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Bilateral impairments in task-dependent modulation of the long-latency stretch reflex following stroke

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HIGHLIGHTS

- Stroke survivors are unable to adapt their long-latency stretch reflex amplitude during tasks that require increased stability.
- Impaired regulation of the long-latency reflex is evident in both the paretic and non-paretic limbs.
- The inability to regulate long-latency stretch reflexes to account for the mechanical properties of the environment may contribute to bilateral deficits in tasks that require proprioceptive feedback and rapid changes in muscle activity to maintain stability.

ABSTRACT

Objective: Modulation of the long-latency reflex (LLR) is important for sensorimotor control during interaction with different mechanical loads. Transcortical pathways usually contribute to LLR modulation, but the integrity of pathways projecting to the paretic and non-paretic arms of stroke survivors is compromised. We hypothesize that disruption of transcortical reflex pathways reduces the capacity for stroke survivors to appropriately regulate the LLR bilaterally.
Methods: Elbow perturbations were applied to the paretic and non-paretic arms of persons with stroke, and the dominant arm of age-matched controls as subjects interacted with Stiff or Compliant environments rendered by a linear actuator. Reflexes were quantified using surface electromyograms, recorded from biceps.
Results: LLR amplitude was significantly larger during interaction with the Compliant load compared to the Stiff load in controls. However, there was no significant change in LLR amplitude for the paretic or non-paretic arm of stroke survivors.
Conclusion: Modulation of the LLR is altered in the paretic and non-paretic arms after stroke.
Significance: Our results are indicative of bilateral sensorimotor impairments following stroke. The inability to regulate the LLR may contribute to bilateral deficits in tasks that require precise control of limb mechanics and stability.

1. Introduction

Cortical stroke disrupts descending motor commands and often interferes with one's ability to stabilize arm postures for various functional tasks such as holding a cup of coffee, driving a car, or walking with a cane. While a healthy motor system is capable of controlling limb muscles in a manner that accurately accounts for the mechanical properties of objects in our environment, this ability is often impaired following stroke. It is well established that the motor system employs flexible control strategies through the development of voluntary motor commands; however, it has recently been suggested that rapid involuntary mechanisms can also compensate for environmental instabilities through regulation of

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stretch reflex sensitivity (Akazawa et al., 1983; Dietz et al., 1994; Doemges and Rack, 1992; Perreault et al., 2008). In particular, long-latency stretch reflexes (LLRs) are modulated to compensate for instabilities in specific directions during the performance of a given task (Krutky et al., 2010). Compensation is achieved by increasing the sensitivity of LLRs in muscles acting to oppose movement in the direction of instability. A number of studies report increases in the amplitude of LLRs during interactions with compliant loads relative to the amplitude of LLRs during interactions with non-compliant, or stiff loads (Dietz et al., 1994; Doemges and Rack, 1992; Perreault et al., 2008). These results suggest that heightened excitability of LLRs may be important for the maintenance of limb stability when the environment does not provide that stability. Understanding how long-latency responses are regulated following stroke may provide additional insight into the neural pathways responsible for impaired motor function during tasks that require increased stability, and to the deficits that can be expected when those pathways are compromised.

Due to the latency and adaptability of the LLR in muscles of the human arm (e.g., biceps, 50–60 ms), it was suggested that these responses are mediated by a transcortical loop (Hammond, 1956). This proposal has since been corroborated by studies in both humans and primates (Cheney and Fetz, 1984; Evarts, 1973; Kimura et al., 2006; Matthews, 1991; Palmer and Ashby, 1992; Pruszynski et al., 2011). Given the involvement of supraspinal pathways, one might expect that cortical lesions could reduce or even eliminate the LLR. Indeed, this has been observed following stroke (Dietz et al., 1991; Hendrie and Lee, 1978; Marsden et al., 1977). Persons with stroke often exhibit delayed voluntary reaction times (Dickstein et al., 1993) and reflex activity some months after stroke (Marsden et al., 1977), suggesting that the circuits responsible for generating the reflex may have been altered and lengthened during the recovery process, resulting in increased processing or transmission delays. Therefore, it also is possible that the LLR in the paretic arm might be delayed if volitional movement and LLRs are mediated through similar neural substrates. This would imply that recovery following stroke involves rerouting of motor and sensory pathways responsible for both volitional movement and postural stability. Our study aimed, in part, to determine if stroke survivors retain the capability for task-specific modulation during this later period, which would help elucidate the efficacy of this rerouting.

