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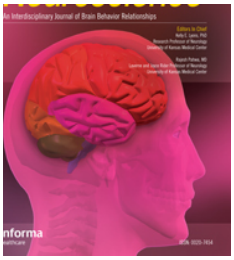

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ORIGINAL ARTICLE

# Upper-extremity spinal reflex inhibition is reproducible and strongly related to grip force poststroke

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*Purpose*: Impaired reflex regulation is assumed to contribute to upper-extremity motor impairment poststroke; however, the relationship between reflex inhibition and motor function remains unclear. To address this question, it is first necessary to determine the reproducibility of reflex responses. The objective of this study was to establish the test–retest reliability of flexor carpi radialis H-reflex inhibition in healthy control and stroke participants and investigate the correlation between H-reflex inhibition and grip strength. *Materials and methods*: Eighteen persons poststroke (mean ± SD: age 63 ± 13 years; 6 ± 5 years poststroke; 13 males) and 16 healthy controls (age: 62 ± 12 years) participated. Reflex inhibition was tested on 2 separate days by conditioning the H-reflex with radial nerve stimulation at two different interstimulus intervals: 13 ms (presynaptic Ia inhibition-PSI) and 0 ms (disynaptic inhibition). Pearson's and intraclass correlation coefficients [two-way mixed model-ICC (1, 2)], and standard error of measurement (SEM) were calculated. *Results*: Relative reliability (ICCs) ranged from good to excellent (0.61–0.78). SEM was low (range 10–19%, stroke; 15–20%, healthy controls). Paretic grip strength and paretic limb PSI revealed a positive correlation ($r = 0.70$; $p < 0.0125$). Disynaptic inhibition and paretic grip strength were not correlated. *Conclusions*: To our knowledge, this is the first study to demonstrate reproducibility of reflex inhibition in individuals poststroke. Furthermore, we quantify smallest real differences, which provide an estimate of the magnitude of effect required to determine a meaningful change, exceeding measurement error. The correlation between PSI and grip strength suggests the potential contribution of PSI to grip force production and upper-extremity motor function.

**KEYWORDS:** Stroke, H-reflex, reliability, presynaptic inhibition, disynaptic inhibition

## Introduction

Following stroke numerous motor sequelae contribute to physical disability and long-term functional motor impairment [1, 2]. Upper-extremity weakness, in particular, impairs wrist and hand function compromising the ability to perform activities of daily living [3].

Upper-extremity motor function is assessed using behavioral and clinical tests, including grip strength. Physiological function, specifically spinal reflex regulation, can be measured using the Hoffmann reflex (H-reflex), a noninvasive technique [4, 5]. Reflex inhibition reflects the net effect of combined facilitatory and inhibitory influences within spinal circuits [5]. Previous studies report impaired reflex inhibition in the flexor carpi radialis (FCR) muscle following stroke [6] compared to healthy controls [4], increased reflex inhibition with wrist splint usage [7], and improvement following a 3-week therapeutic intervention [8].

Here, we studied two types of reflex inhibition: presynaptic inhibition (PSI) – the effect on the presynaptic Ia-afferent terminal, and disynaptic inhibition directed from extensor carpi radialis to FCR [5]. Direct cortical influences on Ia-afferents projecting to FCR motor neurons increase the level of PSI at rest [9]. Following stroke, this cortico-motoneuronal influence can be compromised, resulting in decreased PSI [10]. Voluntary activation of arm muscles (i.e., strength and power) following stroke should thus be related to PSI measured at rest. At least one study has reported that, along with

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improved motor function, PSI can be partially restored in individuals poststroke following rehabilitation [8].

In contrast to PSI, disynaptic inhibition involves activity originating from both agonist and antagonist muscle afferents [11]. Excessive cocontraction between antagonistic muscles has been reported following stroke [12] with impaired disynaptic inhibition argued to be an underlying mechanism [8]. Motor training in both healthy controls [13] and stroke subjects [8] has shown increased disynaptic inhibition in wrist muscles, reducing antagonist muscle cocontraction. Such findings suggest that exercise improves transmission in disynaptic inhibition pathways, and could be an efficacious approach for reducing antagonist cocontraction and improving motor function poststroke.

