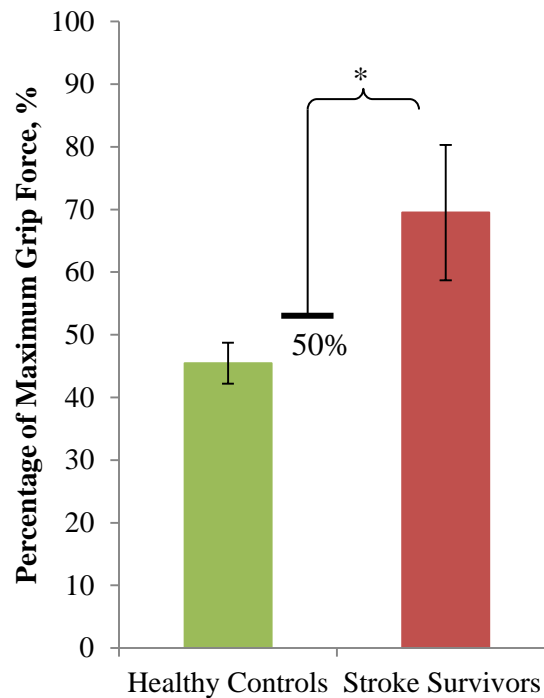
Figure 10: Mean ± SE percentage of force produced during grip at 50% of maximum perceived effort. Stroke survivors produced more than 50% of maximum (t-test with *p*<.05), unlike controls (t-test with *p*>.05). Non-transformed data is shown in the figure.

2.3.4 Altered muscle activity pattern post stroke

Each muscle's activity (in %MVC) is shown for the stroke and control groups in Figure 11a and Appendix B. Muscle activity was significantly dependent upon subject group, effort level, muscle, and the interaction between group and muscle (ANOVA with $p<.05$). While the overall muscle activity was lower for stroke survivors compared with control, the reduction in muscle activity was more pronounced for the EDC and FDI muscles compared to the FDS muscle.

This greater reduction in the FDI and EDC muscle activity than the FDS muscle for stroke survivors is apparent when the relative muscle activity is examined (Figure 11b and Appendix B). Compared to healthy controls, stroke survivors' FDI and EDC activities relative to FDS muscle were significantly lower (ANOVA subject group main effect with $p<.05$ as well as Tukey posthoc $p<.05$ for stroke vs. control for both muscles). The under-activation was greater for the FDI than EDC muscle (ANOVA, muscle main effect and subject group and muscle interaction with $p<.05$). The interaction between group and effort was also found to be significant ($p<.05$) while the effort main effect was not ($p>.05$).

(a)

(b)
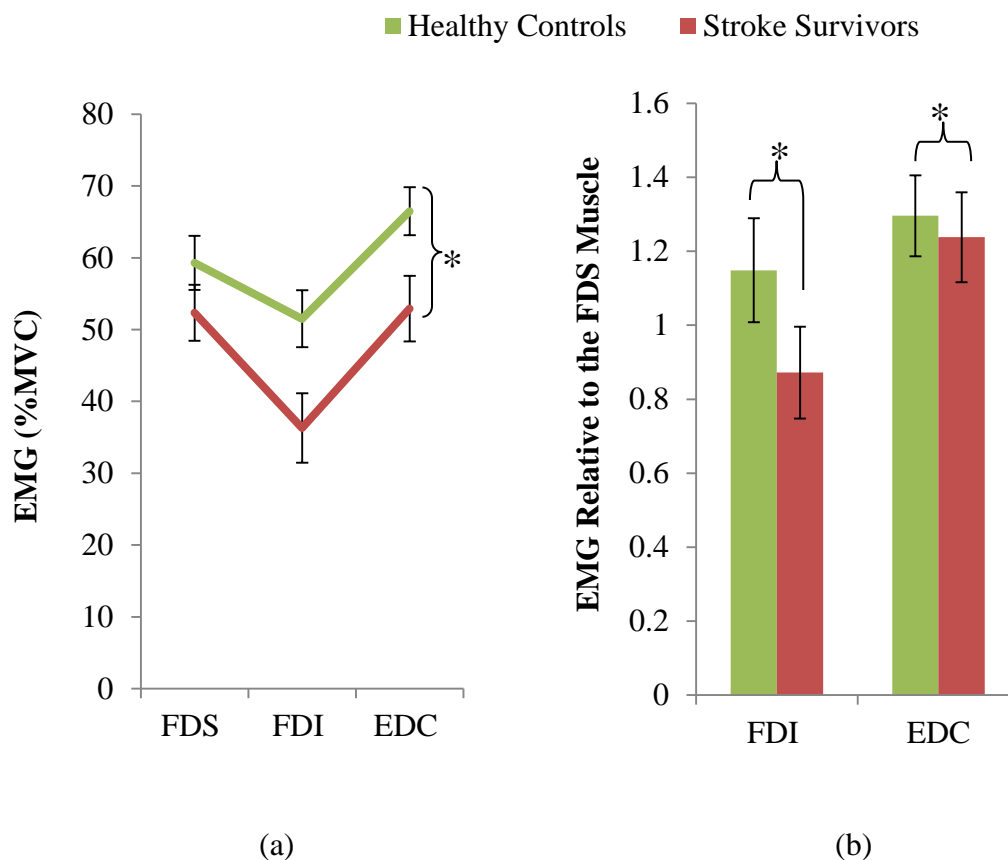
Figure 11: Mean ± SE EMG was reduced for all muscles of the stroke survivors compared with healthy controls (a). Relative to the FDS EMG, mean ± SE FDI and EDC EMG were significantly reduced for stroke survivors compared with controls (significant subject group and finger muscle interaction with p<.05, significant difference in relative FDI and EDC EMG between stroke and control with Tukey post-hoc p<.05) (b), showing an altered muscle activity pattern with a particularly weakened intrinsic FDI muscle and the extrinsic EDC muscle for stroke survivors compared with controls. Non-transformed data is shown in the figure.

2.3.5 Similar skin COF

There was no significant difference in COF between the finger skin and the paper grip surface between the two subject groups (Figure 12 and Appendix B, t-test with $p>.05$). The mean COF of the paper surface with the finger skin was 0.43, similar to previous findings for healthy adults ranging from 0.3 (Buchholz et al. 1988) to 0.5 (Gee et al. 2005). This COF value means that the threshold for slippage (the maximum phalanx

force deviation allowed before slippage, calculated as arctangent of the COF) for the grip

surface used in this study was 23˚ for both stroke survivors and controls.
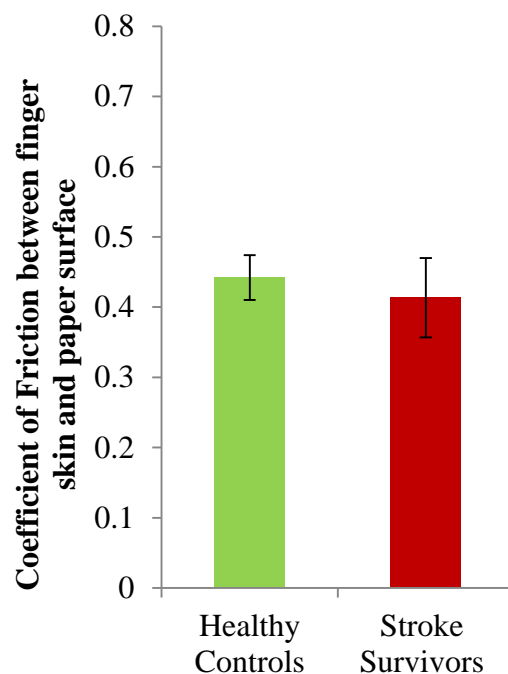
Figure 12: Mean ± SE of the COF between the finger skin and paper surface was similar for stroke survivors and healthy controls (t-test, *p*>.05). Non-transformed data is shown in the figure.

2.4 Discussion

2.4.1 Altered power grip force profile for stroke and potential mechanisms

Post-stroke power grip was characterized by 34% greater phalanx force angular

deviation accompanied by weakened FDI and EDC muscle activities relative to FDS and

inaccurate force estimation, compared to age-matched controls. The skin friction did not

differ, indicating that this change in phalanx force direction is not mediated by skin

friction change. This increased phalanx force deviation was significantly correlated with lower motor function scores. Consistent with the previous study (Boissy et al. 1999), stroke survivors produced reduced maximum grip force compared with the age-matched controls. However, the distribution of the remaining grip force across the fingers and phalanges of the hand was similar to the controls.

One of the possible explanations for this greater phalanx force angular deviation during power grip after stroke is disruption in the coordinated force outputs across individual muscles for the hand. Both the FDI and EDC muscles were under-activated for stroke survivors, with greater weakness observed for the FDI muscle compared to the EDC muscle. Grip requires coordination among all muscles for the hand to produce grip force toward the object, and disruption in the balance among individual muscles' force outputs can directly alter the direction of phalanx forces (Johanson et al. 2001; Valero-Cuevas 2000). For instance, weakening of any single muscle can limit force production in a specific direction (Kutch and Valero-Cuevas 2011). Specifically, the intrinsic muscles are important for directional force control (Long et al. 1970; Milner and Dhaliwal 2002; Valero-Cuevas et al. 2000). Weakening of the intrinsic muscles has been shown to increase digit force angular deviation from the normal direction based on a biomechanical model (Valero-Cuevas et al. 2000), and this weakening has been speculated to contribute to altered digit force deviation in older adults (Cole 2006). Therefore, the increased phalanx force angular deviation may be attributable to the observed altered muscle activation pattern with more weakened intrinsic and extensor muscles relative to the long finger flexor muscles after stroke. This specific pattern of altered muscle activation after

stroke was observed in the past (Seo et al. 2010) and is thought to be mediated by a disinhibited reticulospinal tract resulting in the hyperexcited long finger flexor muscle (Zaaimi et al. 2012) relative to other muscles. In addition, the distal intrinsic muscles may require more corticospinal drive than more proximal muscles (Palmer and Ashby 1992; Turton and Lemon 1999), leaving them more vulnerable to weakness post-stroke. Furthermore, intrinsic muscles may suffer from disproportionately greater weakness due to changes occurring within the muscles: intrinsic muscles are composed predominantly of Type II muscle fibers (Hwang et al. 2013), which have been shown to be particularly prone to atrophy post stroke (Dattola et al. 1993; Hu et al. 2007).

While the altered muscle activation pattern with relatively more weakened intrinsic and extensor muscles appears to have contributed to greater phalanx force deviation following stroke (Figure 11), force distribution over the phalanges and fingers remained unchanged after stroke (Figure 9). This preserved force distribution could be due to the relatively minor contribution of the intrinsic and extensor muscles toward grip force generation compared with the large long finger flexor muscles (Li et al. 2000). Intrinsic muscles account for less than 13% of the metacarpophalangeal joint moment and their contribution toward distal phalanx flexion force production is minimal (Li et al. 2000). Likewise, the extensor muscles appears to contribute very little to the flexion force of the fingers (Chao et al. 1976) but are important in terms of joint stability (Chao et al. 1976). As such, the trend with the major force concentrated on the distal phalanges powered by the extrinsic flexor muscles did not change after stroke in this study. In

general, FDI and EDC weakness does not appear to affect the power grip force
distribution over the fingers and phalanges.

Another explanation for altered phalanx force direction for stroke survivors is a
difference in finger posture with respect to the gripping surface compared to healthy
controls. Although subjects' finger alignment to the three measuring pads was controlled
when the subjects' hand was placed on the dynamometer initially for each trial, it is
possible that the position and orientation of the phalanges shifted during power grip
exertion. For instance, stroke subjects' altered muscle activation could have caused a
curling or rotation of the finger during the grip, affecting the direction of the force vector
and causing an increase in the proximal-distal shear force. Although finger postures were
not recorded in the present study to substantiate this possibility, the results of the present
study show that regardless of the posture, the force vectors applied to the object differed
for stroke survivors compared to healthy controls, which has implication for grip stability
during daily activities involving power grip as discussed in the next section.

An alternative explanation for the greater phalanx force angular deviation during
power grip after stroke is impaired somatosensation post stroke (Carey 1995; Di Fabio
and Badke 1991; Hermsdorfer et al. 2003; Niessen et al. 2008; Turton and Butler 2001).
Somatosensory feedback has previously been shown to be critical in the control of digit
force magnitudes and trajectories during gripping (Enders and Seo 2011; Nowak et al.
2001; Shim et al. 2012; Zatsiorsky and Latash 2004a). The diminished somatosensation

often found for stroke survivors (Carey 1995; Turton and Butler 2001) has been shown to contribute to stroke survivors' excessive force fluctuation (Blennerhassett et al. 2007), inappropriate grip force regulation (Blennerhassett et al. 2007), and improper safety margins (Hermsdorfer et al. 2003). The stroke survivors tested in this study exhibited impaired somatosensation, as seen by the overshooting of grip force estimation at 50% of maximum effort (Figure 10 and Appendix B). The impaired somatosensation could have hindered stroke survivors from correcting their phalanx force direction reaching toward the threshold of slippage, resulting in the increased phalanx force deviation observed in this study.

2.4.2 Functional implications of stroke survivors' altered phalanx force deviation

As previously discussed, the phalanx skin slips against the gripped object surface when phalanx force deviation reaches the cone of friction, calculated as the arctangent of the COF between the finger and the grasped object (MacKenzie and Iberall 1994) (Figure 5). Stroke survivors produced an average $19° ± 2°$ phalanx force deviation, closer to the slip threshold of $23°$ for the grip surface used in the present study, compared with controls who kept their phalanx force deviation low at $14° ± 1°$. The COF between the finger skin and the grip surface was not significantly different between stroke survivors and controls (Figure 12 and Appendix B), indicating that both groups had the same slip threshold and the increased phalanx force deviation was not afforded by increased skin COF post stroke.

This phalanx force deviation near the slip threshold post stroke represents grip instability and likelihood of object dropping. Indeed, a previous study found that excessive digit force deviation for stroke survivors was accompanied by finger slippage of at least 1 cm in 55% of all pinch grips (Seo et al. 2010). The increased phalanx force deviation near the slip threshold also implies potential hand slippage while stroke survivors try to hold onto a support pole in a bus or in the shower, which could lead to falling and serious injury. Given the tight relationship between the phalanx force deviation and grip stability, it is not surprising that stroke survivors with greater phalanx force deviation were found to have lower motor function scores indicating difficulty in hand grip function.

In addition to greater finger-object slippage, greater phalanx force deviation can lead to reduced phalanx normal force. Increasing phalanx force deviation can decrease the phalanx normal force by tilting the force vector such that the force in the normal direction decreases and force in the shear direction increases. Specifically, in the present study, the phalanx force deviation reduced stroke survivors' potential normal force by approximately 14% (calculated by taking the average difference between phalanx resultant and normal force).

Furthermore, applying more grip force than is required by the task, such as the grip force overshoot observed in this study during the 50% maximum grip task, can lead

to an earlier onset of muscle fatigue and decrease one's ability to perform daily activities (Nowak et al. 2003). This could be especially true for the tasks where a high grip effort is required, such as cooking, holding onto a bar while riding the bus or train, or pushing and pulling a cart in a store.

2.4.3 Study Limitations and Future Directions

One limitation of this study is that each trial only recorded phalanx force data from one finger at a time. Recording forces from all fingers at the same time would be preferable. However, the custom-made device does not allow for simultaneous measurements across fingers due to space constraints for the strain gauges inside. Instead, subjects were instructed to grip the device with the whole hand in a consistent manner regardless of what finger was placed on the measuring pads. Another limitation of this study is that only the proximal-distal shear force was recorded, again due to the device limitation. Therefore, the medial-lateral shear force was neglected in this study and phalanx force deviation only took into account the normal force and proximal-distal shear force. Because grip is performed primarily in the finger flexion direction and abduction/adduction strength is severely weakened post stroke (Lang and Schieber 2004a), and because the grip dynamometer was supported against gravity in this study, only a very small amount of shear force is expected to be applied in the medial-lateral direction.

2.5. Conclusions

The present study demonstrated that stroke survivors perform power grip with greater phalanx force deviation compared to age-matched controls, although the slip threshold between the skin and grip surface was not significantly different between stroke survivors and controls. Distribution of phalanx grip force was similar for stroke survivors and healthy controls. Altered muscle activation patterns with reduced activation of the FDI and EDC muscles compared with the FDS muscle may account for increased phalanx force deviation. Impaired somatosensation following stroke may also account for the increased phalanx force deviation as well as the grip force overshoot. Furthermore, impaired posture, shifting the position and orientation of the phalanges during power grip exertion due to stroke survivors' altered muscle activation could have increased phalanx force deviation. Increased phalanx force deviation could reduce the grip strength and increase the likelihood of finger-object slippage, thus leading to reduced grip stability and an increased rate of object dropping or loss of grip. In addition, stroke survivors' grip force overshoot may indicate that they develop muscle fatigue earlier in tasks requiring submaximal force. Decreased phalanx force control and grip force overshoot may limit stroke survivors in completing everyday tasks and progressing in rehabilitation, leading to long-term negative effects on hand function post stroke. The knowledge obtained in this research could be applied to developing more sophisticated rehabilitation therapies or assistive devices that correct altered phalanx force deviations and assist in approximating target grip force levels.

Chapter 3: Effects Of Tactile Sensory Deficit On Phalanx Force Deviation During Power Grip Post Stroke

ABSTRACT

When stroke survivors apply grip force, force from each phalanx is directed further from the direction normal to the object surface, compared to age-matched stroke-free adults. This study examined how tactile sensory deficit, in addition to motor deficit, plays a role in altered phalanx force direction during static power grip. Three groups (stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and age-matched controls) gripped an instrumented cylinder at 100% and 50% maximum efforts. The two stroke groups were similar in motor impairment and grip strength. Each phalanx's normal force and direction as well as muscle activity were recorded. The main finding was that the stroke survivors with tactile sensory deficit gripped with 14% and 24% greater phalanx force deviation compared to stroke survivors without tactile sensory deficit and healthy controls, respectively ($p<.05$). Altered muscle activity pattern compared to controls, with greater weakness in the intrinsic and extensor muscles than in the long flexor muscle, was observed for the stroke survivors with tactile sensory deficit ($p<.05$), but not for the stroke survivors without tactile sensory deficit. All three subject groups estimated 50% maximum power grip force closely, showing that neither stroke groups exhibited proprioceptive sensory deficit. All groups had similar skin friction. In summary, stroke survivors with tactile sensory deficit were found to grip with more altered phalanx force direction than those without, compared to healthy controls, accompanied by an altered muscle activation pattern, which could elevate the risk of dropping grasped objects, hampering their ability to complete daily activities.

3.1. Introduction

Of the 7 million stroke survivors in the U.S. (Roger et al. 2012), many experience unilateral arm and hand functional loss (Gray et al. 1990; Nakayama et al. 1994; Parker et al. 1986) that continues to persist even after physical therapy (Parker et al. 1986). Post stroke hand impairment is further complicated by somatosensory loss in the hands (Carey 1995). The reduced hand function affects stroke survivors' ability to complete common gripping tasks and can lead to increased dependence on others to perform daily living activities.

Approximately 50% to 85% of stroke survivors experience tactile and proprioceptive sensory deficit (Carey 1995; Kim and Choi-Kwon 1996) in addition to motor deficit in their hands and arms (Gray et al. 1990; Nakayama et al. 1994; Parker et al. 1986). While proprioceptive sensory information from the muscle spindle and tendon-organ afferents and the interphalangeal joint receptors pertains to limb positions and muscle force during gripping (Macefield and Johansson 1996), the tactile sensory feedback is the dominant type of sensory feedback used for the initial force scaling and the majority of finger force feedback control (Häger-Ross and Johansson 1996). When the finger comes in contact with an object for grasping, tactile feedback is sent via activation of the mechanoreceptors in the fingertips (Johansson and Westling 1987). This tactile sensory feedback is important for appropriating the necessary finger force magnitudes and direction for a successful grasp (Augurelle et al. 2003; Monzée et al. 2001; Nowak et al. 2001; Robertson and Jones 1994; Zatsiorsky and Latash 2004b). Post

stroke tactile sensory deficit has previously been linked to impaired detection of contact with an object (Turton and Butler 2001), increased latencies in pinch grip activation during lifting tasks (Blennerhassett et al. 2007), problems regulating grip force (Blennerhassett et al. 2007), and increases in safety margin (Hermsdorfer et al. 2003).

In addition to its implications on pinch grip force control, post stroke tactile sensory deficit may account for impairment in power grip force control. In our recent study, it was found that stroke survivors perform static power grip with phalanx forces deviated further from the normal direction with respect to the object surface, referred to as increased phalanx force deviation, compared to healthy controls. Power grip was examined because little knowledge exists with stroke survivors' power grip force control even though stroke survivors with severe impairment are unable to perform pinch grip and are limited to power grip (Gowland et al. 1995; Lang and Schieber 2004b). A successful grasp requires phalanx force to not be deviated from the direction normal to an object's surface, by more than an angle calculated as the arctangent of the coefficient of friction (COF) (MacKenzie and Iberall 1994) (Figure 13). Increased phalanx force deviation can lead to finger slippage and hamper hand grip function (Seo et al. 2010). Also, increased phalanx force deviation results in a tilting of the phalanx force and could reduce the phalanx normal force. This altered power grip for stroke survivors may increase the likelihood of object dropping due to reduced gripping stability and overall grip force. Since tactile sensory feedback has previously been shown to be important for gripping force control (Nowak et al. 2001; Shim et al. 2012), stroke survivors who experience tactile sensory deficit could be at greater risk for altered phalanx force

deviation and object dropping during power compared to stroke survivors with normal

tactile sensation as well as their healthy counterparts.
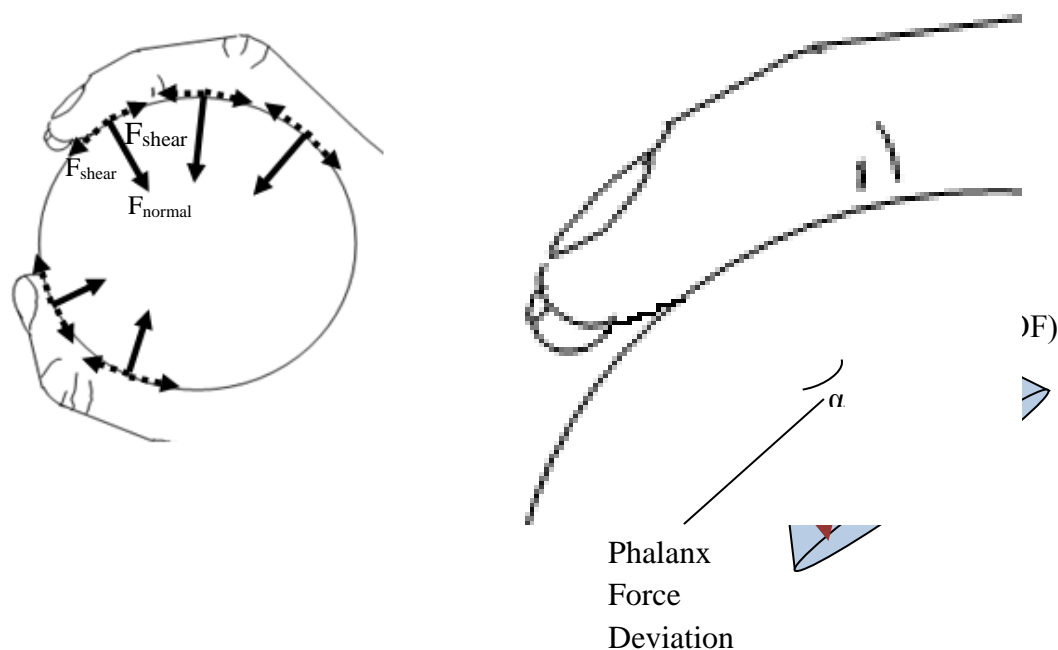
Figure 13. During power grip, there are normal and shear forces produced by the phalanges. (a) Phalanx slippage occurs when phalanx force deviation (α) exceeds a certain angle (θ) determined as arctangent (atan) of the COF between the finger skin and object.

Stroke survivors' increased phalanx force deviation was also previously found to

be accompanied by an altered muscle activation pattern with reduced activation of the

intrinsic and extensor muscles (first dorsal interosseous and extensor digitorum

superficialis) more so than the extrinsic flexor muscle (flexor digitorum superficialis)

(Chapter 2). Altered muscle activation pattern is thought to biomechanically account for

increased phalanx force deviation since the intrinsic and extensor muscles are important for directing and stabilizing the digit force (Kutch and Valero-Cuevas 2011; Long et al. 1970; Milner and Dhaliwal 2002; Valero-Cuevas et al. 2000). Intrinsic muscles may be particularly affected post stroke due to their need for corticospinal drive (Palmer and Ashby 1992; Turton and Lemon 1999) and their dominant Type II muscle fiber composition (Hwang et al. 2013) which has been associated with increased risk of atrophy post stroke (Dattola et al. 1993; Hu et al. 2007). Altered muscle activation previously observed for stroke survivors could have decreased phalanx force control (Chapter 2), and tactile sensory deficit could further alter muscle activation via reduced feedback, leading to increased phalanx force deviation. Furthermore, changes in skin slipperiness could account for increased phalanx force deviation (Cole 1991). However there was no evidence of difference in skin slipperiness between stroke survivors and healthy controls in the previous study (Chapter 2).

It is currently unknown how stroke-induced tactile sensory deficit (Carey 1995; Turton and Butler 2001) plays a role in altering phalanx force direction in *power grip*. The goal of this study was to determine the effects of tactile sensory deficit on stroke survivors' power grip force direction during power grip. Phalanx force direction for stroke survivors with tactile sensory deficit was compared to stroke survivors without tactile sensory deficit and age-matched neurologically-intact healthy controls. Furthermore, muscle activation pattern was examined to determine if stroke induced tactile sensory deficit is associated with an altered muscle activation pattern compared to stroke survivors with no tactile sensory deficit and healthy controls. COF between the

finger skin and the grip surfaces were also measured to determine any decrease in skin

slipperiness that might allow for greater phalanx force deviation between the subject

groups. Determining the role of impaired tactile sensory feedback on power grip is

important to inform stroke rehabilitation practices with the way somatosensation impacts

motor recovery post stroke and could be useful in the development of more effective or

alternative rehabilitation strategies for stroke recovery.

3.2. Methods

3.2.1 Subjects

Fourteen chronic stroke survivors with tactile sensory deficit (mean age $\pm$ SD =

59 $\pm$ 12 years), 9 chronic stroke survivors without tactile sensory deficit (63 $\pm$ 12 years),

and 18 age-matched healthy control subjects (61 $\pm$ 10 years) participated (Table III). Age

was not significantly different between any of the groups (T-Tests with $p>.05$). Mean

motor impairment was quantified by the Chedoke-McMaster Stroke Assessment Hand

Section (Gowland et al. 1995) and the hand and wrist subdivision of the Fugl-Meyer

Assessment (Fugl-Meyer et al. 1975). Mean Chedoke-McMaster score was 5 $\pm$ 2 for both

stroke groups (out of a possible 7, t-test with $p>.05$), and mean Fugl-Meyer score was 17

$\pm$ 7 and 20 $\pm$ 6 (out of a possible 24) for the stroke survivors with tactile sensory deficit

and stroke survivors without tactile sensory deficit groups, respectively (t-test with

$p>.05$). Stroke survivors' median Modified Ashworth Scale (MAS) (Ashworth 1964)

assessing spasticity of the flexor muscles of the forearm was 1 and 0 (out of a possible 4)

for the stroke survivors with and without tactile sensory deficit, respectively (ranging

from 0 to 3 for both groups, Mann-Whitney Test with *p*>.05). Overall, both stroke groups

were similar in terms of motor function. In addition, the FDS, EDC, and FDI muscle

activity was recorded for 13 stroke survivors with tactile sensory deficit, 7 stroke

survivors without tactile sensory deficit, and 13 healthy controls

Table III: Subject Demographics

Group	Subject	Time since most recent stroke (months)	Type of Stroke	Hand Dominance***		Paretic Side	Sex	Age (years)	Fugl-Meyer Score (out of 24)	Chedoke McMaster Score (out of 7)	Monofilament Score	
				Pre Stroke	Post Stroke						Index	Thumb
Stroke survivors with tactile sensory deficit	1	19	Ischemic	R	R	L	Male	79	-	2	3.61	3.61
	2*	16	Ischemic	R	R	R	Female	73	24	6	3.61	3.61
	3*	22	Ischemic	R	R	R	Male	50	21	5	3.61	3.61
	4*	102	Hemorrhagic	L	L	R	Female	66	9	2	3.61	3.61
	5*	82	Ischemic	R	L	R	Male	59	16	5	6.65	6.65
	6*	36	Unknown	R	L	R	Female	67	5	2	4.31	3.61
	7*	53	Ischemic	R	R	L	Male	54	10	2	6.65	6.65
	8*	7	Ischemic	R	R	R	Male	66	24	7	4.31	4.31
	9*	55	Hemorrhagic	L	R	L	Male	32	8	2	6.65	6.65
	10*	162	Hemorrhagic	R	R	L	Male	60	22	7	3.61	3.61
	11*	62	Unknown	R	R	R	Female	60	22	7	3.61	3.61
	12*	142	Ischemic	L	L	R	Female	53	24	7	3.61	3.61
	13*	8	Unknown	R	R	L	Male	65	24	7	3.61	3.61
	14*	44	Ischemic	R	R	L	Female	43	18	3	6.65	6.65
Stroke survivors without tactile sensory deficit	1*	8	Hemorrhagic	R	R	L	Female	49	24	7	2.83	2.83
	2	86	Ischemic	R	R	L	Male	79	-	3	2.83	2.83
	3*	45	Ischemic	R	R	L	Female	58	16	6	2.83	2.83
	4	46	Ischemic	R	R	R	Male	69	13	2	2.83	2.83
	5	80	Hemorrhagic	R	R	R	Male	63	24	7	2.83	2.83
	6*	245	Ischemic	L	L	R	Female	55	24	7	2.83	2.83
	7*	52	Ischemic	R	L	R	Male	81	9	2	3.61	2.83
	8*	172	Hemorrhagic	R	R	R	Female	50	24	7	2.83	2.83
	9*	42	Ischemic	R	R	R	Male	59	23	7	2.83	2.83
	10**	64	Ischemic	R	R	L	Male	62	24	7	3.61	2.83

*Indicates subjects whose EMG and COF measurement was also recorded
**Indicates subjects who only had EMG and COF measurement recorded with no grip force data obtained
**R= "Right hand dominance", L= "Right hand dominance"

All subjects underwent the Semmes-Weinstein monofilament test (Bell-Krotoski et al. 1993). Tactile sensory deficit was determined based on a score of ≥ 3.61 (Dellon 1997) for both the index and thumb finger. Monofilament scores for the stroke survivor group with tactile sensory deficit ranged from 3.61 to 6.65 with a median score of 3.61. All stroke survivors were at least 6 months post stroke. All age-matched controls were neurologically healthy and free from injury in the upper extremity. All subjects signed a consent form and followed a protocol approved by the Institutional Review Board.

3.2.2 Procedure

Subjects were instructed to sit in a chair with the elbow flexed at approximately 90° and the forearm horizontally rested on an arm rest. Subjects gripped a custom-made power grip dynamometer at the maximum and 50% maximum effort for five seconds, while individual phalanges' normal and proximal-distal shear force were recorded for a single finger (Enders and Seo 2011). Visual feedback was not provided to the subjects during gripping. Subjects were instructed to grip in a consistent manner regardless of the finger being measured. Stroke subjects' paretic hand and control subjects' non-dominant hand was used because this hand often acts as the "stabilizing hand" that people use to hold objects, while performing finer manipulation with the dominant hand and non-paretic hand. Two grip surfaces, a paper or a rubber surface, covered the entire surface of the grip device and finger pads. These two surfaces were used because a previous study showed that young healthy individuals adapted the direction of their phalanx forces

depending on the surface (Enders and Seo 2011). Measurement of phalanx normal and shear forces for the all five fingers, two frictional surfaces, and two effort levels (maximum and 50% of maximum power force) were repeated at least two times each, and the order of testing was randomized.

In addition, the FDS, EDC, and FDI muscle activity was recorded during power grip using surface electromyogram (EMG) (Bortec Biomedical Ltd., Calgary, AB, Canada). Surface electrodes were placed on top of the muscle bellies according to Basmajian (1989) after the skin had been cleaned with alcohol swabs. Maximum voluntary contractions (MVC) for each muscle, while contracting against resistance, were also recorded. Both the force and EMG data were recorded at 1000 Hz.

Furthermore, the COFs of the finger skin and the paper and rubber surfaces were determined using a series of finger drags for same set of subjects who had EMG recorded (Table III). Subjects' fingers were pressed against a load cell by the investigator at a normal force level of approximately 2 N. Then shear force was gradually applied until the finger slipped against the surface. The shear to normal force ratio at the point of slip was defined as the COF for that surface. COF was measured twice for each subject and each surface. The COF data for the paper surface was previously reported for the average of the two stroke groups and is investigated here for the two separate stroke groups along with the COF for the rubber surface.

