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A Pilot Study to Measure Upper Extremity H-reflexes Following Neuromuscular Electrical Stimulation Therapy after Stroke

Ann M. Stowe
UT Southwestern Medical Center

L. Hughes-Zahner
Kansas University Medical Center

V. K. Barnes
Kansas University Medical Center

L. L. Herbelin
Kansas University Medical Center,

Sheila M. Schindler-Ivens
Marquette University, sheila.schindler-ivens@marquette.edu

See next page for additional authors

Authors

Ann M. Stowe, L. Hughes-Zahner, V. K. Barnes, L. L. Herbelin, Sheila M. Schindler-Ivens, and B. M. Quaney

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A.M. Stowe
Landon Center on Aging, Kansas University Medical Center, Kansas City, KS

L. Hughes-Zahner
Landon Center on Aging, Kansas University Medical Center, Kansas City, KS

V.K. Barnes
Landon Center on Aging, Kansas University Medical Center, Kansas City, KS

L.L. Herbelin
Department of Neurology, Kansas University Medical Center, Kansas City, KS

S.M. Schindler-Ivens
Department of Physical Therapy, Marquette University, Milwaukee, WI

B.M. Quaney

Landon Center on Aging, Kansas University Medical Center,
Department of Neurology, Kansas University Medical Center,
Kansas City, KS
Department of Allied Health, Washburn University,
Topeka, KS

Abstract

Upper extremity (UE) hemiparesis persists after stroke, limiting hand function. Neuromuscular electrical stimulation (NMES) is an effective intervention to improve UE recovery, although the underlying mechanisms are not fully understood. Our objective was to establish a reliable protocol to measure UE agonist–antagonist forearm monosynaptic reflexes in a pilot study to determine if NMES improves wrist function after stroke. We established the between-day reliability of the H-reflex in the extensor carpi radialis longus (ECRL) and flexor carpi radialis (FCR) musculature for individuals with prior stroke (n = 18). The same-day generation of ECRL/FCR H-reflex recruitment curves was well tolerated, regardless of age or UE spasticity. The between-day reliability of the ECRL H-reflex was enhanced above FCR, similar to healthy subjects [20], with the Hmax the most reliable parameter quantified in both muscles. H-reflex and functional measures following NMES show the potential for NMES-induced increases in ECRL Hmax, but confirmation requires a larger clinical study. Our initial results support the safe, easy, and efficacious use of in-home NMES, and establish a potential method to measure UE monosynaptic reflexes after stroke.

Keywords: Stroke, H-reflex, FCR, ECRL, Upper extremity spasticity, Neuromuscular electrical stimulation

1. Introduction

According to the American Heart Association, stroke is the leading cause of long-term adult disability, with annual healthcare costs exceeding $73 billion. Stroke-related upper extremity (UE) hemiparesis limits voluntary finger and wrist extension, and decreases hand function. Recovery from UE hemiparesis continues for months and is a self-reported major obstacle to quality of life [1], reducing the potential for the stroke survivor to live independently at home [4]. Physical therapy may improve UE hemiparesis, although the 'best

practices' regarding the specific modality, frequency or duration of therapy to reduce stroke-related disability require further investigation.

Neuromuscular electrical stimulation (NMES) is a therapeutic intervention that delivers electrical impulses through the skin to repeatedly activate muscles [2]. NMES facilitates UE motor recovery in paretic limbs during acute [8] and chronic [19,17] stroke. Our data showed that wrist and hand impairment was significantly improved in a small sample of individuals with chronic stroke after only two weeks of NMES therapy [17]. The mechanisms underlying NMES-driven changes in motor function remain unclear, but may include enhanced cortical plasticity [19,10] and motor unit-derived CNS plasticity [15]. We hypothesized that NMES delivery to the UE of chronic stroke survivors would also modulate the excitability of the spinal reflexes, thus reducing spasticity and improving motor function.