Accurate assessment of such neurophysiological effects of rehabilitation requires valid, reliable, and meaningful outcome measures. While reflex inhibition has been used to assess rehabilitation effects [8], these findings were reported without information regarding its stability over time. Reproducibility of the FCR H-reflex latency, amplitude [14, 15], and slope of the H- and M-waves has been reported in healthy controls and persons poststroke [16, 17], but the reliability of FCR H-reflex inhibition has not been established in either population.

The first purpose of this study was to examine reliability of FCR H-reflex inhibition in both healthy controls and persons poststroke. Because grip strength is strongly correlated with other tests of upper-extremity function [3], it is frequently used as a proxy for motor impairment. However, the relationship between impaired reflex inhibition and grip force is not known. Therefore, the second purpose of this study was to determine the relationship between reflex inhibition and grip strength poststroke.

## Materials and methods

### Participants

Eighteen persons poststroke: mean ± SD – age 63 ± 13 years (range 31–81), 13 males, 6 ± 5 years poststroke, 11 left-side paretic, stroke location (10 cortical, 3 subcortical, 5 both) and sixteen healthy controls (62 ± 12 years, range 46–79, 10 males) with no documented neurological/orthopedic impairment participated. Inclusion criteria for stroke participants: single, unilateral hemispheric stroke at least 1 year prior and no evidence of brainstem or cerebello-medullar stroke. Exclusion criteria: participating in an ongoing upper extremity therapeutic study, pain in the arm, fixed joint contractures (especially wrist). In accordance with Declaration of Helsinki, all participants provided written, informed consent approved by the Institutional Review Board at University of Florida and Malcom Randall Veteran's Affairs Medical Center. Demographic information and participant characteristics are reported in Table 1.

Table 1. Demographic information of all participants.

| Healthy controls | Mean ± SD | Range |
|---|---|---|
| Age (years) | 62 ± 10 | 46–79 |
| Gender (M/F) | 10/6 | – |
| **Stroke** | **Mean ± SD** | **Range** |
| Age (years) | 63 ± 13 | 31–81 |
| Gender (M/F) | 13/5 | – |
| Time since stroke (years) | 6 ± 5 | 1–18 |
| Hemisphere affected (L/R) | 7/11 | – |
| Lesion location (# of subjects) | | |
| Cortical | 10 | – |
| Subcortical | 3 | – |
| Both | 5 | – |
| Fugl-Meyer (UE; max 66) | 27 ± 18 | 5–58 |
| Wrist flexor MAS (max 4) | | |
| Paretic | 1.90 ± 1.50 | 1–4 |
| Nonparetic | 00–00 | |
| Grip strength | | |
| Paretic (lbs) | 25 ± 20 | 00–70 |
| Nonparetic (lbs) | 83 ± 21 | 41–106 |
| Paretic* | 0.37 ± 0.28 | 0.00–0.91 |
| Nonparetic* | 1.10 ± 0.31 | 0.77–1.42 |

M = male; F = female; R = right; L = left; MAS = modified Ashworth scale; lbs = pounds

*Strength normalized to age/gender–matched healthy controls–Mathiowetz et al. (1985).

### Instrumentation

Participants were seated with the wrist positioned in neutral flexion/extension; forearm mid-prone; elbow 45° flexion; shoulder abduction and flexion 15° each; and neutral shoulder rotation. H-reflexes were evoked in the right-arm in healthy controls and bilaterally in persons poststroke. A previous study found no significant differences in reflex inhibition between sides in healthy controls [10], hence we chose to test only the right side of healthy controls. A custom-fabricated forearm splint [17] provided stabilization and maintained the forearm in mid-prone.

### Protocol

Testing was conducted on two occasions, at the same time of day, separated by at least 24 h and up to 3 days. Clinical assessments included: upper-extremity Fugl-Meyer (FMA), grip force (Jamar dynamometer, Sammons Preston Rolyan, 4 Sammons Court, Bolingbrook,

IL 60440, USA) [18], Box and Blocks Test [19], and modified Ashworth scale (MAS) [20].

