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Contributions of motoneuron hyperexcitability to clinical spasticity in hemispheric stroke survivors

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HIGHLIGHTS

- Hyperexcitability of motoneurons is one potential contributing mechanism towards muscle spasticity in stroke.
- We quantified the reflex latency of single motor units evoked from a precisely controlled tendon tap on the biceps muscle, as an estimate of motoneuron hyperexcitability.
- The latency of the unitary discharge was systematically shorter in the spastic muscle compared with the contralateral muscle.

ABSTRACT

Objective: Muscle spasticity is one of the major impairments that limits recovery in hemispheric stroke survivors. One potential contributing mechanism is hyperexcitability of motoneurons. Previously, the response latency of the surface electromyogram (EMG) record evoked by joint rotation has been used to characterize motoneuron excitability. Given the limitations of this method, the objective of the current study was to reexamine the excitability of motoneurons in chronic stroke survivors by estimating reflex latency using single motor unit discharge.
Methods: We quantified the excitability of spastic motoneurons using the response latency of a single motor unit discharge elicited by a position controlled tap on the biceps brachii tendon. We applied tendon taps of different amplitudes on the biceps tendons of both arms of the stroke survivors. Unitary reflex responses were recorded using intramuscular EMG recordings.
Results: Our results showed that the latency of unitary discharge was systematically shorter in the spastic muscle compared with the contralateral muscle, and this effect was consistent across multiple tap amplitudes.
Conclusions: This method allowed us to quantify latencies more accurately, potentially enabling a more rigorous analysis of contributing mechanisms.
Significance: The findings provide evidence supporting a contribution of hyperexcitable motoneurons to muscle spasticity.

1. Introduction

Spasticity, affecting up to 43% of chronic stroke survivors (Wissel et al., 2013), is diagnosed clinically as muscular hypertonia, coupled with other reflex disturbances. This hypertonia is defined as a velocity-dependent resistance to stretching due to exaggerated reflex responses (Lance, 1980). There are also concurrent mechanical changes of the muscular–tendon complex, which also contribute to increased muscle tone. Although spasticity can sometimes be beneficial for certain functional movements (e.g., making locomotion and body weight support possible), it is still a major neurological impairment that frequently limits motor functions of many stroke survivors. For example, it can lead to abnormal muscle activation patterns and to disabling body and joint postures in both upper and lower extremities (Knutsson et al., 1979, 1980;

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Finley et al., 2008; Trumbower et al., 2010). These postures, called contractures, can hinder normal functional output and induce disability in daily activities.

Spasticity arises primarily because of increased tonic stretch reflex responses (hyperreflexia), but it can also trigger changes in the mechanical properties of the muscle and connective tissues, as quantified by an increase in the mechanical stiffness of the spastic muscle (Dietz et al., 1981, 2007; Lee et al., 1987; O'Dwyer et al., 1996). Independently, hyperexcitability of the reflex arc, manifested by an increased stretch reflex response has also been recognized as one major contributor to hypertonia (Gottlieb et al., 1978; Powers et al., 1988, 1989; Dietz et al., 2007). One possible mechanism that can contribute to the overall reflex response is increased motoneuron excitability (Katz et al., 1989). This is the focus of our current study.

One standard test of motoneuron excitability is mediated through the evaluation of stretch reflex or H-reflex latency, often combined with measurements of the ratio between the maximum H-reflex and M-wave magnitudes, where a shorter latency and a larger (H/M) ratio represent signs of more excitable motoneurons. However, there have been inconsistent observations regarding the actual reflex latency in the spastic muscles of stroke survivors. In several reports, stretching the soleus muscle via transient ankle joint rotations, or using electrical stimulation of the posterior tibial nerve, studies have reported that both the stretch reflex latency and H-reflex latency were shorter in the spastic muscle compared with the contralateral one (Hui-Chan et al., 1993; Levin et al., 1993; Bakheit et al., 2003). In contrast, others have observed that the H-reflex latency in the spastic gastrocnemius muscle was not different from the contralateral one (Pisano et al., 2000; Bakheit et al., 2005).