Determining monosynaptic reflex changes requires a reliable measure with low variability. We developed a protocol to measure the Hoffman (H)-reflex in both the extensor carpi radialis longus (ECRL) and the flexor carpi radialis (FCR) across days in healthy adults [20]. The assessment of agonist–antagonist muscles is key, as stroke suppresses the ECRL H-reflex and consequent EMG activity [12], which exacerbates FCR spasticity [16]. The first purpose of the pilot study was to determine if ECRL/FCR H-reflexes could be reliably measured between days in the affected extremity of participants with chronic stroke. Our second purpose was to quantify H-reflex plasticity following NMES intervention in the affected UE to determine if H-reflex modulations contribute to the beneficial effects of NMES therapy. Our preliminary data suggest that measuring ECRL/FCR H-reflexes is safe and well tolerated, and UE spasticity does not affect between-day H-reflex measurement. In a small cohort of subjects, NMES increases ECRL activity, although we lack adequate power to conclude a relation to improved motor function. Our results establish a protocol to measure UE H-reflexes in a larger clinical trial to advance our understanding of the mechanisms by which NMES improves functional recovery after stroke-related UE hemiparesis.

2. Methods

Eighteen participants with a confirmed diagnosis of chronic ischemic stroke (25–82 years of age; 10 M; 8 Fe; 62 ± 15 years) participated in this study ([Table 1](#)). All participants but one lived at home, and all gave written informed consent. We enrolled stroke survivors 1–10 years post-stroke. Participants were not excluded based on passive or active range of motion of the affected UE, but exclusion criteria included: (1) pregnancy; (2) cardiac pacemaker; (3) neurodegenerative disease; (4) tennis elbow/carpal tunnel syndrome; (5) UE pain. All participants had a Mini-Mental Status Exam ≥ 24, written physician approval for participation, and no concurrent rehabilitative therapies. The Human Subjects Institutional Review Board for the Kansas University Medical Center approved the experimental design.

Table 1. CVA Participants.

Subjects	Gender	Age, y	Stroke, y	Treatment group	Affected hemisphere	Orpington (0–5.2 points)
1	M	75	7	I/C	Right	2
2	M	75	5	C/I	Right	2.4
3	Fe	58	1	I	Brain stem	2.8
4	M	60	5	C	Right	2.4
5	Fe	46	4	C	Right	2
6	M	63	8	I/C	Bilateral	4
7	M	67	5	C/I	Left	2
8	Fe	43	3	I	Left	2.4
9	Fe	76	10	I	Right	4
10	Fe	25	3	C/I	Right	1.6
11	Fe	77	2	I	Right	3.2
12	M	77	5	I	Right	2

Subjects	Gender	Age, y	Stroke, y	Treatment group	Affected hemisphere	Orpington (0–5.2 points)
13	M	63	6	I	Right	3.2
14	M	57	3	I	Right	3.6
15	Fe	53	3	C/I	Left	3.6
16	Fe	82	6	C/I	Left	3.6
17	M	71	4	C	Left	N/A
18	M	59	3	I	Right	3.2

y, year; M, male; F, female; I, intervention; C, control.

3. Experimental design

On the first day, baseline stroke-related UE dysfunction was established (Table 1). The affected UE ECRL and FCR H-reflexes were measured over two consecutive days ('Baseline') before being randomized by coin toss into Control or Intervention. Intervention underwent NMES 30 min/day/10d, while Control continuously ambulated at a self-selected velocity for 30 min/day/10d. H-reflex and UE evaluation occurred immediately following NMES (Post) and 2 weeks later (Retention). Testing order was counterbalanced between days, and sessions were scheduled at the same time each day. After Retention, Control participants that chose to enter the Intervention group began NMES. Two Intervention participants returned at 3 month and 6 month following testing to participate in Control, at times when we assume there was no effect of NMES on UE motor performance.

At baseline, Orpington Prognostic Test established stroke severity [22]. Additional clinical tests of UE motor performance, speed, and function performed at every timepoint included: (1) Stroke impact scale (SIS), including the physical domain to assess UE function [5]; (2) modified Ashworth spasticity scale (MASS)(6); (3) Box and Block (BB) test to measure manual dexterity (6); (4) Fugl–Meyer (FM) test of sensorimotor impairment (6). Two participants were unable to complete BB due to severely impaired UE range of motion.

