STROKE INDUCED SENSORY DEFICIT DECREASES PHALANX FORCE CONTROL DURING POWER GRIP

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INTRODUCTION

Many stroke survivors experience not only motor deficit but also somatosensory deficits, especially in the hand [1]. Knowledge regarding the role of this sensory deficit in stroke survivors’ ineffective grip control is sparse. Not to mention little knowledge is available for grip control during power grip. New development of a power grip dynamometer in our laboratory allows investigation of grip force control and force direction during power grip [2]. The objective of this study was to determine the influence of stroke induced sensory deficit on (1) phalanx normal force control and (2) phalanx force trajectory deviation during lifting of an object in power grip. These two characteristics were investigated due to their functional implications for object slippage/dropping in impaired hands [3, 4].

METHODS

Thirteen chronic stroke survivors with tactile sensory deficit, 5 chronic stroke survivors with no tactile sensory deficit, and 12 age-matched neurologically-intact controls participated. Tactile sensory deficit was determined by the Semmes-Weinstein monofilament and Two-Point discrimination tests. Mean motor function and hand spasticity assessed by the upper extremity portion of the Fugl-Meyer Assessment (FM) and Modified Ashworth Scale (MAS), respectively, were similar for the two stroke survivor groups.

Subjects held the custom-made cylindrical grip dynamometer [2] vertically against gravity in power grip with the paretic (stroke survivors) or non-dominant (controls) hand, while each phalanx’s normal force (normal to grip surface) and proximal-distal shear force (tangential to grip surface) were recorded for the thumb, index, and middle fingers. The phalanx force trajectory deviation was quantified as angular deviation of the phalanx force from the direction normal to the grip surface (the arctangent of the ratio of proximal-distal direction shear force to normal force). The grip dynamometer weighed 7N and was covered in a rubber surface.

The phalanx normal force magnitude and phalanx force trajectory deviation observed during the holding phase were compared among the three subject groups (stroke survivors with sensory deficit, stroke survivors without sensory deficit, and healthy controls) using ANOVA and Tukey post hoc analysis. Both sets of data were non-normal and thus transformed using a Box-Cox transformation.

RESULTS AND DISCUSSION

Stroke survivors with sensory deficit produced significantly less phalanx normal force during power grip compared to stroke survivors without sensory deficit and healthy controls (Figure 1, Tukey post hoc, p<.05). In other words, only stroke survivors with sensory deficit displayed reduced safety margin compared to control. Phalanx normal force was significantly dependent upon subject group, finger, and phalanx (ANOVA, p<.05).

Reduced safety margin [3] for stroke survivors with sensory deficit reflects an inability to detect micro-slips and greater likelihood for object dropping. The results also suggest that after stroke, those with sensory deficit behave differently than those without sensory deficit. Reduced safety margin for stroke survivors with sensory deficit and somewhat elevated safety margin for stroke survivors without sensory deficit may explain the previous research finding on no significant change in safety margin post stroke overall [5]. These differences in safety margins between the two stroke groups were not influenced by strength differences between the two groups, as additional analysis for maximum power
grip force showed no significant strength capacity differences between the two stroke groups ($p<.05$).

Only stroke survivors with sensory deficit produced significantly greater phalanx force trajectory deviation compared to healthy controls (Figure 2, Tukey post hoc, $p<.05$). Phalanx force trajectory deviation was significantly dependent upon subject group, phalanx, and the interaction between group and finger (ANOVA, $p<.05$). Increased phalanx force trajectory deviation can increase chances of object slippage for those with sensory deficit due to force trajectories lying closer to the direction tangential to the object’s surface [4].

CONCLUSIONS

Stroke induced sensory deficit, but not motor deficit, plays a role in reduced safety margin and increased phalanx force trajectory deviation during power grip, which can increase the likelihood of dropping or slippage of a grasped object. Tactile sensation is important for efficient grip control (i.e. appropriate safety margins [3] and phalanx force trajectory deviation [4]) to avoid object slippage. Hand motor rehabilitation may need to be preceded by somatosensory rehabilitation to maximize the recovery.

REFERENCES


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Figure 1: Mean ± SE phalanx normal force was significantly less for stroke survivors with sensory deficit compared to stroke survivors without sensory deficit and healthy controls (fingers and phalanges pooled) ($p<.05$).

Figure 2: Mean ± SE phalanx force trajectory deviation was significantly greater for stroke survivors with sensory deficit compared to healthy controls (fingers and phalanges pooled) ($p<.05$).