## Evoking H and M waves

**Technical aspects:**
H- and M-waves were evoked with a constant-current stimulator (Digitimer DS7A, Digitimer Ltd., 37 Hydeway, Welwyn Garden City, Hertfordshire AL7 3BE, UK) triggered using a second stimulator (S8800 Stimulator and constant-current SIU, Natus Neurology Incorporated – Grass Products, 200 Metro Center Blvd Unit 8, Warwick, RI 02886, USA) at 0.2 Hz, to minimize reflex depression. A preamplified surface electromyography (EMG) electrode (12 mm disk diameter, 17 mm interelectrode distance, ×20 – Motion Lab Systems, Inc., 15045 Old Hammond Highway, Baton Rouge, LA 70816 USA) was placed longitudinally over the FCR belly. A stimulating electrode (30 mm interelectrode distance, convex surfaces) was placed in the medial bicipital groove and stabilized with a custom-fabricated thermoplastic clamp [17]. The ground electrode was positioned over the acromion process. Data were collected at 10 kHz, band-pass filtered (20 Hz–2000 Hz), and stored on disk for offline analysis (PowerLab A/D converter and LabChart Software, Version 6.1.3, ADInstruments, Inc., 2205 Executive Circle, Colorado Springs, CO 80906, USA).

**Stimulation procedure:**
The stimulating electrode for median nerve was adjusted to evoke FCR H-reflexes in the absence of M-waves. Stimulation intensity was slowly increased to elicit a maximal H-wave ($H_{max}$) and was used as the test intensity for assessing reflex inhibition. Maximal FCR muscle motor response ($M_{max}$) was measured when increasing current intensity increased M-wave amplitude no further. To control for potential effects of background muscle activation, all H-reflex amplitudes were normalized using the average EMG over the period 35 ms prior to stimulation. Peak-to-peak amplitude was calculated for all H-reflexes and M-waves; H reflex amplitude was expressed relative to the peak-to-peak amplitude of $M_{max}$.

**Reflex inhibition parameters:**
To test reflex inhibition, the radial nerve was stimulated in the lower arm on the lateral aspect where the nerve leaves the spiral groove. The tester palpated the extensor carpi radialis muscle belly for a twitch contraction. Motor threshold (MT) was determined as the lowest stimulation intensity at which a muscle twitch contraction was palpable. PSI was induced by conditioning the FCR H-reflex with radial nerve stimulation at 95% of MT (1 ms duration) 13 ms prior to median nerve stimulation [10]. Disynaptic inhibition was induced by stimulating both radial and median nerves simultaneously. The conditioned H-reflex ($H_{conditioned}$) was defined as the H-reflex evoked with accompanying radial nerve stimulation (PSI and disynaptic inhibition) and unconditioned H-reflex ($H_{unconditioned}$) was defined as the H-reflex evoked without accompanying radial nerve stimulation. Reflex inhibition was calculated as: $[(1 - (H_{conditioned}/H_{unconditioned})) \times 100]$ where higher, positive values indicate greater inhibition while negative values indicate facilitation.

## Statistical Analysis

Relative reliability was evaluated using intraclass correlation coefficients [two-way mixed model-ICC (1, 2)] and one-way ANOVA. Absolute reliability, that is within-subject reliability or measurement error, was evaluated by calculating the standard error of measurement (SEM) and smallest real difference (SRD) and constructing Bland-Altman plots [17]. SEM and SRD were calculated using the following formulae:

SEM = $\sqrt{}$(within-subject mean square – from ANOVA table);
%SEM = SEM/Mean (2 days)*100, where mean (2 days) is the mean value across days 1 and 2.
SRD = $1.96 \times \sqrt{2} \times$ SEM;
%SRD = SRD/Mean (2 days)*100

Grip force was normalized to age-, gender- and side-matched normative grip strength data reported by Mathiowetz et al. [21]. Paired *t*-tests were used to assess for differences in grip strength between paretic and nonparetic sides. We also calculated Pearson correlation coefficients between PSI, disynaptic inhibition, and clinical tests. We performed four correlations. After correction for multiple tests (i.e., Bonferroni, $p = 0.05/4$) the level to reach statistical significance was adjusted to $p = 0.0125$. All statistical analyses were performed using SPSS software version 17.0 (IBM SPSS, IBM Corporation, 1 New Orchard Road, Armonk, New York 10504-1722 USA). Plots were prepared using an Excel add-in (XL Toolbox 2008–2011 Daniel Kraus, University of Würzburg, Sanderring 2, 97070 Würzburg, Germany).