3.2.3 Data Analysis

Mean phalanx normal and shear forces for each grip trial were calculated from the two-second period in which the total force (the sum of all normal and shear forces for all phalanges) was the greatest. The phalanx force deviation from the direction normal to the grip surface was quantified as the arctangent of the absolute ratio of shear force to normal force. The absolute phalanx force deviation was used because the frequency of phalanx force being distally directed was similar for all of the groups (64% for healthy individuals, 67% for stroke survivors without tactile sensory deficit, and 57% for stroke survivors with tactile sensory deficit). For the analysis of the EMG data, the root mean square (RMS) with a 20-ms moving window was applied. The RMS EMG was normalized to the MVC level for each muscle calculated as the peak RMS EMG recorded during the MVC trials. The mean EMG in %MVC during the same two-second period from which the force data was selected were used for further analysis. Muscle activation pattern between the subject groups was assessed by examining the raw %MVC data and the relative FDI and EDC muscle activities in relation to the FDS muscle activity (calculated as the ratio of FDI to FDS EMG and that of EDC to FDS EMG in %MVC).

A mixed-design ANOVA was conducted to examine how the phalanx force deviation varied for the three subject groups (stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and healthy controls), surface, effort level, finger, phalanx, and the interactions between subject group and surface, subject group and effort, subject group and finger, and subject group and phalanx. To assess

difference in power grip force level among the three subject groups, secondary mixed-design ANOVA was conducted for the phalanx normal force with the same model. To determine if proprioceptive sensory feedback was different between the subject groups, three separate one-sample t-tests were used to determine if the approximated 50% maximum phalanx resultant force normalized to the phalanx resultant force produced during 100% maximum power grip was different from 50% for each subject group. Additionally, two separate mixed design ANOVAs were used to analyze how muscle activity changed for the FDS, FDI, and EDC muscles depending on the subject groups. One determined if EMG (%MVC) activity was significantly different subject group, effort level, muscle, and interactions between the subject group and the effort level and between the subject group and the muscle. Another ANOVA was used to examine if the muscle activity pattern, the relative FDI and EDC EMGs (normalized to FDS EMG), varied significantly for the subject group, effort level, muscle, and interactions between the subject group and the effort level and between the subject group and the muscle. Finally, a fifth mixed-design ANOVA was conducted to examine differences in COF of the finger skin for the three subject groups, surface, and the interaction between subject group and surface. Phalanx force deviation data, phalanx normal force data, and EMG data sets were skewed based on the Test for Skewness (Tabachnick and Fidell 2007). Therefore, a square root transformation was applied to normalize the phalanx force deviation data and the phalanx normal force data and a log transformation was applied to the muscle activation pattern data. Transformed data were used in the ANOVAs. Tukey post hoc tests determined differences among the three subject groups.

3.3. Results

An overview of the results is as follows. The average phalanx force magnitude and direction during static power grip for the two stroke groups and healthy controls are shown in Figure 14. Consistent with the previous literature (Boissy et al. 1999), stroke survivors' mean phalanx normal force was approximately half of that for controls. The stroke survivors with and without tactile sensory deficit had similar *maximum* phalanx normal forces that were not significantly different from each other. Consistent with the previous study (Chapter 2), the stroke groups gripped with greater phalanx force deviation compared to healthy controls. The new finding of this study is that stroke survivors with tactile sensory deficit produced power grip with significantly greater phalanx force deviation compared to stroke survivors without tactile sensory deficit and healthy controls. Increased phalanx force deviation for stroke survivors with tactile sensory deficit was associated with an altered muscle activation pattern, but not a change in skin slipperiness or force estimation accuracy. The details of these findings are presented below.
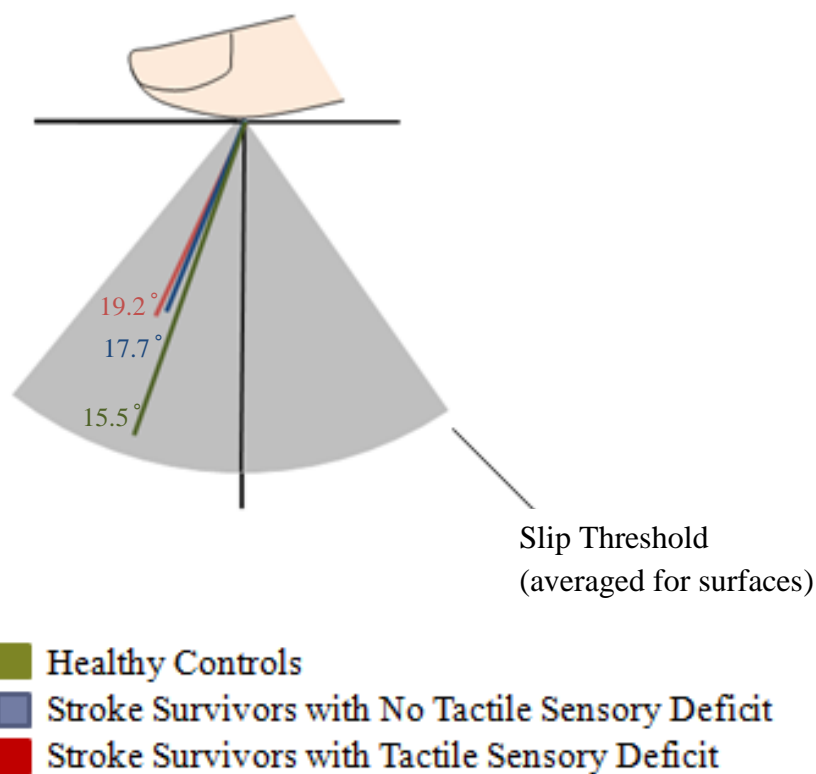
Figure 14: Mean phalanx force deviation, was the greatest for stroke survivors with tactile sensory deficit followed by stroke survivors without tactile sensory deficit and healthy controls. Averaged phalanx force deviation across the effort levels, surfaces, fingers, and phalanges is shown. The averaged phalanx force deviation across the effort levels, surfaces, fingers, and phalanges is shown. The slip threshold is the averaged threshold between the two surfaces.

3.3.1 Increased phalanx force deviation for stroke survivors with tactile sensory deficit compared to stroke survivors without tactile sensory deficit and healthy controls

Phalanx force deviation was significantly dependent upon group, finger, phalanx, surface, and the interactions between group and effort, group and phalanx, and group and finger (Figure 15 and Appendix C, ANOVA with $p < .05$). Stroke survivors with tactile sensory deficit produced the largest phalanx force deviations, followed in order by stroke

survivors without tactile sensory deficit and healthy individuals (Figure 15a and

Appendix C). Compared to healthy controls, phalanx force deviation was significantly

increased by 14% for stroke survivors without tactile sensory deficit and 24% for stroke

survivors with tactile sensory deficit (Tukey-post hoc, p<.05). This trend of stroke

survivors with tactile sensory deficit producing the greatest phalanx force deviation,

followed in order by stroke survivors without tactile sensory deficit and healthy controls,

was observed for all surfaces, efforts, phalanges, and fingers (Figure 15b-d and Appendix

C). Stroke survivors with tactile sensory deficit produced significantly greater phalanx

force deviation compared to healthy controls and stroke survivors without tactile sensory

deficit during 50% maximum power effort and significantly greater phalanx force

deviation compared to healthy controls at maximum power effort (Figure 15c and

Appendix C, group and effort level interaction, Tukey post hoc with $p<.05$). Stroke

survivors with tactile sensory deficit produced significantly greater phalanx force

deviation with all the phalanges compared to healthy controls and for the distal phalanx

compared to stroke survivors without tactile sensory tactile deficit (Figure 15d and

Appendix C, group and phalanx interaction with Tukey post-hoc p<.05). Stroke survivors

with tactile sensory deficit produced significantly greater phalanx force deviation with

the thumb and index finger compared to healthy controls and for the thumb compared to

stroke survivors without tactile sensory deficit (Figure 15e and Appendix C, group and

finger interaction with Tukey post-hoc p<.05). Consistent with the previous study for

healthy young adults (Enders and Seo 2011) phalanx force deviation was greater with the

rubber surface compared to the paper surface (Figure 15b and Appendix C, ANOVA

surface main effect with p<.05 without a significant subject group and surface

interaction), although it was observed that stroke survivors with tactile sensory deficit changed their phalanx force deviation very little (<1%) compared to the stroke survivors without tactile sensory deficit (7%) and healthy controls (13%).
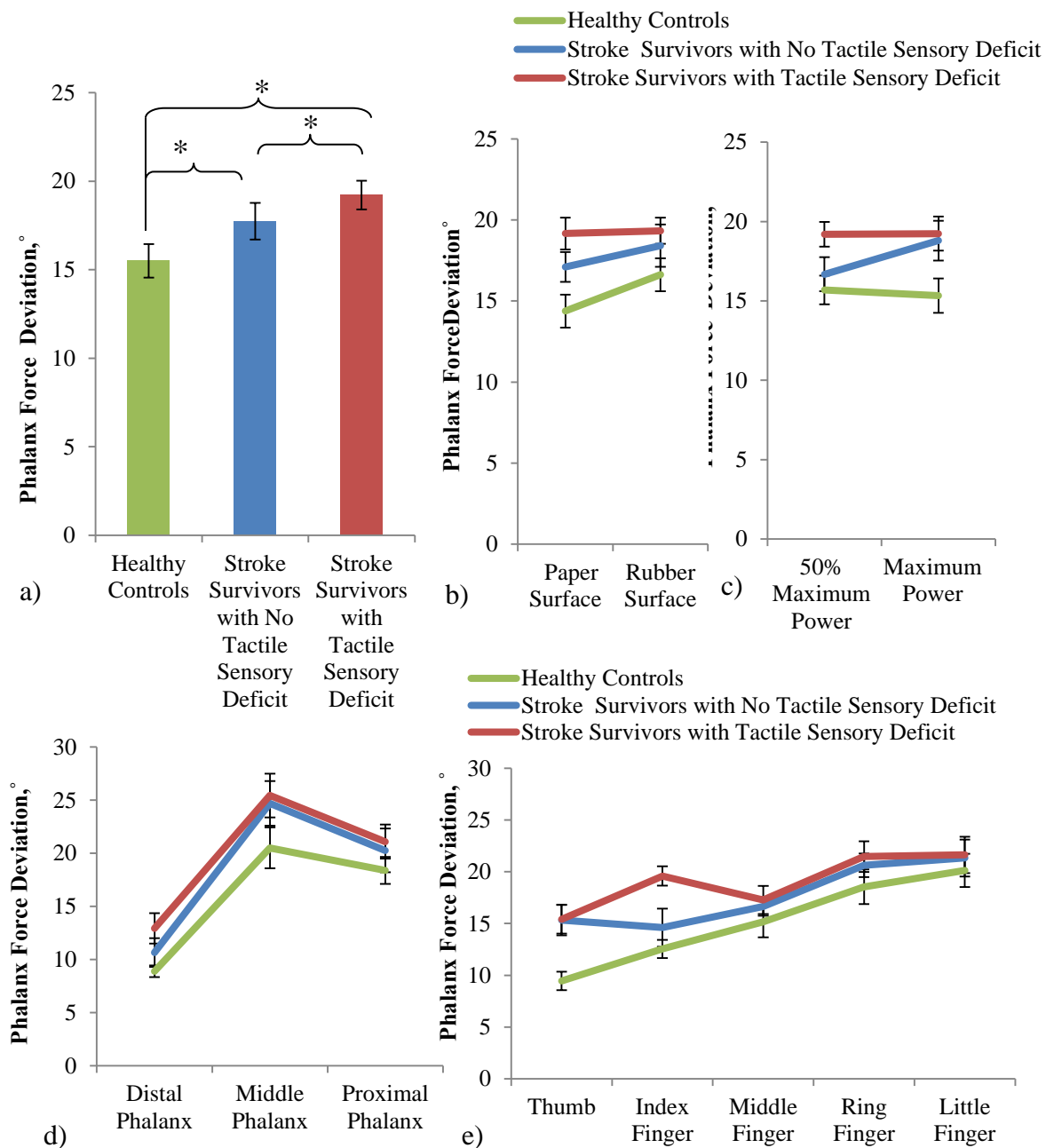
Figure 15: Mean ± SE phalanx force deviation was significantly greatest for stroke survivors with tactile sensory deficit, followed in order by stroke survivors without tactile sensory deficit and healthy controls (ANOVA, subject group main effect with $p<.05$) This trend was observed for all surfaces (b), effort levels (c), phalanges (d), and fingers (e).

3.3.2 Phalanx normal force

Phalanx normal force was similarly reduced for both stroke groups. Phalanx normal force was significantly dependent upon subject group, effort level, finger, phalanx, and the interaction of group and effort, group and phalanx, and group and finger (Figure 16 and Appendix C, ANOVA with $p<.05$). Both stroke survivors with and without tactile sensory deficit performed power grip with reduced phalanx normal force during both effort levels compared to healthy controls (Figure 16c and Appendix C, group and effort level interaction Tukey post-hoc with $p<.05$), while the two stroke groups were not significantly different from one another during either effort level (Figure 16c and Appendix C, group and effort level interaction Tukey post-hoc with $p>.05$). Although their force level was reduced compared to healthy controls, both stroke survivor groups were able estimate 50% of their maximum power grip force well, without a significant difference from 50% (Figure 17 and Appendix C, t-tests p>.05 for all three groups). Stroke survivors with tactile sensory deficit produced significantly less phalanx normal force during power grip with all the phalanges compared to healthy controls and significantly less normal force with the distal phalanx compared to stroke survivors without tactile sensory deficit (Figure 16d and Appendix C, ANOVA, group and phalanx interaction, Tukey post-hoc with $p<.05$). Both stroke groups produced significantly less phalanx normal force from all the fingers compared to healthy controls and were not significantly different from one another for any finger (Figure 16e and Appendix C, ANOVA, group and phalanx interaction, Tukey post-hoc with $p<.05$).
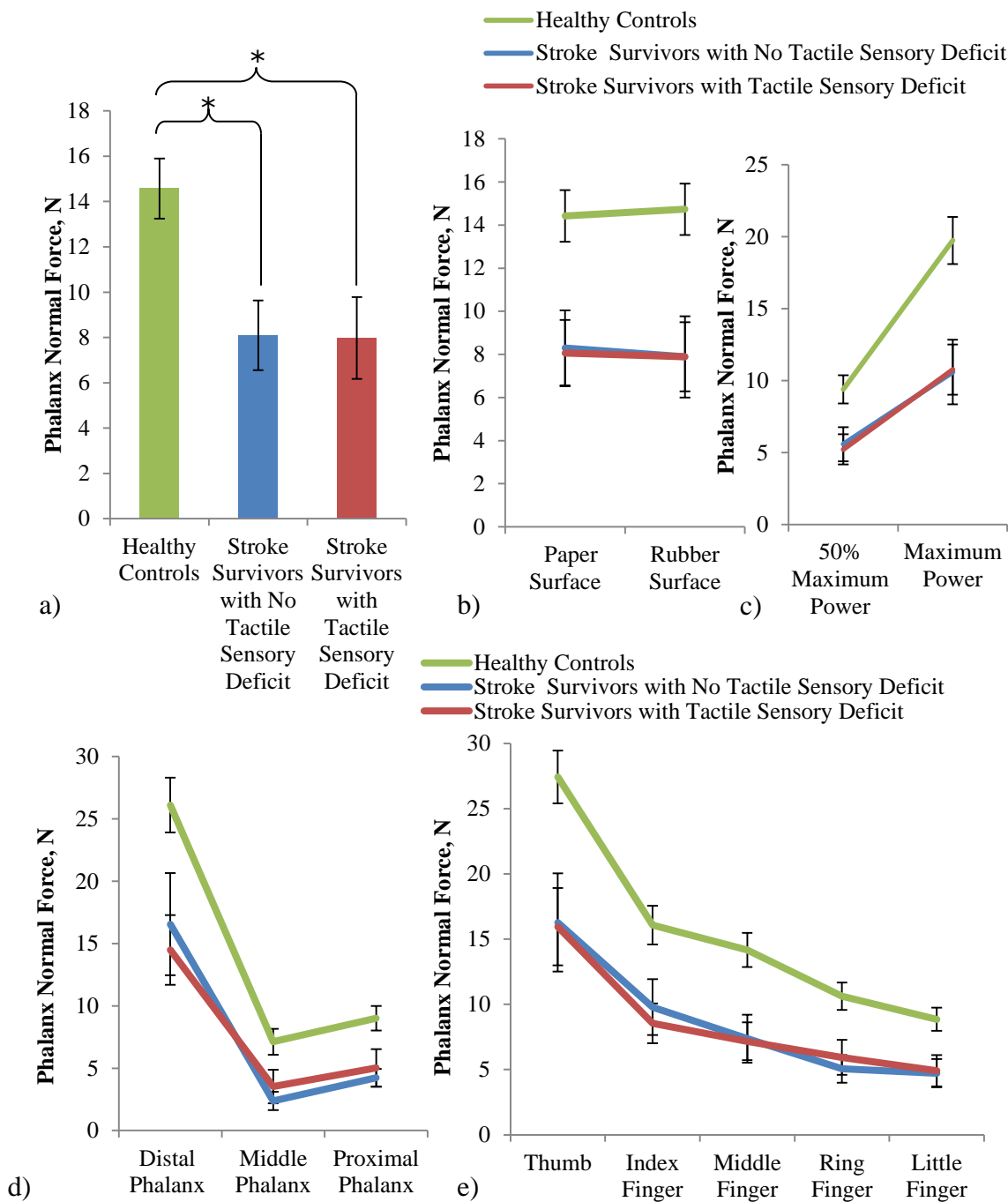
Figure 16: Mean ± SE phalanx normal force was significantly reduced for both stroke survivor groups compared to healthy controls (ANOVA, subject group main effect with $p < .05$). This reduction was similar for both stroke survivor groups and was observed for all surfaces (b), effort levels (c), phalanx (d), or finger (e).
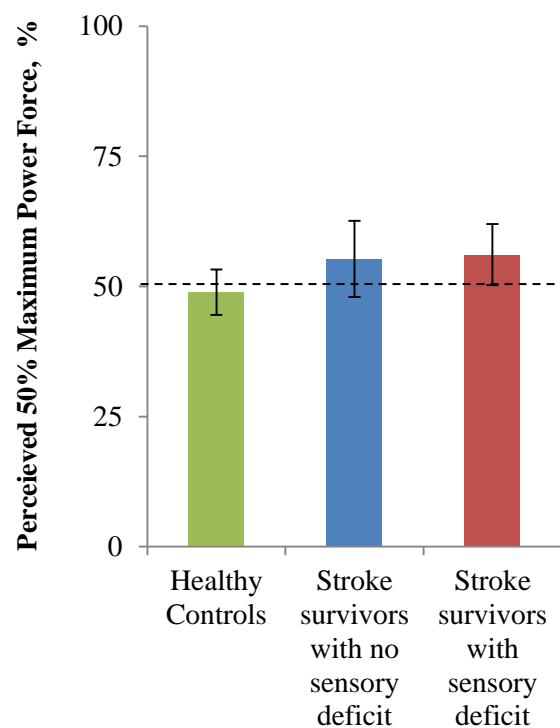
Figure 17: Mean ± SE grip force produced during grip at 50% of the maximum perceived effort normalized to the grip force produced during maximal grip was not significantly different from the target of 50% for all subject groups.

3.3.3 Altered muscle activity pattern for stroke survivors with tactile sensory deficit

Each muscle's activity during static power grip is shown for the three subject groups in Figure 18a (and Appendix C). The EMG (%MVC) was significantly dependent upon subject group, effort level, muscle, and the interaction between subject group and effort level and the interaction between subject group and muscle ($p<.05$). While the overall muscle activity was highest for healthy controls, followed by stroke survivors with tactile sensory deficit and those without, Tukey post hoc results show that the FDI

and EDC muscle activities were significantly reduced for both stroke groups compared to controls (Tukey post hoc, subject group and muscle interaction with $p<.05$), while the FDS muscle activity was not significantly different between any of the subject groups (Tukey post hoc, subject group and muscle interaction with $p>.05$).

This difference in muscle activity pattern for the three subject groups is highlighted in the relative FDI and EDC activity normalized to FDS EMG (Figure 18b and Appendix C). Muscle activation pattern was significantly dependent upon subject group, muscle, and the interactions between group and muscle, and group and effort (ANOVA with $p<.05$). Muscle activation pattern was significantly altered for stroke survivors with tactile sensory deficit compared to stroke survivors without tactile sensory deficit and healthy controls (Figure 18b and Appendix C, ANOVA subject group main effect, Tukey post-hoc with $p<.05$). Specifically, the stroke survivors with tactile sensory deficit have significantly reduced FDI and EDC muscle activities relative to the FDS muscle, compared to both stroke survivors without tactile sensory deficit and healthy controls (Figure 18b and Appendix C, ANOVA, subject group and muscle interaction and Tukey post hoc with $p<.05$). Such altered muscle activation pattern was found for both the 50% maximum and maximum power grip effort levels (ANOVA, subject group and effort level interaction, Tukey post-hoc with $p<.05$). Although the overall muscle activity in %MVC was lower than healthy controls (Figure 18a), stroke survivors without tactile sensory deficit were not significantly different in muscle activation pattern compared to healthy controls (Figure 18b and Appendix C, ANOVA, subject group and muscle

interaction, Tukey post-hoc with $p > .05$), for either effort level (ANOVA, subject group

and effort level interaction, Tukey post-hoc with $p > .05$).
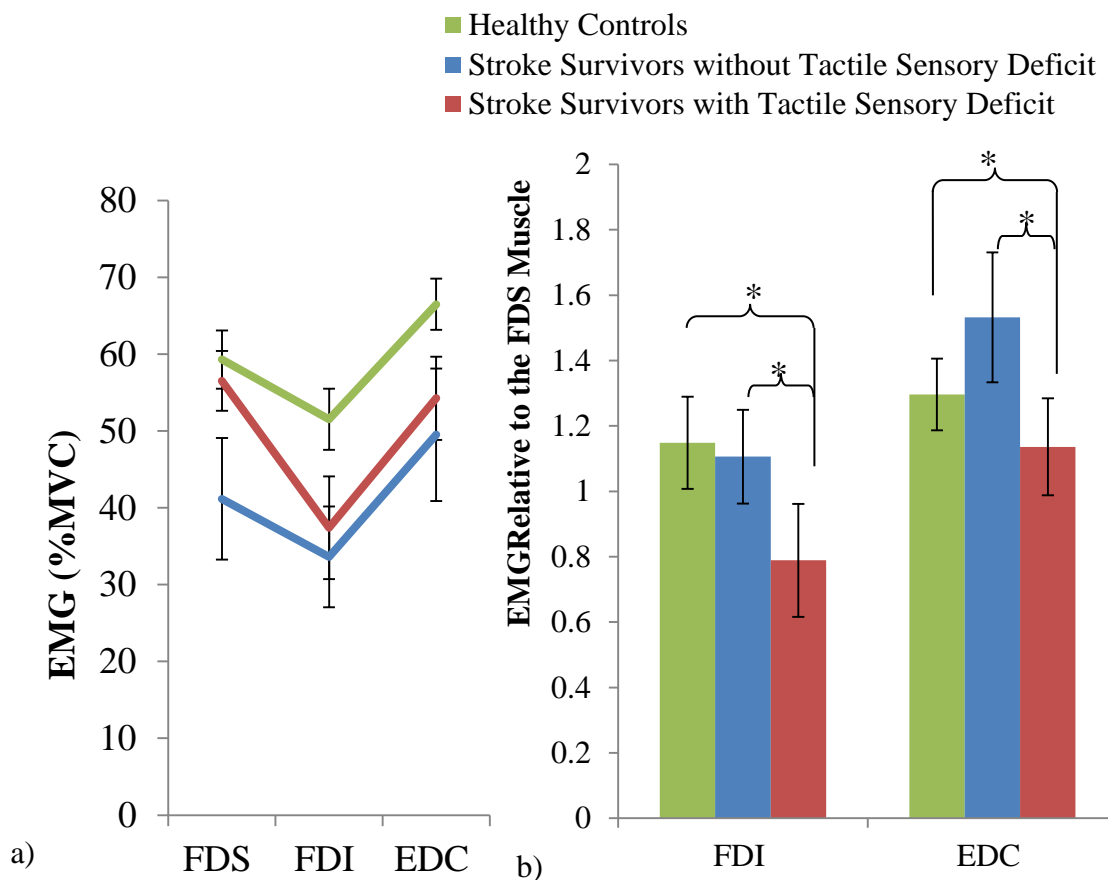
Figure 18: Mean ± SE EMG was reduced for stroke survivors compared to healthy controls for both stroke survivor groups (a). Mean ± SE FDI and EDC EMGs relative to the FDS EMG were significantly reduced for stroke survivors with tactile sensory deficit compared to controls and stroke survivors without tactile sensory deficit (significant subject group main effect with p<.05, significant difference for stroke survivors with tactile sensory deficit group compared to other two groups with Tukey posthoc p<.05 for both relative FDI and EDC EMGs) (b), showing altered muscle activity pattern with particularly reduced intrinsic FDI and extrinsic EDC muscle activities for stroke survivors with tactile sensory deficit compared to controls and stroke survivors without tactile sensory deficit. Non-transformed data is shown in the figure.

3.3.4 Skin COF

There was no signifiant difference in the COF between any of the subject groups for either surface (Figure 19 and Appendix C, ANOVA, group main effect and group and surface interaction with *p*>.05, and only surface having p<.05). The mean COF of the paper surface was 0.43, similar to previous papers (Buchholz et al. 1988; Gee et al. 2005). The mean COF of the rubber surface was 1.00, similar to a previous study that found a COF for the finger skin and rubber of 0.9 (Seo and Armstrong 2009). These COF values indicate that the threshold for slippage (calculated as the arctangent of the COF) was, on average, 23˚ and 45˚ for the paper and rubbers surfaces, respectively.
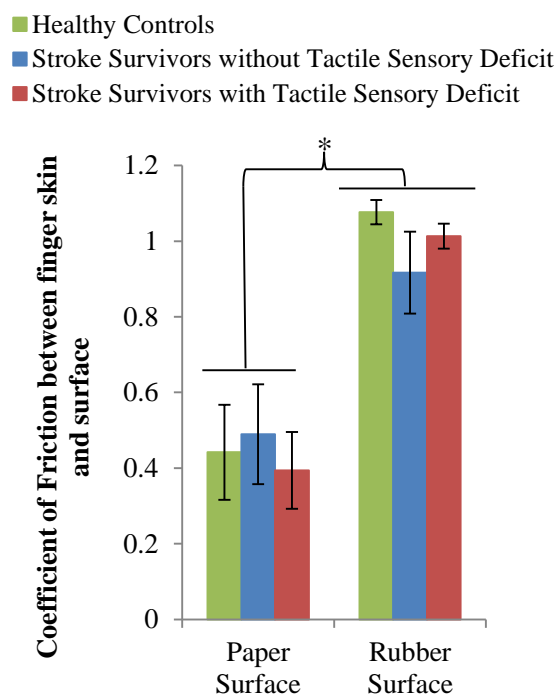
Figure 19: Mean ± SE COF between the finger skin and the paper and rubber surfaces was similar for stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and healthy controls (ANOVA, group main effect and group and surface interaction p>.05). The COF for the rubber surfaces was significantly greater than

the paper surface (ANOVA, surface main effect with p<.05). Non-transformed data is shown in the figure.

3.4. Discussion

Consistent with previous studies, stroke survivors gripped with greater phalanx force deviation compared to healthy controls during static power grip. The new finding of this study is that stroke survivors with tactile sensory deficit gripped with greater deviation compared to stroke survivors without tactile sensory deficit. This greater phalanx force deviation for the stroke survivors with tactile sensory deficit was accompanied by an altered muscle activation pattern, characterized by the underactivated FDI and EDC muscles relative to the FDS muscle, compared to both the stroke survivors without tactile sensory deficit and the healthy controls. Stroke survivors without tactile sensory deficit were not significantly different in muscle activation pattern compared to healthy controls. Differences in the two stroke survivors groups were not the result of differences in motor impairment level, grip strength, skin slipperiness, or proprioceptive sensory feedback.

3.4.1 Phalanx force direction altered more for stroke survivors with tactile sensory deficit

Stroke survivors with tactile sensation deficit performed a static power grip with phalanx force deviated from the normal direction to a greater extent than stroke survivors

with no tactile sensation deficit, compared to healthy controls. The effect of tactile

sensory loss on power grip force control investigated in the present study is in line with

other studies showing that reduced sensation may affect pinch grip force control

(Blennerhassett et al. 2007; Hermsdorfer et al. 2003; Robertson and Jones 1994). Both

stroke survivor groups had similar functional motor scores (described in the subject

section) and exhibited similar reduction in phalanx normal force during power grip for

both effort levels, thus indicating that the group differences in grip control could be

related to reduced sensory feedback (Figure 16c and Appendix C). Also, the difference in

gripping control was most likely not the result of differences in skin slipperiness (Figure

19 and Appendix C), because the threshold for slippage (the maximum phalanx force

deviation allowed before slippage, calculated as arctangent of the COF) was similar for

all subject groups. Therefore, it appears that stroke induced sensory deficit, in addition to

motor deficit, was associated with a loss of phalanx force control. Specifically, those

stroke survivors with *tactile* sensory deficit appeared to have decreased phalanx force

control, since *proprioceptive* sensory feedback (important for force scaling and force

estimation (Levin et al. 1995; Ostry and Feldman 2003)) appears to be similar for all

groups as shown by the similar grip force estimation accuracy for all groups (Figure 17

and Appendix C).

An altered muscle activation pattern was also observed for stroke survivors with

stroke induced tactile deficit in the present study, suggesting that diminished sensory

feedback was associated with greater alteration in muscle activity controlling the fingers

during power grip. When muscle activation pattern was examined by quantifying the

relative muscle activity to the major gripping muscle of the FDS, both the intrinsic FDI and the extrinsic extensor EDC muscles were under-activated compared to the extrinsic flexor FDS muscle for stroke survivors with tactile sensory deficit compared to the other stroke group and healthy controls (Figure 18b and Appendix C). This reduction in the relative muscle activity for the stroke survivors with tactile sensory deficit was somewhat greater for the intrinsic FDI muscle than the EDC muscle (Figure 16b and Appendix C). No statistical difference in the muscle activation pattern was observed between the stroke survivors without tactile sensory deficit and healthy controls. Weakening of any muscle controlling the fingers can hamper finger force production in a specific direction (Kutch and Valero-Cuevas 2011). The intrinsic muscles are important for directional force control and weakening of the intrinsic muscles has previously been shown to lead to increased fingertip force deviation in biomechanical models (Valero-Cuevas et al. 2000) and has been speculated to have reduced force control for older adults (Cole 2006). Therefore the altered phalanx force deviation seen for stroke survivors with tactile sensory deficit could be attributable to the observed altered muscle activation pattern with more weakened intrinsic and extensor muscle activities relative to the long finger flexor muscle activity after stroke. While the EDC and FDI muscle activity was reduced for stroke survivors with tactile sensory deficit, the FDS muscle activity appeared to be preserved (Figure 18a and Appendix C) potentially due to the disinhibited reticulospinal tract (Zaaimi et al. 2012). Different muscle activation pattern despite similar Fugl-Meyer Assessment and Chedoke-McMaster Stroke Assessment Hand Section scores, could be because these motor functional scores were not sensitive enough to detect the differences in muscle activation pattern between the two stroke survivor groups.

Tactile sensory deficit could be linked to the decreased phalanx force direction and altered muscle activation pattern in two ways. First, decreased tactile sensory feedback could have reduced stroke survivors' ability to detect how they are directing their phalanx forces and hampered the closed-loop motor control, resulting in non-corrected phalanx force deviation and altered muscle activation pattern during power grip. This finding complements previous studies in which reduced tactile sensory feedback was associated with impaired closed-loop pinch grip control based on grip surface characteristics (Johansson and Westling 1987), the magnitude and direction of the phalanx force being produced (Augurelle et al. 2003; Blennerhassett et al. 2007; Cole 2006; Hermsdorfer et al. 2003; Monzée et al. 2001; Robertson and Jones 1994), and the finger position with respect to the object surface (Monzée et al. 2001). In addition, our previous study showed that healthy individuals change their phalanx force deviation with a change in surface COF (Enders and Seo 2011), because a higher COF surface allows for greater phalanx force deviations, reducing the amount of muscle coordination needed to direct the phalanx forces in a precise direction (Cole 2006; Milner and Dhaliwal 2002; Valero-Cuevas et al. 2000). Stroke survivors with tactile sensory deficit tended to not change their phalanx force deviation between the two surfaces compared to stroke survivors without tactile sensory deficit and healthy controls (Figure 15b and Appendix C, group and surface interaction with $p>.05$). Therefore, it is possible that stroke survivors with tactile sensory deficit were not able to detect the surface change and adapt their phalanx force deviation during the static power grip task.