4. H-reflex measurement

We previously established methods for measuring ECRL and FCR H-reflexes [20]. For this study, we measured the H-reflex in the affected UE contralateral to the lesioned hemisphere. Briefly, a constant current stimulator and isolation unit was used (Digitimer DS7A, Hertfordshire, England; 50 μA–200 mA; total output capability – 400 V) with bipolar surface electrodes (Ambu; Ballerup, Denmark) placed over the radial [14] or median [9] nerve to elicit ECRL and FCR H-reflexes, respectively (Fig. 1). EMG signals were recorded (DelSys Inc. Boston, MA) and pre amplified before remote differential amplification. Data were sampled online (10,000 Hz) using a 16-bit analog to digital converter (National Instruments; Austin, TX) with a custom-designed data acquisition program (Labview, National Instruments). In order to optimize H-reflex signals and minimize fatigue, yet maintain the UE postures between test sessions, we chose a relaxed ECRL limb position of pronation/wrist extension, and supination/wrist flexion for FCR recordings [20]. A 227 g weight was held and tolerated by all participants except three (subjects 6, 11, 16), who lacked UE strength to hold the weight during testing. No H-reflex was recorded in FCR (subjects 15, 16) or ECRL (subjects 4, 9), but was found in the antagonist muscle; so data from these participants were not excluded. During testing, stimulation intensity was increased in 0.2–0.3 mA increments from below H-reflex threshold to the point on the recruitment curve where H-reflex amplitude declined; then stimulation intensity was increased in larger increments (~5 mA) until maximum M-wave amplitude. Three pulses (1 ms/0.2 Hz) were delivered at each intensity level with a 5 ms inter-pulse interval to minimize muscle fatigue.
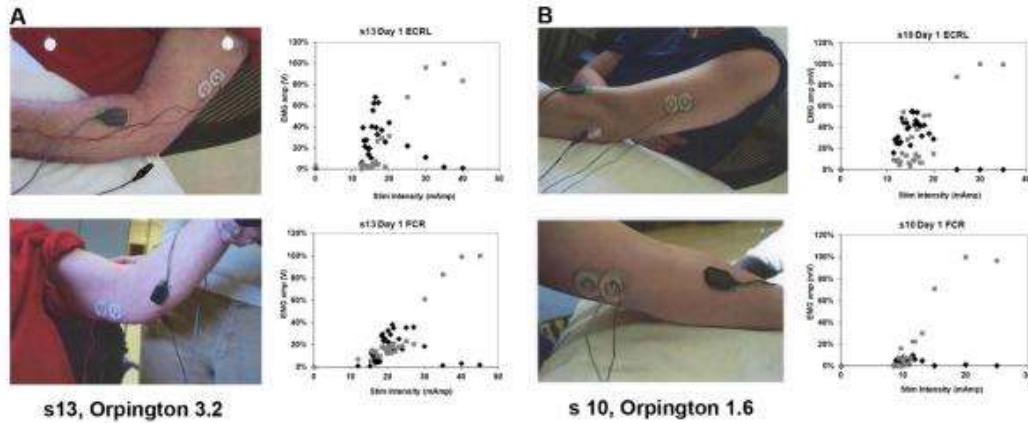
Fig. 1. Stimulation parameters in the affected UE. The stimulating and recording electrode placement are shown in the left panels, and the corresponding H-reflex (black diamonds) and the M wave (grey squares) recruitment curves in the right panels for ECRL and FCR of subjects (A) 13 and (B) 10.

5. NMES protocol

NMES protocol has been previously reported from our laboratory [17]. On Day 2, Intervention participants received the first NMES intervention using a portable electrical stimulator (Rehabilicare; Windham, NH), with electrode placement determined by a physical therapist. A symmetrical, biphasic waveform (300 μs pulse width; 40 Hz; 2 s on/off ramp; 6 s hold; 20 s rest) was applied to the affected UE in alternating extensor/flexor muscle contraction (30 min), approximately 60 muscle contractions/session [15]. After sufficient training, participants self-administered NMES at home, for 9 days/30 min/sessions, with additional training for the spouse/caregiver when requested. Stimulus intensity was adjusted for individual subject tolerance at a level which produced a visible muscle contraction without discomfort.

