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Modification of Spastic Stretch Reflexes at the Elbow by Flexion Synergy Expression in Individuals With Chronic Hemiparetic Stroke

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Abstract

Objective: To systematically characterize the effect of flexion synergy expression on the manifestation of elbow flexor stretch reflexes poststroke, and to relate these findings to elbow flexor stretch reflexes in individuals without neurologic injury.

Design: Controlled cohort study.

Setting: Academic medical center.

Participants: Participants (N=20) included individuals with chronic hemiparetic stroke (n=10) and a convenience sample of individuals without neurologic or musculoskeletal injury (n=10).

Interventions: Participants with stroke were interfaced with a robotic device that precisely manipulated flexion synergy expression (by regulating shoulder abduction loading) while delivering controlled elbow extension perturbations over a wide range of velocities. This device was also used to elicit elbow flexor stretch reflexes during volitional elbow flexor activation, both in the cohort of individuals with stroke and in a control cohort. In both cases, the amplitude of volitional elbow flexor preactivation was matched to that generated involuntarily during flexion synergy expression.

Main Outcome Measures: The amplitude of short- and long-latency stretch reflexes in the biceps brachii, assessed by electromyography, and expressed as a function of background muscle activation and stretch velocity.

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Supplier

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Results: Increased shoulder abduction loading potentiated elbow flexor stretch reflexes via flexion synergy expression in the paretic arm. Compared with stretch reflexes in individuals without neurologic injury, paretic reflexes were larger at rest but were approximately equal to control muscles at matched levels of preactivation.

Conclusions: Because flexion synergy expression modifies stretch reflexes in involved muscles, interventions that reduce flexion synergy expression may confer the added benefit of reducing spasticity during functional use of the arm.

Keywords

Motor disorders; Muscle spasticity; Neurological rehabilitation; Rehabilitation; Robotics; Stroke

After stroke, the contralesional arm often exhibits both spasticity and the constraint to grouped movement patterns, which are known clinically as abnormal muscle synergies. Spasticity is defined as a velocity-dependent increase in resistance to imposed movement, and is representative of a heightened stretch reflex.¹ In abnormal muscle synergies, volitional activation of one muscle group drives involuntary activation of specific muscles acting about other joints.^{2,3} This results in the characteristic poststroke inability to produce fine, fractionated movements. In the flexion synergy, progressive increases in shoulder abductor activation lead to proportional increases in elbow flexion (EF) and wrist and finger flexion.⁴⁻⁶ Given the consistent appearance of spasticity in the paretic elbow flexors and the manifestation of the flexion synergy in the same muscles, elbow flexor stretch reflexes that are already overactive poststroke may be exacerbated further during tasks compromised by flexion synergy expression (eg, combined shoulder abduction [SABD] and elbow extension [EE]).

The interplay between flexion synergy expression and stretch reflexes has only been preliminarily investigated,⁷ and is an important yet unexplored aspect of poststroke motor control given its basic scientific and clinical implications. For example, even though mild flexion synergy expression is correlated with substantial functional impairment,⁸ the functional implications of elbow flexor spasticity remain contentious.⁹⁻¹¹ In part, the relevance of spasticity to the overall poststroke movement disorder is questioned by the equivocal findings of previous investigations studying the influence of background muscle activation on stretch reflex exaggeration in spastic and nonspastic muscles.¹²⁻¹⁹ However, this ambiguity is further perpetuated because previous studies of spasticity were conducted in the absence of flexion synergy expression, and previous studies of flexion synergy expression were likewise designed to avoid the influence of spasticity.

The primary aim of this study was to characterize the effect of increasing flexion synergy expression on evoked stretch reflexes in the paretic biceps (BIC) brachii. We then investigated whether stretch reflexes evoked during flexion synergy expression differ in magnitude from stretch reflexes evoked while maintaining matched levels of volitional muscle preactivation. Finally, we compared the magnitude of synergy and nonsynergy enhanced reflexes poststroke with those evoked in individuals without neurologic or musculoskeletal injury. We hypothesized that stretch reflexes elicited during flexion synergy expression would be larger than those elicited during volitional preactivation, ostensibly

because of increases in synergy-associated descending neuromodulation. By systematically characterizing reflex responses over a wide range of stretch velocities and background muscle activation levels, we sought to decouple any potentially unique flexion synergy-driven effects from those attributable to direct, volitional muscle preactivation.