## Results

No statistical differences in age were revealed between poststroke and healthy controls ($p = 0.93$). Figure 1 shows a representative H-reflex (with and without conditioning) from one healthy control and one subject poststroke.

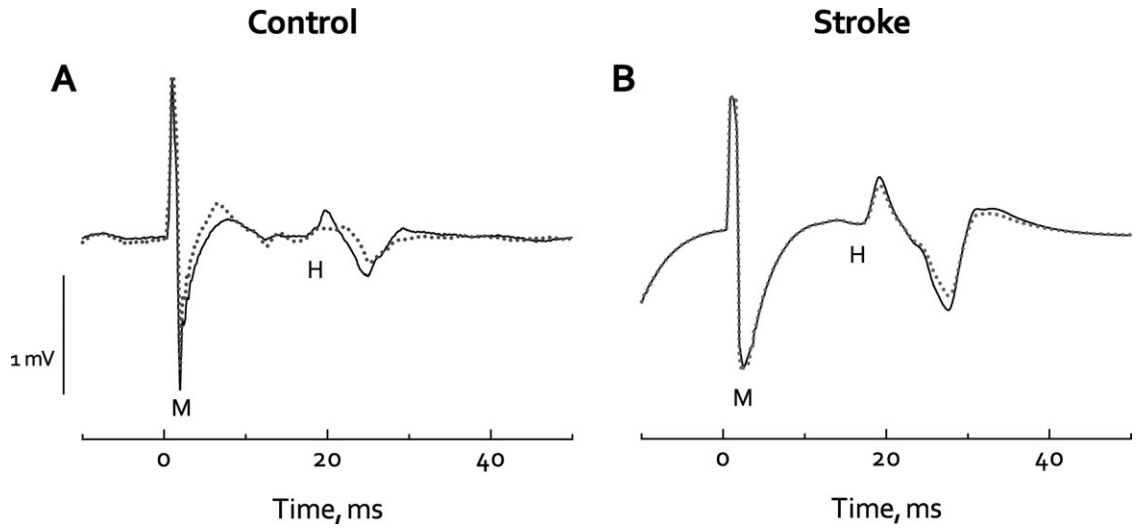


Figure 1. Representative H-reflexes with (dotted line) and without (solid line) conditioning illustrating PSI in one Healthy Control (44%) and one individual post-stroke (14%).

## Healthy controls

### Relative reliability

ICCs for mean PSI and disynaptic inhibition between sessions were 0.77 and 0.74, respectively (Table 2). ICCs exceeding 0.75 reflect excellent reliability [22]. Based on this criterion, ICCs for both PSI and disynaptic inhibition revealed very good to excellent reliability [22].

### Absolute reliability

SEM, expressed as a percentage of the mean, was 15% for PSI and 20% for disynaptic inhibition. SRD, expressed as percentage of the mean scores between days, was 41% and 56%, respectively, for PSI and disynaptic inhibition. Relative and absolutely reliability scores can be found in Table 2.

Bland-Altman plots revealed no systematic trends (Figure 2). The mean reflex amplitude (average of days 1 and 2) for PSI and disynaptic inhibition in healthy controls was 0.79 ± 0.23 (i.e., 21% inhibition) and 0.91 ± 0.33 (i.e., 9% inhibition), respectively.

## Persons Poststroke

Persons poststroke revealed the following characteristics: upper-extremity FMA score 27 ± 18.4 (total 66 points; range 5–58); paretic wrist MAS 1.9 ± 1.5 (total 4 points); Box and Blocks Test – paretic 3.7 ± 9.8 and nonparetic 61.7 ± 6.4 blocks per minute.

We were unable to elicit FCR H-reflexes in four persons poststroke. One additional participant was unable to return for the second testing session. Accordingly, we report results for 13 individuals post-stroke.

### Relative reliability

Test–retest reliability ranged from good to excellent. ICCs for mean PSI and disynaptic inhibition in the paretic arm were 0.61 and 0.68 and in the nonparetic arm were 0.78 and 0.78, respectively (Table 2).

