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Flexion Synergy Overshadows Flexor Spasticity During Reaching in Chronic Moderate to Severe Hemiparetic Stroke

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Abstract

Objective—Pharmaceutical intervention targets arm flexor spasticity with an often-unsuccessful goal of improving function. Flexion synergy is a related motor impairment that may be inadvertently neglected. Here, flexor spasticity and flexion synergy are disentangled to determine their contributions to reaching dysfunction.

Methods—Twenty-six individuals participated. A robotic device systematically modulated shoulder abduction loading during ballistic reaching. Elbow muscle electromyography data were partitioned into windows delineated by elbow joint velocity allowing for the separation of synergy- and spasticity-related activation.

Results—Reaching velocity decreased with abduction loading ($p < 0.001$) such that velocity was 30% slower when lifting the arm at 50% of abduction strength compared to when arm weight was supported. Abnormal flexion synergy increased with abduction loading ($p < 0.001$) such that normalized activation ranged from a median (interquartile range) of 0.07 (0.03–0.12) when arm weight was supported to 0.19 (0.12–0.40) when actively lifting (large effect size, $d = 0.59$). Flexor spasticity was detected during reaching ($p = 0.016$) but only when arm weight was supported (intermediate effect size, $d = 0.33$).

Conclusion—Flexion synergy is the predominant contributor to reaching dysfunction while flexor spasticity appears only relevant during unnaturally occurring passively supported movement.

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Conflict of Interest Statement

None of the authors have financial or other relationships that might lead to a perceived conflict of interest.

Significance—Interventions targeting flexion synergy should be leveraged in future stroke recovery trials.

Keywords

Stroke; Arm; Biomechanical Phenomena; Movement; Muscles; Robotics; Rehabilitation; Neurology

1. INTRODUCTION

Clinicians will be required to quantitatively measure and directly target the contributing underlying motor impairments in individuals with hemiparetic stroke to realize advances beyond conventional care in restoring upper extremity function (Krakauer et al., 2012). In the context of reaching function, impairment in joint individuation is the best predictor of recovery outcome over other common impairments observed in chronic stroke such as weakness and spasticity (Zackowski et al., 2004). The term “spasticity” is defined traditionally as a velocity-dependent hyperactive stretch reflex (Lance, 1980, Thilmann et al., 1991) measured under passive conditions. While this definition has been argued as inadequate (Malhotra et al., 2009) or at least inconsistent with the conventional clinical use that includes increased resting muscle tone and abnormal posturing, (Burke et al., 2013) it is adopted here to differentiate two distinct but concomitant muscle activation impairments in order to elucidate their contributions to reaching dysfunction. Specifically, flexor spasticity, or stretch reflex-related flexor activation, coincides with a more immobilizing muscle activation impairment. Abnormal co-activation of shoulder abductors with distal limb flexors (Dewald et al., 1995) results in a loss of independent joint control (Dewald et al., 2001b) and has been previously described as flexion synergy (Twitchell TE, 1951, Brunnstrom, 1970). Disentangling flexor spasticity and flexion synergy will serve to direct medical and rehabilitation management focused on improving arm function.

Despite the contemporary view that spasticity as defined by Lance (Lance, 1980) does not contribute to abnormal posturing, synkinetic movements, or even disability in general, (Burke et al., 2013) there seems to be a persistent antiquated view that pharmaceutical treatment of spasticity will improve movement function, specifically reaching. This is evident in investigations of Tizanidine Hydrochloride (Gelber et al., 2001) and Botulinum Toxin (Bensmail et al., 2010) where it was hypothesized that both spasticity and arm function would improve. Both investigations failed to demonstrate improvements in arm function as measured by the Action Research Arm Test (ARAT) (Lyle, 1981) despite reductions in spasticity as measured by the Modified Ashworth Scale (Bohannon et al., 1987). The lack of improvement in ARAT suggests that another motor impairment is at play. When reaching against gravity, range of motion is known to be limited by the abnormal coupling of shoulder abduction with elbow flexion (Beer et al., 2004). Perhaps the ineffectiveness in improving reaching function when treating spasticity is because flexion synergy and subsequent loss of independent joint control is the predominant impairment.