Second, previous research has shown how sensory and motor cortical territories shift following the removal of sensory inputs, suggesting that sensory feedback assists in the preservation of the normal cortical representations of both the motor and sensory cortex (Weiss et al. 2004). Therefore, those stroke survivors with reduced tactile sensation could have altered cortical sensorimotor representations, leading to an altered muscle activity pattern and diminished phalanx force control. Furthermore, the distal intrinsic muscles may require more corticospinal drive than more proximal muscles (Palmer and Ashby 1992; Turton and Lemon 1999), leaving them more vulnerable to the reorganizational shifts in the sensorimotor cortical representations and making them especially affected in stroke survivors with tactile deficit (Figure 18b and Appendix C). In summary, a loss of tactile sensory feedback following stroke could have led to altered coordination of muscle activation, resulting in increased phalanx force deviation, due to reduced feedback control and cortical shifts that occurred after stroke. As a future study, making a comparison on the power grip of healthy individuals with and without digital anesthesia simulating tactile sensory deficit could provide insight into the direct interaction of tactile sensation and power grip control.

3.4.2 Functional Implications of Reduced Phalanx Force Control and Clinical Implications

Increased phalanx force deviation can lead to object dropping. Finger force deviating from the direction normal to the surface can lead to increased chance of object

slippage (MacKenzie and Iberall 1994; Seo et al. 2010). Therefore, stroke survivors with tactile sensory deficit may be more at risk than stroke survivors without tactile sensory deficit for dropping objects. This finding for power grip is comparable to the previous finding for pinch grip in which reduced tactile sensation increased frequencies of dropping objects (Augurelle et al. 2003). Furthermore, increased phalanx force deviation can also lead to increased risk of dropping objects by reducing the phalanx normal force. Increased phalanx force deviation tilts the force vector such that the normal force is reduced and force in the shear direction increases. Specifically, in the present study, the phalanx force deviation reduced potential normal force by 29% for stroke survivors with tactile sensory deficit (calculated by taking the average difference between phalanx resultant force and normal force).

Tactile sensory deficits have been shown to negatively impact functional recovery of the upper limb following stroke (Meyer et al. 2014). The results of this study demonstrate the importance of integrating tactile sensory retraining for stroke survivors in addition to motor re-training post stroke. Methods exist to improve hand tactile sensation, such as the transcutaneous electrical stimulation (Conforto et al. 2007), temporary functional de-afferentation (Sens et al. 2012b), and vibrotactile noise (Collins et al. 1996; Enders et al. 2013; Kurita et al. 2013). Combining these tactile sensory enhancement techniques with a sensory re-training paradigm such as that described in (Carey and Matyas 2005; Chanubol et al. 2012) or conventional motor rehabilitation(Wolf et al. 2006) could be beneficial to stroke patients in functional recovery. Alternatively, bypassing stroke survivors' tactile sensory impairment and

increasing functional performance by training to improve force control with visual feedback may have potential to lead to recovery of motor control (Ellis et al. 2005; Seo et al. 2011).

3.5. Conclusion

This study indicates that tactile sensory feedback could be important for control of phalanx force direction and reduction of tactile sensory feedback due to stroke could reduce control of phalanx forces during static power grip. Stroke survivors with tactile sensory deficit exhibited similar motor impairment level, similar reduction in grip strength, and similar skin friction as other stroke survivors without tactile sensory deficit, but had greater phalanx force deviation. Stroke induced tactile sensory deficit was associated with increased phalanx force deviation and an altered muscle activation pattern. These results suggest that not only motor deficit, but also tactile sensory deficit following stroke could be responsible for impaired hand grip for stroke survivors. Stroke survivors with tactile sensory deficit may have an increased incidence of dropping grasped objects compared to those without tactile sensory deficit and healthy controls due to greater phalanx force deviations, and may require additional attention for sensory deficit during their rehabilitation.

Chapter 4: Remote vibrotactile noise improves light touch sensation in stroke survivors' fingertips via stochastic resonance[2]

ABSTRACT

Stroke rehabilitation does not often integrate both sensory and motor recovery. While subthreshold noise was shown to enhance sensory signal detection at the site of noise application, having a noise-generating device at the fingertip to enhance fingertip sensation and potentially enhance dexterity for stroke survivors is impractical, since the device would interfere with object manipulation. This study determined if remote application of subthreshold vibrotactile noise (away from the fingertips) improves fingertip tactile sensation with potential to enhance dexterity for stroke survivors. Index finger and thumb pad sensation was measured for ten stroke survivors with fingertip sensory deficit using the Semmes-Weinstein Monofilament and Two-Point Discrimination Tests. Sensation scores were measured with noise applied at one of three intensities (40%, 60%, 80% of the sensory threshold) to one of four locations of the paretic upper extremity (dorsal hand proximal to the index finger knuckle, dorsal hand proximal to the thumb knuckle, dorsal wrist, volar wrist) in a random order, as well as without noise at beginning (Pre) and end (Post) of the testing session. Vibrotactile noise of all intensities and locations instantaneously and significantly improved Monofilament scores of the index fingertip and thumb tip ($p<.01$). No significant effect of the noise was

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[2] Information presented in this chapter is published as follows and is used with permission from:

seen for the Two-Point Discrimination Test scores. Remote application of subthreshold (imperceptible) vibrotactile noise at the wrist and dorsal hand instantaneously improved stroke survivors' light touch sensation, independent of noise location and intensity. Vibrotactile noise at the wrist and dorsal hand may have enhanced the fingertips' light touch sensation via stochastic resonance and interneuronal connections. While long-term benefits of noise in stroke patients warrants further investigation, this result demonstrates potential that a wearable device applying vibrotactile noise at the wrist could enhance sensation and grip ability without interfering with object manipulation in everyday tasks.

4.1 Introduction

Many of 7 million stroke survivors in the U.S. (Roger et al. 2012) experience not only motor deficit (Gray et al. 1990; Nakayama et al. 1994; Parker et al. 1986) but also sensory deficits (Carey 1995) especially in the hand. Carey and Matyas found that discriminatory sensory loss was observed in almost 50% (24 of 51 subjects) in chronic stroke survivors, compared to almost 85% (57 of 67 subjects) of acute stroke survivors(Kim and Choi-Kwon 1996). Turton and Butler (2001) found in a case study that a stroke survivor had a decreased ability to correctly identify the time and locations of stimuli applied to both the palm and digits of the affected hand (Turton and Butler 2001). When the stroke subject was asked to correctly identify where and when a touch stimulus was applied on their hand, the subject only responded to the tests correctly about 65% of the time (Turton and Butler 2001).

While tactile sensation is critical for hand function, current stroke rehabilitation practices predominantly focus on motor re-training with limited emphasis on sensory re-training and sensorimotor integration. Cutaneous sensory feedback is essential for dexterity, fine finger movements, grip stability, and the setting and maintenance of force production during grip and object manipulation (Augurelle et al. 2003; Monzée et al. 2001). For instance, tactile sensory feedback from receptors in the fingertips is used for motor adaptation to surface characteristics (Johansson and Westling 1984) and dexterous hand movement (Zatsiorsky and Latash 2004b). Tactile sensory deficit experienced by

stroke survivors can lead to inappropriate grip force regulation and inefficient safety margins (Blennerhassett et al. 2006). The reduced sensory feedback experienced in stroke survivors may deteriorate feedback control of finger forces leading to unstable grip and object slipping against the finger, thereby hampering their hand grip function. Therefore, it is necessary to improve tactile sensation for stroke survivors, which may facilitate rehabilitation to improve dexterity, finger force control, and thus, hand function.

Previous research has aimed at increasing tactile sensation through a range of modalities. Anesthetic cream to the forearm has been shown to increase fingertip tactile sensation for healthy individuals (Bjorkman et al. 2004) and stroke survivors (Sens et al. 2012a) by inducing short-term changes in cortical representations (Sens et al. 2012a). Intense sensory retraining for chronic stroke survivors through repetitive sensory exercises (i.e. shape and texture discrimination) over a several weeks time period has also been shown some potential to increase tactile sensation (Byl et al. 2003; Carey and Matyas 2005; Yekutiel and Guttman 1993).

Stochastic resonance is a phenomenon in which addition of noise (e.g., vibrotactile noise) to a weak signal maximizes the detection and transmission of the weak signal (Galica et al. 2009; Moss et al. 2004; Priplata et al. 2002). Collins et al. (1997) found that healthy individuals' tactile sensation can be improved with certain levels of subthreshold vibrotactile noise (below the level at which a person can perceive the vibration), while it can be degraded if noise is too high (i.e., suprathreshold) "masking"

the original signal. Therefore, intensity of noise should be high enough for the signal to cross the threshold but low enough not to swamp the signal and decrease the signal to noise ratio (Collins et al. 1997; Moss et al. 2004; Wells et al. 2005). Previous work has shown optimum vibrotactile noise intensity as low as 50% of the sensory threshold for sensing a vibration at the fingertips (Kurita et al. 2011; Wells et al. 2005), while others have shown as high as 90% of the sensory threshold to be effective (Galica et al. 2009; Liu et al. 2002; Priplata et al. 2002). No consensus has been reached regarding the optimum vibrotactile noise intensity, especially for stroke survivors.

In light of the accumulating evidence for stochastic resonance, a wearable device applying vibrotactile noise to the fingertip has been developed by Kurita et al. (2011). While the device improves tactile sensation at the fingertip pad, a noise-generating device placed at the lateral aspect of the fingertip adversely interferes with object manipulation and dexterous finger movement by blocking physical contact between the finger and object, thus defeating the purpose of somatosensory enhancement. Furthermore, donning and doffing an assistive glove is difficult for stroke survivors, especially those with spasticity (Bhakta et al. 1996; Nathan et al. 2009). Thus, the desirable design would involve remote application of the vibrotactile noise to a location on the back of the hand or wrist that can still enhance tactile sensation. However, it is unknown if remote vibrotactile noise (i.e., away from the fingertip) could influence tactile sensation of the fingertip. In this study, we investigated how vibrotactile noise applied to various noise locations proximal to the fingertips could influence tactile sensation of the fingertip for stroke survivors.

The main objective of this study was to determine the effect of remote subthreshold vibrotactile noise on the tactile sensation of the index and thumb fingertips in stroke survivors. To achieve this objective, subthreshold vibrotactile noise was applied to one of four locations on the paretic upper limb (dorsal hand proximal to the index finger knuckle, dorsal hand proximal to the thumb knuckle, dorsal wrist, or volar wrist) at one of three noise intensities (40%, 60%, or 80% of the sensory threshold). It was hypothesized that remote subthreshold vibrotactile noise improves light touch sensation and spatial discrimination at the index and thumb fingertip pads in stroke survivors.

4.2 Methods

4.2.1 Subjects

Ten chronic stroke survivors (mean age $\pm$ SD = 60 ± 9 years) with sensory deficit participated in this study (Table IV). Hand motor function, evaluated using the hand and wrist subdivision of the Fugl-Meyer Assessment (Fugl-Meyer et al. 1975) (Table IV), was 19 ± 5 (out of a possible 24). All stroke survivors were at least 6 months post stroke. Subjects with history of upper extremity orthopedic conditions were excluded from this study. Subjects' tactile sensory deficit was recorded with the Semmes-Weinstein Monofilaments (Bell-Krotoski et al. 1993) and the Two-Point Discrimination Tests (Bell-Krotoski et al. 1993) for the index finger and thumb. Sensory deficit was defined as abnormal scoring for either of the sensory tests for either the index finger or the thumb.

All subjects signed a consent form and followed a protocol approved by the Institutional

Review Board.

Table IV: Subject demographics

Subject	Gender	Age	Fugl-Meyer	Monofilament (mm)		Two Point Discrimination (mm)		Vibrotactile sensory threshold (A peak to peak)			
				Index	Thumb	Index	Thumb	Dorsal hand proximal to index knuckle	Dorsal hand proximal to thumb knuckle	Volar wrist	Dorsal wrist
1	F	67	9	3.61	3.61	5	6	0.19	0.07	0.17	0.20
2	F	75	24	3.61	3.61	5	6	0.75	0.05	0.14	0.17
3	M	71	13	3.61	3.61	6	8	0.20	0.12	0.20	0.20
4	M	57	23	3.61	3.61	4	3	0.21	0.13	0.19	0.19
5	M	52	21	3.61	3.61	4	5	0.20	0.11	0.15	0.06
6	F	60	16	6.65	6.65	15	10	0.09	0.07	0.19	0.21
7	F	60	22	3.61	3.61	5	5	0.08	0.05	0.14	0.19
8	F	47	20	3.61	3.61	3	5	0.17	0.06	0.07	0.15
9	M	54	24	3.61	3.61	4	4	0.09	0.07	0.07	0.17
10	M	59	16	3.61	3.61	4	5	0.20	0.12	0.16	0.20

4.2.2 Procedure

Subjects' Monofilament and Two-Point Discrimination scores for the index and

thumb fingertips were compared with and without noise. Specifically, sensory scores

without noise were recorded at the beginning (pre) and end (post) of the testing session.

Sensory scores for the pre and post test were compared to ensure no learning effect and

no residual effect of noise after the exposure during the one day testing session. In

between the pre and post sensory tests without noise, sensory scores with noise were

recorded while subthreshold vibrotactile noise was applied to four different locations at

three noise intensities. The subthreshold vibrotactile noise was turned on immediately before each sensory test and was turned off immediately after each sensory test (lasting approximately 1 minute each). The testing session lasted for approximately two hours for each subject.

Subthreshold vibrotactile noise was white noise bandwidth filtered at 0 to 500 Hz, applied with a C-3 Tactor (Engineering Acoustics, Inc. Casselberry, Florida). Due to the characteristics of the C-3 Tactor, the vibration amplitude could have been larger for 100-300Hz which includes the sensitive range of the Pacinian corpuscles. The noise was applied to one of four locations in the paretic upper limb (Figure 20): 1) dorsal hand approximately 2 cm proximal to the index finger knuckle; 2) dorsal hand approximately 2 cm proximal to the thumb knuckle; 3) dorsal wrist, medial to the radial styloid process; and 4) volar wrist, medial to the radial styloid process. These locations were arbitrarily chosen with the intention of developing a future wearable rehabilitation device for stroke survivors. Since the long-term goal of the research is to improve dexterity and grip control, noise locations that would interfere with gripping, such as the fingertip or palm, were avoided. Presentation of noise locations was block randomized.
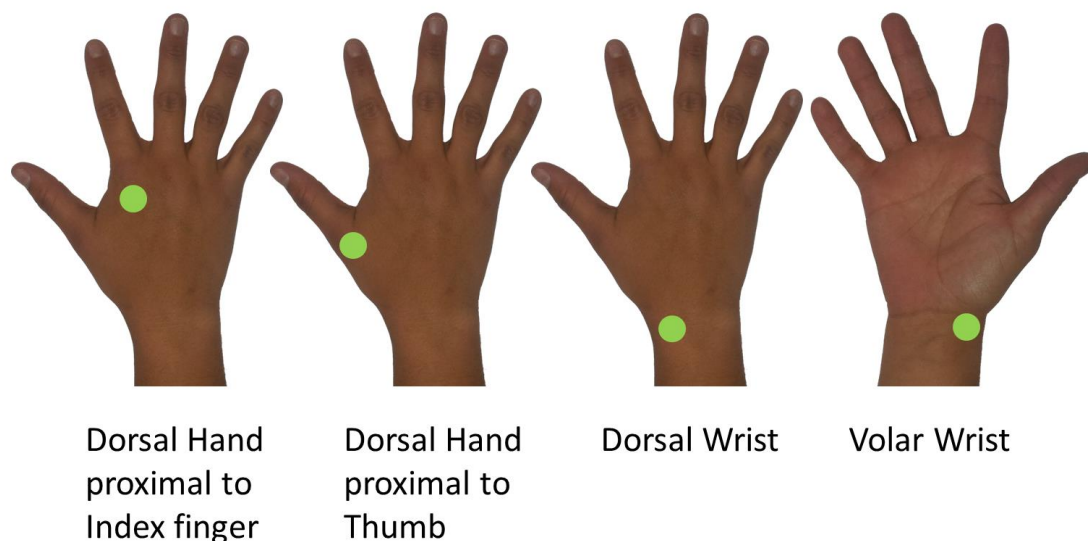
Figure 20: Sensation scores were recorded while remote vibrotactile noise was applied to one of four locations: 1) dorsal hand approximately 2 cm proximal to the index finger knuckle; 2) dorsal hand approximately 2 cm proximal to the thumb knuckle; 3) dorsal wrist, medial to the radial styloid process; and 4) volar wrist, medial to the radial styloid process. Noise intensity was set to 40%, 60%, or 80% of the sensory threshold for each location for each stroke survivor.

Noise intensities were set to 40%, 60%, or 80% of the sensory thresholds specific for each location. The order of testing different noise intensities was randomized within each location block. To determine the sensory threshold, the noise intensity was increased and decreased until the subject was barely able to distinguish between an "off" and an "on" presentation of the vibrotactile noise (i.e., the method of ascending and descending limits (Collins et al. 1997)). Subjects' mean sensory threshold occurred when the Tactor was driven by current of 0.17 A peak-to-peak. There is a linear relationship between the current and amplitude of the vibration. According to the data sheet from the manufacturer, 0.17 A peak-to-peak corresponds to a maximum amplitude of 260 μm. Subthreshold noise intensities were chosen not only so that subjects could not distinguish

between trials with and without noise (Priplata et al. 2002), but also because suprathreshold noise has been shown to degrade performance (Wells et al. 2005).

The Monofilament and Two-Point Discrimination Tests were administered using standard testing measures. For the Monofilament score, beginning with the baseline 2.83 Monofilament (indicating the threshold for "normal sensing"), the Monofilament was applied to the fingertip at least three times and the smallest Monofilament for which the subjects responded "yes" and could identify the correct finger that was touched marked the score (Bell-Krotoski et al. 1993). Similarly, the Two-Point Discrimination test was conducted so that subjects were asked to respond either "one" for a single point and "two" for two points separated by a small distance. One and two point stimuli were alternated randomly. The smallest distance where the subjects responded correctly to the two separated points was used for their Two-Point Discrimination score (Bell-Krotoski et al. 1993). A score of 2.83 (Dellon 1997) and 5 mm (Louis et al. 1984) was considered normal for the Monofilament test and Two-Point Discrimination tests, respectively.

4.2.3 Data Analysis

Monofilament Test scores (ranging from 2.83 to 6.65) were converted to the corresponding estimated logarithmic bending force (ranging from .07 to 300 grams) for the statistical analysis. Paired t-tests showed that neither the Monofilament score nor Two-Point Discrimination Test score without vibrotactile noise at the beginning of the testing session was significantly different from that at the end of the testing session without noise ($p=.33$ and $p=.78$ for the Monofilament and Two-Point Discrimination,

respectively), indicating that there was no learning effect with repeated sensory tests and there was no residual effect of noise on tactile sensation. Therefore, sensory scores pre and post testing sessions were averaged to become the noise off trials.

Two separate repeated measures ANOVAs were completed to determine how stroke survivors' tactile sensation varied with vibrotactile noise. The first ANOVA determined if stroke subjects' Monofilament Test scores varied significantly by noise 'on' and 'off', noise location (nested in the noise 'on' condition), noise intensities (nested in noise 'on'), finger (index or thumb), and their second-order interactions. The same ANOVA was performed for the Two-Point Discrimination Test scores. Specifically, these two ANOVAs were used to determine 1) if noise had an overall effect on the Monofilament and Two-Point Discrimination Test scores, and 2) if different noise locations and intensities had varying effects on the Monofilament and Two-Point Discrimination Test scores. Since the Test for Skewness showed skewed Monofilament ($p<.01$) and Two-Point Discrimination score data ($p<.01$) (Tabachnick and Fidell 2007), log and inverse (1/x) transformations were applied to the Monofilament and Two-Point Discrimination data, respectively, to yield non-significant skew values. Transformed data were used for the ANOVAs. In addition to these ANOVAs, the same analyses were performed using nonparametric Kruskal-Wallis tests, which resulted in the same conclusion (not presented here). As an additional analysis, a Pearson Correlation examined the relationship between improvement in sensation and functional motor score (Fugl-Meyer Assessment).

4.3 Results

4.3.1. Improved Monofilament scores with remote subthreshold vibrotactile noise

Stroke survivors' fingertip mean Monofilament Test scores improved from 3.91 to 3.73 when vibrotactile noise was applied to the paretic hand remotely from the fingertip (subject, noise location, intensity, and fingers pooled) (Figure 21 and Appendix D). Seven out of the ten stroke survivors had improved Monofilament Test score when vibrotactile noise was applied to the paretic hand remotely from the fingertip, for at least one noise location, noise intensity, and finger. The improvement in the Monofilament scores with vibrotactile noise was statistically significant (ANOVA, noise main effect with $p<.01$). All other effects of noise location ($p=.13$), intensity ($p=.48$), finger ($p=.45$), and interactions were not significant (ANOVA with $p>.05$). Monofilament scores improved from mean ± standard deviation of 3.91 ± 0.94 to 3.73 ± 1.03 with vibrotactile noise (subject, noise location, intensity, and fingers pooled). Neither finger (index, thumb) nor the interaction between finger and noise was significant, indicating vibrotactile noise improved light touch sensation for both fingers. Noise location and intensities did not significantly affect the Monofilament scores, indicating that all remote vibrotactile noise at all intensities improved Monofilament score at the fingertips to the similar degree. As described earlier, monofilament scores without vibrotactile noise did not change pre vs. post test ($p=.33$), indicating no learning effect and no after-effect of noise. Improvement in the Monofilament score with noise was not significantly related to the Fugl-Meyer score (Pearson Correlation, p=.84).
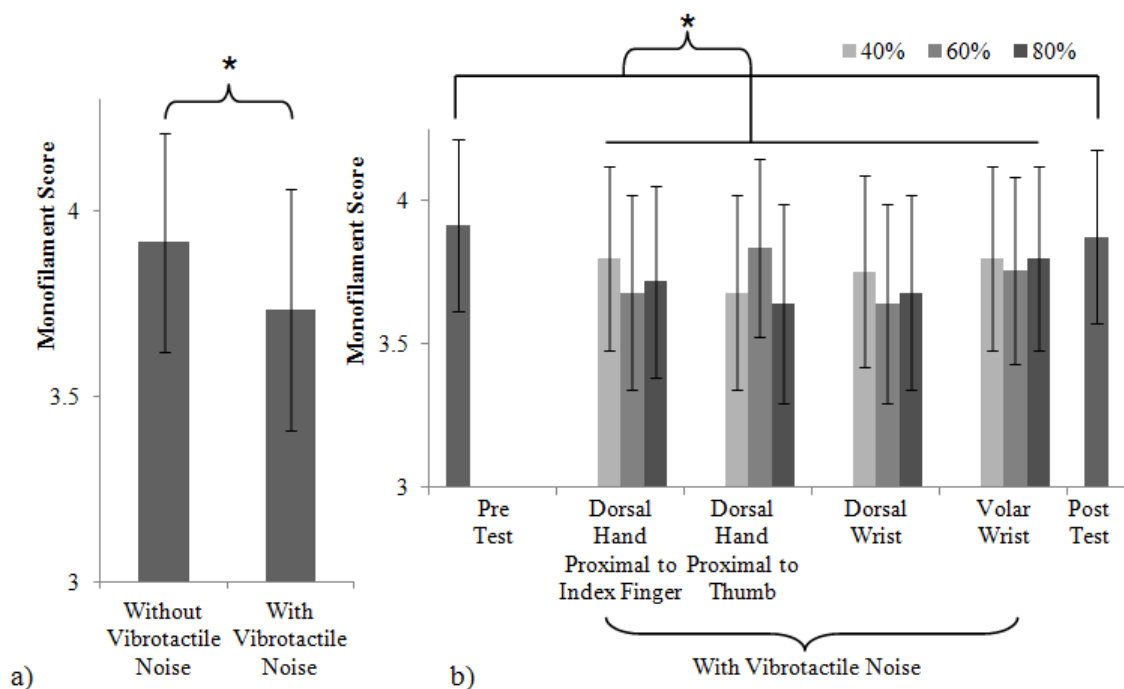
Figure 21: Mean ± SE Monofilament scores significantly decreased with subthreshold vibrotactile noise (noise locations, intensities, fingers, and subjects pooled) ($p<.01$) (a). Noise locations and intensities did not significantly affect the improvement of Monofilament score (fingers and subjects pooled, $p>.05$ for noise location and intensity) (b).

4.3.2 No significant effect of vibrotactile noise on Two-Point Discrimination

 Stroke survivors' Two-Point Discrimination Test score did not significantly change when vibrotactile noise was applied to the paretic wrist and dorsal hand (Figure 22 and Appendix D, ANOVA, noise main effect with p=.84). Mean Two-Point Discrimination was significantly dependent upon finger (ANOVA, finger main effect with $p<.01$) and was significantly higher for the thumb compared to the index finger. Mean Two-Point Discrimination scores were not significantly dependent upon noise intensity ($p=.82$),

location (*p*=.19), or any interactions (*p*>.05). The Two-Point Discrimination scores

without vibrotactile noise did not change pre vs. post test (*p*=.78).

a)

b)
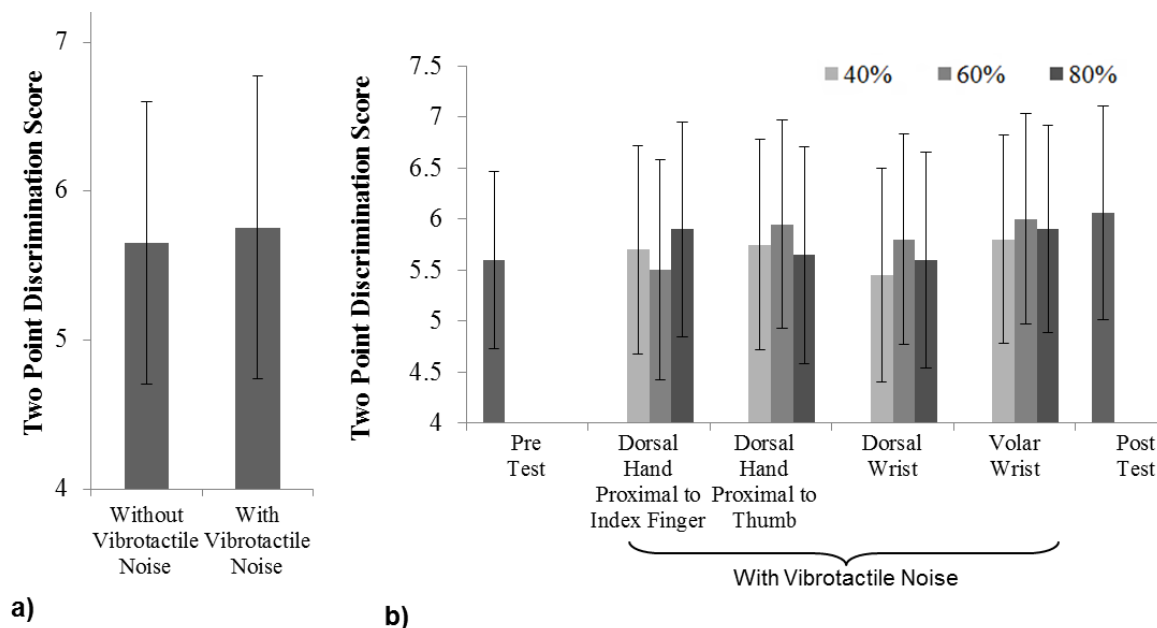
Figure 22: Mean ± SE Two Point Discrimination scores were not significantly affected by the vibrotactile noise (a) nor with noise locations, intensities, fingers, and their interactions (fingers and subjects pooled) (*p*>.05) (b). The Two-Point Discrimination score without vibrotactile noise did not change at the beginning vs. end of the testing session.

4.4. Discussion

4.4.1 Remote subthreshold vibrotactile noise enhanced stroke survivors' light touch sensation at the fingertips

Light touch sensitivity at the pads of the thumb and index fingertips was enhanced with the subthreshold vibrotactile noise at the wrist or dorsal hand, as evidenced by the

improved Monofilament Test score (Figure 21 and Appendix D). All noise intensities

(40%, 60%, and 80% of the sensory threshold) and locations (dorsal hand and wrist)

improved the fingertip light touch sensation. The benefit of the subthreshold vibrotactile

noise was instantaneous, and not influenced by learning or after-effect of noise (as

evidenced by insignificant difference between the Monofilament scores without noise pre

and post test). The largest improvement of 25% in Monofilament score with vibrotactile

noise compared to without vibrotactile noise was found for the vibrotactile noise at the

dorsal wrist at 60% of the sensory threshold and for the vibrotactile noise at the dorsal

hand proximal to the thumb knuckle at 80% of the sensory threshold (Figure 21b and

Appendix D). Hand motor function (as measured by the hand and wrist subdivision of the

Fugl-Meyer assessment) was not found to be related to the degree of sensory

improvement. Therefore, stochastic resonance improved sensation for the stroke

survivors in this study who ranged from 9 to 24 (out of 24) in hand motor function levels.

The clinical implication of this finding is significant. This study finding indicates

that a wearable assistive wrist band applying subthreshold vibrotactile noise can be

developed to enhance touch sensation for stroke survivors' fingertips and assist with their

dexterous hand movement. The advantage of this wearable assistive wrist band compared

to the current glove with a vibrator attached at the fingertip (Kurita et al. 2011) is that the

wrist band minimally interferes with manual dexterity of stroke survivors. In addition, the

vibration is minute at the level that is not perceivable. Thus, this vibration is unlikely to

result in numbness or tissue damage in the long-run.

4.4.2. Potential mechanisms of remote sensory enhancement

It is unlikely that the light touch sensation improved via the vibrotactile noise traveling from the wrist or dorsal hand to the fingertips through the skin, because vibration significantly attenuates across the skin. In general, vibration can improve tactile sensation by directly stimulating the tactile receptors in the finger skin (Kurita et al. 2011). However, Kurita et al. (Kurita et al. 2011) reported that mechanical vibration may lose 90% of its original power when it travels 1 to 2 cm on the skin (Kurita et al. 2011). In our study, the distance between the fingertip and noise locations ranged from 10 to 20 cm. Therefore, it is unlikely that the index and thumb fingertips' sensation would have been affected by transfer of the mechanical vibration through the skin from any of the noise locations to the thumb or index fingertip.

A more likely mechanism for enhanced light touch sensation at the fingertips with remote vibrotactile noise is that the vibrotactile noise at the wrist and dorsal hand may have increased the sensory neurons' excitability not only for the wrist and dorsal hand but also for the fingertips through interneuronal overlap either in the spinal or supraspinal level. For example, Merzenich et al. (Merzenich et al. 1983) found that median, ulnar, and radial nerves, although peripherally separate, appear to overlap in the central nervous system. Specifically, they have shown that immediately after median nerve transaction, significant inputs from the dorsum of the hand (innervated by the radial and ulnar nerve)

appear in the somatosensory cortex area that was previously innervated by the median nerve in monkeys. Such emergence of radial and ulnar nerve representation in the median nerve territory in the somatosensory cortex was immediate, suggesting pre-existing synaptic overlap between the sensory representations of the palmar and dorsal areas of the hand (Merzenich et al. 1983). Unmasking of the pre-existing overlap has been shown in other studies involving healthy persons (Bjorkman et al. 2004) as well as people with stroke (Sens et al. 2012a). In addition it has also been shown that vibrotactile noise results in increased cortical as well as spinal neuronal activities in humans and cats, which demonstrates the effect of stochastic resonance in the central nervous system (Manjarrez et al. 2002a; Manjarrez et al. 2003). Therefore, vibrotactile noise applied to the wrist or dorsal hand may have increased the fingertip sensation by increasing the excitability of the sensory neurons in the central nervous system through stochastic resonance and interneuronal connections.

Another potential mechanism for the enhanced light touch sensation is that vibrotactile noise at the wrist or the dorsal hand may have increased the synchronization of sensory neuron firing between the spinal cord and the somatosensory cortex (Manjarrez et al. 2002a; Manjarrez et al. 2003). The increased synchronization may facilitate neural communication between the spinal and cortical levels (Fell and Axmacher 2011), thereby enhancing detection of light touch stimulation from the fingertips to the somatosensory cortex.

4.4.3. Lack of noise effect on Two-Point Discrimination

Two-Point Discrimination sensation was not significantly affected by the subthreshold vibrotactile noise in this study. This finding aligns with a study done by Kurita et al. (2011) that subthreshold vibrotactile noise enhanced only light touch sensation but not Two-Point Discrimination at the fingertips. A reason for inconsistent results may be that the Monofilament Test and Two-Point Discrimination Test assess different aspects of sensation. The Monofilament Test assesses the threshold of the mechanoreceptors responsible for pressure, whereas the Two-Point Discrimination Test examines spatial resolution of receptive fields for discriminative touch (O'Sullivan and Schmitz 2006). Therefore, the present study's finding suggests that spatial resolution of mechanoreceptors was not affected by the subthreshold vibrotactile noise.