Methods

Participants

We conducted a cross-sectional study with enrollment-matched cohorts of individuals with first-ever chronic hemiparetic stroke and individuals without neurologic or musculoskeletal injury. In total, 20 individuals participated. Ten of these participants (mean $\pm$ SD, 65 $\pm$ 10y) sustained cortical or subcortical stroke 1 year prior to enrollment. Across these participants, upper limb motor impairment ranged from moderate to severe as assessed by the Fugl-Meyer Assessment of motor impairment (16–41/66; n=10), Modified Ashworth Scale (score 0: n=1; score +1: n=2; score 2: n=2; score 3: n=3; 1 score unknown), and strength (mean maximum EF torque $\pm$ SD, 31.2 $\pm$ 13.7Nm). The remaining 10 participants formed a healthy control cohort acquired from a convenience sample (mean age $\pm$ SD, 45 $\pm$ 21y; maximum EF torque, 48.1 $\pm$ 18.8Nm). All participants possessed 90° each of passive EF/EE, SABD, and shoulder flexion/extension, and at least some volitional control of EF/EE and SABD. Current use of antispastic medications was grounds for exclusion. All individuals were screened by a licensed physical therapist to determine eligibility.

All participants gave informed, written consent, and the investigation was approved by the Institutional Review Board of Northwestern University in accordance with the standards established by the 1964 Declaration of Helsinki.

Protocol

The experimental protocol evolved as follows: (1) collection of isometric maximum voluntary torque (MVT) in all participants; (2) assessment of stretch reflexes during flexion synergy expression in individuals with stroke; and (3) assessment of stretch reflexes during volitional elbow flexor preactivation, both in individuals with stroke and in control participants.

For MVT, the forearm, wrist, and hand (paretic arm for stroke; dominant arm for control) were casted and positioned in an isometric testing rig as described previously.²⁰ The tested arm maintained 85° of SABD, 45° of shoulder flexion/horizontal adduction, and 105° of EF (180° being full extension); the forearm was fully pronated. The arm was secured at the wrist to a 6 df load cell, and MVT was collected in EF, EE, and SABD. An active, differential electrode^a was placed on the long head of the BIC brachii to record electromyographic activity during MVT production. MVT was generated in each direction until at least 3 trials were acquired that had peak magnitudes within 10% of one another; the last trial for a given direction could not have the greatest magnitude.

^a.Delsys; Delsys Bagnoli EMG system.

After MVT, participants transitioned to the Arm-Coordination and Training 4-D robotic device robotic device^{7,21–23} for stretch reflex experiments (fig 1A). The Arm-Coordination and Training 4-D robotic device allows free motion of the arm in the vertical direction, can impose vertical forces on its user (eg, manipulating the amount of SABD torque required to lift the arm), and has a dedicated EF/EE perturbation apparatus. For all experiments, SABD, shoulder flexion/horizontal adduction, and forearm pronation angles were matched between the MVT and Arm-Coordination and Training 4-D robotic device setups. Once a participant was positioned in the device, the Arm-Coordination and Training 4-D robotic device delivered a series of 30 passive EE stretches to stabilize stretch reflex excitability prior to reflex assessments (6°/s; approximately 60°–150° extension; 1min between stretches).²⁴

The combined flexion synergy and stretch reflex protocol was performed only in the paretic arm of our stroke cohort because we have previously demonstrated that SABD loading does not result in spontaneous elbow flexor activation in the nonparetic arm or in individuals without stroke.^{6,21} Participants lifted and maintained the paretic limb 5cm above a simulated hard, frictionless table surface created by the Arm-Coordination and Training 4-D robotic device, resulting in an SABD angle of 85°. Simultaneously, the Arm-Coordination and Training 4-D robotic device applied a constant force in the downward direction requiring production of 0%, 12.5%, 25%, 37.5%, or 50% of SABD MVT (eliciting progressively more vigorous flexion synergy expression). In all cases, the robotic device compensated for limb weight; for example, in the 0% SABD MVT condition, the arm felt weightless. For some trials, participants were not required to lift the arm; instead, it rested on the virtual table (table condition).