Testing this proposition requires a quantitative evaluation of muscle activation during a controlled movement task. Prior investigations have measured impairments of weakness,

spasticity, and joint individuation independently and evaluated relationships between them. Concurrent quantification of each phenomena in a single controlled movement task, as performed in this study, allows for causative (effect of abduction loading) as opposed to relational hypotheses to be tested identifying key impairments of reaching dysfunction. The device, ACT^{3D}, (Sukal et al., 2005) is capable of systematically manipulating the amount of abduction loading required during outward reaching (Sukal et al., 2007, Ellis et al., 2008, Ellis et al., 2016). Muscle electromyography can be concurrently acquired and partitioned into time epochs prior to and just after the onset of elbow joint extension allowing for quantification of the contributions of synergy-related vs. spasticity-related flexor activation to reaching performance. Here, evidence is provided for the overwhelming contribution of synergy-related elbow flexor muscle activation that; 1) occurs after actively abducting the shoulder but prior to the onset of elbow extension, and 2) persists throughout movement limiting reaching speed and range. This compelling evidence supports the proposition that abnormal flexion synergy is the predominant impairment of reaching function eclipsing flexor spasticity.

2. METHODS

2.1 Design

The study implemented a prospective, single-site, cross-sectional, quantitative, experimental design that was conducted in a university laboratory setting to test the effect of abduction loading on reaching velocity, flexion synergy, and flexion spasticity in individuals with chronic stroke-related hemiparesis and flexor spasticity.

2.2 Participants

Twenty-six individuals with chronic hemiparetic stroke participated in this study. All participants provided signed consent for the study that was approved by the Institutional Review Board of Northwestern University in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki for research involving human participants. The sample consisted of 4 (15%) women and 22 (85%) men with either right (N=14 or 54%) or left (N=12 or 46%) hemiparesis and an average (standard deviation) age of 56.30 ± 9.30 years. Hemiparesis was evaluated using the arm motor portion of the Fugl-Meyer Motor Assessment (Fugl-Meyer et al., 1975) with an average score of 27.19 ± 6.00 out of 66 representing moderate motor impairment. Spasticity was evaluated using the Modified Ashworth Scale (Bohannon et al., 1987) with an average score of 0.78 ± 1.03 (elbow extensors) and 2.19 ± 0.63 (elbow flexors) out of 5. The scoring of 1, 1+, 2, 3, and 4 were converted to 1–5 to allow for analysis. Elbow joint isometric strength was quantitatively measured using laboratory methods described previously (Ellis et al., 2007) for both extension (26.07 ± 10.77 Nm) and flexion (21.62 ± 8.26 Nm). Pain-free passive range of motion of at least 90° of shoulder flexion and abduction, and neutral internal/external rotation was required to safely interface with the robotic device and participate in the study.

2.3 Experimental Setup

The ACT^{3D} robotic device was employed to quantify reaching function under various abduction loading conditions (Figure 1A,B) (Sukal et al., 2007, Ellis et al., 2008, Ellis et al.,

2016). The participant interface of the ACT^{3D} allows motion of the arm in the horizontal plane and is capable of imposing vertical forces upon its user, thus controlling the abduction loading experienced by the participant during an outward reach. All position data from the ACT^{3D} were recorded at 50 Hz. EMG data were collected using active differential surface electrodes (Delsys, 16-channel Bagnoli EMG System, Boston, MA) with 1-cm inter-electrode distance from brachioradialis, biceps brachii, lateral head of triceps brachii, and long head of triceps brachii with a sampling frequency of 1000Hz.