4.4.4. Limitations and Future Work

One limitation of this study could be the use of the Two-Point Discrimination Test to demonstrate impact on the tactile spatial resolution. Although still used widely in clinics to demonstrate a deficit in spatial acuity, the Two-Point Discrimination has been criticized previously by scientists for the response variable of "one point" or "two points" as an unreliable outcome measure that has high variability both between and within subjects (Craig and Johnson 2000). Additionally, although Monofilament Test Score showed that all subjects had light touch deficit at the beginning, not all subjects had

sensory deficit according to the Two-Discrimination Test. Therefore, the lack of improvement in the Two-Point Discrimination Test with vibrotactile noise could have been due to near-normal starting scores leaving not much room for improvement.

Additionally, this study is limited by examining the effect of remote stochastic resonance on sensation from only two fingers, the index and thumb fingers. Due to limited time to examine each noise level and location, no additional fingers were examined for sensation. As discussed earlier, remote stochastic resonance (at sites on the hand/wrist innervated by the radial nerve) may have influenced both index and thumb fingertip sensation through integration of information from the median, ulnar, and radial nerves in the central nervous system. It can only be postulated that similar improvements found with the index and thumb fingertip may also occur for the middle, ring, and little fingertips through this integration. However, further testing would be necessary to verify.

In this study, the Monofilament scores were recorded from a set of 5 Monofilaments, instead of the set of 20 Monofilament sizes. The 20 Monofilament sizes would have shown greater resolution to the degree of sensory improvement. However, the 5 Monofilament set was still sufficient to show the large changes in sensation for this study.

While the present study demonstrated the immediate effects of vibrotactile noise on sensory enhancement, in order to be applied to a longer term sensorimotor rehabilitation therapy, future studies need to examine the effects of repeated exposure and the long-term benefits of vibrotactile noise in stroke survivors. Although Monofilament scores pre and post the 2-hour test were not significantly different in the present study, longer or repeated exposure to the vibrotactile noise may elicit longer-lasting improvements in fingertip sensation. A sensory re-training program, such as the one described by Carey et al. (Carey and Matyas 2005), could be complimented by the addition of vibrotactile noise. Furthermore, the effect of sensory enhancement on motor function following stroke should be investigated. Specifically, how effectively the enhanced sensation at the fingertips leads to improved dexterity such as precise grip force regulation and coordination (Blennerhassett et al. 2006) could be investigated. Finally, a prototype of a vibrotactile noise wrist band will be developed for clinical evaluation to determine the efficacy of the remote vibrotactile noise for rehabilitation post stroke.

4.5 Conclusions

Remote stochastic resonance phenomenon was investigated to determine if subthreshold vibrotactile noise at the wrist or dorsal hand can enhance the tactile sensation at the fingertip of the stroke survivors. The application of the subthreshold vibrotactile noise at the wrist and dorsal hand instantaneously enhanced the light touch sensation at the fingertip of stroke survivors. This benefit in the light touch sensation was not influenced by learning effect. The most improvement in the light touch sensation at

the fingertip occurred when the dorsal wrist and the dorsal hand proximal to the thumb knuckle were stimulated at 60% and 80% of the sensory thresholds, respectively. This study carries clinical significance, since the finding of this study demonstrates strong potential that a subthreshold vibrotactile noise-generating assistive wrist band may be able to enhance fingertip tactile sensation for stroke survivors and may contribute to enhanced manual dexterity and abilities for activities of daily living.

Chapter 5: Effects of remote subthreshold vibrotactile noise on stroke survivors' altered phalanx force direction during power grip

ABSTRACT

Previously it was found that during static power grip, phalanx forces are directed away from the direction normal to the object surface, more so for stroke survivors with tactile sensory deficit than for those without tactile sensory deficit, compared to age-matched controls. This increased phalanx force deviation represents increased likelihood of object slippage from the hand. Recently, application of vibration to the wrist skin at unfelt intensities was shown to improve stroke survivors' fingertip sensation. The objective of this study was to determine if the subthreshold vibrotactile noise applied to the wrist would improve stroke survivors' phalanx force directions during power grip, especially for those with tactile sensory deficit. Thirteen chronic stroke survivors with tactile sensory deficit, 7 chronic stroke survivors without tactile sensory deficit, and 13 age-matched healthy controls performed maximum power grip on an instrumented cylinder. Phalanx force direction for the thumb, index, and middle fingers and muscle activity were recorded. To confirm tactile sensory enhancement with vibrotactile noise, Monofilament scores for the fingertips and palm were recorded with and without vibrotactile noise for the stroke survivors with tactile sensory deficit. Results showed that vibrotactile noise significantly reduced phalanx deviation, on average, by 7%, for all groups, and improved Monofilament scores for stroke survivors with sensory deficit. There was no significant change in phalanx normal force or muscle activity with vibrotactile noise. Therefore, vibrotactile noise may be useful in reducing phalanx force deviation, improving gripping

stability, and complementing sensorimotor rehabilitation especially for stroke survivors with tactile sensory deficit.

5.1. Introduction

Impaired tactile sensation has previously been shown to affect anywhere from 50% (Carey and Matyas 2011) to 85% (Kim and Choi-Kwon 1996) of stroke survivors. The extent of tactile sensory deficit is related to motor recovery post stroke (Tyson et al. 2008). Furthermore, reduced somatosensation post stroke has been shown to hinder functional gains and recovery during rehabilitation therapies (Carey 1995) and can increase duration of time in rehabilitation (Sommerfeld and von Arbin 2004).

Tactile sensation is important for finger force management during gripping and object manipulation (Johansson 1996; Zatsiorsky and Latash 2004b). Artificially reducing tactile sensation in fingertips of young healthy individuals has led to impaired finger force control (Tseng 2013; Westling and Johansson 1984b) and increased object dropping (Augurelle et al. 2003). Stroke induced tactile sensory deficit has been shown be associated with impaired pinch grip force regulation (Blennerhassett et al. 2007) and impaired power grip force control (Chapter 3). Specifically, during power grip, stroke survivors with tactile sensory deficit have been shown to produce phalanx force with increased deviation from the direction normal to the grip surface compared to stroke survivors without tactile sensory deficit and age-matched healthy controls (Chapter 3). Evidence of a greater reduction in activation of the first dorsal interosseous (FDI) and extensor digitorum communis (EDC) muscles compared to the flexor digitorum superficialis (FDS) muscle accompanied the increased phalanx force deviation for stroke survivors with tactile sensory deficit, but not for stroke survivors without tactile sensory

deficit, compared to age-matched healthy controls (Chapter 3). Proper muscle activation of the finger muscles is critical for controlling phalanx force direction, and weakening or underactivation of any muscle can limit phalanx force output in specific direction (Kutch and Valero-Cuevas 2011). This is especially true for the intrinsic muscles, such as the FDI muscle, whose decreased activation has previously been shown to lead to increased finger force deviation in biomechanical models (Valero-Cuevas et al. 2000). Therefore, the previous study (Chapter 3) concluded that tactile sensory loss could have limited stroke survivors' ability to sense their current phalanx force direction and could have hampered feedback control for precise coordination of muscle activities and phalanx force direction.

Increased phalanx force deviation can lead to object dropping if the deviation exceeds the limit allowed by the coefficient of friction (COF) for that surface. This allowed limit of phalanx force deviation, called the 'cone of friction', is calculated as the arctangent of the COF between finger skin and the object's surface (MacKenzie and Iberall 1994). Phalanx force deviations outside the cone of friction have previously been observed to lead to finger slip for stroke survivors during pinch grip (Seo et al. 2010). Therefore, stroke survivors with tactile sensory deficit could be at a greater risk of finger slippage during power grip due to their greater phalanx force deviation.

One of the recent methods of enhancing tactile sensation is application of subthreshold vibrotactile noise remotely from the fingertips so as not to interfere with

object manipulation. Specifically, vibration applied to the volar or dorsal wrist or dorsum hand skin at the intensities below the level of perception has been shown to result in improved fingertip tactile sensation in chronic stroke survivors with sensory deficit (Enders et al. 2013). The location of noise away from the fingertips and palm avoids interfering with gripping and thus provides potential for a wearable device that improves dexterity and grip control for stroke subjects. This finding of improved sensation with remote noise is similar with stochastic resonance in which the application of white noise can improve the detection of a weak signal, such as a light sensory signal (Galica et al. 2009; Moss et al. 2004; Priplata et al. 2002). Although this method of applying the vibration noise remotely from the fingers was found to improve the fingertip sensation for stroke survivors with sensory deficit (Enders et al. 2013), it is currently unknown if phalanx force control can be improved as well.

The goal of this study was to determine the effect of remote vibrotactile noise on controlling phalanx forces during static power grip. Specifically, this study examined the effect of remote subthreshold vibrotactile noise on phalanx force deviations in stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and age-matched healthy controls. While the impact was anticipated for stroke survivors with tactile sensory deficit, age-matched controls and stroke survivors without tactile sensory deficit were also tested since remote vibrotactile noise is a relatively new technique and its impact on motor control for those without sensory deficit is unknown. In addition, muscle activity was also recorded to examine if changes in phalanx force direction with the noise is associated with changes in muscle activity. Lastly, to determine if the

improvement in phalanx force control was accompanied by an improvement in sensation, the effect of remote vibrotactile noise on the fingertip and palm tactile sensation for the stroke survivors with sensory deficit was examined.

5.2. Methods

5.2.1 Subjects

Thirteen chronic stroke survivors with tactile sensory deficit (mean age ± SD = 56 ± 12 years), 7 chronic stroke survivors without tactile sensory deficit (64 ± 11 years), and 13 age-matched healthy control subjects (57 ± 8 years) participated (Table IV). All stroke survivors were at least 6 months post stroke. The two stroke groups were separated based on the fingertip tactile sensory results from the Semmes-Weinstein monofilaments test (Bell-Krotoski et al. 1993) at the beginning of the testing session (Baseline Monofilament Score in Table V). Those stroke survivors who had a score of ≥ 3.61 (Dellon 1997) for both the index and thumb fingertips were considered to have tactile sensory deficit. All age matched controls were neurologically healthy and had no tactile sensory deficit in the fingertips (< 3.61 Semmes-Weinstein monofilaments test).

Both stroke survivor groups were similar in terms of motor function. Stroke survivors' mean functional motor impairment quantified by the Chedoke-McMaster Stroke Assessment Hand Section (Gowland et al. 1995) was 5 ± 2 (out of a possible 7) for both stroke survivor groups with and without tactile sensory deficit (t-test with $p > .05$

between the two stroke groups). Another functional motor impairment score based on the hand and wrist subdivision of the Fugl-Meyer Assessment (Fugl-Meyer et al. 1975) was 18 ± 7 and 20 ± 7 (out of a possible 24) for the stroke survivors with tactile sensory deficit and stroke survivors without tactile sensory deficit groups, respectively (t-test with $p>.05$ between the two stroke groups). Stroke survivors' muscle spasticity was similar for both stroke survivor groups, with a median and interquartial range (IQR) for the Modified Ashworth Scale (Ashworth 1964) of 0 (IQR, 0 to 3) for the stroke survivors with tactile sensory deficit and 0 (IQR, 0 to 2) for the stroke survivors without tactile sensory deficit (Mann-Whitney Test with $p>.05$). All subjects signed a consent form and followed a protocol approved by the Institutional Review Board.

Table V: Subject Demographics

Group	Subject	Time since most recent stroke (months)	Type of Stroke	Hand Dominance*		Paretic Side	Sex	Age (years)	Fugl-Meyer Score (out of 24)	Chedoke McMaster Score (out of 7)	Baseline Monofilament Score	
				Pre Stroke	Post Stroke						Index	Thumb
Stroke survivors with tactile sensory deficit	B01	36	Unknown	R	L	R	Female	67	5	2	4.31	3.61
	B02	47	Hemorrhagic	R	R	L	Female	51	24	7	3.61	3.61
	B05	48	Hemorrhagic	R	R	R	Male	52	21	5	3.61	3.61
	B06	53	Ischemic	R	R	L	Male	54	10	2	6.65	6.65
	B08	40	Ischemic	L	L	L	Female	61	16	5	6.65	6.65
	B11	67	Ischemic	R	R	L	Male	59	16	6	3.61	3.61
	B12	7	Ischemic	R	R	R	Male	66	24	7	4.31	4.31
	B13	55	Hemorrhagic	L	R	L	Male	32	8	2	6.65	6.65
	B14	162	Hemorrhagic	R	R	L	Male	60	22	7	3.61	3.61
	B16	62	Unknown	R	R	R	Female	60	22	7	3.61	3.61
	B17	142	Ischemic	L	L	R	Female	53	24	7	3.61	3.61
	B19	8	Unknown	R	R	L	Male	65	24	7	3.61	3.61
	B20	44	Ischemic	R	R	L	F	43	18	3	6.65	6.65
Stroke survivors without tactile sensory deficit	B03	245	Ischemic	L	L	R	Female	55	24	7	2.83	2.83
	B07	44	Ischemic	R	R	R	Female	75	24	6	2.83	2.83
	B09	51	Ischemic	R	R	R	Male	60	23	7	2.83	2.83
	B10	126	Hemorrhagic	L	R	L	Female	67	9	2	2.83	2.83
	B15	52	Ischemic	R	L	R	Male	81	9	2	3.61	2.83
	B18	64	Ischemic	R	R	L	Male	62	24	7	3.61	2.83
	B21	172	Hemorrhagic	R	R	R	Female	50	24	7	2.83	2.83

*R= "Right hand dominance", L= "Right hand dominance"

5.2.2 Procedure

Subjects performed maximum power grip on a custom made grip dynamometer (Enders and Seo 2011), with or without remote vibrotactile noise while individual phalanges' normal and proximal-distal shear force data for a single finger as well as surface muscle EMG activities were recorded. Subjects sat in a chair with arm rested and flexed at 90°. Stroke survivors used their paretic hand and control subjects used their non-dominant hand because this hand is normally used to hold the object while the non-

paretic or dominant hand is used to perform finger manipulation tasks. The surface of the dynamometer was covered in a high-friction rubber surface with COF of 1.00 with the finger skin or a low-friction paper surface with a COF of 0.43 with the finger skin (Chapter 3). Only phalanx forces from the thumb, index, and middle fingers were recorded due to these fingers being most involved in producing power grip force (Enders and Seo 2011). Measurement of phalanx normal and shear forces for the 3 fingers, two surfaces, and noise "on" and "off" were repeated at least two times each. The order of noise, surfaces and fingers were randomized. Subjects were blinded to the noise "on" and noise "off" condition because they were unable to distinguish when the noise was "on" during testing.

During all power grip, muscle activation was also measured by recording surface EMG (Bortec Biomedical Ltd., Calgary, AZ) from the FDS, EDC, and FDI muscles at 1000Hz. The surface electrodes were placed on the skin above each targeted muscle's belly according to literature (Basmajian 1989) after skin was prepared with alcohol swabs. Maximum voluntary contractions (MVC) that targeted each muscle separately were collected additionally.

Subthreshold remote vibrotactile noise was white noise bandwidth filtered at 0 to 500 Hz and was applied to the dorsal wrist with an intensity at 60% of the sensory threshold with a C-3 Tactor (Engineering Acoustics, Inc. Casselberry, Florida). To determine the sensory threshold for each subject's dorsal wrist, noise intensity was

increased and decreased in a method of ascending and descending limits (Collins et al. 1997) until subjects could no longer distinguish between when the noise was "on" and "off". The noise intensity was set to 60% sensory threshold on the dorsal wrist (medial to the radial styloid) because this intensity and remote location were found to be among the most effective to improve fingertip sensation in our pervious study (Enders et al. 2013).

Additionally, Monofilament scores for the stroke survivors with tactile sensory deficit were recorded for the index fingertip pad, thumb fingertip pad, upper palm below the index (or the palmar skin over the index finger knuckle), and the thenar eminence region with and without vibrotactile noise using the standard method (Bell-Krotoski et al. 1993; Enders et al. 2013). Monofilament scores were recorded with vibrotactile noise turned off at baseline at the beginning of the testing session, with vibrotactile noise on during testing after completing the power grip trials, and with vibrotactile noise turned off at the end of the testing session.

5.2.3 Data Analysis

Phalanx normal force and phalanx force deviation were determined for both stroke survivor groups and healthy controls. For each grip trial, the mean phalanx normal force and shear force was calculated from a 2-second period in which the total finger force (calculated as the sum of all the phalanx normal and shear forces) was greatest. The extent of phalanx force deviation from the direction perpendicular to the grip surface

was calculated as the arctangent of the absolute ratio of shear force to normal force in that time window. EMG was processed using the root mean square (RMS) with a 20-ms moving window and the mean RMS EMG during the same 2-second time period was computed for each muscle. The RMS EMGs for each of the three muscles during gripping trials were then normalized to that recorded during the MVCs, to calculate the muscle activity in %MVC.

One mixed-design ANOVA determined if the phalanx force deviations varied significantly for the three subject groups (stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and healthy controls), noise (on/off), surface (paper, rubber), finger (index, middle, thumb), phalanx (distal, middle, proximal), and the interactions between subject group and noise, noise and surface, noise and finger, and noise and phalanx. To examine if vibrotactile noise affects the grip force magnitude, a second mixed-design ANOVA determined if the phalanx normal force varied significantly for the three subject groups, noise, surface, finger, phalanx, and the interactions between subject group and noise, noise and surface, noise and finger, and noise and phalanx. Another ANOVA examined how muscle activity varied for the subject groups, noise, muscle, and the interactions between noise and subject group and the interaction between noise and muscle. As an additional analysis, an ANOVA determined if Monofilament scores for the stroke survivors with tactile sensory deficit group varied significantly by noise, location, and their second-order interaction. Monofilament scores (ranging from 2.83 to 6.65) were converted to the bending force (.07 to 300 grams) for statistical analysis. An ANOVA determined that the baseline and

post testing trials without vibrotactile noise were not significantly different from each other for either location ($p=.20$), indicating no residual effect of noise on tactile sensation. Therefore, these trials were combined to become the vibrotactile noise "off" trials for the statistical analysis. The phalanx force deviation data, phalanx normal force, muscle activation, and monofilament scores data were skewed based on the Test for Skewness (Tabachnick and Fidell 2007). Therefore, a square root transformation was applied to normalize the phalanx normal force and phalanx deviation data and the log transformation was applied to the muscle activation data and the monofilament scores. Transformed data was used in the ANOVA.

5.3. Results

The finding of this study is that phalanx force deviation was significantly reduced with the application of the remote vibrotactile noise for all subject groups. This improvement in phalanx force deviation was accompanied by improvement in the hand tactile sensation for stroke survivors with tactile sensory deficit, while phalanx normal force and muscle activity were not significantly affected by the remote vibrotactile noise for any subject group. Consistent with the previous study (Chapter 3), both stroke groups produced significantly less maximum phalanx normal force and significantly greater phalanx force deviation compared to healthy controls. Also consistent with the previous study (Chapter 3), the stroke survivors with tactile sensory deficit produced significantly greater phalanx force deviation (16˚) compared to stroke survivors without tactile sensory deficit (14˚) and healthy controls (12˚).

5.3.1 Phalanx force deviation improvement with vibrotactile noise 'on'

Phalanx force deviation was reduced by noise for all the subject groups (Figure 23and Appendix D). Phalanx force deviation was significantly dependent upon noise, phalanx, and the interaction between noise and finger and the interaction between noise and phalanx (ANOVA with $p<.05$). No other factor or interaction was significant (ANOVA with $p>.05$). Phalanx force deviation was reduced by 9%, on average (ANOVA, noise main effect with $p<.05$) (Figure 23a and Appendix D). Phalanx force deviation was reduced, on average, by 8% for stroke survivors with sensory deficit, 5% for stroke survivors without sensory deficit, and 12% for healthy controls (ANOVA, group and noise interaction with $p>.05$) (Figure 23b and Appendix D). Reduction in phalanx force deviation was the largest for the thumb finger than other fingers (noise and finger interaction $p<.05$) (Figure 23c and Appendix D) and for the middle phalanx than other phalanges (noise and phalanx interaction $p<.05$) (Figure 23d and Appendix D).

a)

b)

c)

d)

e)

f)

g)

h)
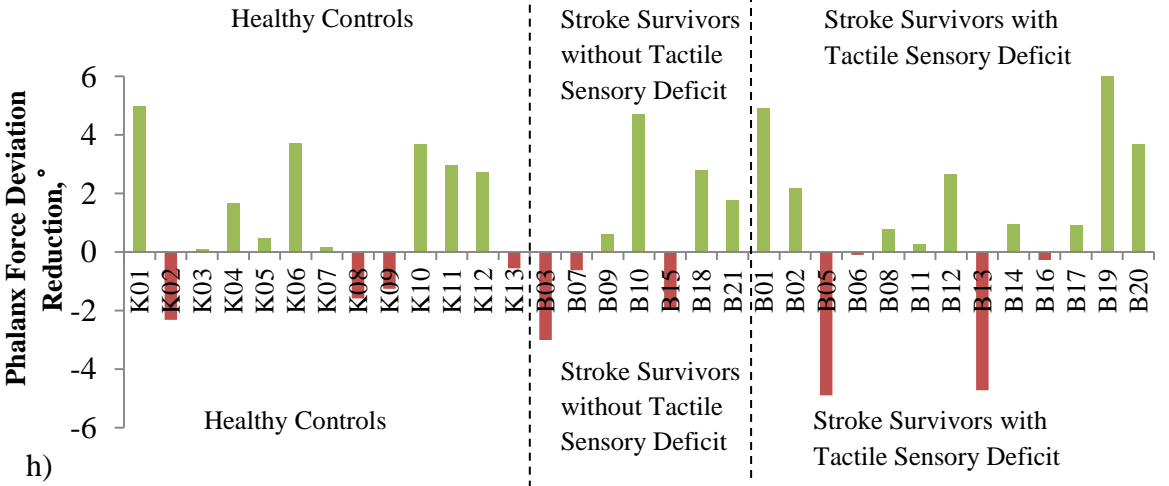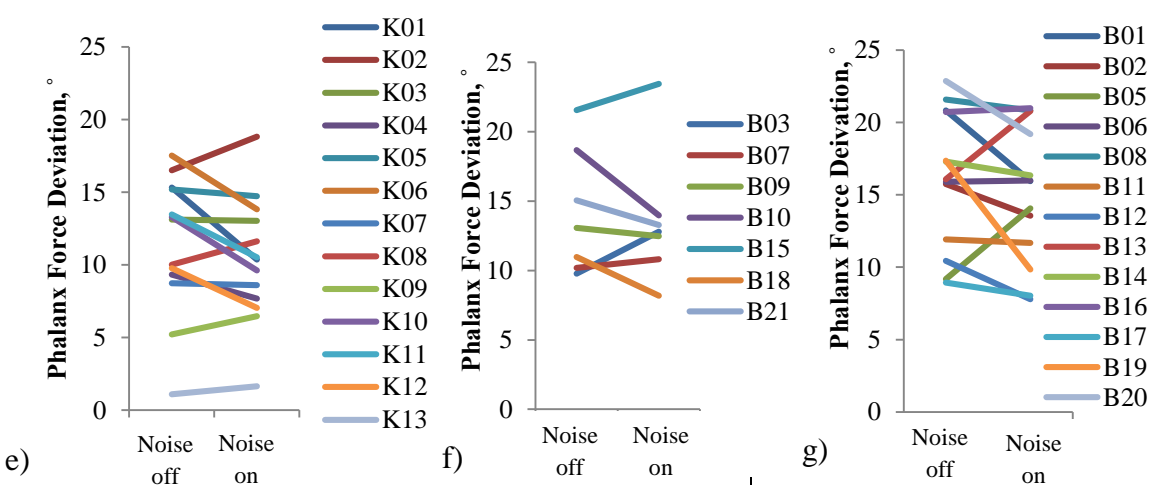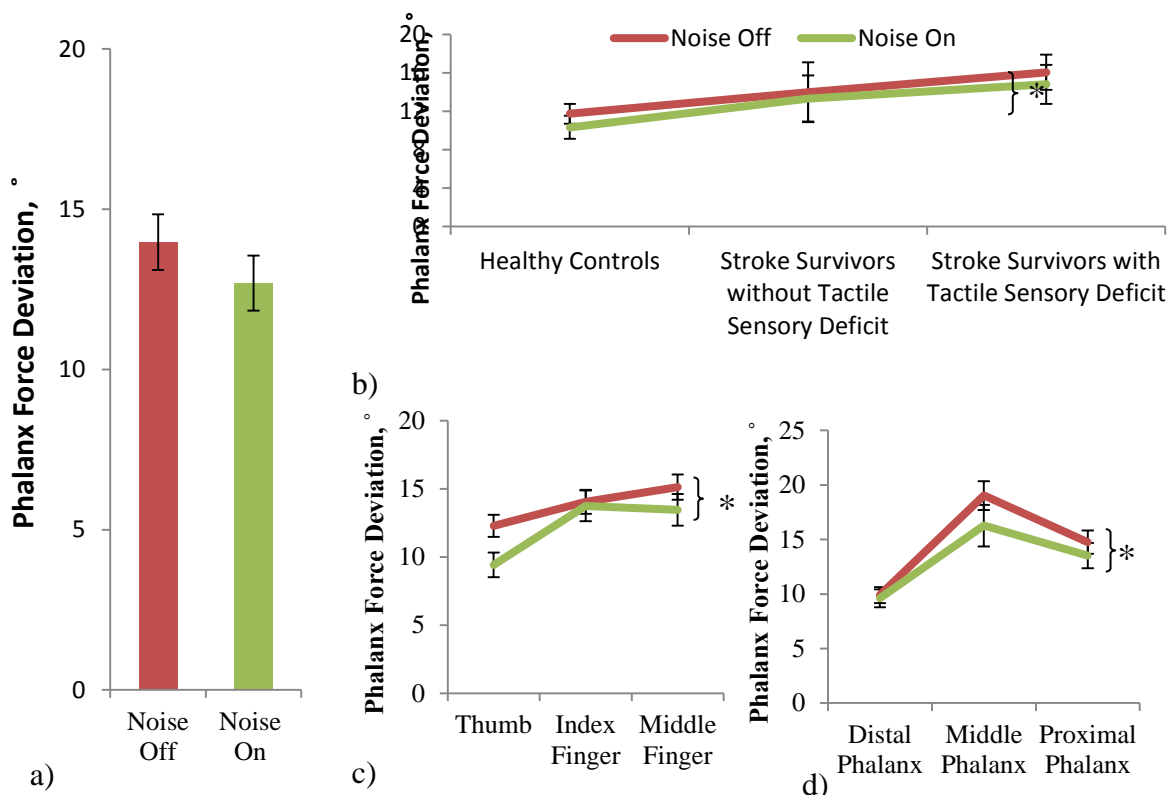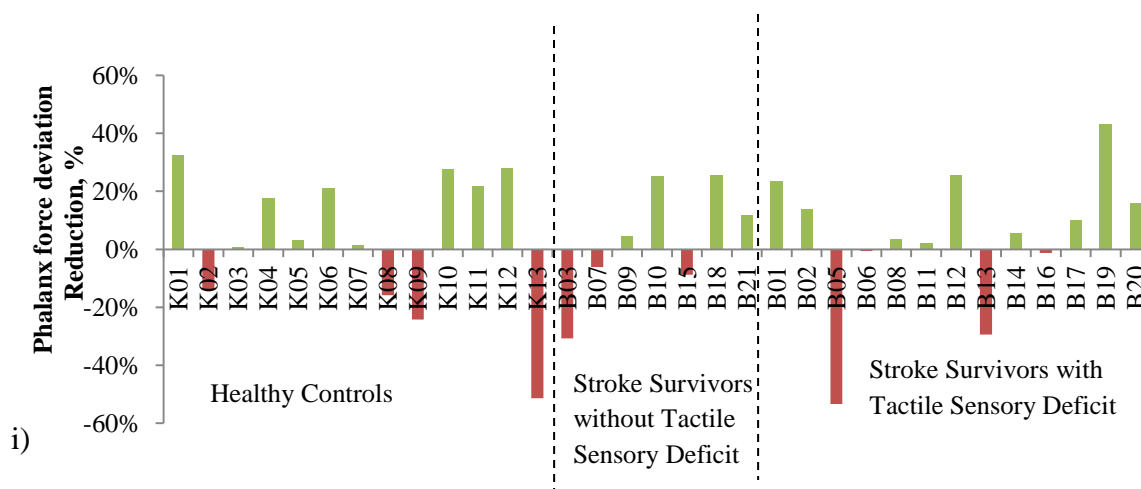
Figure 23: Mean ± SE phalanx force deviation was significantly reduced with remote vibrotactile noise turned on (a). Reduction in phalanx force deviation was observed across all subject groups (b). The extent that phalanx force deviation reduced with the noise differed by fingers (c) and phalanges (d). Individual subjects' change in phalanx force deviation with noise is shown in (e-g) and the change in phalanx force deviation with noise in degrees and percent change is shown in (h) and (i), respectively.

5.3.2 No change in phalanx normal force with vibrotactile noise 'on'

Phalanx normal force did not significantly vary with the application of remote vibrotactile noise ($p>.05$, Figure 24 and Appendix D). Phalanx normal force was significantly dependent upon subject group, finger, and phalanx (ANOVA with $p<.05$). No other factor or interaction was significant (ANOVA with $p>.05$). Phalanx normal force was largest in healthy controls, followed by the stroke survivors with tactile sensory deficit and stroke survivors without tactile sensory deficit (ANOVA subject group main effect with $p<.05$).
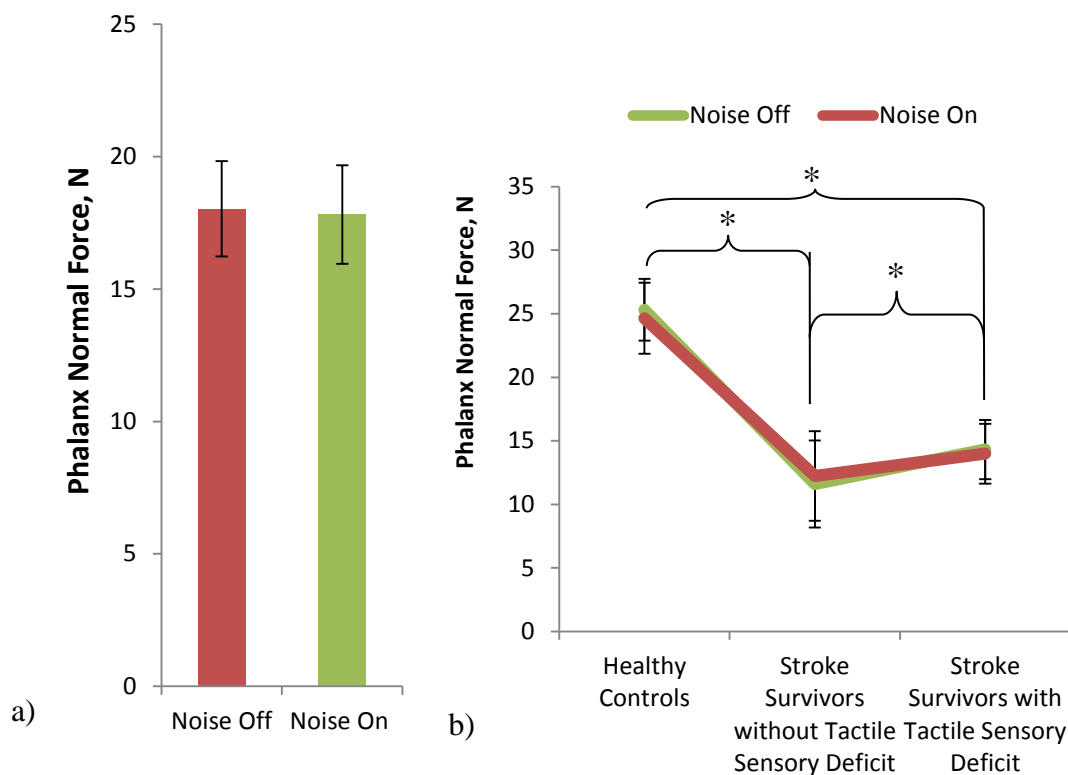
Figure 24: Mean ± SE phalanx normal force did not significantly change with the application of vibrotactile noise (a). Phalanx normal force was significantly greater for the healthy controls, followed in order by the stroke survivors with tactile sensory deficit and the stroke survivors without tactile sensory deficit (b). None of the subject groups significantly changed their phalanx normal force with remote vibrotactile noise.

