Mitigating disruption to voluntary movement caused by velocity-dependent stretch reflex via α-MN collateral projection to γ-MN: A simulation study.

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Background: Muscle spindle afferent signals contribute to the proprioceptive feedback signals that inform the CNS about the position and movement of the body [1]; And, thus, are thought to be important for kinesthesia, posture, and balance control [1-4]. However, their function during voluntary movement is not fully understood [5]. One such fundamental input is velocity-dependent stretch reflexes (i.e., Ia afferents) in lengthening (antagonist) muscles, which are thought to be inhibited by reciprocal inhibition from the shortening (agonist) muscles [6]; if not regulated or inhibited, the reflex feedback produces neural resistance to the muscle stretch that can disrupt or stop joint rotations induced by the shortening muscles and compromise movement accuracy [7]. It remains an open question, however, the extent to which velocity-dependent stretch reflexes disrupt voluntary movement, and whether and how they should be inhibited in limbs with numerous mono- and multi-articular muscles where agonist and antagonist roles become unclear and can switch during a movement. In this work, we investigate (i) how velocity-dependent stretch reflexes perturb limb movements in the general case of numerous multi-articular muscles, and (ii) whether spinal modulation of velocity-dependent stretch reflex gains mitigate these disruptions.

Methods: We address these long-standing fundamental questions using 3D movements against gravity in a 25-muscle computational model of a Rhesus Macaque arm. After simulating 1,100 distinct movements across the workspace of the arm with feedforward α-MN commands, we computed the kinematic disruptions to the arm endpoint trajectories caused by adding positive homonymous muscle velocity feedback (i.e., simple velocity-dependent stretch reflexes) at different static gains to the feedforward α-MN drive (without reciprocal inhibition).

Results: We found that arm endpoint trajectories were disrupted in surprisingly movement-specific, typically large, and variable ways, and could even change movement direction as the reflex gain increased. In contrast, these disruptions became small at all reflex gains when the velocity-dependent stretch reflexes were simply scaled by the α-MN drive to each muscle (equivalent to an α-MN excitatory collateral [8-10] to its homologous γ-MNs but distinct from α −γ co-activation).

Conclusion: We argue this α-MN collateral projection to γ-MN circuitry is more neuroanatomically tenable, generalizable, and scalable than α − γ co-activation and movement-specific reciprocal inhibition. In fact, we propose that this mechanism at the homonymous propriospinal level could be a critical low-level enabler of learning via cerebellar and cortical mechanisms by locally and automatically regulating the highly nonlinear neuro-musculo-skeletal mechanics of the limb. This propriospinal mechanism also provides a powerful paradigm that may begin to clarify how dysregulation of γ-MN drive can result in disruptions of voluntary movement in neurological conditions [5].

References:

[1] Santuz & Akay, 2022; [2] Proske & Gandevia, 2011; [3] Proske & Gandevia, 2012; [4]S trzalkowski et al., 2018; [5] Krakauer et al., 2022; [6] Pierrot-Deseilligny & Burke,2005; [7] Valero-

Cuevas, 2016;[8] Wilson & Burgess, 1962; [9] Bhumbra & Beato, 2018; [10] Beato & Bhumbra, 2022