2.4 Experimental Protocol

Participants were asked to make ballistic arm movements from an initial home position close by their body to an outward target in order to quantify reaching velocity (Ellis et al., 2016). The home position and reaching target were standardized to shoulder and elbow joint angles. The home position was defined by 90° shoulder abduction, 110° of elbow flexion, and 40° of shoulder flexion (horizontal adduction) (Figure 1A). The reaching target required an additional 90° of elbow extension and 40° of shoulder flexion to acquire (10° short of full elbow extension). Upon holding the arm in the home position for 250ms, the reaching target was displayed. Participants were instructed to remain in the home position for approximately one second prior to beginning the reach. The reach was instructed to be of maximal effort moving directly toward and acquiring the target.

Reaching movements were first measured with the arm supported on a horizontal frictionless haptic table emulated by the ACT^{3D} and centered at the participant's shoulder height such that the participant was not required to lift/abduct the arm during the reach. This condition was utilized to determine participant-specific maximum reaching elbow angular velocity for normalization purposes. Next, participants were asked to perform ballistic reaching to the same outward target but while lifting the arm against various abduction loads emulated by the ACT^{3D}. Specifically, once lifting the arm off of the haptic table, the arm would be supported by the ACT^{3D} with a vertical force (upward or downward), making the limb heavier or lighter than normal. These forces were standardized to percentages of their maximum isometric abduction strength. Five different shoulder abduction loads of 0%, 13.5%, 25%, 37.5% or 50% of maximum shoulder abduction were utilized. In doing so, the impact of loss of independent joint control on reaching function was precisely controlled for the acquisition of muscle activation data. Reaching trials were performed in sets of five trials per abduction loading condition and were randomized for each participant. Mandatory rest of 1 minute was taken between sets to prevent fatigue.

2.5. Data Processing

Data from the ACT^{3D} were analyzed using custom software in the MATLAB environment (Mathworks, Inc.). Movement onset and peak angular velocity were determined for each trial based on position data from the reach trajectories (Figure 2A,B). Reaching trajectories were required to remain within a tolerance cone of $\pm 15^\circ$ (black dotted line in Figure 2A) that was ensured through routine participant coaching. Movement onset was identified as the point when velocity deflected beyond 0.1 rad/s in the positive direction from zero (moving in the direction of elbow extension). The maximum elbow extension angular velocity (red

circle in Figure 2A and red line in Figure 2B) was identified for each of the 5 abduction loading conditions and normalized to the maximum angular velocity of all conditions.

EMG signals were analyzed using custom software in the MATLAB environment (Mathworks, Inc.). After baseline correction, raw EMG signals were rectified and filtered with a 250ms zero-phase moving average window. Data were then normalized to the maximum EMG values obtained during the entire protocol. Mean EMG for elbow flexors were calculated for three distinct time periods based on elbow joint kinematics in order to evaluate contribution of elbow flexor muscle activation to reaching function. The first time-period, labeled as “lift,” was the instantaneous moment 125ms after lifting the arm and acquiring the home position (time 125ms truncated in Figure 2B). The second time-period was labeled “movement onset window” representing the first 25ms of movement just prior to the physiological contribution of stretch reflex that is thought to have spinal loop delay of 30ms (Stein et al., 1995) (green and black lines in Figure 2B). The third time-period was labeled “reflex window” conservatively evaluating contributions of stretch reflex by measuring from the end of the movement onset window up to the instant of peak elbow extension angular velocity (red circle in Figure 2A and red line in Figure 2B). Mean EMG data for elbow extensors were calculated for only two time periods, “lift” and “volitional extension window,” since the extensors were the agonist in this task functioning with a concentric contraction where stretch reflex was not physiologically relevant. The volitional extension window began at movement onset extending to peak elbow extension angular velocity.

Change in mean EMG was then calculated to investigate modulatory effects of abduction loading for each specific time-period. The first time-period remained as the instantaneous EMG at 125ms representing synergy-related activation. Change in mean EMG for the second time-period was calculated by subtracting “movement onset window” from “lift” representing continuation of synergy-related EMG after movement onset but prior to stretch reflex. Finally, change in mean EMG for the third time-period was calculated by subtracting “reflex window” from “movement onset window” representing spasticity-related activation. Change in mean EMG was calculated in similar fashion for the extensors for “lift” and “volitional extension window.”

