Published in final edited form as:

J Mot Behav. 2017 ; 49(1): 55–66. doi:10.1080/00222895.2016.1191416.

Effects of Sensory Deficit on Phalanx Force Deviation during Power Grip Post Stroke

Leah R. Enders, Ph.D.¹ and Na Jin Seo, Ph.D.²

¹Department of Industrial and Manufacturing Engineering, University of Wisconsin-Milwaukee, Milwaukee, WI, USA

²Division of Occupational Therapy, Department of Health Professions, Department of Health Sciences and Research, Medical University of South Carolina, Charleston, SC, USA

Abstract

The effect of sensory deficits on power grip force from individual phalanges was examined. We found that stroke survivors with sensory deficits (determined by the Semmes-Weinstein monofilament test) gripped with phalanx force directed more tangential to the object surface, than those without, although both groups had similar motor deficits (Chedoke-McMaster and Fugl-Meyer), grip strength, and skin friction. Altered grip force direction elevates risk of finger slippage against the object thus grip loss/object dropping, hindering activities of daily living. Altered grip force direction was associated with altered muscle activation patterns. In summary, the motor impairment level alone may not describe hand motor control in detail. Information about sensory deficits helps elucidate patients' hand motor control with functional relevance.

Keywords

Stroke; sensory deficit; power grip; hand; EMG

1. Introduction

This study examined how post-stroke sensory deficits, in addition to motor deficits, plays a role in altered phalanx force direction during static power grip. There are 7 million stroke survivors in the U.S. (Roger et al., 2012). More than two thirds experience unilateral arm and hand functional loss that persists even after rehabilitation therapy (Parker et al., 1986, Gray et al., 1990, Nakayama et al., 1994, Lawrence et al., 2001). The reduced hand function diminishes stroke survivors' ability to complete common gripping tasks and can lead to increased dependence on others to perform daily living activities. Post stroke hand impairment is further exacerbated by somatosensory loss in the hand (Carey and Matyas, 2005, Blennerhassett et al., 2007). Sensory deficits are prevalent after stroke, with

Corresponding author: Na Jin Seo, 151B Rutledge Ave., Charleston, SC 29425, Phone: 843-792-0084, Fax: 843-792-0710, seon@muscd.edu.

7. Conflict of Interest Statement

The authors listed in this manuscript participated in the study design, collection, and preparation of the manuscript. The information conveyed in this article has not been submitted for publication elsewhere except for one author's doctoral dissertation.

approximately 50% to 85% of stroke survivors experiencing tactile and proprioceptive sensory deficits (Carey, 1995, Kim and Choi-Kwon, 1996). Sensory impairment hinders motor performance (Hill et al., 2014) and recovery (Tyson et al., 2008). Yet, sensory deficits are largely overlooked in the assessment of limb function and planning of therapy (Schabrun and Hillier, 2009).

In grip, the tactile sensory feedback is used for the initial force scaling and 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 sensory feedback is important for appropriating the necessary finger force magnitudes and direction for a successful grasp (Robertson and Jones, 1994, Monzée et al., 2001, Nowak et al., 2001, Augurelle et al., 2003, Zatsiorsky and Latash, 2004). Post-stroke sensory deficits have previously been linked to impaired detection of contact with an object (Turton and Butler, 2001), prolonged latencies in pinch grip activation during pinching and lifting tasks (Blennerhassett et al., 2007), problems regulating pinch grip force (Blennerhassett et al., 2007), and increased safety margin during pinch grip (Hermsdorfer et al., 2003).

In addition to its implications on pinch grip force control, post-stroke sensory deficits may account for impairment in power grip force control. Power grip is another type of hand grasp involving all fingers and the palm, which is frequently used in activities of daily living (Napier, 1956). Yet, little knowledge exists regarding stroke survivors' power grip force control. 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 1). Our recent study 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 hereafter) compared to age-matched healthy controls (Enders and Seo, 2015). This 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 force direction for stroke survivors may increase the likelihood of object dropping due to reduced gripping stability and overall grip force. As sensory feedback is important for pinch grip force control (Nowak et al., 2001, Shim et al., 2012), stroke survivors who experience sensory deficits could be at greater risk for altered phalanx force deviation and object dropping during power grip compared to stroke survivors with normal sensation as well as their healthy counterparts.