While generating SABD torque, EE position perturbations were applied to the limb from approximately 60° to 150° (fig 1B). The Arm-Coordination and Training 4-D robotic device accelerated the limb at 4500°/s² to peak velocities of 6, 30, 60, 90, 180, or 270°/s. All combinations of velocity and SABD loading were tested (in a random order), with at least 3 trials per combination. Participants were instructed to maintain the required amount of SABD prior to and during the perturbation (with the aid of visual feedback) and not to volitionally flex or extend the elbow during the perturbation.

All participants performed portions of the protocol investigating volitional preactivation and stretch reflexes. Here, the virtual table supported the full weight of the limb at all times. However, participants were required to voluntarily preactivate the BIC by flexing the elbow against the locked Arm-Coordination and Training 4-D robotic device swingarm. Visual feedback was provided for contraction level. While maintaining 5%, 10% to 15%, or 20% of maximum BIC activation (matching the average synergy-evoked BIC activation from the stroke cohort), we applied EE perturbations as previously described. Perturbations were also collected during a relaxed condition (confirmed by electromyography). The sequence of an individual trial was the same as that depicted in Figure 1B, except that the prestretch baseline phase involved volitional EF.

Data analysis

In order, raw BIC electromyographic data were filtered (20–400Hz bandpass; 55–65Hz notch), rectified, smoothed (5ms zero-phase moving average), and normalized to the peak

rectified BIC electromyography obtained during EF MVT.^{25,26} Individual BIC electromyographic time records at each speed and preactivation level were then ensemble averaged per participant. Next, pre-activation was removed by subtracting from the overall electromyographic time record the average electromyography during the 500 milliseconds immediately prior to the perturbation. Electromyographic data were then divided into short-latency reflexes (SLRs; 20–50ms postperturbation onset) and long-latency reflexes (LLRs; 50–150ms), with a single, averaged record at each preactivation/velocity combination per participant. The mean amplitude of SLR and LLR components, respectively, was extracted per participant, and averaged across participants in each cohort.

Linear regression was used to determine the effect of SABD load on spontaneously generated BIC electromyography in the stroke cohort. Three-way repeated-measures analyses of variance were used to assess the effect of preactivation magnitude, perturbation velocity, and preactivation type (ie, SABD loading vs volitional preactivation) on SLRs and LLRs, respectively, within the stroke cohort. Repeated-measures analyses of covariance were used to assess the effect of preactivation magnitude and perturbation velocity on SLRs and LLRs, respectively, between our stroke and control cohorts. Specific parameters for each model can be seen in table 1. For all models, normality was assessed with the Kolmogorov-Smirnov test, sphericity was assessed with the Mauchly test, and α was .05.

Results

Increased SABD loading potentiates stretch reflex magnitude

Figure 2 depicts the average, involuntary BIC electromyography generated in the paretic arm of the stroke cohort under progressively increasing SABD loads. Consistent with the definition of the flexion synergy, BIC electromyography increased in direct proportion to SABD load ($R^2=.98$, $P<.001$). Figure 3 depicts a flexion synergy/stretch reflex experiment in the paretic limb of a representative participant (expanded analyses and experiments are subsequently detailed). Qualitatively, either increasing SABD load at a given perturbation velocity or increasing perturbation velocity at a given SABD load led to an increased reflex magnitude. By extension, as SABD loading increases, the angular velocity at which a stretch reflex can be evoked is reduced. This is the fundamental relation between flexion synergy expression and stretch reflexes. These trends were reflected across the stroke cohort (fig 4). Here, SLR and LLR are presented separately, and averaged across participants. We found significant effects of both SABD loading and velocity for LLR (each $P<.001$) (see table 1), but only a significant effect of velocity for SLR ($P<.001$) (see table 1).

Preactivation normalizes paretic stretch reflexes

We first confirmed that stretch reflexes evoked in the relaxed, paretic BIC exceeded those in individuals without neurologic injury—the defining feature of spasticity (fig 5A). Next, we assessed the effect of volitional preactivation on stretch reflex amplitude in individuals with stroke and those without neurologic injury. In figure 5B, stroke and control cohorts volitionally generated 20% maximum BIC preactivation prior to a 270°/s EE perturbation. This level corresponded to the amount of BIC electromyography that arose spontaneously during 50% MVT SABD loading poststroke. Under these conditions, we observed

qualitatively similar reflex magnitudes between groups. In figure 5C, we present summary data taken only from the stroke cohort at 270°/s, highlighting the similarity between reflexes elicited during volitional and flexion synergy-driven BIC activation ($P=.654$ and $P=.514$ for SLR and LLR, respectively) (see table 1).