2.6 Statistical Analysis

All data were analyzed using IBM SPSS Statistics Version 23. Kinematic/EMG data were not normally distributed based on Shapiro-Wilkes test so non-parametric analyses were utilized and medians (interquartile ranges, 25%–75%) are reported throughout. Sample size was calculated from prior reaching velocity data based on reliability coefficients as an estimator of effect size. With a mean reliability coefficient of 0.94 for the reaching velocity variable, an alpha of 0.05, and a beta of 0.10, a sample size of N=6 was required. Sample size was increased, maximizing study limits, and fixed to N=26 to increase generalizability.

Three main hypotheses were tested: 1) elbow extension slows as a function of abduction loading, 2) spasticity-related flexor activation occurs following the onset of reaching movement, 3) synergy-related flexor activation occurs prior to the onset of motion and is modulated by abduction loading. For each of these hypotheses, Friedman tests of differences

among repeated measures were performed. If a significant Chi Square value was found, post hoc Wilcoxon Paired Sample Sign Ranks tests were performed. A family-wise Bonferonni correction was used for all post hoc comparisons.

3. RESULTS

3.1 Elbow Extension Velocity

There was a significant effect of abduction loading on peak elbow extension angular velocity ($\chi^2(4) = 34.20, p < 0.001$) (Figure 3). Median elbow extension angular velocity decreased significantly from the 0% abduction loading condition (0.77 (0.59–0.97)) to the 37.5% abduction loading condition (0.63 (0.47–0.82)) and to the 50% abduction loading condition (0.54 (0.42–0.69)) with p-values of $p=0.001$ and $p=0.01$ respectively. The decrease in velocity at the 25% and 12.5% abduction loading conditions did not reach significance with p-values of $p=0.055$ and $p=0.469$ respectively.

3.2. Flexor EMG

Mean EMG for biceps brachii and brachioradialis were analyzed in the three time periods of lift, movement onset window, and reflex window to determine if an increase in muscle activity occurred from lift to immediately after the onset of movement and then up to peak elbow angular velocity. EMG analysis for the biceps brachii found an effect of time period on muscle activity at the 0% ($\chi^2(2) = 13.23, p=0.001$), 12.5% ($\chi^2(2) = 9.54, p=0.008$), 25% ($\chi^2(2) = 12.08, p=0.002$), and 37.5% ($\chi^2(2) = 9.77, p=0.008$) abduction loading levels (Figure 4A). An effect of time window was not found for the 50% abduction loading condition ($\chi^2(2) = 5.62, p=0.06$). Post hoc comparisons identified more biceps brachii activity in the reflex window than at the lift time point for only the 0% condition ($Z = -2.4, p=0.016$) with median of 0.10 (0.05–0.14) for the reflex window and 0.07 (0.03–0.12) for the lift time point demonstrating an intermediate effect size ($d=0.33$). No significant differences were found between lift and movement onset windows.