The goal of this study was to determine the effects of the sensory deficit on stroke survivors' power grip force direction during power grip. Phalanx force direction for stroke survivors with sensory deficits was compared to stroke survivors without sensory deficits and age-matched neurologically-intact healthy controls. Furthermore, muscle activation patterns were examined to determine if a greater extent of phalanx force deviation for those with sensory deficits is associated with an altered muscle activation pattern compared to those with no sensory deficits and healthy controls. The COF between the finger skin and the grip surfaces were also measured to determine any increase in skin slipperiness that might allow

for a greater range of acceptable phalanx force deviation between the subject groups (Cole, 1991). Knowledge of the way sensory impairment compromises power grip control is important to inform stroke rehabilitation practices of the need to treat sensory deficits to enable better motor recovery.

2. Methods

2.1 Subjects

Fourteen chronic stroke survivors with sensory deficits (mean age $\pm$ SD = 59 $\pm$ 12 years, 6 females), 9 chronic stroke survivors without sensory deficit (63 $\pm$ 12 years, 4 females), and 18 age-matched healthy control subjects (61 $\pm$ 10 years, 9 females) participated. All stroke survivors were at least 6 months post stroke. All age-matched controls were free of any neurological conditions and upper extremity injury. Age was not significantly different between any of the groups (t-tests with $p > .05$). The mean hand length (from the wrist crease to the tip of the middle finger) was 18.1, 18.2, and 18.1 cm for stroke survivors with sensory deficits, those without sensory deficits, and controls, respectively, and the hand length was not significantly different between any of the groups (t-tests with $p > .05$). All subjects signed a consent form and followed a protocol approved by the Institutional Review Board.

For stroke survivors, 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). The 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 sensory deficit and stroke survivors without sensory deficit groups, respectively (t-test with $p > .05$). Stroke survivors' median Modified Ashworth Scale (MAS) (Ashworth, 1964) assessing spasticity of the forearm flexor muscles was 1 and 0 (out of a possible 4) for the stroke survivors with and without 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 impairment levels.

Presence of sensory deficits was determined by the Semmes-Weinstein monofilament test (Bell-Krotoski et al., 1993). Scores greater than or equal to 3.61 for both the index and thumb finger indicated sensory deficits (Dellon, 1997), while scores less than 3.61 indicated normal sensation. The monofilament scores for the stroke survivors with sensory deficits ranged from 3.61 to 6.65 with a median score of 3.61.

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 (Enders and Seo, 2011, 2015) (Figure 2). The grip dynamometer was a 50.8 mm diameter cylinder with an opening in the middle where 3 pads were horizontally located to contact each phalanx. Each of the three pads was attached to the bottom of the cylinder through a vertical beam. Each beam was instrumented with four strain gauges in the Wheatstone bridge to measure the normal and proximal-distal shear force of the phalanx.

Due to the limited space inside the grip dynamometer, only 3 beams were fitted, which enabled measurement for three phalanges of one finger at a time. The distal-proximal locations of the three pads were adjusted for each subject to make contact with each phalanx.

Subjects performed power grip at the 100% and 50% maximum effort for five seconds, while normal and proximal-distal shear forces were recorded for individual phalanges. 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 dynamometer. Measurement of phalanx normal and shear forces for the all five fingers, two frictional surfaces, and two effort levels (maximum and 50% of maximum) were repeated at least two times each, and the order of testing was randomized.