Finally, figure 6 shows group-average reflex responses across cohorts. Although reflex magnitude increases with either increasing preactivation or perturbation angular velocity across both groups ($P<.001$ and $P<.001$, respectively) (see table 1), it is again apparent that when relaxed, individuals with stroke generate larger reflex responses; once preactivated, however, reflex responses are comparable in stroke and control populations ($P=.171$) (see table 1).

Discussion

Neurophysiological links between spasticity and flexion synergy expression

Spasticity and flexion synergy expression could both arise in part from an increased influence of the reticulospinal system, which has a motor and a neuromodulatory component.²⁷ The motor component is well suited to drive flexion synergy expression because its diffuse spinal projections simultaneously activate flexor and abductor muscles of the ipsilateral upper limb (ie, synergy-involved muscles).^{27–30} The neuromodulatory component is well suited to drive spasticity because the postsynaptic effects of its neurotransmitters, serotonin, and norepinephrine can increase motor unit recruitment and amplify motoneuron output in response to 1a afferent feedback.^{31–33}

We found significantly exaggerated stretch reflexes evoked from rest in our stroke compared with our control cohort, yet negligible differences between reflexes elicited during synergy expression and those elicited at matched levels of volitional preactivation (whether poststroke or in our control cohort). The finding of exaggerated stretch-sensitive reflexes at rest is common poststroke, and is consistent with a tonic neuromodulatory drive to motoneuron pools innervating the paretic musculature.³⁴ The finding that synergy-driven and volitionally driven preactivation led to equivalent reflex amplitudes poststroke has not previously been reported, and may appear paradoxical in the context of the reticulospinal hypothesis. Indeed, we expected reflexes elicited during synergy expression to exceed those elicited at matched levels of volitional preactivation. This is because neuromodulatory drive typically scales with motor output, suggesting that progressive flexion synergy expression would beget continued increases in serotonergic and noradrenergic signaling. It is important to reiterate, however, that although the motor and neuromodulatory components tend to be coactivated, they do not necessarily increase or decrease in a one-to-one manner. Therefore, it is possible that higher levels of background elbow flexor activation could have caused differences in reflex amplitude to re-emerge. However, 2 intriguing alternative explanations could also underlie our findings: (1) reticulospinal neuromodulation may be enhanced yet relatively inflexible post-stroke and/or (2) synaptic inhibition may increase in direct proportion to motor or neuromodulatory drive,^{35,36} giving the appearance of a relatively static neuromodulatory output. Further investigation of both hypotheses is required, however.

Functional and clinical implications

Spasticity assessments typically rely on externally imposed perturbations to evoke stretch reflexes in relaxed muscles. It has classically been assumed that the exaggerated responses observed in this setting contribute in some way to the overall poststroke reaching deficit. However, during functional use of the paretic arm, flexion synergy expression will drive involuntary coactivation of the elbow flexors. In tasks that primarily require EE (eg, reaching, grasping), this coactivation of antagonistic muscles may have competing effects on whether spasticity is likely to interfere with movement.

In one scenario, synergy-driven elbow flexor activation would increase the likelihood of self-evoking a stretch reflex by bringing more motoneurons close to firing threshold. Indeed, this is consistent with our finding of increased stretch-evoked electromyography at a given perturbation velocity as background muscle activation increases (eg, moving from the top row to the bottom row within the column corresponding to a 90 deg/sec perturbation in fig 3). However, involuntary elbow flexor activation during attempted EE will also slow overall extension velocity,³⁷ reducing the likelihood that a stretch reflex will be self-evoked. Preferred and maximum EE velocities during volitional reaching under conditions of negligible flexion synergy expression (ie, the fastest possible case) have recently been reported as approximately 33 to 46°/s and approximately 58 to 76°/s, respectively, in a cohort similar to ours.¹¹ Because these velocities decrease with increasing flexion synergy expression, they are likely to overestimate the volitional extension velocities achieved during functional limb use. Nevertheless, using these speeds in the context of our imposed EE perturbations, we find average SLR and LLR responses of only approximately 5% maximum when flexion synergy expression is minimal, and responses that remain <10% maximum even during the most vigorous flexion synergy expression (50% SABD MVT). Therefore, even if a reflex were to be self-evoked during typical volitional reaching, we would expect its net effect to be low.