Similar to the biceps brachii, there was also an effect of time period on brachioradialis activity at the 0% ($\chi^2(2) = 12.33, p<0.001$), 12.5% ($\chi^2(2) = 29.25, p<0.001$), 25% ($\chi^2(2) = 24.25, p<0.001$), 37.5% ($\chi^2(2) = 16.00, p<0.001$), and 50% ($\chi^2(2) = 17.33, p=0.005$) abduction loading levels. Post hoc comparisons identified more brachioradialis activity in the reflex window compared to the lift time point for all conditions (0%, $Z=-2.457, p=0.014$; 12.5%, $Z=-4.2, p<0.001$; 25%, $Z=-3.629, p<0.001$; 37.5%, $Z=-3.029, p=0.002$; 50%, $Z=-2.857, p=0.004$). Median brachioradialis activity during the reflex window vs. the lift time point was 0.11 (0.07–0.14) vs. 0.08 (0.05–0.11) for the 0% condition, 0.11 (0.08–0.14) vs. 0.06 (0.05–0.11) for the 12.5% condition, 0.12 (0.08–0.20) vs. 0.10 (0.05–0.14) for the 25% condition, 0.13 (0.10–0.20) vs. 0.10 (0.05–0.18) for the 37.5% condition, and 0.11 (0.10–0.20) vs. 0.10 (0.07–0.19) for the 50% condition demonstrating intermediate to large effect sizes respectively (0%, $d=0.34$; 12.5%, $d=0.58$; 25%, $d=0.50$; 37.5%, $d=0.42$; 50%, $d=0.40$). No significant differences were found between lift and movement onset windows.

Change in EMG for biceps brachii and brachioradialis were analyzed in the three time periods of lift, movement onset window, and reflex window to determine if the change in

muscle activity in each condition was modulated by abduction loading. EMG increased for the biceps brachii as a function of abduction loading only during the lift time point ($\chi^2(4) = 45.76$, $p=0.001$) with a median of 0.07 (0.03–0.12) for the 0% condition, 0.09 (0.04–0.17) for the 12.5% condition, 0.15 (0.07–0.30) for the 25% condition, 0.16 (0.06–0.35) for the 37.5% condition, and 0.19 (0.12–0.40) for the 50% condition demonstrating a large effect size ($d=0.59$) (Figure 5). No effect of abduction loading was found for the movement onset or reflex windows.

EMG increased for brachioradialis as a function of abduction loading during the lift time point ($\chi^2(4) = 14.54$, $p=0.006$) with a median of 0.08 (0.05–0.11) for the 0% condition, 0.06 (0.05–0.11) for the 12.5% condition, 0.10 (0.05–0.14) for the 25% condition, 0.10 (0.05–0.18) for the 37.5% condition, and 0.10 (0.07–0.19) for the 50% condition demonstrating an intermediate effect size ($d=0.35$). Similarly, there was an effect in both the movement onset window, ($\chi^2(4) = 9.96$, $p=0.041$) and the reflex window ($\chi^2(4) = 11.58$, $p=0.021$) but with only small to intermediate effect sizes ($d=0.29$ and $d=0.31$ respectively).

3.3 Extensor EMG

Mean EMG for lateral and long head of triceps brachii were analyzed in the two time periods of lift and volitional extension window to determine if an increase in muscle activity occurred from lift (counteracting synergy-related flexor activation) up to peak elbow angular velocity (maximal extension effort). EMG analysis for both the lateral and long head of triceps brachii found a significant increase in muscle activity in the volitional extension window for all abduction loading conditions with Z-score ranges from -3.82 to -4.46 for the lateral head (Figure 4B) and -4.23 to -4.29 for the long head ($p < 0.01$) and with very large effect sizes ranging from $d=0.74$ – 0.88 .

Change in EMG for lateral and long head of triceps brachii were analyzed for the two time periods of lift and volitional extension window to determine if the change in muscle activity during each condition was modulated by abduction loading. Similar to the elbow flexors, EMG increased as a function of abduction loading during the lift time period for both the lateral ($\chi^2(4) = 23.39$, $p < 0.001$) and long head ($\chi^2(4) = 10.08$, $p=0.039$) of triceps brachii. The effect sizes were large and intermediate respectively ($d=0.42$ and $d=0.29$). EMG did not increase as a function of abduction loading during the volitional extension window.