In addition, to assess muscle activation patterns, the flexor digitorum superficialis (FDS), extensor digitorum communis (EDC), and first dorsal interosseous (FDI) muscle activities were 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. Specifically, the MVC for the FDS was obtained by placing the hand on the table with the palm side up and asking the subject to flex the hand against resistance from the researcher as hard as possible while the researcher also stabilized the wrist. Similarly, the MVC for the EDC was obtained by asking the subject to place their hand palm side down on the table and to extend the hand against resistance from the researcher. For the FDI muscle, subjects were instructed to place their hand palm down on the table with fingers together. Then, against resistance from a researcher, subjects were asked to abduct their index finger against resistance as hard as possible. The EMG data were recorded for 13 stroke survivors with sensory deficit, 7 stroke survivors without sensory deficit, and 13 healthy controls. Both the phalanx force and EMG data were recorded at 1000 Hz.

Furthermore, the COFs of the finger pad skin and the paper and rubber surfaces were determined using a series of finger drags for the same set of subjects who had EMG recorded. Subjects' thumb and index fingertip pads were pressed against a 6-axis load cell by the investigator at a normal force level of approximately 2 N. Then the finger was pulled in the proximal direction by the investigator 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 (Enders and Seo, 2015) and is investigated here for the two separate stroke groups along with the COF for the rubber surface.

2.3 Data Analysis

A sample grip trial is shown in Figure 3. 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 sensory deficit, and 57% for stroke survivors with 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.

2.4 Statistical Analysis

Force data—A mixed-design ANOVA was conducted to examine how the phalanx force deviation varied for the three subject groups (stroke survivors with sensory deficit, stroke survivors without 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 among the three subject groups, secondary mixed-design ANOVA was conducted for the phalanx normal force with the same model.

EMG and COF data—In addition to the EMG in %MVC, difference in the muscle activation pattern between the subject groups was assessed by examining 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). Two mixed-design ANOVAs were used, one for EMG (%MVC) and the other for the relative FDI and EDC EMGs normalized to FDS EMG, to analyze how muscle activity changed depending on the subject groups. These ANOVAs examined if EMG activity (in %MVC or relative ratio) was significantly different by subject group, effort level, muscle, and interactions between subject group and effort level and between subject group and muscle. For COF data, a mixed-design ANOVA was used to examine differences in COF of the finger skin for the three subject groups, surface, and the interaction between subject group and surface.

For all statistical analysis: Phalanx force deviation data, phalanx normal force data, and EMG data 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. Results

An overview of the results is as follows. The average phalanx force direction and magnitude during static power grip for the two stroke groups and healthy controls are shown in Figure 4 and Figure 5, respectively. 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 sensory deficits had similar *maximum* phalanx normal forces that were not significantly different from each other. Consistent with the previous study (Enders and Seo, 2015), the stroke groups gripped with greater phalanx force deviation compared to healthy controls. The new finding of this study is that stroke survivors with sensory deficits produced power grip with significantly greater phalanx force deviation compared to stroke survivors without sensory deficits and healthy controls. Increased phalanx force deviation for stroke survivors with sensory deficits was associated with an altered muscle activation pattern, but not a change in skin slipperiness. The details of these findings are presented below.

3.1 Increased phalanx force deviation for stroke survivors with sensory deficits compared to stroke survivors without sensory deficits 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 4, ANOVA with $p < .05$). Stroke survivors with sensory deficits produced the largest phalanx force deviations, followed in order by stroke survivors without sensory deficit and healthy individuals (Figure 4a, $p < .05$ for all three Tukey post hoc comparisons among the three subject groups). Compared to healthy controls, phalanx force deviation was increased by 14% for stroke survivors without sensory deficits and 24% for stroke survivors with sensory deficits (Figure 4a). Group comparisons for each condition are shown in Figure 4b-d. Stroke survivors with sensory deficits produced significantly greater phalanx force deviation compared to healthy controls and stroke survivors without sensory deficits during 50% maximum power effort and significantly greater phalanx force deviation compared to healthy controls at maximum power effort (Figure 4c, $p = .04$ for the group and effort level interaction in ANOVA, $p < .001$ for all Tukey post hoc comparisons mentioned). Stroke survivors with sensory deficits produced significantly greater phalanx force deviation with all the phalanges compared to healthy controls and for the distal phalanx compared to stroke survivors without sensory deficits (Figure 4d, $p = .01$ for the group and phalanx interaction in ANOVA, $p < .001$ for the Tukey post-hoc comparisons mentioned). Stroke survivors with sensory deficits produced significantly greater phalanx force deviation with the thumb and index finger compared to healthy controls and for the index finger compared to stroke survivors without sensory deficits (Figure 4e, $p < .001$ for the group and finger interaction in ANOVA, $p < .001$ for the Tukey post-hoc comparisons mentioned). 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 4b, $p < .001$ for the surface main effect but $p > .05$ for the subject group and surface interaction in ANOVA), although it was observed that stroke survivors with sensory deficits changed their phalanx force deviation very little (<1%) compared to the stroke survivors without sensory deficits (7%) and healthy controls (13%).