Table 2. Relative and absolute reliability scores for all subjects.

| | | | Stroke | | | |
|---|---|---|---|---|---|---|
| | Healthy controls | | Paretic side | | Nonparetic side | |
| Statistic | PSI | Disynaptic inhibition | PSI | Disynaptic inhibition | PSI | Disynaptic inhibition |
| ICCs | 0.77 | 0.74 | 0.61 | 0.68 | 0.78 | 0.78 |
| 95% CI | 0.08 to −0.11 | −0.03 to −0.27 | 0.11 to −0.05 | −0.01 to −0.15 | 0.05 to −0.12 | 0.06 to −0.17 |
| SEM | 0.12 | 0.18 | 0.10 | 0.10 | 0.11 | 0.15 |
| %SEM | 15% | 20% | 11% | 10% | 13% | 19% |
| SRD | 0.33 | 0.51 | 0.29 | 0.29 | 0.31 | 0.43 |
| %SRD | 41% | 56% | 31% | 29% | 35% | 52% |

%SEM and %SRD are reported as percentage of the combined mean H-reflex amplitude from both days of testing; CI = confidence interval.

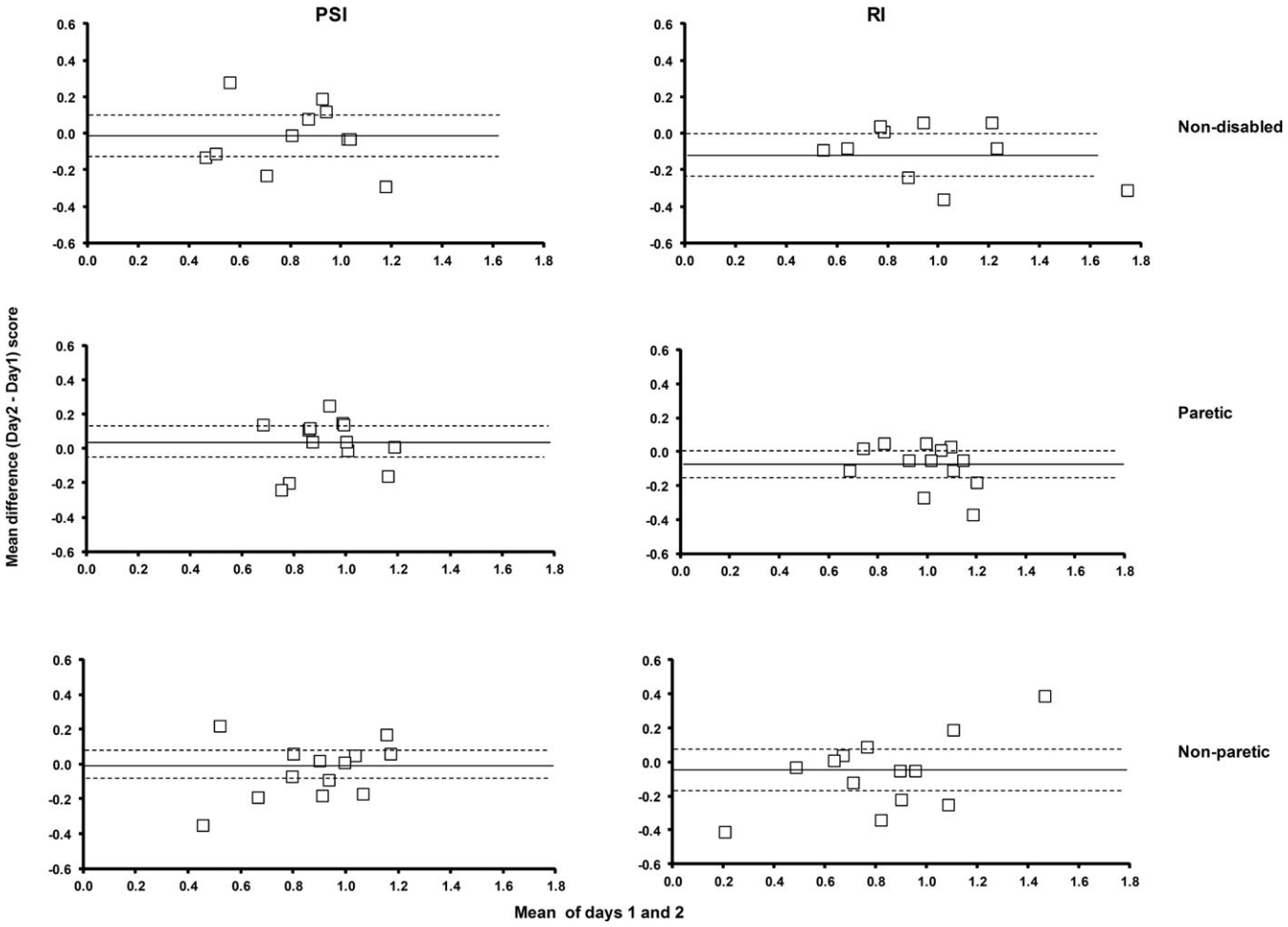


Figure 2. Bland-Altman plots for Presynaptic Inhibition (PSI) and Disynaptic Inhibition (RI). Solid line represents mean difference between days 1 and 2, dashed line represents 95% confidence interval. PSI (mean difference (95% CI)) – healthy controls: (−0.01 (−0.1–0.1)); paretic: (0.03 (−0.1–0.1)); nonparetic (−0.04 (−0.1–0.1). RI (mean difference (95% CI)) – healthy control: (−0.15 (−0.3–0.0); paretic (−0.08 (−0.2–0.0); nonparetic (−0.06 (−0.2–0.1)).

### Absolute reliability

SEM expressed as a percentage of the mean for PSI and disynaptic inhibition in the paretic arm was 11% and 10% and in the nonparetic arm was 13% and 19%, respectively. SRD expressed as percentage of the mean scores between days for PSI and disynaptic inhibition in the paretic arm was 31% and 29% and on the nonparetic arm 35% and 52%, respectively.

Bland–Altman plots revealed no systematic trends between days (Figure 2).

The amplitude (average of days 1 and 2) for PSI and disynaptic inhibition on the paretic side was $0.93 \pm 0.15$ (i.e., 7% inhibition) and $1.00 \pm 0.16$ (i.e., no inhibition) and on the nonparetic side was $0.88 \pm 0.22$ (i.e., 12% inhibition) and $0.82 \pm 0.30$ (i.e., 18% inhibition), respectively.