6. Data processing and analysis

Peak-to-peak amplitudes for between-day reliability were calculated (Matlab; MathWorks-Natick, MA) [20]. Mean EMG amplitude for the H-reflex and M-wave were computed at each intensity and expressed as a proportion of the maximum M-wave to calculate the H-reflex peak amplitude (Hmax), gain (HGN, bestfit slope of the rise to Hmax), and threshold (HTH, *x*-intercept of the HGN), each a descriptor

of the monosynaptic reflex excitability [20]. We also used an alternative visual (visHTH) method to determine the threshold based on the first visual sighting of the H-reflex during EMG data collection [20]. Recruitment curves were analyzed by an unbiased observer who determined atypical recruitment curves due to intermittent EMG signal, high signal:noise ratio, lack of a confirmed EMG plateau for Mmax, or cross-talk EMG signals from other activated muscles. Of the 93 recruitment curves recorded in participants, 71 were established as physiologically representative, a similar percentage to prior results [20].

7. Statistical analysis

Mean values ± standard deviations (SD) were calculated on each day for the FCR and ECRL muscles. Between days 1 and 2, paired *t*-tests were performed (muscle× Day) to assess significant changes for each variable ($p < 0.05$), and repeated measures ANOVA between day 2 (pre), post, and retention time points. Interclass correlation coefficients (ICCs) were calculated between days 1 and 2, as were typical error and typical percent error. The latter computations represented within-subject standard deviation and provided an indication of required treatment effect to be clinically meaningful [7].

8. Results

8.1. Establishing a reliable UE H-reflex measure after stroke

Throughout testing, the EMG signal for the H-reflex and M wave (e.g. latency of signal onset, waveform), and generation of recruitment curves (Fig. 1) in the affected UE did not differ from healthy subjects [20]. Table 2 and Fig. 2 depict between-day group means and individual subject variation for Hmax, HGN, HTH, and visHTH. Typical error and typical percent error are also presented for each H-reflex measure. Overall, the ECRL H-reflex yielded higher ICCs, and thus more reliable measures, than the FCR. For the ECRL muscle, between-day analysis for Hmax was dependable (ICC = 0.71), although the Hmax decreased amplitude on the second day (day 1, 48% Mmax vs. day 2, 38% Mmax; $p < 0.05$). The ECRL HGN also