5.3.3 No change in muscle activity with vibrotactile noise 'on'

Muscle activity was not significantly varied with the application of remote vibrotactile noise (Figure 25 and Appendix D). Muscle activity was significantly dependent upon subject group and muscle (ANOVA with $p<.05$). The healthy controls produced power grip with the largest muscle activity in %MVC, followed in order by the stroke survivors with tactile sensory deficit and the stroke survivors without tactile sensory deficit (ANOVA subject group main effect with $p<.05$). Muscle activity

significantly varied depending on the muscle with larger EDC and FDS activities than the

FDI muscle activity (ANOVA muscle main effect with $p<.05$).
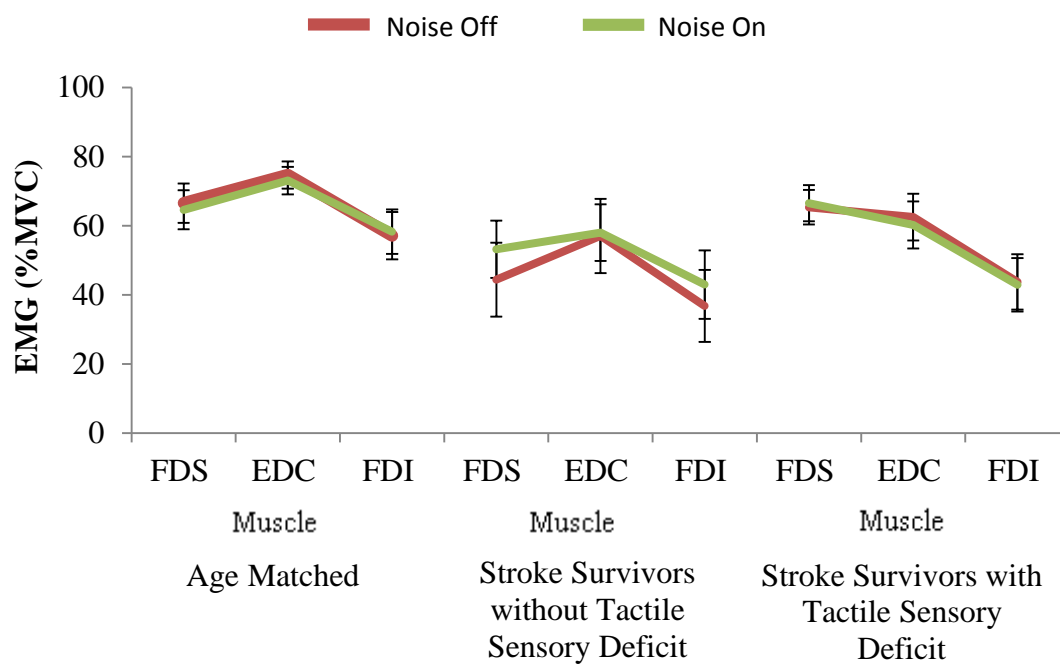
Figure 25: Mean ± SE Muscle EMG activity was not significantly affected by the application of remote vibrotactile noise.

5.3.4 Monofilament score improvement for stroke survivors with sensory deficit with remote vibrotactile noise 'on'

Monofilament scores for stroke survivors with tactile sensory deficit significantly improved with remote vibrotactile noise overall (Figure 26 and Appendix D, ANOVA, noise main effect with $p<.05$). Monofilament score was significantly dependent upon noise and location (ANOVA with $p<.05$), but not the interaction between noise and location (ANOVA with $p>.05$). On average, Monofilament scores improved by 4% with vibrotactile noise and Monofilament score was observed to decrease for the index fingertip, thumb fingertip, and the palm of the hand near the index finger, but did not significantly affect sensation at the thenar eminence location.
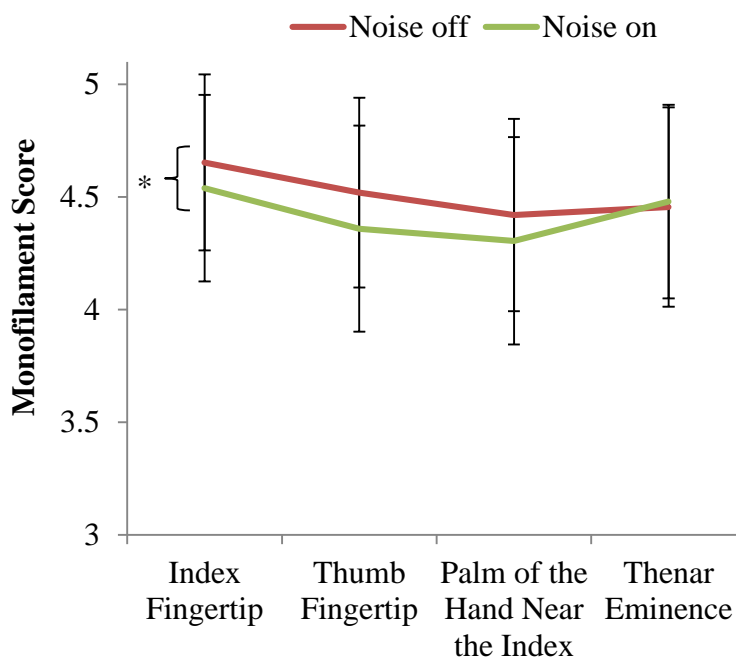
Figure 26: Mean ± SE Monofilament score was overall significantly reduced for the stroke survivors with tactile sensory deficit when vibrotactile noise was present.

5.4. Discussion

Similar to previous findings, stroke survivors with tactile sensory deficit gripped with the largest phalanx force deviation compared to both stroke survivors without tactile sensory deficit and healthy controls. The new finding of this study is that remote vibrotactile noise decreased phalanx force deviation for all of the subject groups. This improvement in phalanx force direction was associated with improved hand tactile sensation for the fingertips and the upper palm with remote vibrotactile noise. Changes in phalanx normal force or muscle activity did not accompany the improvement observed in phalanx force deviation.

5.4.1 Proposed mechanism for finger motor control and sensation improvement

Improving the tactile sensation via remote subsensory vibrotactile noise for the fingertips and the upper palm could have improved phalanx force deviation by facilitating greater feedback in the closed-loop motor control. For instance, increasing tactile sensation could have given individuals more information regarding grip surface characteristics, such as slipperiness of the object surface (Johansson and Westling 1987). Also improving tactile sensation could have provided individuals with greater feedback on how the magnitude and direction of the phalanx force being produced (Augurelle et al. 2003; Blennerhassett et al. 2007; Cole 2006; Hermsdorfer et al. 2003; Monzée et al. 2001; Robertson and Jones 1994) and more information on the position of their phalanx

with respect to the object surface (Monzée et al. 2001). Greater sensory feedback could have improved phalanx force control, reducing the deviation of phalanx forces.

The vibrotactile noise at the wrist could have increased tactile sensation of the fingertips and upper palm by increasing the sensory neurons' excitability via interneuronal overlap between the wrist and hand areas at the spinal or supraspinal level. Increased cortical and spinal activity with the application of vibrotactile noise on the hand has previously been observed in both humans and cats (Manjarrez et al. 2002a; Manjarrez et al. 2003). For instance, when vibrotactile noise added to one portion of the paw (central hindpaw), it was found that noise increased spinal and cortical activation representing another portion of the cat's paw (the third hindpaw digit), showing remote vibrotactile noise (Manjarrez et al. 2003). In addition, when the spine was transected from the brainstem of the cat, this increased activity was absent at the cortical level but still present in the spinal cord, showing the presence of remote vibrotactile noise at the spinal level (Manjarrez et al. 2003). Cortically, overlap in areas responsible of activation for the wrist, palm, and fingertips has been observed for owls and squirrel monkeys (Merzenich et al. 1983) and humans(Sanes et al. 1995), providing further evidence of how vibrotactile noise at the wrist could have affected tactile sensation at the palm and fingertips in the humans in this study. Furthermore, a recent pilot study has shown that applying subthreshold remote vibrotactile noise at the wrist during application of a tactile stimulation to the fingertip, resulted in evidence of increased brain activity, increased sensory feedback, and greater sensorimotor integration and processing for the tactile stimulation (Hur et al. 2013; Tseng 2013). Given the previous evidence of vibrotactile

noise's effects in the spinal and cortical areas, the results from this study are important because they show that applying vibrotactile noise remotely can improve tactile sensation and thus motor control.

In addition to finding the improved tactile sensation at the fingertips, this study also found improved tactile sensation for the upper palm region but not for the thenar eminence location. The fingertips and the upper palm region near the index finger are all innervated by the palmar digital braches of the median nerve, while the thenar eminence region is innervated by the palmar branch of the median nerve, radial nerve, and the musculocutaneous nerve (Netter 1997). It could be that application of the remote vibrotactile noise on the dorsal wrist tended to affect areas mediated by the palmar digital branch of the median nerve. Thus, remote vibrotactile noise at the wrist could have worked through a direct nerve connection, in addition to the potential increased sensation via increased spinal and cortical activity.

It is unlikely, that tactile sensation improved distally by the vibrations traveling through the skin from the wrist to the sensation locations because the vibration significantly attenuates across the skin. It has previously been shown that mechanical vibration loses approximately 90% of its original power when traveling a distance of 1 to 2 cm on the skin (Kurita et al. 2011; Manfredi et al. 2012). Also, if vibrations traveled through the skin to improve tactile sensation at the fingertips, then the largest improvement of tactile sensation would have been expected at the thenar eminence.

However, no significant improvement in tactile sensation was observed for the thenar eminence, making it unlikely that the mechanism of tactile sensation improvement is through vibrations traveling across the skin.

The findings for tactile sensation improvement with remote noise for this study and the previous study (Chapter 4) are similar with tactile sensation improvements seen in other studies with the *direct* application of vibrotactile noise. Previous studies have found improvement in fingertip tactile sensation (Kurita et al. 2011) and motor control (Mendez-Balbuena et al. 2012) when vibrotactile noise was applied *directly* to the fingertip. This study showed similar findings in the finger/hand sensation and finger phalanx force control even when the vibrotactile noise was applied *remotely* at the dorsal wrist.

5.4.2 Lack of effect of remote vibrotactile noise on phalanx normal force and muscle activation

Although phalanx force direction improved with vibrotactile noise, phalanx normal force and muscle activity were not significantly affected. The change in direction was approximately 1-2 degrees for each of the subject group. Therefore, there was very little expected change in phalanx normal force (<1%) and muscle activity. In addition, muscle activity was only recorded for three muscles: the FDS, EDC, and index fingers' FDI muscles. Since all the muscles of the fingers are important for controlling force in a

particular direction (Kutch and Valero-Cuevas 2011; Valero-Cuevas et al. 2000) including the flexor digitorum profundus (FDP), the lumbricals (LUM), the palmer interosseous (FPI), the index finger's extensor indicis (EI), as well as the thumb finger's flexor pollicis longus, extensor pollicis longus and brevis, abductor pollicis longus, the opponens pollicis, the abductor pollicis brevis, the flexor pollicis brevis, and the adductor pollicis, it is possible that the examination of only three muscles may not have been sufficient to capture the way the muscle activation pattern changed with remote vibrotactile noise toward improved phalanx force control. Also, improved phalanx force deviation could have occurred by improved posture of the phalanges such that the phalanges could be oriented with its resultant force perpendicular to the surface during power grip with noise, although experimental evidence for postural change was not obtained in this study. Regardless of a lack of empirical evidence for change in FDS, EDC, and FDI muscle activation or posture with the application of vibrotactile noise, all subject groups were able to improve phalanx force deviation during power grip.5.4.3 Study Limitation

Tactile sensation improvement was only measured for the strokes survivors with tactile sensory deficit and was not measured for the stroke survivors without tactile sensory deficit or healthy controls due to a bottom ceiling effect of the Monofilament test. Individuals in those groups already detected the lowest Monofilament size before noise was applied. However, it is expected that tactile sensation improved for the stroke survivors without tactile sensory deficit and healthy controls based off of previous studies that showed tactile sensation improvement with remotely applied vibrotactile noise for

tactile sensory healthy individuals (Hur et al. 2014; Lakshminarayanan et al. 2015; Wang et al. 2014).

5.4.4 Functional and Clinical Implications

Phalanx force deviation was reduced with vibrotactile noise for stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and age-matched controls and could reduce the risk of dropping objects. Previously stroke survivors, especially stroke survivors with tactile sensory deficit, were found to have increased phalanx force deviation compared to healthy controls (Chapters 2-3). Increased phalanx force deviation has previously been shown to lead to finger slippage in pinch grip (Seo et al. 2010) and can lead to object slippage in the hand if the deviations exceed the cone of friction for that surface (MacKenzie and Iberall 1994; Seo et al. 2010). Therefore, reducing phalanx force deviation with vibrotactile noise could improve object stability in the hand and could reduce the risk of dropping objects, especially for those stroke survivors with tactile sensory deficit whose phalanx force deviations are closer to the slip threshold limit.

Improvement in phalanx force deviation was significant, yet small for both stroke survivor groups. However, this small improvement in phalanx force control was seen immediately with stimulation. Even though tactile sensory feedback is important for finger force control (Blennerhassett et al. 2007; Johansson 1996; Zatsiorsky and Latash

2004b), it could be that there is a ceiling effect for improvement in stroke survivors'

finger force control regardless of the interventions due to damage occurring at the

corticospinal tract (Lang and Schieber 2004b) or for those stroke survivors who

experienced a high level of muscle atrophy in the hand post stroke (Dattola et al. 1993;

Dietz et al. 1986; Hafer-Macko et al. 2008; Hu et al. 2007; Landin et al. 1977).

Therefore, the degree of phalanx force control improvement for stroke survivors may be

limited due to neurological damages that occur post stroke. Furthermore, the

improvement in phalanx force deviation for healthy individuals was significance, yet

small potentially because healthy individuals are already operating close to their optimum

neurological capacity for grip force control.

Even though the improvement was small, the improvement in phalanx force

control was seen immediately with the application of vibrotactile noise to the wrist. A

greater improvement in phalanx force deviation for stroke survivors could occur with

long-term exposure and therapy using vibrotactile noise. There is evidence that

application of remote vibrotactile noise increases brain activity and causes greater

sensorimotor integration and processing (Hur et al. 2013). Over the course of long term-

exposure and therapy, it is possible that vibrotactile noise could potentially reverse some

cortical damage. Furthermore, remote vibrotactile noise applied over a long period of

time within an intensive rehabilitation training paradigm could elicit greater phalanx

force control improvement.

The results in this study can be applied to the development of a wearable therapeutic wrist band device for stroke survivors. The novelty of such a device is that it can improve *motor control* in addition to *tactile sensory* feedback without impeding natural range of motion of the fingers. Also, such a device could improve efficiency in rehabilitation therapy sessions by reducing the need for multiple intervention techniques to target motor control and tactile sensation separately and could be beneficial to motor therapies that already aim at regenerating tactile sensory and motor connections simultaneously. For instance, the constraint-induced movement therapy has been found to be effective in stroke hand recovery (Kunkel et al. 1999) and also promotes cortical reorganization (Liepert et al. 2000). In addition, improving tactile sensory feedback could encourage cortical reorganization for stroke survivors with tactile sensory deficit. Sensory feedback assists in the preservation of the normal cortical representations of both the motor and sensory cortex (Weiss et al. 2004). Therefore, stroke survivors with tactile sensory deficit could have altered cortical sensorimotor representations leading to the altered muscle activation and their diminished force control, and improving tactile sensory feedback via a training paradigm with remote vibrotactile noise could encourage shifts towards the normal sensorimotor brain mapping. Adding subsensory remote vibrotactile noise to a sensory-motor training paradigm could facilitate functional and tactile sensation recovery.

A wearable device that has the potential to improve tactile sensory and motor control can be especially important during the immediate recovery time after a stroke. Immediately after a stroke, there is a considerable amount of brain reorganization

occurring in the first 4 months post stroke (Cicinelli et al. 1997). Furthermore, it has been

postulated that sensory reorganization may occur before motor reorganization after stroke

(Weiller 1998) and is critical in motor recovery (Tyson et al. 2008). Because the

mechanism of motor and sensory improvement with the remote vibrotactile noise is

proposed to occur through increased brain activity and sensori-motor processing and

integration (Hur et al. 2013), this wearable device could further promote neural

regeneration during this critical time

5.4.5 Future Directions

Remote vibrotactile noise was found to improve fingertip motor control of the

phalanx force deviations during a static power grip. However, this study did not examine

the effect of remote vibrotactile noise on dynamic grip control and movement.

Furthermore, it is unknown if the improvement changes seen in the phalanx deviation can

lead to improvement object manipulation. In the future, the effect of this noise on

dynamic movement will be examined during a dynamic task that requires both precision

manual handling and tactile sensory feedback such as the Box and Block Test

(Mathiowetz et al. 1985a).

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5.5. Conclusion

Remote subthreshold vibrotactile noise applied on the dorsal wrist improved phalanx force deviation during static maximal power grip for healthy controls, stroke survivors without tactile sensory deficit, and stroke survivors with tactile sensory deficit. Remote vibrotactile noise also improved hand tactile sensation for stroke survivors with tactile sensory deficit. Reducing phalanx force deviations can lead to a reduced chance of object slippage. This study is clinically important since improving tactile sensation and motor control remotely could be designed into a wristband-like wearable device to improve gripping for stroke survivors. Improving motor control post stroke can lead to independence in completing daily living activities.

Chapter 6: Effects of subsensory remote vibrotactile noise on stroke survivors' dynamic grip[3]

ABSTRACT

Remote subsensory vibrotactile noise applied to the wrist has previously been shown to improve fingertip and upper palm tactile sensation and phalanx force control during static power grip. This study examined if stroke survivors' clinical hand grip function could be improved. Ten stroke survivors with tactile sensory deficit performed the Nine Hole Peg Test (NHPT) and the Box and Block Test (BBT) with remote subsensory vibrotactile noise applied to the dorsal wrist. Tactile sensation was also recorded with and without noise. Results showed that stroke survivors improved their BBT score by 2% and improved their time to complete the NHPT by 14% ($p<.05$). Vibrotactile noise at the wrist improved motor control of the hand potentially by increased spinal and/or cortical motor and sensory activity. Tactile sensation of the fingertips did not improve with noise potentially due to the prolonged exposure to noise and the adaptation of the tactile afferents, or direct effects to motor control without perceptual changes. Regardless, remote subsensory vibrotactile noise could be a useful rehabilitation tool used to improve stroke survivors' ability to manipulate objects during daily living, encouraging long term functional recovery and making it a promising rehabilitation tool.

6.1. Introduction

Many stroke survivors experience somatosensory deficit (Carey 1995) in addition to motor deficit in their hands and arms (Gray et al. 1990; Nakayama et al. 1994; Parker et al. 1986). Stroke survivors have been previously been observed to move arms and hands slower (Schaefer et al. 2012), with reduced coordination (Cirstea et al. 2003), delayed proactive and reactive responses to perturbations (Grichting et al. 2000; Hermsdorfer et al. 2003), and excessive force during safety margin tasks (Nowak et al. 2003) compared to healthy individuals. Impaired tactile sensory feedback further contributes to stroke survivors' reduced phalanx force control (Chapter 2-5) and impaired manipulation (Hermsdorfer et al. 2003). Reduced ability to perform object manipulation tasks may hamper stroke survivors' ability to complete daily living tasks.

Increasing tactile sensation of the fingertips and the upper palm via remote vibrotactile noise (Chapter 3-4) has previously been shown be associated with improved phalanx force control for stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and healthy controls (Chapter 5). Vibrotactile noise applied remotely may improve fingertip and upper palm sensation and motor control of the fingers via increasing the excitability of the tactile sensory neurons in the central nervous system through stochastic resonance and interneuronal connections (Hur et al. 2013; Manjarrez et al. 2003; Manjarrez et al. 2002b; Tseng 2013). Specifically, the application of remote subsensory vibrotactile noise at the wrist has shown evidence

during a pilot study of increased brain activity, increased sensory feedback, and greater sensorimotor integration and processing for a tactile stimulation at the fingertip (Hur et al. 2013; Tseng 2013). Therefore, remote subsensory vibrotactile noise has been shown to increase tactile sensation and hand motor control via increased excitability in the central nervous system and could be a useful rehabilitation device for stroke survivors. However, the previous investigation on how remote vibrotactile noise impacts motor control only determined the effect of noise on a static grip force task requiring a maximum grip effort. It is currently unknown if remote subsensory vibrotactile noise can result in an improved ability to complete a dynamic movement task as well. Therefore, the effect of remote vibrotactile noise on dynamic movement control should be investigated.

The goal of this study was to determine the effect of remote vibrotactile noise on stroke survivors' ability to perform dynamic movement tasks. Specifically, the objective of this study was to determine the effect of remote subthreshold vibrotactile noise on stroke survivors' ability to complete the Box and Block Tests (BBT) and the Nine Hole Peg Test (NHPT). Additionally, to determine if an improvement in the ability to perform a dynamic gripping task was accompanied by an improvement in sensation, the effect of remote vibrotactile noise on the fingertip tactile sensation was examined.

6.2. Methods

6.2.1 Subjects

Ten chronic stroke survivors (mean ± standard deviation (SD) age of 63 ± 9 years), with fingertip tactile sensory deficit, participated in the study (Table VI). Mean ± SD motor impairment, quantified by a hand and wrist subdivision of the Fugl-Meyer Assessment (Fugl-Meyer et al. 1975), was 21 ± 4 (out of a possible 24) for those who completed both the BBT and NHPT. All stroke survivors were at least 6 months post stroke. All subjects underwent the Semmes-Weinstein monofilaments test (Bell-Krotoski et al. 1993). Sensation deficit was based on a baseline Monofilament score of > 2.83 (Dellon 1997) for both the index and thumb finger (Table VI). Monofilament scores for the stroke survivors with sensory deficit group ranged from 3.22 to 6.65 with a median score of 3.61. All subjects signed a written consent form and followed protocol approved by the Institutional Review Board.

Table VI: Subject Demographics

Subject	Time since most recent stroke (months)	Type of Stroke	Hand Dominance*		Paretic Side	Sex	Age (years)	Fugl-Meyer Score (out of 24)	Chedoke McMaster Score (out of 7)	Baseline Monofilament Score	
			Pre Stroke	Post Stroke						Index	Thumb
1	24	Hemorrhagic	R	R	L	M	60	22	7	3.61	3.61
2	6	Unknown	R	R	L	M	62	16	6	3.61	3.61
3	16	Ischemic	L	R	L	F	62	16	5	6.65	6.65
4	7	Hemorrhagic	R	R	L	F	52	24	7	3.61	3.61
5	15	Hemorrhagic	R	R	L	M	59	22	5	3.22	3.22
6	7	Unknown	R	R	R	M	55	14	6	3.22	3.22
7	3	Hemorrhagic	R	R	R	M	81	23	7	4.31	4.17
8	2	Hemorrhagic	R	R	L	M	66	24	7	3.22	3.22
9	22	Ischemic	L	L	R	M	60	24	7	3.61	3.61

*R= "Right hand dominance", L= "Right hand dominance"

6.2.2 Functional Tests

Subjects were asked to perform the BBT and the NHPT with and without vibrotactile noise applied to the wrist (Figure 27). Due to the difficultly level of the tasks, one participant was unable to perform the BBT and two subjects were unable to complete the NHPT. The BBT and the NHPT provide a reliable measurement of manual dexterity (Chen et al. 2009; Desrosiers et al. 1994) and were administered according to the literature (Mathiowetz et al. 1985a; b; Oxford Grice et al. 2003a; b). The BBT score was calculated as the total number of blocks moved from one box, across a wooden barrier, to another box in 60 seconds (Mathiowetz et al. 1985a). The score from the NHPT was calculated as the total time subjects picked up, placed, and removed nine pegs from nine holes (Oxford Grice et al. 2003b).
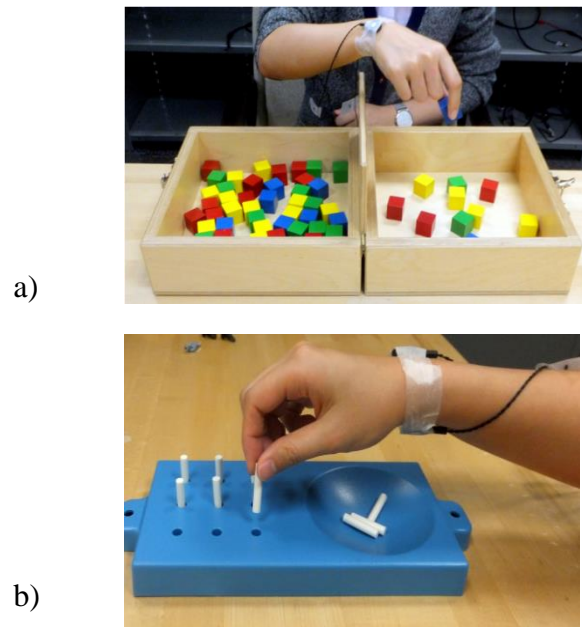
Figure 27: Subjects' performed hand motor tasks using the Box and Block Test (a) and the Nine Hole Peg Test (b)

6.2.3 Vibrotactile noise

Remote subthreshold vibrotactile noise was applied on the dorsal and volar wrists of the stroke subjects using C-3 Tactors (Engineering Acoustics, Inc, Casselberry, FL). Similar to our previous studies (Enders et al. 2013; Enders and Seo 2014), the remote subthreshold vibrotactile noise was white noise bandwidth filtered at 0 to 500 Hz and set to an intensity of 60% the sensory threshold for sensing vibration for the wrist dorsal and wrist volar location, individually. This intensity has previously been shown to be effective in improving fingertip tactile sensation (Enders et al. 2013; Wells et al. 2005) and phalanx force deviation (Chapter 5). Similarly, the previous study showed improvement in sensation and phalanx force deviation when the vibrotactile noise was applied to the wrist area (Chapters 4-5) (Enders et al. 2013). These functional tests were repeated in four blocks where the vibrotactile noise on was turned 'off' for the first and

final block, and turned 'on' for the second and third block of testing. Total exposure time
to the vibrotactile noise was the time to complete a block, around 15-25 minutes. A
practice block was given prior to testing to limit learning effects.

6.2.4 Statistical Analysis

A Kruskal-Wallis test was used on the multivariate data to determine if BBT and
NHPT score significantly varied with and without noise across all subjects. There was no
statistical difference between the first and the last blocks of testing with noise 'off' and
also between the second and third blocks with noise 'on' (Mann-Whitney U test with
$p<.05$ for both). Thus, the blocks were not included as a factor in the Kruskal-Wallis
analysis. In addition, an ANOVA was used to measure if fingertip tactile sensation, as
measured by the 20-set of Semmes-Weinstein monofilaments, improved with and without
subthreshold vibrotactile noise. Monofilament Test scores (ranging from 3.22 to 6.65)
were converted to the corresponding estimated logarithmic bending force (ranging from
.16 to 300 grams) for the statistical analysis.

6.3. Results

Subthreshold vibration significantly improved stroke survivors' ability to perform
the BBT and the NHPT (Kruskal-Wallis, $p<.05$). With remote subthreshold vibrotactile
noise, stroke survivors improved BBT score by 4% (an average of 1-2 blocks)(Figure 28a

and Appendix D). Seven out of the 9 subjects who performed the BBT improved their

score by an average of 7%, while two subjects decreased their BBT score by 2% (Figure

28c,e and Appendix D). With remote vibrotactile noise, stroke survivors improved their

ability to complete the NHPT by 9 seconds (14%), on average (Figure 28b and Appendix

D). Improvement on the NHPT was observed for seven out of the eight subjects who

were able to complete this task (Figure 28d,f and Appendix D). Mean ± SE monofilament

score, did not significantly change (4.2 ± 0.3 to 4.1 ± 0.4) with remote vibrotactile noise

(ANOVA with $p>.05$).
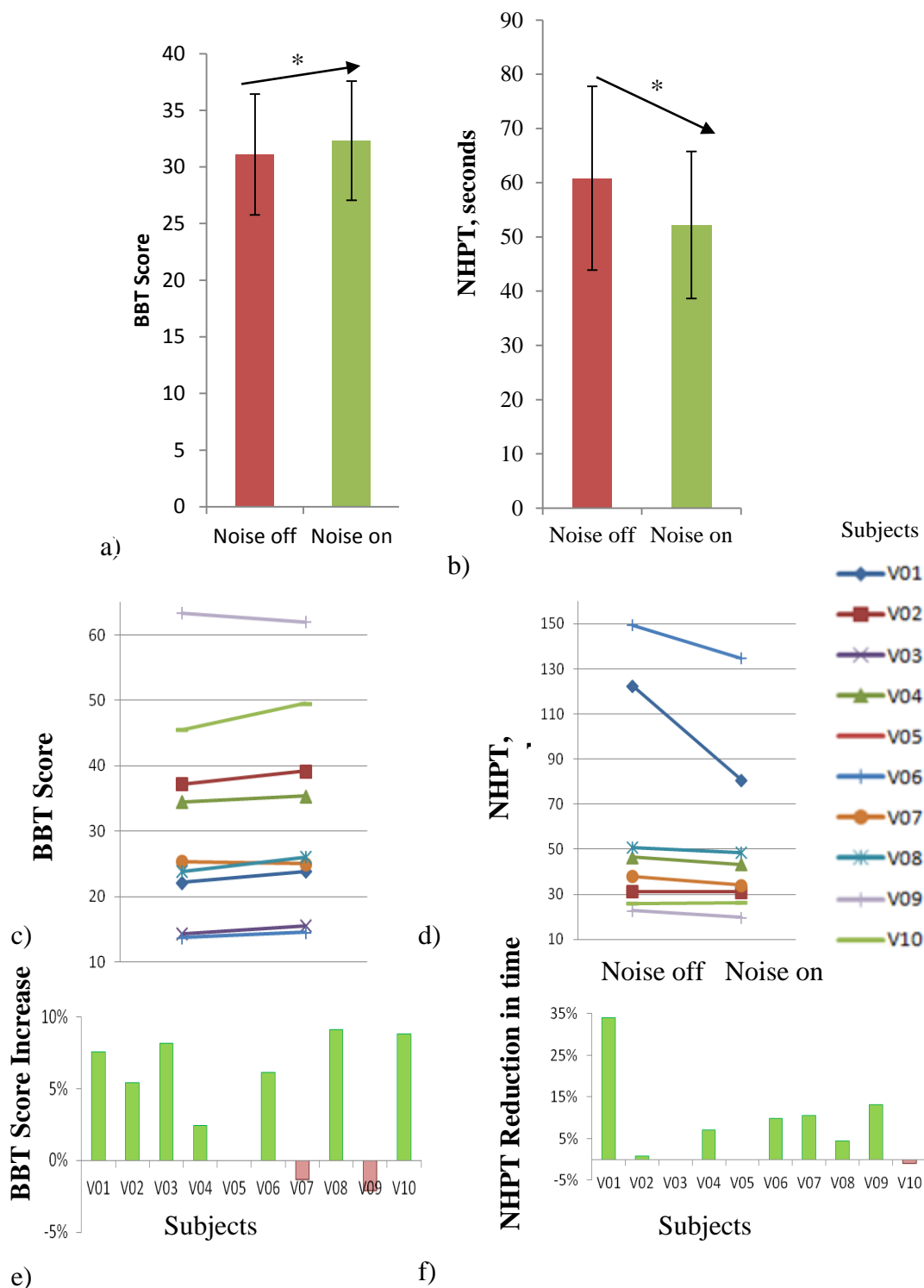
Figure 28: Mean ± Standard Error (SE) BBT score (a) and NHPT time (b) significantly improved with the application of remote vibrotactile noise. Individual subjects' data is shown for the BBT scores and NHPT in c) and d), respectively. Individual subjects' percent change in the BBT score and the time to complete the NHPT is shown in e) and f), respectively.

6.4. Discussion

Manual dexterity improved with remote subthreshold vibrotactile noise for stroke survivors with sensory deficit. This study shows that subthreshold vibrotactile noise can improve stroke survivors' ability to complete a dynamic task that involves grasping objects and moving them to either a broad (BBT) or precise (NHPT) target area. Monofilament score did not improve with prolonged (15-20 minutes) application of the remote vibrotactile noise.

6.4.1 Potential mechanism of motor dexterity improvement

This research study confirms previous research that remote vibrotactile noise targeting the sensory system has effects in the motor system. Previously, it has been found that when subsensory noise was added directly to the fingertip, improvements were also seen in motor control system (Galica et al. 2009; Hur et al. 2014; Mendez-Balbuena et al. 2012; Priplata et al. 2006). For instance, subsensory vibrotactile noise applied to the fingertip has shown improvement in controlling finger position (Mendez-Balbuena et al. 2012). Also, subsensory vibrotactile noise applied to the lower extremity has been shown to reduce variability in gait measures and balance (Galica et al. 2009; Gravelle et al. 2002; Priplata et al. 2002; Priplata et al. 2006), by increasing tactile sensory and proprioception feedback. In addition to direct vibrotactile noise, remote application of

subsensory noise on the forearm has been shown to improve muscle reaction time and stabilization of a handle after perturbation (Hur et al. 2014).