3.2 Phalanx normal force

Phalanx normal force was similarly reduced for both stroke groups compared to control. 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 5, ANOVA with $p < .01$). Both stroke survivors with and without sensory deficits performed power grip with reduced phalanx normal force during both effort levels compared to healthy controls (Figure 5c, $p < .001$ for the group and effort level interaction in ANOVA, $p < .001$ for Tukey post-hoc comparisons between control and stroke survivors without sensory deficit and between control and stroke survivors with sensory deficits, for each effort level), while the two stroke groups were not significantly different from one another during either effort level (Figure 5c, $p > .05$ for Tukey post-hoc comparison between stroke survivors without vs. stroke survivors with sensory deficits, for each effort level). Stroke survivors with sensory deficits 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 sensory deficits (Figure 5d, $p < .001$ for the ANOVA group and phalanx interaction, $p < .001$ for the Tukey post hoc comparisons mentioned). Both stroke groups produced significantly less phalanx normal force from all the fingers compared to healthy controls (Figure 5e, $p < .001$ for the ANOVA group and finger interaction, $p < .001$ for the two Tukey post-hoc comparisons) and were not significantly different from one another for any finger ($p > .05$ for the Tukey post-hoc comparison between the two stroke groups).

3.3 Altered muscle activity pattern for stroke survivors with sensory deficits

Each muscle's activity during static power grip is shown for the three subject groups in Figure 6a. 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 ($p < .05$ in ANOVA). Specifically, the activation pattern across the three muscles was similar between the healthy controls and stroke survivors without sensory deficits, whereas those with sensory deficits exhibited reduction of the FDI and EDC muscle activities relative to the FDS muscle compared to the other groups (Figure 6a).

This different muscle activity pattern for the sensory deficit group with the reduced FDI and EDC muscle activities relative to the FDS muscle is highlighted in the relative EMG data (Figure 6b). The relative muscle activation pattern was significantly dependent upon subject group, muscle, and the interactions between group and muscle and between group and effort (ANOVA with $p < .05$). Tukey post hoc results showed that the muscle activation pattern was significantly altered for stroke survivors with sensory deficits compared to stroke survivors without sensory deficits and healthy controls (Figure 6b, $p = .01$ for the subject group main effect in ANOVA, $p < .001$ for Tukey post-hoc comparisons between stroke survivors with vs. without sensory deficits and between stroke survivors with sensory deficits vs. healthy controls), while no statistical difference was seen between stroke survivors without sensory deficits and healthy controls ($p > .05$ for Tukey post hoc comparison between stroke survivors without sensory deficits and healthy controls). Specifically, the stroke survivors with sensory deficits had significantly reduced FDI and EDC muscle activities relative to the FDS muscle,

compared to both stroke survivors without sensory deficits and healthy controls (Figure 6b, $p=.004$ for the subject group and muscle interaction in ANOVA, $p<.001$ for Tukey post hoc comparisons between stroke survivors with and without sensory deficits and between stroke survivors with sensory deficits and healthy controls for each muscle, and $p>.05$ for Tukey post hoc comparison between stroke survivors without sensory deficits and healthy controls for each muscle). Such an altered muscle activation pattern for stroke survivors with sensory deficits was found for both the 50% maximum and maximum power grip effort levels (with the same Tukey post hoc comparison results among the three groups found for each effort level).