exhibited fair reliability (ICC = 0.74) despite large individual between-day changes in amplitude. Neither HTH nor visHTH method for determining threshold could be reliably measured in the ECRL. The FCR Hmax showed fair reliability (ICC = 0.62), with a <5% decrease in amplitude between testing sessions. Unlike the ECRL HGN, however, there was no reliability in the FCR HGN (ICC = −0.04), though the between-day reliability for FCR HTH improved (ICC = 0.49). Overall, the FCR visHTH was the most-reliable measure for this muscle (ICC = 0.76), with no effect between days 1 and 2.
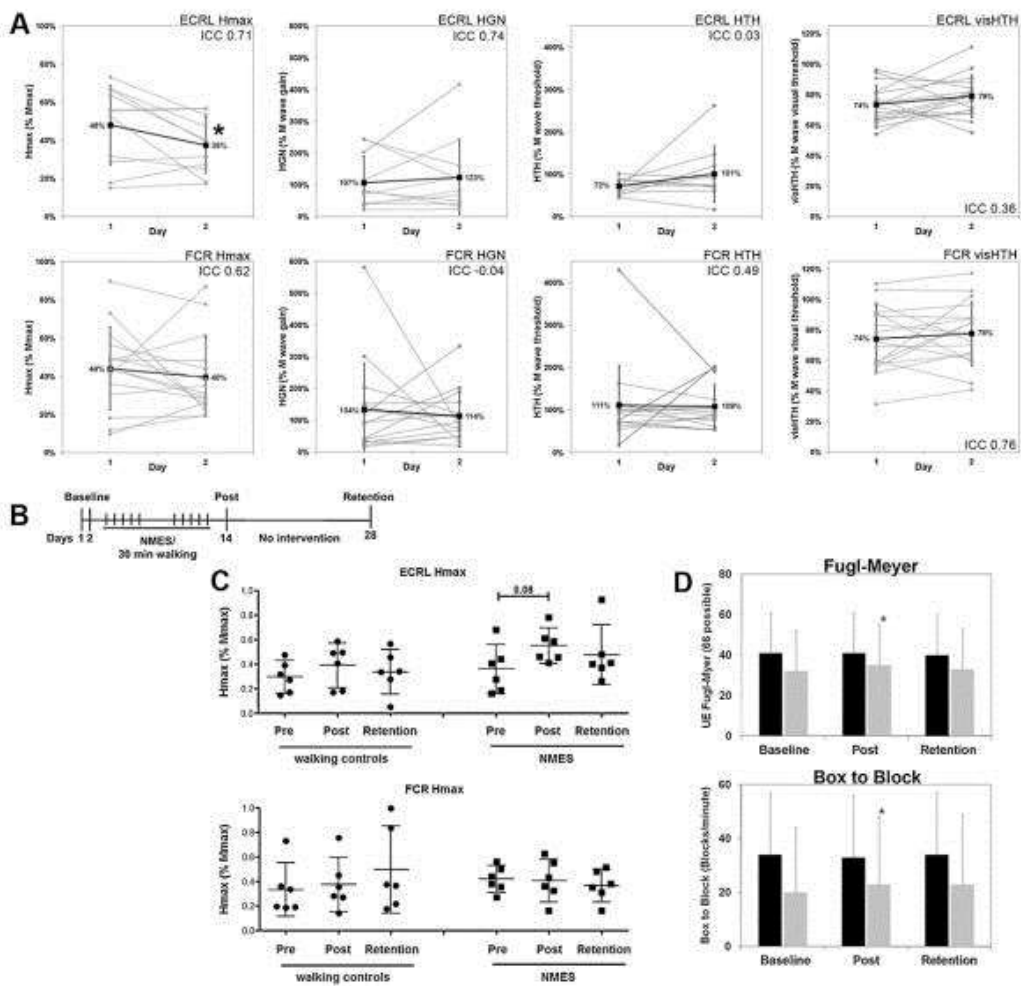
Fig. 2. H-reflex measures in the affected UE. (A) Panels show subject variation between the first two days of baseline testing. Individual subjects (grey lines), group means/SD (black lines), and ICC values are shown. (B) Schematic for the NMES study. (C) For subjects in the final analysis, NMES, but not walking increased ECRL Hmax. (D) NMES improves UE sensorimotor impairment (Fugl-Meyer) and UE speed/coordination (Box and Block). *p < 0.05.

Table 2. Baseline UE H-reflex parameters during chronic stroke.

	n	Day 1 (mean/SD)	Day 2 (mean/SD)	Effect of day on baseline measures	ICC	Typical error	Typical percent error
ECRL							
Hmax	11	48 ± 21%	38 ± 15%	$F_{1, 10} = 2.33$; $p = 0.04$	0.71	10.52%	21.91%
HGN	11	107 ± 84%	123 ± 117%	$F_{1, 10} = 0.57$; $p = 0.58$	0.74	67.22%	62.72%
HTH	10	72 ± 18%	101 ± 66%	$F_{1, 9} = 1.38$; $p = 0.20$	0.03	47.26%	65.53%
visHTH	16	74 ± 13%	79 ± 14%	$F_{1, 15} = 1.35$; $p = 0.20$	0.36	11.07%	15.04%
FCR							
Hmax	15	44 ± 22%	40 ± 21%	$F_{1, 14} = 0.79$; $p = 0.44$	0.62	15.96%	36.17%
HGN	15	134 ± 147%	114 ± 82%	$F_{1, 14} = 0.46$; $p = 0.65$	−0.04	121.47%	90.75%
HTH	14	111 ± 97%	109 ± 52%	$F_{1, 13} = 0.11$; $p = 0.91$	0.49	63.43%	56.93%
visHTH	17	79 ± 30%	80 ± 24%	$F_{1, 16} = 0.28$; $p = 0.78$	0.76	13.39%	18.01%

8.2. H-reflex and UE motor performance measurements following NMES

In an effort to establish an unbiased method to include only participants with reliable measures, we removed 3 participants with a between-day change greater than 2 SD of the group mean from the larger SD of either day 1 or day 2 (subjects 3, 5, 14). Twelve additional participants did not pass our predetermined criteria for physiologically representative recruitment curves (e.g. lack of Mmax, high signal:noise ratio) at either the Post or Retention times for a specific muscle (Fig. 2B). Therefore, only half (i.e. 12 out of 24)

participants, with good reliability and physiologic recruitment curves at every time point, were included in the final cohort.

