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Stretch-reflex threshold modulation during active elbow movements in post-stroke survivors with spasticity

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HIGHLIGHTS

- Tonic stretch-reflex thresholds in post-stroke spasticity occurred within the joint range at rest.
- Threshold modulation during active movements was related to clinical spasticity and motor impairment.
- Characteristics of threshold modulation provide information about post-stroke sensorimotor deficits.

ABSTRACT

Objectives: Voluntary movements post-stroke are affected by abnormal muscle activation due to exaggerated stretch reflexes (SRs). We examined the ability of post-stroke subjects to regulate SRs in spastic muscles.
Methods: Elbow flexor and extensor EMGs and joint angle were recorded in 13 subjects with chronic post-stroke spasticity. Muscles were either stretched passively (relaxed arm) or actively (antagonist contraction) at different velocities. Velocity-dependent SR thresholds were defined as angles where stretched muscle EMG exceeded 3SDs of baseline. Sensitivity of SRs to stretch velocity was defined as μ. The regression through thresholds was interpolated to zero velocity to obtain the tonic SR threshold (TSRT) angle.
Results: Compared to passive stretches, TSRTs during active motion occurred at longer muscle lengths (i.e., increased in flexors and decreased in extensors by 10–40°). Values of μ increased by 1.5–4.0. Changes in flexor TSRTs during active compared to passive stretches were correlated with clinical spasticity ($r = -0.68$) and arm motor impairment ($r = 0.81$).
Conclusions: Spasticity thresholds measured at rest were modulated during active movement. Arm motor impairments were related to the ability to modulate SR thresholds between the two states rather than to passive-state values.
Significance: Relationship between spasticity and movement disorders may be explained by deficits in SR threshold range of regulation and modifiability, representing a measure of stroke-related sensorimotor deficits.

1. Introduction

Spasticity is a common complication of stroke, occurring in ~20–50% of patients in the first year (Wissel et al., 2013) and often associated with other sensory and motor impairments (e.g., muscle weakness, loss of dexterity). Spasticity is generally assessed by resistance or EMG responses to passive muscle stretches and has been attributed to exaggerated spinal stretch reflexes (SRs) and alterations in intrinsic muscle properties (Dietz and Sinkjaer, 2007). For example, motor units of spastic muscles often have an impaired ability to relax (Lewek et al., 2007), prolonged spontaneous firing (Mottram et al., 2010) and low firing rates (Young and Mayer, 1982; Gemperline et al., 1995). Neural mechanisms

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underlying spasticity include deficits in the regulation of inhibitory reflex pathways (e.g., reciprocal Ia inhibition, presynaptic inhibition) and hyper-excitability of α-motoneurons (MNs; Nielsen et al., 2007).

A causal relationship may exist between spasticity and limitations in performance of daily activities (Pandyan et al., 2005). However, clear understanding of the relationship between spasticity and movement deficits remains elusive (O'Dwyer et al., 1996; Mirbagheri et al., 2001; Dietz and Sinkjaer, 2007). This situation might be changed by considering spasticity and movement production within the same conceptual framework such as the *threshold position control theory* (an extension of the Equilibrium-point hypothesis). The theory is based on findings that voluntary muscle activation originates from central shifts in the spatial stretch-reflex threshold (SRT), defined as the muscle length (or corresponding joint angle) at which muscle activity emerges in response to stretch (Feldman, 2015; Raptis et al., 2010; Fig. 1). The SRT is velocity-dependent (dynamic SRT or DSRT; e.g., Powers et al., 1989), such that the length at which the muscle begins to be activated decreases with increasing stretch velocity (see Eq. (1)). DSRT has a velocity-independent component called the tonic stretch reflex threshold (TSRT; e.g., Matthews, 1959). It has been shown that central changes in SRTs underlie voluntary movements in humans (Asatryan and Feldman, 1965). By shifting TSRT, the nervous system pre-determines the spatial (angular) range in which the muscle can generate active forces (Fig.1A). Shifts in TSRT can be accomplished by different descending systems (vestibulo-, reticulo- cortico- and rubro-spinal) that mediate influences on α-MNs mono- and/or poly-synaptically as well as pre-synaptically or via γ-MNs (Feldman and Orlovsky, 1972; Capaday, 1995).

Within this framework, spasticity can be understood as an impaired ability to increase SRTs in affected muscles to prevent active responses to passive muscle stretching (Powers et al., 1988, 1989; Levin and Feldman, 1994; Musampa et al., 2007; Fig. 1B, right panel), as occurs in healthy subjects. However, the framework also implies that in addition to muscle relaxation, SRT modulation also underlies the control of voluntary movements. In particular, the muscle that is inactive at a given length, can be activated by shifting the threshold below this length. Therefore, a reduced capacity to modulate SRTs might contribute to movement disorders in spastic patients (Jones and Yang, 1994; Sinkjær et al., 1996; Faist, 1999; Morita, 2001; Burne, 2005). In contrast, procedures facilitating SR modulation can improve motor function, as observed in chronic spinal cord injury (Manella et al., 2013). The possibility remains that SRTs are also modulated during movements in spastic muscles and limitations in this capacity may be related to movement disorders.