The magnitudes of both PSI and disynaptic inhibition across both sessions are summarized in Table 3.

## Grip strength

Asymmetric grip strength impairment poststroke was reflected in an ~60% lower paretic grip force compared to the nonparetic side. Mean paretic grip force (pounds, nonnormalized) was $25.5 \pm 19.8$ lbs while nonparetic grip force was $82.6 \pm 20.7$ lbs. Normalized paretic grip strength ($0.37 \pm 0.28$) was significantly lower than the nonparetic side ($1.11 \pm 0.32$; $p < 0.0001$). Both raw and normalized grip force were significantly greater on the nonparetic side, but nonparetic grip strength did not differ significantly from age-referenced normative data ($p = 0.25$).

## Correlation

A significant, positive correlation was revealed between normalized paretic side grip strength and PSI (Pearson's

Table 3. Summary of reflex inhibition across sessions.

| Healthy controls | Day 1 | Day 2 |
|---|---|---|
| PSI | 0.76 ± 0.27 | 0.81 ± 0.24 |
| Disynaptic inhibition | 0.95 ± 0.37 | 0.90 ± 0.32 |
| Stroke | Day 1 | Day 2 |
| PSI – paretic | 0.92 ± 0.16 | 0.94 ± 0.17 |
| PSI – nonparetic | 0.90 ± 0.20 | 0.86 ± 0.26 |
| Disynaptic inhibition – paretic | 1.04 ± 0.19 | 0.96 ± 0.15 |
| Disynaptic inhibition – nonparetic | 0.86 ± 0.26 | 0.79 ± 0.38 |

coefficient – $r = 0.70$; $p = 0.002$; Figure 3). While not statistically significant, our data also suggest a negative correlation between paretic grip strength and nonparetic side PSI ($r = -0.50$; $p = 0.034$; Figure 3). In contrast, the correlation between grip strength and disynaptic inhibition was poor for both paretic ($r = -0.11$, $p = 0.35$) and nonparetic sides ($r = 0.27$, $p = 0.17$).

## Discussion

Our main findings are: (1) PSI and disynaptic inhibition can be reliably evoked in both healthy controls and persons poststroke – in both paretic and nonparetic arms; (2) paretic grip strength is positively correlated with paretic PSI, measured at rest.