Hemispheric stroke often results in bilateral motor deficits in the upper extremity (Desrosiers et al., 1996; Jebsen et al., 1971; Sunderland, 2000). These deficits might be due in part to altered regulation of reflex pathways. There is evidence, for example, that the short latency stretch reflex (SLR) in elbow flexors of the ipsilesional arm is depressed in stroke survivors (Thilmann et al., 1990) and attenuation of the LLR is occasionally observed bilaterally (Marsden et al., 1977). Yet, whether or not stroke survivors retain the ability to modulate LLRs in the ipsilesional arm to account for different physical environments has yet to be determined. If modulation is impaired bilaterally, it could be argued that communication between bilateral brain structures is necessary for the proper regulation of the LLR and efficacy of rerouting after hemispheric stroke. Given the role of the LLR in compensating for environmental instability, impairments of stretch reflex regulation in the ipsilesional upper limb are also likely to have implications for stroke survivors' ability to effectively interact with directionally unstable objects (e.g., single-point canes) during their daily activities.

The purpose of this study was to examine if stroke survivors retain the ability to modulate LLR amplitude in accordance with the level of mechanical stability provided during a postural task. Based on previous demonstrations of stretch reflex modulation in response to changes in the amount of stability provided by the external environment, we hypothesized that elbow perturbations applied in a stiff (stable) environment would elicit smaller long latency stretch reflexes in neurologically healthy individuals than the same perturbations applied in a compliant (less stable) environment. We also hypothesized that identical perturbations of the paretic arm of stroke survivors would elicit long latency reflexes in stiff and compliant environments not different in amplitude. While the flexibility of reflex control in the non-paretic arm is difficult to predict, motor pathways within and descending from the non-lesioned cerebral hemisphere remain intact following stroke, enabling cortical contributions to reflex control in the non-paretic arm to be preserved. We therefore hypothesized that perturbations of the non-paretic arm of stroke survivors would produce long latency reflexes of smaller amplitude in a stiff relative to a compliant environment. The results of this study have implications for understanding how the stabilizing role of feedback mechanisms is altered following stroke.

2. Methods

2.1. Study Population

Experiments were performed on 8 adults with chronic stroke and 8 healthy, age-matched control subjects (Table 1). Ethical approval for the study was received from the Northwestern University Institutional Review Board (IRB protocol STU00009204); informed consent and HIPAA authorization were obtained from subjects prior to their participation. Control subjects had no history of upper limb or neurological impairments. Stroke subjects underwent an evaluation by a licensed physical therapist to determine their eligibility. Stroke survivors were included if they had sustained a unilateral stroke as defined from chart review, had full passive range of motion of tested shoulder and elbow without pain or shoulder subluxation, some voluntary movement of elbow and shoulder, cortical injury resulting in motor deficits of the upper extremity, no receptive aphasia, and the ability to follow verbal and visual commands. All subjects with stroke had at least mild spasticity in elbow muscles as defined by a cumulative Ashworth score of greater than or equal to 1. Subjects were excluded if they had history of unilateral neglect (spatial and motor), inability to provide informed consent, and/or significant medical complications. We recorded Fugl-Meyer scores (Fugl-Meyer et al., 1975) as a reliable clinical measure of arm motor impairment (Duncan et al., 1983).

2.2. Equipment

Subjects were seated comfortably with their trunk secured to an adjustable chair (Biodex, Shirley, NY) using padded chest and lap straps. The target arm (paretic or non-paretic arm of stroke subjects; dominant arm of control subjects) was positioned in the horizontal plane with the shoulder at 70° of abduction and 0° of flexion, the elbow joint at 90°, and the forearm slightly pronated (Fig. 1A and B). The upper arm was placed in a height-adjustable trough support to ensure a constant position of the shoulder joint. A fitted orthotic splint extending from the fingers to the middle of the forearm was used to maintain the wrist joint in a neutral position and to attach the forearm to a linear actuator (Copley Thrust-Tube TB3806; Copley Controls, Canton, MA). A 10 cm steel plate located on the underside of the cast, centered at the wrist joint, was secured to the top surface of the actuator via a precision bearing that allowed rotation in the horizontal plane.

The linear actuator was used to apply elbow extension perturbations in two different mechanical environments (Compliant and Stiff). The actuator was mounted on an adjustable aluminum frame and was oriented such that perturbations were applied in