3.4 Skin COF

There was no significant difference in the COF between any of the subject groups for either surface (Figure 7, ANOVA group main effect and group and surface interaction with $p>.05$, and only surface having $p<.001$). The mean COF of the paper surface was 0.43, similar to previous studies (Buchholz et al., 1988, Gee et al., 2005). The mean COF of the rubber surface was 1.00, similar to a previous study (Seo and Armstrong, 2009).

4. Discussion

Consistent with the previous study (Enders and Seo, 2015), 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 sensory deficits gripped with greater deviation compared to stroke survivors without sensory deficits. This greater phalanx force deviation for stroke survivors with sensory deficits was accompanied by an altered muscle activation pattern, characterized by underactivated FDI and EDC muscles relative to the FDS muscle, compared to both the stroke survivors without sensory deficits and the healthy controls. Stroke survivors without sensory deficits were not significantly different in the muscle activation pattern compared to healthy controls. Differences in the two stroke survivors groups were not the result of differences in motor impairment levels, grip strength, or skin slipperiness.

4.1 Phalanx force direction altered more for stroke survivors with sensory deficits

Stroke survivors with sensation deficits performed a static power grip with phalanx force deviated from the normal direction to a greater extent than stroke survivors without sensation deficits, compared to healthy controls. The effect of 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 (Robertson and Jones, 1994, Hermsdorfer et al., 2003, Blennerhassett et al., 2007). Both stroke 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 (Figure 5c), thus indicating that the group differences in grip control could be related to reduced sensory feedback. Also, the difference in gripping control was most likely not the result of differences in skin slipperiness (Figure 7), 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.

An altered muscle activation pattern was also observed for stroke survivors with sensory deficits in the present study, suggesting that diminished sensory feedback was associated with greater alteration in muscle activity controlling the fingers during power grip. When the muscle activation pattern was examined by quantifying the relative muscle activity to the major gripping muscle of the FDS, it was seen that both the intrinsic FDI and the extrinsic extensor EDC muscles were under-activated compared to the extrinsic flexor FDS muscle for stroke survivors with sensory deficits compared to the other stroke group and healthy controls (Figure 6b). This reduction in the relative muscle activity for the stroke survivors with sensory deficits was somewhat greater for the intrinsic FDI muscle than the EDC muscle (Figure 6b). The intrinsic muscles such as the FDI have been shown to be important for directing the large force generated by the long finger flexor muscles, and the extensor muscles such as the EDC have been shown to be important for stabilizing the joints (Milner and Dhaliwal, 2002). Specifically, reduced activity of the intrinsic muscles including the FDI muscle has previously been shown to lead to increased fingertip force deviation as much as 9° in biomechanical models (Valero-Cuevas et al., 2000). While reduced activity of any single muscle can hamper finger force production in a specific direction, reduced activity of the FDI and EDC muscles severely compromises the finger force production ability as much as, if not to more than, the FDS muscle (Kutch and Valero-Cuevas, 2011). Therefore, the altered muscle activation pattern with underactivated FDI and EDC muscle activities relative to the long finger flexor muscle activity appears to compromise the ability to direct and stabilize the phalanx force and lead to increased phalanx force deviation in stroke survivors with sensory deficits. While the EDC and FDI muscle activity was reduced for stroke survivors with sensory deficits, the FDS muscle activity appeared to be preserved (Figure 6a) potentially due to hyperexcitability of the FDS muscle (Kamper and Rymer, 2001, Cruz et al., 2005, Zaaime et al., 2012). Different muscle activation patterns despite similar Fugl-Meyer Assessment and Chedoke-McMaster Stroke Assessment Hand Section scores could be because these motor functional scores are not sensitive enough to detect the differences in muscle activation patterns between the two stroke groups.