Table 3 depicts group means for baseline (Pre), Post and Retention time points for all H-reflex parameters. Fig. 2C shows the group analysis for ECRL and FCR Hmax within the 95% confidence interval ($n = 6$ per group). While ECRL Hmax increased by 9% following walking in control participants, NMES increased the post Hmax by 19% ($p = 0.08$), and maintained this elevation by 12% at retention. Changes in ECRL Hmax were the only H-reflex changes to exceed typical error, which was 10.52% of Mmax for this measure. The FCR Control Post Hmax increased 5%, and additionally 12% at retention. Immediate post-NMES, FCR Hmax was unchanged from baseline, but Retention FCR Hmax decreased 5%.

Table 3. H-reflex parameters in the affected UE following NMES.

	Group	n	Baseline (mean/SD)	Post (mean/SD)	Retention (mean/SD)	Repeated measures ANOVA
ECRL						
Hmax	Control	6	30 ± 13%	39 ± 17%	34 ± 17%	$F_{2, 5} = 1.55$; $p = 0.26$
	Intervention	6	36 ± 19%	55 ± 14%	48 ± 23%	$F_{2, 5} = 1.54$; $p = 0.26$
HGN	Control	6	50 ± 39%	134 ± 131%	60 ± 44%	$F_{2, 5} = 2.18$; $p = 0.16$
	Intervention	6	84 ± 56%	91 ± 84%	95 ± 62%	$F_{2, 5} = 0.04$; $p = 0.96$
HTH	Control	5	58 ± 25%	55 ± 41%	47 ± 27%	$F_{2, 4} = 0.43$; $p = 0.66$
	Intervention	5	81 ± 16%	54 ± 19%	67 ± 27%	$F_{2, 4} = 1.45$; $p = 0.29$
visHTH	Control	10	73 ± 16%	67 ± 17%	74 ± 18%	$F_{2, 9} = 1.49$; $p = 0.25$
	Intervention	13	71 ± 14%	63 ± 15%	64 ± 17%	$F_{2, 12} = 1.28$; $p = 0.30$
FCR						
Hmax	Control	6	33 ± 21%	38 ± 21%	50 ± 34%	$F_{2, 5} = 3.00$; $p = 0.10$
	Intervention	6	42 ± 10%	41 ± 17%	37 ± 13%	$F_{2, 5} = 1.49$; $p = 0.27$
HGN	Control	5	108 ± 75%	133 ± 138%	193 ± 92%	$F_{2, 4} = 1.11$; $p = 0.38$

	Group	n	Baseline (mean/SD)	Post (mean/SD)	Retention (mean/SD)	Repeated measures ANOVA
	Intervention	6	99 ± 61%	152 ± 213%	36 ± 37%	$F_{2, 5} = 1.40$; $p = 0.29$
HTH	Control	5	90 ± 15%	70 ± 21%	84 ± 16%	$F_{2, 4} = 1.77$; $p = 0.23$
	Intervention	5	79 ± 13%	74 ± 37%	79 ± 29%	$F_{2, 4} = 0.14$; $p = 0.87$
visHTH	Control	9	73 ± 17%	77 ± 17%	71 ± 16%	$F_{2, 8} = 0.93$; $p = 0.41$
	Intervention	14	76 ± 22%	74 ± 20%	85 ± 18%	$F_{2, 13} = 1.44$; $p = 0.26$

A significant between-group effect in FM scores following NMES ($F_{2, 14} = 7.47$; $p < 0.001$) for the Intervention group showed improvement (baseline vs. Post; $p < 0.01$) that did not remain at retention (Table 4). There was also a trend for improvement in specific FM hand function following NMES ($p = 0.051$; Fig. 2), but not in UE wrist function or coordination and speed. While we have previously shown an effect of NMES on UE sensorimotor impairment when assessed using MASS [17] Post, we did not find a similar change after NMES in this study. NMES did, however, improve UE motor performance and coordination for the BB test. The blocks per minute in the Intervention group increased between baseline and Post ($p < 0.01$) while the Control group showed no change.