4. DISCUSSION

A robot-mediated kinematic/kinetic reaching paradigm was employed in order to disentangle the contributions of synergy-related and spasticity-related muscle activation to reaching performance. When elevating the arm against an abduction load in preparation for an outward reach, there is a substantial and abnormal activation of elbow flexors that occurs prior to the onset of elbow extension movement in individuals with stroke. The activation of elbow flexors increases as a function of initial abduction loading with only very minimal increases in activity following the onset of movement even though the participants were strongly encouraged to reach ballistically. This spasticity-related flexor activation was very small in comparison to the initial synergy-related activation and mostly occurred in brachioradialis. Spasticity-related activation was not modulated by abduction loading, as

was the synergy-related activation, likely because of the slowing elbow extension velocity as a function of increasing abduction loads. These effects demonstrate a codependency of synergy- and spasticity-related elbow flexor activation on the level of proximal shoulder abduction requirements. For example, relative contributions of both spasticity- and synergy-related activation to reaching can be calculated by a simple quotient of means. Contribution of synergy-related EMG can be represented as the quotient of “lift” over “reflex window” while spasticity-related EMG can be represented as the quotient of change in EMG over total EMG for the “reflex window.” Therefore, for the 0% abduction-loading condition, where the limb was made weightless, movement velocity was fastest with synergy contributing 79% and spasticity contributing 21% of the abnormal biceps activation occurring during elbow extension. This balance shifts substantially at 50% abduction-loading significantly slowing movement velocity with synergy then contributing 96% and spasticity contributing 4% of the abnormal biceps activation occurring during elbow extension. In fact, spasticity-related biceps activity was only found to be significantly contributing at the 0% abduction level.

These data suggest that flexor spasticity is playing only a small role in reaching dysfunction. Its contribution to the impairment of elbow extension movement is rapidly eclipsed by flexor synergy at higher and more functionally meaningful abduction loads. It should then be no surprise that a reduction in clinically assessed spasticity (modified Ashworth test) where the limb is passively supported would not be corroborated by improvements in reaching function that is measured clinically via reach-grasp tasks against gravity (Action Research Arm Test). These data suggest that only when the arm is passively supported during reaching, will spasticity play a meaningful role. In fact, it has been previously reported that during reaching on a frictionless table there is a disruptive hooking in the ballistic reach trajectory due to a rapid stop of elbow extension thought to be from biceps stretch reflex activation (Beer et al., 2004). These effects disappear once reaching is performed against gravity with velocity and excursion being grossly reduced (Beer et al., 2004, Beer et al., 2007). More so, when quantitatively evaluating reaching with a custom rehabilitation robotic device, (Sukal et al., 2005) reaching range of motion progressively diminishes as a function of abduction loading (Sukal et al., 2007) and reflects conventional clinical methods of evaluating arm function (Ellis et al., 2008).

Abnormal elbow flexor activation diminishes reaching speed and range of motion perhaps due to changes in supraspinal motor system drive following stroke (Sukal et al., 2007). In other related conditions such as hemiparetic cerebral palsy, it has been suggested that reaching movement speed is slowed, not due to decreased extensor activation, but instead due to abnormal elbow flexor activation from a supraspinal source (Kukke et al., 2011). Quantitative pioneering work in hemiparetic stroke discovered abnormal co-activation of deltoid with biceps brachii in individuals with stroke and suggested an increased reliance on contralesional corticobulbospinal motor pathways (Dewald et al., 1995). More recent work in individuals with chronic severe stroke employed the asymmetric tonic neck reflex, a developmental reflex that abnormally emerges in brain injury, (Shevell, 2009) implicating the contralesional lateral/medullary corticoreticulospinal motor pathways as the likely mechanism underlying abnormal co-activation of deltoid with distal limb flexors (Ellis et al., 2012). This phylogenetically-older motor pathway is more diffusely branched at the spinal

cord such that direct stimulation of pontomedullary reticular formation in the macaque monkey produces the same shoulder/elbow flexion-synergy patterning observed in humans with stroke (Davidson et al., 2006, Davidson et al., 2007, Herbert et al., 2010).