Sensory deficits could be linked to the altered phalanx force direction and altered muscle activation pattern in two ways. First, decreased 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 direction and 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 grip force being produced (Robertson and Jones, 1994, Monzée et al., 2001, Augurelle et al., 2003, Hermsdorfer et al., 2003, Cole, 2006, Blennerhassett et al., 2007), and the finger position with respect to the object surface (Monzée et al., 2001).

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 cortices

(Weiss et al., 2004). Therefore, stroke survivors with reduced sensation could have altered cortical sensorimotor representations, leading to an altered muscle activity pattern and diminished phalanx force control. Specifically, 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 reorganization in the sensorimotor cortex and making them especially affected in stroke survivors with sensory deficits (Figure 6b). In summary, a loss of sensory feedback following stroke could have led to reduced feedback control and adverse cortical reorganization, resulting in altered coordination of muscle activity and increased phalanx force deviation.

4.2 Functional and Clinical Implications

Increased phalanx force deviation can lead to slips and object dropping. Finger force deviating from the direction normal to the surface leads to increased risk of object slippage (MacKenzie and Iberall, 1994, Seo et al., 2010). Therefore, stroke survivors with sensory deficits may be more at risk than stroke survivors without sensory deficits for dropping objects. Specifically, the mean phalanx force deviation was 15.5°, 17.7°, and 19.2° for healthy controls, stroke survivors without sensory deficits, and stroke survivors with sensory deficits, respectively. The threshold for slippage for commonly encountered surfaces such as polyethylene, polished stainless steel, wool, aluminum, and suede is 16.7°, 17.2°, 18.0°, 18.3°, and 21.3°, respectively, based on the COFs reported in literature (Comaish and Bottoms (1971) for polyethylene and wool, El-Shimi (1977) for stainless steel, Buchholz et al. (1988) for aluminum and suede). Thus, on average, stroke survivors with sensory deficits may experience finger slipping and drop objects finished with these surfaces except for suede, while healthy controls would not and stroke survivors without sensory deficits may experience slipping only with polyethylene and polished stainless steel. This finding of increased phalanx force deviation with sensory deficit 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). Note that the stroke survivors studied in this study had, on average, moderate impairment of the hand, and the impact of sensory deficits on power grip force control in stroke survivors with severe impairment may be examined in future studies.

Furthermore, increased phalanx force deviation can also contribute to reduced 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 sensory deficits (calculated by taking the average difference between phalanx resultant force and normal force).

Reduced sensation, along with altered muscle activation patterns and increased phalanx force deviation as seen in the present study, may also interfere with in-hand manipulation, especially when vision is occluded, such as fastening parts, buttoning, zipping, and retrieving objects from pockets or bags. Thus, it follows that stroke survivors without sensory deficits are more likely to report high levels of performance and satisfaction with

valued activities including activities of daily living, compared to stroke survivors with sensory deficits (Hill et al., 2014).

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 support the importance of treating sensory impairment for stroke survivors in addition to motor impairment. Methods exist to improve hand sensation, such as transcutaneous electrical stimulation (Conforto et al., 2007), temporary functional de-afferentation (Sens et al., 2012), subsensory vibrotactile stimulation (Collins et al., 1996, Enders et al., 2013, Kurita et al., 2013, Lakshminarayanan et al., 2015), and tactile coactivation (Dinse et al., 2006, Dinse and Tegenthoff, 2015). The timing of such sensory rehabilitation may optimally be at the acute phase to preserve the sensory cortical representations of the upper limb (Weiss et al., 2004). Combining these sensory enhancement techniques with a sensory re-training (Carey and Matyas, 2005, Chanubol et al., 2012) or conventional motor rehabilitation (Wolf et al., 2006, Seo et al., 2014) could be beneficial to stroke patients in functional recovery. Alternatively, in lieu of treating sensory impairment, training with visual feedback to improve force control may have potential to lead to recovery of motor control (Ellis et al., 2005, Seo et al., 2011).