We measured DSRTs from which we computed TSRTs in spastic muscles at rest (i.e. when patients were instructed to completely relax arm muscles) and determined whether and how the thresholds (TSRTs) were modified during active movement. Since movement production depends on the capacity to modulate TSRTs, we assumed that in patients who can produce elbow movements, some residual ability to regulate TSRTs in spastic muscles would be retained (Morita, 2001). Three hypotheses were tested: (1) TSRTs determined at rest identify the joint angle at which spasticity begins to be manifested; (2) active movement influences TSRTs at which spasticity is manifested, and (3) differences in TSRTs between passive and active movement will be related to clinical movement deficits.

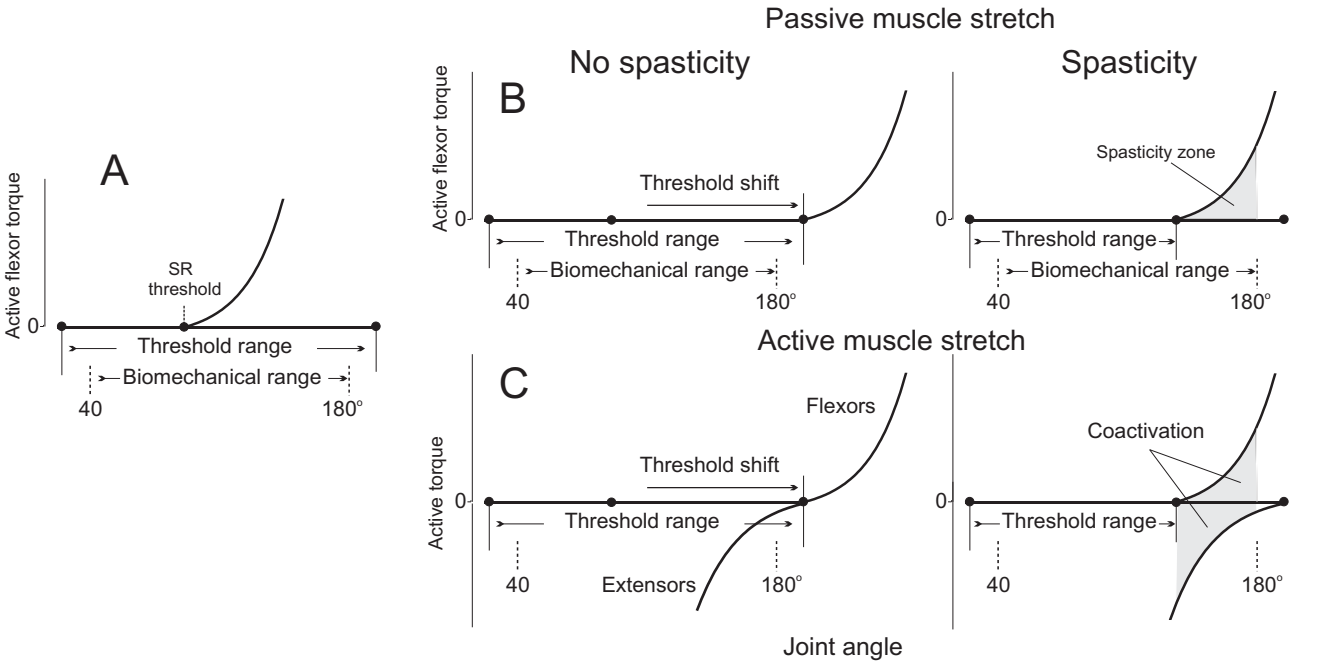

Fig. 1. Threshold position control (schematic). (A) Activity and torque is generated when the muscle length exceeds the velocity-dependent threshold muscle length also called the dynamic spatial stretch-reflex threshold (DSRT). Static (tonic) muscle activity and torque increase with the increasing difference between the actual and the tonic stretch reflex threshold (TSRT). Panel A shows that, in healthy individuals, the range of central TSRT regulation exceeds the biomechanical limits of the joint. (B) Left: To prevent flexor activation during passive stretching (full muscle relaxation during elbow extension), healthy subjects shift TSRT to the right, beyond the upper biomechanical joint limit. Right: In contrast, due to a deficit in central inhibitory mechanisms, TSRT is not shifted far enough and occurs abnormally within the biomechanical range, resulting in spasticity beyond the TSRT angle (spasticity zone, shaded area), i.e., patients cannot relax (de-activate) spastic muscles stretched beyond certain muscle lengths, even at low velocities. Left: Active stretching of flexors is produced when subjects intentionally extend the joint. This is achieved by facilitating extensor and de-facilitating/inhibiting flexor motoneurons, shifting flexor and extensor TSRTs to the right. Right: Extensor muscles are activated to overcome spastic flexor resistance during active extension into the spasticity zone.