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Flexion synergy overshadows flexor spasticity during reaching in chronic moderate to severe hemiparetic stroke

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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.

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

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

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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, 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 Hydrocholride (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 and Smith, 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, 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 flexor 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 and Smith, 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

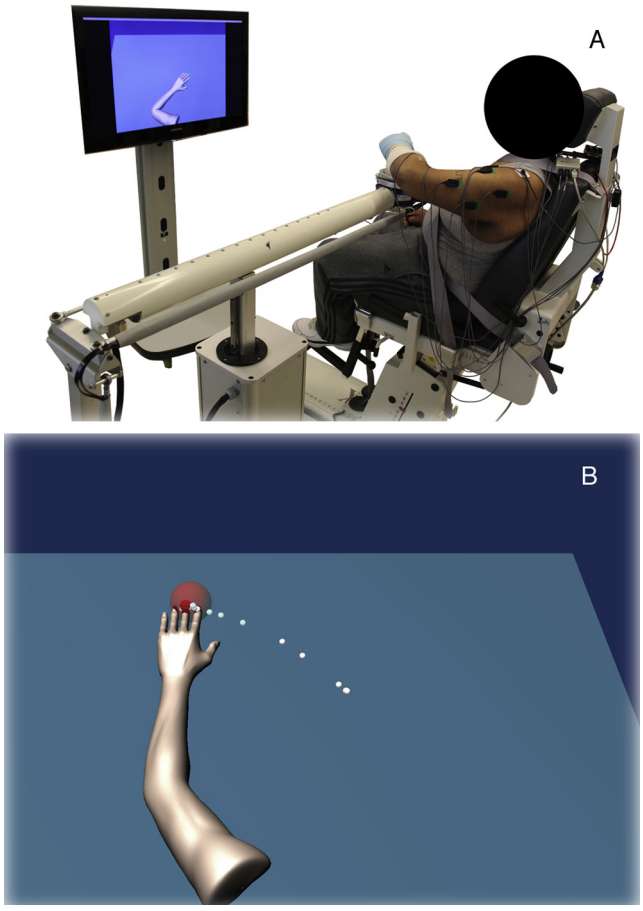

Fig. 1. Setup of a participant in the ACT^{3D} (A). Visual feedback of the arm avatar reaching toward an outward target with a display of the reaching trajectory (B).