### Physiologic Effects on H-reflex Inhibition

Our results reveal lower PSI magnitude and amplitude of conditioned reflex responses (range 7–24% inhibition) compared to those reported by Lamy et al. (range: 16–43% inhibition) [10]. These differences are likely methodological. Stimulation frequency and aging are both known to reduce the magnitude of PSI [5], thus these factors may contribute to our observations of less inhibition. Contrasted with Lamy, we evoked H-reflexes at lower stimulation frequency (0.2 Hz vs. 0.3 Hz [10]) and studied participants approximately 10 years older. Stroke chronicity may also contribute to differences in our respective results. Participants studied by Lamy et al. [10] were 6.5 months (median) poststroke, whereas all our participants were >1 year, poststroke. Lamy further divided participants into acute and chronic groups reporting equally impaired PSI regardless of chronicity [10]. However, the median in their chronic group was 17 months poststroke. Thus, the effect of greater stroke chronicity (>2 years) on PSI remains untested.

While previous studies have reported significant FCR PSI and disynaptic inhibition (~75%) in healthy individuals [23], we observed markedly less inhibition (21% PSI and 9% disynaptic inhibition). These differences are consistent with age-related changes in the spinal reflex circuitry [5] which motivate carefully age-matching healthy controls and stroke participants to account for effects of aging, in the absence of pathology, and greater incidence of stroke with advancing age [1].

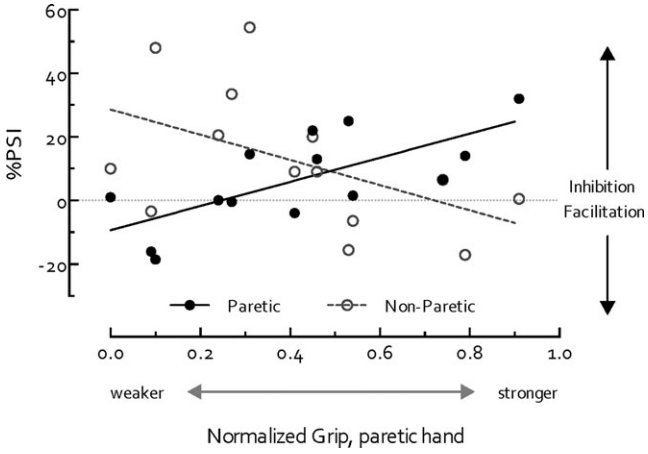


Figure 3. PSI correlates with paretic grip strength. Paretic side grip force normalized to age-, gender-, and side- matched reference data from healthy individuals [18] correlates strongly with PSI measured at rest.

### PSI – mechanism and relationship with grip strength

**Resting muscle:**

The strong positive correlation between paretic grip strength and PSI suggests that reduced PSI, measured at rest, is associated with lower grip force production poststroke. Cortical influences on spinal motor pools are known to increase PSI at rest [24]. Wrist and hand muscles have well-developed direct corticomotoneuronal connections [24], but stroke-related impairment of these descending pathways may decrease PSI and contribute to impaired grip force, as we observed. A previous study [22] showed that improvement in motor function poststroke also increased PSI supports our findings of a positive relationship between greater grip strength and greater PSI. Lower PSI of the wrist flexors would result in increased activation of the wrist flexors. Effective grip force production involves integration of descending neural drive with afferent signals in the spinal circuitry. Position-specific tasks, such as gripping, require synergistic coactivation between wrist extensor and flexor muscles so that optimal wrist extension for effective muscle length-tension relationship can be maintained [25]. Thus, increased activation of wrist flexors can disrupt the length-tension

relationship of the long finger flexors and indirectly impair grip force production.

**Contracting muscle**

In active, contracting muscle, PSI decreases with increased (agonist and antagonist) wrist muscle activation [26]. Reduced PSI in the active state is argued to allow a greater contribution of Ia-afferent activity to monosynaptic stretch reflexes, facilitating force production [26]. Here we measured PSI at rest. The relationship between impaired grip force and PSI during active muscle contraction likely differs. Although PSI is decreased poststroke when tested during rest; the effect of stroke on PSI tested during active muscle contraction is not known. Future studies should explore the effect of active muscle contraction on the magnitude of PSI poststroke.