An inability to adequately modulate reflex excitability has also been suggested as a means by which spasticity can cause functional impairment.^{15,38–40} In the lambda model this refers to the tonic stretch reflex threshold, which is reached at a more acute joint angle in individuals with spasticity.⁴¹ The lambda model has not been investigated in the context of flexion synergy expression, nor was our study intended to do so. However, as previously mentioned, free reaching with the paretic arm increases spontaneous elbow flexor electromyographic activity while reducing EE velocity³⁷ and range of motion. A change in any one of these parameters (or a combination thereof) could alter the apparent location of the tonic stretch reflex threshold, rendering it challenging to decouple its potential functional effect during reaching. Changes in neuromodulatory and inhibitory drive poststroke may result in functional impairment by compressing rate modulation and altering intrinsic motoneuron properties,^{35,36} whereas changes in spinal interneuron excitability may exacerbate abnormal muscle coactivation patterns and/or hyperactive stretch reflexes by shifting reciprocal inhibition toward net facilitation.^{42,43}

Study limitations

An important limitation of this study is that we did not attempt to elicit stretch reflexes during volitional EE. However, our goal was to quantify the effect of flexion synergy expression on stretch reflexes in the context of individuals without neurologic injury. In this case, it is not feasible to maintain substantial agonist-antagonist cocontraction during reaching; therefore, we would have been unable to provide an appropriate control for our synergy-driven reflex responses. A second limitation of our study is that all perturbations were delivered from the same starting angle for a given participant. This arrangement prevented us from quantifying spatial reflex thresholds, which have been used as an alternative means of quantifying spasticity (eg, lambda model).^{16,44,45} However, because our reflex assessments required demonstrable background muscle activation (except for trials specifically in the relaxed BIC), reflexes were initiated nearly immediately regardless of perturbation velocity. Therefore, the potential dependence of reflex response on joint angle would have been obscured, resulting in a poor characterization of spatial threshold effects.

Conclusions

Our results demonstrate that the magnitude of stretch reflexes elicited during flexion synergy expression is no greater than that of stretch reflexes elicited at matched levels of volitional pre-activation, whether poststroke or in individuals without neurologic impairment. Although more work is warranted to understand the role(s) of flexion synergy expression and spasticity in functional arm use, our results reinforce the emerging notion that spasticity itself may have little to do with functional motor deficits.^{9,46} Based on our findings, we suggest that interventions seeking to improve the overall reaching deficit poststroke consider prioritizing flexion synergy expression over spasticity. In addition to the direct benefits of flexion synergy reduction, this could indirectly mitigate potential effects of spasticity by reducing involuntary elbow flexor activity.

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List of abbreviations:

BIC	biceps
EE	elbow extension
EF	elbow flexion
LLR	long-latency reflex
MVT	maximum voluntary torque
SABD	shoulder abduction
SLR	short-latency reflex

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Fig 1.

Experimental setup, protocol. (A) Schematic representation of the Arm-Coordination and Training 4-D robotic device. (B) Participants either lifted the limb against an applied force (causing flexion synergy expression) or volitionally flexed the elbow while EE perturbations were delivered. Shown: stretch reflex elicited in response to a 270°/s perturbation. Abbreviations: And. vel., angular velocity; EMG, electromyography.

Fig 2.

Flexion synergy expression at the elbow. Because SABD loading in the paretic arm increases poststroke, the BIC is involuntarily activated. Data are collected from the 2-second lift and hold period that preceded perturbation, as shown in figure 1B. Data are normalized to the BIC EMG generated during each participant's isometric maximum voluntary EF contraction. Abbreviations: EMG, electromyography; max, maximum; s.e.m., standard error of the mean.

Fig 3. Single participant flexion synergy/stretch reflex results. As SABD load level or perturbation velocity increases, BIC reflex amplitude increases. Each subplot represents the baseline corrected BIC EMG at the combination of SABD load level and perturbation velocity indicated by the axes grid. SABD load level increases down the rows and is indicated by the right-side y-axis label; perturbation velocity increases left to right across the columns and is indicated by the top x-axis label. Abbreviations: EMG, electromyography; max, maximum. Adapted with permission from Institute of Electrical and Electronics Engineers.⁷

Fig 4.