Table 4. NMES improves motor performance in the affected UE.

	Baseline (mean/SD)	Post (mean/SD)	Retention (mean/SD)	Repeated measures ANOVA
Control				
SIS hand function	66 ± 38	71 ± 39	66 ± 37	$F_{2, 9} = 2.18$; $p = 0.17$
physical domain	70 ± 26	72 ± 25	75 ± 23	$F_{2, 9} = 0.21$; $p = 0.82$
% Recovery	68 ± 20	66 ± 22	67 ± 22	$F_{2, 9} = 0.06$; $p = 0.94$
Modified Ashworth	3.5 ± 1.1	3.3 ± 3.6	3.4 ± 3.5	$F_{2, 9} = 0.70$; $p = 0.52$

	Baseline (mean/SD)	Post (mean/SD)	Retention (mean/SD)	Repeated measures ANOVA
BB unaffected	51 ± 15	51 ± 17	50 ± 16	$F_{2,9} = 0.06$; $p = 0.94$
Affected	34 ± 23	33 ± 23	34 ± 23	$F_{2,9} = 0.06$; $p = 0.95$
FM Hand function	9.6 ± 5.0	9.6 ± 5.0	8.7 ± 4.9	$F_{2,9} = 0.22$; $p = 0.81$
Coordination	4.1 ± 2.2	3.5 ± 2.4	3.4 ± 2.3	$F_{2,9} = 0.86$; $p = 0.46$
Wrist	6.0 ± 3.4	5.6 ± 3.4	5.9 ± 3.2	$F_{2,9} = 0.45$; $p = 0.65$
Total	41.0 ± 19.9	40.7 ± 20.0	40.0 ± 20.1	$F_{2,9} = 0.02$; $p = 0.98$
Intervention				
SIS hand function	37 ± 38	38 ± 38	38 ± 38	$F_{2,14} = 0.50$; $p = 0.62$
physical domain	59 ± 24	57 ± 24	57 ± 24	$F_{2,14} = 0.04$; $p = 0.96$
% Recovery	55 ± 20	58 ± 18	59 ± 19	$F_{2,14} = 0.47$; $p = 0.64$
Modified Ashworth	5.2 ± 3.9	4.7 ± 3.9	4.9 ± 3.9	$F_{2,14} = 0.89$; $p = 0.43$
BB unaffected	50 ± 14	50 ± 16	52 ± 15	$F_{2,14} = 0.04$; $p = 0.96$
Affected	19 ± 24	20 ± 25	20 ± 26	$F_{2,14} = 9.8$; $p = 0.002$[*]
FM hand function	6.1 ± 5.3	6.4 ± 5.6	6.7 ± 5.7	$F_{2,14} = 0.2.14$; $p = 0.16$
Coordination	3.2 ± 2.1	2.9 ± 2.1	2.7 ± 2.3	$F_{2,14} = 0.89$; $p = 0.43$
Wrist	3.8 ± 3.7	4.6 ± 3.7	4.1 ± 3.7	$F_{2,14} = 1.54$; $p = 0.25$
Total	31.9 ± 20.3	34.1 ± 20.4	32.6 ± 20.4	$F_{2,14} = 0.7.47$; $p = 0.006$[*]

SIS, Stoke impact scale; BB, box and block; FM, Fugl–Meyer.

[*]$p < 0.05$.