The mechanism of improvement could be the result of increased cortical or spinal activation, previously shown to occur with vibrotactile noise (Manjarrez et al. 2002a; Manjarrez et al. 2003). Specifically, the subthreshold vibrotactile noise on the wrist may have increased somatosensory integration and processing for the whole hand area (Hur et al. 2013; Tseng 2013), increasing dexterity of the fingers. This is based off previous findings that applying subthreshold vibrotactile noise at the fingertip, increases tactile sensation at the fingertip location(Collins et al. 1996). Since increasing somatosensory feedback has been shown to increase cortical excitation and activation in the motor cortices (Kaelin-Lang et al. 2002), it could be that applying vibrotactile noise at the wrist increased motor activation and sensation for the whole hand-wrist area via increased cortical activation.

6.4.2 Lack of tactile sensory improvement seen after prolonged vibration noise exposure

Contrary to previous studies, monofilament score did not significantly improve with the subsensory vibrotactile noise, potentially due to adaptation. In the previous studies (Chapters 4-5) (Enders et al. 2013), sensation was examined after the subject threshold vibrotactile noise was applied for only a short time (~1 min). In the present study, sensation was measured after a much longer exposure to the remote vibrotactile

noise (approximately 15-20 minutes). It could be that fingertip tactile sensation improved

when the vibrotactile noise was first applied and then reached a point of adaptation,

which has been observed with prolonged exposure to stimulation (Dinse and Merzenich

2002). Regardless, improvement in manual dexterity continued without the measurable

improvement in tactile sensation. It is also possible that an improvement in functional

performance occurred through bypassing stroke survivors' tactile sensory impairment via

the visual feedback provided during the task (Ellis et al. 2005; Seo et al. 2011), paired

with remote vibrotactile noise.

6.4.3 Clinical Applications of Remote Subsensory Vibrotactile Noise

Because of the improvements observed in this study and previous research

studies, remote subsensory vibrotactile noise has a promising future as a rehabilitation

device for stroke survivors. Previous methods of sensory enhancement using intense

levels of transcutaneous electrical nerve and muscular stimulation and high volume

vibration of forearm muscles have shown improvements in hand and arm function for

stroke survivors (Chae et al. 1998; Conforto et al. 2007; Santos et al. 2006; Yozbatiran et

al. 2006). However, the intensity of the stimulations needed for these devices to produce

the desired effects, often leads to discomfort, making them less practical in terms of a

long-term rehabilitation method. Remote vibrotactile noise is subsensory and non-

detectable to a user, making it a comfortable tactile sensory enhancement technique.

Furthermore, one of the benefits of remote subsensory vibrotactile noise is that it is away

from restricting movement of the fingers during gripping, allowing stroke survivors to

wear such as device during normal rehabilitation therapy or throughout the day. Also due to the vibrotactile noise being below the sensory threshold, discomfort of wearing such a device for a long exposure is limited unlike other types of motor enhancing stimulations (Chae et al. 1998).

6.5. Conclusion

Stroke survivors' manual dexterity, measured by the BBT and the NHPT, improved significantly with the application of remote subsensory vibrotactile noise to the wrist. Therefore, remote subsensory vibrotactile noise appears to improve motor control potentially due to increased sensory and motor cortical activation. This technology could be applied as a wearable rehabilitation device to promote motor recovery post stroke and increase stroke survivors' ability to complete daily living activities in the long term.

Chapter 7: Entrepreneurial activity

1. TheraBracelet, LLC.

A timeline portraying the sequence of entrepreneurial events is shown in Figure 29. In October of 2012, an invention disclosure to UWM was filed for the technology idea of applying remote vibrotactile stimulation as a rehabilitation device. Then in April 2013, UWM Research Foundation filed a provisional patent on behalf of Dr. Na Jin Seo and I. Over the next couple months, companies were interviewed for the opportunity to market and develop the rehabilitation device. One of these groups was a student group from The University of Louisville's Entrepreneurship MBA program. This program allows for a group of MBA students in their final year of the program to seek out an invention idea and market this idea on a national and world-wide business venture capital circuit. I was also requested to officially become part of the competition team and became a visiting student in their program. A license option agreement was signed between Matthew Raggard, Kacie Neutz, Nicholas Phelps, myself, and the University of Wisconsin Research Foundation to take the invention, named TheraBracelet, to market on the competition circuit. In October of 2013 an Operating Agreement was signed that outlined how the company will conduct business, how the individual financial and managerial responsibilities are divided, the equity shares, and how the dissolution of the company would be conducted if such would occur. TheraBracelet, LLC became an official and legal company entity in October 2013, with Matthew Raggard as CEO, Kacie Neutz as COO, Nicholas Phelps as CFO and Leah Enders as CSO (therabracelet.com).

During this time, a business plan, an executive summary, and a business plan presentation was developed for presenting TheraBracelet to the competition circuit.
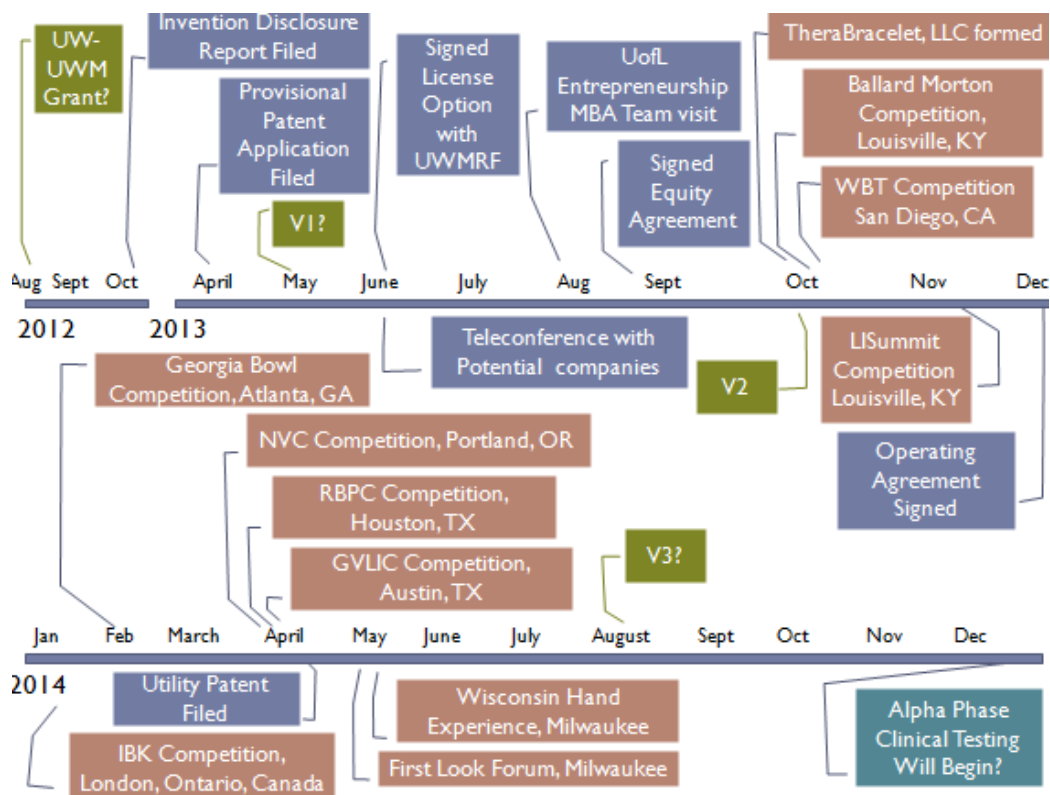
Figure 29: Timeline of Entrepreneurial activities with TheraBracelet from 2012-2014. Green squares are associated with prototype development, blue squares are associated with UWM Research Foundation activities, and red squares are associated with Therabraclet, LLC activities.

From October 2013 until April 2014, TheraBracelet, LLC attended seven competitions held in the United States and Canada. TheraBracelet, LLC placed third in the final competition at the Global Ventures Labs Investment Competition in Austin, TX and closed out the competition circuit with over $110,000 (alliance.rice.edu/2014_RBPC_Winners). The largest of these earnings was $100,000 from an investor, The Mercury Fund, who will continue to support TheraBracelet as we

move into our clinical testing of the device on patients. My personal responsibility as CSO is to communicate the technology behind TheraBracelet and the research findings in non-technical terms during presentations, tradeshow events, and investor meetings. I was also responsible for assembling the investor packet that includes raw data and detailed finding explanations for the Mercury Fund.

2. TheraBracelet Prototype Development

Coinciding with this time period, a UW-Madison UW-Milwaukee Inter-institutional Research Grant between Dr. Na Jin Seo of UW-Milwaukee and Dr. John Webster of UW-Madison allowed for the development of a working prototype. Funding for prototype development was also provided by an WiSys Technology Foundation, Inc's Applied Research Grant (ARG) to Dr. Seo and Dr. Webster. This prototype was built by Dr. John Webster and his graduate student, Fa Wang following the conceptual and functional design outlined by Na Jin Seo and I. Working together with input from fellow researchers, TheraBracelet has developed into a device that is nearly ready for use in clinical testing.

2.1 Laboratory Version

The initial device that was used throughout the research studies was a C-3 Tactor (Engineering Acoustics, Inc. Casselberry, Florida) (Figure 30b) that required an

additional mp3 playing device (Figure 30a). The negative with this setup was the

vibration amplitude of the C-3 vibrotactile device was too small and some patients were

unable to feel to the maximum amplitude.

Figure 30: The C-3 Tactor (Engineering Acoustics, Inc. Casselberry, Florida) was used for all stimulation research studies. Characteristics of the device were not conducive for a proper therapeutic device.

2.2 TheraBracelet Version 1

The first prototype for TheraBracelet was an electromagnet transducer that

oscillated a small metal plate against the skin, and fed through a sweat band cuff (Figure

31). An mp3 playing device was still needed to play the sound file that transmitted the

noise signal. The problems with this version were that the house box for the circuitry was

bulky (Figure 31b), the small metal plate was against the skin and held in place against

the cuff fabric with superglue (Figure 31c), the small metal plate was detachable and

easily lost (Figure 31d), and the vibration amplitude was still not strong enough for some

stroke survivors to feel. Although the battery was too large, the life was sufficient (~8 hrs

for all day use) (Figure 31b). In addition, this device was not fMRI compatible for some

concurrent fMRI studies.
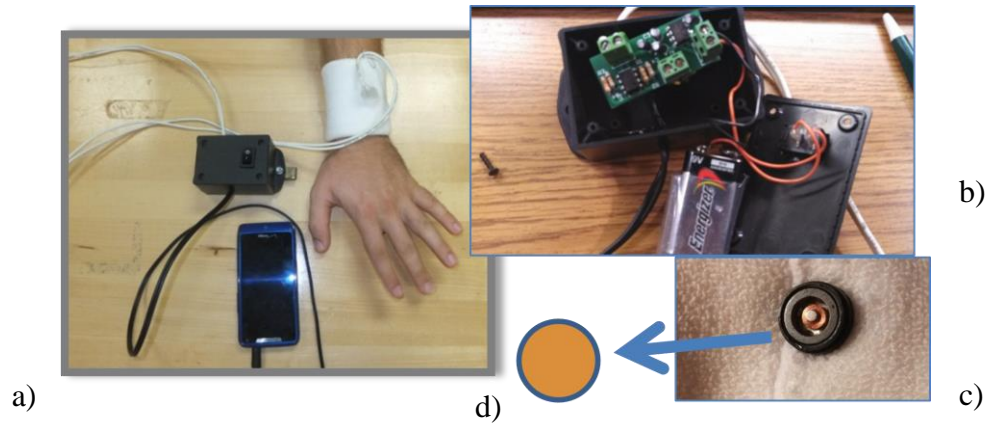
Figure 31: TheraBracelet Version 1 (a) with large, inconvenient battery and bulky circuitry box (b), electromagnetic vibrators (c), and a detachable metal plate (d).

2.3 TheraBracelet Version 2

The second TheraBracelet prototype built by Dr. John Webster and Fa Wang was

piezoelectric (detached could used for the fMRI setup), used a small, easy replaced 3.7V,

the piezolelectric could be embedded into the cuff between layers so as not to come into

direct contact with skin, and the circuitry was much more compact. The problems with

this version was that this device was still dependent upon an external mp3 device, the

tactor was still not strong enough to be felt by some individuals, the battery life (~6) hrs

w as insufficient for long-term use, the circuitry needed secure housing, the electrical

connections were unreliable and repairs were difficult.
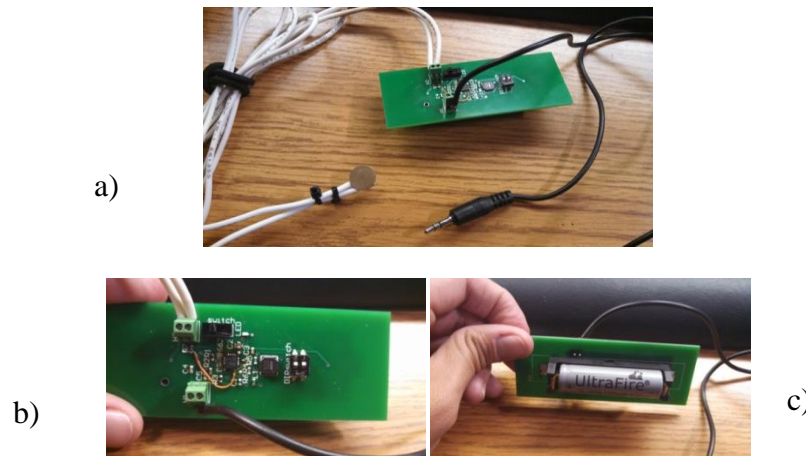
Figure 32: TheraBracelet Version 2 uses a piezoelectric vibrator (a) with small,
unprotected circuitry box (b), and an easily replaced battery (c).

2.4 TheraBracelet Version 3

The third TheraBracelet prototype has a portable controller box that contains a

noise file that can be easily controlled from the controller buttons on the box (Figure 33a)

and an electromagnetic transducer that has the metal plate securely fixed (Figure 33b-c).

The TheraBracelet Version 3 allows for the person to find the sensory threshold and then

adjust the level of the noise. Also the battery is easily replaced on the back of the

controller box and the connections between the electromagnetic vibrator is secure both on

the vibrator and the controller box end. The current problems currently being addressed

with this version are that the control box is still too large for comfort, there are more

buttons on the control than needed and cause confusion, the battery life (~4 hrs) is still

insufficient, and the plate secured on the electromagnetic device is able to be bent and broken easily.
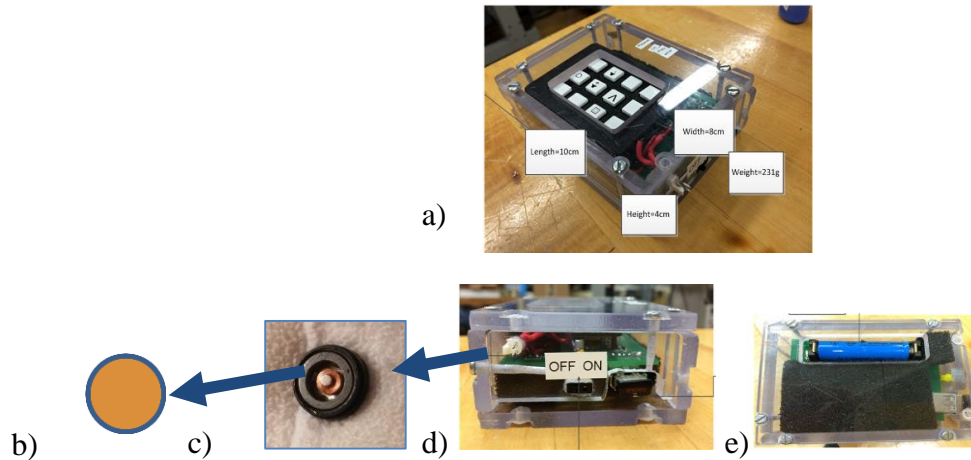
Figure 33: TheraBracelet Version 3 has a independently controlled noise file that is enclosed in a protective case (a), uses an electromagnetic piezoelectric vibrator (c) with the plate attached (b), an external on/off switch (d), and an easily replaced battery (e).

2.5 TheraBracelet Final Desired Version

The final TheraBracelet product will be able to be worn completely on the wrist and include the vibration device, controller, sound file, and battery all in one housing unit similar to a watch (Figure 34). Users will be able to adjust the noise level of the device using an interface that walks users through finding their sensory threshold. In addition, the device will have a long battery life (>8 hrs for all day use) and the battery will be rechargeable by plugging in the device at night. The device will be lightweight (about the weight of a watch), durable, and waterproof.

Figure 34: The final desired TheraBracelet device with batter, amplitude adjusting

controlling, sound file, and battery all able to be housed on the wrist.

Chapter 8: Overall Conclusions

The main findings of this dissertation are that stroke survivors' power grip is altered compared to healthy individuals, especially for those who experience tactile sensory deficit, and enhancing sensation via remote vibrotactile improves grip control making it a promising rehabilitation technique. Improving rehabilitation therapies could improve stroke survivors' independence in completing daily activities.

After stroke, there is a considerable amount of complex brain reorganization (Dettmers et al. 1997; Liepert et al. 2000; Rossini et al. 1998). This reorganization often leaves decreased excitability of the motor cortex in the stroke-affected hemisphere and decreased cortical representation of the paretic limb (Liepert et al. 2000), leading to impaired peripheral neural control. Damage to the sensory cortex area can also lead to changes in the cortical representations of the paretic limb in the motor cortex (Nelles et al. 1999; Weiss et al. 2004). These cortical activation changes can result in altered activation of the muscles controlling grip, change paretic muscle fiber composition, impair sensory feedback from the paretic limb and sensory integration to other areas in the brain, and result in further disuse of the limb. Stroke induced tactile sensory deficit and altered muscle activation and could have contributed to the altered phalanx force control observed for stroke survivors (Figure 1), as described in the following several paragraphs.

Decreased phalanx force control via intrinsic muscle weakness

Increased phalanx force deviation could also have occurred due to altered muscle activation and atrophy specifically affecting the intrinsic muscle group of the hands over the extrinsic muscles. Specific weakness of the intrinsic muscles could reduced phalanx force control and lead to increased phalanx force deviation during power grip, due their role in stabilizing the MCP joint, producing flexion force, and directing fingertip forces (Stack 1962; Valero-Cuevas et al. 2000). The intrinsic hand muscles may be particularly affected by stroke compared to the extrinsic muscles, potentially due to their muscle fiber type composition and greater need for corticospinal activation. Specifically, intrinsic muscles have been shown have a greater concentration of Type II muscle fibers compared to extrinsic muscles (Hwang et al. 2013). Previous reports have shown that muscles composed predominantly of Type I fibers may be better preserved and muscles with a higher concentration of Type II muscle fibers may be more susceptible to atrophy in instances of increased age (Larsson et al. 1978; Lexell 1995), diabetes (Bus et al. 2002), and stroke (Dattola et al. 1993; Dietz et al. 1986; Hafer-Macko et al. 2008; Hu et al. 2007; Landin et al. 1977). Changes in muscle fiber composition post stroke occur not only due to limb disuse after stroke, but also due to changes in the corticospinal activation targeting specific muscle fiber types (McComas et al. 1973). In addition to greater potential atrophy of their muscle fibers, the intrinsic muscles may be further weakened compared to the extrinsic muscles post stroke, due to their need for greater corticospinal drive compared to the more proximal muscles of the arms (Palmer and

Ashby 1992; Turton and Lemon 1999). Therefore, the reduced cortical activity regarding the paretic limb, could have affected the intrinsic muscles to a greater extent compared to the extrinsic muscles controlling the hand. Specific weakness of the intrinsic muscles could have resulted in destabilization of the MCP joint, decreased flexion force, and misdirection of fingertip forces (Stack 1962; Valero-Cuevas et al. 2000).

Decreased phalanx force control via diminished tactile sensory feedback

Since tactile sensory feedback is critical in the control of phalanx force magnitudes and deviations needed during gripping (Johanson et al. 2001; Zatsiorsky and Latash 2004b), and it appears that stroke survivors' diminished tactile sensory feedback (Carey 1995; Turton and Butler 2001) decreased phalanx force control further, leading to the greater deviation. Since both stroke survivors groups were similar in terms of level of motor functional recovery, and the stroke survivors who experienced some level of tactile sensory deficit had significantly greater phalanx force deviation than those without sensory deficit, it can be proposed that sensory feedback from the fingers is a contributor in directing finger forces during power grip. Furthermore, the ability to approximate their 50% maximum grip effort was similar between both stroke groups, leading greater evidence that the reduced phalanx force control was due to differences in a deficit of tactile and not proprioceptive sensory feedback. Since tactile sensory feedback is important for updating the CNS for the adjustment of the grip forces to prevent object slippage (Johansson and Westling 1984), stroke survivors with tactile sensory deficit may be more at risk of dropping objects. In addition, research has shown how cortical

territories shift following the removal of sensory inputs, suggests that sensory feedback assists in the preservation of the normal cortical representations of both the motor and sensory cortex (Weiss et al. 2004). Therefore, stroke induced tactile sensory feedback could also have decreased representations of the paretic hand in the motor cortex, increasing the degree of altered neural control of the hand muscles, and cause the increase in phalanx force deviation observed for stroke survivors with sensory deficit.

Decreased phalanx force control via impaired grip posture

Although posture was controlled for in the context of this dissertation, impaired grip posture could also decrease phalanx force control during everyday activities for stroke survivors. Altered muscle activation has previously been shown to result in finger muscle coactivation (Kamper and Rymer 2001; Lang and Schieber 2004b) and spasticity (Mottram et al. 2009). Increased coactivation among the fingers' flexor and extensor muscles (Kamper and Rymer 2001) and the abduction/adduction muscles (Lang and Schieber 2004b) could have decreased stroke survivors' ability to achieve an optimal posture when gripping the device, leading to a decreased control of phalanx forces. Similarly, hyperexcitability of the stretch reflex causing involuntary muscle contraction and flexion of the fingers, known as spasticity (Bhakta 2000; Brown 1994), could also impede stroke survivors' ability to correctly coordinate finger force production, leading to improper posture, and increased phalanx force deviations.

Potential mechanisms of improved fingertip and upper palm tactile sensation and motor function via remote subsensory vibrotactile noise

This dissertation investigated how *remotely* applied subsensory vibrotactile noise improved fingertip and upper palm tactile sensation and motor control. This knowledge builds upon previous studies which have found similar improvements with *direct* application of subsensory vibrotactile noise (Collins et al. 1996; Kurita et al. 2013; Liu et al. 2002; Mendez-Balbuena et al. 2012; Wells et al. 2005). Subsensory vibrotactile noise applied *directly* to the fingertip has been shown to improve the transmission and detection of weak sensory signals (Collins et al. 1996) and improve the tactile sensation by directly stimulating the tactile receptors in the skin (Kurita et al. 2013; Liu et al. 2002; Wells et al. 2005). Furthermore, improvement in tactile sensation at the fingertips, with vibrotactile noise applied *directly* to the fingertip, has been shown to yield improvements in motor control (Kurita et al. 2013; Mendez-Balbuena et al. 2012; Priplata et al. 2002). For instance, subsensory vibrotactile noise applied to the fingertip has shown improvement in controlling finger position (Mendez-Balbuena et al. 2012). Also, subsensory vibrotactile noise applied to the feet has been shown to reduce variability in gait measures (Galica et al. 2009) and subsensory noise applied to both the knee and the feet has been shown to improve balance in healthy (Gravelle et al. 2002; Priplata et al. 2002; Priplata et al. 2006), by increasing tactile sensory and proprioception feedback. Increasing sensation peripherally also has impacts cortically, such as increased cortical excitation and activation in the motor cortices (Kaelin-Lang et al. 2002).

The effects of *remote* subsensory vibrotactile noise on tactile sensation and motor control have not been previously investigated. Previously, it has been shown that remote vibrotactile noise applied to the arterial baroreceptor on the neck of healthy individuals was able to improve pressure detection changes in the heart, detected by the cardiopulmonary baroreceptor, via the interaction of the two baroreceptors' neuronal inputs located in the brainstem (Hidaka et al. 2000). Subsensory vibrotactile noise applied to the forearm of healthy individuals elicited improvements of muscle reaction time and stabilization of a handle after perturbation of the handle (Hur et al. 2014). This dissertation research has shown that application of the remote vibrotactile subsensory noise to the wrist of stroke patients improved fingertip and upper palm tactile sensation (at least short term) and improved motor control of the hands. In addition, healthy controls' phalanx force control also improved with remote subsensory vibrotactile noise. The exact mechanism of how remote subsensory vibrotactile noise improves tactile sensation of the fingertips and upper palm and hand motor function is currently unknown.

Remote subsensory vibrotactile noise may have improved wrist sensation and then increased fingertip and upper palm tactile sensation and motor control via increased spinal and cortical excitation and increased corticomuscluar synchronization (Figure 35). Increasing tactile sensation peripherally also has impacts cortically, such as increased cortical excitation and activation in the motor cortices (Kaelin-Lang et al. 2002). Therefore, remote subsensory vibrotactile noise could have stimulated the tactile receptors in the skin at the wrist increasing sensation at the wrist (Kurita et al. 2013; Liu et al. 2002; Wells et al. 2005) and then increased motor control and tactile sensation at

the fingertips via increased cortical activity in the motor areas. The application of vibrotactile noise on the hand of humans and the paws of cats, has been shown to increase cortical and spinal neuronal activities, demonstrating the effect of noise in the central nervous system (Manjarrez et al. 2002a; Manjarrez et al. 2003). It has also been shown that vibrotactile noise applied to one portion of a cat's paw increased spinal and cortical activity for another portion of the cat's paw (Manjarrez et al. 2003), demonstrating how vibrotactile noise has the potential to *remotely* affect other areas on the same limb via increased activity in the central nervous system. Recently, a pilot study has shown that applying subsensory remote vibrotactile noise at the wrist during noise to the fingertip resulted in evidence of increased brain activity, increased sensory feedback, and greater sensorimotor integration and processing (Hur et al. 2013; Tseng 2013). In addition to increased cortical activity, remote vibrotactile noise may also increase the neuronal synchronization between spinal and cortical activity (Manjarrez et al. 2002b). Increased synchronization may facilitate neural communication between the spinal and cortical levels (Fell and Axmacher 2011) and improving corticomuscluar synchronization can lead to improved motor control (Mendez-Balbuena et al. 2012).
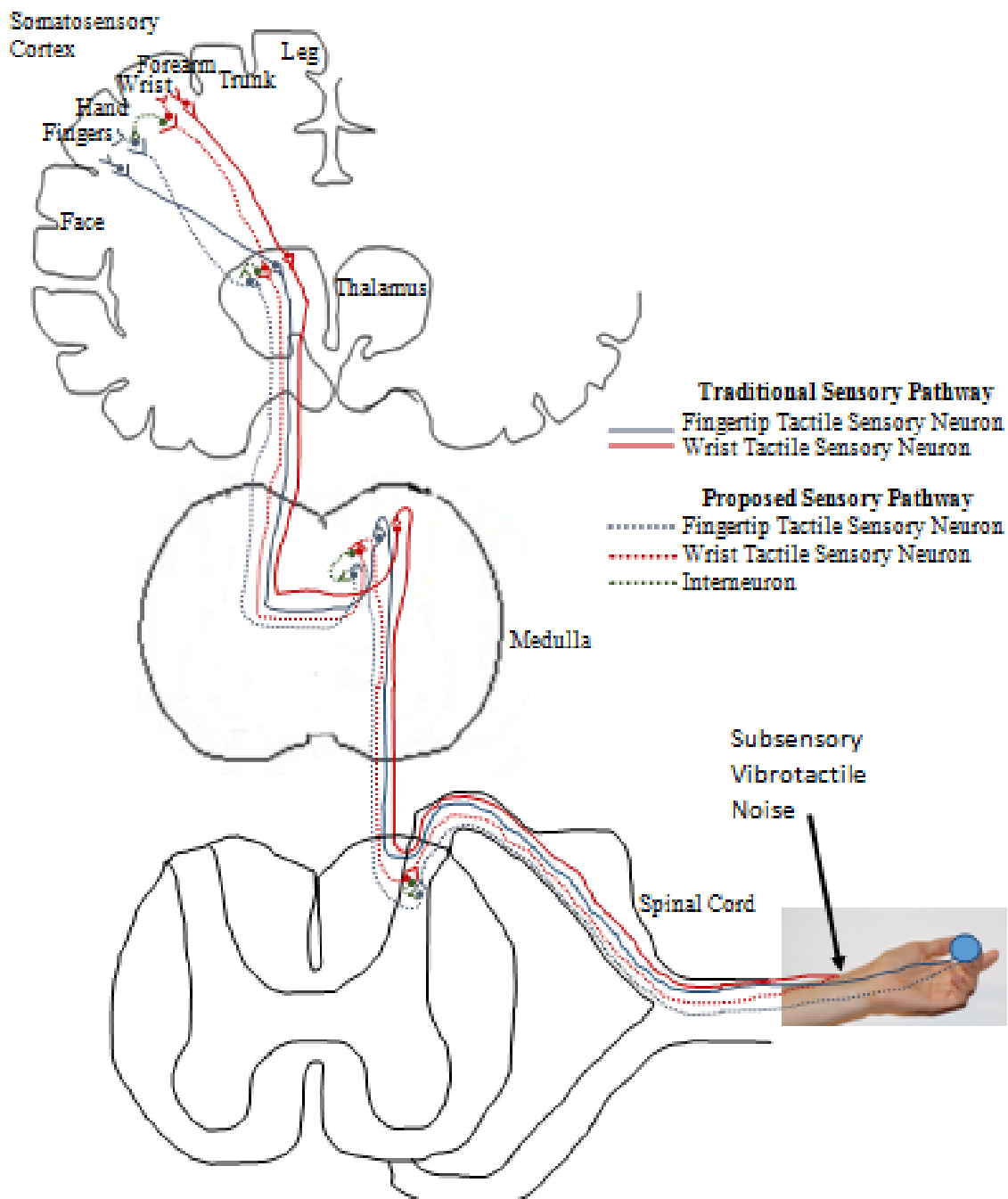
Figure 35: Application of remote vibrotactile noise at the wrist could have improved tactile sensation (at the fingertips and upper palm) and motor control of the hand via increased excitation and synchronization of the interneuronal connections in the spinal and cortical pathways. The proposed mechanism with internueural connections is shown in 'dotted lines' with the traditional sensory pathway shown in 'solid lines'.

Practicality of remote subsensory vibrotactile noise for clinical adoption

Currently, main stroke rehabilitation strategies focus more on motor-retraining rather than sensorimotor integration, even though motor recovery post stroke has been found to depend on the extent of somatosensory deficit (Tyson et al. 2008). Furthermore, sensory reorganization may actually precede and generate motor reorganization post stroke (Weiller 1998). Therefore, a therapy device that has the potential to improve sensation and motor control simultaneously, such as one with remote subsensory vibrotactile noise, could greatly benefit stroke survivors and promote motor recovery.

Remote subsensory vibrotactile noise may also be beneficial as a rehabilitation device because it allows for grip comfort. A preliminary device applying subsensory noise has been developed by Kurita et al. (2013). Although this device has been shown to improve fingertip tactile sensation, the placement of the device on the side of the fingers could be problematic for use as a rehabilitation device. Such a device blocks physical contact between the finger and object, defeating the purpose of improving sensation. Also since stroke patients often experience spasticity in fingers which causes finger flexion (Mottram et al. 2009), there may not be sufficient space around the fingers for a device. Applying the subsensory vibrotactile noise remotely, allows for the stroke survivors to have full range of motion of the hands. In addition to placement, the remote subsensory vibrotactile noise is not felt, decreasing the disturbance to the user. Other methods of increasing tactile sensation and motor control peripherally for stroke survivors use

suprasensory nerve or muscle stimulation that can cause discomfort (Chae et al. 1998) and may cause unintentional muscle spasms (Pike 1978).

Limitations and Future Directions of Remote Subsensory Vibrotactile Noise Research

One of the limitations of this research is that tactile sensory deficit and tactile sensory improvement was only investigated for fingertip and palm light touch, but not directly for other types of sensation that could be important in grip control such as proprioception. The effect that vibrotactile noise has on tactile sensory deficit was investigated, since impaired tactile sensory feedback is more common post stroke than impaired proprioception (Tyson et al. 2008). Also, the stroke groups in this research did not show evidence of impaired proprioception by being able to relatively perceive their 50% maximum grip effort. Proprioception assessment for fingertips is often difficult as proper measurement can require a complex testing modality (Clark et al. 1986) or could be hindered by spasticity of the fingers due to equipment requirements (Wycherley et al. 2005). However, a future study may target how remote subsensory vibrotactile noise could improve proprioception, which is important for balance and hand motor control and has been found to improve with direct remote subsensory vibrotactile noise (Mendez-Balbuena et al. 2012; Priplata et al. 2002; Priplata et al. 2006). Additionally, a future study could look at how healthy older individuals' and stroke survivors' cortical activity changes with remote subsensory vibrotactile noise using electroencephalography (EEG).