5. Conclusion

This study indicates that sensation is important for control of phalanx force direction and reduction of sensation post stroke could reduce control of phalanx force direction during static power grip. Stroke survivors with sensory deficits exhibited similar motor impairment levels, similar reduction in grip strength, and similar skin friction as stroke survivors without sensory deficits, but had greater phalanx force deviation. Stroke induced 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 sensory deficit following stroke is responsible for impaired hand grip for stroke survivors. Due to greater phalanx force deviation, stroke survivors with sensory deficits may have an increased risk of dropping grasped objects compared to those without sensory deficits and healthy controls, which hampers their ability to complete daily activities and may require additional attention for sensory deficit during their rehabilitation.

Acknowledgments

This study was supported by funding from an American Heart Association Midwest Affiliate Predoctoral Fellowship 12PRE9320004, a Grant-In-Aid award from the American Society of Biomechanics, the University of Wisconsin-Milwaukee Research Growth Initiative, and the Mary E. Switzer Distinguished Fellowship from the National Institute on Disability and Rehabilitation Research grant number H133F110005. However, those contents do not necessarily represent the policy of the Department of Education, and you should not assume endorsement by the Federal Government. Also, this project was supported by the National Center for Advancing Translational Sciences, National Institutes of Health, through Grant Number 8UL1TR000055. Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH. In addition, the authors would like to thank Dr. Thomas Armstrong and Mr. Charles Woolley for their assistance in developing the custom-made grip dynamometer.

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Figure 1.

During power grip, phalanges produce force (F_{phalanx}) that can be decomposed to normal force (F_{normal}) and shear forces (F_{shear}). Stable grip requires that the phalanx force deviation ($\alpha = \arctangent \text{ of } F_{\text{shear}}/F_{\text{normal}}$) be less than the slip threshold ($\theta = \arctangent \text{ of the coefficient of friction between the finger skin and object}$). If the phalanx force deviation (α) exceeds the slip threshold angle (θ), the phalanx would slip.

Figure 2:
A custom made grip dynamometer measured phalanx forces from each phalanx of a single finger.

Figure 3:

An example trial for a stroke survivor with sensory deficit. On the top, the normal force (solid line) and shear force (segmented line) of the proximal phalanx of the middle finger are shown. On the bottom, the FDS (dotted line), EDC (segmented line) and FDI (solid line) RMS EMGs normalized to the maximum voluntary contraction are shown. The two vertical lines mark the two second window in which the average data were obtained.

Figure 4:

Mean $\pm$ SE phalanx force deviation was largest for stroke survivors with sensory deficits, followed in order by stroke survivors without sensory deficits and healthy controls (a, significant Tukey post hoc comparisons are marked with stars). Mean $\pm$ SE phalanx force deviation is also shown for each surface (b), effort level (c), phalange (d), and finger (e).

Figure 5:

Mean $\pm$ SE phalanx normal force was reduced for both stroke groups compared to healthy controls (a, significant Tukey post hoc comparisons are marked with stars). This reduction was similar for both stroke groups for all surfaces (b), efforts (c), phalanges (d), and fingers (e).

Figure 6:

Mean $\pm$ SE EMG for healthy controls, stroke survivors without sensory deficits, and stroke survivors with sensory deficits (a). Mean $\pm$ SE FDI and EDC EMGs relative to the FDS EMG were reduced for stroke survivors with sensory deficit compared to controls and stroke survivors without sensory deficit (b, significant Tukey post hoc difference for stroke survivors with sensory deficit group compared to other two groups are marked with stars for both relative FDI and EDC EMGs), showing altered muscle activity pattern with particularly reduced FDI and EDC muscle activities for stroke survivors with sensory deficit compared to controls and stroke survivors without sensory deficit. Non-transformed data is shown in the figure.

Figure 7:

Mean $\pm$ SE COF between the finger skin and the paper and rubber surfaces was similar for stroke survivors with sensory deficits, stroke survivors without sensory deficits, and healthy controls. The COF for the rubber surfaces was significantly greater than the paper surface (with the star indicating significant difference). Non-transformed data is shown in the figure.