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9. Discussion

The purpose of our study was two-fold: to establish a reliable protocol to measure UE H-reflexes after stroke, and to explore our hypothesis that H-reflex modulations contribute to the beneficial effects of NMES therapy. The H-reflex represents the monosynaptic connection between Ia muscle spindle fibers and the homonymous innervating motoneuron; the electrical equivalent of the spinal stretch reflex [3]. Lower extremity soleus H-reflex measurements have been useful in understanding spasticity and poor motor function after neurological injury [21,18]. UE H-reflex is only easily measured within ECRL and FCR, but not other forearm muscles [11], which has limited previous investigation [14,11]. We report that measuring ECRL and FCR H-reflexes in a single session was well tolerated in participants with prior stroke, despite the length of testing session, severity of deficit, or age of participant.

Full recruitment curves characterize multiple facets of a muscle's monosynaptic reflex excitability, including maximum recruited motoneurons (Hmax), stimulus thresholds for excitability (HTH), and the ease of additional motor unit recruitment (HGN) [20]. In contrast to healthy participants, chronic stroke-related hemiparesis reduced the number of statistically reliable H-reflex parameters. In particular, only ECRL Hmax and HGN, and to a lesser extent FCR Hmax and visHTH, were quantified in a consistent manner. The enhanced reliability of the ECRL over the FCR occurred previously [20] and may reflect anatomical influence on nerve stimulation and/or EMG recording. The mean ECRL and FCR Hmax for our participants were 48% and 44%, respectively; twice the response in healthy participants [20]. This expected elevated UE Hmax [21] may be related to stroke induced spasticity of the affected limb [16].

The ECRL H-max magnitude declined significantly during baseline testing. It is well known that H-reflex amplitude is influenced by factors such as body orientation, limb position, activity in test muscles and muscles remote to test muscles, and anxiety. While we were successful in controlling the position of our subjects and we made an effort to control background activity in the test muscle, we did not monitor muscle activity or anxiety. Increased familiarity with the procedures on day 2 could have reduced subjects' anxiety, resulting in

smaller values for ECRL Hmax. Moreover, the *p*-value of 0.04, with no adjustment for experiment-wise error, suggests that the day 1 to day 2 drop in ECRL Hmax could be caused by type 1 error. If this effect is observed in subsequent studies, future protocols should monitor background muscle activity and ensure adequate familiarization with procedures to minimize uncontrolled influences on H-reflexes.

Establishing between-day reliability for four participants was technically challenging in terms of obtaining a stable EMG signal and generation of true Mmax. While the latency was similar to healthy participants [20,14,9] and did not differ from results in stroke [12], consistently eliciting adequate EMG signals related to the reflex was difficult. This loss of response was evenly distributed between ECRL and FCR and did not correlate with functional deficit (data not shown). Offline analysis revealed several additional participants without true Mmax despite what appeared to be maximum peak-to-peak amplitude in the M wave during data collection. For future studies, designing a data collection program that generates real time recruitment curves during testing sessions will guarantee true Mmax.

A 2008 meta-analysis of clinical trials for NMES intervention following stroke failed to show efficacy [13], though more recent publications show improved UE functional outcome in both acute [8] and chronic stroke survivors [19,17], including NMES delivered only to the ECRL [6]. A larger clinical trial using this protocol may determine if NMES promotes plasticity in the monosynaptic reflex in a use-dependent manner, concomitant with enhanced motor recovery after stroke, particularly considering the trend for increased ECRL Hmax after NMES (*p* = 0.08). Moreover, NMES-induced changes in ECRL Hmax exceeded typical error, which suggests that these changes are clinically meaningful though a larger cohort of participants is needed to make sure that the effect is real and to adequately power the ANOVA. Power analysis of the ECRL Hmax change suggests that a minimum of 18 participants would adequately assess modulation of this variable by NMES.

Both H-reflex testing of agonist–antagonist UE muscles, and inhome NMES therapy, are well tolerated in participants with prior stroke and offer new possibilities for research into reflex-driven

plasticity during UE pathology and treatment of UE functional disability.

Highlights

- First protocol to measure agonist–antagonist H-reflexes in the hemiparetic arm.
- In-home NMES therapy is well tolerated after stroke and improves UE function.
- NMES strengthened the maximum H-reflex amplitude in the ECRL of the affected arm.
- Agonist-antagonist UE H-reflex testing offers new possibilities for research into modulation of reflexes after stroke.

Acknowledgments

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