The previous pilot study (Hur et al. 2013; Tseng 2013), showed increased activity associated with improved sensorimotor processing when remote subsensory vibrotactile noise was applied to one healthy young individuals. A similar protocol examining how cortical activity changes with noise for stroke survivors and healthy individuals would yield greater understanding of the neurological pathway in which remote vibrotactile noise improves tactile sensation and motor control.

Another limitation of this research is that the long terms benefits of remote vibrotactile noise are currently unknown. The improvements observed in this research did not appear to be long lasting, as motor control and sensation returned to normal when the vibrotactile noise was removed. However, in these experiments exposure to remote subsensory vibrotactile noise was short term (1- 25 minutes, depending on the study). It could be that the long term benefits of vibrotactile noise occur after a longer exposure time, such as continuously for two hours, or when paired with more intense motor training (i.e. 15 minutes of a continuous motor task). Previous experiments in determining the effectiveness of peripheral stimulation have shown improvements retained 30 days after an intense 2 hour somatosensory training (Conforto et al. 2007). Furthermore, the stroke survivors in the research reported in this dissertation were all in the chronic stage of recovery (>6 months post-stroke event) and since there is a great deal of cortical reorganization occurring in the first months of stroke (termed the acute stage of recovery)(Ward et al. 2003), it is possible that applying remote vibrotactile noise earlier could induce greater and more permanent results. Currently, a clinical study to determine the effects of remote subsensory vibrotactile noise on acute stroke survivors is

being designed. Approximately 15 stroke survivors will have remote subsensory vibrotactile noise applied to their wrists during their normal physical therapy sessions over a two week period. Two weeks following this two week session, measurements of functional recovery will be tested again and compared to a control group of stroke survivors who received normal physical therapy with no remote subsensory vibrotactile noise applied to the wrist.

Final Conclusions

Stroke survivors, especially those with tactile sensory deficit, exhibit reduced phalanx force control during power grip, increasing risk of dropping objects. Applying remote subsensory vibrotactile noise improved phalanx force control during static power and improved gripping during a dynamic task. Furthermore, vibrotactile noise improved fingertip and upper palm tactile sensation for stroke survivors with tactile sensory deficit. Remote subsensory vibrotactile noise improved tactile sensation and motor control potentially via stochastic resonance and interneuronal connections. Therefore, vibrotactile noise may be useful in improving gripping stability and could be a useful tool in sensorimotor rehabilitation, especially for stroke survivors with tactile sensory deficit. In the future, the rehabilitation device designed using the concept of vibrotactile noise will be used in clinical testing. In addition, an EEG study will be conducted to further understand the cortical pathway of how vibrotactile noise improves stroke survivors' motor control. This dissertation contributes to the long term goal of increasing stroke survivors' independence in completing daily living activities.

Conflict of Interest

Work included in Aim 3 has been used in the development of a pending patent for

TheraBracelet (U.S. Patent Application No. 14/256,156, Entitled: Wearable Device for

Improving Tactile Sensitivity) and I have financial interest in, or financial conflict with,

in the subject matter or materials discussed in the manuscript section Aim 3 as a co-

inventor.

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Appendix A. Dissertation Overview

Aims	Known	Unknown	Dissertation Contribution
To characterize altered power grip post stroke.	Stroke have increased digit force deviation leading to increased slippage during pinch grip (Seo 2010) and reduced pinch grip force control and timing (Nowak 2003, Hermsdorfer 2003) Force distribution between the index and the thumb tip not affected by stroke during pinch grip (Seo 2010)	Do stroke survivors exhibit altered force control or an altered distribution of grip force in power grip?	Phalanx force control is reduced for stroke survivors compared to healthy as found by increased phalanx force deviation. Force distribution across the phalanges and fingers remained similar to healthy individuals.
To determine the role of sensory deficit in stroke survivors' power grip force control.	Stroke survivors experience diminished tactile sensory feedback (Carey 1995; Turton and Butler 2001) and the extent of tactile sensory deficit is related to motor recovery post stroke (Tyson 2008) Sensory feedback assists in the preservation of the normal cortical representations of both the motor and sensory cortex (Weiss 2004). Important for updating the CNS for the adjustment of the grip forces to prevent object slippage (Johansson and Westling 1984) Stroke tactile sensory deficit decreased pinch grip force control (Hermsdorfer 2003) Reduced tactile sensation can lead to object dropping (Augurelle 2003)	How does stroke induced sensation deficit affect power grip force control?	Stroke survivors with tactile sensory deficit exhibited greater phalanx force deviation during power grip compared to stroke survivors without tactile sensory deficit and healthy controls.
To determine the effect of sensory enhancement on power grip force control (via application of remote subsensory vibrotactile noise)	Increasing sensation could facilitate information regarding grip surface characteristics (Johansson and Westling 1987), magnitude and directional feedback on phalanx force being produced (Augurelle 2003; Blennerhassett 2007; Cole 2006; Hermsdorfer 2003; Monzée 2001; Robertson and Jones 1994) and information about finger position and alignment with respect to the object surface (Monzée 2001) Subsensory vibrotactile noise has been found to maximize the detection and transmission of weak sensory signals and can increase tactile sensation (Galica 2009; Moss 2004; Priplata 2002, Collins 1997) Application of vibrotactile noise has been shown to improve gait (Galica 2009), balance (Priplata 2002, Priplata 2006, Gravelle 2002), and controlling of finger position (Mendez-Balbuena 2012) Evidence shows that vibrotactile noise may increase cortical and spinal activation (Manjarrez 2002a; Manjarrez 2003, Hidaka 2000) and sensorimotor integration and processing (Hur 2013, Tseng 2013)). Also vibrotactile noise may improve neuronal synchronization (Manjarrez 2002b) which can facilitate neural communication between the spinal and cortical levels (Fell and Axmacher 2011)	Can applying the subsensory vibrotactile noise at a remote location (at the wrist) improve sensation at the fingertips/hand? Can remote subsensory vibrotactile noise improve phalanx force deviation during power grip? Can remote subsensory vibrotactile noise improve the ability to manipulate objects during a dynamic movement?	Applying subsensory vibrotactile noise remotely improved fingertip and upper palm tactile sensation for stroke survivors with tactile sensory deficit Noise improved power grip phalanx force control for stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and healthy controls Noise improved stroke survivors' with tactile sensory deficit ability to complete two separate dynamic movement tasks

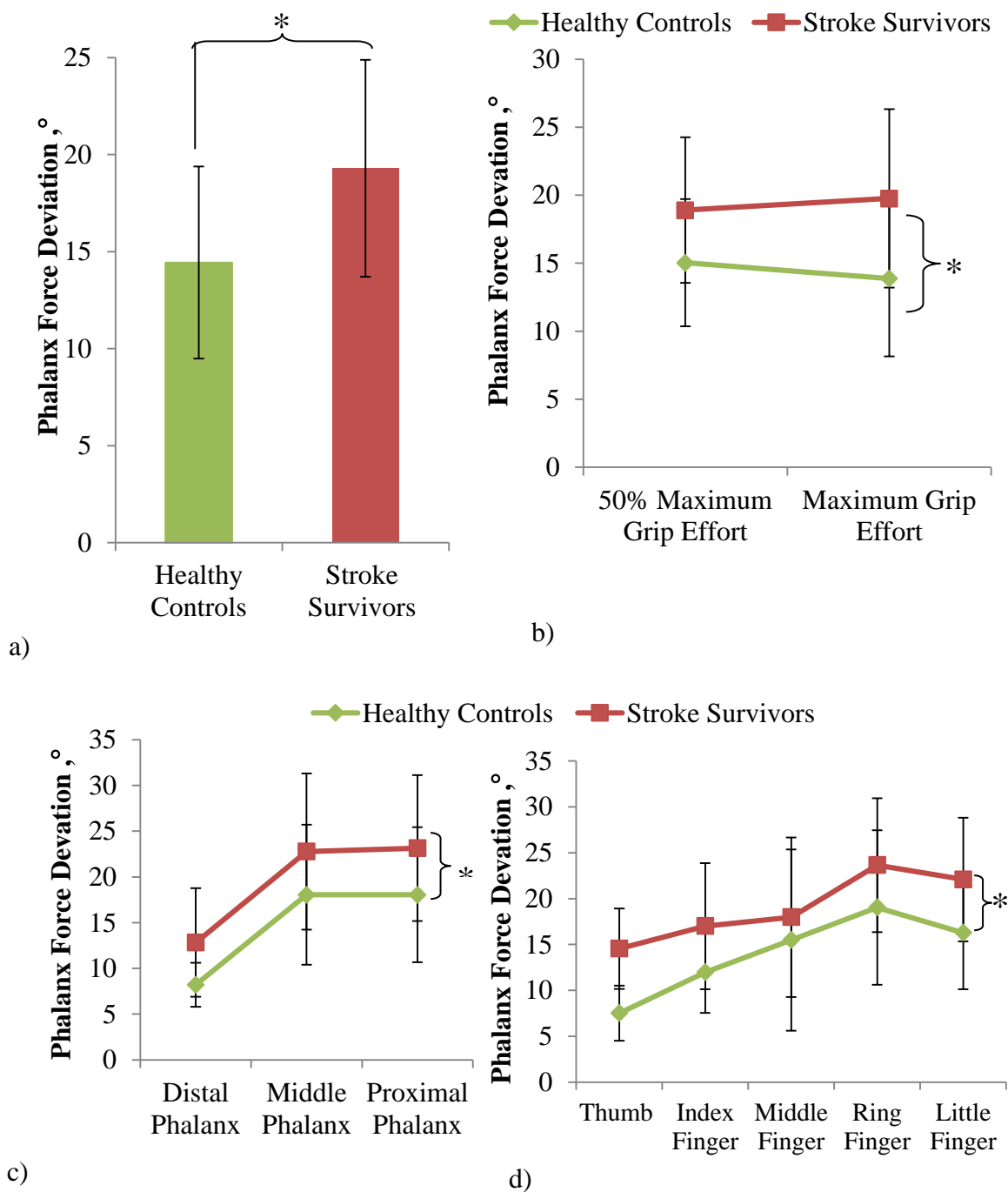**Appendix B. Aim 1 Results with Standard Deviation bars**

a)

b)

c)

d)

Figure 35: Phalanx force angular deviation was significantly greater for stroke survivors compared with controls (ANOVA subject group main effect with p<.05) (effort levels, fingers, phalanges, and subjects pooled) (a), for both 50% and maximum grip effort (b), for all three phalanges (c), and especially for the thumb, index, and little fingers (ANOVA, subject group and finger interaction with p<.05, posthoc significance marked with stars) (d). Non-transformed mean ± SD data is shown in the figure.
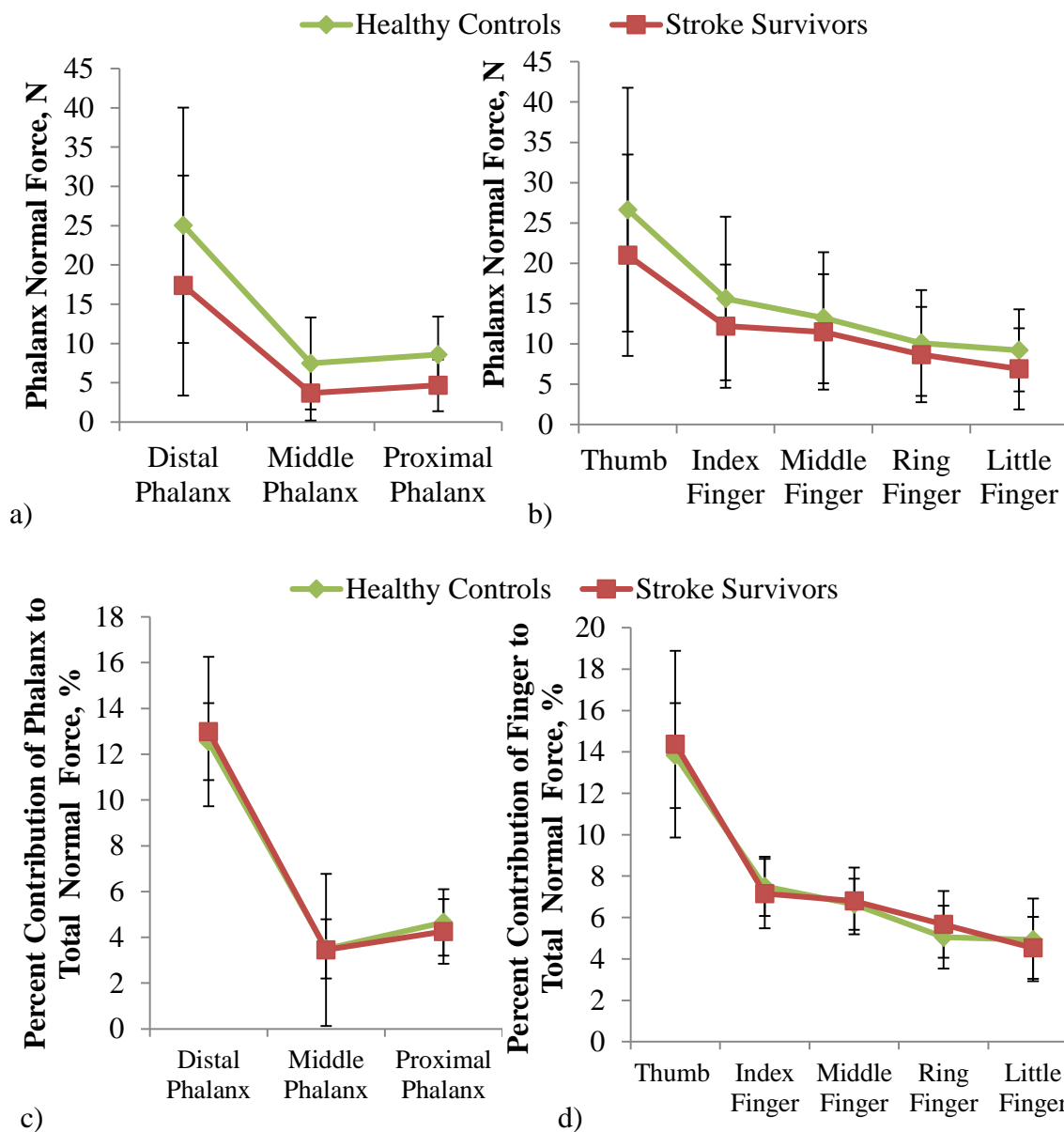
Figure 36: The distribution of phalanx normal force across the phalanges (a and c) and fingers (b and d) for stroke and control subjects. Percent contribution (c and d) of the individual phalanges to total normal force was not significantly dependent upon the interaction of subject group and phalanx or the interaction of subject group and finger (ANOVA with $p > .05$) (d). Non-transformed mean $\pm$ SD data is shown in the figure.
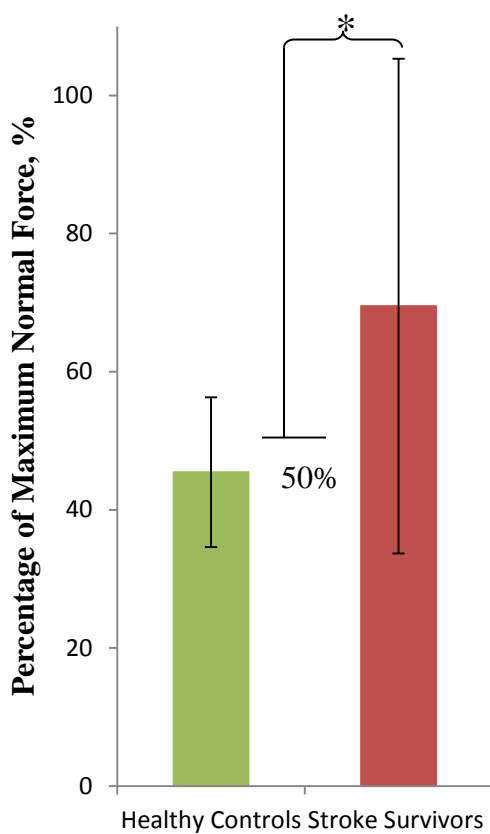
Figure 37: Mean ± SD percentage of force produced during grip at 50% of maximum perceived effort. Stroke survivors produced more than 50% of maximum (t-test with *p*<.05), unlike controls (t-test with *p*>.05). Non-transformed data is shown in the figure.
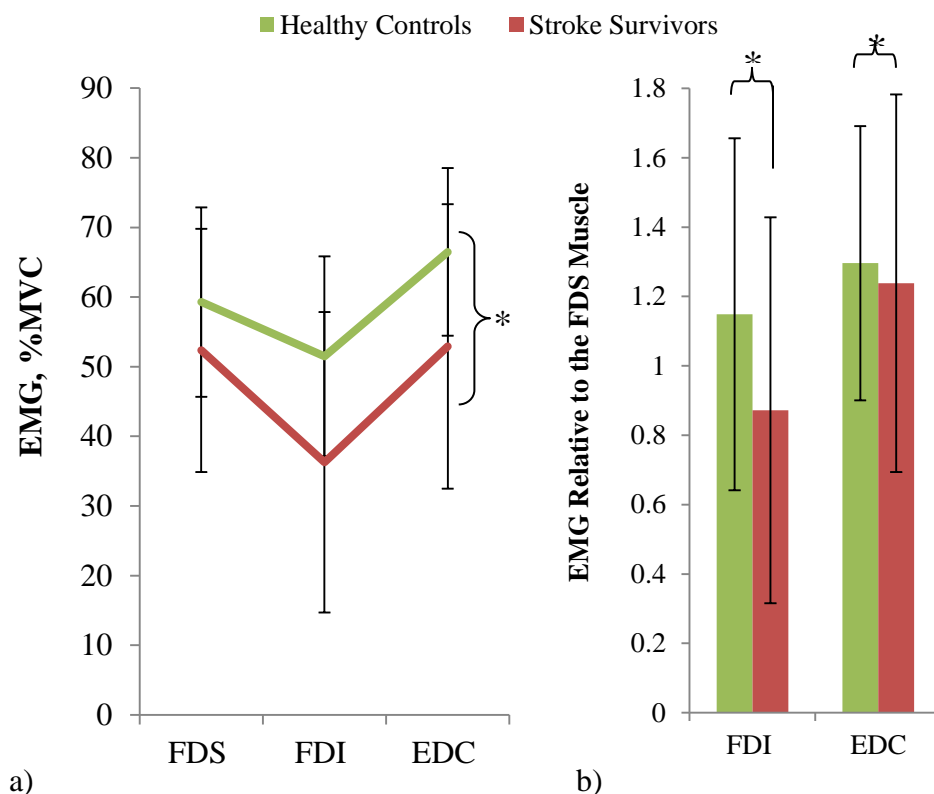
Figure 38: Mean ± SD EMG was reduced for all muscles of the stroke survivors compared with healthy controls (a). Relative to the FDS EMG, mean + SD FDI and EDC EMG were significantly reduced for stroke survivors compared with controls (significant subject group and finger muscle interaction with p<.05, significant difference in relative FDI and EDC EMG between stroke and control with Tukey post-hoc p<.05) (b), showing an altered muscle activity pattern with a particularly weakened intrinsic FDI muscle and the extrinsic EDC muscle for stroke survivors compared with controls. Non-transformed data is shown in the figure.
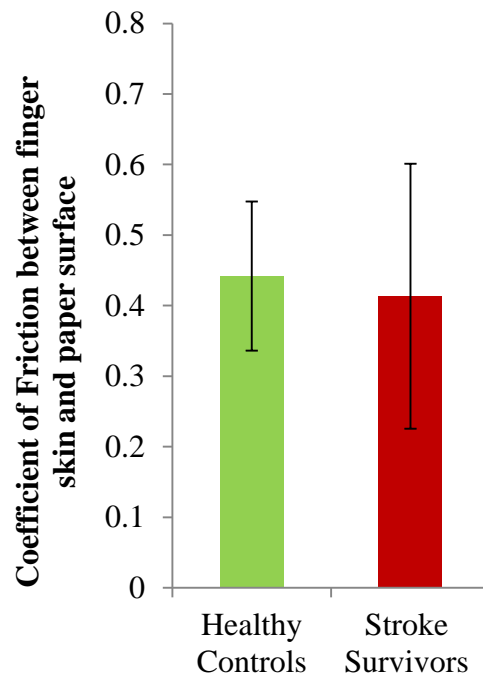
Figure 39: Mean ± SD of the COF between the finger skin and paper surface was similar for stroke survivors and healthy controls (t-test, *p*>.05). Non-transformed data is shown in the figure.

Appendix C. Aim 2 Results with Standard Deviation bars
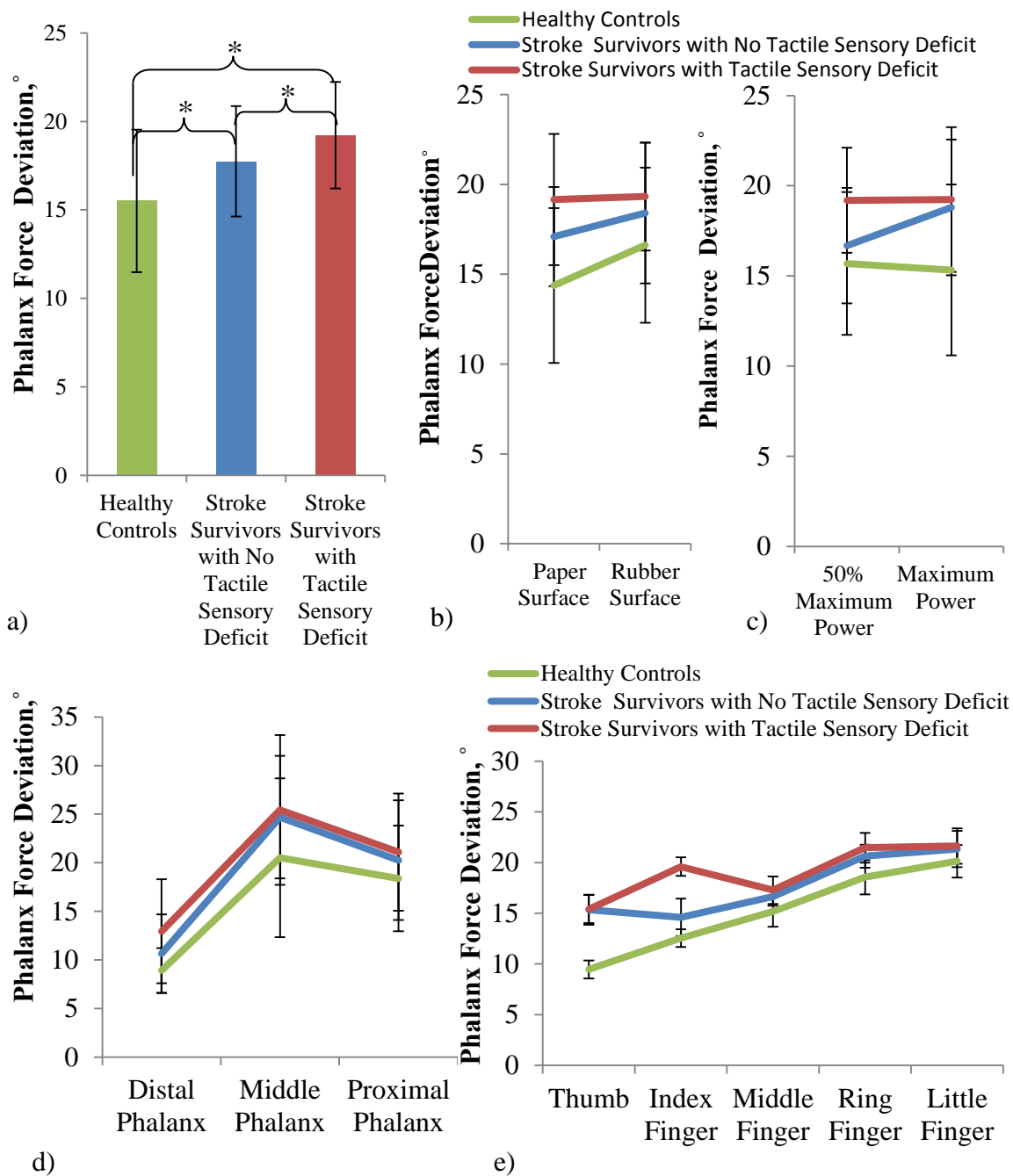

Figure 40: Mean ± SD phalanx force deviation was significantly greatest for stroke survivors with tactile sensory deficit, followed in order by stroke survivors without tactile sensory deficit and healthy controls (ANOVA, subject group main effect with p<.05) This trend was observed for all surfaces (b), effort levels (c), phalanges (d), and fingers (e).
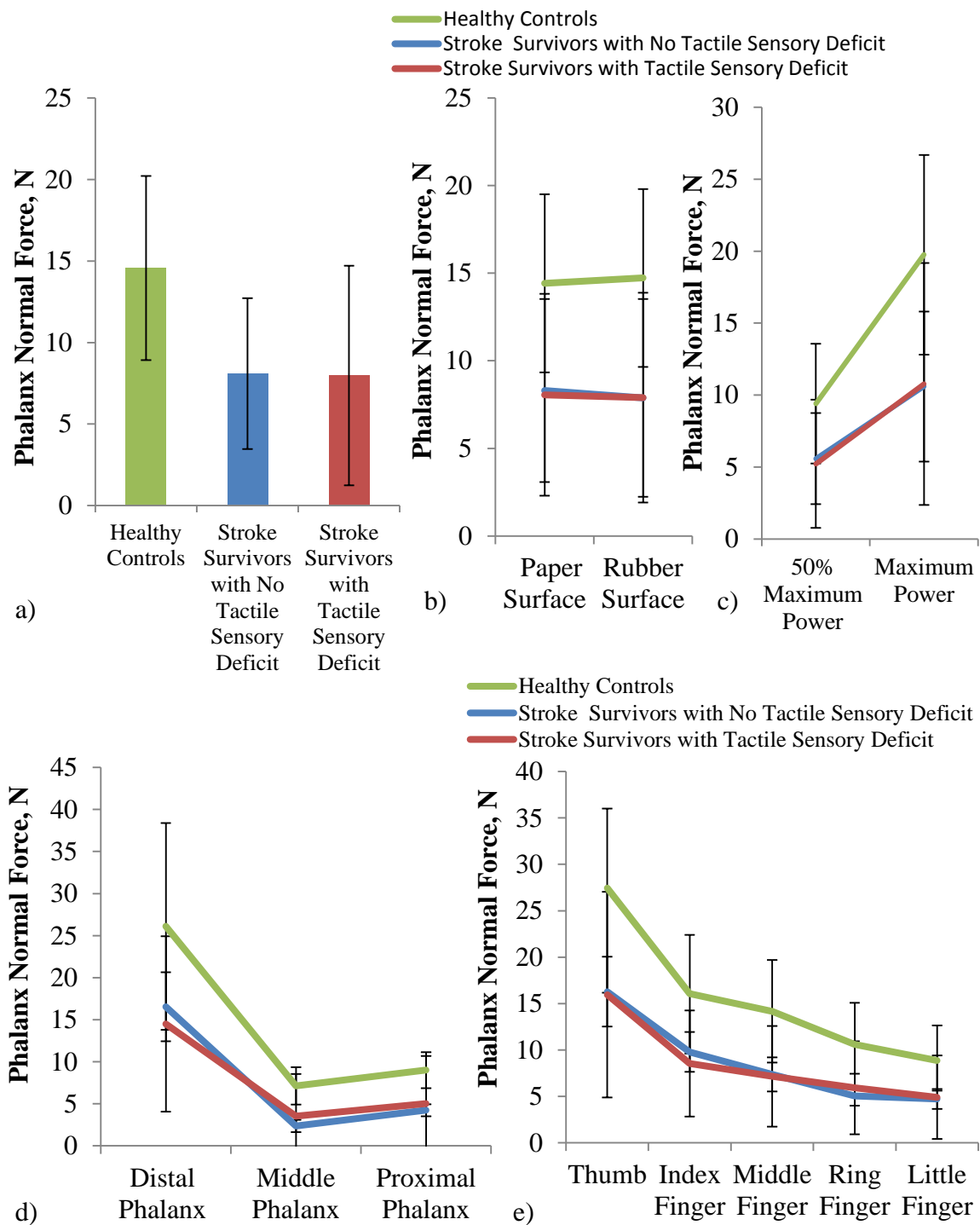
Figure 41: Mean ± SD phalanx normal force was significantly reduced for both stroke survivor groups compared to healthy controls (ANOVA, subject group main effect with p<.05). This reduction was similar for both stroke survivor groups and was observed for all surfaces (b), effort levels (c), phalanx (d), or finger (e).
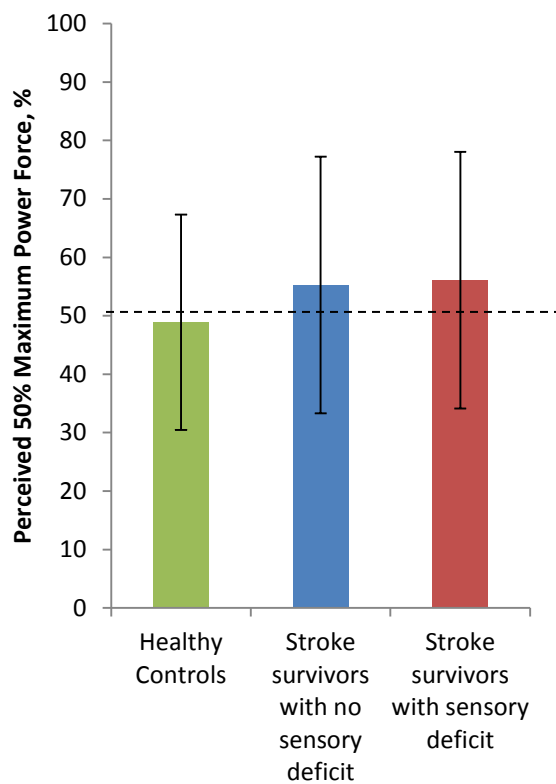
Figure 42: Mean ± SD grip force produced during grip at 50% of the maximum perceived effort normalized to the grip force produced during maximal grip was not significantly different from the target of 50% for all subject groups.
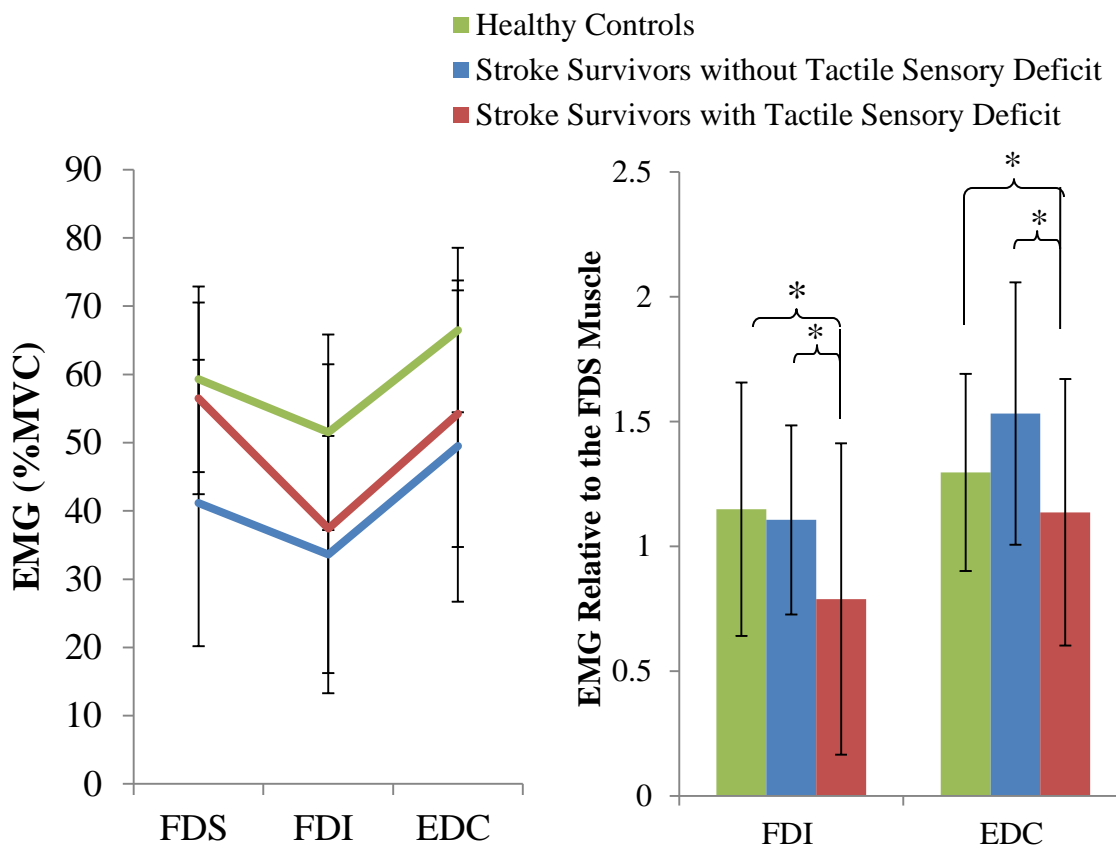
Figure 43: Mean ± SD EMG appeared to be reduced for stroke survivors compared to healthy controls for both stroke survivor groups (a). Mean ± SD FDI and EDC EMGs relative to the FDS EMG were significantly reduced for stroke survivors with tactile sensory deficit compared to controls and stroke survivors without tactile sensory deficit (significant subject group main effect with p<.05, significant difference for stroke survivors with tactile sensory deficit group compared to other two groups with Tukey posthoc p<.05 for both relative FDI and EDC EMGs) (b), showing altered muscle activity pattern with particularly reduced intrinsic FDI and extrinsic EDC muscle activities for stroke survivors with tactile sensory deficit compared to controls and stroke survivors without tactile sensory deficit. Non-transformed data is shown in the figure.
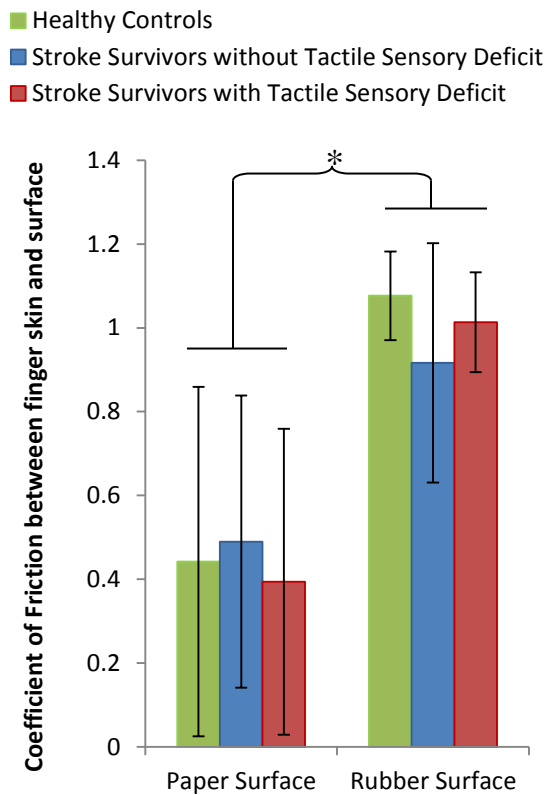
Figure 44: Mean ± SD COF between the finger skin and the paper and rubber surfaces was similar for stroke survivors with tactile sensory deficit, stroke survivors without tactile sensory deficit, and healthy controls (ANOVA, group main effect and group and surface interaction p>.05). The COF for the rubber surfaces was significantly greater than the paper surface (ANOVA, surface main effect with p<.05). Non-transformed data is shown in the figure.