PSI onto Ia-afferents projecting to flexor motor neurons is also decreased at the onset of wrist extensor muscle activation when the wrist flexors are inactive [26]. This observation suggests that activation of wrist extensors decreases flexor PSI. However, it is not clear if cocontraction of wrist flexors and extensors creates an additional decrease in flexor PSI. Furthermore, it has been suggested that the PSI mediating interneurons may be shared between wrist flexors and extensors [26]. These findings are congruent with the functional role of the wrist muscles. Effective grip force requires cocontraction of wrist flexors and extensors, thus these muscles work synergistically, rather than antagonistically, during gripping [27].

Although not statistically significant after correction for multiple comparisons, our data also suggest a negative correlation between paretic grip strength and nonparetic side PSI. This inverse relationship between paretic and nonparetic side PSI compared to paretic grip is consistent with compensatory responses in the spinal circuitry (i.e., reactive plasticity [28, 29]). Of note, both disynaptic inhibition and PSI were impaired on the so-called nonparetic side. Chronic decreased use of the paretic limb may also exacerbate the pathological reduction of PSI due to stroke. For example, reduced PSI has been reported in response to wrist and ankle immobilization in healthy controls [30]. With greater paretic side weakness, the nonparetic side is increasingly employed during daily functional activities. Repeated compensatory use of the nonparetic limb would enhance nonparetic side motor function and lead to improved efficacy of motor pathways [29].

A recent study reported normalization of PSI (at rest), grip strength, and upper-extremity motor function after a 3-week intervention in persons chronic poststroke [8]. Pathologically reduced PSI, revealed as reflex facilitation prior to training, increased significantly (~40%) posttraining [8]. In agreement with their findings [8] our results illustrate that several of the more impaired individuals poststroke revealed H-reflex facilitation, rather than inhibition, on both sides (Figure 3). Taken together, the strong positive correlation we observed between paretic grip strength and PSI and the concomitant increase in grip force and PSI reported by Fujiwara et al. [22], leads us to hypothesize that increased PSI, at rest, could reveal a mechanism of recovery and therefore could be used to monitor neurophysiological changes underlying rehabilitation.

Based on our calculations, a change of 31% (%SRD) indicates a change in PSI exceeding measurement noise. Fujiwara et al. (2008) reported an ~40% improvement in PSI postintervention which, interpreted in light of the current results, suggests their participants exhibited a genuine physiological change. Future studies can use %SRD criteria to determine whether changes in PSI are physiologically meaningful.

## Level of disynaptic inhibition poststroke

Our results also suggest that, unlike PSI, disynaptic inhibition may not contribute significantly to functional performance involving wrist joint movements poststroke. Production of grip force involves simultaneous cocontraction of wrist flexor and extensor muscles to maintain an optimal length tension relationship of the long finger flexors. Since grip requires some level of synergistic activation of antagonist muscles, the disynaptic inhibition pathway may not be a critical mechanism underlying this task. Unlike other joints, tasks requiring alternate wrist flexion/extension movements are relatively few, whereas the majority of the activities of daily living involve adoption of an extended wrist position [31]. Consistent with these observations, immobilization in healthy individuals has shown a loss of PSI but not disynaptic inhibition [30]. Thus, it appears that abnormal antagonist muscle activity, or impaired disynaptic inhibition, is not directly related to functional impairments involving wrist movements such as grip strength.

## Study Limitations

Our study was done in subjects with chronic stroke and future studies need to examine reliability and association with grip strength in an acute patient group. Since age is related to neurological changes, our results cannot be generalized to persons poststroke with ages $<50$ years or $>70$ years. The impact of hand dominance – both in premorbid dominance in persons post-stroke and healthy control subjects – on reliability was not assessed in this study.

## Conclusion

Here we established test–retest reliability of two types of reflex inhibition, of which PSI is strongly correlated with paretic grip strength. %SEM and %SRD reported here have not been previously established for reflex inhibition. A change in paretic PSI >31% (%SRD) indicates a meaningful, physiological change in PSI. Results of the present study thus aid discrimination of genuine, physiological changes contributing to recovery of motor function poststroke.

### Declaration of Interests

Dr. Phadke has received a grant in the past from Allergan Inc. for a study not related to the current work. Other authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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