Other investigators have reported that increased agonist/antagonist co-activation of biceps/triceps about the elbow joint impedes reaching movement however did not measure biceps co-activation with deltoid (Silva et al., 2014). Substantial co-activation at the elbow joint was observed in the present study however it began prior to the onset of elbow extension movement during the lifting portion of the task. The current results emphasize that the primary impairment is not the co-activation of muscles across the elbow joint per se, but instead, the initial abnormal co-activation of deltoid and biceps that precipitates biceps/triceps co-activation with the attempt to hold static and then extend the elbow joint. Others have employed mathematical data reduction approaches such as non-negative matrix factorization to characterize muscle synergies in the paretic arm but were unable to identify abnormal deltoid/biceps co-activation (Roh et al., 2013). While it has been shown that abnormal abduction/elbow flexion torque coupling occurs at even small abduction efforts, (Beer et al., 1999, Dewald et al., 2001a, Ellis et al., 2007) the effect may have gone undetected due to the low force application and small study sample size of 10 participants.

A better understanding of muscle activation impairments following stroke may lead to a refined approach to ameliorating arm function. Attempts to specifically target flexion-synergy in chronic severe stroke have demonstrated improvement in reaching function despite the persistence of spasticity (Ellis et al., 2009). Considering the present results with that of intervention work targeting flexion synergy, future work should investigate the effectiveness of targeting abnormal flexion synergy as opposed to flexor spasticity in the restoration of arm function. Addressing flexor spasticity may best be reserved for palliative approaches.

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Highlights

- Robotic movement analysis disentangles flexion synergy from spasticity during reaching in stroke.
- Flexion synergy eclipses flexor spasticity during reaching with abduction loading in stroke.
- Stroke rehabilitation should target flexion synergy over spasticity to improve arm function.

Figure 1. Setup of a participant in the ACT^{3D} (A). Visual feedback of the arm avatar reaching toward an outward target with a display of the reaching trajectory (B).

Figure 2.

Example position data from a single reaching trajectory under 50% abduction loading (A) with the peak elbow angular velocity labeled with a red circle followed by a single elbow extension angular velocity profile (B) with time 0s (truncated) representing the successful lift and acquisition of the home position target (large green dot in A). The reaching target is indicated with a large black dot whereas the endpoint of the actual reach is labeled with a small black dot. The grey dotted lines represent the accepted range of reaching movements. Movement onset is labeled with a green vertical line while peak angular velocity is labeled

with a red vertical line in (B) and a red circle in (A). EMG data was evaluated at lift (from time 0s to green vertical line), first 25ms of movement onset (between green and black vertical lines), and from the end of movement onset window (black vertical line) to peak angular velocity (red line) in order to determine the EMG contributions of synergy-related flexor activation (lift) and spasticity-related flexor activation (end of movement onset to peak velocity).

Elbow Extension Angular Velocity by Loading Condition

Figure 3. Mean (standard error) elbow angular velocity decreases as a function of abduction loading. The asterisks indicate significant decreases in velocity from the 0% abduction loading condition.

Figure 4.

Mean (standard error) biceps brachii EMG (A) is larger following the onset of elbow extension for all abduction loading levels except the 50% condition representing spasticity-related flexor activation superimposed upon initial synergy-related flexor activation. Asterisks indicate a significant effect of time period. Mean (standard error) lateral head of triceps brachii EMG (B) (shown here for ease of subsequent comparison) is larger following the onset of elbow extension for all abduction loading levels representing consistent maximum volitional drive of extensors during the ballistic reaching task. Asterisks indicate a

significant difference between each individual time period. Change in EMG, while not directly illustrated, can be visually appreciated and was not modulated by abduction loading.

Change in EMG by Time Window

Figure 5.

Mean (standard error) biceps brachii EMG increases as a function of abduction loading when first lifting the arm but prior to the onset of reaching (Lift) representing abnormal progressive synergy-related flexor activation. Mean (standard error) change in biceps brachii EMG in the first 25ms of reaching movement (Onset) and then up to peak elbow extension angular velocity (Reflex) is not modulated by abduction loading.