These inconsistent findings may arise in part from unreliable estimates of reflex latency from the surface electromyogram (EMG). For example, the surface EMG is typically used to record the reflex responses, but the rise time of the reflex response is typically slow, due to progressive recruitment of different motor units, many with very small size. As a result, the reflex latency is highly sensitive to the reflex onset criterion, and this criterion is often set differently in different studies. Additionally, the non-selectivity of the stimulus input may also bias the latency estimates. During either joint rotations or nerve stimulations, multiple non-targeted muscles are activated inevitably (Perry, 1993), and the reciprocal excitatory and inhibitory projections between muscles may further bias the estimates of motoneuron excitability of the targeted muscle. Therefore, these approaches may provide inaccurate estimates of the physiological status of the spastic spinal motoneuron.

To overcome these limitations, we estimated the reflex latency of single motor unit discharges elicited by precisely controlled tendon taps, delivered to a single muscle. Specifically, we examined the reflex latency in passive spastic biceps brachii muscle, and we compared it with the contralateral muscle of chronic stroke survivors. We applied tendon taps with amplitudes that were small enough (i.e., 0.5, 1, and 2 mm) to only elicit single motor unit discharges. These single motor unit recordings provided a very sharp rise time of the unitary reflex response, and allowed us to derive a highly reliable estimate of the reflex latency. The specificity of the single muscle stimulation using a precisely controlled tapper ensured the consistency of the stimulus input, and eliminated the potential activation of non-targeted muscles.

Using this technique, we compared the reflex latency differences between the spastic and contralateral biceps of ten stroke survivors. Our results showed that the reflex latency in the spastic muscle was significantly shorter compared with the contralateral muscle in seven stroke subjects, and this latency difference was reversed in two stroke subjects. It was not significantly different in one subject. The findings provide evidence for the existence of hyperexcitable motoneurons, as one of the potential neural mechanisms that can contribute to spasticity in stroke survivors.

The relevance of these latency observations to our understanding of the mechanisms of spasticity will also be discussed. In particular, the relative contributions of sustained depolarization of hyperexcitable motoneurons, as compared with enhancement of the size or rise time of stretch-evoked excitatory postsynaptic potentials (EPSPs) will be explored. The findings provide evidence and a potential tool to identify the reflex contributions to spasticity, which can further inform decision making for spasticity management, including physical or pharmacological therapies, botulinum toxin injections, or surgery. Additionally, our results further revealed limitations of current clinical assessment techniques (e.g., Modified Ashworth Scores), that are unable to distinguish neural (e.g., hyperreflexia) from mechanical contributions (e.g., contracture) of muscle hypertonia. More quantitative assessment approaches are needed for better diagnosis and clinical decision making.

2. Methods

2.1. Participants

Ten chronic hemispheric stroke survivors (8 male, 2 female) volunteered to participate in this study. Inclusion criteria for the stroke subjects were: spasticity present in the upper extremity (Modified Ashworth Scale $\geqslant 1$), stroke onset longer than 6 months, medically stable, no concurrent medical illnesses, no significant cardiorespiratory, metabolic, orthopedic, or other neurological disease, and no history of multiple or recurrent vascular episodes. The demographic profiles of the stroke subjects are summarized in Table 1. The participants gave informed consent via protocols approved by the Institutional Review Board under the Office for the Protection of Human Subjects at Northwestern University.

A research physical therapist performed the clinical evaluation prior to the experimental testing. This included:

1. Spasticity: an assessment of spasticity at the elbow, using the Modified Ashworth Scale (Bohannon et al., 1987), and an estimate of the magnitude of the biceps deep tendon reflex, elicited with a clinical hammer, using a 4 point scale (Walker, 1990). The lower boundary for inclusion of the stroke subjects was a Modified Ashworth score of 1 and a deep tendon reflex score of 2+.
2. Motor impairment: the physical therapist further assessed the upper arm impairment with the Fugl-Meyer Assessment Scale (Fugl-Meyer et al., 1975) and the Chedoke–McMaster Stroke Assessment Scale (Gowland et al., 1993).

3. Experimental setup

3.1. Linear motor for tendon tap

Participants were seated upright in a Biodex chair with their shoulder placed in 45° of abduction and neutral rotation, with the elbow in 120° of extension, and the wrist in 45° of supination and 0° of flexion/extension. The forearm was cast from just below the elbow to the most distal point of the finger and attached to a ring-mount interface to standardize arm position, and minimize activity of muscles. A position controlled linear actuator (Linmot Inc.), as shown in Fig. 1A, was positioned perpendicular to the biceps tendon. To ensure that the probe was placed consistently in contact with the tendon across testing sessions, the anatomical location of the muscular–tendon junction was marked on the skin guided by the ultrasound imaging (Supersonic Imagine Inc.) of the