Group average flexion synergy/stretch reflex results. Across the stroke cohort ($n=10$), BIC EMG increases as SABD load or perturbation angular velocity increases. Each colored rectangle within a panel represents the group average evoked BIC EMG at the given SABD load and perturbation velocity combination. The color data from both panels is interpreted by the scale bar at right, with cooler colors representing lower relative EMG activity and warmer colors representing higher EMG activity. The left panel represents SLR responses; the right panel, LLR responses. SABD load level increases down the rows and is indicated for both panels by the left-side y -axis label; perturbation velocity increases left to right across the columns and is indicated for both panels by the bottom left x -axis label. Color intensity: normalized BIC EMG magnitude (% max); each block represents the across-participant mean BIC EMG amplitude at a given SABD load and perturbation velocity. Abbreviations: EMG, electromyography; max, maximum.

Fig 5. Preactivation normalizes stretch-evoked BIC EMG between stroke and control participants. (A) Under relaxed conditions, stretch reflexes are exaggerated poststroke compared with controls; (B) once preactivated (approximately 20% max BIC EMG), reflex magnitude is comparable. Color code and gray identifiers indicate matched participants. EMG records: average response per participant to 270°/s perturbation. (C) Mean LLR responses to a 270°/s perturbation are shown for the stroke cohort during flexion synergy expression and during volitional background muscle activation; results are displayed as a function of background BIC activation level. Abbreviations: EMG, electromyography; max, maximum; s.e.m., standard error of the mean.

Fig 6.

Group-average volitional preactivation/stretch reflex results. Across the stroke cohort (top panel) and the control cohort (bottom panel), stretch-evoked BIC EMG increases as volitional preactivation (subplot rows) or perturbation angular velocity (subplot columns) increases. Although reflex amplitude remains exaggerated in the relaxed BIC poststroke, reflexes become indistinguishable across groups once preactivated. Color intensity: normalized BIC EMG magnitude; each block represents the across-participant mean BIC EMG amplitude for a given preactivation. Abbreviations: EMG, electromyography; max, maximum.

Table 1

Statistical analyses and results

Comparison	Test	Main Effect	Main Effect	Main Effect	Interaction	Interaction	Interaction	Interaction
SLR in stroke cohort. Within-subject factors: preactivation magnitude; velocity; between-subject factors: preactivation type (SABD vs volitional BIC activation); dependent: SLR magnitude.	3-way rm- ANOVA	Preactivation magnitude; $P<.001$	Velocity; $P<.001$	Preactivation type; $P=.65$	Preactivation magnitude by Preactivation type (SABD vs volitional); $P=.001$	Velocity by Preactivation type (SABD vs volitional); $P=.27$	Preactivation magnitude by velocity; $P=.07$	Preactivation magnitude by Velocity by Preactivation type; $P=.001$
LLR in stroke cohort. Within-subject factors: preactivation magnitude; velocity; between-subject factors: preactivation type (SABD vs volitional BIC activation); dependent: LLR magnitude.	3-way rm- ANOVA	Preactivation magnitude; $P<.001$	Velocity; $P<.001$	Preactivation type; $P=.51$	Preactivation magnitude by Preactivation type (SABD vs volitional); $P=.39$	Velocity by Preactivation type (SABD vs volitional); $P=.76$	Preactivation magnitude by velocity; $P=.15$	Preactivation magnitude by Velocity by Preactivation type; $P=.51$
SLR between stroke and control cohorts. Within-subject factors: velocity; between-subject factors: cohort; covariate: preactivation; dependent: SLR magnitude.	rm- ANCOVA	Stroke vs control on reflex magnitude; $P=.76$	Preactivation magnitude (between-cohort covariate); $P<.001$	Velocity; $P=.01$	Velocity by Preactivation magnitude; $P<.001$	Velocity by cohort (stroke vs control); $P=.24$	NA	NA
LLR between stroke and control cohorts. Within-subject factors: velocity; between-subject factors: cohort; covariate: preactivation; dependent: LLR magnitude.	rm- ANCOVA	Stroke vs control on reflex magnitude; $P<.001$. Note: control is larger	Preactivation magnitude (between-cohort covariate); $P<.001$	Velocity; $P=.01$	Velocity by Preactivation magnitude; $P<.001$	Velocity by cohort (stroke vs control); $P=.001$	NA	NA

Abbreviations: ANCOVA, analysis of covariance; ANOVA, analysis of variance; rm, repeated-measures.