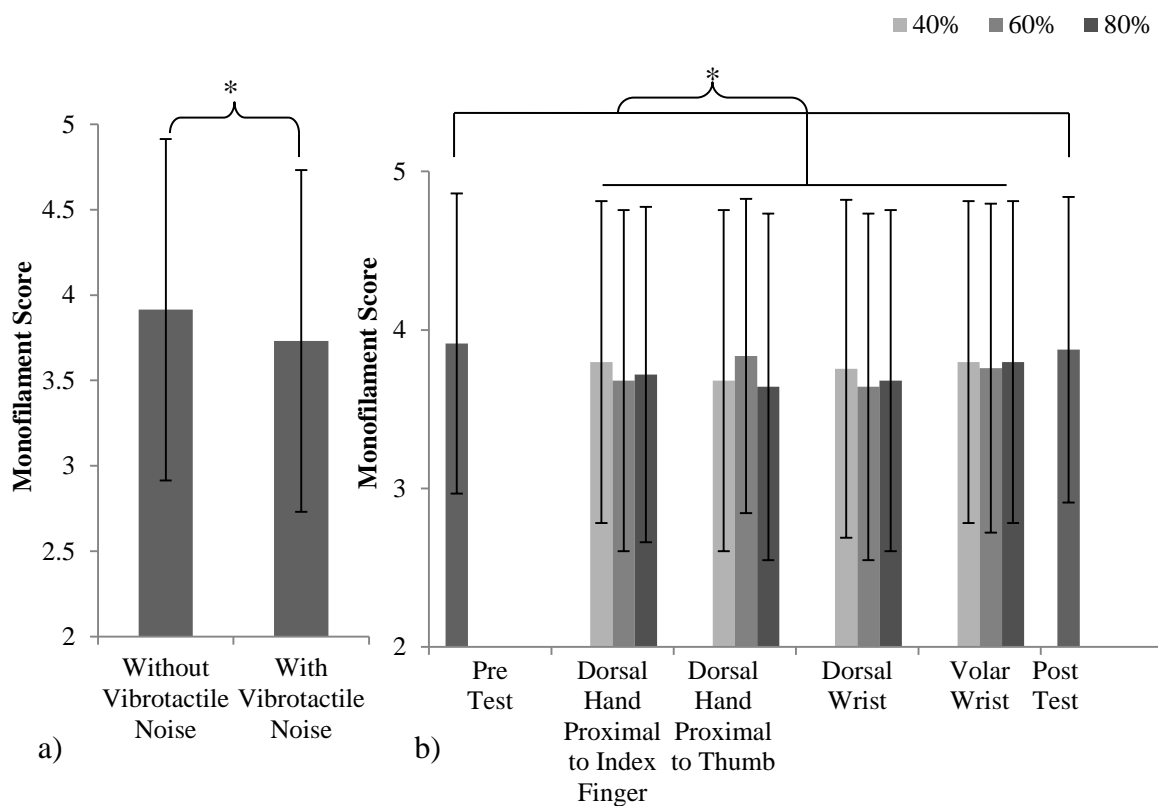**Appendix D. Aim 3 Results with Standard Deviation bars**

Figure 45: Mean ± SD Monofilament scores significantly decreased with subthreshold vibrotactile noise (noise locations, intensities, fingers, and subjects pooled) ($p<.01$) (a). Noise locations and intensities did not significantly affect the improvement of Monofilament score (fingers and subjects pooled, $p>.05$ for noise location and intensity) (b).
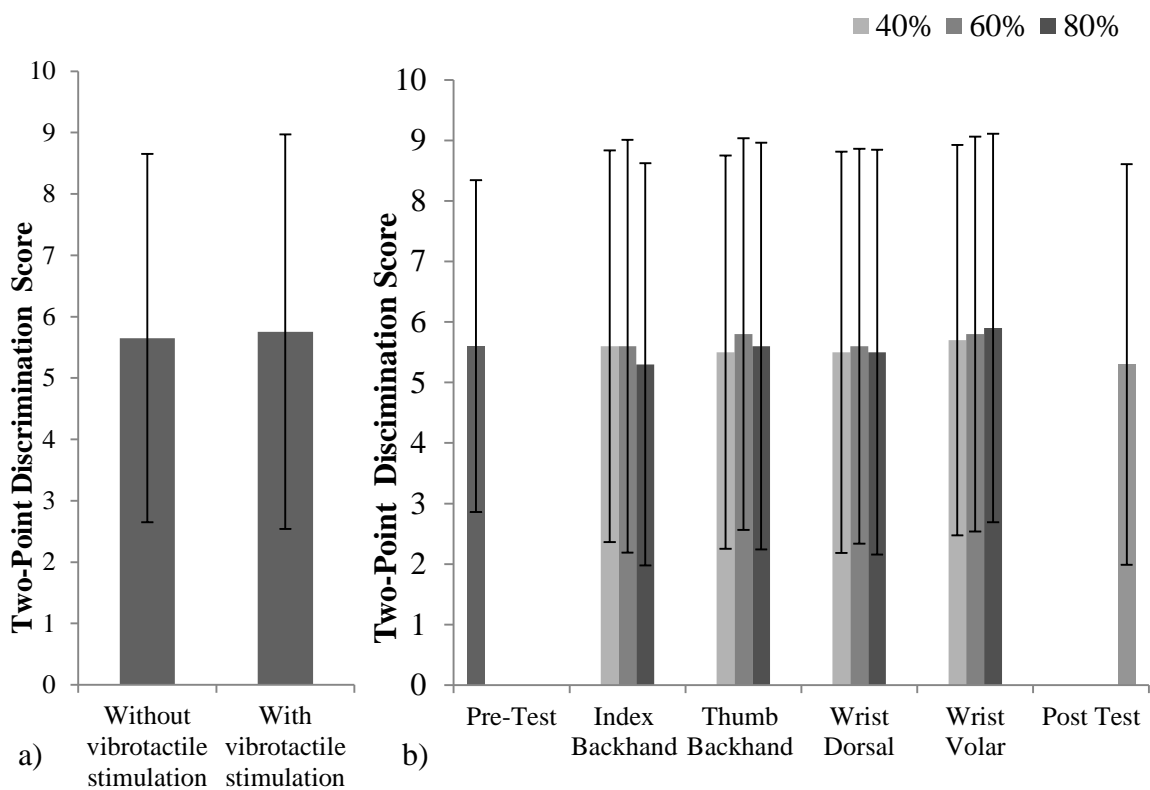
Figure 46: Mean ± SD Two Point Discrimination scores were not significantly affected by the vibrotactile noise (a) nor with noise locations, intensities, fingers, and their interactions (fingers and subjects pooled) (p>.05) (b). The Two-Point Discrimination score without vibrotactile noise did not change at the beginning vs. end of the testing session.
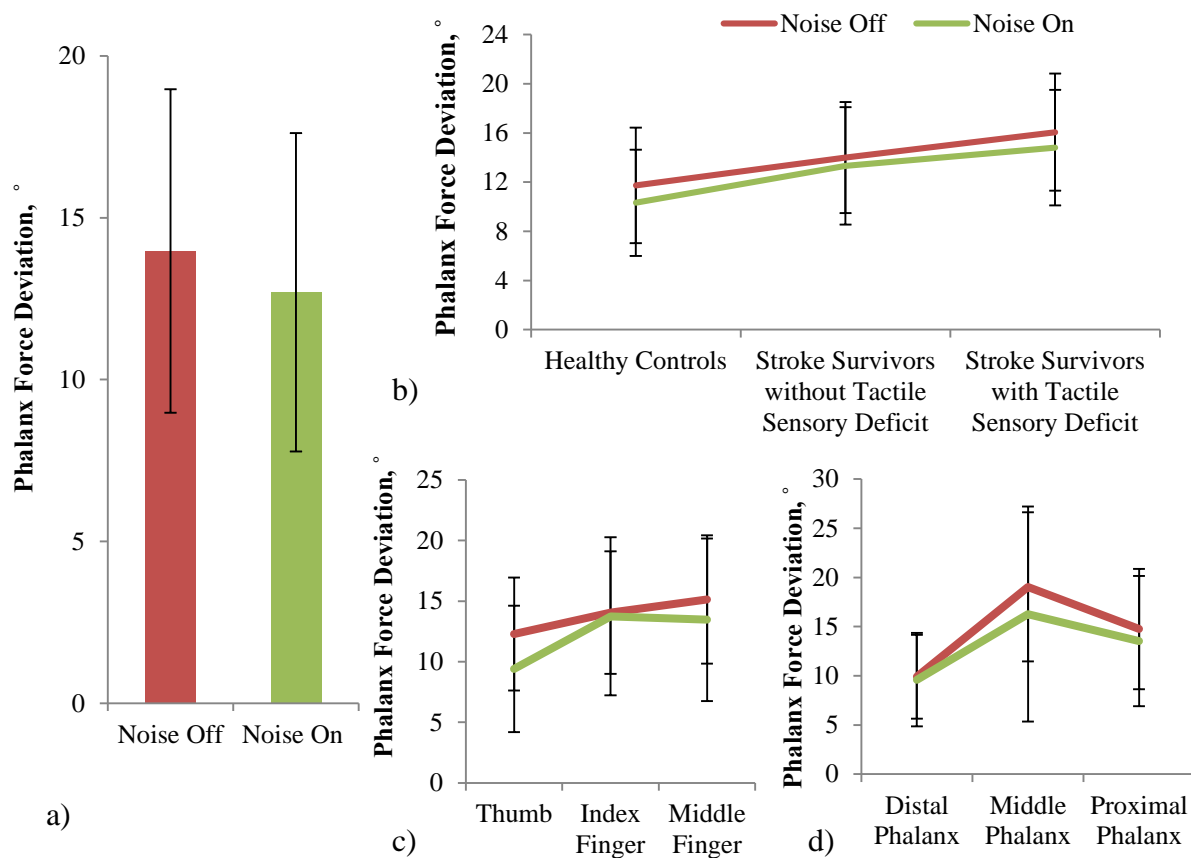
Figure 47: Mean ± SD phalanx force deviation was significantly reduced with remote vibrotactile noise turned on (a). Reduction in phalanx force deviation was observed across all subject groups (b). The extent that phalanx force deviation reduced with the noise differed by fingers (c) and phalanges (d).
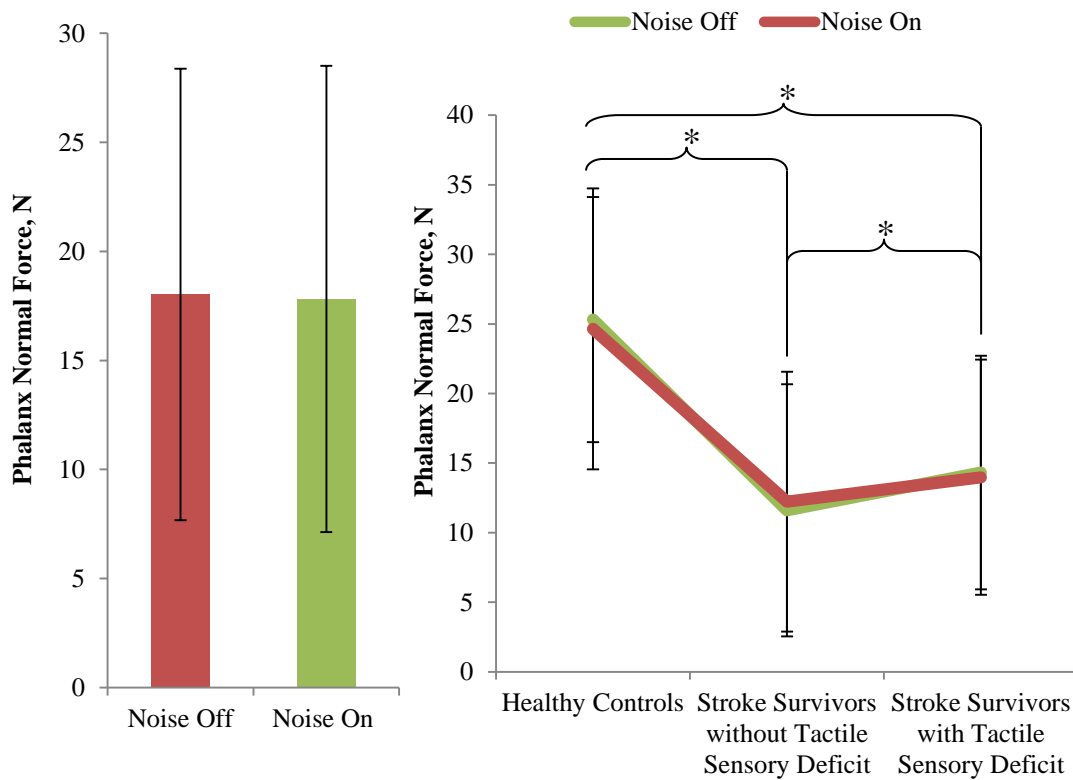
Figure 48: Mean ± SD phalanx normal force did not significantly change with the application of vibrotactile noise (a). Phalanx normal force was significantly greater for the healthy controls, followed in order by the stroke survivors with tactile sensory deficit and the stroke survivors without tactile sensory deficit (b). None of the subject groups significantly changed their phalanx normal force with remote vibrotactile noise.
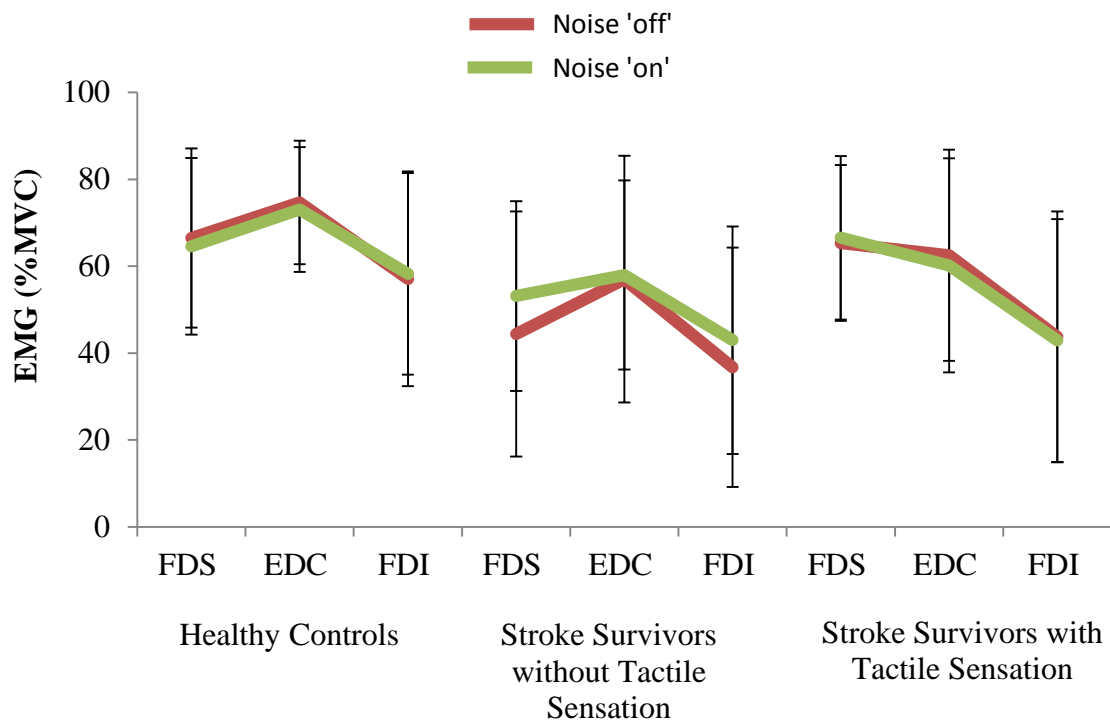
Figure 49: Mean ± SD Muscle EMG activity was not significantly affected by the application of remote vibrotactile noise.
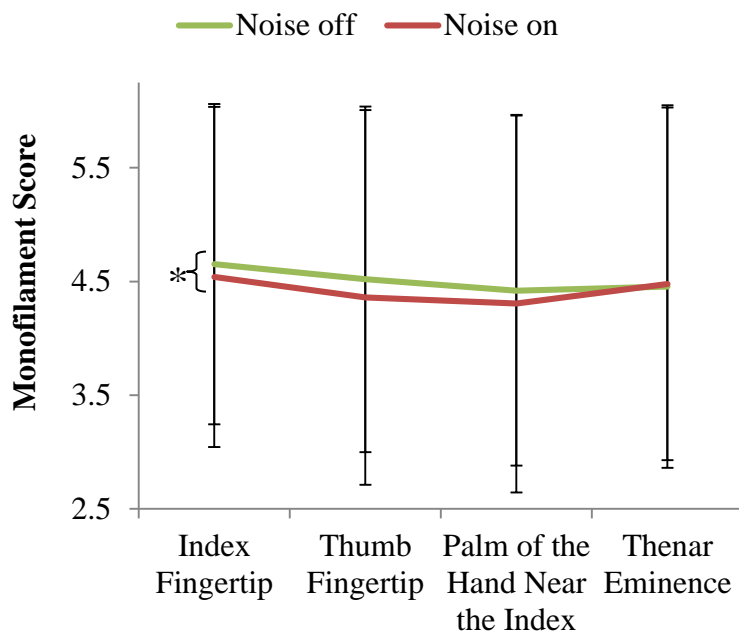
Figure 50: Mean ± SD Monofilament score was overall significantly reduced for the stroke survivors with tactile sensory deficit when vibrotactile noise was present.
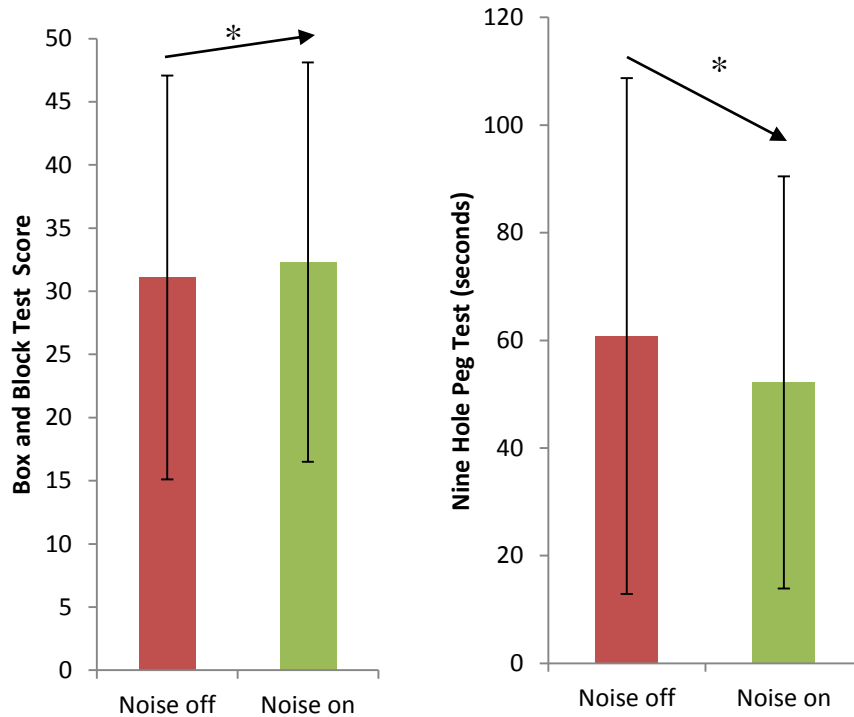
Figure 51: Mean ± SD BBT score (a) and NHPT time (b) significantly improved with the application of remote vibrotactile noise. Individual subjects' data is shown for the BBT scores and NHPT in c) and d), respectively. Individual subjects' percent change in the BBT score and the time to complete the NHPT is shown in e) and f), respectively.

CV
LEAH R. ENDERS

A. Education and Training

INSTITUTION AND LOCATION	DEGREE	YEAR(S)	FIELD OF STUDY
University of Pittsburgh, Pittsburgh	B.S.	05/09	Bioengineering/Biomechanics
University of Wisconsin-Milwaukee, Milwaukee	M.S.	12/10	Biomechanics/Rehabilitation
University of Wisconsin-Milwaukee, Milwaukee	Ph.D.	12/14	Biomechanics/Rehabilitation

B. Positions and Honors

| ACTIVITY/ OCCUPATION	BEGINNING DATE (mm/yy)	ENDING DATE (mm/yy)	FIELD	INSTITUTION/ COMPANY	SUPERVISOR/ EMPLOYER
Undergraduate Research Assistant	2006	2009	Bio-engineering	Human Movement and Balance Laboratory, University of Pittsburgh	Rakié Cham, Ph.D. and April Chambers, Ph.D.
Teaching Assistant	2009	2010	Industrial Engineering	University of Wisconsin-Milwaukee	Department of Industrial & Manufacturing Engineering
Research Assistant	2009	Current	Biomechanics/ Rehabilitation	Hand Rehabilitation Laboratory, University of Wisconsin-Milwaukee	Na Jin Seo, Ph.D.
Chief Scientific Officer	2013	Current	Research/ Engineering	TheraBracelet, LLC	Matthew Raggard, CEO

Honors

2014	University of Wisconsin-Milwaukee, College of Engineering and Applied Science Academic Excellence Award
2013-14	University of Wisconsin-Milwaukee Distinguished Dissertation Fellowship Award

2012-13	American Heart Association Midwest Affiliate 2011 Predoctoral Fellowship
2013-14	American Society of Biomechanics Student Travel Award
2013	University of Wisconsin-Milwaukee, College of Engineering and Applied Science Research Poster Competition, 2nd place award
2012	American Society of Biomechanics Grant-In-Aid Award
2012	University of Wisconsin-Milwaukee, College of Health Sciences, Spring Scientific Research Symposium, 1st place award
2012	IEEE Larry House Student Poster Competition, Milwaukee School of Engineering, 1st place award
2009-2013	University of Wisconsin-Milwaukee Chancellor's Graduate Award
2010-2013	University of Wisconsin-Milwaukee Travel Award
2010	University of Wisconsin-Milwaukee Outstanding Student Award

Academic and Professional Membership/Activity

2010- Present	Member, American Society of Biomechanics
2012- 2013	Member, Society for Neuroscience
2010- 2012	Community Outreach Chair and Member, Engineers without Borders
2010- 2012	Volunteer with Growing Power, Inc.
2009- 2011	Chair member, Human Factors and Ergonomics Society
2009- 2010	Member, Biomedical Engineering Society
2008- 2011	Volunteer with Habitat for Humanity
2008- 2009	Developed a program called the Interactive Projective Overlay project with a team of students for a senior project at the University of Pittsburgh. This is a simulation training product for tracheal intubation for medical trainees
2006	Volunteered with Hurricane Katrina Relief
2005	Volunteered as a mentor in an after-school tutoring program.

C. Publications

Journal Publications:

1. **Enders LR,** Hur P., Johnson M.J., and Seo N.J, "Remote vibrotactile noise improves light touch sensation in stroke survivors' fingertips via stochastic resonance", Journal of NeuroEngineering and Rehabilitation, 2013, 10:105.

2. **Enders LR,** Seo NJ, "Phalanx force magnitude and trajectory deviation increased during power grip with an increased coefficient of friction at the hand-object interface", Journal of Biomechanics, 2011, 44(8):1447-53.

3. Seo NJ, Fathi-Firoozabad M, **Enders LR,** Motawar B, Kosmopolous M, "Digit force deviation correlates with upper extremity function scores in chronic stroke survivors", Journal of Biomechanics, *Accepted*

4. Seo NJ, Kosmopolous M, **Enders LR,** Hur P. "Effect of Remote Sensory Noise on Hand Function Post Stroke", Frontiers in Human Neuroscience, *Accepted, In Press*

5. Slota, GP, **Enders LR**, Seo NJ, "Improvement of hand function using different surfaces and identification of difficult movement post stroke in the Box and Block Test", Applied Ergonomics, 2013, 45(4):833-838.

6. Seo NJ, **Enders LR,** "Hand grip function assessed by the Box and Block Test (BBT) is affected by object surfaces", Journal of Hand Therapy, 2012, 25(4) 397-405.

7. Seo NJ, Shim JK, Engel AK, **Enders LR, "**Grip surface affects maximum pinch force", Human Factors: The Journal of Human Factors and Ergonomics Society, 2011, 53 740-748.

8. Samosky JT, Baillargeon E, Bregman R, Brown A, Chaya A, **Enders L**, Nelson DA, Robinson E, Sukits AL, and Weaver RA, "Real-time "x-ray vision" for healthcare simulation: an interactive projective overlay system to enhance intubation training and other procedural training," Studies in Health Technology Informatics, 2011, 163, 549-551.

9. **Enders LR** and Seo NJ. "Altered phalanx force direction during power grip following stroke", Experimental Brain Research, *In Revision*

10. **Enders LR** and Seo NJ. "Effects of tactile sensory deficit on phalanx force deviation during power grip post stroke", *To be submitted*

11. **Enders LR** and Seo NJ. "Effects of subthreshold vibrotactile noise on healthy controls and stroke survivors' altered phalanx force deviation during power grip", *To be submitted*

Master's Thesis
Enders L.R. "Effect of hand-object friction on grip force application and hand function" Master's Thesis, University of Wisconsin-Milwaukee, 2010.

Doctoral Dissertation
Enders L.R. "Role of Sensation in altered phalanx grip force in persons with stroke" Doctoral Dissertation, University of Wisconsin-Milwaukee, 2014.

Conference Proceedings
1. **Enders L.R.,** Seo N. J. "Remote Vibrotactile Noise Improves Phalanx Force Deviation during Power Grip in Stroke Survivors". 7th World Congress of Biomechanics 2014, Boston, MA.

2. Kosmopoulos M., Hur P., **Enders L.R.,** Seo N. J. "Effect of Remote Subthreshold Vibrotactile Noise on Hand Function Post-Stroke". 7th World Congress of Biomechanics 2014, Boston, MA.

3. **Enders L.R.,** Seo N. J. "Stroke induced sensory deficit decreases phalanx force control during power grip". 37th Annual Meeting of the American Society of Biomechanics 2013, Omaha, NE.

4. Lambert L.R., Slota G.P., **Enders L.R.,** Seo N. J. "Altered power grip force distribution across the phalanges and increased proximal phalanx trajectory deviation for older individuals." 37th Annual Meeting of the American Society of Biomechanics 2013, Omaha, NE.

5. **Enders L.R.**, Seo N.J. "Stochastic Resonance Improves Sensation and Pinch Grip in Stroke Survivors". The 42nd Annual Meeting of the Society for Neuroscience, New Orleans, LA.

6. Brandenburg A.R., **Enders L.R.**, Seo N.J. "Use of high-friction objects to improve hand grip function assessed by the Box and Block Test for older adults". The 42nd Annual Meeting of the Society for Neuroscience, New Orleans, LA.

7. Weyers B.L., **Enders L.R.**, Seo N.J. "Age-Related Changes in the Control of Pinch Grip Force". The 42nd Annual Meeting of the Society for Neuroscience 2012, New Orleans, LA.

8. Lambert L.R., **Enders L.R.**, Seo, N.J. "Inter-limb transfer of grip coordination as a rehabilitation strategy for women following stroke". The 42nd Annual Meeting of the Society for Neuroscience 2012, New Orleans, LA.

9. **Enders L.R.** Seo N.J. "Effects of stroke-induced sensory and motor deficit on phalanx force magnitude and angular deviation during power grip" Annual Meeting of the American Society of Biomechanics 2011, Long Beach, CA.

10. Pounds C., **Enders L.R.,** Seo N.J. "Lack of Modulation of Maximum Pinch Force Depending on Grip Surface in Stroke Survivors" Annual Meeting of the American Society of Biomechanics 2011, Long Beach, CA.

11. **Enders L.R.**, Engel A.K.J., Seo N.J., "High Coefficient of Friction at the Hand-Object Interface Contributes to Increased Maximum Grip Force by the Phalanges During Power Grip," Annual Meeting of the American Society of Biomechanics 2010, Providence RI.

12. **Enders L.R.**, Cary D.T., Seo N.J., "The Box and Block Test Score is Dependent upon Block Surface," Annual Meeting of the American Society of Biomechanics 2010, Providence RI.

13. Engel A.K.J. **Enders L.R.**, Keenan, K.G., Seo, N.J., "Grip surface friction affects maximum tip pinch force," Annual Meeting of the American Society of Biomechanics 2010, Providence RI.

14. **Enders L.R.**, Berger K., Chambers A., Redfern M., McCrory J.L. "Biomechanical Evidence of Waddling During Pregnancy" Annual Biomedical Engineering Society Meeting, Pittsburgh 2009.

D. Patents

Full Utility Patent Application filed: April 2014, Inventors: Leah Enders and Na Jin Seo, Title: "Wearable Device for Improving Tactile Sensitivity" U.S. Patent Application No. 14/256,156.

E. Research Support

Role of tactile sensation in altered phalanx grip force in persons with stroke (Enders PI), University of Wisconsin-Milwaukee Distinguished Dissertation Fellowship, 2014.

Delays in Muscle Relaxation: A Novel Approach to Neuromechanism-based Stroke Rehabilitation (Seo PI, Enders Research Assistant), American Heart Association Scientist Development Grant, 2011-2015.

Role of tactile sensation in altered phalanx grip force in persons with stroke (Enders PI, Seo Advisor), American Heart Association Midwest Affiliate 2011 Predoctoral Fellowship, 2012-2013.

Role of tactile sensation in altered phalanx grip force in persons with stroke, American Society of Biomechanics 2012 Grant-In-Aid Award, 2012-2013.

Development of gender-specific rehabilitation strategies for women following stroke (Seo PI, Enders Research Assistant), Wisconsin Women's Health Foundation, 2010-2012.

Ergonomic Interventions for Altered Grip Force Direction in Stroke Survivors (Seo PI, Enders Research Assistant), University of Wisconsin-Milwaukee Research Growth Initiative, 2010-2011.

Stretch-Induced Neurophysiologic Recovery in Persons with Stroke (Seo PI, Enders Research Assistant), University of Wisconsin System Administration, 2009-2010.

Investigation of Hand Functional Grip and Release in Persons with Stroke (Seo PI, Enders Research Assistant), American Heart Association, 2009-2010